The Lasting Legacy of Seasonal Influenza: In-utero Exposure and Human Capital Development.

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Abstract

Unfavorable circumstances in the fetal period have been shown to have impacts over the entire life course. Usually, these impacts are identified via catastrophes affecting cohorts such as pandemics, famines, and natural or manmade disasters. This study is the first to demonstrate long term effects of seasonal influenza, a more moderate threat that recurs every year and is preventable through vaccination. Using rich administrative data from Denmark, I show that *in utero* exposure to influenza is associated not only with worse health at birth, but with 10% lower earnings, a 7% decrease in labor market participation, and a strong 43% increase in welfare dependence. The 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. Birth outcomes are most strongly affected by third trimester exposure, while labor market outcomes are most affected by second trimester exposure. These findings suggest that influenza exposure has the potential to damage the fetus through multiple mechanisms and that much of the damage on human capital is not visible at birth. Informing mothers, doctors and policy makers about these large economic long-term costs of maternal influenza infections in order to decrease infection rates among pregnant women might have large potential effects on their offspring's human capital development.

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1. Introduction

The nine months spent *in utero* are increasingly recognized as a critical period that affects a person's health and economic outcomes over the entire life course. Indicators of health at birth such as birth weight have been found to predict future outcomes including earnings, employment, education and the health of the next generation.[1] In the economic literature, fetal shocks leading to poor health at birth are often identified via catastrophic events such as pandemics, famines, natural disasters, nuclear accidents or terrorist attacks.[2] I study the impact of a common and more moderate fetal shock: *in utero* exposure to seasonal influenza.

Influenza is a seasonal virus with annual epidemics that usually start in the fall and peak during the winter months, infecting 5-15% of the population. Recent research suggests that pregnancy renders women more susceptible to influenza and that influenza infections induce premature labor and harm the fetus (Rasmussen et al., 2008, Currie and Schwandt, 2013, Kay et al. 2014). Little is known, however, about the long-term effects on the offspring's human capital development. So far, long-term effects have only been studied in the context of influenza pandemics (Almond, 2006, Kelly, 2011) but not for seasonal influenza during ordinary non-pandemic years. Yet, the highly predictable annual reoccurrence of seasonal influenza and the ability to prevent infection via vaccination make it particularly relevant for public policy.

[1] Currie and Hyson 1999, Case, Fertig and Paxson, 2005, Currie and Moretti, 2007, Oreopoulos et al., 2008, Black et al., 2007, Royer, 2009, Figlio et al., forthcoming. For a comprehensive review of the broader literature see Almond and Currie, 2011.

[2] *Pandemics*: Almond, 2006, Kelly, 2011. *Famines*: Banerjee et al., 2007, Chen and Zhou, 2007, Almond et al. 2010, Lindeboom et al. 2010. *Natural disasters*: Torche, 2011, Simeonova, 2011, Currie and Rossin-Slater, 2013. *Nuclear accidents*: Almond, Edlund and Palme, 2009, Black et al., 2014. *Terror attacks*: Lauderdale, 2006, Camacho, 2008, Currie and Schwandt, 2014.

In this paper I use rich Danish register data for all individuals born between 1980 and 1993 in Denmark to explore the effects of *in utero* exposure to influenza on health at birth and on human capital outcomes in young adulthood. I first ask whether the strong seasonal pattern in annual influenza outbreaks in society at large implies a corresponding seasonal pattern in birth outcomes, at a time when there was essentially no vaccination of pregnant women for influenza in Denmark. Then I identify individual women who were hospitalized with influenza-like illness during pregnancy. I use this information to compare the health at birth and long-run human capital outcomes of those exposed to maternal influenza infection *in utero* to that of siblings who were not exposed. I provide balancing regressions documenting that disadvantaged mothers are more likely than others to be hospitalized with influenza and that using sibling comparisons eliminates this selection.

I find shorter gestation lengths for cohorts nearing full term during the peak of the influenza season compared to siblings born at a different time of the year. This seasonality in gestation length is more pronounced in years with stronger influenza outbreaks. Maternal infection with seasonal influenza occurring late in the pregnancy may have a negative effect on health at birth. Turning to the analysis of individual mother's influenza infections, I find a very similar pattern. Infants exposed to maternal influenza have poorer health at birth than siblings who were not exposed--rates of preterm birth and low birth weight are about twice as high. These effects are entirely driven by exposure during the third trimester.

Following exposed children and their siblings into young adulthood I find strong long-run effects on labor market outcomes. Those who suffer *in utero* exposure to maternal influenza infections earn 10% less than siblings who were not exposed. At the same time, income from government transfers is significantly increased. A corresponding pattern is observed in labor market

attachment. Participation is decreased by 5.3 percentage points (or 7%) while the probability of receiving welfare or disability pension as the main income source is increased by 3.8 percentage points (or by 43%). Educational outcomes at the extensive margin, such as years of schooling, are affected only for children of less educated mothers. In this subgroup there is also a considerably larger estimated impact on labor market outcomes, but the estimated effects on health at birth are only slightly stronger. This pattern of a small differences in health at birth that grow over time is in line with Heckman's (2007) capacity formation model and the underlying idea that "skill begets skill."

These long-run effects on human capital development are strongest for the second trimester, the period that corresponds to the most rapid fetal brain development.[3] Moreover, the estimated long-run effects remain strong when I control for birth weight and prematurity, suggesting that these measures do not capture the entire negative effect of exposure on the fetus.

These findings provide the first evidence that *in utero* exposure to maternal influenza affects long-term human capital development not only in the context of devastating pandemics but also during common non-pandemic years. Documenting these impacts of common seasonal influenza epidemics matters because unlike pandemics, they can be addressed by vaccination campaigns. It is particularly striking that much of the damage done by *in utero* influenza exposure is not captured by common measures of health at birth including birth weight and prematurity, making it less visible to doctors, mothers, and policy makers.

The long-run effects of *in utero* exposure to maternal influenza on labor market outcomes are economically significant. The 10% reduction in income is comparable to the estimated returns to an

[3] There are no significant effects of first trimester hospitalizations. However, maternal infections during the first trimester are likely to induce miscarriages (Bloom-Feshbach et al., 2011), which might imply a positive survival bias that cancels out potential negative effects.

entire year of schooling in the U.S. (Card, 2001). The long-run effects are also comparable to the estimated effects of low birth weight--an indicator of poor fetal development that is commonly used in the literature (Almond and Currie, 2011). A further benchmark is the average effect of maternal influenza in the population, estimated at the cohort level by the regression of labor market outcomes on a national influenza index at different pregnancy trimesters. These aggregate estimates are imprecise but they mirror the effects that are observed in the micro-level regressions.

Influenza vaccinations have been shown to protect pregnant women against infections and there are no known side-effects on the fetus (Rasmussen 2008). My findings therefore suggest that fostering influenza vaccinations among pregnant women would have positive long-term effects on their offspring's human capital development.

The paper proceeds as follows: Section 2 provides background information about the related literature and about seasonal influenza. Section 3 presents an overview of the data sources and Section 4 describes the methods used. Section 5 presents the results, followed by a conclusion in Section 6.

2. Background

The study most closely related to the estimated short-term effects on birth outcomes is that of Currie and Schwandt (2013), which analyzes the seasonality in health at birth among birth cohorts in New England. They document shorter gestation lengths among cohorts of pregnant women who near full term during the height of the influenza season. I find a very similar seasonality in the Danish data (see end of the Background Section). Moreover, I show that the same pattern of short-term effects is observed when analyzing influenza infections of individual mothers.

Long-run effects of *in utero* exposure to influenza on human capital development have so far only been documented in the context of influenza pandemics.[4] In a pioneering work, Almond (2006) uses a cohort level approach to document strong effects of *in utero* exposure to the 1918/19 "Spanish Flu" pandemic on human capital and health outcomes in the U.S. According to his estimates, exposure to maternal influenza decreases earnings in the long-run by 5-9%.[5] This is close to the 10% wage loss that I estimate and Almond (2006) notes that his estimates might be attenuated due to the devastating impact of the pandemic in terms of maternal mortality. Lin and Liu (2014) find similar effects of the 1918/19 "Spanish Flu" on human capital and adult health outcomes for Taiwan, a country that was not involved in the second world war. Kelly (2011), on the other hand, analyzes U.K. cohorts that were exposed to the 1957/58 "Asian Flu" pandemic, mainly during their second trimester. She finds only marginal effects on birth weight while the effects on test scores are stronger and detectable in the overall sample. Her results are plausible given my finding that second trimester exposure is not detectable in measures such as birth weight and gestational age even though it has a strong effect on human capital development.

One challenge of cohort-based studies such as Almond (2006) and Kelly (2011) is that it is often difficult to control for the type of mothers who select into different conception periods, as pointed out by Brown and Thomas (2011). I directly address this issue by including mother fixed effect, which can be thought of as a perfect control for the mother's type. Moreover, I show that the estimated effects of influenza in aggregate cohort level data correspond to micro-level estimates based on influenza hospitalizations of individual mothers.

[4] While this study is the first to document long-term effects of common seasonal influenza as opposed to pandemics, long-run effects have also been found for other common fetal insults. These include pollution (Isen et al., 2014, Currie et al., 2014), malnutrition (Almond and Mazumder, 2011, Almond et al. 2014, Baten, Crayen, and Voth, 2014), alcohol (Nilsson, 2014) and smoking (Bharadwaj et al., 2014).

