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The Long Term Impact of the 1918 Influenza Pandemic in Norway

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Abstract

A growing literature show the negative effect of prenatal health shocks on childhood and adult outcomes. Several studies exploit disease outbreaks to find causal effects of *in utero* exposure on various outcomes. We build on the existing literature by applying theories of *in utero* health effects to Norwegian data. This thesis uses the 1918 Spanish Influenza pandemic as a natural experiment to investigate the impact of prenatal health shock on various long term outcomes in Norway. Influenza is considered a negative shock to the intrauterine environment, and has been shown to lower both cognitive and non-cognitive abilities. We use unique historical data provided by Statistics Norway that contains information on the influenza mortality and morbidity rate, the number of still births and population estimates from 1912 to 1919. In addition, we have collected data from the Statistical Bulletin which provides information on tax income in all municipalities. This data is matched to the 1960 census, allowing us to estimate effects on year of education, marital status, number of children and employment status. We find that exposure to prenatal influenza leads to a reduction in years of education for men. This effect is twice as large for the poorest municipalities in Norway, indicating that negative shocks are more severe for the lower socioeconomic groups. Since early intervention is far more beneficial for both social and economic outcomes, than interventions later in life, measures to prevent these serious implications are relatively cheap and easily attainable, and should be offered to all pregnant women.

Acknowledgements

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1 Introduction

Facilitating a good and worthy life through health measures, education and institutional quality is important both for the individual and for society as a whole. It is essential that we increase our understanding for the mechanisms of what forms us. A large and growing literature presents evidence of the positive effects of early childhood intervention, for instance in reducing crime, increasing high school completion rates and improving adult health [Campbell et al., 2014]. The aim of this paper is to investigate the effects of *in utero* exposure to influenza on long term outcomes. More specifically: *"How does prenatal exposure to influenza impact years of education, marital status, number of children and years of education in Norway?"*

The technology of skill formation, which is described by Cunha and Heckman [2007], shows that ability gaps between individuals occur early in life, and that early intervention is much more beneficial and cost effective than treatment in adolescent years. Disadvantaged children in particular stand to benefit from early intervention. Furthermore, the return from intervention mechanisms varies across different periods of childhood, and that interventions made in adolescent years have a far higher return if preceded by intervention early in life as well. The basis for future health and cognitive ability is laid already in the womb [Barker, 1997]. Several papers both in the medical and the economic literature investigate the impact of disease and malnutrition during pregnancy, documenting sensitive and critical periods in the development of human capabilities (see Almond and Currie [2011] for an overview). Some found that exposure in the first trimester lead to a higher rate of miscarriages [Bloom-Feshbach et al., 2011], while others found negative effects on long-term outcomes when the fetus was exposed to negative health shocks in the second trimester [Almond, 2006; Almond and Mazumder, 2005; Black et al., 2013; Kelly, 2010]. Currie and Schwandt [2013] found an increase in preterm births for

exposure in the third trimester.

The link between fetal development and outcomes later in life is often referred to as the the fetal origins hypothesis. It states that severe conditions of adult health, such as heart disease and type 2 diabetes, could be triggered by a range of various environmental effects *in utero* [Barker, 1992]. Smoking, poor nutrition and alcohol have negative long term effects for the child, including poor cognitive and non cognitive abilities, low birth weight, lower education and lower income [Almond and Currie, 2011]. The exact effect of the various health shocks is hard to test, especially in the long term, because factors such as genes, socioeconomic status, parental education and neighbourhood environment also impact future outcomes. It is therefore a huge challenge to separate the effect of a negative shock *in utero* from the other correlating factors.

Economists have used natural experiments in order to separate the effect of biological and social factors from the actual effect of the intrauterine environment. Natural experiments have the potential to hit all levels of society in equal measure, thus providing an excellent opportunity to look at the average effect of prenatal exposure. Examples of previous research include the effect of radioactive fallout [Black et al., 2013; Nyagu et al., 2002], famine [Stein, 1975], negative income shock [Banerjee et al., 2010] and disease outbreaks [Almond, 2006; Almond and Mazumder, 2005; Kelly, 2010; Richter and Robling, 2013].

In recent years, a larger focus has been made on the consequences of prenatal exposure to influenza. Douglas Almond [2006] investigated the effect of the Spanish flu using US census data. He found that the cohort exposed the pandemic *in utero* were 4-5 per cent less likely to finish high school than the surrounding cohorts. Similarly, Kelly [2010] uses UK data from the Asian flu to examine the effect of in utero exposure of flu on birth weight and cognitive development for children up to

the age of 11. She had access to more detailed data and were able to pin point the in utero effect of the flu to more specific time periods of the pregnancy. She found that exposure to the flu during the 2nd trimester only had an effect on birth weight if the mother smoked or were of low height. In contrast she found that exposure did have an effect on cognitive development of the child regardless of the health of the mother. We conduct a similar study using Norwegian data to see how exposure of influenza during the Spanish Influenza pandemic in Norway affected the return to education for individuals. To the best of our knowledge, a similar study has not been done in Norway before.

Given previous literature and knowledge on the medical consequences of *in utero* exposure to maternal health shocks, we expect to find negative effects in cognitive abilities, such as years of schooling, if the mother is exposed to influenza in the second trimester. We do not necessarily expect to find negative effects in the first trimester, as there is a risk of culling of the weakest.[1] Effects in the third trimester have been found to have the largest effect on physical measures at birth, such as birth weight [Kelly, 2010]. Since we do not have data for birth weight, we will not be able to investigate this. Further, exposure toward the end of the pregnancy lead to an increased risk of preterm birth. As we are investigating events in the early 20th century, the chance of surviving a preterm birth was low. We may therefore have a strong selection bias both for the first and third trimester.

We consider several long term outcomes including years of education, marital status, number of children and working status. We find that exposure to influenza leads to fewer years of education for men, an effect that is stronger for the lower socioeconomic groups. For women we find that exposure to influenza reduces the chance of being married. We collected unique data from Statistics Norway on the number of registered flu cases from 1912 to 1918, and combine this with the 1960 census. An

[1] Culling: Higher mortality among the weakest, and only the strongest survive

important and necessary feature of our data is that we know the month and municipality of birth for all individuals in the census, thus enabling us to retrace the effect of the prevailing health conditions in the local medical authority they were born in. In our analysis we are able to couple the month of birth with any of the nine months prior to the individuals birth. We can then seek out effects of *in utero* exposure to influenza specifically for each month of the pregnancy and for each trimester.

The impact of prenatal exposure to influenza has important policy implications. The long term effects of *in utero* exposure may lead to lower education and higher probability of work disabilities for the children of the exposed mother [Almond, 2006; Kelly, 2010; Schwandt, 2014]. Further, Richter and Robling [2013] found strong intergenerational effects of influenza in Sweden. They found lower education for the grandchildren of those exposed to the 1918 Spanish flu, so the effect of exposure seems to be persistent. Given the lasting impact of insults to the intrauterine environment, it is therefore important to restrict the chance of getting influenza while pregnant through vaccination.[2] This is important not only for the individual, but also for society as a whole. Karlsson et al. [2013] found increased poverty rates and negative effects on capital return in Sweden following the 1918 influenza pandemic. Pregnant women are more susceptible to influenza than the general population [Schwandt, 2014], so vaccination for pregnant women is already quite common, and is offered for free to those who wish to get immunized. However, if the consequences are as severe as some studies suggest, immunization should be even more widespread.

This paper is organized as follows: Section 2 presents a summary of the most important characteristics of the 1918/1919 influenza pandemic and an overview of the Fetal Origin Hypothesis. Section 3 presents existing literature on the effect of maternal exposure to influenza and other health shocks *in utero*. Section 4 presents

[2]Influenza vaccination has no known side effects for pregnant women [Rasmussen et al., 2008].

the data, some of which is collected by ourselves through non-digitalized reports provided by Statistics Norway. In Section 5, we proceed to present the empirical approach, including the theoretical framework and the specification of the model we use in our regression analyses. In Section 6 we present the findings from our analyses. Finally, we discuss our findings in Section 8.

2 Background

2.1 The Spanish Influenza Pandemic

The Spanish Influenza pandemic hit worldwide between May and December 1918, the most prominent and deadly period being in the later half of 1918.[3] Updated estimates indicate that over 500 million people were infected, and that 50 - 100 million died globally [Johnson and Mueller, 2002]. The flu mortality rate seems to vary from region to region. For instance, in the in the United States, it is believed that around 28 per cent of the population were infected during the pandemic [Jordan, 1927], but only a 0.5 per cent died [Almond, 2006]. Compared with estimates provided by Johnson and Mueller [2002] this a relatively low death rate. The 1918 Influenza pandemic was more deadly than regular seasonal strands of influenza because it was an H1N1 virus which attacked the lungs as well as the bronchus, leading to many people dying of pneumonia as a consequence of the infection [Morens and Fauci,

[3]The exact origin of the virus remains unknown today. The virus had an outbreak in the United States in Kansas in January 1918. It is believed that the virus later reached Europe through American soldiers who were sent to Europe during the later part of World War I. The first known cases of the Spanish flu in Europe were in Bordeaux and Brest in France in April 1918, both in military camps [Patterson and Pyle, 1991]. The virus is named the 'Spanish Flu' as result of the first official reports of the disease originating from Spain. Participating countries in WW1 were trying to hide and censor the fact that a threatening decease were spreading among their troops. Spain, however, were not a part of the war and reported about the decease before any other countries. It would then seem to origin from Spain, and the name 'Spanish flu' stuck.

2007].

