The Long Lasting Influenza: The Impact of Fetal Stress during the 1918 Influenza Pandemic on Socioeconomic Attainment and Health in Sweden 1968-2012

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ABSTRACT

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The observation in the 1940s, that children to mothers having *rubella* in the first part of the pregnancy experienced elevated health risks in later life led to a growing interest into whether fetal exposure to other – less severe – diseases could cause health problems as well. Epidemiological studies of the fetal origins of later life health that followed found that, while this indeed was the case, the effect was rather modest. A frequent weakness with many of these studies is furthermore that they only demonstrate associations, not causal relationships. Recent studies by economists and demographers, using quasi-experimental design to overcome this weakness, show that fetal conditions not only affect health in later life but also education and socioeconomic attainment. There is, however, a lack of consistency in the results. While some are showing strong effects, others show weak or no effects at all. Whether this is due to omitted variables, such as the socioeconomic status of parents or data quality problems is unclear. Thus, the question remains: does fetal stress caused by less severe diseases such as influenza, have long lasting impact on health and socioeconomic attainment? In this study we use a quasi-experimental design to test whether exposure to the 1918 influenza pandemic during the fetal stage influenced later life attainment using detailed data on the entire population living in Sweden anytime between 1968 and 2012. In addition, we use rich contextual data on morbidity and mortality, as well as on the socioeconomic status of parents, for the period 1914 to 1922 in order to address issues of selection. We find that the children of mothers exposed during pregnancy to influenza suffered from worse adult health and, for males, also increased mortality at old ages, particularly in cancer. Their income attainment was, however, only weakly – and positively - affected by fetal influenza exposure. We therefore conclude that observed health disadvantage is likely to have been a direct effect of fetal exposure to the 1918 influenza pandemic, remaining latent until later in life.

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Introduction

In the 1940s, it was found that the fetus was not as well protected during gestation as previously thought. In an important study by Gregg (1941), children to mothers having *rubella* in the first part of the pregnancy were found to experience elevated risks of health problems. Later studies found that *Thalidomide*, which was prescribed to women for morning sickness, could cause severe birth defects (McBride 1961; Von Lenz and Knapp 1962). It was also found that mothers consuming drugs or excessive amounts of alcohol were increasingly likely to give birth to children with health defects (see Almond and Currie 2011). In the wake of the early studies, the field investigating the *fetal origins hypothesis* grew rapidly, within epidemiology typically using birth weight as the key indicator of fetal development (Barker 1994). The underpinning theory was that low birth weight is associated with high blood pressure, which is a predictor of cardiovascular disease. The overall finding was that while low birth weight indeed is associated with higher mortality from cardiovascular and other diseases, the effect is modest (Christensen 2005; Godfrey and Barker 2000; Kuh and Ben-Schlomo 2004) .

Studies by economists for the U.S. have demonstrated that conditions during the fetal stage also affected socioeconomic outcomes, including educational and socioeconomic attainment, and that these effects could be large (Currie 2009; see Almond and Currie 2011). Using low birth weight as the indicator, this has been shown to have pronounced effects on educational (Currie and Moretti 2007; Johnson and Schoeni 2007; Royer 2009) and labor market outcomes (Johnson and Schoeni 2007). Studies for Great Britain have corroborated these findings, showing that children with low weight at birth performed worse in school, were less likely to be employed and earned less (Currie and Hyson 1999; Case et al 2005; Palloni et al 2009). Other studies have only found small effects of birth weight. A study of Norway, for example, finds that a 10% lower birth weight - a sizeable difference from the norm - is associated with only 1.2% lower IQ for males, 0.9% less earnings and 0.3% shorter height (Black et al 2007). Thus the results are mixed, possibly because the empirical design of many of these studies does not allow for causal interpretations.

Almond (2006) used a quasi-experimental approach focusing on the consequences of exposure to a specific event, the 1918 influenza pandemic, to obtain causal estimates of the link between fetal stress and later life socioeconomic and health outcomes. Using data for the US, he analyzed adulthood educational, earning, and health differentials depending on successive birth cohorts' exposure to the influenza pandemic. He found that males born in 1919, thus affected by the influenza during the fetal stage, were 3-4 % less likely to graduate

from high school and earned 1-3 percent less than surrounding cohorts (Almond 2006: Tables 1 and 2).

Brown (2011) questioned Almond's findings, arguing that he failed to take into account the compositional changes in parenthood caused by the drafting of army recruits to the First World War. More specifically, the criticism stated that the recruitment of American soldiers in the First World War was socioeconomically biased, since the authorities avoided recruiting married men from poor families. Consequently, this changed the social composition of the 1919 birth cohort, so that the share of children from poor families increased. Almond and Currie (2011:163) responded that while the effects found by Brown indeed are smaller, they still exist.

Several later studies of the long-term effects of the 1918 influenza on education and income corroborate Almond's findings (Nelson 2010 for Brazil; Liu and Lin 2014 for Taiwan; Neelson and Stratman 2012 for Switzerland). While the size of the effect in Brazil was large—the cohort exposed to the flu in utero earned 26 percent less than surrounding cohorts—it was minor in Taiwan. A study of the 1918 pandemic in Sweden, using county level data, found unexpected negative effects on poverty rates, expected positive effects on capital returns in a medium-term time perspective, but no effects on earnings (Karlsson, Nilsson, Pichler 2014). Another study of Sweden using individual level data for a sample born between January 1916 and September 1919 found a 2.4% negative effect on high school graduation for men only, and no effects on earnings (Richter and Robling 2015). In conclusion, previous research on the socioeconomic effects for individuals exposed to the 1918 pandemic during the fetal stage have produced a very mixed picture, with effects that sometimes are large, sometimes non-existent or even reversed.

The 1918 influenza pandemic also influenced mortality in later life for cohorts exposed during the fetal stage. A study by Myrskylä et al (2013) using the National Health Interview Surveys for the US to analyze cause-specific deaths find elevated mortality in non-cancer diseases for children born in the second quarter of 1918 and the first quarter of 1919. Another study on health outcomes, using annual data for twenty-eight countries found, however, no influence of the 1918 flu (Cohen et al 2010). Thus, also when it comes to mortality outcomes, the results are mixed.

The aim of this study is to test *the fetal origins hypothesis,* largely following the approach used by Almond, using data on the entire population in Sweden between 1968 and 2012. Examining Sweden provides a few important advantages compared to many other contexts. Firstly, as Sweden did not actively participate in the First World War, the concern

concerning a major change in the parental socioeconomic composition around the time of the influenza pandemic should not be relevant. We can also confirm this by using aggregated data on the occupation of fathers to new-born children. Second, since month and county of birth is available in the data, we are able to model the trimester of exposure to the influenza pandemic, whereas previous studies primarily have relied on annual or quarterly data. Thirdly, instead of using census or survey data, we use longitudinal data with detailed information not only on educational and income attainment, but also on hospitalization and cause of death.

The rapid spread of the 1918 influenza and its age-pattern

The 1918 pandemic is likely to have claimed at least 40 million lives worldwide during its short presence from the summer of 1918 until the spring the following year (Tauenberger and Morens 2006). In Sweden, the official number of deaths from the disease amounted to 34,000 out of a population of approximately 5.8 million. It started on Midsummer's eve, when a worker infected with the flu arrived from Kristiania (today's Oslo) to visit his relatives in a small coal-mining village in southern Sweden. In a few days' time, more than 50 people in the village showed symptoms of having been infected, and a few weeks later reports of the influenza came from all parts of Sweden (Åman 1990:42).

A distinguishing feature of the 1918 influenza was that the first wave occurred unusually early, already in the summer; the influenza period in Sweden normally occurs during the winter. Furthermore, due to the low initial death toll, it was hardly recognized. The second, very virulent, wave started in September and reached its peak in October/November 1918 with about 160 deaths per 100,000 individuals. During late spring of 1919, a third and comparably mild wave of the flu arrived, after which mortality returned to normal levels. As in other parts of the world, the influenza spread across Sweden in a short period of time, as shown by mortality rates in Figure 1. The spread of the disease thus followed the same pattern in Sweden as elsewhere.

- Figure 1 here

Another distinguishing feature of the 1918 influenza was its age-pattern. While most influenza epidemics do not change the typical u-shape pattern of age-specific mortality, the 1918 influenza elevated mortality in early-to-mid adulthood ages. Figure 2 shows mortality in influenza and pneumonia added together. The reason is that in some cases the cause of death

was registered as influenza, in others pneumonia. The latter include, however, all deaths in pneumonia and not only those related to the influenza. Taken together, it shows the typical w-shape reported from all over the world. Several explanations have been proposed to account for the disproportionately high mortality among young adults, e.g. partial immunity among older adults who had experienced the Russian flu of 1889-90, or vulnerability among young adults having tuberculosis, or overactive immune systems among young adults, or T-cell deregulation (Gagnon et al 2013).

- Figure 2 here

A comparison of all-cause mortality in 1918 with 1917 cast some doubts about the w-shaped age-pattern of the 1918 influenza itself. Evidently, overall mortality among infants and elderly are not discernably higher in 1918 than the year before, as shown in Figure 3. Infant mortality in Sweden was in fact slightly lower in 1918 than the year before, just like in Denmark and Finland, while in the US it was only slightly higher than the year before. Apparently, new-borns rarely died from the 1918 influenza, possibly being protected by their mother's antibodies. Mortality among the elderly is, likewise, not higher in 1918 than in the year before. Thus, it is doubtful that the mortality age-pattern that resulted from the 1918 influenza pandemic indeed was w-shaped in Sweden. What is certain is that mortality in among young adults and children were elevated creating an inverted u-shape age-pattern. Both males and females in childbearing ages suffered from higher mortality, males more than females. Contemporary sources report that, among females, pregnant women were particularly vulnerable (Almond 2006: 681; Åman 1990).