[5] Almond (2006) finds that cohorts exposed in utero to the Spanish Flu have 1.6-3% lower wages. He assumes that one in three pregnant women got infected during the pandemic which implies a wage loss of 5-9% per actual case of *in utero* exposure.

Significant long-term effects of moderate and common health insults *in utero* are at odds with the traditional health capital framework proposed by Grossman (1972). This framework models health as a stock that depreciates over time, such that initial health shocks fade out. In contrast, Heckman's (2007) capacity formation model allows the effect of shocks to grow over time. The model posits a conventional constant elasticity of substitution production function to produce health, with health inputs at different life stages as inputs. A simplified Cobb-Douglas version of the capacity formation model can be written as

$$H_{adult} = I_{prenatal}^{\gamma} I_{postnatal}^{1-\gamma} \quad (1)$$

where H is health, and I are health inputs.

In this specification the health returns of postnatal inputs are complementary to the level of prenatal inputs. This means that differences in health at birth, e.g. due to differences in fetal conditions, may amplify over time as postnatal health inputs are accumulated. H may also stand for other adult outcomes that depend on pre- and postnatal inputs, such as cognitive skills. Equation (1) states that prenatal impairments of cognitive skills make later skill acquisition more difficult or that "skill begets skill."

2.1. 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 since not every infected person sees a GP and not every GP delivers a report. But it is indicative 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.[6]

Vaccine producers try to predict each year's influenza strain, with varying success (Luksza and Lässig, 2014). In Denmark before the early 2000s, however, influenza vaccination was recommended to only a few risk groups, excluding pregnant women, and patients had to pay for the vaccine (Rønne 2000). It is therefore safe to assume that during the time period analyzed in this study influenza vaccination rates were close to zero, in particular among pregnant women.

It is now known that pregnancy renders women more susceptible to and more severely affected by influenza (Neuzil et al. 1998, Fiore et al. 2009, Kay et al. 2014). Understanding the biological mechanisms behind this relationship is a central question of current medical and epidemiological research (Rasmussen 2008). In a recent study, Kay et al. (2014) find evidence that immune cells in pregnant women *over*react to influenza infections--an unexpected finding given that immune responses to other infections are typically suppressed by pregnancy to protect the fetus. This overreaction may cause additional inflammation in the pregnant mother that may harm the

[6] There is also some variation in the exact timing and the (age-specific) strength, depending on the mutation of the virus and the residual resistance in different cohorts of the society (see Figure A1 for the disaggregated times series of the influenza index). But the overall seasonality is highly predictable from year to year.

fetus rather than efficiently fighting the virus (which usually does not pass through the placenta to the fetus).

Influenza infections have been found to trigger adverse birth outcomes, in particular preterm labor, both for pandemic (Harris, 1919) and seasonal influenza (Rasmussen et al., 2008). Currie and Schwandt (2013) provide evidence of such effects at the society-wide level. While the causal pathways from influenza infections to premature labor are not yet fully understood, recent studies suggest that the inflammation caused by influenza infections is linked to the cascade of events triggering labor, a mechanism that is particularly relevant when nearing full term (Goldenberg et al., 2008, Uchide et al., 2005).

In terms of long-run health effects of *in utero* exposure to influenza, much of the existing medical literature has so far focused on its relationship to 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 a review of the literature see Ebert et al., 2005). The neural migration forming the brain and the synaptogenesis growth, i.e. the creation of new synapses, hit their peak during the second trimester (Tau and Peterson, 2009) and the inflammatory processes triggered by maternal influenza infections are believed to impair this main growth period of the brain. There is less evidence of long-term effects of exposure during the first trimester, when organs and vital functions are developed. Strong impairments during this early developmental stage may lead to miscarriages, implying a positive survival effect if the weakest fetuses are most likely to be fatally affected. In a recent study Bloom-Feshbach et al. (2011) analyze birth data from the US and three Scandinavian countries and find consistent evidence of strong increases in miscarriage rates among cohorts of women that were

exposed to pandemic influenza during their first pregnancy trimester, but not among those that were exposed in the second or third trimester.

In sum, there is some evidence that third trimester exposure to maternal influenza leads to preterm birth, while second trimester exposure may be particularly harmful for the brain development and first trimester effects could be overshadowed by positive survival bias. But overall the jury is still out on the magnitude of harms to the fetus, and when *in utero* influenza exposure is most harmful.

Figure 2 replicates the analysis of Currie and Schwandt (2013), showing the seasonal pattern in gestation length and influenza prevalence at birth in the Danish data. In order to obtain each figure, the infants' gestation length and the influenza prevalence at birth is regressed on month of conception dummies in models that include maternal fixed effects (see Appendix Section I for details). The figure plots the coefficients from these regressions.[7] Figure 2 indicates that gestation lengths are shorter for conception cohorts that are born during the influenza season. In particular, gestation length reaches a trough for conceptions in April and May. These conceptions near full term in January and February when the influenza season is at its peak. 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.[8], indicating a negative short-run effect of influenza exposure towards the end of pregnancy on a cohorts' average gestation length. This aggregate cohort-level approach is less well suited to an analysis of long-term human capital effects, however. Human capital during young

[7] The focus on the conception month is important to account for the strong seasonality in the conception rate that would bias results at the birth month level.

[8] Currie and Schwandt (2013) report a January-May difference of -0.08 weeks for birth data from New England over the past two decades in a partially vaccinated population, while I find a difference of -0.094 weeks in gestation length for cohorts conceived in January vs. May for Denmark during a time with essentially no influenza vaccination.

adulthood is related to month of birth for other reasons, e.g. due to schooling laws (Fredriksson and Öckert, 2014). Thus, one would have to partial out the average seasonality and rely on differences between stronger and weaker years such as in Figure 3. (This figure is already estimated with little precision for gestation length, see Table A1). Moreover, human capital might also be affected by influenza earlier in pregnancy thus adding further complexity to such a seasonality analysis. For that reason I focus my analysis on individual women's influenza infections. I present results using the overall influenza index at the end of the analysis.

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 which are available for the overall adult population (age 18+) from 1980 up to 2012 when the sample cohorts were of age 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.

3.2 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 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 often result from in-vitro fertilization, 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 (Currie 2009). The personal identifier of the mother allows siblings to be matched in the birth data.

3.3. Influenza index and maternal influenza infections during pregnancy

The only influenza surveillance measure available for the analyzed time period is the index based on general practitioners' reports of influenza-like illness mentioned in the background section. These ILI cases include patients with symptoms that subjectively appear to be influenza-related to the general practitioners, 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. However, the availability of this ILI index is exceptional for that time period. For the U.S. there is no surveillance data before the mid-1990s, and researchers have to rely on indices based on influenza and pneumonia deaths (Weinberger et al., 2012). Moreover, ILI reports remain the international standard for influenza surveillance (Paget et al., 2007).

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 world-wide 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 (Babock et al., 2006), I define influenza-like illness to include the diagnosis codes for influenza (ICD-8: 470-474), pneumonia (ICD-8: 480-486), unspecified respiratory symptoms (ICD-8: 460-462,464,465,466) and unspecified viral symptoms (ICD-8: 071,074-079). Notice that influenza is the most common pathogen of pneumonia during pregnancy (Goodnight and Soper, 2005). 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. To avoid unnecessary jargon I will refer to ILI as "influenza" in the remainder of the paper.

Figure 4 compares the seasonality of the national influenza index and the fraction of pregnant women with an influenza diagnosis at birth. Maternal influenza cases follow the general seasonal pattern neatly with the lowest level during the summer months, an increase in the fall and a peak in February.

3.4. 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 limited to the date and the type of the most recently awarded degree as well as a variable that indicates whether the individual was enrolled in formal education on October 31st of the previous year. 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 31st to the same year, leads to similar results (though it does not allow to include 2012 data for which school enrollment on October 31st is not available). Years of schooling are constructed by totaling years in school after age 18 and adding it to the years of schooling implied by the degree at age 18 (9 years if highest degree is compulsory schooling and the individual is not in school at age 18; 18-6 years if the individual is in school at age 18 or completed high school that year).

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.

3.5. Sample restrictions and descriptive statistics

The sample is restricted to conceptions between 1/1980 and 12/1992 to mothers of age 18 or older. Maternal hospital admissions, a central variable of this analysis, are not observed before 1/1980 or for mothers below age 18. 12/1992 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 conceptions of a given conception month are observed in the available birth data which is important for the seasonality analysis.[9] 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 descriptive statistics for the newborns. 51.3% are boys, 4.5% are born preterm and 4.4% 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 30% above the Danish rate (5.9%). The rates for white U.S. women

[9] For example, for conceptions in 4/1979, a month not included in the sample, only full-term births would be observed in 1/1980. Likewise, for conceptions in 4/1993, only preterm births would be observed up to 12/1993.

with at least a high school education are more similar (Chen, Oster and Williams, 2014). More than 97% of the births are matched to the population and income registers at age 18 and above. Table 1 (b) shows mother characteristics. There are 468,412 mothers overall, or on average 1.54 children per mother. Six percent of the mothers are foreign born (compared to 15.5% in the U.S.), and the average age is 27.51.