The characteristics of the 1918 Spanish flu pandemic deviate from the regular seasonal flu in several ways. In addition to a devastating impact on people with a weaker immune system, which is mostly young children and the elderly, a disproportionate number of the infected where in the age group 15-44 [Brainerd and Siegler, 2002]. In fact, nearly half of the fatalities were adults between 20 and 40 years old [Simonsen et al., 1998], giving the age distribution of flu fatalities an unusual W-shape instead of the U-shape expected to be found during a regular flu season. This means that the pandemic hit women of childbearing age just as hard as the elderly and the young. Pregnant women are more exposed to the influenza than other groups, and it is estimated that approximately 25 per cent of pregnant women were infected by the Spanish flu [Richter and Robling, 2013].[4] This feature makes the pandemic an ideal natural experiment to estimate the effects of *in utero* exposure to influenza.

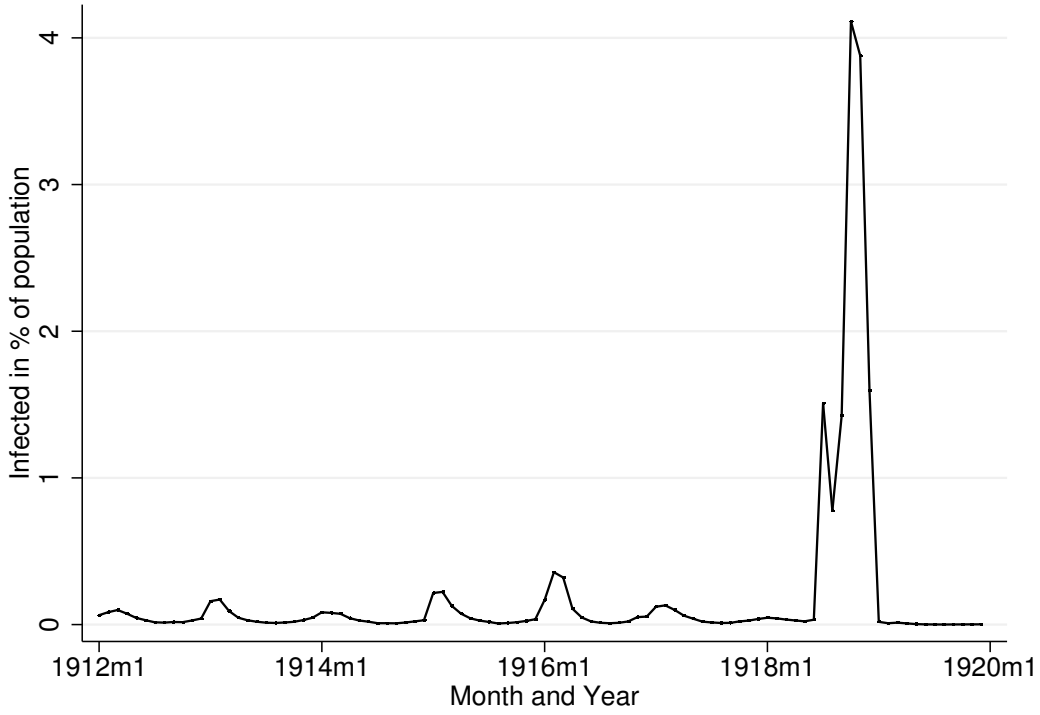
Norway, like most other countries, was hit hard by the pandemic. The Spanish flu hit Norway in three main epidemics. The first one in June and July in 1918, often referred to as the 'summer epidemic'. The two latter epidemics hit in late august, the 'autumn epidemic', and in the winter of 1918-1919, the 'winter epidemic'. The autumn epidemic was by far the worst with morbidity rates up to 21 per cent in one month. In total, about 500 000 people were infected over the course of the three epidemics, out of which approximately 7400 died.[5] In 1918, the Norwegian population was approximately 2,6 million [Statistics Norway, 1922a].

Figure 1 presents the monthly average share of influenza cases across local medical districts from 1912-1919. The share of influenza prior to the pandemic is significantly lower than in 1918. There are also clear signs of seasonality, with one clear spike in

[4] Almond [2006] found a 30 per cent infection rate among women of childbearing age in the US.

[5] Mamelund [1998] estimated 1 million infected persons and 13 00-15 000 deaths in Norway. These numbers also approximates deaths that is indirectly caused by influenza, such as pneumonia.

Figure 1: Influenza morbidity rate by month and year from 1912 to 1918 in Norway



Note: Monthly data for 1919 and 1920 are missing

Source: Statistics Norway medical rapports, 1912-1918

influenza cases each year. The spike in 1918 clearly shows how much larger and more significant the number of infected persons were during the Spanish flu compared to regular seasonal flu. The two main epidemics in 1918 are clearly noticeable. Unfortunately, monthly data for 1919 is missing from the Statistics Norway medical reports, so the infection rate from the last wave is not included in the figure.

2.2 The Fetal Origins Hypothesis

The Fetal Origin Hypothesis postulates that negative health shocks during pregnancy have a negative impact on the health of the fetus later in life. A growing field of study investigate the long term impact of various factors that impact health later

in life. Barker [1992] is given much of the credit for formulating the 'Fetal Origins Hypothesis' late in the 1980's, saying that during critical periods of the pregnancy, lack of nutrients or oxygen will affect the development of the various organs of the fetus. Examples of negative shocks are maternal influenza or pneumonia, starvation and poor maternal nutrition in general. 'Programming' of the fetus affects both the physiology and the metabolism of the fetus, and may result in non-insulin dependent diabetes and cardiovascular disease later in life [Barker, 1997]. This hypothesis has been expanded to include specific consequences of negative shocks to the intrauterine environment. Barker [1997] found that exposure in the first trimester might lead to increased risk of hemorrhagic stroke because of raised blood pressure, exposure in the second trimester increases the risk of coronary heart disease via insulin resistance, and for exposure in the third trimester he finds increased risk of both coronary heart disease and stroke. These are all health conditions that are not apparent until later in life, so exclusively looking at various factors at birth may therefore not be sufficient to map the full effect of prenatal exposure. Further, these consequences increase the risk of dying at a young age.

Research has shown that the human brain is most vulnerable between 8 and 25 weeks in gestation [Tau and Peterson, 2009]. Brain development can further be grouped into two critical periods, the first from 8 to 15 weeks in gestation, and the other from 16 to 25 weeks in gestation [Otake and Schull, 1998]. From weeks 8 to 15 in gestation the increase of nerve cells is at its highest. Otake and Schull [1998] found that this was the period with strongest effect on cognitive abilities. This finding is supported by Eriksen et al. [2009] who used Norwegian register data and intelligence scores from the military, and found lower intelligence scores among men born after the Hong Kong Flu.[6] They found that the strongest impact is apparent if exposure

[6]The Hong Kong Flu originated in China in 1968 and was the third influenza pandemic in the 20th century. The Pandemic killed one to four million people world wide [Encyclopedia Britannica, 2016]

to influenza happened in the third to fourth month of pregnancy. The creation of the synapses is the most active during the second period, from weeks 16 to 25 [Tau and Peterson, 2009]. Schwandt [2014] found a decline in labour force participation if the mother is exposed in the second trimester. Some have found that exposure in the second trimester leads to higher susceptibility to schizophrenia, but this finding is disputed [Ebert and Kotler, 2005].

The model of capital formation, described by Heckman [2007], puts forth the idea that 'capabilities beget capabilities', and is well suited to study the fetal origins hypothesis. This means that there is a snowball effect both for positive and negative events early in life. Heckman [2007] states that "the capability formation process is governed by a multistage technology". Based on this, as well as several other factors, he describes a constant elasticity of substitution production function to produce later outcomes, such as health, cognitive skills etc. The simplest model includes two inputs; investments *in utero* and investments later in life:

$$H_{adult} = A\Big[\gamma I^{\varphi}_{prenatal} + (1-\gamma) I^{1-\varphi}_{postnatal}\Big]^{1/\varphi} \tag{1}$$

where H is health outcomes and I are health inputs. γ and φ show the relationship between the input variables.

An important feature of the model is that "capital formation is *dynamic complimentary*", which implies that capabilities that are produced early in life, help raise the productivity of investment later in life. This means that those who are disadvantaged at birth or in early childhood are more likely to suffer more later in life due to a negative shock or lack of positive stimuli in early childhood. Differences in health at birth due to differences in fetal conditions may intensify over time.

3 Literature Review

The literature on early development is extensive and views many aspects of the theory. We will restrict our focus to fetal health, the consequence of low birth weight and the consequences of negative shocks to the intrauterine environment which follows an influenza infection.

Negative physical and cognitive effects of health shocks *in utero* are of interest to economists as well as to medical professionals.Birth weight in particular has been shown to be an important indicator for future outcomes. A large body of literature has found that children with low birth weight have poorer health, cognitive deficits and more behavioural problems than children with higher birth weight [Case et al., 2005; Currie and Hyson, 1999]. However, because of correlations between birth weight, parental income, socioeconomic status and genetic variables, a causal relation effect of birth weight is challenging to determine. Using a twin-study, Black et al. [2007] found that a 10 per cent increase in birth weight increase earnings by 0.9 per cent, high school graduation by 1.2 per cent and IQ of men by 1.2 per cent. Despite the seemingly causal link between birth weight and later outcomes, not all conditions are reflected in the birth weight. Some incidents are relevant for later outcomes but will not influence birth weight, while others affect birth weight but are not relevant for outcomes later in life [Rasmussen, 2001]. This is in line with research by Stein [1975], who found that exposure to famine late in the pregnancy lead to low birth weight but does not affect health later in life. However, starvation early in pregnancy lead to high birth weight, but poorer health later in life. The timing of the negative shocks therefore seems to be important.