- Figure 3 here

The sensitivity of women who were pregnant has potentially important implications for this article, as the study population consists of individuals who survived the during the fetal stage and into late adulthood. Consequently, if in exposure to the influenza during the fetal stage caused a culling of the weakest fetuses, those surviving could be positively selected in terms of health characteristics. On potentially important indicator of such selection is represented by the stillbirth rate, as exposure to the 1918 influenza pandemic during the later stages of pregnancy may have resulted in an increased number of stillborn babies, particularly during the months of peak influenza exposure. Figure 4 uses monthly data on still births and

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live births, obtained from official Swedish statistics during the time period 1916 –1920, presented as still births expressed as a share of every 1,000 live births. The figure indicates a distinct increase in the stillbirth rate during the most severe influenza months, particularly accentuated during October 1918. More specifically, during the time period displayed, the stillbirth rate typically hovers between 20 and 25 still born children per 1,000 live born babies, increasing considerably in October 1918 to a rate of almost 35. Based on the expectation that stillbirths predominantly reflect fetuses succumbing to influenza exposure during the *later* stages of pregnancy, Figure 4 indicates the possibility of culling among fetuses exposed during the third trimester.

- Figure 4 here

Another related indicator of fetal selection is represented by the sex ratio at birth. This derives from male fetuses being weaker than female, why an accentuated (negative) deviation from the normal male to female ratio of around 52 percent could be an indication of the birth cohort having experiencing a high incidence of spontaneous abortions. As the influenza by and large peaked during the last quarter of 1918, the sex ratio could be expected to be affected from late 1918 and potentially until September/October the next year, thus approximately nine months after. The expectation that the response should become manifest with a delay is motivated by the fact that fetuses are particularly sensitive to insults during the earlier phases of pregnancy. Figure 5 shows the sex ratios of each monthly birth cohort born between January 1916 and December 1920, calculated based on data from official Swedish statistics. The data indicates a sex ratio which consistently hovers at around 51.5 percent, never going below (above) 50 (53) percent during the period displayed. Thus, the data fails to show a greater incidence of spontaneous abortions as a result of the influenza, contrary to the hypothesis that birth cohorts affected during the earlier stages of pregnancy were characterized by particularly strong selection mechanisms.

- Figure 5 here

According to official sources at the time, only 9 percent of the Swedish population was reported as having been infected by the flu; the figure for Stockholm city is as low as 1.43 percent (Åman 1990: 58). Officially reported morbidity figures have, however, been deemed to be far too low, even by contemporary authorities, both in Sweden and the US (Åman 1990;

Crosby 1989). Local and workplace surveys have provided much higher figures (Alling 1919, Gibson 1919, Widstrand 1919). In Sweden, according to some sources, about 75 percent of the population showed clear symptoms of the 1918 influenza (Åman 1990: 58-59).

At *Höganäsverket*, a coal mine and factory located in the southwest of Sweden, 61 percent of all males and 50 percent of all females, families included, were reported as having evident symptoms of the 1918 flu by the company physician (Alling 1919, Bilaga I). Among men aged 15-20, the sickness rate was 81 percent. Similar figures were reported in other workplaces in Sweden, as shown in Figure 6.

- Figure 6 here

White and blue collar workers at Höganäsverket were reported to have the same incidence rate, again, a pattern representative of Sweden as a whole (Alling 1919, Gibson 1919). Among the mine workers, 56 % were reported to have been on sick leave for an average of 14 days (Alling 1919, Bilaga II; Höganäsverkets arkiv 1919). Out of those being ill, one-third was reported as suffering badly from the disease. While the attach rate (the proportion of persons with clinical signs) of a normal influenza is 30-60 percent (Gagnon et al 2013), it is likely that it was higher in the case of the 1918 flu.

The rapid spread of the 1918 pandemic meant that by the end of the year, 80 percent of all deaths had occurred. Thereafter, the number of deaths fell, only to resurge somewhat during the third wave in March-April 1919. Despite the substantial underreporting of persons having influenza symptoms, as discussed above, the morbidity rate followed an almost identical time pattern, as shown in Figure 7. By the end of 1918, some 83 percent of those ever-reported ill to the medical authorities had contracted the disease. Thus the 1918 influenza was very short and intensive in Sweden, as elsewhere.

- Figure 7 here

The use of the 1918 influenza as a quasi-experiment
Employing the 1918 flu exposure as a quasi-experiment is based on a number of assertions. First, the event should not be anticipated. Second, when it takes place, there should be no means to avoid it. Third, there should not be any concurrent factor that could influence the outcome of the event (no omitted variable). We believe that the first two do not pose any major problem. Of course, knowledge of the disease was widespread already in September, with newspapers reporting about it, but the disease was difficult to avoid and treat (Åman

1990). While the authorities closed down theatres and cinemas, for the most part, people went on with their daily lives much as usual. The question about an omitted variable, raised by Brown (2011) in his critique of Almond, is of greater concern. Would this be a problem when analyzing Sweden? While Sweden did not participate in the First World War, more men were nonetheless recruited to the army than usual. The magnitude was modest, however, and recruits could not be excluded on social grounds. Still, other factors might cause the social composition of new parents to change, whether it was an effect of the war, or the flu, or some other unknown factor.

To answer the question of whether the socioeconomic composition of parents changed in 1918, as in the US, we use information on the occupation of fathers of new-borns from the aggregate national statistics provided in *Befolkningsrörelsen* between 1915 and 1920. The data shows not only in which of the six economic sectors the father worked, but also their occupation, classified into a total of 63 categories. Based on this information we can distinguish between different social groups. For example, in agriculture, which was the largest sector at the time, we can distinguish between various types of landowners and workers. Factory owners, likewise, are separately reported within the industrial sector, and so are higher civil servants in the service sector. In other sectors, e.g. in trade, it is more difficult to distinguish between the well-off and the workers, and hence, we include results using an alternative calculation.

Figure 8 shows that the proportion of new-born children to well-off fathers increased slightly in 1917-18 and declined in 1920. An alternative calculation, with merchants excluded from the well-off group, shows a similar pattern. The proportion of children to well-off parents is changing 1-2 percent units over time at the most, the largest change taking place between 1919 and 1920. Thus the potential problem of changes in social composition of parenthood during the 1918 flu is if not negligible, at least very modest.

- Figure 8 here

Data and methods

The data used in the analyses comes from the Swedish Interdisciplinary Panel (SIP), by and large covering all individuals living at any time in Sweden from 1968 until 2012. SIP contains detailed individual level information on demographic events including county, year and month of birth, time and cause of death, as well as information on hospitalization, educational attainment, income, and information from quinquennial censuses between 1960 and 1990.

The sample selected for the analysis consists of all individuals born in Sweden between 1914 and 1922. Similar to related analyses, the sample is centered on the cohorts exposed to the 1918 influenza pandemic, in 1918-1919. Exposure to the influenza pandemic is defined in two different ways. Initially, we follow the definition most frequently used in Almond's (2006) seminal paper, examining the long-term consequences among those born during the first quarter of 1919. Across the world, the most severe wave of the influenza had subsided by January 1919, and hence those born during this quarter by and large are characterized by exposure which occurs exclusively during the fetal stage, in particular during late pregnancy.

Ideally, however, the temporal as well as geographical spread of the 1918 influenza pandemic needs to be taken into account when defining an individual's exposure. Therefore, for the second definition of influenza exposure, we use county-level mortality data in order to define the timing and duration of the most severe 1918 autumn/winter wave of the 1918 influenza pandemic. For each county, we define the onset of the pandemic to occur from the month when the CDR exceeds 1.75 deaths per 1,000 inhabitants. While the threshold may appear arbitrary, during 1917 and the first half of 1918, the median value was 1.09, never exceeding 1.74. The onset for all counties occurs between September and November of 1918, where for the large majority, this occurred in October. The end of the influenza period is defined as occurring from the month where the CDR again falls below the threshold of 1.75 deaths per 1,000, which happens no later than after January 1919[1]. The monthly CDR for all counties during the twelve months centered on the influenza pandemic is displayed in Figure 1, located in a previous section. Furthermore, based on the individual's county and month of birth, we are able to more precisely pinpoint if and when they were exposed to the 1918 influenza pandemic. More specifically, among those exposed during the fetal stage, we distinguish between the trimesters of exposure. This distinction is potentially important, as the fetal growth process is characterized by different processes, implying that the consequences of experiencing an insult may differ depending on the timing of exposure.

The dependent variables for the study are represented by measurements of adulthood health as well as socioeconomic outcomes. Whereas health outcomes are examined among both men and women, the analysis of socioeconomic outcomes is limited to males. When analyzing health outcomes, individuals are followed from age 54 and until turning age 90, death or censoring. Health is measured both through morbidity and mortality, where the former is obtained through annual information on hospitalizations. For hospitalizations,

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[1] Sensitivity analyses have been run using CDR thresholds of 1.5 and 2.0, not altering results qualitatively or quantitatively.

information is available on both the duration and underlying diagnosis of all hospitalization spells. In the analysis, we however only rely on i) whether individual i was hospitalized during year t, and ii) the combined duration of all hospitalization spells of individual i during year t. The reason for not exploiting information on the underlying diagnosis is due to left censoring, that we are unable to accurately measure the *onset* of disease due to the unavailability of information prior to 1968. A further caveat is represented by the hospitalization register only covering all Swedish counties from 1987, with a gradually increasing coverage from its start in 1968. Thus, individuals are only included in the analysis provided that their county of residence is covered by the hospitalization register during the actual observation year. The analysis focuses on the probability of hospitalization during the current year and is performed using logistic regression models. Sensitivity analyses using different operationalizations of the hospitalization variable produces essentially identical results, and is presented in the Appendix. Here, multinomial logistic regression models distinguish between different durations of hospitalizations, whereas Tobit regression models are used to analyze the number of days of hospitalization.