As shown in panel (c) there are 1,756 cases of influenza hospitalizations during pregnancy (referring to at least one admission per pregnancy). There are 222 hospitalizations during the first trimester of pregnancy, 335 during the second, and 1,274 during third trimester. 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 likely higher rates in a risk group like pregnant women), these cases of influenza hospitalizations (about 0.25%) 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 was introduced in Denmark only in the late 1990s and before that, i.e. during my sample period, doctors had little incentive to code their diagnoses accurately. 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. Overall, I find that the micro-level estimate based on influenza hospitalizations is in a similar range

as the average influenza effect in society at large, suggesting that these two opposing factors offset each other.

Panel (d) of Table 1 shows descriptive statistics for matched long-run outcomes during young adulthood. There are in total 5,396,536 matches, or 7.5 matches per individual. Average age is 22.27, given that only earlier born cohorts are observed at higher ages. About half of the sample is receiving education, with an average of 14 years of schooling. Of those who are not receiving education, 76% are participating in the labor force (either employed or unemployed) while 8.7% receive their main income from welfare or disability pension.

4. Empirical approach

The following empirical model is used to analyze the effect of maternal influenza infections during pregnancy on birth outcomes and human capital development.

$$Y_{i,t} = \alpha + \beta Influenza_i + \mu_{mom} + \delta X_{i,t} + \varepsilon_{i,t} \quad (2)$$

where $Y_{i,t}$ are measures of health at birth as well as labor market and educational outcomes at different ages in early adulthood. $Influenza_i$ are maternal influenza hospitalizations at different points during pregnancy. μ_{mom} are mother fixed effects. 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 conception year and the conception month (including dummies for the year and month of birth instead of those for conception does not affect results). In the long-run regressions I additionally add fixed effect 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 the fact that there are multiple

observations on individuals. To compare these micro-level estimates to the aggregate influenza effect, I also present regressions in which I replace maternal influenza hospitalizations by the national influenza index.

β measures the causal 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 or her marital status 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 these kinds of 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. Pischke and Schwandt (2014) show that a more powerful test is provided by balancing regressions that use these controls as a dependent variable on the left-hand side of the regression equation. The power discrepancy between this kind of balancing regressions and the coefficient movement tests is particularly large if the control variables are noisy proxies of the true underlying potential confounders, which might often be the case. 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 this selection is eliminated by within-mother comparisons.

5. Results

Table 2 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, less educated, and more likely to live in the Copenhagen area (Denmark's capital and most densely populated area), and they earn 16% less. They are slightly older while there is no significant association with marital status. Panel B repeats the balancing regressions with a broad set of controls, including all maternal characteristics except the respective dependent variable. Coefficients decrease only slightly compared to the bivariate regressions and 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 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.

Table 3 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 likelihood of being preterm by 4 percentage points. Further, they weight 77 grams less and are 2.9 percentage points more likely to have low birth weight. These effects are large. Given baseline rates of 4.5% and 4.4%, prematurity and low birth weight rates are increased by 89% and 66%, respectively.

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 to control for covariates the way a multivariate regression model does but in the current setting the most relevant control is the mother fixed effects. 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 81g which is close to the point estimate of 77g reported in Table 3. Moreover, the distributions diverge particularly strongly below the 2500g cutoff which explains the strong effect on the rate of low birth weight.

Epidemiological and medical literature concerning “fragile males” has found that fetal shocks often harm male fetuses more than female fetuses, which can lead to a higher proportion of fetal losses among male fetuses and thus a negative effect on the gender ratio at birth (Kraemer

2000). Column (5) of Table 3 shows that the estimated effect on the gender ratio at birth 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 (gender-specific effects on birth and human capital outcomes are reported below).

One concern might be that influenza diagnoses proxy for other, more severe health conditions that may induce hospitalizations and influenza becomes diagnosed merely "on the side." In Appendix Table A2 I repeat the birth outcome regressions controlling for the other diagnoses that are most often coded along with influenza, such as pregnancy complications or prenatal care inspections. The resulting estimates decrease only slightly, suggesting that the estimated effects of influenza do not merely proxy for these other diagnoses. Notice that the inclusion of these additional controls may bias the estimated effect of influenza towards zero if influenza infections are the primary reason for hospitalization, which seems a likely scenario. Column (3) of Table A2 indicates that influenza also does not merely proxy for hospitalization per se. In fact, controlling for hospitalization slightly increases estimated effects because mothers who are never hospitalized during pregnancy tend to have lower socio-economic status.

Table 4 shows regressions of birth outcomes on dummies indicating influenza hospitalizations during the first, second, and third trimester. As the estimates in columns (1) to (4) show, effects are entirely driven by influenza admissions during the third trimester. None of the other trimester effects are significant or of a similar size as the third trimester effect. Notice however that the second trimester effect on gestation length is particularly small, while it is still of about half the size of the third trimester effect for birth weight. In columns (5) to (8) I use influenza infections six to ten months after conception as an instrument for influenza in the third trimester, i.e. between month six and birth. This IV strategy, pioneered by Currie and Rossin-Slater (2013), corrects for a

potential bias induced by the fact that shorter gestation lengths shorten the time period during which a mother may get infected with influenza. 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. Since 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.

Overall, the results in Tables 3 and 4 indicate strong effects of influenza infections during pregnancy on birth outcomes that are driven by third trimester infections, i.e. *in utero* exposure to infections close to birth. This pattern is in line with the seasonal comovement of gestation length and influenza spread in the birth month shown in Figure 2 and with the existing literature on first trimester influenza and prematurity. Moreover, the fetus gains the most weight during the third trimester which explains the similar effect pattern for birth weight.

The analysis thus far examines the effect of individual influenza hospitalizations on health at birth. However, a major contribution of this study is that we can also examine long-term outcomes. Panel A of Table 5 shows the effect of maternal influenza hospitalizations on labor market outcomes at age 18 and above, excluding years when an individual receives education (results are similar in the overall sample). The estimate in column (1) indicates that young adults who were exposed to influenza infections *in utero* have wages about 10% lower than their siblings who were not exposed. This is a strong effect, similar in size to the return of an entire year of schooling. The effect overall income including government transfers, on the other hand, is less than half the size of the wage effect and not significantly different from zero, suggesting that transfer income might partly compensate for the wage losses induced by influenza exposure *in utero*. 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 more than 23%.

Columns (4) to (6) show effects on employment status. There is a strongly significant, negative effect of about 5.3 percentage points on the probability of being in the labor force (that is either employed or unemployed). The likelihood of receiving welfare or a disability pension as the main income source is increased by almost 4 percentage points. Given a baseline of 8.7%, the effect on transfer receipts corresponds to an increase of almost 50%. 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 conditional on joining the labor force. In other words, affected adults tend to leave the labor force if it would take them longer to find a job.

Figure 6 shows the distribution of log earnings for exposed and unexposed siblings. 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 -0.102 reported in Table 5. This figure shows that the earnings effect is not driven by one particular income group but by a uniform shift of the entire distribution.

An important question is whether these strong long-term effects on human capital run through the effects on the birth outcomes described above or whether they are also present conditional on observable birth outcomes. To answer this question I include flexible controls for birth outcomes, i.e. fixed effects for the exact week of gestation and for 300-gram birth weight groups, in Panel B of Table 5. The effects on the labor market outcomes are remarkably robust to the inclusion of these detailed birth outcomes controls. Estimates decrease only slightly compared to Panel A, and they remain highly significant, indicating that the effects on labor market outcomes may work on top of the effects on observable health at birth. However, as Pischke and Schwandt

(2014) 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.[10]

Another way to examine whether the effects on labor market outcomes run through health at birth is to analyze effects by trimester. The first three columns in Table 6 show that as in the case of birth outcomes there are significant negative effects of third trimester exposure that are of similar size as the overall effects. However, unlike for birth outcomes there are significant effects of second trimester exposure that are larger in magnitude than the third trimester effect. This pattern is plausible given that in the second trimester the brain is developed and the synaptogenesis is growing strongest, which makes this period the most relevant for long-term effects on cognitive ability. The second trimester effect is particularly strong for wages. *In utero* exposure to maternal influenza in the second trimester is more than twice as detrimental to wages as exposure during the third trimester, while it is about 50% and 10% above the third trimester effect for labor force participation and welfare receipt, respectively. Columns (4) to (6) of Table 6 additionally control for health at birth directly, which decreases third trimester effects slightly while it leaves the second trimester effect unchanged.

In sum the results in Tables 5 and 6 suggest that *in utero* exposure to maternal influenza has strong effects on the labor market outcomes of young adults. These long-run effects on human capital are not entirely driven by the impact that is observable in health at birth but they are also present conditional of birth outcomes. The strong long-run effects the second trimester exposure are in line with the medical literature that predicts particularly strong effects on later cognitive

[10] 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 (Pischke and Schwandt, 2014).

development due to the brain development during that pregnancy period. There are no significant first trimester effects, but negative effects may cancel out with a positive survival bias if infections induce miscarriages (Bloom-Feshbach et al., 2011). 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 potential positive effect on miscarriages.