A challenge when investigating long term outcomes of the intrauterine environment is separating the correlation between factors such as parental education and their children's outcomes. A groundbreaking study by Almond [2006] used the 1918

influenza pandemic as a natural experiment for testing the fetal origins hypothesis. Almond used U.S. Census micro data identifying state of birth from 1917 to 1919 and samples of the 1960 and 1970 censuses to investigate various adult outcomes, and found large negative effects of exposure to influenza *in utero*. By comparing the surrounding birth cohorts to those born in the year after the pandemic, he found that the 1919 cohort are 4-5 per cent less likely to complete high school than the trend would predict. Further, he found that men in the 1919 birth cohort where 5 per cent more likely to have a work-limiting physical disability and 8 per cent more likely to have a work-preventing disability. A critical assumption by Almond [2006] is that of random selection, as the influenza pandemic seemingly hit all socioeconomic groups and geographical areas independent of each other. This assumption was challenged by Brown and Thomas [2011] who claimed that due to World War I, the men remaining in the U.S. were, on average, less able than the men who fought in the war. They found that those who were exposed *in utero* belonged to a lower socioeconomic group, their fathers had a lower income, were less likely to be white and less likely to have been a WWI veteran. Using the same data as Almond but controlling for socioeconomic status (SES), Brown and Thomas [2011] find smaller and insignificant effects of *in utero* exposure to influenza.[7]

Following the paper by Almond [2006], other researchers have investigated the short and long term effects of influenza exposure for pregnant women. Kelly [2010] examines the consequence of the Asian Influenza Pandemic in 1957 on childhood development.[8] She used the National Child Development Study (NCDS) in Great Britain which followed 17 400 individuals born in march 1958. Most of the cohort were therefore in their second trimester when the Asian Flu hit Britain between

[7]The critique by Brown and Thomas (2011) should not apply in the Norwegian context as Norway was neutral in WWI. No Norwegian men were recruited during the war and as a consequence there were no military fatalities. However, approximately 2000 men died at sea during WW1.

[8]Asian Influenza was a pandemic of influenza originating in China in 1957 and was the second influenza pandemic in the 20th century. An estimated one to two million people died of the H2N2 strand worldwide.

September and November in 1957, and therefore *in utero* in the second trimester, which is the most important period of gestation for development of the brain [Nyagu et al., 2002]. Kelly [2010] explores the effects of influenza exposure in the second trimester on childhood outcomes. She investigated the various effects captured by birth weight, and how maternal health can limit the effects of influenza. She used a linear regression model with the number of registered pneumonia cases in each local authority as explanatory variable, and controls for family background through mothers characteristics. Kelly [2010] found that the epidemic has a negative effect on birth weight, but only if the mother is short of height and smoked during pregnancy. In contrast to the physical results, the epidemic had negative effect on the test score regardless of the mothers health. Furthermore, Richter and Robling [2013] found strong intergenerational effects of *in utero* exposure to influenza. They found that potential maternal exposure reduces educational attainment for the offspring by 2.4 months. Richter and Robling [2013] did control for the number of children in each family, but the results of are nonetheless indicative of serious and long lasting implications for prenatal exposure.

A large literature examines the relationship between season of birth, health and socioeconomic outcomes. Some indicate a connection between the season of birth and the socioeconomic background of the mother. Currie and Schwandt [2013] established that mother fixed effects eliminates selection into conception month, and found that seasonal effects were still present. Further, they found shorter gestation length for the children exposed to influenza late in the pregnancy independent of mothers' socioeconomic background. Schwandt [2014] investigated the effect of *in utero* exposure to seasonal influenza, controlling for mother fixed effects. He had access to very detailed Danish birth registry data, enabling him to identify when in the pregnancy influenza exposure happens. He found shorter gestation lengths for cohorts exposed close to full term. Moreover, Schwandt [2014] found that *in*

utero exposure in the second trimester greatly decreases to probability of being in the labour force.

4 Data

Our primary source of data is the Medical Statistical Reports from Statistics Norway for the years 1912 to 1920. For the analysis of long term outcomes, we link this information to the 1960 Norwegian Census. Further, we use information from the Bulletin of Statistics Norway for information on number of tax payers and the amount of tax paid in each municipality. The Medical Statistical Reports and the Statistical Bulletin are available only as pdf-scans of the original rapports. In order to use the data in our analysis, it was necessary to digitally transfer the data to a spreadsheet manually. We include data for cohorts born between 1913 and 1919 in Norway and who were still alive in 1960. Consequently, all those who have died in accidents, of illness or during World War II are not in the sample. Those born outside Norway are not included in the analysis because our identification strategy depends on knowing the municipality of birth.

One challenge with the data, is that local medical districts have changed structure over the years. In 1912, there were far less local medical districts than in 1920. For the vast majorities of cases, one medical district has been divided in to two or more smaller ones by 1920. By collapsing the data by 1912 medical districts, we ensure that all data are linked to the correct geographic entity. The data is thus specified according to the 1912 level of detail, giving us 173 different medical districts throughout the entire country.

Medical Statistical Reports

The Medical Statistical Reports provides information on influenza morbidity and

mortality rates and share of still born babies, as well as population numbers. The morbidity rates are available for every month in each local medical authority, enabling us to follow the influenza outbreak month by month. For the years 1919 and 1920, data for each month is only available at country level, but not on a medical authority level. Since influenza was not a reportable disease in the early 1900s, there is some uncertainty regarding the accuracy of the reported incidents. Consequently, we do not know if the morbidity rate reported represents the total number of cases or just a few cases. For instance, some local authorities report the same number of deaths from influenza as the total number that are infected from the flu, while others only report a 1 per cent mortality rate. This is indicative of a reported lower bound morbidity rate. We therefore collected data for deaths associated with influenza, as they may serve as a better indicator for the frequency of cases of flu. The deaths are reported for each local authority, but are only available on a yearly basis. Deaths are mandatory to report along with cause of death, and for that reason it may serve as a good proxy to provide more accurate numbers for cases of influenza and measles.

The Medical Statistical Reports also provide data on the number of still and live births for each local medical authority on a yearly basis. We collected this data to see if there were any indications of culling. The number for still and live births have some uncertainty related to them, as the definition of a still birth may vary across medical districts. Some report a still birth if the baby lived for five minutes after birth, while others would report it as a live birth. There is also the question of when a birth is counted as a spontaneous abortion and when it is a premature birth. Despite this ambiguity, the number of deaths caused by influenza remain a less noisy indicator of the magnitude of influenza in each municipality. Doctors in each medical authority will not change their practice in such a time span, and the differences should therefore be controlled for when controlling for municipality fixed

effects. We use still births as an outcome variable and see if we find evidence of higher mortality among those exposed to influenza *in utero*.

Statistical Bulletin

Data for tax income is available for the years 1915 and 1920 for each municipality, and includes information on the number of tax payers and the total tax paid. From this we are able to calculate the tax paid per worker and use this as an indicator for the level of skilled labour in each medical district. Unfortunately, we do not have data in the medical reports regarding the parental education for cohorts born from from 1912 to 1920. Since we are unable to control for the level of education of the parents, we use the tax information as a proxy variable for SES of the medical district.

1960 Census

The 1960 census provides us with a range of outcome variables on an individual level. We have data with 364,504 observations of individuals born between 1900 and 1922. Cohorts born before 1912 and after 1920 are dropped from our sample, so our final sample consists of 153,877 observations. The variables of interest are years of education, marital status, working status and number of children. Summary statistics for all variables are provided in Table 1. Up until 1969 it was compulsory to complete seven years of primary education in Norway, and higher education was available to both boys and girls. However, it was more common for boys to enroll in additional education, while most girls settled for the compulsory seven years. Marital status is a categorical variable and provides information whether the individual is single, married, divorced or widowed. In order to simplify our analysis we generate a dummy variable equal to 1 if a person has ever been married, and 0 otherwise. Working status indicates whether the individual is actively working or unemployed.

Table 1: Summary Statistics: The 1960 Census

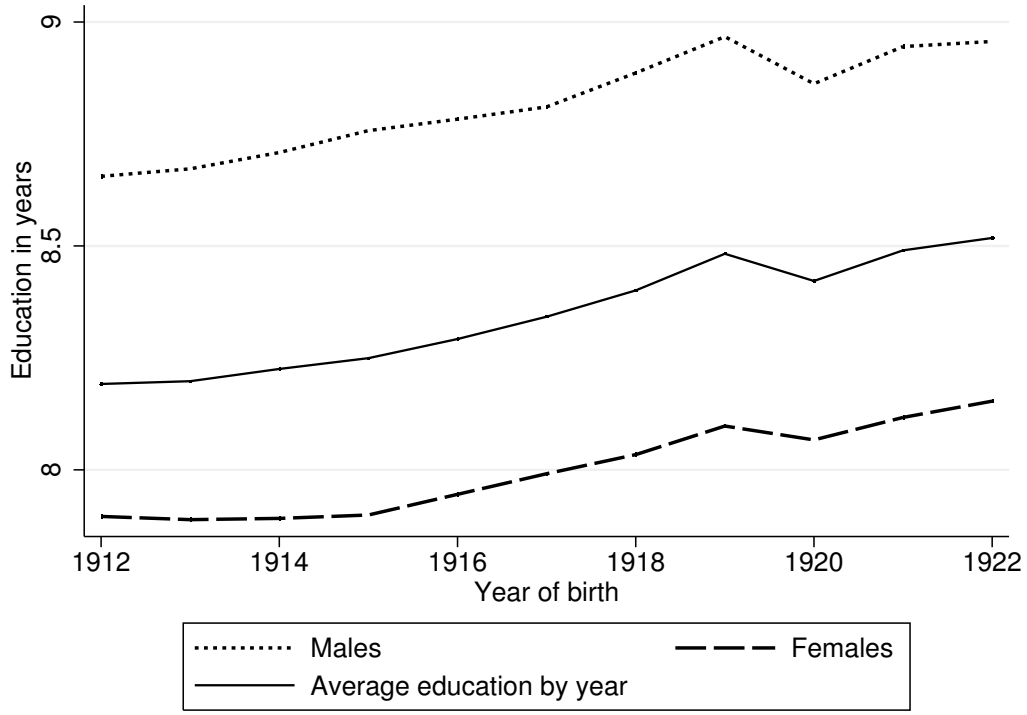
	Mean	Standard deviation	Count	Min	Max
Years of Education:					
Men	8.81	2.66	63113	7	18
Women	7.97	1.72	87404	7	18
Number of Children	2.32	1.61	73284	0	19
Working Status:					
Men	0.99	0.01	29275	0	1
Women	0.57	0.49	6294	0	1
Marital Status	0.89	0.31	153877	0	1
Observations	153877				

Note: The sample include all cohorts born between 1913 to 1919. Data for education is provided in years of completed education.

