



Short communication

The effects of in utero exposure to the 1918 influenza pandemic on family formation

Jason M. Fletcher

La Follette School of Public Affairs, Department of Sociology, Center for Demography and Ecology, Center for Demography of Health and Aging, University of Wisconsin-Madison, 1180 Observatory Drive, Madison, WI, 53706, United States

ARTICLE INFO

Article history:

Received 12 December 2017
 Received in revised form 19 April 2018
 Accepted 11 June 2018
 Available online xxx

Keywords:

In utero exposure
 Life course analysis
 Family formation
 1918 influenza pandemic

ABSTRACT

A growing literature ties in utero conditions to life course outcomes, including education, earnings, and adult health and mortality. A smaller literature has begun to examine the intergenerational impacts of in utero conditions. A link between these two literatures—the impacts of in utero conditions on family formation—has had few examinations but offers a potential set of mechanisms for the intergenerational reach of early conditions. This paper draws from the 1960 US Decennial Census to examine whether individuals exposed in utero to the 1918/19 influenza pandemic had different family formation patterns than adjacent unexposed cohorts. The findings suggest small overall effects on marriage rates, number of children, and several measures of “type” of spouse for men, but moderate effects for women. For example, women with in utero exposure during their first trimester marry men with 0.2 fewer years of schooling than those not exposed. The findings show that exposed individuals have spouses with lower schooling than unexposed counterparts, this effect is particularly large for women, and it increases the likelihood of marrying spouses with very low levels of schooling.

© 2018 Elsevier B.V. All rights reserved.

1. Introduction and background literature

A large and growing literature has shown evidence of life course effects of poor in utero conditions (Almond and Currie, 2011 for review). A key set of studies have used the 1918/1919 influenza pandemic as an in utero shock that was largely unanticipated and short lived in order to estimate causal effects on life course outcomes. Almond (2006) estimated that in utero exposure to the influenza pandemic in the US resulted in lower educational attainments as well as increased the likelihood of disability and lower incomes fifty or more years later. Related research (Mazumder et al., 2010; Almond and Mazumder, 2005; Fletcher, 2014) has linked this exposure to additional measures of adult health and mortality. Much of this work has been supportive of the importance of the fetal origin hypothesis—that early exposures shape long term health and socioeconomic trajectories, possibly through the early programming of critical body systems and epigenetic signatures that persistent throughout the lifecourse.

A smaller literature has taken findings from animal models that point to the possibility that insults to one generation may impact future generations to begin to examine the intergenerational effects of the influenza pandemic. Richter and Olof Robling (2013)

were the first to identify an effect of prenatal exposure to the 1918 flu pandemic on the outcomes of the subsequent generation. The authors use historical influenza morbidity data matched to birth information and find that maternal in utero exposure in the second trimester lowers educational attainment for female offspring but not for male offspring. They also find an analogous result for paternal exposure and male outcomes: second trimester exposure lowers educational attainment for male offspring.¹ Cook et al. (2016) use a sample from Wisconsin and find that children of exposed individuals had lower educational attainment, as did grandchildren.

An important question raised by the emerging evidence of intergenerational effects of in utero exposure is whether the mechanism is biological or social (or both). A possible biological mechanism is through epigenetic markers—where the environment “turns off/on/up/down” levels of genetic expression—that may be able to pass between parents and children. While there is currently limited evidence of this mechanism in humans,² many studies using animal models, particularly mice, have shown these

¹ van den Berg and Pinger (2016) examine multigenerational effects of famine during pre-puberty in Germany during World War I.

² Sen et al. (2015) presents supporting evidence from 35 mother-infant pairs in Detroit.

E-mail address: jason.fletcher@wisc.edu (J.M. Fletcher).

effects (Stewart et al., 1980). An alternative mechanism linking in utero exposure to an insult to the outcomes of the next generation is through social mechanisms. For example, exposed individuals could suffer in the marriage market and thus the offspring would have “lower quality” parents. While there has been much investigation of the impacts of the influenza pandemic in the US, investigations of family formation have not been undertaken.

Indeed, much of the evidence in the literature that focuses on impacts of in utero exposures on marriage and fertility outcomes is from less developed settings³ or from earlier historical periods.⁴ Marital status was found to be negatively impacted by early childhood exposure to the 1959–61 China famine (Almond et al., 2007; Brandt et al., 2016). Lee (2014) found increases in the number of children born and reductions in spousal education for women in utero during the harshest part of the Korean War.⁵ This paper extends the literature by estimating the impacts of in utero exposure to the 1918/1919 influenza pandemic on marriage and fertility outcomes using data from the 1960 Census. Results suggest that this domain of outcomes were affected in exposed females but not exposed males. The results suggest a need for further investigation for the sources of these gender differences.

A potential implication of these results suggests a difficulty of interpreting

intergenerational effect estimates in other work. Specifically, models of transgenerational inheritance of epigenetic signatures often rely on a sex-difference between the number of generations between the exposure and the outcome to obtain evidence for inheritance (e.g. Richter and Olof Robling, 2013). Because pregnant females carry two generations (fetus and sex cells of the fetus) during an environmental exposure, researchers often seek a fourth generation's outcomes (exposed mother, daughter, child-of-daughter, grandchild-of-daughter) to confirm transgenerational effects. Since an exposed male only carries a single generation of sex cells, researchers can examine impacts on the (not directly exposed) third generation to confirm transgenerational effects. The findings of this paper, that in utero shocks may have sex-specific social effects, further cloud the ability of prior work to take advantage of sex-specific effects across generations to confirm transgenerational transmission.