Table 7 shows the estimated effects on educational outcomes. For the overall sample there are no significant effects on the probability of being in school or on years of schooling between ages 18 and 20. Education in Denmark is free and students are entitled to generous stipends. Hence, there might even be more of an incentive to continue schooling for the less able if they cannot easily find a job or if they are unlikely to earn high wages in the labor market. This mechanism might be particularly relevant for children of more educated parents for whom higher education is the baseline rather than the exception. For children with a less advantaged parental background, on the other hand, these educational outcomes might be a more relevant margin. In line with this reasoning, the estimates in columns (3) and (4) show that there are negative effects of maternal influenza infections on educational outcomes when restricting the sample to less educated mothers. Trimester-specific effects in columns (5) and (6) are imprecise due to the reduced sample size, but as for labor market outcomes, point estimates are strong for the second trimester.

The strong effect heterogeneity shown in Table 7 could also be driven by a stronger initial effect on the birth outcomes of children with less advantaged parental backgrounds. For example, disadvantaged mothers may tend to develop stronger infections, or other types of disadvantages (such as poor nutrition or stress) may interact with influenza to produce poor outcomes. In this case a similar heterogeneity should be observable in health at birth. Table 8 columns (1) to (4) show that effects on birth outcomes are indeed stronger for less educated mothers, but the difference in effects

is only about 20% and therefore relatively small compared to the three-fold difference in the education effect. The heterogeneity in the estimated effects of influenza exposure on labor market outcomes on the other hand, is very strong (about twice as large for children of less educated mothers), as shown in columns (5), (7), and (9). Moreover, part of the effect seems to run through the impact on education, as point estimates decrease by about 10% when I control for the child's own education in columns (6), (8) and (10).

To sum up, the results in Table 7 and 8 show that the difference in the estimated effects of *in utero* exposure between advantaged and disadvantaged mothers is small at birth but increases over time. This pattern is in line with Heckman's (2007) capacity formation model that states that a lower initial skill level makes later skill acquisition more difficult. Another complementary factor contributing to the increasing effect difference might be if more educated parents invest more resources to compensate for ability differentials between their children. Alternatively, there could be already stronger impacts on cognitive endowment at birth among affected children of less educated mothers that are simply not observable in birth weight and gestation length. But the similarity of the estimated effects on these observable birth outcomes suggests that influenza infections are not much stronger in disadvantaged mothers per se. Hence, for this explanation to hold influenza infections would have to interact with other disadvantages of less educated mothers (such as poor nutrition or stress) in a way that affected brain development more strongly than birth outcomes.

The gender of the newborn may be another potentially relevant dimension for effect heterogeneity. Even though I do not find significant effects on the gender ratio at birth (Table 3), there might be gender differences in effect strength that are not dramatic enough to be detectable in differential rates of still birth. Columns (1) to (4) of Appendix Table A3 show that the effect of influenza during pregnancy on birth outcomes is stronger for boys in terms of point estimates, but

that the differences are not significant in any of the four regressions. The long-run effect on male human capital outcomes is considerably larger for wages, with an interaction coefficient that is significant at the 10% level. However, the difference is smaller and not significant for the other long-run outcomes. For welfare receipt the point estimate suggests that men are less affected than women. Overall, the results in Table A3 suggest that males might be more affected both at birth and in the long run (in line with the literature on the 'fragile males'), but confidence intervals are too large to provide conclusive evidence.

How large are the long-run effects that I find compared to the impact of low birth weight, a measure that is often used to indicate poor fetal development (Currie and Moretti, 2007, Oreopoulos et al., 2008, Black et al., 2007, Royer, 2009, Figlio et al., forthcoming)? In Table 9 I compare the effects of influenza during pregnancy (Panel A) to the impact of low birth weight (Panel B), including mother fixed effects in all regressions. The results indicate that the effects of influenza during pregnancy are generally stronger than the effects of low birth weight, but the estimates are of the same order of magnitude for each outcome. Moreover, the effect patterns across the different outcomes are remarkably similar. For example, the effect on log wages is much stronger than the effect on log income in both panels. And in both cases, the effect on the probability of being unemployed is close to zero and not significant. This comparison shows that neither the size of the maternal influenza effect nor the structure across outcomes is unreasonable. Instead, the estimated effects are similar to a well-established indicator of poor fetal development in both dimensions.

Another important implication of the above analysis is that influenza diagnoses during the second trimester have the strongest long-run effects on labor market outcomes. Here, a comparison of the long-run impacts with the effects of low birth weight is not informative, because birth weight is a summary measure at delivery that does not provide information about the time during pregnancy

that fetal growth was hindered. Moreover, given that the fetus gains the most weight in the third trimester, birth weight is not likely to serve as a good proxy for fetal shocks that occur earlier in pregnancy.

A better trimester-specific comparison is provided by estimates of the long-run impact of the national influenza index, which can be matched to the different trimesters of an individual woman's pregnancy. Table 10 shows the estimated effects of the influenza index on birth and human capital outcomes using the same specification as in the micro-level regressions shown in the previous tables. As expected (see discussion above), the estimates are imprecise, in particular when month fixed effects are included. However, the pattern of third and second trimester effects mirrors the estimated effects from the micro-level regressions in Tables 4 and 6. Birth outcomes are most affected by the influenza index during the third trimester, while for labor market outcomes, the point estimates are strongest for the second trimester, though the confidence intervals are large and the differences in coefficient size are not statistically significant.

Comparing the point estimates in the micro- and aggregate-level analysis, it is natural to ask what fraction of pregnant women would have to experience influenza in the micro-level analysis in order to match the average effect estimated using the influenza index? Multiplying the influenza index estimates by 6 (the three-month average of the index in the months around the peak of the influenza season) and comparing them to the corresponding trimester effects in Tables 4 and 6 suggests that about 1 in 10 to 1 in 30 pregnant women would have to be infected during the influenza season in order to match the aggregate impact of the influenza index (see Appendix Table A4).

In terms of influenza infection rates, values of 1 in 10 to 1 in 30 are at the lower end of the official estimates that assume average rates of 1 in 5 to 1 in 20 in the overall population and higher rates for risk groups such as pregnant women (Russell et al., 2008). This suggests that the micro-level estimate based on influenza hospitalizations is moderately stronger than the average effect in the overall population but that both estimates are in a similar range.

6. Conclusion

Using rich administrative data from Denmark, I document long-run effects of *in utero* exposure to seasonal influenza on human capital development. Maternal influenza infections during pregnancy are associated not only with worse health at birth, but 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 not only in the context of devastating pandemics but also during common non-pandemic years. Importantly, much of the damage caused by *in utero* exposure to maternal influenza is not detectable in common measures of health at birth such as birth weight and gestation length, suggesting that influenza exposure has the potential to damage the fetus through multiple mechanisms.

These results 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 Center 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). A major concern of mothers is that the vaccine may harm the fetus, despite broad information campaigns stating that there are no harmful side-effects. Informing doctors and pregnant women about the potential lasting costs of not getting vaccinated rather than emphasizing that the vaccine does not harm the fetus might be a more effective strategy to raise vaccination rates. Developing effective ways to get this message to pregnant women and their doctors should be a priority for future research.