The mortality analysis is performed using information provided by the death register, covering all deaths from January 1 1968 and until the end of 2012. Individuals are considered risk from turning 54 years of age and are censored when turning 90 years of age, or exiting the dataset through – for example – emigration. The analysis focuses both on all-cause as well as cause specific mortality, where we distinguish between deaths due to cancer, cardiovascular, or other causes. The all-cause mortality models are estimated by means of Cox proportional hazards regression technique, whereas the cause-specific analysis is performed through Cox proportional hazards competing risk models. More specifically, we employ the duplication method outlined by Lunn and McNeil (1995), permitting the estimation of the effect of 1918 influenza pandemic exposure on all three competing outcomes separately. Thus, while competing risks typically are analyzed through a separate analysis for each event, treating the competing events as censored, this model allows for greater opportunities for statistical inference. In this case, this is represented by the ability to statistically test whether the effect of influenza exposure is statistically significantly different between the outcomes of interest, through Wald tests on total effects.

Turning to the final set of outcomes for the analysis, socioeconomic attainment is measured through income recorded at ages 55 and 60 and through being in a high socioeconomic status (SES) occupation, observed in the 1970 census. This is defined through being in a proprietary position within agriculture, industry and service occupations, or being

in a military occupation. As a result, the birth cohorts examined in the analysis on occupational attainment are observed at different - yet not before attaining peak performing - ages, between ages 48 and 56, an unavoidable feature of the data. The income data has been inflation adjusted to 1975 prices and is analyzed by means of Tobit regression to account for the considerable share of censored zero values, whereas the attainment of a high SES occupation is analyzed by means of binary logistic regression.

All estimated models control for centered birth year and centered birth year, squared. Furthermore, all models include combined county, year and month of birth fixed effects, in order to account for seasonality and county-specific factors. The reason for this is that birth season has a social gradient, which consequently affects health and income (Buckles and Hungermann 2013).

Descriptive statistics

Table 1 displays variable means for the study population, which differs somewhat in terms of sample size depending on the outcome being examined. Standard deviations are only reported for continuous variables, in brackets. Common across all samples is that the population analyzed consistently is restricted to individuals born between 1914 and 1922, as well as how influenza exposure is defined. Across all samples, the population is centered at 1918, and each annual birth cohort contributes with between 10 and 13 percent of each sample. Indeed, there are no indications that either the 1918 or the 1919 cohort – exposed to the 1918 influenza pandemic during the fetal stage or during infancy – are decimated compared to surrounding birth cohorts. As can also be observed in the table, the share of individuals who were exposed to the 1918 influenza pandemic is highly similar across the samples analyzed. More specifically, approximately five percent of each sample was exposed during one of the trimesters of gestation, respectively, compared to the approximately 2.5 percent who were born during the first quarter of 1919, the definition most frequently encountered in the related previous research.

- Table 1 here

The analyses of socioeconomic outcomes, income attainment and reporting a high SES occupation, is performed in a cross-sectional setting. Incomes are analyzed for men at the ages 55 and 60 and the sample means indicate an annual income of about 44,000 SEK (in 1975 prices). Using data from the 1970 census, the data also shows that around 20 percent of

the males in the examined birth cohorts had attained a high SES occupation. Of greater interest is, however, to what extent the data indicated that exposure to the influenza pandemic is associated with later life adversity. Figure 9 displays the log mean attained income – fixed at 1975 prices - at age 55 (on the left axis) as well as the mean probability of being in a high SES occupation (on the right axis), for all year and quarters of birth examined in the paper. The black vertical line represents the cohort born during the first quarter of 1919, in previous research typically considered to be the one most severely affected by the pandemic. For inflation adjusted log mean income, the data reveals an almost monotonically increasing trend across birth cohorts, clearly without any indications of the outcome of the Q1 cohort of 1919 diverging from the trend. A similar absence of such indications can be observed regarding the share attaining a high SES occupation.

- Figure 9 here

Returning to Table 1, across all measurements of hospitalization, men are experiencing greater morbidity, as would also be expected. While the differences between the genders are not dramatic, in the age range examined for all cohorts – between the ages 54 and 90 – about 20 and 18 percent of the men and women, respectively, experience a hospitalization spell during any given year. Figure 10 displays a generally declining probability of hospitalization across birth cohorts for both men and women, also containing considerable volatility, albeit not necessarily consistent with a seasonal pattern. Thus, while the increasing hospitalization share among men born during the first quarter of 1919 is clearly above the trend, the extent of this is emphasized by the previous observation, exhibiting an unusually low value. Furthermore, a similarly elevated value can be observed for the cohort born during the second quarter of 1920, not plausibly attributable to the influenza pandemic. Among women, no indications of any consequences resulting from influenza pandemic can be observed. The mean days of hospitalization, displayed in Figure 11, indicates a monotonically decreasing trend, from 3.9/4.2 for the female/male 1914 Q1 cohort to 2.6/2.8 for the 1922 Q4 cohorts. Neither shows any indications that the cohort born during the first quarter of 1919 was any different from surrounding cohorts.

- Figure 10 and 11 here

The last two columns of Table 1 display the summary statistics for the mortality analysis, displaying males' overall higher mortality. Of the sample of individuals observed at age 54 and followed until death, age 90 or censoring, 86 and 71 percent of the males and females, respectively, die. The data also indicate that death due to cardiovascular disease represent the most common cause of death, representing more than double the share of deaths due to cancer. Figure 12 shows the development of the mean age at death for the examined birth cohorts, again displayed by quarter of birth. While showing an increasing tendency over time, this is indeed quite modest, not indicative of any noteworthy deviation from the overall trend experienced by the cohort born during the first quarter of 1919.

- Figure 12 here

Results

Income attainment

Table 2 presents the results from Tobit regression analyses on males' income attainment at ages 55 and 60, respectively. For each outcome, exposure to the 1918 influenza pandemic is defined as experienced by individuals born during the first quarter of 1919, in line with related previous research. Due to the access to county specific mortality data, exposure has also been more specifically defined, allowing us to identify the trimester of exposure, thus representing the second definition of exposure.

- Table 2 here

Regardless of the age at measurement, individuals born during the first quarter of 1919 are observed with an *increased* income attainment, shown in Models 1 and 3 and clearly at odds with the à priori expectation. Whereas the coefficient is insignificant at age 55, it is both statistically and quantitatively significant at age 60. More specifically, males born during Q1 1919 enjoy an 862 SEK higher annual income (in 1975 prices) than surrounding birth cohorts, which translates to an approximately two percent income premium compared to the average at this age. When proceeding to the more precise definition of exposure, in Models 2 and 4, it emerges that this premium in particular is enjoyed by males exposed during the first trimester of pregnancy, with similar estimates both at ages 55 and 60. The income premium amounts to about 2.4 percent, to be compared to 1.4 and 1.8 percent at ages 55 and 60, respectively, if exposed during the second trimester. Among those exposed during the third trimester, no

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significant effects are found. Given the fetus' greater sensitivity during the earlier stages of pregnancy, the results are consistent with selection being the main drivers underlying the results observed among individuals exposed during trimesters one and two.

High SES occupation

An outcome representing a different yet related measurement of labor market performance is measured through the attainment of a high SES occupation. As previously reported the outcome is dichotomous and measured using data from the 1970 census. The results, presented in Table 3, are consistent with those previously obtained for income attainment as far as the absence of evidence showing that fetal exposure to the 1918 influenza pandemic is associated with adverse socioeconomic outcomes are concerned. However, whereas the strongest positive effects for income were observed among those exposed during trimester one, as regards the probability of attaining a high SES occupation, a significantly elevated probability is only observed among males exposed during trimester two. More specifically, compared to surrounding birth cohorts, exposure during the second trimester is associated with a 4.6 percent increase in the probability of attaining a high SES occupation. While the estimates for exposure during trimester one and three (as well as being born during Q1 1919) are statistically insignificant, the point estimates show an increased rather than decreased probability of high SES occupation attainment among individuals exposed to the 1918 influenza pandemic.

- Table 3 here

We have so far not found any evidence supporting the hypothesis that exposure to the 1918 influenza pandemic caused an adversity in terms of socioeconomic attainment during late adulthood. Instead, the results so far show that males exposed during the earlier part of pregnancy became positively selected and thus experienced improved socioeconomic outcomes in the ages here examined, compared to surrounding birth cohorts.

Hospitalization

Table 4 presents results for males as well as females, estimating the probability of being hospitalized during any given year between the age of 54 and 90. With the results regarding socioeconomic outcomes in mind, the results for males on morbidity through hospitalization are quite striking. More specifically, as indicated in Model 1, males born during the first

quarter of 1919 not only experience significantly higher incomes, but also a 3.6 percent higher risk of hospitalization. Similarly, an increased (3.1 percent) probability of hospitalization among individuals exposed to the 1918 influenza pandemic is found for the second trimester.

- Table 4 here

Largely similar results are found among women, however showing differences in when the female fetus was particularly vulnerable to 1918 influenza pandemic exposure. Whereas exposure for individuals born during the first quarter of 1919 resembles that observed for males, with a 2.9 percent increase in the risk of hospitalization, shown in Model 3, the analysis by trimester of exposure indicates females exposed during the last trimester as particularly disadvantaged. More specifically, as indicated by Model 4, females exposed during this last trimester of pregnancy experience a 3.2 percent increase in the probability of hospitalization. For both males and females, identical models have been run using alternative outcome variables, with days' of hospitalization during the current year operationalized as a continuous as well as a categorical variable. The models, provided in Table A1/A2, Appendix, strongly confirm the results already discussed in this subsection. More specifically, among males, influenza exposure during the second trimester is systematically linked to longer hospitalization spells, with the same being the case among women exposed during the third trimester.

