The Lasting Legacy of Seasonal Influenza: In-utero Exposure and Labor Market Outcomes

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Abstract

Pregnancy conditions have been shown to matter for later economic success, but many threats to fetal development that have been identified are difficult to prevent. In this paper I study seasonal influenza, a preventable illness that comes around every year and causes strong inflammatory responses in pregnant women. Using administrative data from Denmark, I identify the effects of maternal influenza on the exposed offspring via sibling comparison, exploiting both society-wide influenza spread and information on individual mothers who suffer strong infections during pregnancy. In the short term, maternal influenza leads to a doubling of prematurity and low birth weight, by triggering premature labor among women infected in the third trimester. Following exposed offspring into young adulthood, I observe a 9% earnings reduction and a 35% increase in welfare dependence. These long-term effects are strongest for influenza infections during the second trimester and they are partly explained by a decline in educational attainment, pointing to cognitive impairment. This effect pattern suggests that maternal influenza damages the fetus through multiple mechanisms, and much of the damage may not be visible at birth. Taken together, these results provide evidence that strong infections during pregnancy are an often overlooked prenatal threat with long-term consequences.

JEL classifications: I10, J24, J3, J13

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1 Introduction

The Fetal Origins Hypothesis posits that pregnancy is a critical period in which the basis for later health and cognitive development is set. Maternal health behaviors during pregnancy, including smoking, drinking, and malnutrition, have been shown to have lasting impacts on the offspring's human capital development.[1] Research also shows that pollution or catastrophic events (e.g. pandemics, natural disasters, and terror attacks) affect the life trajectories of those exposed in utero, but these factors are often difficult to address via public policy. Relatively little attention has been paid to preventable infectious diseases, such as seasonal influenza, that may harm fetal development through the inflammations they cause in pregnant mothers.

Seasonal influenza is the leading infectious disease in the developed world, it comes around every winter and affects 5%-15 % of the population (Russell et al. 2008). Importantly, pregnancy renders women more susceptible and more vulnerable to strong influenza infections (Rasmussen et al. 2008; Currie and Schwandt 2013; Kay et al. 2014). Even though the virus itself usually does not pass through the placenta, it triggers a strong inflammatory response in pregnant women. The detrimental effects of this inflammatory response on health at birth have been documented by a literature dating back almost a century (Harris 1919). Still, the long-term effects on human capital development have been studied so far only in the context of devastating pandemics that are rare, unpredictable, and unpreventable (Almond 2006). This paper investigates the long-term effects of the ordinary influenza that occurs every year and is preventable via vaccination.

To that end, I use administrative data from Denmark that allows me to follow all individuals born from 1980 to 1993 and their mothers, from the pregnancy period through birth into young adulthood when labor market outcomes are observed. I exploit two separate sources of identifying variation: surveillance data of the society-wide influenza spread (based on General Practitioners' reports) that I link to the pregnancy periods of different cohorts, and information on individual mothers who are hospitalized with influenza infections during pregnancy. For both approaches, I compare siblings differentially exposed to influenza seasons or maternal infections in order to control for maternal selection into conception months and individual infections, respectively.

I start the analysis by illustrating that annual influenza waves drive the seasonality in ges-

[1] See Almond and Currie (2011) and Almond et al. (2017) for reviews of the literature. Cunha and Heckman (2007) and Heckman (2007) provide the leading model of how differences in initial endowments impact human capital development over the life cycle.

tation length that is observable across cohorts born in different months, with shorter gestation lengths for births during the peak of the influenza season. This pattern is very similar to recent evidence from the U.S. and suggests that influenza infections during the third trimester of pregnancy may trigger premature labor.

Next, I focus on influenza infections at the micro-level, observed in individual pregnant mothers. I find that these infections have strongly negative effects on the offspring both in the short- and the long-term. In-utero exposure to maternal influenza is associated with a doubling of both prematurity and low birth weight, as well as an increase in postnatal mortality. When I follow exposed children and their siblings into young adulthood, I find strong effects on labor market outcomes. Those who suffered in-utero exposure to maternal influenza infections earn about 9% less than siblings who were not exposed. Labor market participation decreases by 3 to 4 percentage points, while welfare dependence increases by 35%.

Short- and long-run effects depend in different ways on the timing of the influenza infection. Observable birth outcomes, such as birth weight and gestation length, are mainly impacted by maternal infections in the third trimester. This pattern is in line with the observed seasonality in gestation length as well as the medical understanding of how infections late in pregnancy trigger premature labor. Labor market outcomes, on the other hand, are most strongly affected by infections in the second trimester, the period of fetal development with the strongest neural brain development (De Santis and Di Gianantonio 2005; Almond et al. 2009; Black et al. 2013). Second trimester infections also decrease completed years of schooling, and including education as a control in the labor market regressions attenuates the second trimester effects. No significant effects on education appear for influenza exposure in the third trimester. These results suggest that the strong second trimester effects on labor market outcomes might run through cognitive skills. First trimester infections affect neither short- nor long-term outcomes. But the analysis of birth rates provides suggestive evidence that infections early in pregnancy might lead to miscarriages (in line with Bloom-Feshbach et al., 2011). This implies that the weakest and most affected fetuses might not be observed and that detrimental impacts on those who survive to birth might be canceled out by positive selection effects.

In a third step, I analyze long-term effects at the cohort level. Despite the strong and visible correlation of seasonal influenza spread with cohorts' average gestation length, the cohort-level approach is less powerful for the analysis of long-term outcomes. The extended timing of relevant exposure (both third and second trimesters matter) and seasonal

confounders occurring after birth (such as school entry rules) strongly reduce the amount of identifying variation. As a consequence, the analysis of long-term effects at the cohort level is less precise than at the level of individual mothers. However, the overall effect pattern is remarkably similar, both in terms of the outcome-specific timing as well as the relative effect magnitudes across outcomes. The comparison of estimated magnitudes in the two different approaches suggests that the micro-level effects are slightly stronger than the impact of the average influenza infection among pregnant women, in line with the idea that hospitalizations identify particularly strong cases, and implies a reduction in average wages of around 1% in the most exposed cohorts.

The labor market effects of exposure to strong maternal infections that I find are economically significant. A 9% earnings reduction is close to the estimated returns to an entire year of schooling in the U.S. (Card 2001). This effect size appears reasonable in comparison to several central estimates from the fetal origins literature. Almond's (2006) seminal analysis of the 1918/19 Spanish Flu pandemic documents that the offspring of mothers who caught the Spanish Flu during pregnancy had 5%-9% lower earnings in adulthood.[2] Moreover, the stronger effects of second trimester infections that I find are very similar to the effects of alcohol consumption during the first half of pregnancy documented by Nilsson (forthcoming) for Sweden (see Section 5.8). I further show that the long-run effects of maternal influenza are in a similar range as the effects of being born with low birth weight, a proxy for poor fetal development that has been prominently used in the literature.[3] Moreover, the long-run impact on earnings is in a similar range as the effects of high-quality early educational interventions (Chetty et al. 2011; Heckman et al. 2010).[4] Finally, in a recent study, Isen et al. (forthcoming) show that cohorts experiencing less in-utero exposure to air pollution following the 1970 Clean Air Act enjoyed a 1% increase in average cohort income. This implies that in terms of average effects at the cohort level, seasonal influenza waves have a similar impact on the most exposed cohorts as higher levels of air pollution had before the Clean Air Act.

This paper provides three primary contributions. It is the first to document the detrimental long-term effects of in-utero exposure to a moderate infectious disease that spreads through society every year. Previous research has mainly focused on the 1918/19 Spanish

[2]This estimate is based on a 30% infection rate and might provide a lower bound given the relatively strong increase of maternal mortality during the 1918/19 Spanish Flu (Almond 2006).

[3]For example, see Black et al. (2007), Currie and Almond (2011), and Figlio et al. (2014).

[4]Chetty et al. (2011) find that assignment to high-quality kindergarten teachers in the Project STAR increased income in early adulthood by 7%, while Heckman et al. (2010) documents a 15% income increase for the Perry Preschool intervention.

Flu pandemic, one of the biggest public health disasters in modern history, with a global death toll of 50 million (Johnson and Mueller 2002). The comparability of Almond's (2006) estimates to my results indicates that the long-term effects of strong infections during regular seasonal influenza periods are as serious as the impacts of maternal infections during a rare and devastating pandemic, conditional on survival. More broadly, the findings of this paper suggest that strong infections during pregnancy are an often overlooked prenatal threat with long-term consequences.

Second, the existing literature usually identifies long-term effects of pregnancy conditions either at the cohort level (e.g. policy changes, disasters, pollution) or at the individual level (e.g. birth weight differences between siblings). To my knowledge, this paper is the first to exploit separate identifying variation both at the cohort and at the individual level, finding reassuringly similar effect patterns across both approaches. Comparing the magnitudes of both sets of estimates sheds light on the share of the population that needs to be affected at the individual level in order to generate the cohort-level effects.

Third, the rich information provided by the Danish register data allows me to show that a given threat can damage the fetus via multiple mechanisms, depending on the timing of the infection. Part of that damage is not visible at birth and only detected in economic outcomes in the long term. This finding also supports the conclusion of Conti et al. (2016) that fetal development needs to be traced by measures above and beyond birth weight or gestation length.

Overall, my results suggest that public policies that decrease maternal influenza infections during pregnancy might have large impacts on human capital development. Pregnant women became an official risk group in the mid-2000s and cheap influenza vaccination is readily available, but the majority of women in the U.S. remain unvaccinated, often trading off a perceived marginal benefit of the vaccine against fears of dramatic negative side effects (CDC 2013). Therefore, cost-effective policy responses could include information campaigns in combination with the promotion of measures that do not rely on the vaccination of pregnant women, such as protective behavior (e.g. washing hands) and the vaccination of significant others to achieve herd immunity.

The paper proceeds as follows: Section 2 provides background information about seasonal influenza. Sections 3 and 4 describe data and methods followed by the results in Section 5. Section 6 concludes.

2 Seasonal Influenza

Influenza is a virus that mutates while circulating around the world so that previously obtained immunity is largely lost. This leads to annual outbreaks of seasonal influenza during the winter months, infecting 5%-15% of the entire population (Russell et al. 2008). Influenza pandemics occur when new influenza strains are transmitted from animals to humans. There have been four pandemics over the past century (1918/19, 1957/58, 1968/69, 2009/10), none of which fell into the time period analyzed in this study. Figure 1 shows the seasonality of influenza in Denmark based on per capita cases of influenza-like illness reported by general practitioners (GP). This index understates the scale of influenza infection because not every infected person sees a GP and not every GP delivers a report, but it is representative of the typical seasonal pattern with low influenza activity from May to August, the start of the influenza season in September, and a peak in the winter months around February. Figure A1 shows the disaggregated time series of the influenza index. Most of the variation is driven by seasonality, which is highly predictable from year to year.[5] However, there is also some variation in the exact timing and strength, depending on the mutation of the virus.[6]

It is well established that pregnancy renders women more susceptible to and more severely affected by influenza (Neuzil et al. 1998; Fiore et al. 2009; Lindsay et al. 2006; Kay et al. 2014). Understanding the biological mechanisms behind this relationship is a central question of current medical and epidemiological research (Rasmussen et al. 2008; Kay et al. 2014). While pregnancy usually suppresses a woman's immune system, Kay et al. (2014) find that immune cells of pregnant women react more strongly to influenza infections than non-pregnant women. This overreaction is a highly inefficient response, as the virus usually does not pass through the placenta, but the strong immune response causes additional inflammation that may disturb the pregnancy and harm the fetus.

While the causal pathways from influenza infections to fetal impairments are not yet fully understood, there is agreement that the mother's inflammatory response to the infection plays a key role (Kourtis et al. 2014). Strong inflammation is a relatively undirected way to fight a virus, spreading through the entire body and destroying both viral as well as non-viral

[5] Shaman and Kohn (2009) and Barreca and Shimshack (2012) suggest that absolute humidity may be one factor contributing to this seasonality.

[6] In societies with high vaccination rates, some variation in the strength of outbreaks might also be driven by the degree to which the vaccine matches the each year's influenza strain (Łuksza and Lässig 2014). However, in Denmark before the early 2000s, influenza vaccination was recommended to only a few risk groups, not including pregnant women, and patients had to pay for the vaccine (Rønne 2000). It is safe to assume that during the time period analyzed in this study, influenza vaccination rates were close to zero, in particular among pregnant women.

cells. The inflammatory response to influenza has the potential to damage not only the lungs (where most of the virus is located) but also other organs such as the heart (Neuzil et al. 1998; Warren-Gash et al. 2009) or the placenta and thus might directly impair fetal development.

Additionally, strong inflammation might activate the maternal immune system, which is hostile to the fetus due to the father's genes in the embryo.[7] The maternal immune system only tolerates the fetus due to regulatory helper cells that surround the placenta during pregnancy (Trowsdale and Betz 2006). If influenza-induced inflammations outweigh these regulatory cells, the fetus is no longer protected from assaults by the maternal immune system. As a result, the fetus might be under double attack, suffering not only from the inflammations but also from an inflammation-induced antifetal immune response.

With respect to short-term effects, influenza infections in pregnant mothers have been found to trigger adverse birth outcomes, in particular, preterm labor. These effects have been documented both for pandemics (Harris 1919; Rasmussen et al. 2008) and seasonal influenza (Steinhoff et al. 2012; Currie and Schwandt 2013), though not all studies are conclusive (Fell et al. 2017). Currie and Schwandt (2013) show in U.S. data that the overall seasonality of prematurity rates is associated with influenza spread in the month of birth, in line with an impact of influenza infections late in pregnancy when mothers near full term. For influenza infections during the first trimester of pregnancy, on the other hand, there is some evidence of an increase in miscarriage rates (Bloom-Feshbach et al. (2011)).

In terms of long-run health effects of in-utero exposure to influenza, much of the existing medical literature has focused on its relationship to mental health and schizophrenia. While the existence and the timing of such effects remains controversial, many studies find particularly pronounced effects of second trimester exposure, a period that is crucial for the development of the brain (for reviews see Ebert and Kotler 2005 and Flinkkilä et al. 2016).[8] The neural migration forming the brain and the synaptogenesis growth, i.e. the creation of new synapses, peak during the second trimester (Tau and Peterson 2010), and the maternal response to an influenza infection might impair this main growth period of the brain (Cordeiro et al. 2015).

The study of economic long-term effects has been pioneered by Almond (2006). His analysis of the economic long-term effects of the 1918-19 outbreak in the U.S. did more than provide one of the main contributions to the economic literature on the early origins

[7]The father's genes make the fetus a "semiallogenetic" object that would be immediately rejected by the mother's immune system if it was, for example, a mismatched organ transplant (Trowsdale and Betz 2006).

[8]Animal studies provide experimental evidence of the detrimental effects of maternal influenza on brain development (Shi and Patterson 2003; Fatemi et al. 2005; Short et al. 2010).

of human capital development. It also initiated a branch of follow-up papers studying long-term impacts of pandemics in other countries or other times. Nelson (2010), Percoco (2016), Richter and Robling (2013), Neelsen and Stratmann (2012), and Lin and Liu (2014) find similar effects of the 1918-19 pandemic for Brazil, Italy, Sweden, Switzerland, and Taiwan, respectively, mitigating concerns that the long-term effects measured in the U.S. could simply represent a cohort effect, driven by WWI veterans returning home (Brown and Thomas 2011). In another subsequent paper, Kelly (2011) analyzes U.K. cohorts that were exposed to the more moderate 1957/58 "Asian Flu" pandemic, mainly during their second trimester. She finds only marginal effects on birth weight while the effects on educational outcomes are stronger and detectable in the overall sample. My finding that second trimester exposure is not detectable in birth outcomes despite a strong effect on human capital development can explain this effect pattern.

In sum, there is accumulated evidence that pregnant women face an elevated risk of severe influenza infections and that these infections induce a strong inflammatory response that triggers adverse birth outcomes. Early in pregnancy this inflammatory response may lead to miscarriages. Less is known about potential long-term health effects, and the analysis of economic long-term effects has so far been limited to pandemics.

3 Data

My primary data source is the birth records of all Danish births from 1980 to 1993 obtained from the Danish Medical Birth Register. For the analysis of long-term outcomes, the Birth Register is linked to the Income Register and the Population Register for the overall adult population (age 18+) from 1980 to 2012 when the sample cohorts were between ages 19 to 32. In order to obtain maternal characteristics that are not contained in the Birth Register, the Income and Population Registers are also linked to the mothers delivering births. Further, I use the National Patient Register, which is also available from 1980 to 2012, for the population age 18 and above, to link births to maternal hospitalizations during pregnancy.