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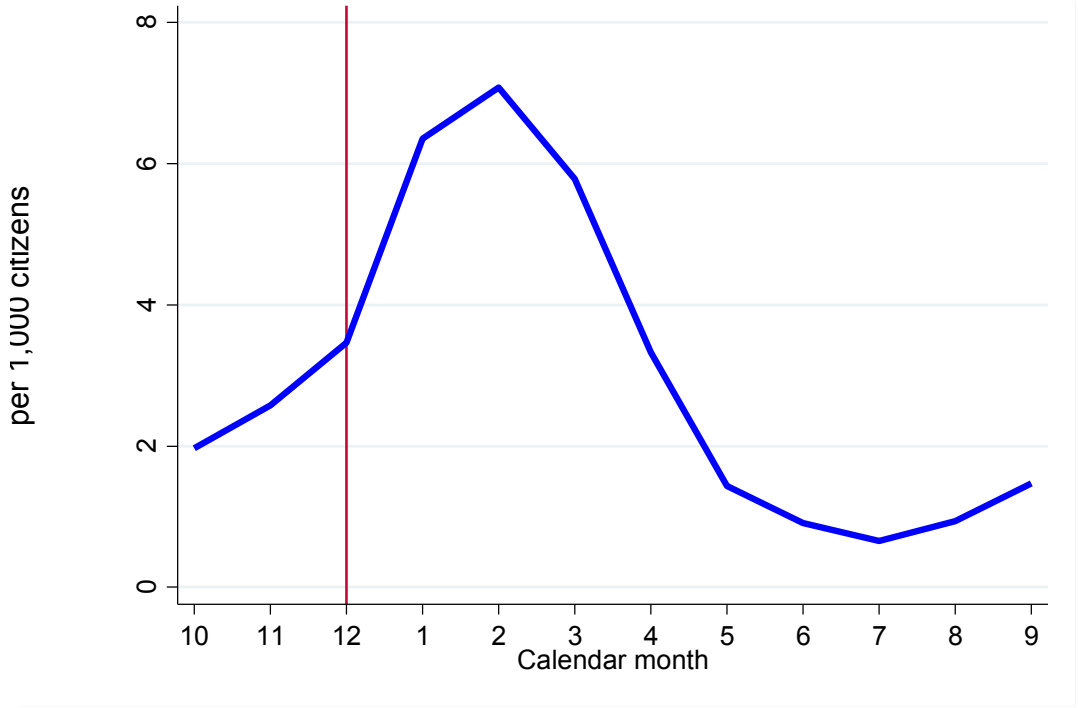
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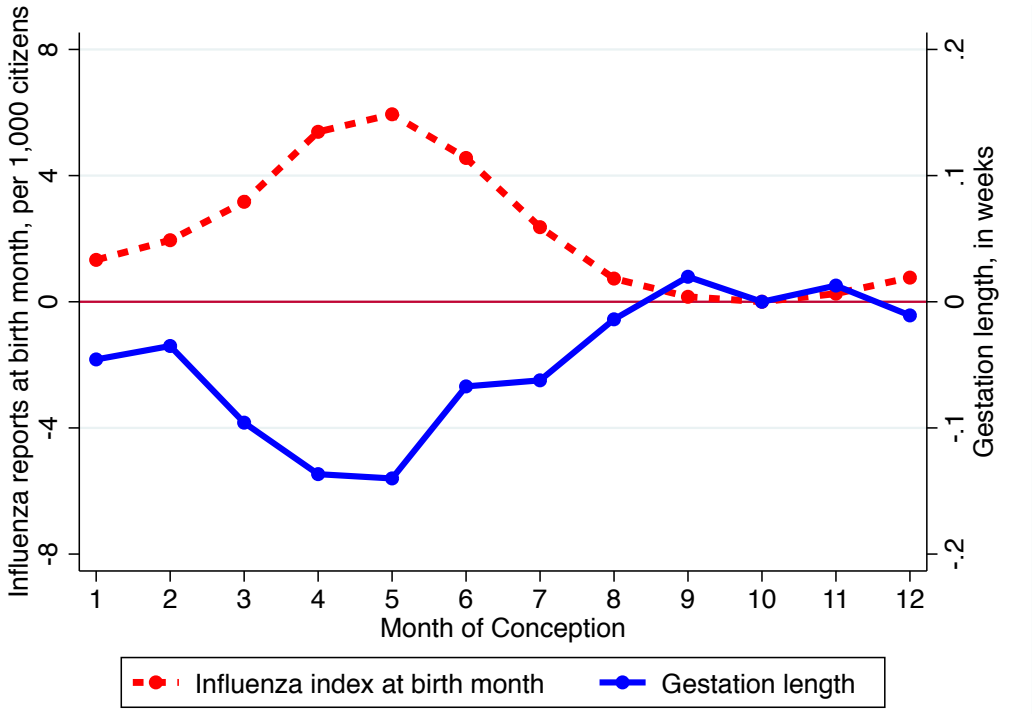
8. Tables and Figures

Figure 1: Seasonality of influenza spread in Denmark



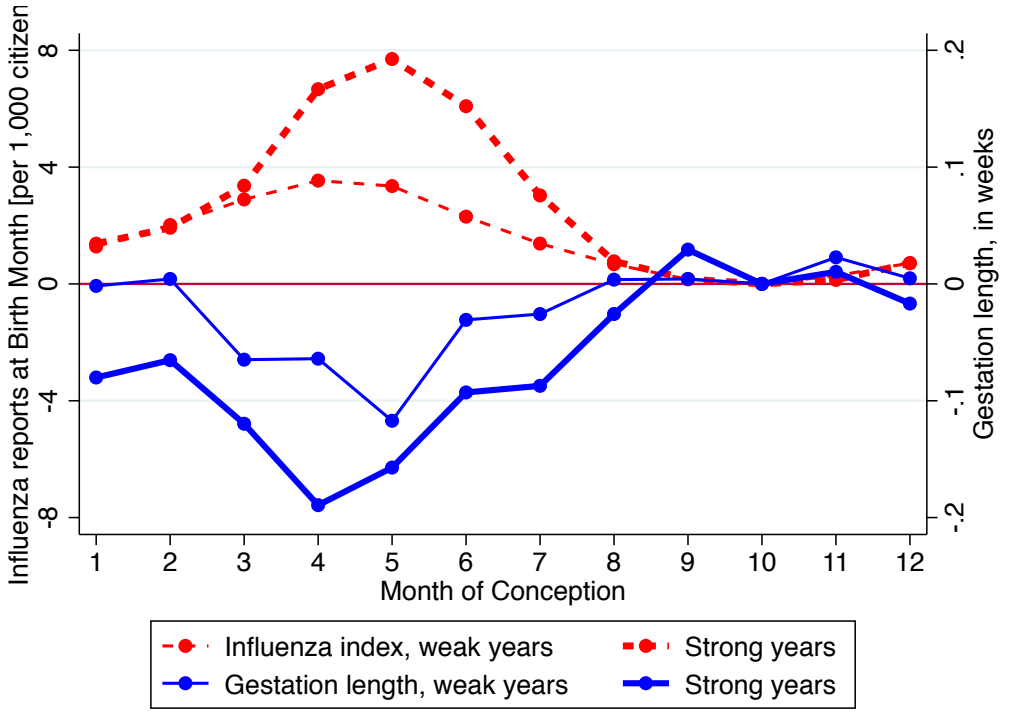
Notes: Monthly cases of patients with influenza-like illness reported by Danish general practitioners for the years 1980-1993 are divided by the current Danish population and averaged by calendar month. For the disaggregated times series see Appendix Figure A1.

Figure 2: Influenza index at birth and gestation length



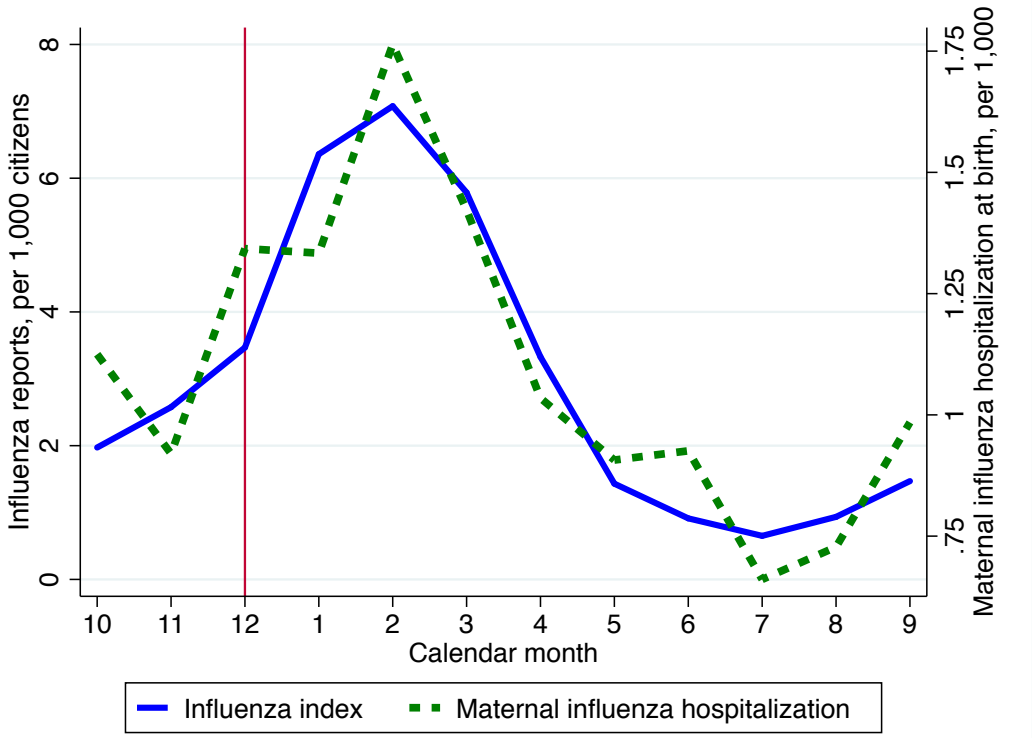
Notes: Coefficients from regressions of the influenza index at birth (left y-axis) and the gestation length (right y-axis) on conception month dummies are displayed. Both regressions include fixed effects for the mother, parity and gender of the newborn, mother's age group, education, region of residence 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.