Mortality
The mortality analyses focuses on outcomes that are operationalized both as all-cause and cause specific mortality. Similar to the morbidity analyses, individuals are considered at risk from turning age 54 and subsequently followed until death, turning age 90 or censoring. The modelling strategy again follows that employed in previous sections, with Models 1 and 3 in Table 6 estimating the change in the mortality hazard among men and women who were born during the first quarter of 1919, respectively. Model 1, displayed in Table 5, indicates that the mortality response among males, elevating the hazard with a statistically significant 3.8 percent, is substantially more accentuated compared to that observed for women in Model 3. Here, the parameter fails to be statistically significant, also showing a considerably more modest elevation of the mortality risk (1.2 percent).

Proceeding to Models 2 and 4, exploiting more precise information on the timing of fetal exposure to the 1918 influenza pandemic, the results for males again strongly resemble

those obtained in the morbidity analysis. More specifically, exposure during the second trimester appears to be particularly harmful and associated with a 3.1 percent increase in the mortality hazard. That exposure during this trimester among males is exceptional is further underlined by the close to null point estimates for individuals exposed during trimesters one or three, both being statistically insignificant. Model 4 displays corresponding results for females, confirming that the weaker mortality response among females previously observed is not concealing a pattern only observable through a more precise definition of exposure. Indeed, whereas the point estimates for females exposed during trimesters show an increased mortality risk, the size of the effect is negligible (<1 percent) and statistically insignificant.

- Table 5 here

Lastly, the effect of the influenza on cause specific mortality is estimated by means of competing risk Cox proportional hazards regression, using the so-called Lunn-McNeill method. The analyses distinguishes between mortality due to cardiovascular disease, cancer or other causes, and presented in Table 6 are the total effects for respective form of influenza exposure and its effect on the cause specific mortality hazard. As the effects for the non-base outcomes are obtained through interaction effects, the parameter standard errors are only reported for the base effects (cancer mortality). Instead of standard errors, Wald test statistics are reported for cardiovascular and other cause mortality.

- Table 6 here

Model 1 is estimated on the male sample, considering individuals born during the first quarter of 1919 as exposed to the 1918 influenza pandemic. While not estimated with enough precision to be statistically significant, we find elevated mortality risks in cancer and cardiovascular disease, both amounting to around 4 percent. A similar tendency is observed among women (Model 3), where those born during the first quarter of 1919 experience an elevated mortality risk in cancer and cardiovascular disease. Again, the parameters are, however, statistically insignificant. Whereas a statistically insignificant increase in mortality risk due to other causes amounting to one percent is observed among males, a slight decrease is observed among females. Models 2 and 4, estimated on males and females separately, again indicates stronger mortality responses among males. Indeed, influenza exposure during trimester two stands out as the period during which this insult is associated with the most

disadvantageous outcomes for males, most strongly (and statistically significant) driven by cancer mortality. Also for cardiovascular mortality is the effect the strongest for males exposed during trimester two, yet both weaker than for cancer mortality and not statistically significant according to the Wald test. Among females, presented in Model 4, effects that are both smaller in size and statistically insignificant can be observed, along with a rather less consistent pattern regarding the timing of exposure and the consequences thereof. More specifically, whereas a small but statistically insignificant mortality risk in cancer disease (1.7 percent) among females exposed to the 1918 influenza during the second trimester is obtained, the third trimester of exposure appears to be the most strongly connected to an increased mortality risk in cardiovascular disease (2.2 percent) and other causes (0.7 percent).

Summary and discussion

In 2009, countries across the world feared the outbreak of another influenza pandemic. The fears were partly linked to the huge immediate effects in terms of deaths it may cause, and partly to possibly long lasting consequences of those surviving the disease. As a result, nations spent huge amounts of money on vaccination campaigns and other forms of preparations to limit the consequences of a pandemic outbreak.

While the immediate costs of the 1918 influenza pandemic were evident, claiming at least 40 million lives worldwide during its short presence from the summer of 1918 until the spring the following year, the long lasting effects are less well-known. They have, however, recently been analyzed by epidemiologists, demographers, and economists with a great deal of variation in the results. While some studies find strong *scarring effects* on income and health, others find only weak or no effects at all. Others again find that the *selection effect* dominates. Whether this is due to data problems or omitted variables, for example for not taking changes in parental socio-economic status into account is unclear. Thus the question remain: how strong are the long term effects of fetal stress during the 1918 influenza pandemic on health and prosperity?

Testing the fetal origins hypothesis on Sweden, we use data for a country for which we at an aggregated level know the occupational composition of parents to the children born during the time of the 1918 influenza. By using data for the entire country, and not certain states, or counties, or a retrospective survey, we not only get access to large amounts of information but we also avoid sampling problems. Furthermore, by having access to more detailed information on time and place of birth, as well as adulthood attainment of income,

occupation, health, and mortality, we are able to model the exposure, and the outcomes thereof in more detail.

In terms of health outcomes, our results are consistent with the fetal origins hypothesis that scarring and not selection is predominant. More specifically, among both sexes, exposure to the 1918 influenza while *in utero* resulted in higher morbidity, as measured by hospitalization. In terms of mortality, significant effects are only found among males, and the effects are overall quite small. The all-cause mortality risk is never more than 3.8 percent higher for those exposed compared to surrounding cohorts. Myrskylä et al (2013) find an effect of 9 percent, using retrospective data for a sample of US born. The results from the U.S., however, potentially suffer from a bias resulting from compositional changes in the socioeconomic status of children born during the First World War. For Sweden we know that such changes are small, the proportion of children born to well-off parents is only slightly higher, a few percentage points, in 1917 to 1919 than in the years before and after. From analyses of aggregated cohort mortality we also know that these cohorts have similar survival rates over the life course, improving life expectancy by 4-5 months per year, and only with small reductions for the cohort born in 1914 and 1918. Thus the 1918 influenza pandemic in Sweden shortened adult life, but only moderately.

While Almond found negative and significant, but modest, effects of the 1918 pandemic on adult incomes, the negative effect has been shown to be considerably stronger elsewhere, e.g. in Brazil where the flu cohort earned - arguably perversely - 26 % less than surrounding cohorts (Nelson 2010). Our results for Sweden, display a lack of indications showing that individuals who were exposed to the influenza pandemic in utero suffered from lower earnings in late adulthood. Indeed, the results – only estimated on males – show that individuals who were exposed during the first (and second) trimester experienced higher earnings, both at age 55 and at age 60. Similar results were also found regarding the ability to attain a high socioeconomic occupation.

The descriptive analysis showed the possibility of fetuses exposed during the final stages of pregnancy as being selected, due to the peak in stillbirths in October of 1918. Furthermore, due to the greater sensitivity of male fetuses, it was hypothesized that such selection mechanisms should be accentuated among boys. Comparing male and female health outcomes, it is clear that the period of exposure associated with the strongest adulthood health responses is the second trimester for males and the third trimester for females. While not conclusive evidence, it appears to be possible that the lowered response among males exposed in trimester three can be linked to aforementioned fetal selection. Turning to the

socioeconomic outcomes, only estimated for males, however complicates this story. The cohorts exposed during the final trimester do not display similar indications of positive selection in terms of socioeconomic outcomes as for health. Instead, it is the cohorts who were exposed during trimesters one and two who experience superior income and occupational attainment. This is particularly interesting, as the sex ratio at birth provided no indications of increasing spontaneous abortions among cohorts exposed to the 1918 influenza during early gestation.

While the effect of in utero exposure to the 1918 influenza on adult hospitalization and mortality is conclusive and supports the fetal origins hypotheses, the size of the effects appear to be relatively modest. More specifically, the largest effect on hospitalizations for both men and women is about a three percent increase, which should be compared to the mean hospitalization rate at between 18 and 20 percent. In terms of the influence of fetal influenza exposure on socioeconomic outcomes, the results are consistent across outcomes, but somewhat difficult to reconcile with the à priori expectations. Consequently, while exposure to the 1918 influenza pandemic while in utero indeed did alter an individual's life trajectory, it hardly did so in as powerful a fashion as has been found in some of the previous literature. In conclusion, the results on Sweden show that is difficult to identify whether scarring or selection is the dominant mechanism. This fits well with the previous studies on Sweden, by Karlsson et al (2014) and by Richter and Robling (2015). Furthermore, our results also highlight that it is important to discuss how big the effects of a given insult are compared to other factors, and not only focus on the direction of the effect and significance.

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Table 1: Sample means

	High SES occupation	Income, age 55	Income, age 60	Hospitalization		Mortality	
	Men	Men	Men	Men	Women	Men	Women
High SES occupation (%)	21.4						
Income (1000's)		44.6	43.8				
		[35.5]	[30.6]				
Hospitalization during current year (%)				19.8	17.5		
Days of hospitalization during current year (%):							
0 days				80.2	82.5		
1-5 days				7.5	6.1		
6-10 days				4.1	3.7		
11-20 days				3.7	3.4		
21-30 days				1.7	1.6		
31 days or more				2.9	2.7		
Days of hospitalization during current year				3.4	3.1		
				[14.4]	[17.5]		
Mortality, all cause (%)						85.5	71.0
Mortality, cancer (%)						22.8	18.4
Mortality, cardiovascular (%)						51.7	41.3
Mortality, others (%)						11.0	11.4
Birth year	1918.1	1918.1	1918.1	1918.3	1918.3	1918.1	1918.1
	[2.6]	[2.6]	[2.6]	[2.5]	[2.6]	[2.6]	[2.6]
Born Q1 1919 (%)	2.6	2.6	2.6	2.6	2.6	2.6	2.5

Exposure trimester 1 (%)	5.1	5.1	5.1	5.0	5.0	5.1	5.1
Exposure trimester 2 (%)	4.9	4.9	4.8	4.9	5.0	4.9	4.9
Exposure trimester 3 (%)	5.3	5.1	5.2	5.2	5.2	5.2	5.2
Observations	322,458	333,574	322,048	6,928,291	8,772,984	338,409	358,264
Individuals	322,458	333,574	322,048	319,957	345,607	338,409	358,264

Notes: The summary statistics and the number of observations for the competing risk cause specific mortality analysis are reported for the sample prior to the duplication procedure, resulting in sample sizes amounting to 1,015,227 for men and 1,074,792 for women.