Education is quoted in years of schooling, from primary school to higher level education. Figure 2 presents the average education per year for both men and women, and additionally the average years of education irrespective of sex. The trend is positive for both sexes, but men have consistently more education than women. The trends also move in approximately the same upward sloping direction, indicating that both sexes are exposed to similar effects regarding years of education. The graph shows a decline in average years of education for men, and slightly less for women, for those born in 1920. The drop in education is interesting, but it is beyond the scope of this thesis to identify the cause of it. The drop should not cause problems for our analysis, since we are able to control for year specific effects.

Figure 4 shows the fertility of women born 1912-1922. The trend shows a steady increase in number of children up until 1919, where the trends turns to be negative. Even though the decline starts in 1919, which is the year of birth for the majority of the Spanish flu cohort, the trend continues to be negative after 1919 within the time range of our data. It seems likely that the decline is caused by other factors than only the maternal flu exposure. From a descriptive point of view it is hard to say if maternal exposure to the Spanish flu causes any effect in either direction, but we will

Figure 2: Average education by year for individuals born between 1912 to 1922 in Norway, separated for males and females



Source: 1960 census data

be able to test whether the reduction in fertility differs by the treatment intensity in our empirical analysis. Fertility is an interesting measure as it captures both the health and the social status of the mother. In the article "An Economic Analysis of Fertility", Gary S. Becker argues that fewer children per mother yields higher quality children in terms of socioeconomic status [Becker, 1960]. Moreover, he argues that having fewer children is an attribute of parents with higher socioeconomic status as they chose a "quality over quantity" approach. We might then expect to find a correlation between maternal exposure to influenza and number of children later in life. More specifically, we should expect the correlation to be positive, so higher share of influenza leads to having more children on average.

Working status specifies whether the individual is currently working or if he or she

Figure 3: Average number of children by year for individuals in the 1960 census born between 1912 and 1922 in Norway

Source: 1960 census data

is unemployed. For a majority of the individuals in our data, the working status was either stated as undisclosed or it were not stated at all. Thus, the average share of employed given in table 1 only consists from the individuals where the working status is given as either 'employed' or 'not employed'. If the working status is disclosed randomly, so that the smaller selection is representative for the average population, we do not have a problem. However, 99 per cent of all men reported that they are employed, indicating a bias in our data. Estimates indicating effect on employment status should therefore be considered with caution.

Marital status is a dummy variable indicating 1 if a person is married, divorced or widowed, and 0 if a person is single. Table 1 shows that 89 per cent of the population is or has been married. From an economic point of view, marital patterns has

important implications with respect to population growth through the number of children, labour force participation and inequality of income. Becker [1973] argues that marriage can be analysed as any other commodity because of two principles. First, that marriage is voluntary (either from the person itself or their parents) so that the theory of preferences can be applied. Second, seeking a partner happens in a well functioning market where all participants want to find the best mate. Becker [1973] further states that "positive assortive mating is the most common and applies to IQ, education, height, attractiveness and other characteristics". Based on this, lower IQ and fewer years of education as well as other undefined capabilities affect the probability of finding a partner. Brandt and Siow [2008] find that exposure to famine in China reduces the marital attractiveness of a person, possibly due to lower intelligence. Given the theory regarding marriage, we expect to find a negative effect on marital status among those who were exposed to prenatal influenza.

A problematic feature of the 1960 census is that the individuals have to be alive in 1960 to be part of the data set. Only people born between 1912 and 1920 who were still alive in 1960 are included our data set. We risk to losing valuable data concerning those who died in the period between when they were born and 1960. If those who died were of significantly lower health, and this correlates with being exposed maternally to influenza, a "selection effect" could lead to an upward bias in our estimates.

Further, better general health conditions may lead to negative scarring effect. "Scarring" is the effect of the infant and childhood environment on survivors of disease. Improved general health lead to less scarring and improved long term outcomes [Bozzoli et al., 2009]. A decrease in infant mortality may follow from an upswing in the general health environment. That is, lower infant mortality is associated with better general health, thus leading to the health of the survivors being better. Hatton [2011] found that the scarring effect was stronger than the selection effect in the

early 20th century.

5 Empirical Approach

5.1 Theoretical Framework

In order to estimate the effect of maternal influenza on various long term outcomes, we take advantage of our panel data structure and use standard ordinary least squares (OLS) regressions. The OLS computes the estimates which minimise the sum of squared residuals and will provide us with estimates of the effect of a change the independent variables *ceteris paribus* [Wooldridge, 2014]. The Spanish Influenza pandemic is considered a natural experiment so it should work well as our identification strategy. There are several reasons for the suitability of the Spanish flu as an identifications strategy. First, the pandemic was an unexpected and severe health shock. Second, it lasted for a very short period of time, enabling us to compare the treated to the untreated with very little noise due to time difference. Third, the pandemic was socially neutral, thus the estimated the results are less confounded by the SES of the parents.[9] Fourth, unusually many women of child bearing age were infected with the virus. Consequently, the impact should be large enough to be captured in our analysis.

OLS

We use ordinary least squares (OLS) to estimate the effect of maternal influenza. In order to get a causal interpretation we need unbiased estimates, which can only be retrieved if the model is linear in parameters, consists of a random sample, and

[9]Mamelund [2006] found that the mortality rate was higher for those born in the poorer areas of Oslo. This finding potentially confounds results using mortality data, but causes less of a concern when morbidity data is used in the analysis.

if the error term is unbiased and homoskedastic.[10]

A vital criteria for unbiased estimates is the the zero conditional mean assumption. This condition states that the error term must be normally distributed with a mean of zero, $E[\varepsilon_{it} \mid x_{it}] = 0$, so the average of any unobserved factor in the model must be equal to zero for every value of the independent variables. In other words, the error term, which consists of all the variables not specified in the model, must not correlate with any of the regressors. If the error term is correlated with the outcome variable and at least one of the independent variables, we will get biased estimates often referred to as omitted variable bias. This would result in either an exaggerated or an understated coefficient estimate, and we would not be able to trust the results of the regression. Unfortunately, there is no certain way of knowing the extent of the omitted variable bias, nor can we be sure what potentially drives the bias. Omitted variable bias can be restricted by including a set of observable control variables that potentially correlate with the regressors. By adding such variables to the model, we take them out of the error term, ε_{it}, so that the zero conditional mean assumption is more likely to hold [Wooldridge, 2010]. However, if the bias in the error term stems from unobserved factors, we will not be able to control for them. Methods of removing the unobserved bias from the error term will be discussed in section on fixed effects.

Another potential confounding factor in the estimates is selection bias. In regression analysis, we look at the average effect of treatment. Since we cannot see the outcome of both assigning and not assigning treatment on the same person, we use averages assuming that the average of a random sample provides representative estimates given treatment to an average person [Angrist and Pischke, 2008]. The assumption of random sampling is therefore vital for unbiased estimates. This implies that if some individuals can choose to be in either the treatment or the control group, the

[10]Homoskedasticity is achieved by using the robust command in STATA

estimation suffers from selection bias. Selection bias can be illustrated using the potential outcome framework where Y_{0i} is the outcome if i is not treated, Y_{1i} is the outcome if i is treated, and D is a dummy =1 for treatment.

$$\underbrace{E[Y_{1i}|D_i = 1] - E[Y_{0i}|D_i = 1]}_{\text{Average causal effect}} = \underbrace{E[Y_{1i}|D_i = 1] - E[Y_{0i}|D_i = 0]}_{\text{Average treatment effect}} - \underbrace{E[Y_{0i}|D_i = 1] - E[Y_{0i}|D_i = 0]}_{\text{Selection bias}}$$

True random sampling will remove the selection bias, and provide us with results we can trust [Angrist and Pischke, 2008]. Since we use the Spanish flu as our identification strategy we must assume that the flu hit the population randomly and that it did not distinguish between poor and rich. If, for instance, lower socioeconomic groups had a higher morbidity rate than the higher socioeconomic groups, the estimated results would be biased by the family background for each individual and not just the pure effect of the influenza epidemic. This selection bias could potentially alter the estimated results significantly, even change the sign of the coefficient.

Fixed Effects

Our data set contains both cross sectional units i, and a time dimension, t. This panel data structure allows us to control for time invariant unobservable factors in the error term. The error term, ϵ_{it}, can be divided in to two types of unobserved factors, an idiosyncratic error term and an unobserved time constant fixed effect. Mathematically, its shown as

$$\epsilon_{it} = a_i + u_{it}$$

u_{it} is the idiosyncratic error term, and varies both across panels and time. The idiosyncratic error term must be normally distributed with zero mean, and be uncorrelated with any independent variable in all time periods. In other words, it must be i.i.d.[11] a_i is the unobserved time constant fixed effect and captures unobserved differences across units, but remains constant over time. Since the unobserved fixed omitted variables remains unchanged over time, we can eliminate it from our model. This is done by obtaining *time-demeaned data*. A general example of a model is presented by Wooldridge [2014],

$$Y_{it} = \beta_1 x_{it} + a_i + u_{it}$$

where Y_{it} is the outcome variable, x_{it} is the independent variable, a_i is the unobserved fixed effect and u_{it} is the idiosyncratic error term. If we average the equation for each i over time, we obtain:

$$\bar{Y}_i = \beta_1 \bar{x}_i + a_i + \bar{u}_i$$

a_i is fixed over time, so it remains constant in both equations. To obtain the time demeaned data we simply subtract the mean from all observations.

$$Y_{it} - \bar{Y}_i = \beta_1 (x_{it} - \bar{x}_i)(a_i - a_i) + u_{it} - \bar{u}_i$$

The fixed effect, a_i, disappears from the model, and we are left with the time-demeaned data

$$\ddot{Y}_{it} = \beta_1 \ddot{x}_{it} + \ddot{u}_{it}$$

When we time-demean data, we lose one degree of freedom (df) since we subtract one

[11] Independent and identically distributed.

time-period from the data. This rather small issue is removed if dummy regression is used, rather than the fixed effects option. Dummy variable regression includes a dummy for each municipality and for each time period, thus controlling for fixed effects for each i and t. This approach creates many independent variables (one for each observation and time period), and is considered the most accurate way of controlling for unobserved fixed effects [Wooldridge, 2014].