3.1 Natality data

The Birth Register provides information on each newborn, such as the exact date of birth, parity, gender, gestation length and birth weight, as well as information about the mother such as age and a personal identifier. A central variable of my analysis is the date of conception, which I calculate by subtracting the gestation time, recorded in weeks, from the

exact date of birth. Births with missing gestation length are excluded. This affects 2.8% of the sample. Further I omit multiple births, which affects 2.3% of the sample. Gestation length and birth weight are chosen as the main birth outcomes of interest since these are the most commonly examined birth outcomes and they have been associated with health and economic outcomes later in life (Almond and Currie 2011). The personal identifier of the mother allows siblings to be matched in the birth data.

3.2 Influenza measures and the National Patient Register

The influenza surveillance measure available for the analyzed time period is the index based on GPs' reports of influenza-like illness (ILI) mentioned in the background section. These ILI cases include patients with symptoms that subjectively appear to be influenza-related to the GP, such as diagnosed influenza or unspecified viral or respiratory symptoms, as well as conditions resulting from influenza infections such as pneumonia. The Danish Staten Serum Institute, which provided these data, cautions that the collection of the general practitioners' reports and their digitalization impart measurement error. The availability of this ILI index is exceptional for that time period (e.g. for the U.S. no surveillance data exists before the mid-1990s). To date, ILI reports remain the international standard for influenza surveillance (Lazer et al. 2014), as influenza is rarely tested for in patients admitted to the hospital (Rothberg et al. 2008).

To identify influenza-like illness infections in pregnant women I merge the birth data with the National Patient Register. This register provides information on the universe of somatic (i.e. non-psychiatric) hospital admissions in Denmark and is considered the most comprehensive hospital register worldwide for my observation period (Lynge et al. 2011). It reports personal identifiers, admission and discharge dates as well as ICD-8 codes for one or more diagnoses. Following the literature (e.g. Babcock et al. 2006), I define ILI to include the diagnosis codes for influenza (ICD-8: 470-474), pneumonia (ICD-8: 480-486), unspecified respiratory symptoms (ICD-8: 783) and unspecified viral symptoms (ICD-8: 079). Notice that influenza is the most common pathogen of pneumonia during pregnancy (Goodnight and Soper 2005). I provide results for an extended ILI definition, which includes codes for the common cold, acute sinusitis, and acute tracheitis, all of which can be caused by influenza (ICD-8: 460, 461, 464). I also provide results for a reduced ILI definition, which excludes unspecified symptoms. Using mother's personal identifier and the admission date, I match ILI hospitalizations to a woman's overall pregnancy as well as to individual pregnancy trimesters. I refer to ILI as "influenza" in the remainder of the paper.

3.3 Population and income registers

The Population and Income Registers provide information on the long-run labor market and educational outcomes of the newborn. Labor market outcomes are measured at the end of the year and include accumulated wages, income (wages plus non-wage income such as government transfers), and the employment status that an individual held for the largest part of the year. I use income and wages in logarithmic terms, excluding non-positive values.

Educational information is provided in the Population Register, including the date of the most recently awarded degree, the total years of education leading to that degree, as well as a variable that indicates whether the individual was enrolled in formal education on October 31 of the previous year.[9]

I further use the Population and Income Registers to merge socio-economic mother characteristics that are not contained in the birth register. These include the mother's origin as well as her municipality of residence, education, marital status and income, measured at the year of birth.

3.4 Sample restrictions and summary statistics

The sample is restricted to conceptions between January 1980 and December 1992 to mothers age 18 or older. Maternal hospital admissions, a central variable of this analysis, are not observed before January 1980 or for mothers under age 18. December 1992 is chosen as the end point of the sample period in order to obtain a balanced number of included conception months. Notice that the chosen conception period also guarantees that all births of a given conception month are observed in the available birth data.[10] As mentioned above, I further exclude multiple births and births with missing information on the gestation length.

These restrictions yield a sample of 719,854 births. Table 1 (A) shows that 64% of children have a sibling born in the sample period. Because my main estimation strategy is based on sibling comparison, I restrict my analysis sample to these cases, yielding 460,618 births. Table 1 (B) indicates that in this sample, mothers are observed on average giving 2.2

[9] I use this variable as an indicator for current school enrollment given that the lagged academic year covers more than the first half of the current year. Alternative timing, e.g. matching school enrollment on October 31 to the same year, leads to similar results (though it does not allow us to include 2012 data for which school enrollment on October 31 is not available). Years of schooling are interpolated for years in which no degree is awarded. For example, if an individual received a 12-year degree in 2008 and a 14-year degree in 2010, I assign 13 years of education for year 2009.

[10] For example, for conceptions in April 1979, a month not included in the sample, only full-term births would be observed in January 1980. Likewise, for conceptions in April 1993, only preterm births would be observed up to December 1993.

births; 29% of the newborns are female. 4.2% are born preterm and 3.9% with low birth weight. As a comparison, the prematurity rate in the U.S. in 1985 was more than twice as large (9.7%), while the low birth weight rate was about 50% above the Danish rate (5.9%). The Danish sample is more comparable to the U.S. subgroup of white women with a high school education (Chen et al. (2016)). Of these births, 2.2% are not observed at age 18+, while in 1.56% of cases the newborn is not observed but a sibling is. The latter share proxies for the 18-year mortality rate per 100 children (age 0 to 17). The respective mortality rate for Denmark overall between 1980 and 1992 is 1.3 (HMD 2016), including only-children, which tend to have lower mortality. Virtually all mothers have been hospitalized during pregnancy (98.3%, including birth), while 40% of deliveries involve some form of complications (ICD-8: 651-659). Foreign-born mothers make up 6.4% (compared to 15% in the U.S.), and the average age is 27.2.

Panel (C) of Table 1 shows descriptive statistics for matched long-run outcomes during young adulthood. There are in total 3,423,350 matches, or 7.43 observations per individual. Average age is 22.11, given that only earlier-born cohorts are observed at higher ages. About half of the sample is enrolled in school (the age distribution of that variable is similar to schooling rates of whites in the 2010 U.S. Census), with an average of 12.4 years of education. Labor force participation is 63% (either employed or unemployed) and less than 1% are unemployed, but for 4.4%, welfare receipts are the main income source. Labor market outcomes in the subsample of individuals who are not enrolled in school are reported in the bottom row of Table 6. In this subsample, 74% are in the labor force, while unemployment and welfare rates are 2% and 8%, respectively.

3.5 Influenza seasonality and gestation length in Denmark

In Figure 2, I replicate the analysis of Currie and Schwandt (2013) using the Danish data. The coefficients from regressions of gestation length and the influenza spread on month of birth dummies are plotted. The regression also includes mother fixed effects in order to control for maternal selection (see Appendix Section A for details). Figure 2 shows that gestation length and influenza spread at the month of birth are virtually mirrored. Gestation lengths are shorter for cohorts born during the influenza season, with a pronounced trough in February when influenza spread peaks. Figure 3 shows that this seasonality in gestation length is more pronounced in years with stronger influenza seasons. This pattern is very

similar to the seasonal effects documented by Currie and Schwandt (2013) for the U.S.,[11] indicating a negative short-run effect of influenza exposure toward the end of pregnancy on a cohort's average gestation length.

3.6 Individual-level Influenza cases

Table 2 shows the number of influenza cases that I can identify in the data. There are 1,589 cases of influenza hospitalization during pregnancy (referring to at least one admission per pregnancy) in the overall sample and 1,008 cases in the sibling sample. The reduced and extended influenza definitions subtract and add about a third of cases, respectively. In the sibling sample, there are 123 hospitalizations during the first trimester of pregnancy, 207 during the second, and 716 during the third. The sum of influenza cases by trimester is slightly larger than the number of influenza cases during pregnancy because some women are hospitalized with influenza in more than one trimester. Given infection rates of 5%-15% in the overall population (and unlikely lower rates in a risk group like pregnant women), these cases of influenza hospitalizations (about 0.24%) are, as one would expect, only the "tip of the iceberg." Not all pregnant women with influenza infections will be hospitalized. In cases of hospitalization, the doctor may overlook influenza symptoms, for example, when focusing on labor-related symptoms late in pregnancy. And doctors may not code all diagnosed symptoms. Compensation based on diagnosis-related groups (DRG) was introduced in Denmark only in 2000 and before that, i.e. during my sample period, doctors had little incentive to code their diagnoses accurately. Even for the years after the introduction of DRGs the Patient Register is considered incomplete (Lynge et al. 2011). This kind of measurement error is likely to attenuate my estimates towards zero because many mothers that I count as influenza-free might actually have been infected during pregnancy. On the other hand, mothers with more severe influenza infections are probably more likely to be hospitalized and accurately diagnosed. In this case, my measure tends to identify particularly severe infections that might have a stronger effect on the fetus than the average influenza infection. Figure 4 compares the fraction of pregnant women hospitalized with an influenza diagnosis with the seasonality of the national influenza index during weak and strong seasons. As is apparent, maternal influenza cases follow the seasonal pattern neatly, both in weak and strong years. This pattern suggests that despite likely measurement error, identified cases do represent seasonal influenza. Moreover, I show that using the national influenza index

[11] Currie and Schwandt (2013) report a difference of -0.08 weeks between the strongest and least affected month for birth data from New England over the past two decades in a partially vaccinated population, while Figure 2 shows a difference of -0.12 weeks for Denmark during a time with essentially no influenza vaccination.

instead of individual level infections in the outcome regressions yields very similar effect patterns.

4 Empirical approach

The following empirical model is used to analyze the effect of maternal influenza infections during pregnancy on birth and long-term outcomes.

$$Y_{i,t} = \alpha + \beta Influenza_i + \mu_{mom} + \delta X_{i,t} + \epsilon_{i,t} \quad (1)$$

where $Y_{i,t}$ are measures of health at birth as well as labor market and educational outcomes at different ages in early adulthood. $Influenza_i$ is a dummy indicating maternal influenza hospitalizations during pregnancy. μ_{mom} are mother fixed effects. $X_{i,t}$ are dummies for parity, the gender of the newborn, mother's age group ($<$20, 20-24, 25-34, $>$35), education ($<$10, 10-12, 13-16, $>$16, missing), four regions of residence, and marital status, the conception year and the conception month. In the long-run regressions I additionally add fixed effects for the calendar year and the current age. Standard errors are clustered at the municipality level in short-run regressions and at the individual level in the long-run regressions to account for multiple observations per individuals. I also present regressions in which I include separate dummies for influenza hospitalizations during the first, second, and third trimester.

4.1 Identification and balancing tests

β measures the effect of maternal influenza hospitalizations on the offspring's health at birth and human capital development if these hospitalizations are orthogonal to factors that may affect child outcomes independently, e.g. predetermined maternal characteristics. One reason this orthogonality assumption might not hold is that different types of women tend to conceive in different months (Buckles and Hungerman, 2013, Currie and Schwandt, 2013) and therefore experience the influenza season at different points in pregnancy. Further, not every pregnant woman may be at the same risk of contracting influenza and being hospitalized in a given month. It is therefore important to include mother fixed effects that control for the mother's type, comparing siblings born to the same mother. However, there might still be time-varying mother characteristics such as the mother's region of residence, her marital status, or her income that may affect the likelihood of contracting influenza as well as child outcomes in a direct way.

Researchers often test the orthogonality assumption by adding potential confounders as controls on the right-hand side of the regression equation. If the coefficient of interest does not move much, the estimate is considered to be reliable. Pei et al. (2017) show that balancing regressions that use these controls as a dependent variable generally provide more powerful orthogonality tests. The power discrepancy between balancing regressions and the coefficient comparison tests is particularly large if the control variables are poorly measured proxies of the true underlying potential confounders, which is usually the case (there would not be an identification problem to begin with if all confounders were available and well measured). I therefore start the analysis with balancing regressions for several maternal characteristics, both with and without including mother fixed effects. These balancing regressions show whether there is a selection of mothers into influenza hospitalizations and to what extent sibling comparisons eliminate this selection.

4.2 Cohort analysis

Regressions of short- and long-run outcomes on measures of society-wide influenza spread provide an alternative specification that does not rely on influenza infections of individual mothers. The starting point of this paper was the observation that the seasonality of average gestation lengths in the population is negatively correlated with influenza seasonality, suggesting influenza induces preterm labor when mothers are infected late in pregnancy. It it therefore a natural next step to extend this cohort-level approach to further outcomes. However, there are two complications when linking society-wide influenza spread to other short- as well as long-term outcomes. First, outcomes might be differently affected by infections at different parts of pregnancy. At the same time, the average spread of influenza at different parts of pregnancy is highly correlated. Due to the strong seasonality of influenza, months of high influenza spread are always preceded by months with low influenza spread. This makes it difficult to identify trimester-specific effects from the overall influenza seasonality. A second complication for the analysis of long-term outcomes are other seasonal effects that occur between birth and young adulthood, such as differences in health and human capital accumulation due to school entry laws (Bedard and Dhuey 2006; Schwandt and Wuppermann 2016). These confounding factors make it impossible to attribute seasonal variation in labor market outcomes to in-utero exposure to influenza waves that follow the typical seasonal pattern.

Using deviations in the society-wide influenza spread from its average seasonal patterns addresses both of these complications. Figure A.1 shows monthly GP reports of influenza-like illness (a disaggregated version of Figure 1), illustrating variation in both the strength

and in the timing of annual influenza waves. The deviations in influenza spread from its average seasonality are largely unpredictable (Viboud et al. 2003) and thus provide exogenous variation. At the same time, these residual fluctuations in a single time-series provide less variation, diminishing the statistical power of such analysis. Also, the focus on deviations increases the role of measurement error in this measure based on subjective reports of GPs, potentially leading to attenuation. Finally, only a minority of mothers suffer influenza infections, implying small differences in average outcomes that are difficult to detect even if individual influenza infections have strong effects.

With these caveats in mind, I provide regressions based on equation (1) but with the average monthly influenza spread, $FluIndex$, in the first, second and third trimester of pregnancy (matched via month of conception) instead of an indicator for individual influenza infections:

$$Y_{i,t} = \alpha + \beta_1 FluIndexT1 + \beta_2 FluIndexT2 + \beta_3 FluIndexT3 + \mu_{mom} + \delta X_{i,t} + \epsilon_{i,t} \quad (2)$$

The same control variables as in the micro-level regressions are used, including dummies for the month of conception, which partial out average seasonality. Additionally, $X_{i,t}$ includes a quadratic time trend at the conception date level to account for non-linear time trends that would confound monthly variation within individual years. Standard errors are clustered at the year-month cells to account for the fact that the influenza index only varies at that level.[12]

4.3 Fertility analysis

Previous research analyzing the 1918/19 Spanish Flu pandemic suggests that maternal influenza infections during the first trimester might increase the risk of miscarriages (Bloom-Feshbach et al. 2011). Moreover, Almond et al. (2016) find miscarriage effects of morning sickness, usually a milder condition than strong influenza infections. In the context of my analysis, first trimester effects on miscarriages would be relevant, implying that those who are most affected are not observed in the birth data. Moreover, if the weakest fetuses are at the highest risk of miscarriage, those who survive first trimester exposure might be pos-

[12]Monthly GP reports only exist at the national level for the time period of this analysis. I can construct regional indices using hospitalization data, but these are not predictive of individual infections if I control for the national level index (as one would expect, given that influenza spreads quickly in a small country like Denmark and regional deviations might largely reflect measurement error or variation in reporting rather than actual cases).

itively selected, biasing estimates against finding negative effects. However, miscarriages early in pregnancy are not observed in my data. Early on, mothers might not even know themselves that they are pregnant and pregnancy related hospitalization rates, such as prenatal care checks, appear in the data only towards the second trimester. However, I can identify influenza cases among women of fertile age and test for a relationship with birth rates in the months after the infection in the following way.

For every influenza hospitalization of a fertile-age woman that I observe in the data, I match three control group women who are born in the same year and month as the infected woman. I then follow the influenza and control group women over the course of 36 months, and regress separately for each month a dummy indicating the occurrence of a birth in that month on a dummy indicating the past influenza infection. In total, I identify 13,978 women with influenza infections (including women that do not give birth subsequently), which are matched to 41,837 control women.