Source: Swedish Interdisciplinary Panel

Table 2: Tobit regression analysis output. Dependent variable: Income (in 1,000 SEK, 1975 prices). Zero incomes are censored.

	Men, age 55		Men, age 60	
	Model 1	Model 2	Model 3	Model 4
Almond exposure (birth Q1 1919)	0.551		0.862**	
	(0.427)		(0.384)	
Exposure during trimester 1		1.030***		1.037***
		(0.341)		(0.300)
Exposure during trimester 2		0.598		0.785**
		(0.366)		(0.331)
Exposure during trimester 3		-0.395		0.099
		(0.340)		(0.298)
Observations	333,574	333,574	322,048	322,048
Pseudo R2	0.003	0.003	0.002	0.002

Note: All models control for month and county of birth, year of birth and year of birth squared.

Source: Swedish Interdisciplinary Panel

Table 3: Logit regression analysis output, odds ratios. Dependent variable: Attainment of a high SES occupation in 1970.

	Men	
	Model 1	Model 2
Almond exposure (birth Q1 1919)	1.015 (0.029)	
Exposure during trimester 1		1.021 (0.022)
Exposure during trimester 2		1.046* (0.025)
Exposure during trimester 3		1.009 (0.022)
Observations	322,458	322,458
Pseudo R2	0.009	0.009

Note: All models control for month and county of birth, year of birth and year of birth squared.
Source: Swedish Interdisciplinary Panel

Table 4: Logit regression analysis output, odds ratios. Dependent variable: Hospitalization during current year.

	Men		Women	
	Model 1	Model 2	Model 3	Model 4
Almond exposure (birth Q1 1919)	1.036***		1.029**	
	(0.012)		(0.012)	
Exposure during trimester 1		0.992		1.006
		(0.009)		(0.008)
Exposure during trimester 2		1.031***		0.999
		(0.010)		(0.009)
Exposure during trimester 3		1.000		1.032***
		(0.009)		(0.009)
Observations	6,928,291	6,928,291	8,772,984	8,772,984
Pseudo R-squared	0.0434	0.0434	0.0426	0.0426

Note: All models control for month and county of birth, year of birth and year of birth squared.

Source: Swedish Interdisciplinary Panel

Table 5: Cox proportional hazards output, hazard ratios. Dependent variable: All-cause mortality, age 54-90.

	Men		Women	
	Model 1	Model 2	Model 3	Model 4
Almond exposure (birth Q1 1919)	1.038***		1.012	
	(0.012)		(0.011)	
Exposure during trimester 1		0.987		0.998
		(0.009)		(0.009)
Exposure during trimester 2		1.031***		1.006
		(0.010)		(0.009)
Exposure during trimester 3		0.996		1.009
		(0.009)		(0.009)
Observations	338,408	338,408	358,264	358,264
Failures	321,923	321,923	317,680	317,680
Log pseudolikelihood	-3839840.6	-3839840.3	-3856333.9	-3856333.4

Note: All models control for month and county of birth, year of birth and year of birth squared.

Source: Swedish Interdisciplinary Panel

Table 6: Cox proportional hazards output, hazard ratios. Dependent variable: Cause specific mortality, age 54-90.

	MEN						WOMEN					
	Model 1			Model 2			Model 3			Model 4		
	Cancer	Cardio-vascular	Other	Cancer	Cardio-vascular	Other	Cancer	Cardio-vascular	Other	Cancer	Cardio-vascular	Other
Almond exposure (birth Q1 1919)	1,039	1,040	1,010				1,019	1,016	0,987			
	0,025	0,532	0,021				0,027	0,072	0,037			
Exposure during trimester 1				1,005	0,975	0,980				1,010	0,990	1,001
				0,018	0,411	0,172				0,020	0,056	0,001
Exposure during trimester 2				1.055***	1,023	1,003				1,017	0,999	1,003
				0,021	0,277	0,003				0,022	0,001	0,002
Exposure during trimester 3				1,010	0,998	0,956				0,997	1,022	1,007
				0,018	0,004	0,827				0,020	0,249	0,020
Observations	1 015 224			1 015 224			1 074 792			1 074 792		
Individuals	338 408			338 408			358 264			358 264		
Failures	289 353			289 353			254 455			254 455		
Log pseudolikelihood	-3488487,8			-3487861			-3126775,9			-3126774,1		

Note: The hazard ratios displayed for Cardiovascular and Other are obtained through multiplying the base effect (Cancer) with the corresponding interaction effect (Cardiovascular/Other). The test of statistical significance is obtained through Wald tests, compared to Chi square test statistics (df=1). Standard errors reported for the base effects (cancer mortality), Wald test statistics reported for the other causes of death.
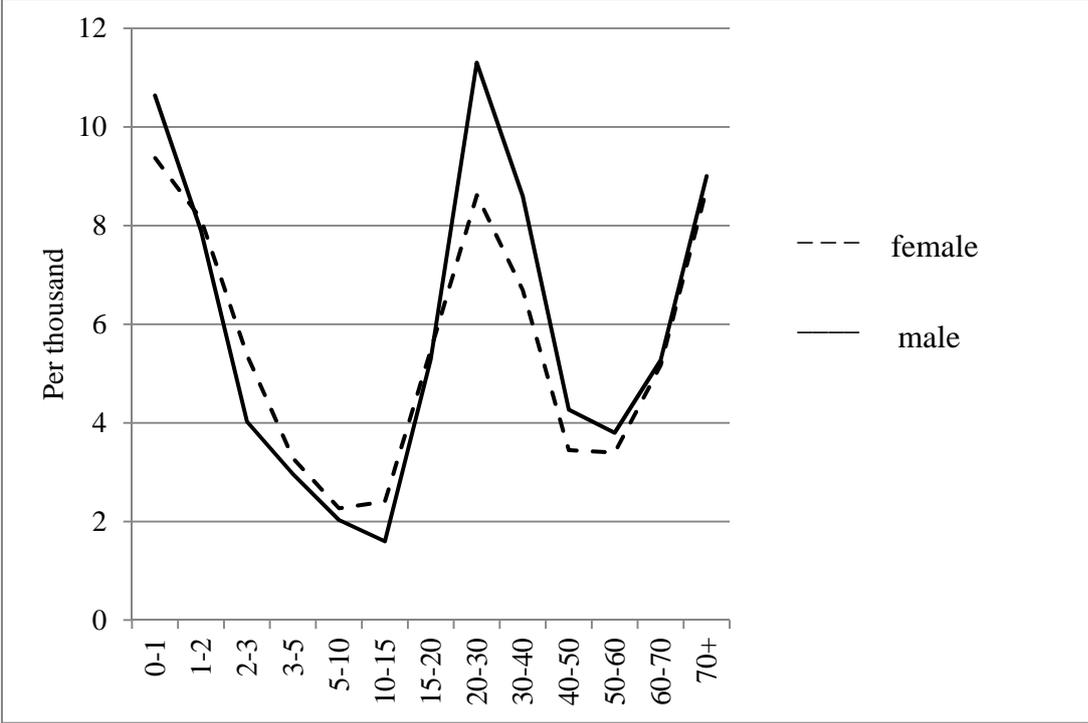
Source: Swedish Interdisciplinary Panel