By further including an interaction term with the cross sectional and time period dummies, the model captures, in our case, municipality specific time trends. The estimated coefficients is then the deviations from an underlying secular trend [Wooldridge, 2010].

Clustering

When estimating the effects of an aggregate explanatory variable on individual-specific dependent variables, a downward bias can occur due to a possible unobserved state-level effect in the error term [Wooldridge, 2003]. For instance, the error term in a community one year is very like to be correlated with the error term for the same community next year.

$$\begin{aligned} Y_{gm} &= \alpha + \beta x_g + \gamma z_{gm} + \nu_{gm} \\ m &= 1, ..., M_g \\ g &= 1, ..., G \end{aligned}$$

where g indexes the group and m is the index for observations within a group. The problem arises in the error term which can be written

$$\nu_{gm} = c_g + u_{gm} \qquad m = 1, ...M_g$$

where c_g is an unobserved group effect. This correlation problem can be solved by clustering on group level. Clustering allows for correlation within each group, but not between. Thus, any potential problem with serial correlation within each municipality is dealt with. Failing to cluster could potentially sharply increase standard errors.

5.2 Our Model

To measure the long-run effects of prenatal exposure to influenza on education, employment, marital status and the number of children per woman, we exploit the magnitude of the Spanish flu and the randomness with which it hit the population. We use a similar approach as Kelly [2010], but we have observations over a longer period of time.

We estimate the following model:

$$Y_{it} = \alpha + \beta_1 Flu_{it} + \gamma_t + \lambda_i + \eta_{iy} + \varepsilon_{it} \tag{2}$$

where Y_{it} represents our outcome variables years of education, marital status, number of children and working status for individuals born in municipality i at time t. Flu_{it} is the prevalence of influenza in the medical district of birth. We investigate the effects for *in utero* exposure in all months of the pregnancy and for each trimester. We choose to focus on exposure 8-25 weeks (2-6 months) in gestation in our primary specification due to the evidence provided by the medical literature which suggests that this is the most crucial period for the development of the brain.

γ_t is a set of year dummies controlling for cohort fixed effects and λ_i is a set of municipality dummies controlling for municipality fixed effects. Thus ordinary time

shocks are controlled for by the year fixed effect and municipality specific unobserved factors are controlled for by the municipality fixed effect (see section 5.1 about fixed effects). In addition, we include an interaction term with the municipality dummies and the year dummies in order to allow for seasonal factors that differ by area. Thus we identify the deviation from the underlying trend of the variables.

β_1 is our variable of interest and measures the causal effect of influenza on future outcomes as long as influenza exposure is orthogonal to other factors potentially affecting the outcomes of the children. A potentail source of selection bias is parental SES. Currie and Schwandt [2013] found that women from different socioeconomic groups give birth at different times of the year. However, in 1912 there was no reliable birth control method available making it harder to time the season of birth, than for mothers in later years. Controlling for season of birth or mother fixed effects should therefore not be necessary in our analysis.[12] Further, the Spanish flu hit randomly for all socioeconomic groups, so SES should not be a source of bias in the error term.

Because of the short duration of the pandemic, we are able to compare people born within months of each other, but who has experienced very different *in utero* environments. The randomness and duration of the pandemic seriously limits the omitted variable bias in our analysis, so much that we claim the zero conditional mean assumption holds.

Our sample includes both men and women, but as there is little variation in the number of years of schooling and employment status for women born in the early 1900s, we choose to separate our analysis by sex. Further, number of children is registered per woman so it makes little sense to include this outcome variable in the analysis for men.

[12]This was tested by Richter and Robling [2013] who investigated the effects of Spanish flu in Sweden. They found no indication of seasonal effects driving their results.

6 Results

6.1 Main specification

In following section present the results from our analyses. First, we present the results from the main specification, where each month of maternal exposure to influenza is regressed separately and all municipalities are included in the sample. Further, we investigate potential heterogeneity effects. We divide the sample between the rich and poor municipalities to see if there are different effects among the socioeconomic groups. We also display estimates for rural and urban areas separately. For all regressions, we separate the sample by sex.

Table 2 presents the estimated impacts of maternal influenza exposure on education, marital status, employment and the number of children per woman in 1960 using our main specification (see equation 2). The analysis is conducted for each month of exposure, and each cell in the table comes from a separate regression and represents the coefficient of share of flu in each municipality. Robust standard errors are clustered by municipality, allowing for arbitrary correlations of the error terms for those born in the same municipality. All regressions include a full set of cohort and municipality specific fixed effects, in addition to an interaction term with all municipality and year dummies. We report months 2-6 because these are the months proven to be of most importance by the medical literature.[13]

In table 2, columns 1 and 2 present completed education in years. Column 3 and 4 present the dummy variable 'married' and is the probability of ever having been married in 1960, including those who are married, divorced and widowed. Column 5 shows the number of children per woman, and columns 6 and 7 show working

[13]For an overview of regressions results for all months in gestation, see tables A1 and table A2 in the appendix.

status in 1960. 'Employed' is a dummy and equals 1 if the person if employed and 0 otherwise. 'Married', 'No. of Children' and 'Employed' are reported per 100. Each variable is separated by sex, except number of children where we only have data for women. It is important to note that 'Pregnancy month' is the term we use from the *assumed* beginning of the pregnancy given a normal 9 month gestation. However, we do not know the gestation length, so the months are counted backwards from the time of birth. Consequently, the results show exposure in the months before birth.

Table 2: Long term outcomes using the full sample

Month after assumed conception	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.022	0.007	-0.109	-0.150	-0.701	0.005	0.639
	(0.015)	(0.006)	(0.163)	(0.102)	(0.636)	(0.004)	(0.720)
Pregnancy month 3	0.002	0.002	0.281**	-0.208*	-0.563	0.005	-0.700
	(0.012)	(0.007)	(0.121)	(0.120)	(0.590)	(0.006)	(0.740)
Pregnancy month 4	0.007	0.012	0.015	-0.045	0.068	0.005	-0.554
	(0.015)	(0.008)	(0.119)	(0.105)	(0.490)	(0.004)	(0.744)
Pregnancy month 5	-0.021**	0.001	-0.140	-0.111	0.083	0.007	-0.703
	(0.010)	(0.006)	(0.117)	(0.096)	(0.553)	(0.005)	(0.676)
Pregnancy month 6	0.005	-0.001	0.105	-0.029	0.230	0.006	-0.947
	(0.009)	(0.005)	(0.101)	(0.109)	(0.562)	(0.0045)	(0.814)
Observations	75281	102223	76946	104569	34786	7307	85940

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born from 1913 to 1919. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month before birth. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Education

We find that exposure to influenza in 4 months before birth (pregnancy month 5) has a significant negative effect on years of education on a 5% significance level, but only for men. The lack of significant effects for women may be explained by the lack of variation in the sample. Women at the time only had basic education, and only a very few took higher education. The magnitude of the coefficient for years of education for men suggest that a 1% point increase in the share of influenza morbidity reduces the total years of education with approximately 1 week (0.02 years). However, this is an intention-to-treat effect of pregnant women who were exposed to the flu. That is, the effect we find is the average effect on *the whole* population, whereas only a part of the pregnant population where exposed to influenza. Hence, to get the full treatment effect on the treated, we would need to divide our results by 0.25.[14] When accounting for the estimated 25% infection rate among pregnant women, we find that *in utero* exposure to influenza 4 months before birth leads to a reduction in years of education by 1 month. This reduction in completed years of education following prenatal exposure to influenza is in line with the findings by Schwandt [2014] and Almond [2006], although our estimates are smaller.[15] One possible explanation for this is the selection bias in our data. As previously discussed, we only have information on individuals still alive in 1960, so the most severely affected individuals are not in our sample. If exposure to maternal influenza is indeed associated with worse health later in life, we should expect to see a higher mortality at a young age for those exposed to influenza *in utero*. The surviving population will on average have better health compared to the entire cohort.

The medical literature indicate negative effects of prenatal exposure 2-6 months be-

[14]Richter and Robling [2013] estimate that approximately 25% of women of childbearing age in Sweden were infected. Norway and Sweden are very similar countries, so assuming the same morbidity rates among Norwegian women is logical.

[15]The estimates found by Almond [2006] are larger than the rest of the existing literature. One possible explanation is the bias due to WWI pointed out by Brown and Thomas [2011].

fore birth, however we do not find negative effects of exposure for all these months. There could be several reasons for the insignificant, and sometimes positive, coefficient estimates in our analysis. In the first trimester, maternal exposure to influenza may lead to and increased risk of miscarriage [Bloom-Feshbach et al., 2011]. This culling of the weakest may be the source of a severe selection bias. The group of survivors performs better and have better health *ceteris paribus*, so the effect of maternal exposure to influenza is seemingly positive. Similar arguments apply to exposure toward the end of the pregnancy. Maternal exposure to influenza late in the pregnancy increase the probability for premature birth [Currie and Schwandt, 2013]. Premature birth in 1918 where associated with substantially worse health and higher mortality, thus leading to a selection bias through mitigation of the weakest children. Indications of culling after exposure in the first trimester, and higher mortality rate after exposure in the third trimester can be seen in table A1 in the appendix, where we find *positive* and significant estimates for exposure to influenza.