5 Results

5.1 Balancing tests

Table 3 presents balancing regressions of different maternal characteristics on influenza hospitalizations during pregnancy. Panel A shows coefficients from bivariate regressions that do not include additional controls. These regressions indicate a considerable degree of selection suggesting that disadvantaged women are more likely to be hospitalized with influenza during pregnancy. Mothers who are admitted are significantly more likely to be foreign born and less educated, and they have 18% lower earnings. They tend to live in more populated areas, and there is no significant association with marital status or age. Panel B repeats the balancing regressions with a broad set of controls, including all maternal characteristics except the respective dependent variable. Coefficients decrease somewhat compared to the bivariate regressions, but they remain strongly significant. These results suggest that the inclusion of observable mother characteristics does not eliminate selection into influenza hospitalizations during pregnancy.

Panel C of Table 3 shows regressions that additionally include mother fixed effects, comparing maternal characteristics between different pregnancies of the same mother. Since a mother's origin is constant over time, mother fixed effects perfectly control for this characteristic, and it is not possible to estimate this regression. The other characteristics are time varying so they might systematically correlate with influenza hospitalizations. However,

estimates are small and insignificant in all cases, suggesting that the inclusion of mother fixed effects does effectively control for maternal selection into influenza during pregnancy. Further balancing tests, by trimester of influenza infection, are reported in Table A.2 and discussed below.

5.2 Short-term effects on birth outcomes

Panel A of Table 4 shows effects of influenza during pregnancy on birth outcomes. All regressions include baseline controls for mother and birth characteristics and mother fixed effects. These regressions indicate that infants who were exposed to maternal influenza infections in utero have poorer health at birth compared to their siblings who were not exposed. Gestation is reduced by about a third of a week, increasing the risk of preterm birth by 4.5 percentage points. Further, they weigh 84.5 grams less and are 3.5 percentage points more likely to have low birth weight. These effects are large. Given baseline rates of 4.2% and 3.9%, prematurity and low birth weight rates are about doubled.[13]

A transparent way to illustrate a binary treatment effect is to show the distributions of the treatment and the control outcome in the raw data. A raw data plot does not allow us to control for covariates in the same way as multivariate regressions, but in the current setting, the most relevant control is the mother fixed effect. This fixed effect can be conveniently accounted for by plotting the distributions only for sibling pairs where at least one sibling is exposed and at least one is unexposed. Figure 5 shows the birth weight distribution for exposed and unexposed siblings. The distribution for exposed siblings is shifted to the left, with a mean difference of 81 g, which is close to the point estimate of 84.5 g reported in Table 4 (the Kolmogorov-Smirnov test for equality of distribution is rejected with p=0.021). Moreover, the distributions diverge particularly strongly below the 2500 g cutoff, in line with the strong effect on the rate of low birth weight.

The "fragile males" hypothesis prominent in the epidemiological literature suggests that fetal shocks tend to harm male more than female fetuses, leading to a higher proportion of fetal losses among male fetuses and thus a negative effect on the share of boys born (Kraemer 2000). Column (5) of Table 4 shows that the estimated effect on the probability of a male newborn is indeed negative, but it is imprecisely estimated and the effect is not significantly different from zero, suggesting that maternal influenza infections do not have a strong effect on gender-specific abortions. The last column of Table 4 indicates that exposure

[13]OLS regressions without mother fixed effects are reported in Table A.3. As expected given the negative selection documented in the balancing regressions, effects on birth outcomes tend to be larger.

to maternal influenza increases the likelihood of not being observed at age 18 or above by 1.6 percentage points. Given the siblings comparison framework, this effect refers to cases when the influenza-exposed sibling is not observed but the unexposed sibling is, proxying for mortality (see discussion of Table 1). The flu-induced doubling of this rate (from a 1.6% baseline, see Table 1) points to a considerable impact on postnatal health. But compared to the mortality effect of low birth weight this effect is relatively moderate (see discussion of Table 10 below).

Much of a fetus' weight gain occurs during the last weeks of pregnancy. One question is therefore, whether the effect on birth weight is simply driven by the shortening of the gestation length or whether there is also an independent effect conditional on the pregnancy length. Panel B of Table 4 shows regressions including dummies for individual weeks of gestation to control flexibly for gestation length. The coefficients in the birth weight regressions attenuate considerably and become insignificant, suggesting that much of the effect runs through gestation length. At the same time, the point estimate of -26.4 g in the birth weight regression, one-third of the unconditional effect in Panel A, points to a potential residual impact on birth weight that might go beyond the reduction mechanically induced by the shortening of the pregnancy period. Similarly, the effect on postnatal mortality in column (6) attenuates and becomes statistically significant, but taking the point estimate at face value suggests that a considerable share of the overall effect might not be induced by the impact on gestation length.

5.3 Short-term effects by trimester of exposure

To further explore the structure of influenza's impacts on birth outcomes I analyze the timing of the influenza infections in Table 5, which shows regressions of birth outcomes on dummies indicating influenza hospitalizations during the first, second, and third trimester. Remarkably, the effects on gestation length and prematurity are entirely driven by infections during the third trimester, which have a strongly significant impact, while effects are insignificant and close to zero for the second trimester and reversed (and insignificant) for first trimester infections. This is in line with the existing literature and the cohort level results in Figures 2 and 3, which suggest that influenza infections trigger premature labor late in pregnancy but not at earlier stages. A similar pattern is observed for the effects on birth weight, with significant impacts only for third trimester infections, though second trimester point estimates are less attenuated than for the gestation effects. This pattern suggests that part of the birth weight impacts not running through gestation length (discussed in the previous ta-

ble) might originate from second trimester infections. Not surprisingly then, controlling for gestation length in Panel B of Table 5 has more of an effect on the third than on the second trimester effects, even though there is also some attenuation of the latter.

5.4 Long-term effects on labor market outcomes

The analysis so far has documented medically plausible, strong effects of maternal influenza infections on health at birth that differ by the timing of the infection. How do these in-utero impairments impact economic long-run outcomes when entering the labor market? One complication in the context of the Danish data is that due to the young age of the analyzed cohorts (18 to 32), about half of the sample is still in school with many of those in school having wage incomes from some side job. Since these student jobs typically pay less, more able individuals who stay in school longer might be associated with lower incomes than those who have already fully entered the labor market, implying a downward bias. In Table 6, I therefore present results both for the overall sample as well as for the subsample of individuals who are out of school.

The estimate in the first column Table 6 indicates that the wages of young adults who were exposed to influenza infections in utero are about 9% lower than those of their siblings who were not exposed. This is a strong effect, similar in size to the return of an entire year of schooling (Card 2001). The effect on overall income including government transfers in column (2), on the other hand, is less than one-third the size of the wage effect and it is not significantly different from zero. And the estimate in column (3) shows that non-wage income, i.e. the difference between an individual's overall income and wage that mainly represents government transfers, increases by 12.6%. These results suggest that the Danish social security system largely compensates for the ability differences caused by in-utero exposure to influenza. A similar role of social safety nets moderating fetal impairments has also been documented for Sweden in two recent studies (Bharadwaj et al. 2015; Nilsson forthcoming).

Columns (4) to (6) show effects on employment status. There is a strongly significant, negative effect of about 2.8 percentage points on the probability of being in the labor force (that is either employed or unemployed). The likelihood of receiving welfare payments as the main income source is increased by 1.5 percentage points. Given a baseline of 4%, the effect on transfer receipts corresponds to an increase of about 37%. These results suggest that young adults who were exposed to maternal influenza infections in utero are less likely to work and instead are considerably more likely to depend on government transfers. Column

(6) shows that there is no significant effect on the likelihood of being unemployed. Instead of searching extensively for a job, disadvantaged adults seem to leave the labor force and obtain welfare if they cannot find a job quickly. This mechanism is also in line with the high share of people in these young cohorts relying on government transfers that is four times as large as the share of the unemployed. In fact, public debate in Denmark in recent years has criticized young adults for not even trying to find a job, and instead, too easily accepting welfare when they come out of school.

Panel (B) of Table 6 repeats these regressions in the sample of individuals who are not currently enrolled in an educational degree program. As expected, point estimates increase across all regressions. The overall pattern, however, looks very similar. Figure 6 shows the distribution of log earnings for exposed and unexposed siblings in this sample. The distribution for exposed siblings is shifted to the left, with an average difference of 0.117 log points, which is close to the point estimate of -0.093 reported in Table 6 (Kolmogorov-Smirnov test for equality of distributions rejected with $p<0.01$). This figure shows that the earnings effect is not driven by one particular income group but by a shift of the entire distribution.

5.5 Long-term effects by trimester of exposure

Table 7 shows that the long-run effects on labor market outcomes strongly depend on the timing of the exposure. The first three columns of Table 7 show estimates for the entire sample, while the last three columns exclude those who are in school. To avoid clutter, I focus on the outcomes that are most affected in the previous table. Across all columns, the third trimester effects are similar to the overall effect in Table 6, and they are significant in most cases. This is in line with the short-term analysis that indicated strong effects of third trimester infections. Remarkably, however, unlike for birth outcomes, there are significant effects of second trimester exposure that are 50%-150% larger in magnitude than the third trimester effect. This pattern supports previous studies that find that cognitive abilities are particularly strongly affected by impairments during the second trimester, the pregnancy period with the strongest brain development (De Santis and Di Gianantonio 2005; Almond et al. 2009). First trimester infections, on the other, do not seem to have effects on any labor market outcomes.

The differential impact of second and third trimester infections on short- and long-term outcomes is important for several reasons. The observed pattern suggests that influenza induced inflammations can impair the fetus in multiple ways. It is not that influenza infections

simply cause premature labor which in turn leads to lower incomes, but there is different damage depending on the timing of the infection. Moreover, much of the damage is not visible in commonly recorded birth outcomes. Figure 7 compares the effect pattern across pregnancy trimesters for the prematurity rate and log wages. The strong impact of third trimester infections on premature birth illustrated in the left panel is an outcome visible at birth to doctors and mothers. However, the dramatic impairment of human capital development of second trimester infections apparent in the right figure is only uncovered by an economic long-term analysis. Finally, the differential effect pattern in the short- and long-run also supports the conclusion from the balancing regressions that effects are unlikely to be driven by selection. If second and third trimester infections were simply a proxy for a disadvantaged parental background, it should be similarly correlated with outcomes in the short and long run.

5.6 Impacts on education and health at birth as potential pathway

The patterns described in the previous table are in line with detrimental impacts of second trimester infections on brain development, which are not visible at birth but have lasting effects on human capital development. An initial cognitive disadvantage might reinforce itself over time if it induces the impaired individual to obtain less education. Table 8 reports effects of in-utero exposure to influenza on the probability of attending school in a given year and on completed years of education. Estimated effects of influenza exposure during overall pregnancy in (Panel A) are negative, but quantitatively small and not statistically significant. Trimester-specific estimates in Panel (B) show more pronounced negative effects for second trimester infections that are significant at the 5% level in the education regression. The point estimate indicates that those who suffered exposure to maternal influenza in the second trimester obtain on average a fifth of a year less education compared to their unexposed siblings, and they are 2.5 percentage points less likely to attend school in a given year. The point estimates for third trimester infections are close to zero, positive and insignificant. There is a marginally significant reversed effect of first trimester infections on education attainment, perhaps representing sampling variation or a potential positive selection effect (first trimester effects are discussed in the next section).

This effect pattern is in line with the idea that the strong long-term labor market impacts of second trimester infections run through an impairment of cognitive skills. But the estimated effects are relatively small and if educational attainment was an important margin one might also expect effects of the third trimester. When interpreting these results, it is

important to keep in mind that education in Denmark is free and that students are entitled to generous stipends when enrolled in a degree program. Therefore, educational attainment in Denmark might be compensating rather than reinforcing ability differences. The less able in a pair of siblings has an incentive to continue schooling and enjoy free stipends if he cannot land as well-paying a job in the labor market as his sibling and if more education increases the chances that he eventually does. This interpretation is in line with the marginal effects of being born with low birth weight on educational outcomes that I discuss further below (Table 10).

In Table 9, I explore more directly to what extent the effects on labor market outcomes might be mediated by health at birth and educational attainment. In the first three columns I include fixed effects for the exact week of gestation and for 300 g birth weight groups, while columns (4) to (6) include fixed effects for years of education. The effects on the labor market outcomes are remarkably robust to the inclusion of birth outcomes controls. Coefficients hardly change, suggesting that effects on labor market outcomes may work on top of the effects on observable health at birth. However, as Pei et al. (2017) point out, this kind of coefficient movement test might not be very informative if observable birth outcomes are poorly measured proxies of actual health at birth.[14] Including educational controls, however, attenuates the second trimester effects by about one-third while the third trimester effects remain largely unchanged. This pattern supports the notion that part of the strong second trimester effects on labor market outcomes indeed run through educational attainment.

5.7 Effects on fertility

Previous research suggests that strong influenza infections early in pregnancy induce miscarriages (Bloom-Feshbach et al., 2011). Such an effect could explain why there are generally no significant impacts of first trimester infections, with often reversed point estimates. If infections lead to miscarriages then the most affected cases are not observed. Moreover, such miscarriage effect might induce a positive selection bias if the weakest embryos are most likely to be aborted. The fact that the fewest cases of maternal influenza hospitalizations are observed during the first trimester (conditional on the pregnancy resulting in a live birth) is in line with a miscarriages effect as well. Figure 6 provides a more direct test, following

[14]Measurement error in birth weight and gestation length may be limited -- this information is usually accurately reported because mothers and doctors care about it -- but it might not correlate strongly with the latent unobserved health of the newborn. Further, the strong effects on birth outcomes shown in Tables 2 and 3 are not contradicting measurement error -- mismeasurement in this setting has less of an effect on the left than on the right-hand side of the equation (Pei et al. 2017).

women of fertile age who are observed with an influenza infection over time and comparing their pregnancy rates to a matched set of women of the same age but without an infection. The blue circles show the fertility rate in the group of matched women, fluctuating around a rate of about 6 births per 1,000 women.

Women who are observed with a strong influenza infection, on the other hand, have an increased risk of giving birth in the months after the hospitalization. This makes sense given that pregnant women are more likely to develop strong influenza infections and influenza infections can lead to a shortened gestation length. Seven months after the infection, however, the pregnancy rate drops sharply and falls below the baseline rate in months eight and nine. This pattern is in line with strong influenza infections inducing miscarriage in the first, but not in the second or third trimester. It is likely that women who lose their baby in the first trimester would continue to seek pregnancy, which would be associated with a fertility rate above baseline in the months following the miscarriage-induced drop in births. Indeed, the fertility rate is elevated by about 25% above baseline 11 to 15 months after the infections. Taken together, identifying miscarriages during the first trimester is empirically challenging, but following infected and uninfected women over time provides suggestive evidence of such a miscarriage effect, which can explain the insignificant and often reversed estimates of first trimester infections. This pattern is also in line with the miscarriage impacts that Almond et al. (2016) document for morning sickness, a relatively mild condition compared to strong influenza infections.

5.8 Effect size

How large are the long-run effects that I find? Is it plausible for early conditions to result in a 9-10% earnings difference during young adulthood? As discussed in the introduction, the effects that I find are similar to the long-run impacts of maternal infections during the 1918/19 Spanish Flu pandemic (Almond 2006) and in a similar range as the estimates for high-quality early childhood interventions studied by Chetty et al. (2011) and Heckman et al. (2010). Another important benchmark is provided by Nilsson (forthcoming) who analyzes a reform in Sweden that lead to a substantial increase in alcohol consumption among mothers below age 21. My second-trimester estimates are remarkably similar to the effects that Nilsson (forthcoming) finds for maternal drinking during the first half of pregnancy. Nilsson (forthcoming) documents a 24.4% earnings loss, a 7.2 p.p. decrease in labor force participation, a 3.5 p.p. increase in welfare dependence, and 0.3 fewer years of schooling. The corresponding estimates for second trimester influenza infections are 24.8%, 6.3 p.p.,, 3.2

p.p., and 0.2 years respectively.[15]

A further important benchmark, which I can directly analyze in the Danish data, is the effect of being born with low birth weight. Birth weight is a commonly used proxy for poor fetal development and has been studied in many contexts. (Currie and Moretti 2007; Oreopoulos et al. 2008; Black et al. 2007; Royer 2009; Figlio et al. 2014; Bharadwaj et al. 2013). In Table 10, I compare the effects of in-utero exposure to influenza (Panel A) to the impact of being born with low birth weight (Panel B). All regressions include mother fixed effects. The results in the first five columns of Table 10 indicate that the effects of influenza on labor market outcomes are in about the same range as the effects of low birth weight. Moreover, the effect patterns across the different outcomes are similar. For example, the effect on log wages is much stronger than the effect on log income in both panels. Postnatal mortality (column 6), on the other hand, is much more strongly affected by low birth weight (10 percentage points) than by influenza exposure (1.6 percentage points). Among low birth weight babies in my sample, 15% weigh less than 1,500 g (called "very low birth weight"), a condition associated with high neonatal mortality. The rate of very low birth weight among those exposed to maternal influenza during pregnancy is less than 2%. Effects on educational outcomes are reported in the last two columns. Unlike for influenza, low birth weight has a significantly negative effect on these outcomes. However, the effects are very small. The probability of school attendance is reduced by less than one percentage point, given a baseline rate of 54.7%. Obtained schooling is reduced by merely 0.055 years, or half a month. These marginal effects support the idea that the Danish educational system is not reinforcing initial ability differences and that neither the strong wage effects of influenza infections nor those of low birth weight are mediated through educational attainment. Overall, these comparisons show that both the size of the influenza effect and the structure across outcomes is reasonable. The estimated effects are similar to a well-established indicator of poor fetal development in both dimensions.