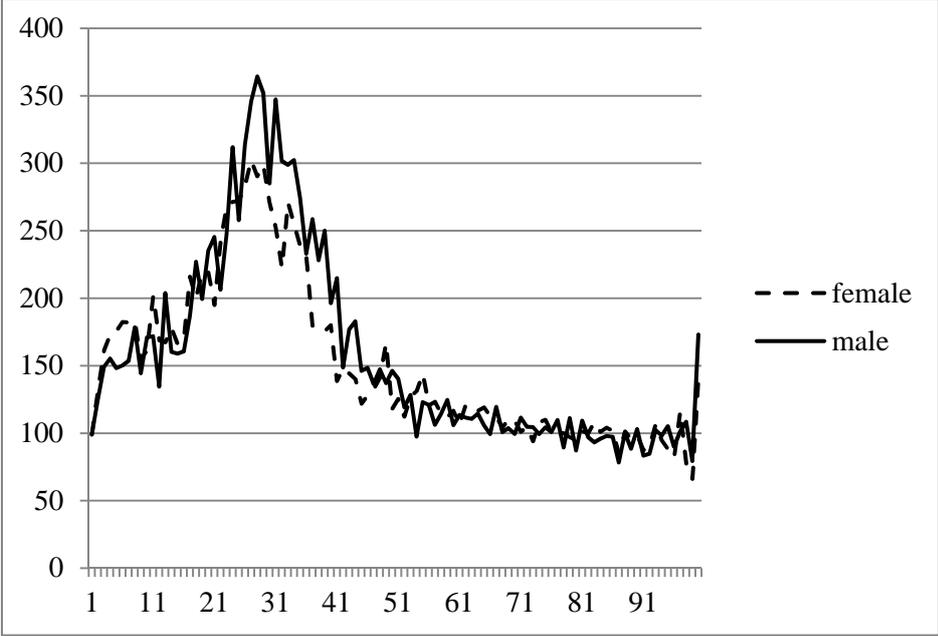Figure 1: Crude Mortality Rate (per 1,000), by county
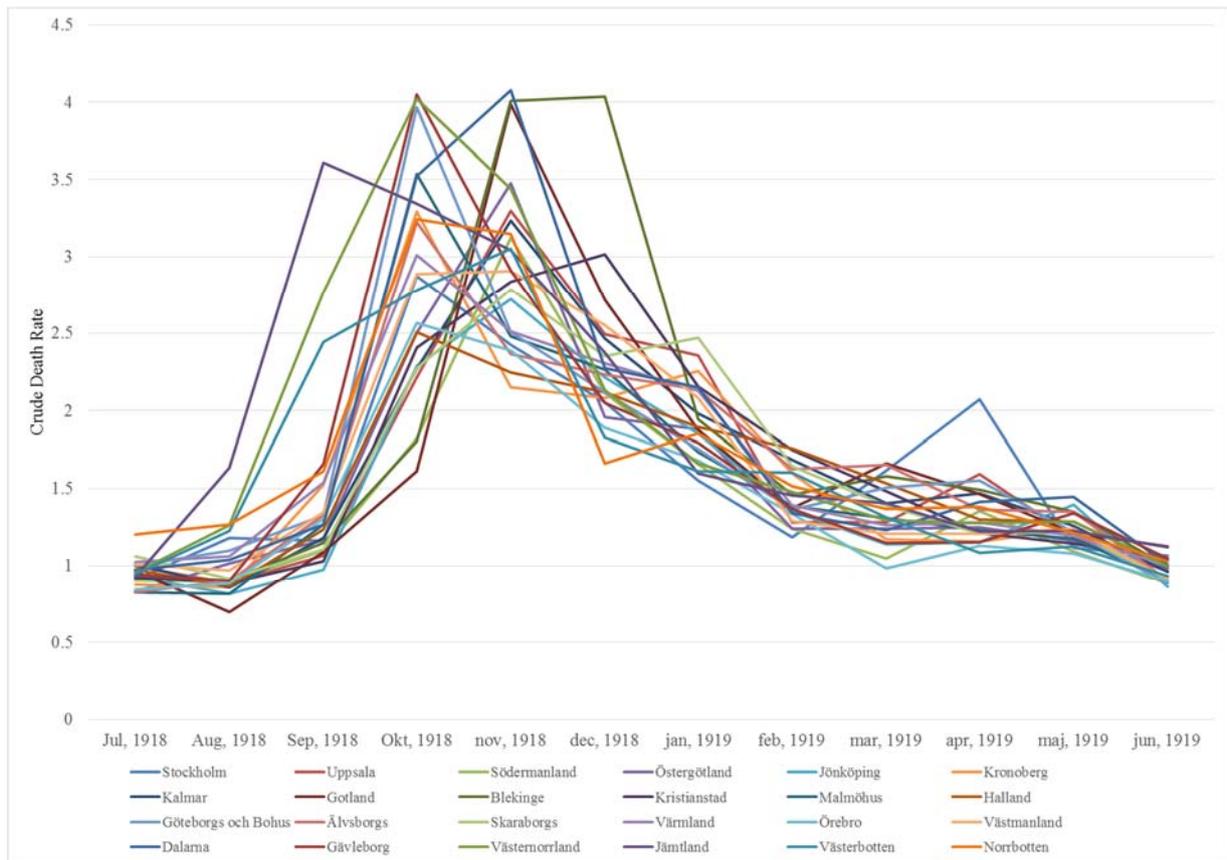
Source: Sveriges officiella statistik: Befolkningsrörelsen åren 1918-1920

Figure 2. Influenza and pneumonia mortality in 1918 by age and sex, Sweden.
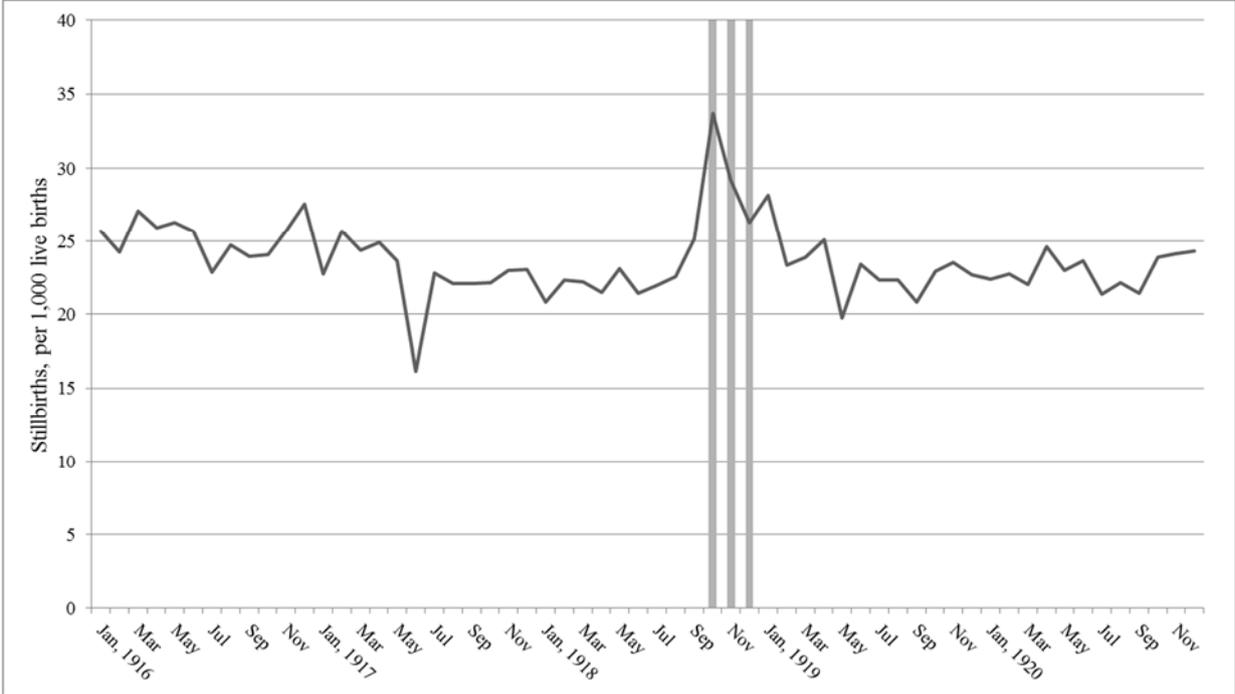

Source: Sveriges Officiella Statistik: Befolkningsrörelsen 1918.

Figure 3. All-cause mortality by age in 1918, Sweden, 1917=100

Source: Sveriges Officiella Statistik: Befolkningsrörelsen 1917, 1918.

Figure 4. Stillbirths per 1,000 live births, by month. Sweden 1916-1920. The shaded vertical bars represents the period October-December 1918.

Source: Sveriges Officiella Statistik: Befolkningsrörelsen 1916-1920.

Figure 5. Sex ratio at birth, by month, Sweden 1916-1920. The shaded vertical bars represents the period October-December 1918.

Source: Sveriges Officiella Statistik: Befolkningsrörelsen 1916-1920.

Figure 6. Influenza morbidity rate by age at different work places in Sweden.
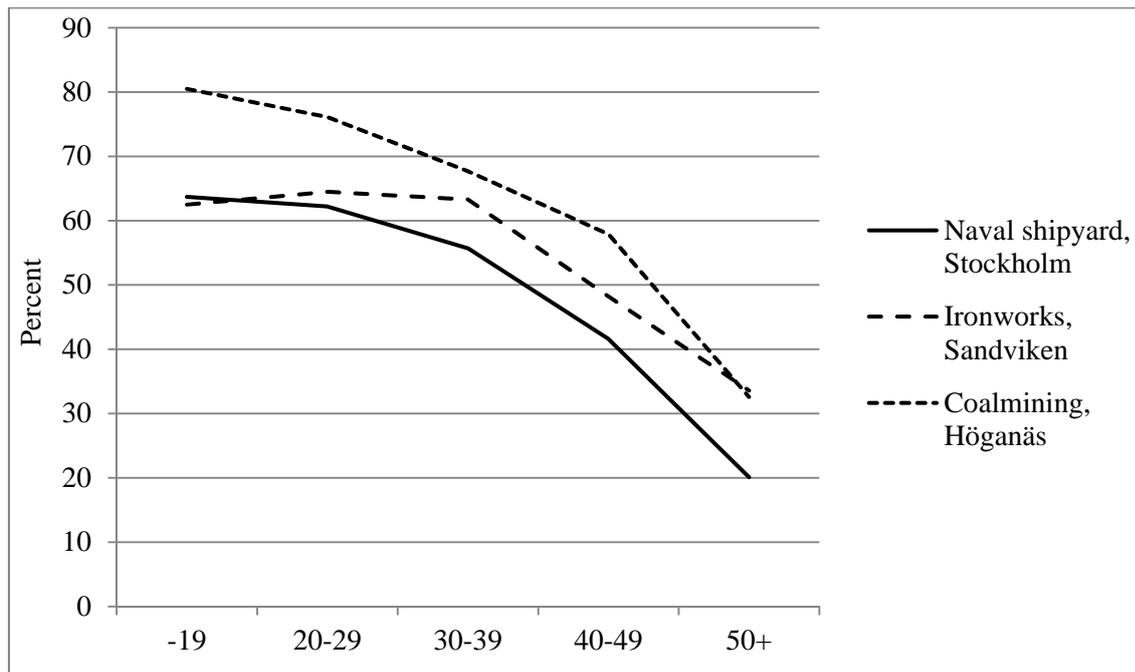
Sources: Alling 1919, Gibson 1919, Widstrand 1918.