Marital Status and Number of Children

If exposed to influenza *in utero*, we expect to find a negative effect on the share of individuals who got married. Marital attractiveness could be lower due to worse health and education [Brandt and Siow, 2008]. We find a small negative effect for women who were exposed to influenza 6 months prior to birth. Given a 1% point increase in influenza morbidity 6 months before birth (pregnancy month 3), women are on average 0.2% less likely to get married at a 10% significance level. Adjusting for intent-to-treat-effects, women are 0.8% less likely to get married if exposed to *in utero* influenza. The same month for men is positive and statistically significant at a 5% level. It shows that men are on average 0.28% (1.12% adjusted to intent-to-treat effects) more likely to get married if the share of influenza exposure increases by 1% point. The opposing signs of the coefficients might indicate an underlying

mechanism which we are not able to identify.[16] None of the estimated coefficients for number of children are statistically significant.

Employment

We find extremely small and insignificant effects on employment both for men and women. Comparing the number of observations for the various explanatory variables, it is clear that there are many missing values for employment. Our data reveal that most of those who have reported their employment status are employed. In fact 99% of all those who reported their employment status are actively working. One possible explanation is that the unemployed are reluctant to report their employment status. Schwandt [2014] found that prenatal influenza exposure have significant negative effects on employment, especially in second trimester exposure. Unfortunately we do not have the level of detail in our data set to find similar results.

6.2 Heterogeneity

Tax income

A large body of literature finds that children from families with lower SES experience stronger negative effects of prenatal shocks and poor childhood health with regards to long-run outcomes (see eg. Case et al. [2005]; Currie and Hyson [1999]; Currie and Moretti [2005]). We therefore divide our sample into different subgroups to investigate whether poorer municipalities suffer more from an influenza pandemic than the rich. We use the average tax income per worker in each municipality as a proxy for high and low socioeconomic status. First we divide the sample at the median and find similar effects to our main specification in terms of education (see table 3). Years of education is reduced for men, and not for women. We find no

[16] See figure 4 for the trend in marital status for men and women.

significant effects for marital status, employment or in the number of children.

We further divide our sample to only include to lowest quartile of tax income. Table 4 shows that the effect of prenatal exposure to influenza has significant negative effects for exposure 5 and 4 months before birth. Five months before the birth, a 1% increase in influenza morbidity leads to 0.037 years (approx. two weeks) decrease in education, for men. Adjusting for intent-to-treat effects, this indicates an almost 2 months reduction in education. When compared to the effect of exposure in our main specification, the effect is approximately twice as large for the poorest municipalities. Four months before birth, a 1% increase in influenza morbidity leads to decrease of 0.039 years of education (approx 2 months when accounting for intent-to-treat estimates). The stronger effects for the lower income municipalities could be the driver behind the negative effect of influenza exposure in the main analysis. These findings are consistent with existing literature, which also finds stronger effects for the children with parents of lower SES, see eg. Black et al. [2013].

Note that the sample size is substantially smaller when dividing the sample into subgroups. This could have an effect on the significance of our result, as the fewer observations give less certainty to our findings. Further, 'Employment' for men have coefficients equal to zero. This is because the effects are so small due to very little variation in the sample.

Table 3: Long term outcomes when the sample is restricted to the lowest median

Trimester	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.043**	0.008	0.189	-0.189	-1.650*	-2.28e-06	-0.001
	(0.019)	(0.009)	(0.22)	(0.181)	(0.946)	(0.0001)	(0.002)
Pregnancy month 3	0.009	0.007	0.253	-0.186	-1.160	-0.001	-0.002
	(0.018)	(0.010)	(0.223)	(0.207)	(0.796)	(0.001)	(0.002)
Pregnancy month 4	-0.019	0.010	0.120	-0.183	-0.144	-0.001	-0.002
	(0.015)	(0.011)	(0.201)	(0.211	(1.00)	(0.001)	(0.002)
Pregnancy month 5	-0.027*	-0.004	-0.083	-0.152	0.044	-0.0001	-0.002
	(0.016)	(0.010)	(0.219)	(0.172)	(1.03)	(0.0001)	(0.002))
Pregnancy month 6	-0.002	-0.004	0.118	0.139	-0.117	0.001	-0.002
	(0.014)	(0.008)	(0.198)	(0.144)	(1.02)	(0.001)	(0.001)
Observations	33869	45696	75757	46615	38404	15424	2887

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education. The sample includes cohorts born between 1913 and 1919 in the four largest cities in Norway at the time. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: Long term outcomes when the sample is restricted to the lowest quartile

Trimester	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.077***	0.011	0.0489	-0.479	-0.154	0	-2.24
	(0.028)	(0.013)	(0.358)	(0.329)	(1.39)	(.)	(2.53)
Pregnancy month 3	0.026	0.017	-0.115	-0.186	-0.817	0	-3.71
	(0.021)	(0.014)	(0.383)	(0.281)	(1.27)	(.)	(2.55)
Pregnancy month 4	-0.037**	0.008	-0.543*	0.200	-0.520	0	-2.00
	(0.018)	(0.014)	(0.310)	(0.262)	(1.59)	(.)	(4.46)
Pregnancy month 5	-0.039***	-0.003	-0.326	-0.151	1.15	0	-2.49
	(0.014)	(0.0116)	(0.311)	(0.042)	(1.47)	(.)	(2.66)
Pregnancy month 6	0.001	-0.008	-0.108	0.162	-0.023	0	-1.51
	(0.019)	(0.010)	(0.313)	(0.214)	(1.56)	(.)	(1.90)
Observations	17303	23410	17633	23871	19507	7846	1406

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education. The sample includes cohorts born between 1913 and 1919 from the municipalities with the lowest tax income quartile. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth. Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Rural and urban areas

Hygiene and access to doctor could vary great between rural and urban areas in 1918. In order to investigate if there are heterogeneous effects depending on the size of the municipality, we run regressions when the sample is restricted to the four largest cities in Norway at the time; Oslo, Bergen, Trondheim and Stavanger. The results are presented in table 5. For the urban areas we find a strong negative effect in years of education for exposure 6 months before birth (pregnancy month 3). The estimates suggest an average reduction in education of 0.046 years (approximately 2 weeks). This translates into 2 months when accounting for intent-to-treat effects. Women in the cities are 0.5% less likely to get married if exposed to influenza *in utero* 4 and 5 months before birth (1.1% when accounting for the intent-to-treat effects). Further, women are 2.63% (10.52% accounting for intent-to-treat effects) less likely to be employed if exposed 3 months before birth. However, this estimate might not be very accurate due to few observations.

We also investigate the long term effects on influenza exposure *in utero* for the rural areas in Norway (see table 6). Rural areas are defined as all areas except Oslo, Bergen, Trondheim and Stavanger. The effect on education is in line with the long term outcome presented in table 2, albeit a littler higher. Four months before birth (pregnancy month 5), a 1% percent increase in influenza morbidity leads to a decline of 0.023 years of education (approx. 1.2 weeks) on average, suggesting a full treatment effect of approximately 4.8 weeks less education. Further, a 1% percent increase in influenza morbidity leads to decline of 1 child pr hundred women, when exposed 6 months before birth (pregnancy month 3), although this is only significant on a 10% level. For men, exposure 6 months before birth (pregnancy month 3) leads to an increase in the probability to be married. However, due to our suspicions of other underlying effects, we should be cautious to interpret any causal relationship on marital status.

Table 5: Long term outcomes when the sample is restricted Oslo, Bergen, Trondheim and Stavanger

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men (1)	Women (2)	Men (3)	Women (4)	Women (5)	Men (6)	Women (7)
Pregnancy month 2	0.001	0.045***	-0.279	-0.372	0.0272**	0	2.10**
	(0.031)	(0.004)	(0.623)	(0.166)	(0.474)	(.)	(0.624)
Pregnancy month 3	-0.046***	0.043*	0.324	-0.150	2.11	0	-0.836
	(0.004)	(0.018)	(0.264)	(0.294)	(1.95)	(.)	(1.02)
Pregnancy month 4	0.073	0.044	-0.256	-0.528*	0.142	0	-1.25
	(0.058)	(0.028)	(0.229)	(0.172)	(0.946)	(.)	(0.722)
Pregnancy month 5	-0.010	0.009	-0.158	-0.511**	0.428	0	0.726
	(0.034)	(0.014)	(0.232)	(0.153)	(1.20)	(.)	(1.55)
Pregnancy month 6	0.035**	-0.002	0.162	-0.193	2.32	0	-2.63**
	(0.009)	(0.009)	(0.115)	(0.519)	(1.03)	(.)	(0.762)
Observations	16650	22389	17101	23011	18434	11237	1981

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes cohorts born between 1913 and 1919 from the municipalities with the lowest tax income quartile. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 6: Long term outcomes when the sample is restricted to rural areas

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.0257	-0.001	-0.085	-0.115	1.24**	0.006	0.272
	(0.0162)	(0.006)	(0.167)	(0.114)	(0.624)	(0.005)	(0.915)
Pregnancy month 3	0.0112	-0.003	0.276**	-0.217	-0.937*	0.005	-0.616
	(0.0125)	(0.007)	(0.137)	(0.132)	(0.553)	(0.007)	(0.895)
Pregnancy month 4	-0.006	0.007	0.063	0.039	0.071	0.006	-0.315
	(0.0112	(0.006)	(0.131)	(0.110)	(0.562)	(0.005)	(0.906)
Pregnancy month 5	-0.023**	0.001	-0.141	-0.054	0.0527	0.008	-0.926
	(0.0100)	(0.006)	(-0.131)	(0.104)	(0.612)	(0.008)	(0.773)
Pregnancy month 6	-0.001	-0.001	0.094	0.006	-0.102	0.007	-0.551
	(0.00894)	(0.006)	(0.119)	(0.104)	(0.600)	(0.006)	(0.936)
Observations	16650	79073	17101	80782	66867	7812	5281

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from rural areas, which is all municipalities less Oslo, Bergen, Trondheim and Stavanger. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

7 Robustness Checks

We conduct several specification checks to verify the robustness of our findings. First, we consider the effects without controlling for a municipality specific time trend. Then we run regressions using quadratic time trend as some of our outcome variables are non-linear. Further, we regress each trimester of exposure on our outcome variables thus looking at longer time periods. There may be measurement errors regarding time of birth and regarding short gestation.[17] Looking at the entire trimester should, to a larger extent, allow for these inconsistencies. We also analyse the effects of maternal influenza using deaths caused by influenza in order to control for measurements errors in morbidity rates.