5.9 Cohort regressions

Table 11 shows regressions of short- and long-term outcomes on the trimester-specific nationwide influenza spread instead of individual infections. Despite the caveats of this cohort-level approach described in Section (4.2), which imply imprecise estimates, the overall pat-

[15] Note that in my sample many individuals have not yet completed their education, potentially explaining the slightly smaller effect on completed years. Further note that Nilsson (forthcoming) estimates "intent-to-treat" cohort effects. However, given the strong uptake of drinking during the analyzed reform it seems likely that the average treatment effects at the individual level are not substantially larger.

tern across trimesters and across different outcomes is remarkably similar to the micro-level estimates.

Effects on birth cohorts are reported in the first 6 columns. The relevant estimates are the specifications with conception month fixed effects in columns 2, 4, and 6 (columns 1, 3, and 5 show estimates without month dummies to illustrate their relevance). Overall, the estimated effects closely mirror the estimates from the micro-level. In both cases, effects are strongest for the third trimester. Moreover, there is no second trimester effect on gestation length, while the effect of the second trimester on birth weight is smaller and less precisely estimated but it is not attenuated to zero.[16] Effects on labor market outcomes and education in columns (7) to (10) are imprecisely estimated. However, in all cases the second trimester effect has the correct sign and the point estimate is larger than for the first and third trimester. The second trimester effect on education is most precisely estimated and significant at the 5% level.

How do these cohort-level effects compare across outcomes in terms of their effect size? To answer this question, Table A.13 compares the estimates from the cohort- and micro-level regressions, focusing on the third trimester for birth outcomes and on the second trimester for long-term outcomes (the periods and outcomes when effects tend to be most significant, respectively). Despite the large confidence intervals around the cohort-level estimates, the relative size compared to the micro-level estimates is similar. Across all outcomes, the estimated impact of maternal influenza infections is about 50 to 85 times larger than the effect of the nationwide influenza index. This gives further confidence that the cohort-level effects do not simply represent arbitrary point estimates, but that they are driven by similar mechanisms as the effects estimated more precisely at the micro-level.

What do the cohort-level estimates in Table 11 tell us about the effects of seasonal influenza waves on average cohort outcomes? The national influenza index, averaged across trimesters, varies by around 5 units over the year. This suggests that the effect of individual maternal infections is 10 to 17 times as large as the influenza-induced fluctuation in average outcomes (dividing the effect ratios in Table A.13 by 5). In particular, taking the point estimate of the second trimester effect on wages in Table 11 at face-value implies that seasonal influenza decreases the average earnings of the most exposed cohorts (those with their second trimester during the peak of the flu season) by 1.4%.[17]

[16] Results for low birth weight (omitted) are very imprecise and there is no specific pattern of point estimates. This might not be surprising, given that micro-level estimates in Table 5 for that outcome are only about half the size of the effect on prematurity and the latter is already only marginally significant in the cohort regression.

[17] Note that the cohort-level effects are identified by deviations from the average seasonal pattern (regressions

The comparison of the two sets of estimates further allows for a back-of-the-envelope calculation of how the effects of strong influenza infections, estimated at the individual level, compare to the effect of an average influenza infection in the society. If we think of the cohort effects as a "reduced form" and the micro-level effects as an estimate of the average treatment effect, this would imply a "first stage" of 0.06 to 0.1 (if the treatment effect is expressed as the ratio of the "reduced form" divided by the "first stage," see Angrist and Pischke (2008)). In other words, if the micro-level estimates indeed represent the average effect of influenza infections, then no more than 6%-10% of pregnant women in the most affected cohorts should be infected. This is at the lower end of plausible infection rates for the most affected conception cohorts, given estimates of around 5%-15% for the overall population (Russell et al. 2008) and likely higher rates among risk groups such as pregnant women. In turn, this suggests that the effects identified at the micro-level via maternal hospitalizations are larger than the average effect of all maternal infections in the population. This seems plausible as only strong infections lead to hospitalizations.[18]

5.10 Robustness

5.10.1 Trimester-specific selection

Interpreting these trimester-specific results, one might worry about selection bias as there are relatively few infections during the first and second trimester and Table 3 only shows balancing regressions pooling all influenza cases. Appendix Table A.2 therefore presents balancing regressions for trimester-specific influenza indicators. The first four columns show that mothers are similarly selected across all three trimesters, while the last four columns indicate that this selection is eliminated when mother fixed effects are included. Another concern could be that third trimester effects might be attenuated, induced by the fact that shorter gestation lengths shorten the time period during which a mother may get infected with influenza. Appendix Table A.4 therefore shows estimates using influenza infections six to ten months after conception as an instrument for influenza in the third trimester, i.e. between month six and birth (see e.g. Currie and Rossin-Slater (2013) for more details on

include month fixed effects). The effects of these deviations might be stronger than the effects of regular seasonal fluctuations if pregnant women adapt protective behavior more to the regular seasonality than to unexpected deviations.

[18] One should also keep in mind that the identification of individual influenza cases in the data involves measurement error, which is likely to attenuate estimated effects. This might explain why the individual level estimates are not even larger compared to the average effects measured at the cohort level (larger effects at the individual level would in turn imply a lower overall infection rate).

this IV strategy). Indeed, the third trimester effects increase by about one-fifth, in line with a small attenuation bias in the regressions that do not account for the endogeneity of gestation length. Because the change is small and the overall pattern of effects is not affected, I use the more transparent OLS specification in the remainder of the analysis.

5.10.2 Effect heterogeneity

Effect sizes might differ across subgroups of the sample. As discussed above, sex ratios at birth are balanced suggesting that maternal influenza infections do not lead to higher miscarriage rates of male fetuses. However, males might still be differently affected than females both in the short- and the long-run. Table A.5 shows influenza effects interacted with a dummy that is one for a male newborn. None of the interactions in the birth outcomes regressions are significant. However, point estimates in the gestation and birth weight regressions are sizable and they have the same sign as the main effect. One interpretation is that male fetuses are more affected but that the analysis lacks statistical power to precisely estimate these differences. The estimates in the last three columns show significant gender differences for labor market outcomes. The effects on wages and labor force participation is much stronger for males, which would be in line with a stronger initial effect. Increases in welfare rates, on the other hand, are mostly driven by females, perhaps because it is less of a stigma for women to have welfare as their main income source.

Another interesting subgroup is families of low socio-economic status. Maternal influenza infections might develop more dramatically in poorer mothers, perhaps due to an interaction of the disease with other unfavorable conditions of poor neighborhoods such as pollution or poorly functioning health-care providers. Further, even for a given initial shock, long-term effects might be more detrimental as poorer parents might have fewer resources to support a disadvantaged child. Differences due to parental responses would be more important the greater the visibility of a disadvantage. This means that differences due to compensating investments might be stronger for third trimester infections that have strong effects on visible birth outcomes than for the second trimester. Table A.6 shows regressions for birth and labor market outcomes that include the interaction of infections with an indicator that is one if the mother belongs to the bottom 30% of her cohort defined by her age and year of birth. Overall, the interaction terms indicate that effects are stronger for poorer families, both in terms of short-term impacts on health at birth as well as long-term labor market outcomes. At face value, the effects on health at birth are about twice as large for low-income mothers, while mark-ups on the long-term effects vary more. However, these differences

are not precisely estimated and none of the interaction terms are significant, except for the gestation regression in the first column.

Keeping in mind that a further stratification of the data might not be informative given the low level of precision, Appendix Table A.7 reports interaction terms for trimester-specific effects. The first column reports the effects on log wages. Even though they are estimated imprecisely, the point estimates of the third trimester terms suggest that the effect is about twice as large for low-income offspring. The second trimester effect, however, entirely loads on the main effect, indicating a highly significant 17% reduction of wages for high-income offspring, while the interaction term is positive and not significantly different from zero. This pattern of stronger long-term effects of more visible third trimester infections for low-income offspring but equally strong effects of less visible second trimester infections is in line with the hypothesis that high-SES parents are better than low-SES parents in compensating prenatal shocks that are visible at birth, but less so for shocks that do not affect birth outcomes. A similar pattern is observed for overall income in column (2), though none of the estimates are significant. For labor force participation, the second trimester effects are also loaded on the main effect, but the pattern is less clear for the third trimester (perhaps again due to a lack of precision). For welfare receipt, the pattern is actually reversed with stronger second trimester effects for low-SES mothers and equal or weaker effects in the third trimester. To sum up, it seems that the overall influenza impact is somewhat stronger for low-income mothers but this subgroup analysis is not precise enough to show how exactly this effect is shaped across the different pregnancy periods.[19]

5.10.3 Controlling for other diagnoses

One general identification concern of my analysis is that influenza diagnoses proxy for other, more severe health conditions that may induce hospitalizations and influenza is diagnosed merely ”on the side.” Moreover, hospitalizations could be stressful events with negative effects on fetal development (Aizer et al. Forthcoming; Black et al. 2016; Persson and Rossin-Slater forthcoming). In Appendix Table A.9, I compare the baseline estimates with regressions that control for other diagnoses that are most often coded along with influenza during pregnancy (Panel B) as well as for any prebirth hospitalization (Panel C). These other diagnoses include pregnancy complications, delivery complications and prenatal care inspections. Prebirth hospitalizations exclude hospitalizations within three days prior to birth.

[19] Appendix Table A.8 further presents results with interaction terms for birth order, stratifying the sample into first and higher order births. No significant effect differences are apparent between the two subgroups.

Notice that the inclusion of these additional controls may attenuate the estimated effect of influenza towards zero as these covariates might be themselves impacted by influenza infections ("bad controls", in the language of Angrist and Pischke (2008)). Indeed, the resulting estimates decrease slightly in the birth outcomes regressions but they remain highly significant in all cases. Moreover, the corresponding attenuation in the labor market regressions are only marginal (columns 5 to 7), suggesting that the estimated effects of influenza do not merely proxy for these other diagnoses.

5.10.4 Alternative specifications

The baseline sample consists of all sibling pairs, including those without any influenza cases. These unexposed sibling pairs do not provide identifying variation but they help to estimate the broad set of fixed effects and covariates that are included to achieve greater precision. Appendix Tables A.10 and A.11 repeat the baseline regressions including only sibling pairs with one exposed and one unexposed sibling. Overall, both effect sizes as well as the pattern of effects across different outcomes are very similar. Non-parametric results based on the smaller exposed sibling sample have been presented in Figures 5 and 6, providing very similar effects to the overall sibling sample, too.

Appendix Table A.12 provides the baseline regressions using alternative influenza definitions. Panel B excludes unspecified viral and respiratory symptoms, while Panel C includes the diagnosis codes for the common cold, sinusitis, and tracheitis (conditions that can be caused by influenza). The effects on birth outcomes in Panel B are slightly stronger than in the baseline regression (in particular the effect on postnatal mortality is increased by about one-third), suggesting that this specification identifies somewhat stronger health effects. The long-term effects on labor market outcomes, on the other hand, are of similar size or slightly smaller, potentially a result of the increased mortality effect. The extended influenza specification in Panel C, which might identify on average less dramatic cases, results in somewhat attenuated effects both in the short- and in the long-run.

6 Conclusion

Using rich administrative data from Denmark, I document long-run effects of in-utero exposure to seasonal influenza on labor market outcomes and human capital development. Maternal influenza infections during pregnancy are associated not only with worse health at birth, but also with lower earnings, decreased labor market participation, and substantially higher

rates of welfare dependence. These effects are remarkably similar whether they are estimated either at the cohort level or by tracking offspring of mothers who were known to have been infected. These findings provide the first evidence that maternal influenza infections impair the offspring's human capital development during common non-pandemic years.

The differential effects that I find depending on the timing of the infection suggest that influenza has the potential to damage the fetus via multiple mechanisms. Infections in the third trimester trigger preterm births, a health condition associated with increased infant mortality but with moderate impacts on human capital development. Second trimester infections, on the other hand, seem to primarily impair cognitive development, with no significant effects on health at birth but strong impacts on labor market outcomes and education.

The effect pattern also suggests that much of the damage caused by maternal influenza infections might not be visible at birth. Doctors and parents might be alarmed about premature labor and low birth weight, but the more important cognitive impacts of second trimester infections may initially remain unnoticed. This limits the extent to which parents can respond to developmental disadvantages of their child. Parents may try to compensate ability differences across siblings in response to observed differences in birth outcomes such as low birth weight (Bharadwaj et al. forthcoming), but they may be less likely to respond to developmental impairments that are not visible at birth. The lack of compensatory parental investments could be an amplifying force underlying the large human capital effects of second trimester infections that I find. At the same time, recent research suggests that parental investment responses might be compensating or reinforcing under multiple dimensions (Yi et al. 2015; Aizer and Cunha 2012; Heckman and Mosso 2014). Studying the parental responses in data sets that provide proxies for parental investments might be a promising path for future research.

Overall, the results of this paper suggest that fostering influenza vaccinations among pregnant women would be an efficient public policy for improving children's human capital development and reducing disparities in adult economic outcomes. In the U.S., influenza vaccination has been recommended for pregnant women by the Centers for Disease Control since 2004, and vaccination rates have increased since the 2009/2010 pandemic. Yet about 30% of doctors fail to pass on these recommendations to pregnant women and about 50% of pregnant women have remained unvaccinated in recent years (CDC 2013). Developing effective ways to inform pregnant women and their doctors about the potential lasting costs of not getting vaccinated should be a priority for public policy.

Another policy component could be the promotion of measures that do not require vac-

cination of pregnant women. Official campaigns on vaccination benefits might not be convincing for people exposed to conspirative, unsubstantiated news stories about vaccination side-effects that are increasingly spread online (e.g. http://www.vaccines.news/, accessed 2/1/2017). For these groups, more effective measures might be the promotion of protective behavior such as washing hands and the vaccination of pregnant women's significant others so that the disease cannot reach the unvaccinated pregnant women. Reducing the spread of seasonal influenza in the overall population will not just be beneficial for pregnant women's offspring, but is also likely to lower health-care costs and improve productivity during flu season (Pichler and Ziebarth 2016).

The broader takeaway of this study is that maternal infections in general might be an important and underestimated prenatal threat. Influenza effects are relatively easy to detect in aggregate outcomes due to the strongly non-linear nature of its seasonal and pandemic spread. Less seasonal diseases, on the contrary, might be equally detrimental for fetal development but less visible in aggregate data. Yet other infectious diseases, such as rubella or Zika, are well-known for devastating malformations caused by first trimester infections, while potential second trimester effects might only be detectable in economic long-term analyses. This implies that the benefits of vaccination campaigns might be understated if these potential long-term effects on the next generation are not taken into account. This is an important message in times of vaccination controversy, public healthcare defunding, and an ever more globally integrated world population facilitating the spread of new diseases.