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



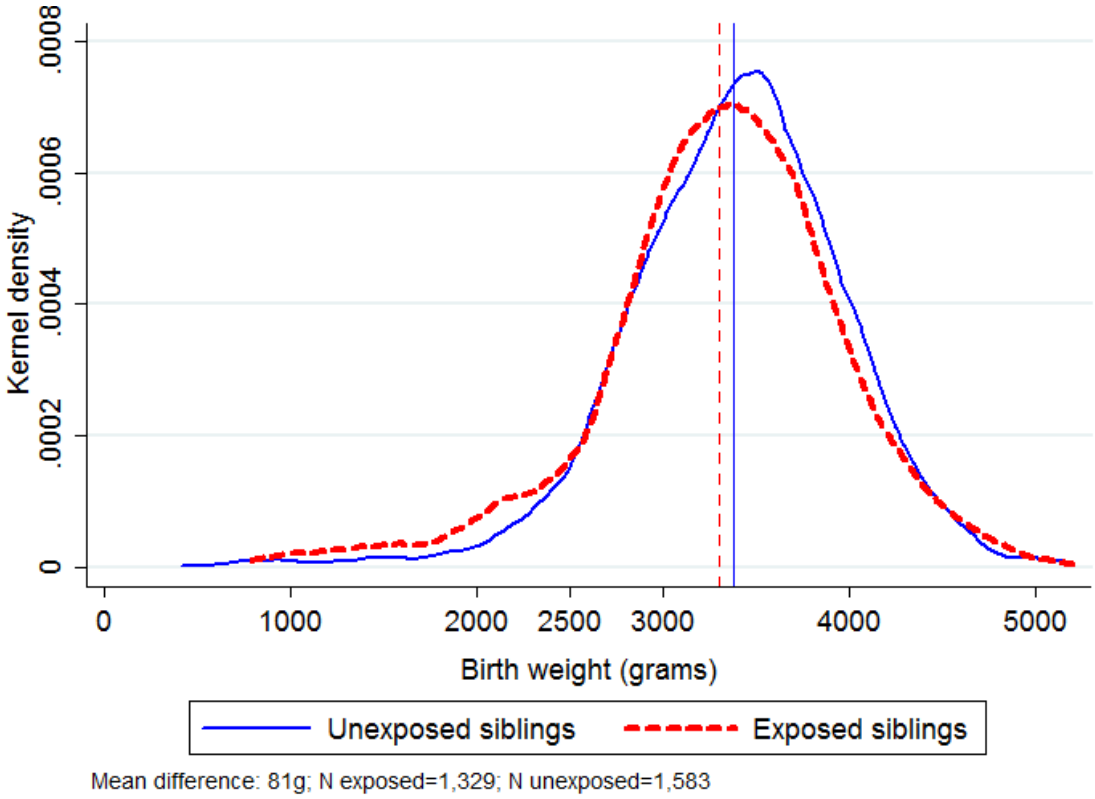
Notes: Displayed are coefficients from regressions of the influenza index at birth (left y-axis) and the gestation length (right y-axis) on conception month dummies, interacted with a dummy for conceptions in years that are followed by a strong influenza season ('80, '82, '83, '84, '85, '86, '89, '92; 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



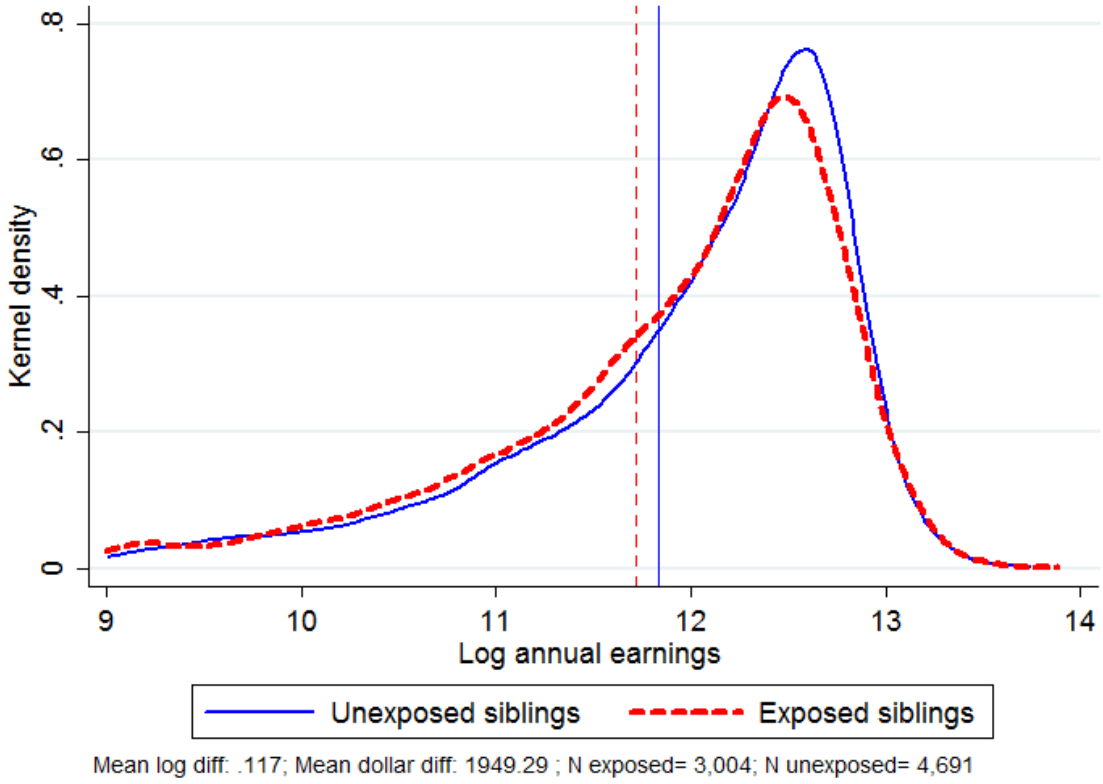
Notes: For comments on the influenza index see Figure 1. Maternal influenza diagnosis at birth refers to hospitalizations at the date of birth with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms.

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. The mean difference is 81 grams which is close to the point estimate of -76.72 in column (3) of Table 3.

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. The mean difference is 0.117 which is close to the point estimate of -0.102 in column (1) of Table 5.

Table 1: Descriptive Statistics

(a) Births, N=719,854	Mean	Std dev.
Boys	0.513	
Gestation (weeks)	39.63	1.81
Premature (<37 weeks)	0.045	
Birth weight (gram)	3,439	562
Low birth weight (<2500g)	0.044	
Observed at age 18+	0.974	
(b) Mothers, N=468,412	Mean	Std dev.
Children per mother	1.54	
Foreign born	0.06	0.25
Low education (9 yrs)	0.354	
Age	27.51	4.72
Birth in hospital	0.981	
(c) Influenza hospitalizations	n	
during pregnancy	1,756	
during 1st trimester	222	
during 2st trimester	335	
during 3st trimester	1,274	
(d) Matches at age 18+: 5,396,536	Mean	Std dev.
Observations per matched PID	7.50	
Age	22.27	3.38
Years of education	13.92	2.78
In school	0.54	
If not in school		
In labor force	0.76	
Unemployed	0.024	
On welfare/disab. pension	0.087	
Log income	11.97	1.00
Log wage	11.85	1.13

Notes: The sample includes all individuals conceived between 1/1980 and 12/1992, born to mothers of age 18+. 'Influenza hospitalizations' refers to hospitalizations with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms

Table 2: Balancing regressions of maternal characteristics on influenza during pregnancy

Dependent variable	Foreign mother (1)	Mother's yrs of education (2)	Residence in Copenhagen (3)	Mother's log earnings (5)	Mother's age (4)	Mother is married (5)
A. Mother characteristics on influenza						
Influenza during pregnancy	0.057 ***	-0.340 ***	0.017 **	-0.152 ***	0.024 *	0.131
	(0.008)	(0.069)	(0.008)	(0.027)	(0.012)	(0.113)
B. Mother characteristics on influenza + baseline controls						
Influenza during pregnancy	0.037 ***	-0.233 ***	0.018 **	-0.105 ***	-0.012	-0.067
	(0.006)	(0.065)	(0.008)	(0.023)	(0.010)	(0.095)
C. Mother characteristics on influenza + baseline controls + mother FEs						
Influenza during pregnancy	-	0.007	0.005	-0.029	-0.015	-0.006
	-	(0.052)	(0.006)	(0.043)	(0.022)	(0.019)
N	718,280	683,151	715,433	639,534	719,854	719,854
Mean dep. var.	0.064	11.67	0.111	11.55	27.51	0.526

Notes: "Influenza" refers to hospitalizations with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms. 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 3: 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)
Influenza during pregnancy	-0.288 ***	0.041 ***	-76.72 ***	0.029 **	-0.024
	(0.098)	(0.012)	(25.83)	(0.012)	(0.029)
Baseline controls	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes
N	719,854	719,854	718,777	718,777	702,786
Mean dep. var.	39.63	0.045	3,439	0.044	0.513

Notes: "Influenza" refers to a dummy variable indicating hospitalizations with an influenza-like illness diagnosis, including ICD-8 codes for influenza, pneumonia, and unspecified respiratory and viral symptoms. 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 individuals conceived between 1/1980 and 12/1992, born to mothers of age 18+. Robust standard errors are clustered at the municipality level.

Table 4: Effect of influenza on birth outcomes, by trimester

Dependent variable	Gestation	Prematurity	Birth weight	Low birth weight	Gestation	Prematurity	Birth weight	Low birth weight
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Influenza during								
First trimester	0.293	-0.017	23.75	-0.010	0.298 *	-0.017	23.69	-0.010
	(0.248)	(0.030)	(57.26)	(0.036)	(0.176)	(0.019)	(38.48)	(0.024)
Second trimester	-0.018	0.008	-37.91	0.029	0.000	0.008	-35.74	0.027
	(0.280)	(0.025)	(62.31)	(0.031)	(0.173)	(0.016)	(38.61)	(0.021)
Third trimester	-0.445 ***	0.058 ***	-101.95 ***	0.031 **	-0.577 ***	0.066 ***	-123.08 ***	0.041 ***
	(0.113)	(0.015)	(32.23)	(0.015)	(0.083)	(0.011)	(22.89)	(0.011)
Baseline controls	yes	yes	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes	yes	yes
Third trimester IV					yes	yes	yes	yes
Mean dep. var.	39.63	0.05	3,439	0.04	39.63	0.05	3,439	0.04

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. Third trimester IV regressions use influenza hospitalizations in month 6 to 10 after conception as an instrument for third trimester influenza hospitalizations, i.e. between gestation month 6 and birth. N=719,854 in the gestation regressions and N=718,777 in the birth weight regressions. For further comments see Table 3.