Trends

Running the regressions without the interaction term in equation 2, we find estimates when cohort and municipality fixed effects are controlled for. The results are presented in table A7. Compared to our main specification, the estimated coefficients are smaller and the standard errors are a little larger. However, our main findings of significant negative effects on years of education for men is clear also here.

To test whether we have specified the functional form of our main specification correctly, we extend the specification to include a quadratic time trend, in order to allow for non-linear trends in the variables (see table A8). Again, we find similar results to our main specification, thus differences in time trends do not drive the effects of influence on long term outcomes. Note that the standard deviations are very large when using a quadratic time trend, indicating less precise estimates.

[17] Richter and Robling [2013] use only trimester in their analysis in order to avoid problems due to misclassification.

Trimester

We run regressions on each trimester because it includes a longer time period, thus allowing for some measurement error regarding the time and month of birth or gestation lengths. The results are shown in table A9. For the second trimester, it is evident that none of the results are statistically significant. That is, when combining data for the second trimester (months 4-6) we are not able to isolate any effects on outcome later in life. The negative effects for exposure 4 months before birth may be drowned by the insignificant effects we found for exposure in months 3 and 5 before birth. For the first and third trimester there is a *positive* and significant effect of exposure on the years of education later in life. This is most likely due to culling of the weakest. The effect on marital status and number of children are not statistically significant.

Influenza Deaths

As discussed in section 4, there may be measurement errors in the registered number of influenza cases. Some doctors have reported influenza cases only if, and when, a death occurred, while others have recorded substantially more incidents. Further, there may be other noise such as mislabelling or misinterpreting the symptoms. In order to attempt to eliminate this noise, we run regressions using the number of deaths by influenza as a proxy for the severity of the illness in each municipality. Cause of death was compulsory to report at the time, but general influenza was not. This may result in less error in death statistics. Unfortunately, we only have yearly data on deaths caused by influenza for each municipality. We therefore have to compare cohorts year by year. The results are presented in table A10 and show that there are no significant effects of the share of influenza deaths on any of our outcome variables. Hence, it is possible that there is either too much measurement error in the mortality rate, or that our data is not detailed enough.

In order to investigate whether there is visible selection bias by culling, we regress

influenza on still births. Still births are only measured per year, and we have significantly fewer observations in our regression. This is because both variables are registered only for each municipality, not for individuals. The results are shown in table A11. We do not find that increased influenza morbidity has a significant impact on the share of still born. There could, however, still be strong effect on child mortality, so this finding does not necessarily indicate a lack of culling in our sample.

8 Conclusion

A large body of literature has shown that prenatal shocks have negative effects on adult outcomes. Using historical records we estimate long term effects of maternal exposure to influenza in Norway. We use the Spanish Influenza pandemic in 1918 as a natural experiment because it arrived without warning, impacted all socioeconomic groups, and had prevalent effects on women of childbearing age. Our findings suggest that negative effects of exposure are mainly found when the fetus is exposed in the second trimester. In our main specification we look at deviations from the trend and find that men exposed 4 months prior to birth get a 1 month reduction in education. This represent the average full treatment effect.These effects are twice as large for the poorest municipalities in Norway. We find a small negative effect of exposure six months before birth on the marital status, also just for men. Findings of negative effects in years of education as well as stronger effects for the poor are consistent with previous studies carried out on maternal exposure to influenza and other negative *in utero* health shocks.

A prominent attribute of our results is that most of the valid outcomes are found for males only. For the difference in return to education, a potential source of

the difference is the lack of variation in the female population. Since most women finished seven years of schooling, but very few have higher education, the variation in the sample is lower than for men. The medical literature suggest strongest impact on the brain during fetal development in weeks 8-15 after conception. Due to strong selection biases, we are not able to see the impact for all these weeks. There are several reasons for this. First, our sample consists of individuals still alive at the age of 42, so the weakest may have died by 1960. Second, negative maternal health shocks early in the pregnancy may lead to higher chances of miscarriages. Third, exposure late in the pregnancy could cause preterm birth, thus increasing the risk of childhood mortality. In our sample we have the strongest and most able children, so any negative consequences on long term outcomes in our analysis is the lower bound estimate for the long term impact on prenatal influenza.

Our findings, together with numerous other studies on shock to the intrauterine environment, suggest that there should be more focus preventing influenza for pregnant women. This measure could avert the potential negative effects associated with poor maternal health. It is particularly important to provide vaccination for women from a low socioeconomic group, as their children experience the most severe consequences from exposure.

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Appendix

Table A1: All months in gestation for men

	(1) Education 1960	(2) Married	(3) Employed
Pregnancy month 1	0.034***	-0.028	0.002
	(0.012)	(0.133)	(0.002)
Pregnancy month 2	0.022	-0.109	0.005
	(0.015)	(0.163)	(0.004)
Pregnancy month 3	0.002	0.285**	0.005
	(0.012)	(0.121)	(0.006)
Pregnancy month 4	0.007	0.015	0.005
	(0.015)	(0.119)	(0.004)
Pregnancy month 5	-0.021**	-0.140	0.007
	(0.010)	(0.117)	(0.005)
Pregnancy month 6	0.005	0.105	0.006
	(0.009)	(0.101)	(0.005)
Pregnancy month 7	0.012	0.065	0.006
	(0.010)	(0.144)	(0.004)
Pregnancy month 8	0.029***	0.175	0.003
	(0.010)	(0.125)	(0.002)
Pregnancy month 9	0.028***	0.020	0.001
	(0.010)	(0.114)	(0.017)

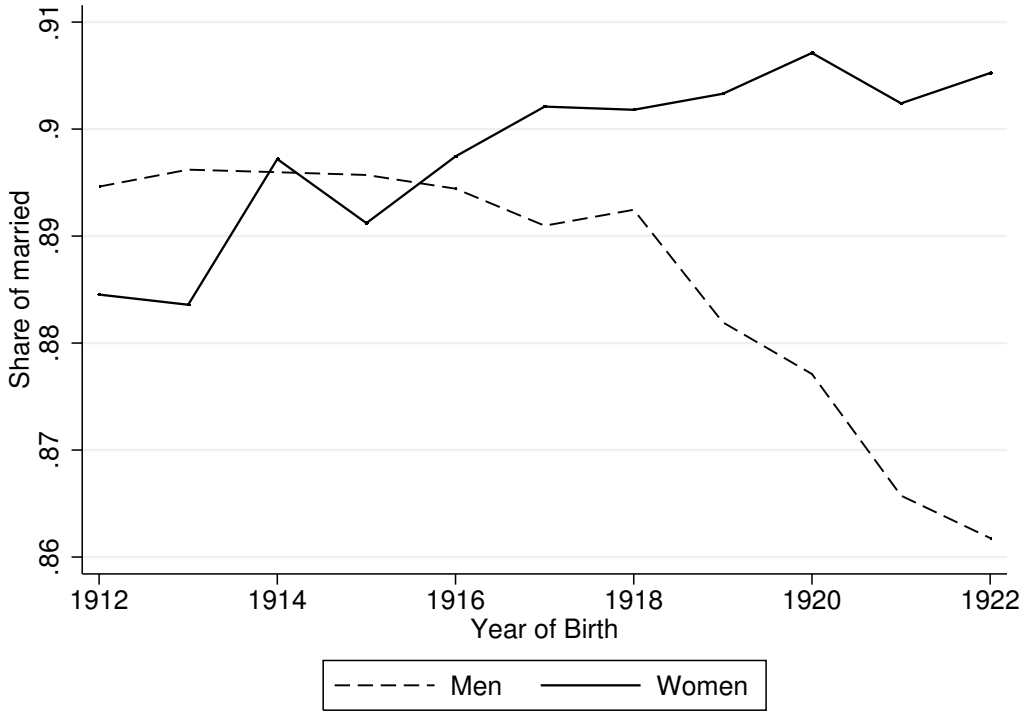
Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all men born between 1913 and 1919 from all municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A2: All months in gestation for women

	(1) Education 1960	(2) Married	(3) No. of Children	(4) Employed
Pregnancy month 1	0.004	0.0107	0.503	0.693
	(0.007)	(0.0939)	(0.555)	(0.970)
Pregnancy month 2	0.007	-0.152	-0.701	0.639
	(0.006)	(0.102)	(0.636)	(0.724)
Pregnancy month 3	0.002	-0.208*	-0.563	-0.700
	(0.007)	(0.120)	(0.590)	(0.740)
Pregnancy month 4	0.012	-0.045	0.068	-0.55
	(0.008)	(0.105)	(0.491)	(0.744)
Pregnancy month 5	0.001	-0.111	0.083	-0.703
	(0.006)	(0.096)	(0.553)	(0.676)
Pregnancy month 6	-0.001	-0.0289	0.230	-0.947
	(0.005)	(0.109)	(0.562)	(0.814)
Pregnancy month 7	0.008	-0.139	0.403	-0.649
	(0.007)	(0.098)	(0.519)	(0.574)
Pregnancy month 8	0.016**	-0.109	0.867	-0.455
	(0.006)	(0.110)	(0.629)	(0.718)
Pregnancy month 9	0.007	0.069	-0.285	-0.285
	(0.006)	(0.103)	(0.755)	(0.076)
Observations	100099	102388	84112	7181