Figure 7: Cumulative distribution of morbidity and mortality due to the Spanish flu from July 1818 to June 1919.
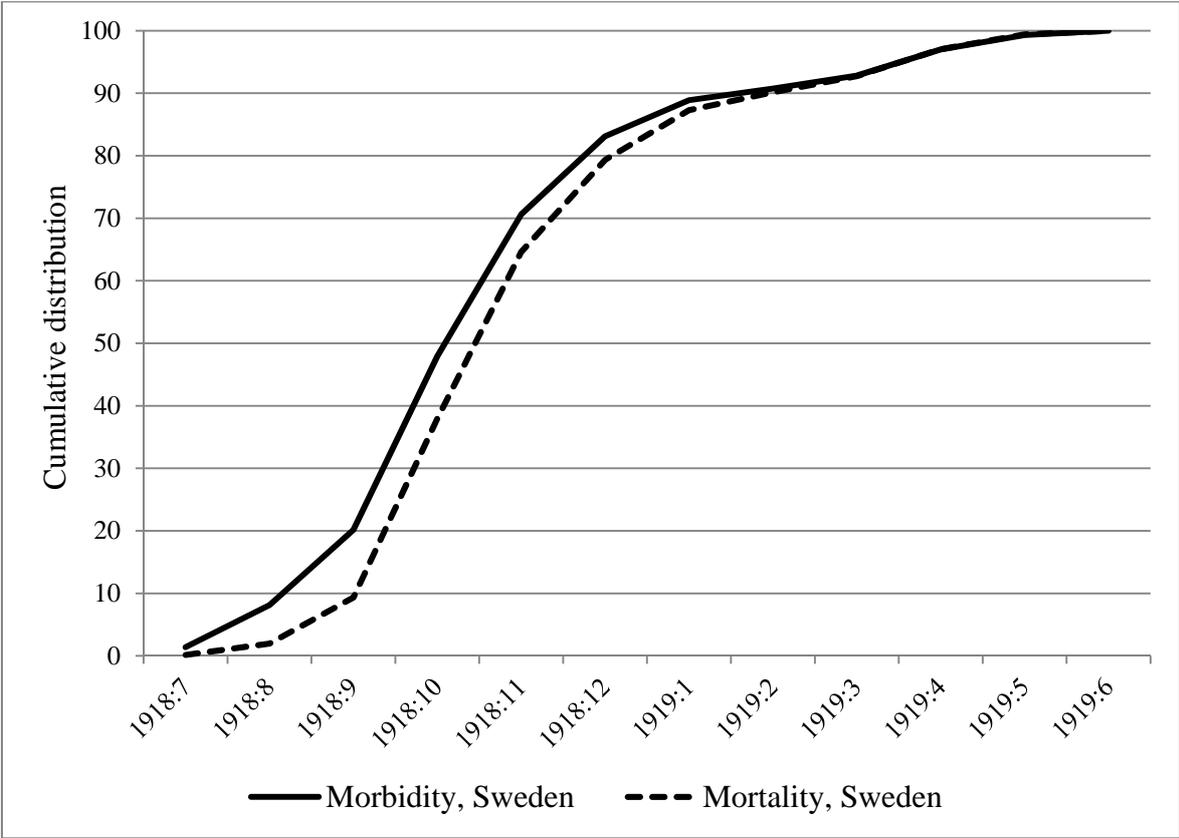
Sources: Lindhagen 1926, SOS Allmän hälso- och sjukvård år 1918, 1919, for Sweden

Figure 8. Percentage of well-off fathers of all fathers to new-born children, 1915-1920, Sweden
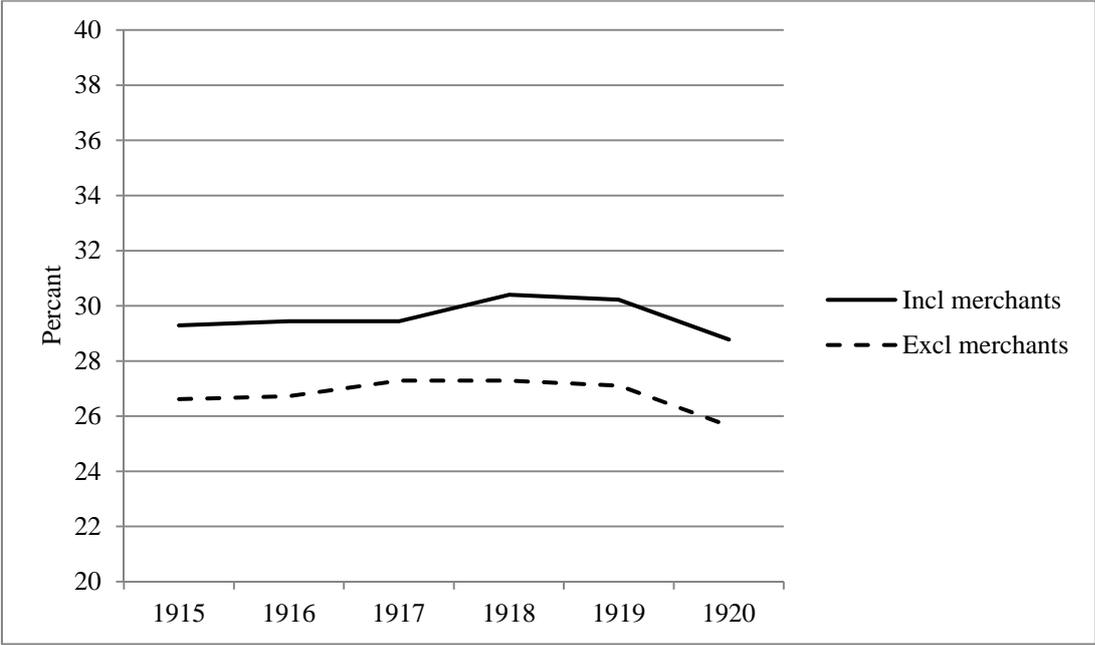