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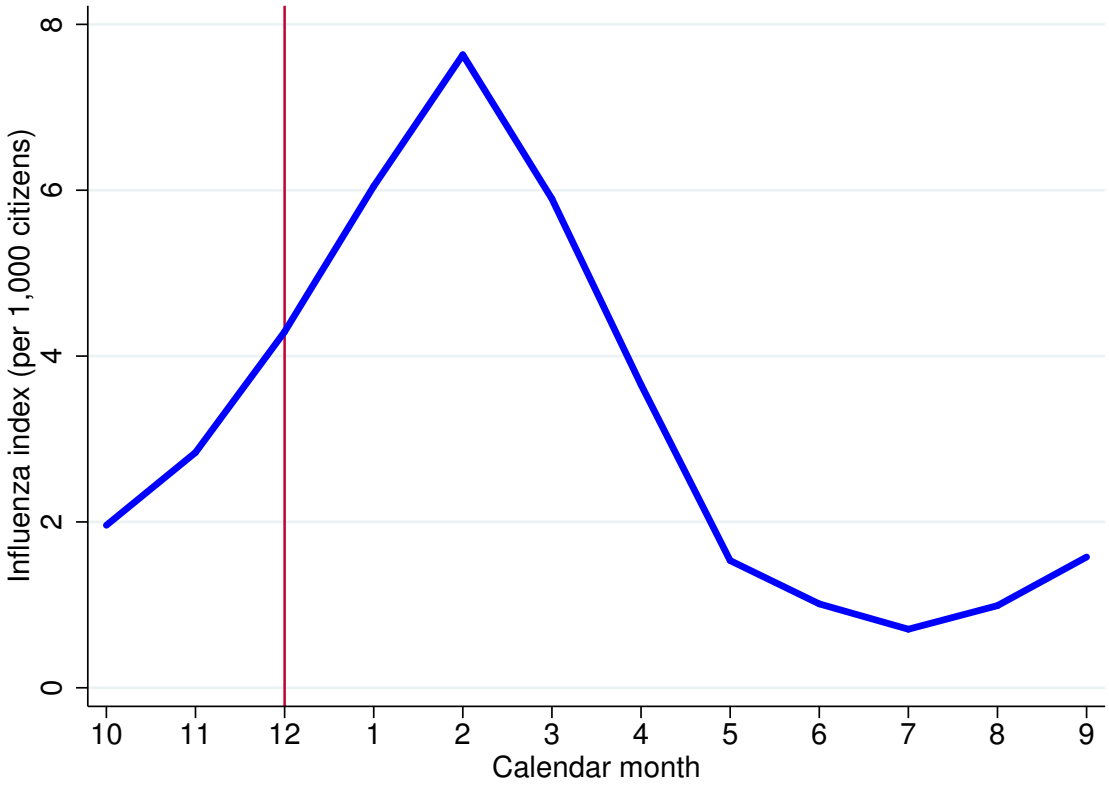
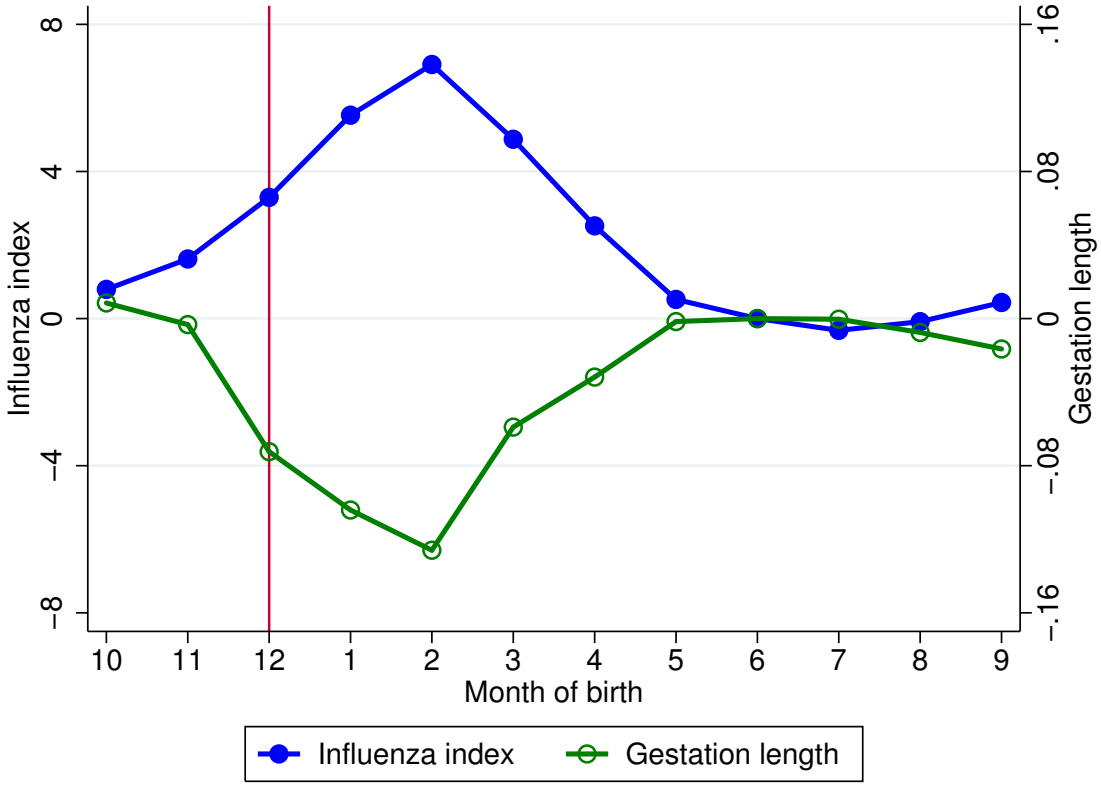
Figure 1: Seasonality of influenza spread in Denmark



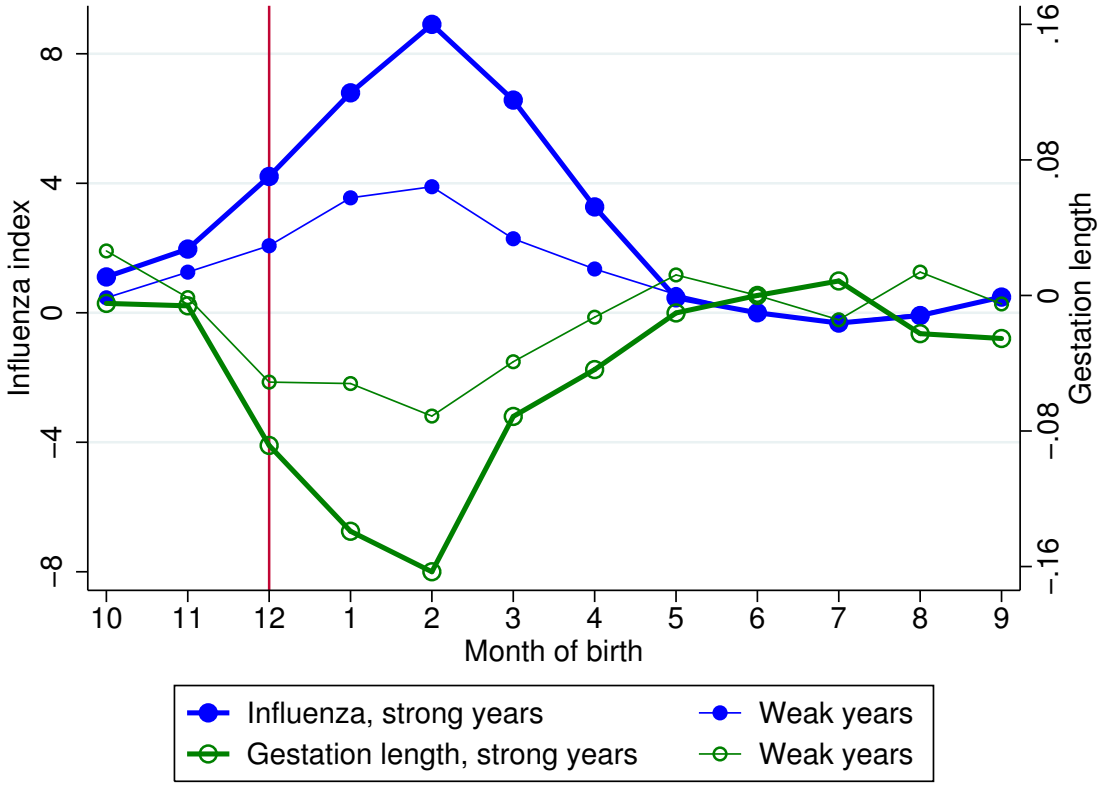
Notes: Monthly cases of patients with influenza-like illness reported by Danish general practitioners for the years 1980 - 1993 are divided by the current Danish population and averaged by calendar month. For the disaggregated times series see Appendix Figure A1.

Figure 2: Influenza index at birth and gestation length



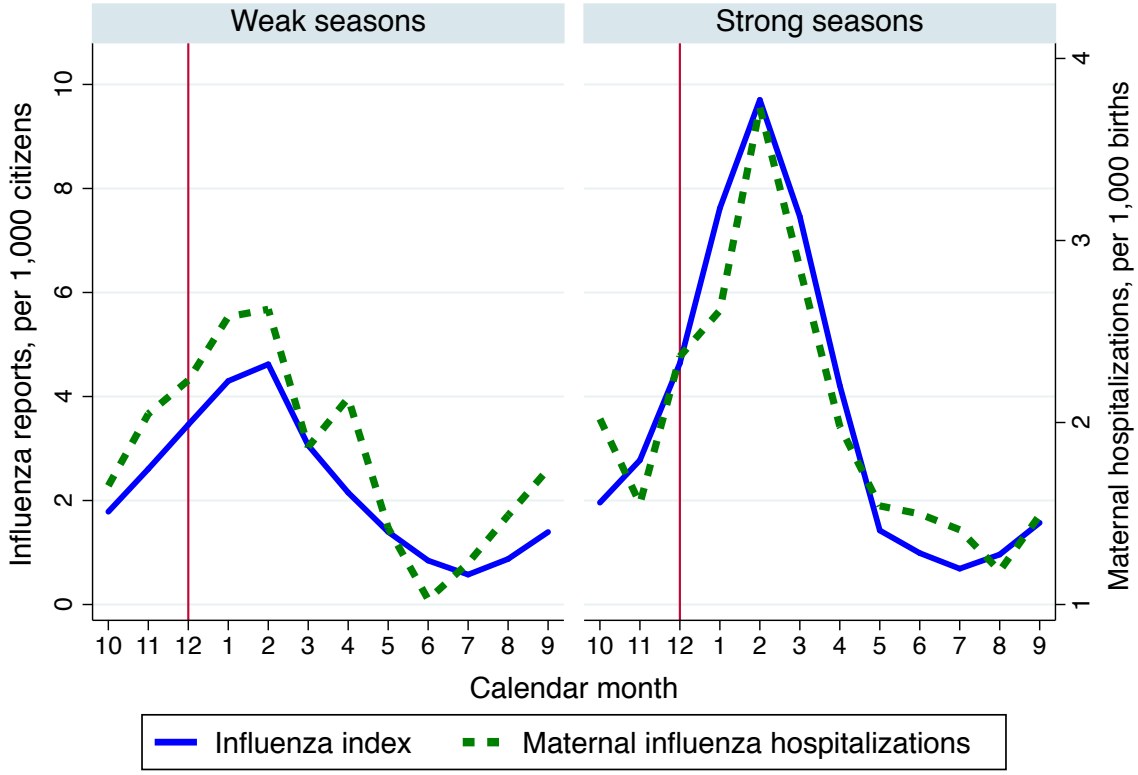
Notes: Coefficients from regressions of the (left y-axis, in monthly reports per 1,000 citizens) and gestation length (right y-axis, in weeks) on birth month dummies are displayed. Additional controls and sample specification as in Figure 2. Numeric results reported in Appendix Table A1.

Figure 3: Influenza index and gestation length, weak vs. strong influenza seasons



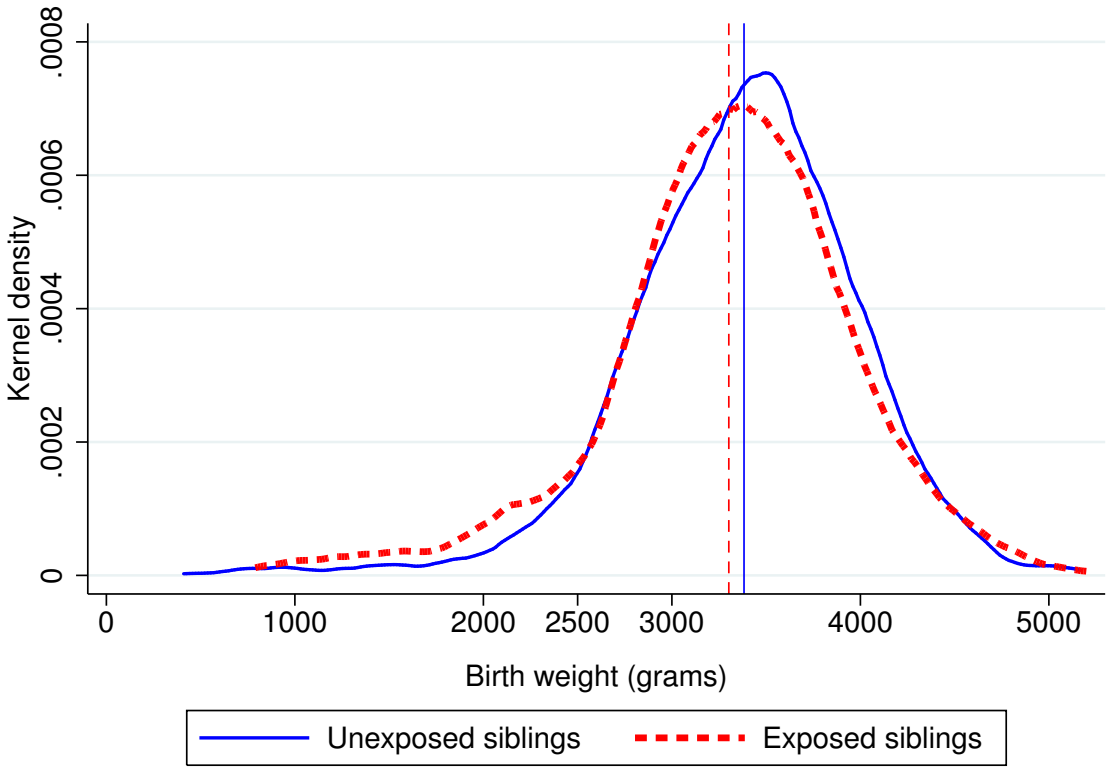
Notes: Displayed are coefficients from regressions of the influenza index (left y-axis, in monthly reports per 1,000 citizens) and gestation length (right y-axis, in weeks) on month dummies, interacted with a dummy for strong influenza season (’80, ’82, ’83, ’84, ’85, ’86, ’89, ’92; see Appendix Figure A1). Both regressions include fixed effects for the mother, parity and gender of the newborn, mother’s age group, education, region of residence, and marital status, the conception year, and a quadratic time trend at the monthly level. For corresponding regression results, see Appendix Table A1, columns (2), (3), (5) and (6). The sample includes all cohorts conceived between 1/1980 and 12/1992, born by mothers of age 18+. N=719,854 in all regressions.