Table 5: Effect of influenza on labor market outcomes

Dependent variable	Log wage	Log income (incl. transfers)	Log non-wage income	Labor force participation	On welfare / disab. pension	Unemployed (if in labor force)
	(1)	(2)	(3)	(4)	(5)	(6)
A. Baseline						
Influenza during pregnancy	-0.102 **	-0.041	0.235 ***	-0.053 ***	0.038 ***	0.004
	(0.040)	(0.032)	(0.072)	(0.014)	(0.012)	(0.005)
B. Controlling for health at birth						
Influenza during pregnancy	-0.095 **	-0.043	0.229 ***	-0.051 ***	0.035 ***	0.004
	(0.039)	(0.032)	(0.072)	(0.014)	(0.011)	(0.005)
Baseline controls	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes
N	2,091,838	2,475,481	2,410,859	2,491,578	2,422,250	1,849,269
Mean dep. var.	11.85	11.97	9.14	0.76	0.087	0.024

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 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 individuals conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to year 2012. Individuals are excluded when receiving education. Regressions in panel B control for health at birth, i.e. indicators for the exact week of gestation and for 300g birth weight groups.

Table 6: Effect of influenza on labor market outcomes, by trimester

Dependent variable	Log wage	Labor force participation	On welfare / disab. pension	Log wage	Labor force participation	On welfare / disab. pension
	(1)	(2)	(3)	(4)	(5)	(6)
Influenza during						
First trimester	0.059	-0.007	0.007	0.052	-0.009	0.008
	(0.096)	(0.035)	(0.027)	(0.096)	(0.034)	(0.027)
Second trimester	-0.252 ***	-0.074 **	0.048 *	-0.255 ***	-0.074 **	0.048 *
	(0.077)	(0.030)	(0.027)	(0.077)	(0.030)	(0.027)
Third trimester	-0.093 *	-0.052 ***	0.042 ***	-0.080	-0.048 ***	0.037 ***
	(0.050)	(0.017)	(0.014)	(0.049)	(0.016)	(0.014)
Baseline controls	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes
Health at birth				yes	yes	yes
N	2,091,838	2,491,578	2,422,250	2,091,838	2,491,578	2,422,250
Mean dep. var.	11.85	0.76	0.087	11.85	0.76	0.087

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 and 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 individuals conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to year 2012. Individuals are excluded when receiving education. 'Health at birth' controls are indicators for the exact week of gestation and for 300g birth weight groups.

Table 7: Effect of influenza on educational outcomes

	All mothers		Mothers with low education			
Dependent variable	In school age 18-20	Education yrs age 18-20	In school age 18-20	Education yrs age 18-20	In school age 18-20	Education yrs age 18-20
	(1)	(2)	(3)	(4)	(5)	(6)
Influenza during pregnancy	-0.014	-0.047	-0.047 ***	-0.192 **		
	(0.010)	(0.045)	(0.018)	(0.079)		
Influenza during						
First trimester					-0.007	-0.033
					(0.046)	(0.205)
Second trimester					-0.052	-0.171
					(0.040)	(0.171)
Third trimester					-0.044 **	-0.193 **
					(0.021)	(0.096)
Baseline controls	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes
N	2,043,490	2,043,490	731,051	731,051	731,051	731,051
Mean dep. var.	0.75	12.22	0.66	11.87	0.66	11.87

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 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 individuals conceived between 1/1980 and 12/1992, born to mothers of age 18+, and observed at age 18+ up to year 2012. Mothers with low education are those with at most 9 years of schooling at latest observed birth.

Table 8: Effect of influenza on birth and labor market outcomes in sample of low educated mothers

	Sample: Mothers with low education									
	Birth outcomes				Labor market outcomes					
Dep. var.	Gestation	Pre-maturity	Birth weight	Low birth weight	Log wage		Labor force participation		Welfare/ disability pension	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Influenza during pregnancy	-0.341 **	0.051 **	-91.49 *	0.036 *	-0.208 ***	-0.182 ***	-0.104 ***	-0.090 ***	0.079 ***	0.065 ***
	(0.168)	(0.022)	(47.42)	(0.021)	(0.060)	(0.060)	(0.020)	(0.019)	(0.019)	(0.018)
Ratio to effect in overall sample	1.18	1.24	1.19	1.24	2.03	1.78	1.95	1.69	2.10	1.71
Baseline controls	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
Own education						yes		yes		yes
N	254,609	254,609	254,236	254,236	885,244		1,111,060		1,076,678	
Mean dep. var.	39.53	0.054	3,376	0.056	11.79		0.708		0.114	

Notes: Mothers with low education are those with at most 9 years of schooling at latest observed birth. "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. The sample includes all cohorts conceived between 1/1980 and 12/1992, observed at birth in columns (1)-(6) and at age 18+ up to year 2012 in columns (5)-(10). Robust standard errors are clustered at the municipality level in columns (1)-(4) and at the individual level in columns (5)-(10). In columns (5)-(10) individuals are excluded when receiving education. 'Own education', included as controls in columns (6), (8) and (10), are dummies for the individual's years of education and highest degree.

Table 9: Comparison of long-term influenza effects with long-term effects of low birth weight and prematurity

Dependent variable	Log wage	Log income (incl. transfers)	Log non-wage income	Labor force participation	On welfare / disab. pension	Unemployed (if in labor force)
	(1)	(2)	(3)	(4)	(5)	(6)
			A. Regressions of outcomes on influenza			
Influenza during pregnancy	-0.102 **	-0.041	0.235 ***	-0.053 ***	0.038 ***	0.0044
	(0.040)	(0.032)	(0.072)	(0.014)	(0.012)	(0.0053)
			B. Regressions of outcomes on low birth weight			
Low birth weight	-0.064 ***	-0.020 **	0.178 ***	-0.045 ***	0.041 ***	0.002
	(0.012)	(0.008)	(0.021)	(0.004)	(0.003)	(0.002)
Baseline controls	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes
N	2,091,838	2,475,481	2,410,859	2,491,578	2,422,250	1,849,269
Mean dep. var.	11.85	11.97	9.14	0.76	0.09	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 and 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. Sample includes all cohorts conceived between 1/1980 and 12/1992, observed at age 18+ up to year 2012. Individuals are excluded when receiving education.

Table 10: Regressions of birth and human capital outcomes on influenza index

	Birth outcomes						Human capital outcomes		
Dep. var.	Gestation		Prematurity		Birth weight		Log wage	Lab. force participation	Welfare/ disab. pen.
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Monthly influenza cases per 1,000 Danes, during									
1st trimester	0.0056	0.0003	-0.0003	-0.0001	-0.093	-0.808	-0.0021	-0.0005	0.0004
	(0.0039)	(0.0041)	(0.0004)	(0.0004)	(0.780)	(0.954)	(0.0023)	(0.0006)	(0.0005)
2nd trimester	0.0078 ***	0.0025	-0.0004	-0.0001	-1.046	-1.476	-0.0030	-0.0012 **	0.0005
	(0.0030)	(0.0046)	(0.0003)	(0.0005)	(0.711)	(1.131)	(0.0025)	(0.0005)	(0.0005)
3rd trimester	-0.0126 ***	-0.0101 *	0.0008 *	0.0008	-2.017 **	-2.312 *	-0.0006	-0.0003	-0.0006
	(0.0045)	(0.0055)	(0.0004)	(0.0006)	(0.872)	(1.193)	(0.0029)	(0.0006)	(0.0006)
Baseline controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month FEs		Yes		Yes		Yes	Yes	Yes	Yes
N	719,854	719,854	719,854	719,854	718,777	718,777	2,091,838	2,491,578	2,422,250

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. 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 monthly 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 monthly level. The sample includes all cohorts conceived between 1/1980 and 12/1992, observed at birth in columns (1)-(6) and at age 18+ up to year 2012 (excluding years when receiving education) in columns (7)-(9).