Source: Sveriges Officiella Statistik: Befolkningsrörelsen 1915-20, Table 22

Figure 9. Log mean income and share in high skilled occupation, by birth year and quarter. The shaded vertical bar represents individuals born during Q4, 1918.
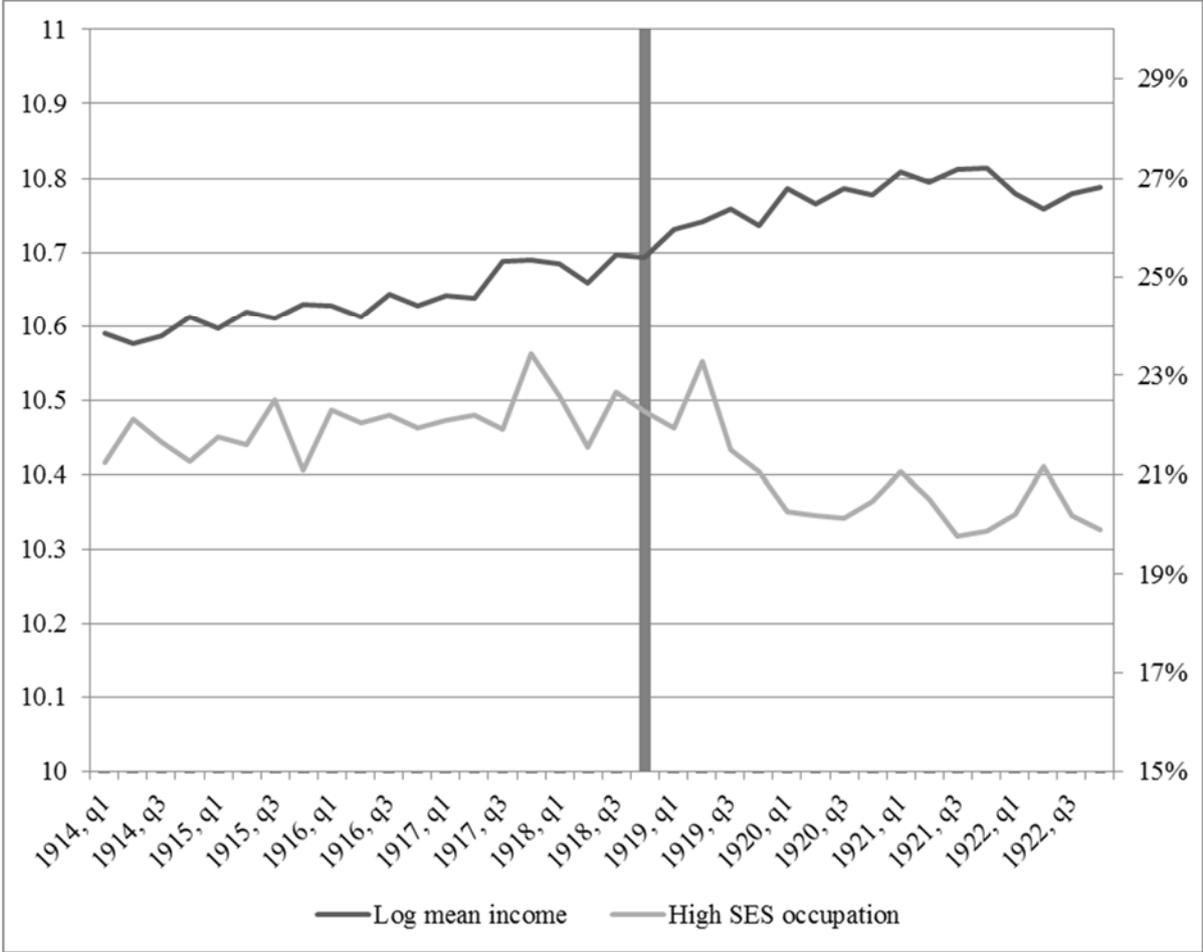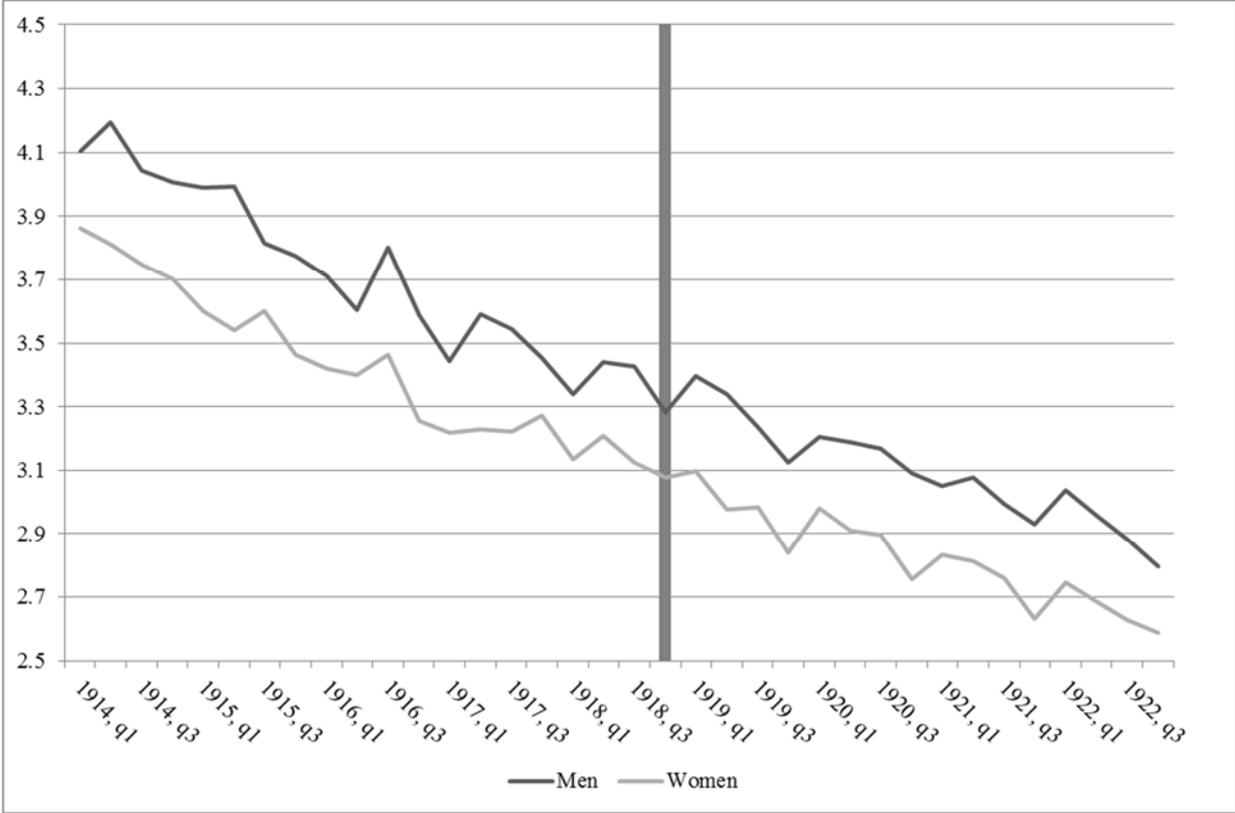
Figure 10. Share hospitalized during current year, by birth year and quarter. The shaded vertical bar represents individuals born during Q4, 1918.
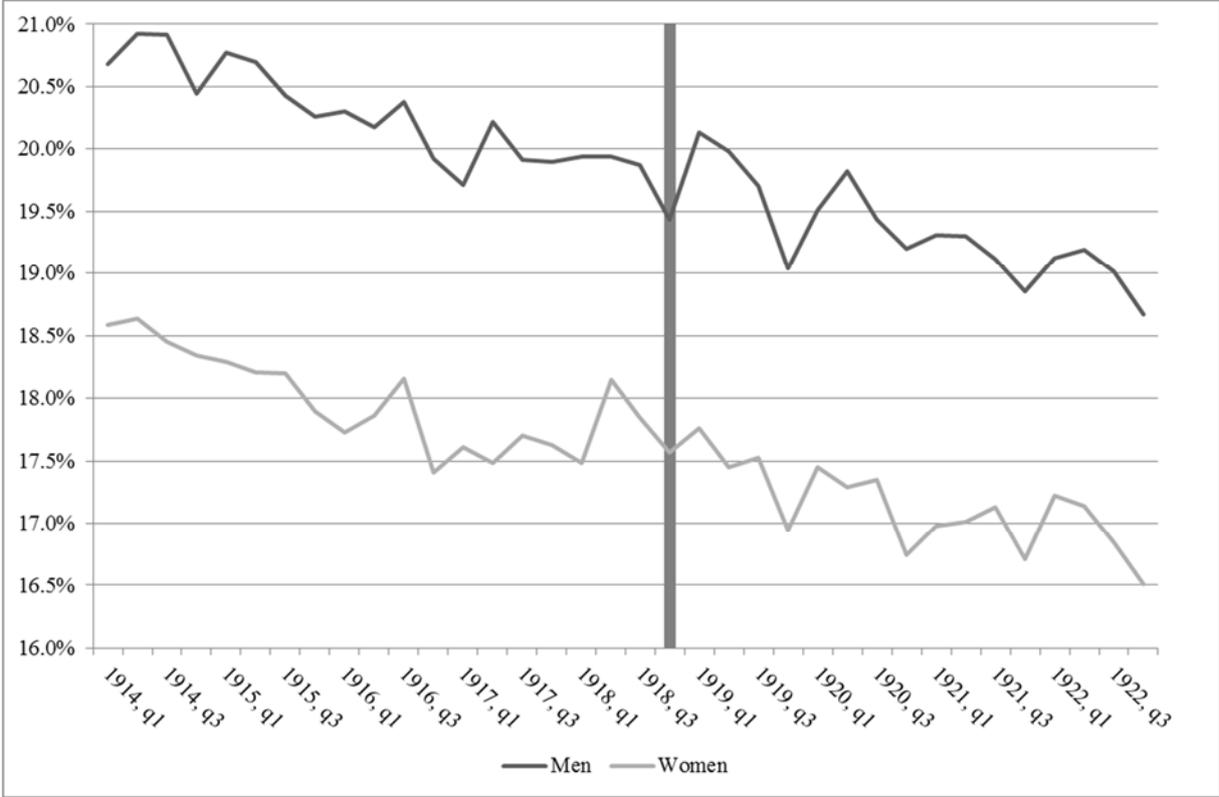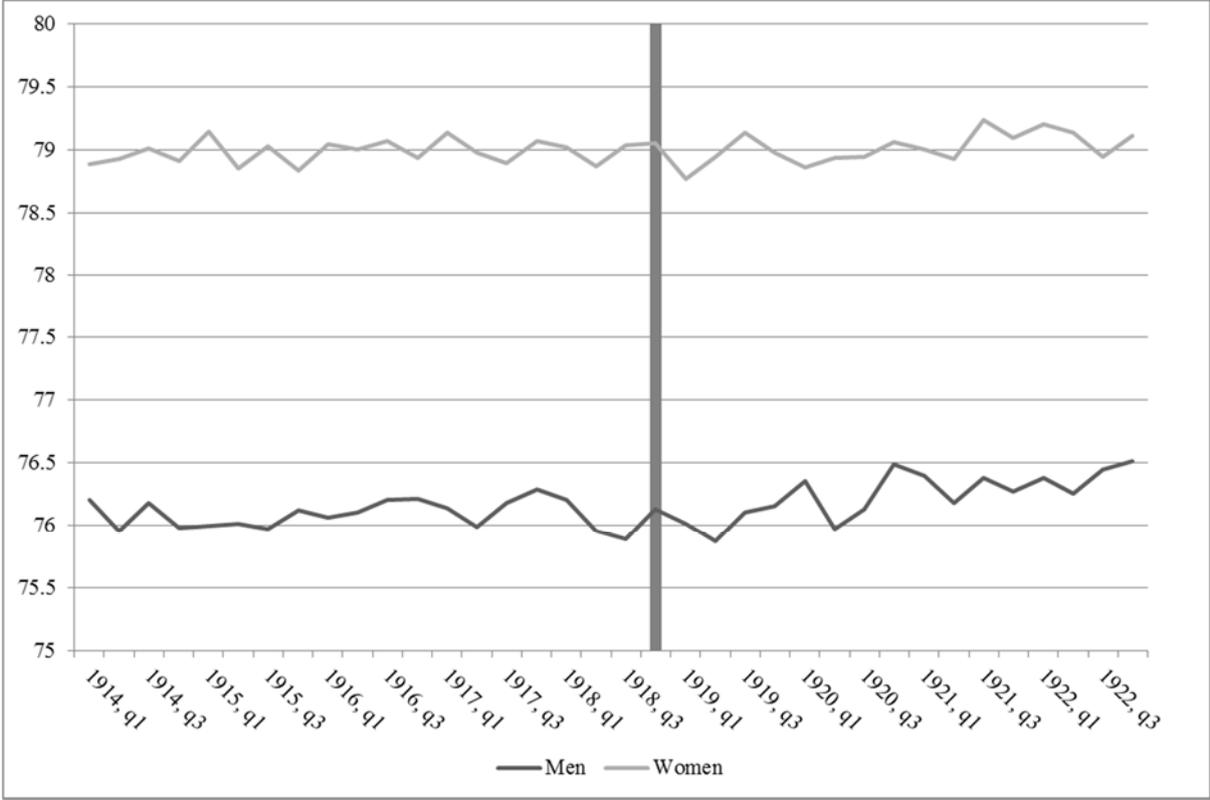

Source: Swedish Interdisciplinary Panel.

Figure 11. Mean days of annual hospitalization between the age 54 and 90, by birth year and quarter. The shaded vertical bar represents individuals born during Q4, 1918.

Source: Swedish Interdisciplinary Panel.

Figure 12. Mean age at death, by birth year and quarter. The shaded vertical bar represents individuals born during Q4, 1918.

Source: Swedish Interdisciplinary Panel.