Figure 4: Influenza index and maternal influenza diagnoses



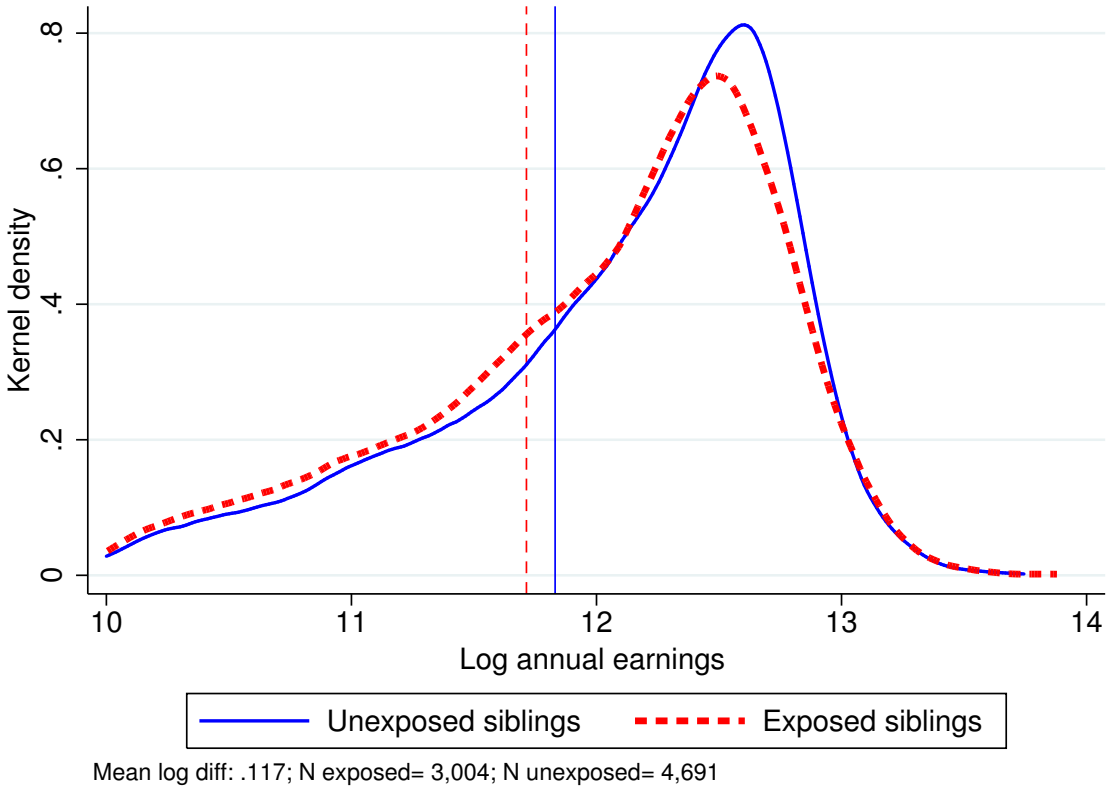
Notes: The influenza index is based on General Practitioners' reports of influenza-like illnesses (for more details see notes of Figure 1). Maternal influenza hospitalizations refer to pregnant women that are hospitalized with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms.

Figure 5: Birth weight distribution of children exposed to maternal influenza during pregnancy and of their siblings.



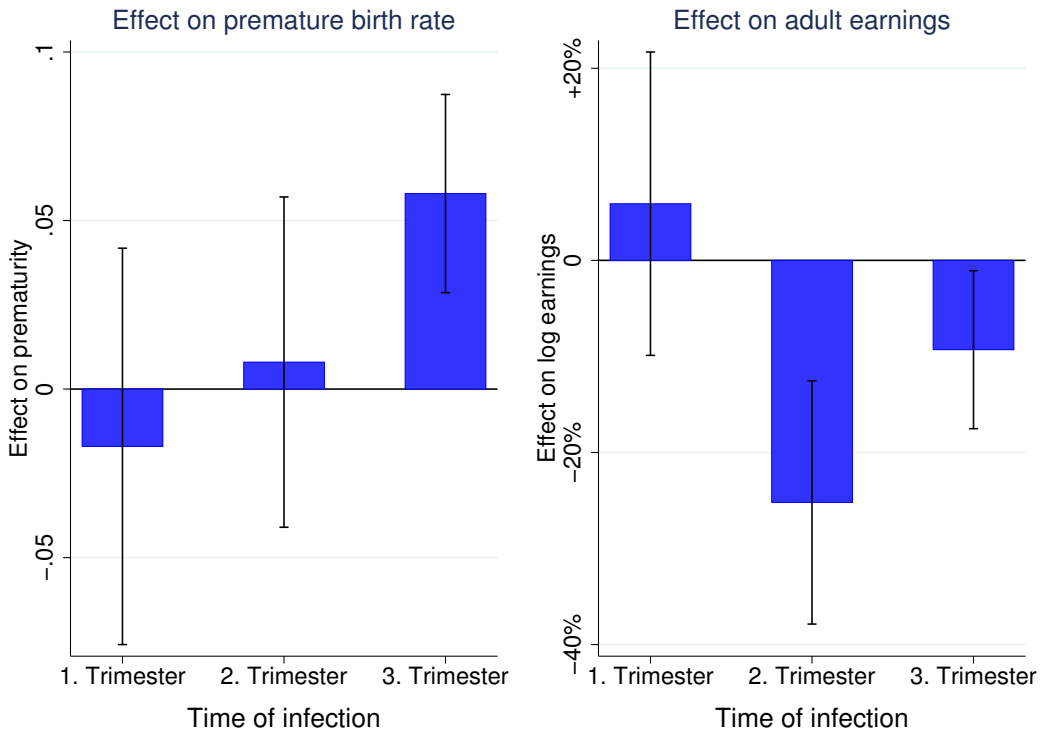
Notes: This figure shows the birth weight distribution of children that have been exposed to maternal influenza in utero and of their siblings that have not been exposed. There are more unexposed (N=1,583) than exposed (N=1,329) siblings because there are more cases of 3+ siblings with only one exposed sibling than cases with only one unexposed sibling. KS test for equal distribution rejected with p=0.021. The mean difference is 81 grams, indicated by the vertical lines.

Figure 6: Log earnings distribution of young adults exposed to maternal influenza during pregnancy, and of their siblings.



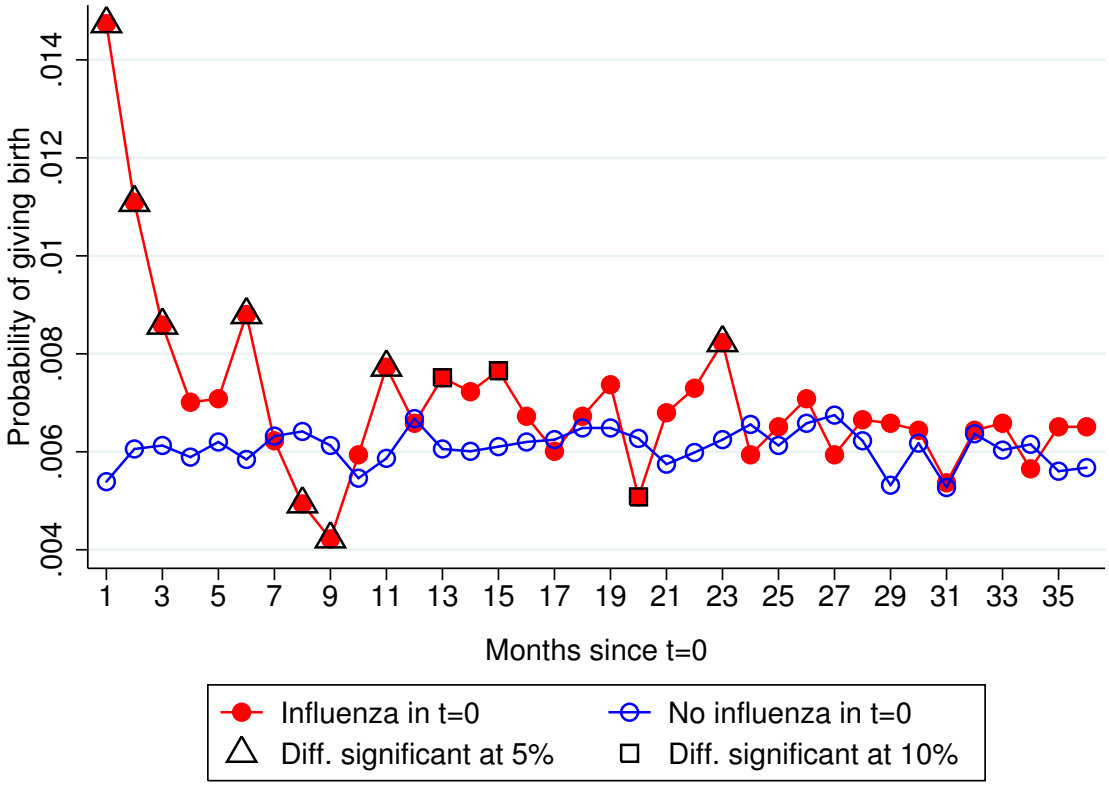
Notes: This figure shows the log earnings distribution of young adults that have been exposed to maternal influenza in utero and of their siblings that have not been exposed. There are more unexposed (N=4,691) than exposed (N=3,004) siblings because there are more cases of 3+ siblings with only one exposed sibling than cases with only one unexposed sibling. KS test for equal distribution rejected with $p<0.01$. The mean difference is 0.117, indicated by the vertical lines.

Figure 7: Effect of influenza on prematurity rate and adult earnings, by trimester



Notes: This figures plots the coefficients reported in Table 5 column (2) and Table 7 column (4), respectively, along with 95% confidence intervals.

Figure 8: Influenza infections and subsequent fertility



Notes: This figure shows the probability of giving birth over time for women of age 18 - 35. Red solid circles represent "treatment group" women who are diagnosed with influenza in t=0 (N=13,978). Blue hollow circles refer to 41,837 "control group" women without influenza who are matched to infected women in t=0 based on their date of birth.

Table 1: Summary Statistics

Variable	Mean	Std dev.	Min.	Max.
(A) Overall sample (all conceptions 1980-1992), N= 719,854				
Children per mother	1.54	0.67	1	9
Sibling born in sample period	0.640			
(B) Sibling sample (sibling born in sample period), N=460,618				
Children per mother	2.202	0.466	2	9
Newborn is female	0.486			
Mother's first birth	0.398			
Gestation (weeks)	39.66	1.78	19	55
Premature (<37 weeks)	0.042			
Birth weight (gram)	3,461	557.9	310	7,000
Low birth weight (<2500g)	0.039			
Not observed at age 18+	0.022			
Not observed at age 18+, but sibling is	0.016			
Hospitalized during pregnancy	0.983			
Delivery with complications	0.400			
Mother foreign born	0.064			
Mother's age	27.17	4.48	18	58
(C) Observed at age 18+ (1998-2012), N= 3,423,350				
Observations per matched person	7.43			
Age	22.11	3.25	18	32
Currently in school	0.547	1.88	7	21
Years of education	12.36			
In labor force	0.630			
Unemployed	0.009			
Receiving welfare benefits	0.044			
Log income (in 2010 USD)	9.70	1.22	-1.69	14.3
Log wage (in 2010 USD)	9.97	1.03	-1.69	15.3

Notes: The overall sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+. For labor market outcomes in the subsample of individuals who are out of school see Panel (B) of Table 6.

Table 2: Influenza cases

	Overall sample		Sibling sample	
	n	percent	n	percent
Influenza during pregnancy	1,598	0.222	1,008	0.219
Influenza, reduced definition	1,210	0.168	781	0.170
Influenza, extended definition	2,004	0.278	1,265	0.275
Influenza during 1st trimester	198	0.028	123	0.027
Influenza during 2nd trimester	307	0.043	207	0.045
Influenza during 3rd trimester	1,155	0.160	716	0.155

Notes: For sample definitions see previous table. "Influenza during pregnancy" refers to hospitalizations with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms. "Reduced definition" excludes codes for unspecified symptoms, "extended definition" includes codes for common cold, sinusitis, and tracheitis.

Table 3: Balancing regressions of maternal characteristics on influenza during pregnancy

Dependent variable	Foreign mother	Mother's yrs of education	Mother's age	Mother is married	Low pop. density districts	Mother's log earnings
	(1)	(2)	(3)	(4)	(5)	(6)
A. No controls						
Influenza during pregnancy	0.044***	-0.320***	0.155	0.020	-0.018**	-0.128***
	[0.007]	[0.065]	[0.122]	[0.012]	[0.008]	[0.027]
B. Baseline controls						
Influenza during pregnancy	0.021***	-0.214***	-0.085	-0.015	-0.017**	-0.080***
	[0.006]	[0.061]	[0.101]	[0.010]	[0.007]	[0.023]
C. Mother FEs + baseline controls						
Influenza during pregnancy	-	0.017	0.002	-0.017	-0.003	-0.029
	-	[0.042]	[0.016]	[0.017]	[0.003]	[0.036]
N (A. and B.)	718,280	683,151	719,854	719,854	719,854	639,534
N (C.)	460,322	440,308	460,618	460,618	460,618	412,043
Mean dep. var.	0.064	11.7	27.500	0.527	0.041	11.5

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for year and month of conception, and (unless chosen as dependent variable) region of birth, parity, gender, maternal age at birth, origin, education, and marital status. The sample includes all mothers of age 18+ with conceptions between 1/1980 and 12/1992. In Panel (C.) the sample is further restricted to sibling pairs. Sample sizes vary across columns due to missing values in the dependent variable. Robust standard errors are clustered at the municipality level.

Table 4: Effect of influenza on birth outcomes

Dependent variable	Gestation length (wks) (1)	Prematurity (<37 wks) (2)	Birth weight (gr) (3)	Low birth weight (<2500 gr) (4)	Child is a boy (5)	Not observed at age 18+ (6)
A. Baseline						
Influenza during	-0.319***	0.045***	-84.483***	0.035***	-0.020	0.016**
pregnancy	[0.091]	[0.010]	[22.238]	[0.011]	[0.024]	[0.008]
B. Controlling for gestation length						
Influenza during	-	-	-26.358	0.007	-0.022	0.011
pregnancy	-	-	[17.325]	[0.008]	[0.024]	[0.007]
Baseline controls	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes
N	460,618	460,618	459,987	459,987	450,596	460,618
Mean dep. var.	39.7	0.042	3,461	0.039	0.514	0.016

Notes: "Influenza" refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the month and year of birth, region of birth, parity, gender (omitted in column 5), maternal age at birth, origin, education, and marital status. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+. Robust standard errors are clustered at the municipality level.

Table 5: Effect of influenza on birth outcomes, by trimester

Dep. var.	Gestation (wks)	Prematurity (<37 wks)	Birth weight (gr)	Low birth weight (<2.5kg)	Not observed at age 18+
	(1)	(2)	(3)	(4)	(5)
A. Baseline					
Influenza during					
First trimester	0.322	-0.033	42.007	-0.016	0.001
	[0.231]	[0.027]	[56.007]	[0.034]	[0.027]
Second trimester	-0.024	0.011	-46.506	0.037	0.009
	[0.238]	[0.021]	[53.662]	[0.024]	[0.017]
Third trimester	-0.495***	0.064***	-112.97***	0.038***	0.020**
	[0.099]	[0.013]	[28.183]	[0.013]	[0.008]
B. Controlling for gestation length					
Influenza during					
First trimester	-	-	-5.748	0.012	0.001
			[51.313]	[0.034]	[0.023]
Second trimester	-	-	-24.049	0.018	0.003
			[32.768]	[0.017]	[0.015]
Third trimester	-	-	-31.515	0.000	0.015*
			[21.977]	[0.009]	[0.008]
Baseline controls	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes
N	460,618	460,618	459,987	459,987	460,618

Notes: "Influenza" during the first, second, and third trimester refers to dummy variables indicating hospitalizations with an influenza-like illness diagnosis during the first, second, and third trimester, respectively. For further comments see Table 3.