9. Appendix

Appendix Section I: 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

$$Y_i = \alpha + \sum_{m=1}^{12} \beta_m D[cmonth_m] + \delta X_i + \mu_{mom} + \varepsilon_i$$

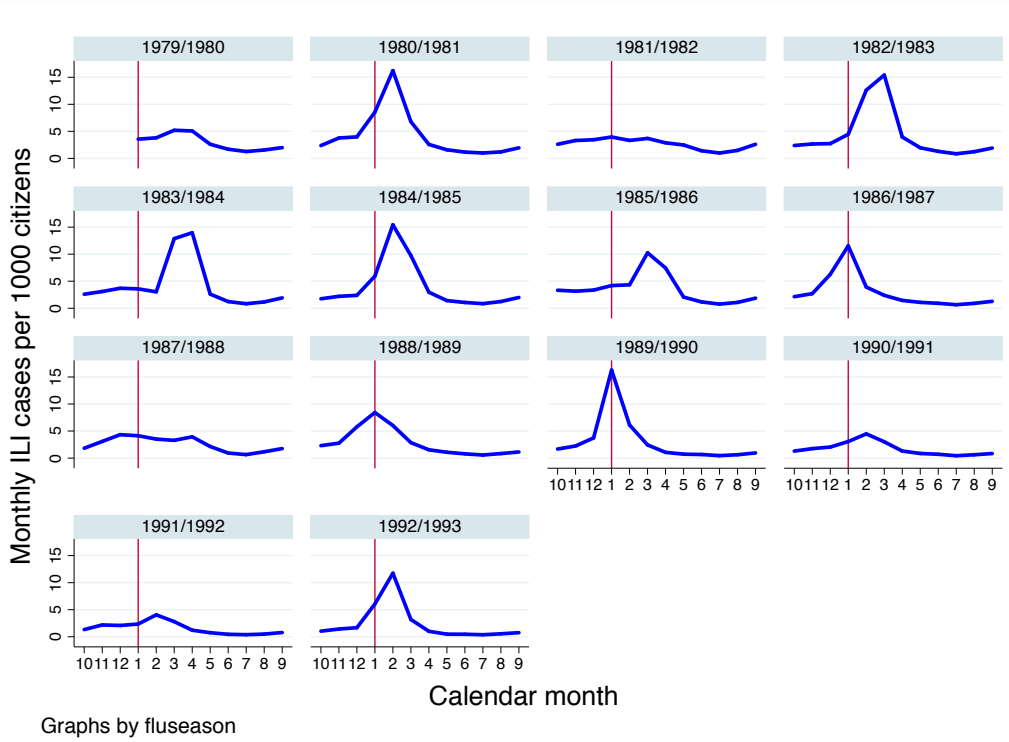
where the index *i* refers to the newborn. Y_i refers to the gestation length when analyzing the seasonality in health at birth, and to the national influenza index in the month of birth when analyzing the seasonality of influenza spread. $D[cmonth_m]$ are dummies for the conception month.

For the interpretation of seasonality effects it is important to focus on the month of conception rather than the month of birth. An analysis at the month of birth level might be strongly confounded because seasonal fluctuations in the conception rate induce a mechanical relationship of birth month and the rate of premature births (Currie and Schwandt, 2013). Fluctuations in the conception rate induce fluctuations in the number of pregnancies that are at risk of premature delivery eight months after conception. When measured at the birth month the prematurity rate relates the number of premature births coming from one conception cohort to the number of full-term births coming from an earlier conceived cohort. Hence, a month with a high conception rate, such as December, will imply a high prematurity rate eight months ahead and a small rate nine months ahead even in the absence of any causal seasonal impact on prematurity. Focusing on the month of conception accounts for differences in the conception rate because it relates preterm births to the full-term births of the same conception cohort.

μ_{mom} are mother fixed effects that account for time-fixed differences in socio-economic characteristics of mothers that tend to conceive in different month (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 conception year, and a quadratic time trend at the monthly level. Standard errors are clustered at the monthly level to account for the fact that the influenza index only varies at that level. The regression results of this analysis are displayed in Table A1 and illustrated in Figures 2 and 3.

Appendix Section II: Figures and Tables

Figure A1: Influenza index; influenza-like illness cases reported by general practitioners per 1,000 Danish citizens, 1980-1993



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.

Table A1: Regressions of influenza index and gestation length on conception month dummies

Dep. var.	Influenza index at month of birth			Gestation length (wks)		
Model		Weak vs. strong flu years			Weak vs. strong flu years	
	Overall	Main effect	Strong seasons	Overall	Main effect	Strong seasons
	(1)	(2)	(3)	(4)	(5)	(6)
Month of conception						
January	1.327 ***	1.282 ***	0.095	-0.046	-0.002	-0.078 *
	(0.271)	(0.186)	(0.329)	(0.122)	(0.121)	(0.044)
February	1.951 ***	2.016 ***	-0.093	-0.035	0.004	-0.070
	(0.300)	(0.195)	(0.366)	(0.112)	(0.116)	(0.044)
March	3.168 ***	2.893 ***	0.469	-0.096	-0.065	-0.055
	(0.482)	(0.505)	(0.841)	(0.100)	(0.103)	(0.050)
April	5.387 ***	3.537 ***	3.130 **	-0.137	-0.064	-0.125 ***
	(0.856)	(0.678)	(1.422)	(0.088)	(0.088)	(0.037)
May	5.944 ***	3.351 ***	4.350 ***	-0.140 **	-0.117	-0.040
	(0.827)	(0.372)	(1.195)	(0.071)	(0.072)	(0.040)
June	4.558 ***	2.303 ***	3.782 ***	-0.067	-0.031	-0.062 *
	(0.809)	(0.236)	(1.217)	(0.061)	(0.062)	(0.036)
July	2.364 ***	1.381 ***	1.648	-0.062	-0.026	-0.061
	(0.627)	(0.348)	(1.044)	(0.047)	(0.049)	(0.038)
August	0.735 ***	0.672 ***	0.102	-0.014	0.004	-0.029
	(0.274)	(0.258)	(0.362)	(0.035)	(0.041)	(0.037)
September	0.157	0.157	0.009	0.020	0.004	0.025
	(0.275)	(0.230)	(0.346)	(0.030)	(0.036)	(0.039)
October			reference month			
November	0.257	0.280	-0.144	0.013	0.023	-0.012
	(0.293)	(0.223)	(0.333)	(0.023)	(0.029)	(0.038)
December	0.764 ***	0.714 ***	-0.007	-0.011	0.005	-0.022
	(0.281)	(0.240)	(0.337)	(0.034)	(0.042)	(0.040)

Notes: Columns (1) and (3) show coefficients from regressions of the influenza index and the gestation length on conception month dummies. Columns (2) and (4) display the main effects from a model with an interaction for years followed by strong influenza seasons ('80, '82, '83, '84, '85, '86, '89, '92). The interaction term of these models is displayed in columns (3) and (5). 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 A2: Effect of influenza on birth outcomes, controlling for other diagnoses and hospitalization at birth

	Effect of influenza during pregnancy		
	Baseline	Control for other diagnoses	Control for hospitalization
	(1)	(2)	(3)
Gestation length (weeks)	-0.288 ***	-0.261 ***	-0.282 ***
	(0.099)	(0.099)	(0.091)
Prematurity (<37wks)	0.0413 ***	0.0366 ***	0.0403 ***
	(0.0119)	(0.0121)	(0.0116)
Effect on birth weight	-76.65 ***	-66.24 **	-75.27 ***
	(25.86)	(26.22)	(22.14)
Low birth weight (<2500g)	0.0287 **	0.0236 *	0.0292 ***
	(0.0119)	(0.0122)	(0.0108)
Baseline controls	yes	yes	yes
Mother FEs	yes	yes	yes

Notes: 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 cohorts conceived between 1/1980 and 12/1992. Robust standard errors are clustered at the municipality level. Other diagnoses are "pregnancy complications", "prenatal care inspection" and "delivery related."

Table A3: Effect of influenza on birth outcomes and human capital accumulation, by gender

	Birth outcomes				Human capital outcomes			
Dependent variable	Gestation	Pre-maturity	Birth weight	Low birth weight	Log wage	Lab. force participation	Welfare/ disab. pension	In school (age 18-20)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Influenza during pregnancy	-0.161	0.029 *	-63.38 *	0.018	-0.029	-0.031	0.053 ***	-0.008
	(0.152)	(0.016)	(38.59)	(0.017)	(0.059)	(0.020)	(0.018)	(0.015)
Influenza during pregnancy *boy	-0.134	0.012	-19.40	0.011	-0.142 *	-0.044	-0.031	-0.011
	(0.187)	(0.024)	(50.47)	(0.023)	(0.079)	(0.027)	(0.023)	(0.020)
Baseline controls	yes	yes	yes	yes	yes	yes	yes	yes
Mother FEs	yes	yes	yes	yes	yes	yes	yes	yes
N	702,786	702,786	701,919	701,919	2,091,838	2,491,578	2,422,250	2,070,494
Mean dep. var.	39.63	0.045	3,439	0.044	11.81	0.74	0.08	0.66

Notes: 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 cohorts conceived between 1/1980 and 12/1992, observed at age 18+ up to year 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 A4: Effect size comparison of maternal influenza and influenza index

	Birth outcomes			Labor market outcomes		
	Gestation	Prematurity	Birth weight	Log wage	Lab. force participation	Welfare/ disab. pen.
	(1)	(2)	(3)	(4)	(5)	(6)
A. 3rd trimester effect of						
[a] Maternal influenza diagnosis	-0.5767 ***	0.0658 ***	-123.08 ***	-0.0934 *	-0.0521 ***	0.04 ***
[b] Nation-wide influenza index	-0.0101 *	0.0008 *	-2.31	-0.0006	-0.0003	0.00
Ratio [a] /[b*6]	9.5	14.3	8.9	25.6	32.0	-10.9
B. 2nd trimester effect of						
[a] Maternal influenza diagnosis				-0.2515 ***	-0.0740 **	0.0481 *
[b] Nation-wide influenza index				-0.0030	-0.0012 **	0.0005
Ratio [a] /[b*6]				14.1	10.6	15.1

Notes: Row [a] of panels A. and B. displays estimates reported in columns (5) to (7) of Table 4 and columns (1) to (3) of Table 6. Row [b] displays estimates reported in Table 10. The ratio describes the size of the micro-level effect of maternal influenza relative to the effect of the nation-wide influenza index in the months around the peak of the influenza season. The index effect is multiplied by 6, which is the average value of the index that a mother would face in a given pregnancy trimester that includes the peak of the influenza season.