2. Empirical methods

The empirical analysis largely follows Almond (2006) and subsequent work that focuses the data on a narrow window of birth cohorts around the 1918/1919 influenza pandemic. The key assumption is that the pandemic arrived quickly, exposed a narrow set of birth quarter-cohorts, and then vanished quickly. The speed of the pandemic and that it was unanticipated would suggest that birth cohorts surrounding the 1918/1919 “treated” group can serve as appropriate counterfactual controls to allow a causal interpretation of the findings. Since the marriage and fertility outcomes of these birth cohorts are also affected by the Great Depression (when the exposed cohorts were approximately 15–20 years old) (e.g. Hill, 2015), having these control groups who were also affected by the Great Depression can potentially allow a separation between exposure to the pandemic and the Great Depression.

The main analysis uses a 10-year window on each side of the pandemic, but additional analyses are shown that narrows and

³ Donaldson and Keniston (2014) find evidence of investments in child quality in India due to the influenza pandemic.

⁴ Bengtsson and Dribe (2006) examine the impacts of exposure to small pox on fertility in the 18th and 19th century.

⁵ A larger literature has explored the impacts of childhood exposure (rather than a focus on in utero exposure) on later marriage market outcomes and have shown sex differences in the effects (e.g. Van den Berg and Gupta, 2015).

Table 1

Descriptive Statistics 1960 Census, Birth Cohorts.1908–1927.

Variable	Obs	Mean	Std Dev	Min	Max
Educational Attainment	442,907	10.70	3.02	0	17
Any Children	442,907	0.72	0.45	0	1
Number of Children	442,907	1.85	1.74	0	9
Married	442,907	0.85	0.36	0	1
Age at First Marriage	410,672	23.11	5.06	14	50
Male	442,907	0.49	0.50	0	1
Non White	442,907	0.10	0.31	0	1
Age	442,907	40.91	5.43	32	51
Born 1918 Q4	442,907	0.01	0.12	0	1
Born 1919 Q1	442,907	0.01	0.11	0	1
Born 1919 Q2	442,907	0.01	0.11	0	1
Born 1919 Q3	442,907	0.01	0.11	0	1
Born 1919 Q4	442,907	0.01	0.12	0	1
Born 2Q	442,907	0.25	0.43	0	1
Born 3Q	442,907	0.26	0.44	0	1
Born 4Q	442,907	0.24	0.43	0	1
Birth Year	442,907	1918	5.41	1909	1927

widens this windows to assess the robustness of the main results. The key controls are for birth year and birth quarter, and the analysis is first pooled and also stratified by gender. A key estimating equation from the literature is given by the following form:

$$y_{it} = \beta_0 + \beta_1(YOB = 1918/19)_i + \gamma T_{it} + \varepsilon_{it}$$

The primary focus is on the coefficient β_1 , which measures the effect of influenza exposure on a number of outcomes for i individuals. Year of birth time trends and their square are denoted by γT_{it} , and ε_i is representative of a clustered error term (on birth year).

More specifically, we will further separate the YOB indicator into five quarter of birth indicators, as the influenza pandemic began in the final quarter of 1918 and lasted into the second quarter of 1919. Thus, potentially exposed individuals were born between the fourth quarter of 1918 (only exposed during the third trimester) and the fourth quarter of 1919 (mainly exposed in the first trimester). The estimating equation is thus:

$$y_{it} = \beta_0 + \beta_1(1918, Q4)_i + \beta_2(1919, Q1)_i + \beta_3(1919, Q2)_i + \beta_4(1919, Q3)_i + \beta_5(1919, Q4)_i + \gamma_1 T_{it} + \gamma_2 Q_{it} + \varepsilon_{it}$$

where birth quarter fixed effects (Q_i) are also added to the equation, as Buckles and Hungerman (2013) have shown that the composition of births differ by quarter of birth.

3. Data

The data for this study are drawn from the public use version of the 1% US 1960 Decennial Census available at IPUMS (Ruggles et al., 2015).⁶ Alternative datasets that were considered were the 1940, 1950, and 1970 Census files. The 1940 Census is now complete count, though it only allows a snapshot of outcomes in an early stage of the life course (i.e. the respondents who were exposed to the 1918/19 influenza pandemic are surveyed when they are 21/22 years old). Since the average age-at-first marriage for women during this time is 21 (see Table 1 below), the fertility and marriage outcomes would be severely censored. A limitation with the 1950 Census is that every household has one “sample-line” person who answered questions, so that the analysis that explores impacts on spousal characteristics would be unavailable; in addition, quarter of birth is unavailable for individuals over the age of one. The 1970

⁶ <https://usa.ipums.org/usa/sampdesc.shtml#us1960a>.

Download English Version:

<https://daneshyari.com/en/article/7348108>

Download Persian Version:

<https://daneshyari.com/article/7348108>

[Daneshyari.com](https://daneshyari.com)