Table 6: Effect of influenza on labor market outcomes

Dependent variable	Log wage	Log income (incl. transfers)	Log non-wage income	Labor force participation	Receiving welfare benefits	Unemployed (if in labor force)
	(1)	(2)	(3)	(4)	(5)	(6)
A. Entire sibling sample						
Influenza during pregnancy	-0.087***	-0.025	0.126***	-0.028***	0.015**	0.002
	[0.027]	[0.020]	[0.046]	[0.010]	[0.006]	[0.002]
N	2,934,255	3,409,363	3,345,837	3,422,403	3,423,511	3,422,403
Mean dep. var.	9.70	9.97	7.54	0.63	0.04	0.01
B. Excluding those in school						
Influenza during pregnancy	-0.093**	-0.031	0.191***	-0.039***	0.026**	0.003
	[0.041]	[0.033]	[0.072]	[0.014]	[0.011]	[0.003]
N	1,302,779	1,541,946	1,501,515	1,551,861	1,552,543	1,551,861
Mean dep. var.	10.20	10.30	7.34	0.74	0.08	0.02

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table 7: Effect of influenza on labor market outcomes, by trimester

	Entire sibling sample			Excluding those in school		
Dep. var.	Log wage	In labor force	Receiving welfare	Log wage	In labor force	Receiving welfare
	(1)	(2)	(3)	(4)	(5)	(6)
Influenza during						
First trimester	0.001	-0.012	0.003	0.145	0.007	0.008
	[0.071]	[0.027]	[0.016]	[0.098]	[0.036]	[0.029]
Second trimester	-0.132**	-0.046**	0.030**	-0.248***	-0.063**	0.032
	[0.054]	[0.020]	[0.013]	[0.079]	[0.029]	[0.025]
Third trimester	-0.088***	-0.021*	0.012	-0.096*	-0.038**	0.028**
	[0.033]	[0.012]	[0.008]	[0.052]	[0.017]	[0.014]
Mother FEs + baseline ctrls.	yes	yes	yes	yes	yes	yes
N	2,934,255	3,422,403	3,423,511	1,302,779	1,551,861	1,552,543

Notes: "Influenza" during the first, second, and third trimester refers dummy variables indicating hospitalizations with an influenza-like illness diagnosis during the first, second, and third trimester, respectively. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table 8: Effect of influenza on educational outcomes

Dependent variable	Currently in school (1)	Years of education (2)
A. Overall effect		
Influenza during pregnancy	-0.002	-0.005
	[0.008]	[0.040]
B. Trimester-specific effect		
Influenza during		
First trimester	0.008	0.196*
	[0.021]	[0.106]
Second trimester	-0.025	-0.201**
	[0.016]	[0.081]
Third trimester	0.004	0.028
	[0.009]	[0.048]
Mother FEs + baseline controls	yes	yes
N	3,449,790	3,426,851
Mean dep. var.	0.55	12.40

Notes: "Influenza" refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table 9: Effect of influenza on labor market outcomes, conditional on health at birth and education

	Controlling for health at birth			Controlling for education		
Dependent variable	Log wage	Labor force participation	Receiving welfare benefits	Log wage	Labor force participation	Receiving welfare benefits
(Entire sibling sample)	(1)	(2)	(3)	(4)	(5)	(6)
Influenza during						
First trimester	-0.001	-0.013	0.004	-0.022	-0.021	0.009
	[0.070]	[0.027]	[0.016]	[0.072]	[0.027]	[0.015]
Second trimester	-0.132**	-0.045**	0.030**	-0.104**	-0.032	0.023*
	[0.054]	[0.020]	[0.013]	[0.053]	[0.020]	[0.013]
Third trimester	-0.088***	-0.020*	0.011	-0.087***	-0.021*	0.011
	[0.033]	[0.012]	[0.008]	[0.033]	[0.012]	[0.007]
Mother FEs + baseline ctrls.	yes	yes	yes	yes	yes	yes
Birth weight + gestation FEs	yes	yes	yes			
Years of education FEs				yes	yes	yes
N	2,934,255	3,422,403	3,423,511	2,934,255	3,422,403	3,423,511

Notes: "Influenza" during the first, second, and third trimester refers dummy variables indicating hospitalizations with an influenza-like illness diagnosis during the first, second, and third trimester, respectively. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table 10: Comparison of long-term influenza effects with effects of low birth weight

Dep. var.	Log wage	Log income (incl. transfers)	Log non-wage inc.	Labor force participation	Welfare receipt	Not observed at age 18+	Currently in school	Years of education
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
A. Regressions of outcomes on influenza								
Influenza	-0.087***	-0.025	0.126***	-0.028***	0.015**	0.016**	-0.002	-0.005
during pregnancy	[0.027]	[0.020]	[0.046]	[0.010]	[0.006]	[0.008]	[0.008]	[0.040]
B. Regressions of outcomes on low birth weight								
Low birth weight	-0.055***	-0.017***	0.151***	-0.035***	0.030***	0.101***	-0.009***	-0.055***
	[0.007]	[0.005]	[0.012]	[0.003]	[0.002]	[0.004]	[0.002]	[0.011]
Mother FEs + baseline controls	yes	yes	yes	yes	yes	yes	yes	yes
N	2,934,255	3,409,363	3,345,837	3,422,403	3,423,511	459,987	3,447,007	3,424,086
Mean dep. var.	9.70	9.97	7.54	0.630	0.044	0.016	0.547	12.400

Notes: "Influenza" refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table 11: Regressions of birth and human capital outcomes on influenza index

	Birth outcomes						Long-term outcomes			
Dep. var.	Gestation (wks)		Prematurity *100 (i.e. in %)		Birth weight (gr)		Log wage	Labor force, *100	Receiving welfare*100	Education years*100
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Influenza index, during										
1st trimester	0.0054*	0.0006	-0.0261	-0.0062	-0.161	-0.827	-0.0023	-0.0280	0.0660	-0.2483
	[0.0030]	[0.0033]	[0.0288]	[0.0331]	[0.611]	[0.783]	[0.0028]	[0.0719]	[0.0594]	[0.1689]
2nd trimester	0.0077***	0.0022	-0.0354	-0.0007	-1.068*	-1.639*	-0.0029	-0.0791	0.1028	-0.4196**
	[0.0024]	[0.0036]	[0.0247]	[0.0359]	[0.576]	[0.914]	[0.0034]	[0.0821]	[0.0706]	[0.1925]
3rd trimester	-0.0114***	-0.0095**	0.0681*	0.0792	-1.789**	-2.278**	-0.0003	0.0421	-0.0066	-0.2143
	[0.0036]	[0.0044]	[0.0356]	[0.0486]	[0.702]	[0.965]	[0.0039]	[0.0878]	[0.0827]	[0.2256]
Baseline controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month FEs		Yes		Yes		Yes	Yes	Yes	Yes	Yes
N	460,618	460,618	460,618	460,618	459,987	459,987	1,302,779	1,551,861	1,552,543	3,400,584

Notes: Coefficients from regressions of birth and labor market outcomes on the national influenza-like illness index during different times of the pregnancy are displayed. The influenza index refers to General Practitioners' reports of influenza-like illness, per 1,000 Danes. See Appendix Figure A1 for the times-series of the index. Baseline controls are the region of birth, parity, gender, maternal age at birth, origin, education, marital status, a quadratic time trend at the conception date level, and conception year fixed effects in columns (1) - (6) and fixed effects for the current calendar year and the current age in columns (7) - (9). Robust standard errors are clustered at the conception year*monthly level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, observed at birth in columns (1) - (6) and at age 18+ up to 2012 (excluding years when receiving education) in columns (7) - (9).

Appendix

A Empirical specification of the seasonality analysis in Figures 2 and 3

Following Currie and Schwandt (2013), I analyze the seasonality of influenza spread and gestation length with the following empirical model

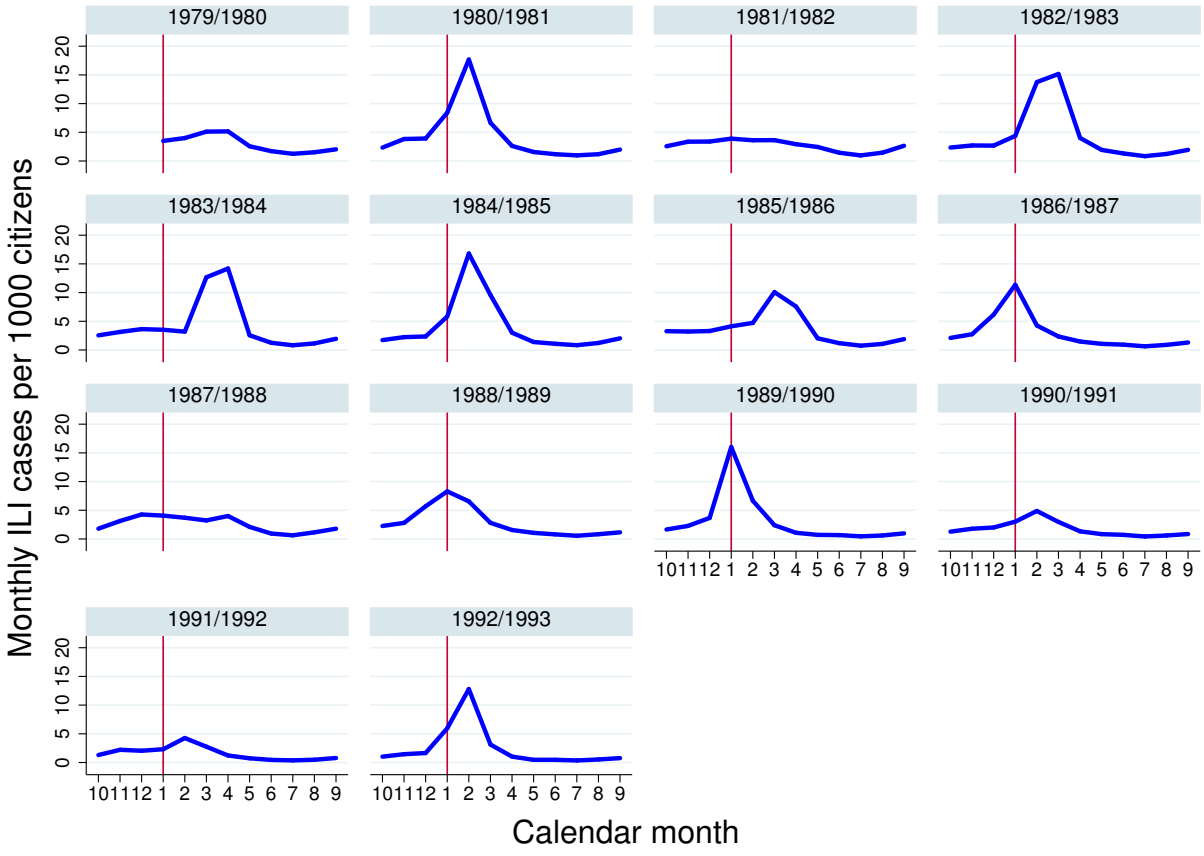
$$Y_i = \alpha + \sum_{m=2}^{12} \beta_m D[month_m] + \delta X_i + \mu_{mom} + \epsilon_i$$

where the index i refers to the newborn. Y_i refers to the gestation length when analyzing the seasonality in health at birth, and to the national influenza index in the month of birth when analyzing the seasonality of influenza spread. $D[month_m]$ are dummies for the individual's month of birth. Currie and Schwandt (2013) provide an analysis at the conception month level as birth month effects could be biased by seasonal fluctuations in the conception rate. However, in the Danish context, resulting patterns are similar at the conception and birth month level; this is why I show the more intuitive pattern by birth month (Appendix Fig. A.2 shows results by conception month). μ_{mom} are mother fixed effects that account for time-fixed differences in socio-economic characteristics of mothers that tend to conceive in different months (Buckles and Hungerman 2013; Currie and Schwandt 2013).

X_i are dummies for parity, the gender of the newborn, mother's age group ($<$20, 20-24, 25-34, $>$35), education ($<$10, 10-12, 13-16, $>$16, missing), four regions of residence and marital status, the birth year, and a quadratic time trend at the monthly level. The time trend at the monthly level is important, because the birth year fixed effects only account for level differences between years but not continuous time trends occurring within the year. Without including the trend-variable at the monthly level, this within-year time trend would end up be picked up as a seasonality effect. Standard errors are clustered at the monthly level to account for the fact that the influenza index only varies at that level. In an extended version of this model, I interact the birth month with a dummy indicating strong influenza seasons. The regression results are reported in Table A.1 and illustrated in Figures 2 and 3.

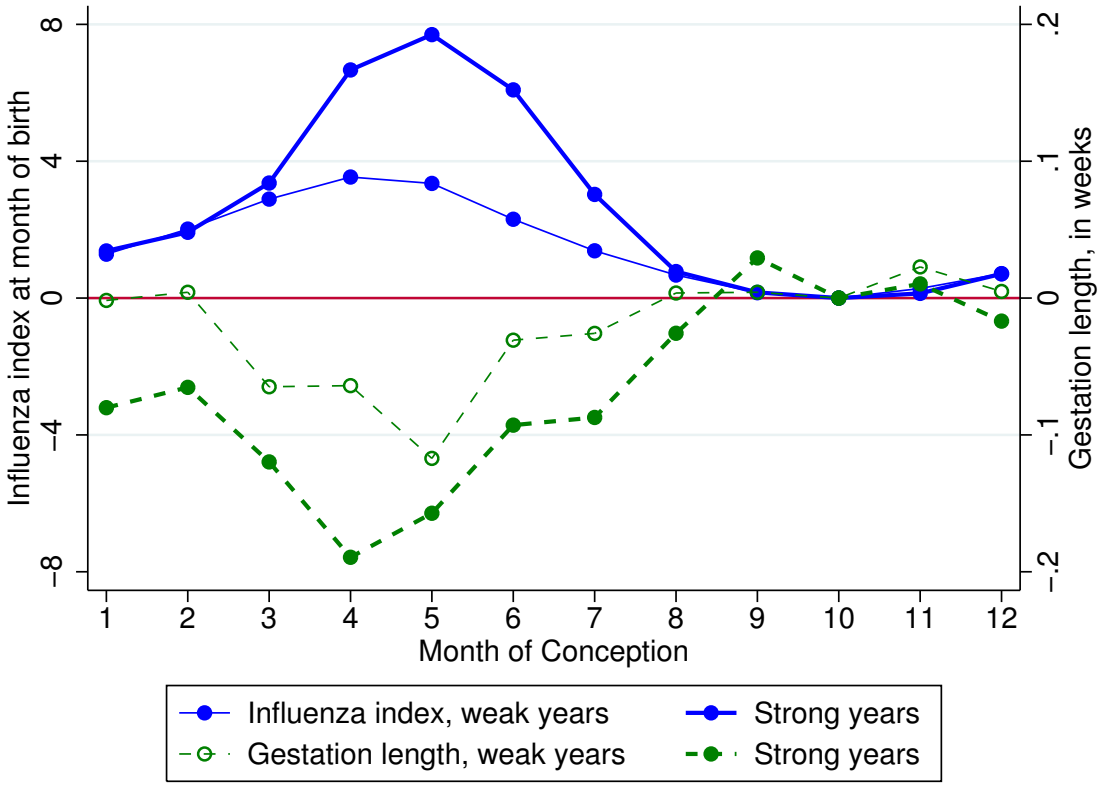
B Appendix Figures and Tables

Figure A.1: Influenza spread in Denmark from 1979 to 1993



Notes: Monthly cases of influenza-like illness (ILI) reported by Danish general practitioners are divided by the contemporaneous Danish population. ILI reports are based on surveillance data collected and provided by the Danish Staten Serum Institut.

Figure A.2: Seasonality of influenza and gestation length, matched at month of conception



Notes: Coefficients from regressions of the influenza index (left y-axis) and the gestation length (right y-axis) on birth month dummies are displayed. Both regressions include fixed effects for the mother, parity and gender of the newborn, mother's age group, education, region of residence and marital status, the conception year and a quadratic time trend at the monthly level. For the corresponding regression specification see Appendix Section I and for the results see Appendix Table A1, columns (1) and (4). The sample includes all cohorts conceived between 1/1980 and 12/1992, born by mothers of age 18+. N=719,854 in all regressions.

Table A.1: Regressions of influenza index and gestation length on conception month dummies

Dep. var.	Influenza index at month of birth			Gestation length (weeks)		
Model		Weak vs. strong flu seasons			Weak vs. strong flu seasons	
	Overall	Main effect	Strong seasons	Overall	Main effect	Strong seasons
	(1)	(2)	(3)	(4)	(5)	(6)
Month of birth						
January	5.530***	3.550***	3.244*	-0.104	-0.052	-0.087***
	[1.149]	[0.851]	[1.911]	[0.070]	[0.068]	[0.033]
February	6.909***	3.894***	5.015***	-0.126**	-0.071	-0.092**
	[1.192]	[0.541]	[1.844]	[0.055]	[0.059]	[0.042]
March	4.875***	2.286***	4.284***	-0.059	-0.039	-0.032
	[0.989]	[0.241]	[1.521]	[0.043]	[0.042]	[0.035]
April	2.521***	1.353***	1.917	-0.032	-0.013	-0.031
	[0.866]	[0.374]	[1.435]	[0.031]	[0.032]	[0.032]
May	0.522	0.565**	-0.104	-0.002	0.012	-0.022
	[0.344]	[0.245]	[0.371]	[0.023]	[0.027]	[0.037]
June	Reference month					
July	-0.320	-0.293	-0.026	0.000	-0.014	0.023
	[0.330]	[0.233]	[0.345]	[0.023]	[0.029]	[0.035]
August	-0.083	-0.032	-0.054	-0.008	0.014	-0.036
	[0.327]	[0.202]	[0.317]	[0.031]	[0.037]	[0.034]
September	0.438	0.457*	0.027	-0.017	-0.005	-0.020
	[0.326]	[0.253]	[0.348]	[0.044]	[0.047]	[0.034]
October	0.794**	0.462	0.644	0.008	0.026	-0.031
	[0.316]	[0.498]	[0.797]	[0.055]	[0.059]	[0.038]
November	1.621***	1.255**	0.713	-0.003	-0.001	-0.005
	[0.333]	[0.523]	[0.862]	[0.068]	[0.076]	[0.049]
December	3.296***	2.070***	2.140	-0.072	-0.051	-0.037
	[1.029]	[0.794]	[2.233]	[0.081]	[0.086]	[0.039]

Notes: Columns (1) and (4) show coefficients from regressions of the influenza index and the gestation length on birth month dummies. Columns (2), (3), (4), and (5) display the effects from a model with an interaction for strong influenza seasons (seasons '80, '82, '83, '84, '85, '86, '89, '92). All regressions include fixed effects for the mother, parity and gender of the newborn, mother's age group, education, region of residence, and marital status, the conception year, and a quadratic time trend. Standard errors are clustered at the monthly level. N=719,854 in all regressions.