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all women born between 1913 and 1919 from all municipalities in Norway.. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Figure 4: Share of married individuals in the 1960 census



Source: 1960 census data

Table A3: Long term outcomes when the sample is restricted to the highest median of tax income

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.008	0.005	-0.292	-0.118	-0.063	0.008	1.070
	(0.022)	(0.009)	(0.0002)	(0.122)	(0.794)	(0.008)	(0.700)
Pregnancy month 3	-0.003	-0.001	0.313**	-0.207	-0.146	0.008	0.038
	(0.015)	(0.010)	(0.137)	(0.148)	(0.776)	(0.011)	(0.908)
Pregnancy month 4	0.024	0.013	-0.054	0.023	0.186	0.008	-0.225
	(0.021)	(0.011)	(0.140)	(0.123)	(0.519)	(0.007)	(0.783)
Pregnancy month 5	-0.018	0.004	-0.183	-0.093	0.107	0.011	-0.298
	(0.011)	(0.007)	(0.126)	(0.118)	(0.631)	(0.008)	(0.679)
Pregnancy month 6	0.008	4.17e-04	0.092	-0.135	0.458	0.008	-0.600
	(0.010)	(0.007)	(0.111)	(0.149)	(0.638)	(0.007)	(1.03)
Observations	41412	57250	42410	58694	38404	19362	4469

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from the highest tax income municipalities in Norway divided at the median. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A4: Long term outcomes when the sample is restricted to the second quartile of tax income

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.019	0.007	0.299	0.030	-2.906**	-0.0003	0.731
	(0.028)	(0.012)	(0.269)	(0.211	(1.29)	(0.0003)	(2.15)
Pregnancy month 3	-0.006	0.002	0.560**	-0.216	-1.550	-0.002	-1.44
	(0.028)	(0.015)	(0.238)	(0.296)	(1.03)	(0.002)	(1.86)
Pregnancy month 4	-0.005	0.012	0.636***	-0.537*	0.111	-0.001	-1.83
	(0.022)	(0.017)	(0.215)	(0.285)	(1.28)	(0.001)	(1.82)
Pregnancy month 5	-0.018	-0.004	0.129	-0.158	-0.899	-0.0004	-0.111
	(0.029)	(0.015)	(0.278)	(0.249)	(1.38)	(0.0004)	(18.3)
Pregnancy month 6	-0.005	-0.001	0.303	0.094	-0.255	0.001	-1.52
	(0.021)	(0.013)	(0.243)	(0.194)	(1.41)	(0.001)	(1.25)
Observations	16566	22286	17633	22744	18897	7578	1481

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from second quartile tax income municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A5: Long term outcomes when the sample is restricted to the third quartile of tax income

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	-0.020	-0.002	-0.491*	-0.147	-0.956	-0.003	0.637
	(0.015)	(0.011)	(0.281)	(0.187)	(1.09)	(0.007)	(1.46)
Pregnancy month 3	-0.002	-0.007	0.278	-0.249	-0.800	-0.003	0.210
	(0.020)	(0.012)	(0.241)	(0.230)	(0.904)	(0.007)	(1.23)
Pregnancy month 4	0.012	0.006	0.0703	0.131	0.053	0.002	-0.491
	(0.022)	(0.011)	(0.215)	(0.160)	(0.881)	(0.002)	(0.981)
Pregnancy month 5	-0.005	-0.003	-0.138	0.076	0.352	0.003	-1.33
	(0.016)	(0.012)	(0.178)	(0.168)	(0.972)	(0.031)	(0.921)
Pregnancy month 6	0.011	-0.008	0.0328	-0.107	-0.554	0.001	-0.329
	(0.016)	(0.009)	(0.220)	(0.184)	(0.978)	(0.001)	(1.48)
Observations	17429	22286	17825	24807	20640	8125	1713

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from the third quartile tax income municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A6: Long term outcomes when the sample is restricted to the highest quartile

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.037	0.013	-0.087	-0.093	0.836	0.018	1.33**
	(0.035)	(0.013)	(0.302)	(0.166)	(1.10)	(0.015)	(0.644)
Pregnancy month 3	-0.004	0.006	0.0003**	-0.173	0.710	0.017	-0.050
	(0.023)	(0.017)	(0.138)	(0.180)	(1.21)	(0.020)	(1.41)
Pregnancy month 4	0.033	0.019	-0.156	-0.076	0.356	0.013	-0.019
	(0.035)	(0.017)	(0.176)	(0.174)	(0.556)	(0.013)	(1.11)
Pregnancy month 5	-0.030	0.011	-0.224	-0.249*	-0.068	0.018	0.537
	(0.018)	(0.008)	(0.183)	(0.142)	(0.812)	(0.015)	(0.716)
Pregnancy month 6	0.006	0.011	0.140	-0.175	1.56**	0.013	-0.784
	(0.014)	(0.009)	(0.107)	(0.241)	(0.663)	(0.013)	(1.42)
Observations	23983	33027	24585	33887	27522	11237	2756

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from the richest quartile municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A7: Long term outcomes no trend

Assumed pregnancy month	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 2	0.025*	0.008	-0.134	-0.146	-0.621	0.006	0.287
	(0.015)	(0.006)	(0.162)	(0.103)	(0.658)	(0.005)	(0.720)
Pregnancy month 3	0.005	0.003	0.261**	-0.192	-0.452	0.006	-0.497
	(0.013)	(0.007)	(0.119)	(0.120)	(0.617)	(0.005)	(0.737)
Pregnancy month 4	0.009	0.013	-0.003	-0.047	0.102	0.004	-0.540
	(0.015)	(0.008)	(0.117)	(0.104)	(0.505)	(0.004)	(0.672)
Pregnancy month 5	-0.019**	0.002	-0.145	-0.130	0.117	0.005	-0.600
	(0.010)	(0.006)	(0.116)	(0.096)	(0.564)	(0.004)	(0.666)
Pregnancy month 6	0.007	-0.001	0.095	-0.048	0.277	0.005	-0.738
	(0.009)	(0.005)	(0.100)	(0.110)	(0.564)	(0.004)	(0.709)
Observations	75281	102946	76946	105309	86566	19362	7356

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from all municipalities in Norway. Each cell represents a separate regression with year and municipality dummies. Municipality specific time trend is not included. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backwards from the month of birth. Robust standard errors in parentheses. All standard errors are clustered on municipality level
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A8: Quadratic trend

Assumed pregnancy months	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pregnancy month 4	0.008	0.012	0.0133	-0.0438	0.0744	0.00468	-0.574
	(0.596)	(0.135)	(91.10)	(67.50)	(87.90)	(27.40)	(44.50)
Pregnancy month 5	-0.020**	0.001	-0.139	-0.109	0.109	0.00668	-0.693
	(0.034)	(0.844)	23.30)	(25.90)	(84.40)	(14.10)	(30.70)
Pregnancy month 6	0.005	-0.002	0.111	-0.0264	0.251	0.00566	-0.945
	(0.576)	(0.754)	(27.60)	(80.90)	(65.50)	(.)	(24.60)
Observations	75281	102946	76946	105309	86566	34786	7356

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 1000 and education is in years of completed education.
The sample includes all cohorts born between 1913 and 1919 from all municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with quadratic trend. 'Pregnancy month' is the share of population contracting the flu in each month in gestation. The time of exposure is counted backward for the month of birth.
Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A9: Long term outcomes when separated by trimester

	Education 1960		Married		No. of Children	Employed	
	Men	Women	Men	Women	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
1st Trimester	0.041**	0.009	0.105	-0.242	-0.546	0.008	0.388
	(0.018)	(0.009)	(0.218)	(0.164)	(0.922)	(0.008)	(1.30)
2nd Trimester	-0.005**	0.008	-0.001	-0.113	0.243	0.011	-1.46
	(0.016)	(0.010)	(0.161)	(0.137)	(0.728)	(0.009)	(1.19)
3rd Trimester	0.046**	0.021**	0.169	-0.124	0.915	0.007	-1.03
	(0.014)	(0.009)	(0.181)	(0.154)	(0.857)	(0.007)	(1.05)
Observations	75825	103607	77502	105987	87150	35014	7395

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education. The sample includes all cohorts born between 1913 and 1919 from all municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. 'Trimester' is the share of population contracting the flu in each trimester in gestation. The time of exposure is counted backward for the month of birth. The table represents exposure by semester in order to eliminate some of the potential error due to misclassification. Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A10: Long term outcomes using influenza deaths

	(1) Education 1960	(2) Married	(3) No. of Children	(4) Employed
Men	0.153	-0.926		0.0213
	(0.097)	(1.48)		(0.0253)
Observations	68260	68260		31288
Women	0.060	-0.581	7.10	-2.77
	(0.055)	(1.23)	(6.01)	(10.1)
Observations	89979	89979	75231	6379

Note: 'Married', 'Employed' and 'No. of Children' are estimates per 100 and education is in years of completed education. The sample includes all cohorts born between 1913 and 1919 from all municipalities in Norway. Each cell represents a separate regression and year and municipality dummies, as well as an interaction term with year and municipality dummies. Hence results show deviations from the trend. The influenza mortality rate is used as the independent variable. The mortality rate is available for each year.
Robust standard errors in parentheses. All standard errors are clustered on municipality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A11: Infant mortality

	(1)
	Share of still born
Total flu	-0.207
	(1.009)
Observations	169406

Note: The influenza morbidity rate is regressed on the share of still born. The sample includes all cohorts born between 1913 and 1919 from all municipalities in Norway. Year and municipality dummies, as well as an interaction term with year and municipality dummies are included in the regression. Hence results show deviations from the trend. Data on the number of still born deaths are available on a yearly basis. Robust standard errors in parentheses. All standard errors are clustered on municipality level.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.