Table A.2: Balancing regressions of maternal characteristics on influenza during pregnancy, by trimester

	No controls				Baseline controls + mother fixed effects			
Dep. var.	Foreign mother	Mother's yrs of education	Low pop. density districts	Mother's log earnings	Foreign mother	Mother's yrs of education	Low pop. density districts	Mother's log earnings
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Influenza during	0.041*	-0.574***	-0.030*	-0.205***	-	0.038	-0.014	0.011
First trimester	[0.022]	[0.167]	[0.015]	[0.074]	-	[0.077]	[0.013]	[0.091]
Second trimester	0.064***	-0.213	-0.016	-0.132**	-	0.080	0.002	-0.027
	[0.020]	[0.146]	[0.012]	[0.059]	-	[0.082]	[0.007]	[0.071]
Third trimester	0.034***	-0.285***	-0.014*	-0.103***	-	0.003	-0.003	-0.030
	[0.008]	[0.087]	[0.007]	[0.027]	-	[0.052]	[0.004]	[0.040]
N	718,280	683,151	719,854	639,534		460,618	460,618	412,043

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for year and month of conception, and (unless chosen as dependent variable) region of birth, parity, gender, maternal age at birth, origin, education, and marital status. The sample includes all mothers of age 18+ with conceptions between 1/1980 and 12/1992. Sample sizes vary across columns due to missing values in the dependent variable. Robust standard errors are clustered at the municipality level.

Table A.3: Effect of influenza on birth outcomes, with and without mother fixed effects

Dependent variable	Gestation length (wks) (1)	Prematurity (<37 wks) (2)	Birth weight (gr) (3)	Low birth weight (<2500 gr) (4)	Child is a boy (5)	Not observed at age 18+ (6)
A. Baseline controls (no mother FEs)						
Influenza during pregnancy	-0.529***	0.059***	-150.322***	0.061***	0.014	0.008***
	[0.056]	[0.008]	[16.169]	[0.007]	[0.013]	[0.003]
B. Baseline controls + mother FEs						
Influenza during pregnancy	-0.319***	0.045***	-84.483***	0.035***	-0.020	0.016**
	[0.091]	[0.010]	[22.238]	[0.011]	[0.024]	[0.008]
N	460,618	460,618	459,987	459,987	450,596	460,618
Mean dep. var.	39.7	0.042	3,461	0.039	0.514	0.016

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table A.4: Effect of influenza on birth outcomes, instrumenting third trimester exposure

Dep. var.	Gestation (wks)	Prematurity (<37 wks)	Birth weight (gr)	Low birth weight (<2.5kg)	Not observed at age 18+
	(1)	(2)	(3)	(4)	(5)
A. Baseline					
First trimester	0.322	-0.033	42.007	-0.016	0.001
	[0.231]	[0.027]	[56.007]	[0.034]	[0.027]
Second trimester	-0.024	0.011	-46.506	0.037	0.009
	[0.238]	[0.021]	[53.662]	[0.024]	[0.017]
Third trimester	-0.495***	0.064***	-112.966***	0.038***	0.020**
	[0.099]	[0.013]	[28.183]	[0.013]	[0.008]
B. Third trimester instrumented					
First trimester	0.330	-0.033	42.220	-0.016	0.002
	[0.184]	[0.021]	[45.72]	[0.025]	[0.020]
Second trimester	-0.017	0.011	-44.970	0.036*	0.009
	[0.167]	[0.017]	[40.15]	[0.021]	[0.012]
Third trimester	-0.598***	0.070***	-134.2***	0.049***	0.023***
	[0.103]	[0.014]	[25.7]	[0.012]	[0.009]
Mother FEs + baseline controls	yes	yes	yes	yes	yes
N	460,618	460,618	459,557	459,557	460,618

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012. In Panel B the third trimester influenza indicator is instrumented with a dummy that is one if the mother was diagnosed with influenza 6 to 10 months after conception.

Table A.5: Effect of influenza on birth and labor market outcomes by gender

	Birth outcomes				Labor market outcomes		
Dep. var.	Gestation	Pre-maturity	Birth weight	Low birth weight	Log wage	Lab. force participation	Welfare receipt
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Influenza during pregnancy	-0.156	0.034**	-63.070**	0.023*	-0.029	-0.010	0.033***
	[0.123]	[0.014]	[31.252]	[0.013]	[0.039]	[0.014]	[0.010]
Influenza * male	-0.143	0.004	-19.464	0.006	-0.113**	-0.036*	-0.036***
	[0.148]	[0.019]	[41.408]	[0.017]	[0.054]	[0.019]	[0.012]
Mother FEs + baseline controls	yes	yes	yes	yes	yes	yes	yes
N	450,596	450,596	450,124	450,124	2,934,255	3,422,403	3,423,511
Mean female	39.70	0.035	3,406	0.038	9.57	0.597	0.051
Mean male	39.70	0.041	3,531	0.034	9.83	0.660	0.037

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age, as well as the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012.

Table A.6: Effect of influenza on birth and labor market outcomes for low-income mothers

	Birth outcomes				Labor market outcomes		
Dep. var.	Gestation	Pre-maturity	Birth weight	Low birth weight	Log wage	Lab. force participation	Welfare receipt
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Influenza during	-0.199**	0.033***	-64.827**	0.025**	-0.074**	-0.023*	0.009
pregnancy	[0.100]	[0.011]	[25.590]	[0.013]	[0.033]	[0.012]	[0.007]
Influenza * low	-0.338*	0.031	-55.302	0.027	-0.040	-0.017	0.016
income mother	[0.188]	[0.023]	[50.983]	[0.025]	[0.058]	[0.021]	[0.014]
Mother FEs + baseline controls	yes	yes	yes	yes	yes	yes	yes
N	460,618	460,618	459,987	459,987	2,934,255	3,422,403	3,423,511
Mean dep. var. overall	39.70	0.042	3,461	0.039	10.20	0.74	0.08
Mean dep. var. low inc.	39.60	0.045	3,452	0.043	9.67	0.61	0.06

Notes: Low-income mothers belong to the bottom 30% in their age and cohort group. ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status and in columns (5)-(6) fixed effects for the current calendar year and current age.

Table A.7: Effect of influenza on labor market outcomes by trimester, interacted with low-income mother

Dependent variable (Entire sibling sample)	Log wage (1)	Log income (incl. transfers)	Lab. force participation (2)	Welfare receipt (3)
Influenza during				
First trimester	0.000	0.060	0.002	-0.014
	[0.081]	[0.073]	[0.033]	[0.019]
First trimester * low income mother	0.007	-0.023	-0.040	0.050
	[0.161]	[0.103]	[0.058]	[0.034]
Second trimester	-0.173***	-0.029	-0.043*	0.007
	[0.060]	[0.042]	[0.022]	[0.013]
Second trimester * low income mother	0.125	0.009	-0.009	0.065**
	[0.123]	[0.079]	[0.046]	[0.030]
Third trimester	-0.060	-0.018	-0.020	0.015*
	[0.041]	[0.031]	[0.015]	[0.009]
Third trimester * low income mother	-0.086	-0.040	-0.002	-0.007
	[0.070]	[0.052]	[0.025]	[0.017]
Mother FEs + baseline controls	yes	yes	yes	yes
N	2,934,255	3,409,363	3,422,403	3,423,511

Notes: Low-income mothers belong to the bottom 30% in their age and cohort group. ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age and the month of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Sample includes all sibling pairs conceived between 1/1980 and 12/1992, observed at age 18+ up to 2012.

Table A.8: Effect of influenza on birth and labor market outcomes, interacted with birth order

	Birth outcomes					Labor market outcomes		
Dependent variable	Gestation	Pre-maturity	Birth weight	Low birth weight	Not observed at age 18+	Log wage	Lab. force participation	Welfare receipt
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Influenza during pregnancy	-0.102	0.049*	-57.704	0.028	0.022	-0.087	-0.007	0.021*
	[0.179]	[0.027]	[45.876]	[0.021]	[0.015]	[0.072]	[0.024]	[0.012]
Influenza * later born sibling	-0.326	-0.006	-40.083	0.011	-0.009	0.000	-0.022	-0.006
	[0.229]	[0.036]	[59.504]	[0.030]	[0.018]	[0.070]	[0.023]	[0.010]
Mother FEs + baseline ctrls.	yes	yes	yes	yes	yes	yes	yes	yes
N	460,618	460,618	459,987	459,987	460,618	2,934,255	3,422,403	3,423,511

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the current calendar year, current age and the month of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. Sample includes all sibling pairs conceived between 1/1980 and 12/1992, observed at age 18+ up to 2012. Robust standard errors are clustered at the municipality level in columns (1) - (4) and at the individual level in columns(5) - (8). In columns (5) - (7) individuals are excluded when receiving education; and restricted to age 18-20 in column (8).

Table A.9: Effect of influenza on birth and labor market outcomes, controlling for other diagnoses and prebirth hospitalization

	Birth outcomes				Labor market outcomes		
Dep. var.	Gestation length (wks) (1)	Prematurity (<37 wks) (2)	Birth weight (gr) (3)	Low birth weight (<2500 gr) (4)	Log wage (5)	Lab. force participation (6)	Welfare receipt (7)
A. Baseline							
Influenza during pregnancy	-0.319*** [0.091]	0.045*** [0.010]	-84.483*** [22.238]	0.035*** [0.011]	-0.087*** [0.027]	-0.028*** [0.010]	0.015** [0.006]
B. Controlling for common diagnoses during pregnancy							
Influenza during pregnancy	-0.292*** [0.083]	0.040*** [0.010]	-75.345*** [21.507]	0.027*** [0.010]	-0.086*** [0.027]	-0.028*** [0.010]	0.015** [0.006]
C. Controlling for any pre-birth hospitalization							
Influenza during pregnancy	-0.233*** [0.083]	0.035*** [0.010]	-61.674*** [21.902]	0.022** [0.010]	-0.084*** [0.027]	-0.027*** [0.010]	0.015** [0.006]
N	460,618	460,618	459,987	459,987	2,930,592	3,417,961	3,419,068

Notes: ”Influenza” refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis. Baseline controls are fixed effects for the month and year of birth, region of birth, parity, gender, maternal age at birth, origin, education, and marital status. The sample includes all sibling pairs conceived between 1/1980 and 12/1992. Robust standard errors are clustered at the municipality level. “Other diagnoses” controls are dummies for pregnancy complications (ICD-8: 630-639), delivery complications (ICD-8: 651-659), and ”prenatal care inspection” (ICD-8: Y60-Y69). “Prebirth hospitalization” is a dummy for hospitalizations during pregnancy up to three days prior to birth (sample mean: 0.243).

Table A.10: Effect of influenza on birth outcomes, overall vs. exposed sibling sample

Dep. var.	Gestation length (wks) (1)	Prematurity (<37 wks) (2)	Birth weight (gr) (3)	Low birth weight (<2500 gr) (4)	Not observed at age 18+ (5)
A. Sibling sample					
Influenza during	-0.319***	0.045***	-84.483***	0.035***	0.016**
pregnancy	[0.091]	[0.010]	[22.238]	[0.011]	[0.008]
N	460,618	460,618	459,987	459,987	460,618
B. Exposed sibling sample					
Influenza during	-0.240***	0.043***	-77.426***	0.034***	0.013
pregnancy	[0.083]	[0.010]	[22.652]	[0.010]	[0.009]
N	2,366	2,366	2,360	2,360	2,366

Notes: All regressions include baseline controls and mother fixed effects. The sibling sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+. The exposed sibling sample only includes sibling pairs with one sibling exposed and one sibling not exposed to maternal influenza.

Table A.11: Effect of influenza on labor market outcomes, overall vs. exposed sibling sample

Dep. var.	Log wage	Log income (incl. transfers)	Log non-wage income	Labor force participation	Receiving welfare benefits	Unemployed (if in labor force)
	(1)	(2)	(3)	(4)	(5)	(6)
A. Sibling sample						
Influenza during	-0.087***	-0.025	0.126***	-0.028***	0.015**	0.002
pregnancy	[0.027]	[0.020]	[0.046]	[0.010]	[0.006]	[0.002]
N	2,934,255	3,409,363	3,345,837	3,422,403	3,423,511	3,422,403
B. Exposed sibling sample						
Influenza during	-0.093***	-0.020	0.152***	-0.026***	0.015**	0.003*
pregnancy	[0.028]	[0.021]	[0.048]	[0.010]	[0.006]	[0.002]
N	14,553	17,705	17,345	17,815	17,818	17,815

Notes: All regressions include baseline controls and mother fixed effects. The sibling sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+. The exposed sibling sample only includes sibling pairs with one sibling exposed and one sibling not exposed to maternal influenza.

Table A.12: Effect of influenza on birth and labor market outcomes, alternative influenza specifications

	Birth outcomes					Labor market outcomes		
	Gestation length	Prematurity (<37 wks)	Birth weight (gr)	Low birth weight	Not obs. at age 18+		In labor	Receiving
						Log wage	force	welfare
Dep. var.	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
A. Baseline								
Influenza during	-0.319***	0.045***	-84.483***	0.035***	0.016**	-0.087***	-0.028***	0.015**
pregnancy	[0.091]	[0.010]	[22.238]	[0.011]	[0.008]	[0.027]	[0.010]	[0.006]
B. Reduced influenza definition								
Influenza during	-0.386***	0.051***	-99.800***	0.041***	0.022**	-0.082***	-0.026**	0.009
pregnancy	[0.103]	[0.012]	[28.560]	[0.012]	[0.009]	[0.030]	[0.011]	[0.007]
C. Extended influenza definition								
Influenza during	-0.232***	0.033***	-65.859***	0.025***	0.015**	-0.048**	-0.019**	0.014**
pregnancy	[0.084]	[0.010]	[19.851]	[0.010]	[0.007]	[0.024]	[0.009]	[0.006]
N	460,618	460,618	459,987	459,987	460,618	2,934,255	3,422,403	3,423,511

Notes: All regressions include baseline controls and mother fixed effects. Baseline controls are fixed effects for the month and year of conception, region of birth, parity, gender, maternal age at birth, origin, education, and marital status as well as (in the labor market regressions) calendar year and age dummies. Robust standard errors are clustered at the individual level. The sample includes all sibling pairs conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to 2012. "Influenza during pregnancy" refers to hospitalizations with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms. "Reduced definition" excludes codes for unspecified symptoms, "extended definition" includes codes for common cold, sinusitis and tracheitis.

Table A.13: Comparison of micro- and cohort-level estimates

	Birth outcomes			Long-term outcomes		
	Gestation	Pre-maturity	Birth weight	Log wage	In labor force	Years of education
	(1)	(2)	(3)	(4)	(5)	(6)
B. 2nd trimester effect of						
[a] Maternal influenza infection				-0.248***	-0.063**	-0.201**
[b] Nation-wide influenza index				-0.0029	-0.000791	-0.0042**
Ratio [a] /[b]				**85.5**	**79.6**	**47.9**
A. 3rd trimester effect of						
[a] Maternal influenza infection	-0.495***	0.064***	-112.966***			
[b] Nation-wide influenza index	-0.0095**	0.000792	-2.2783**			
Ratio [a] /[b]	**52.1**	**80.8**	**49.6**			

Notes: This table compares estimates from Tables 5 and 7 (based on individual mothers' influenza infections) with estimates from Table 11 (based on society-wide influenza spread). The table focuses on the third trimester for birth outcomes and on the second trimester on long-term outcomes, the pregnancy periods when effects tend to be most significant, respectively. The nationwide influenza index fluctuates by around 5 units over the year. Given ratios of 50 to 85, the cohort impact for the most affected cohorts equals to about 1/10 to 1/17 of the individual level effect.