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Is the 1918 Influenza Pandemic Over? Long-Term Effects of *In Utero* Influenza Exposure in the Post-1940 U.S. Population

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This paper uses the 1918 influenza pandemic as a natural experiment for testing the fetal origins hypothesis. The pandemic arrived unexpectedly in the fall of 1918 and had largely subsided by January 1919, generating sharp predictions for long-term effects. Data from the 1960–80 decennial U.S. Census indicate that cohorts in utero during the pandemic displayed reduced educational attainment, increased rates of physical disability, lower income, lower socioeconomic status, and higher transfer payments compared with other birth cohorts. These results indicate that investments in fetal health can increase human capital.

I. Introduction

According to the fetal origins hypothesis (Barker 1992), certain chronic health conditions can be traced to the course of fetal development. Randomized experiments with animals have supported the hypothesis, but its relevance for humans remains controversial because of obstacles to evaluation. Chief among these are (i) omitted factors, such as genetic

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endowments, that may "stack" nonexperimental studies toward positive findings and (ii) the inherent difficulty of detecting delayed effects, particularly when the period of latency is long.

The 1918 influenza pandemic presents an exceptional opportunity to evaluate effects of the prenatal environment using U.S. Census data. Twenty-five million persons in the United States contracted the debilitating influenza strain and survived. Some of the highest infection rates were observed among women of childbearing age, one-third of whom contracted influenza. As census micro data identify both the place and quarter of birth of respondents, these can be linked to the timing and geographic variation in influenza infection.

Two distinct features of the 1918 pandemic severely limit the scope for omitted variables bias. First, the pandemic struck without warning[1] in October 1918 and had largely dissipated by the beginning of 1919 (figs. 1*a* and 1*b*), implying that cohorts born just months apart experienced markedly different in utero conditions. This presents a severe test of the fetal origins hypothesis since the design generates sharp predictions for differences in adult outcomes among individuals born within months of one another. Second, the severity of the pandemic varied widely and idiosyncratically across states. Pregnant mothers in Kansas, for example, experienced more than 10 times the increase in mortality rates than mothers in Wisconsin. This second approach uses geographic variation to identify within-cohort differences in fetal exposure to the pandemic. In order to bias estimates, omitted factors would have to follow the same abrupt and idiosyncratic patterns as the pandemic.

The two estimation approaches reveal large impacts of this negative shock to the health endowment on a range of census outcomes. Fetal health is found to affect nearly every socioeconomic outcome recorded in the 1960, 1970, and 1980 Censuses.[2] Men and women show large and discontinuous reductions in educational attainment if they had been in utero during the pandemic. The children of infected mothers were up to 15 percent less likely to graduate from high school. Wages of men were 5–9 percent lower because of infection. Socioeconomic status (as measured by the Duncan occupation index [Duncan 1961]) was substantially reduced, and the likelihood of being poor rose as much as 15

[1] As late as September 28, 1918, the *Journal of the American Medical Association* claimed that nothing more worrisome than the regular flu had arrived and that influenza had "already practically disappeared from the Allied Troops" ("The Epidemic of Influenza," 1918, 1063). The spring 1918 "herald wave" of the pandemic (Olson et al. 2005) was identified only in hindsight; Jordan (1927), commissioned by the American Medical Association to study the worldwide pandemic, noted that the spring outbreak would have received scant attention had it not been for the massive upsurge in deaths that followed.

[2] These are the only postpandemic censuses to report quarter of birth of adult respondents (see Sec. IV).

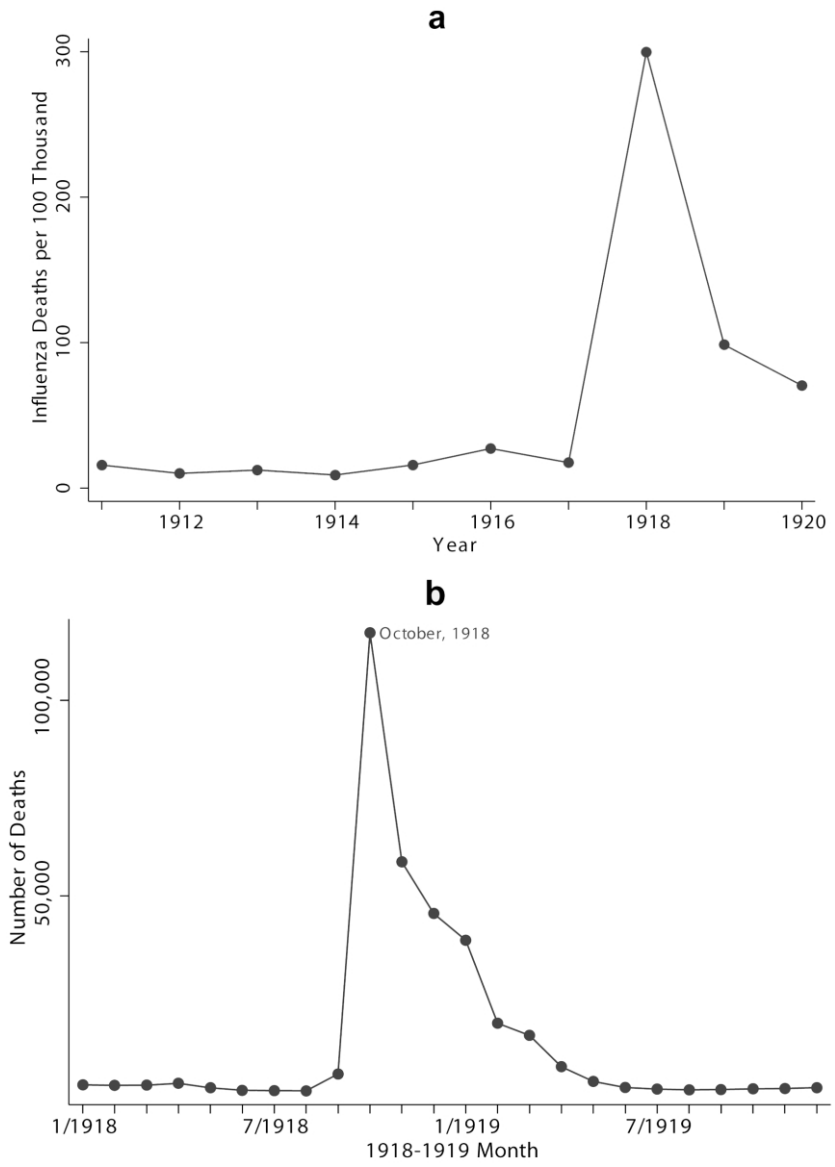

FIG. 1.—U.S. influenza deaths: *a*, by year; *b*, by month

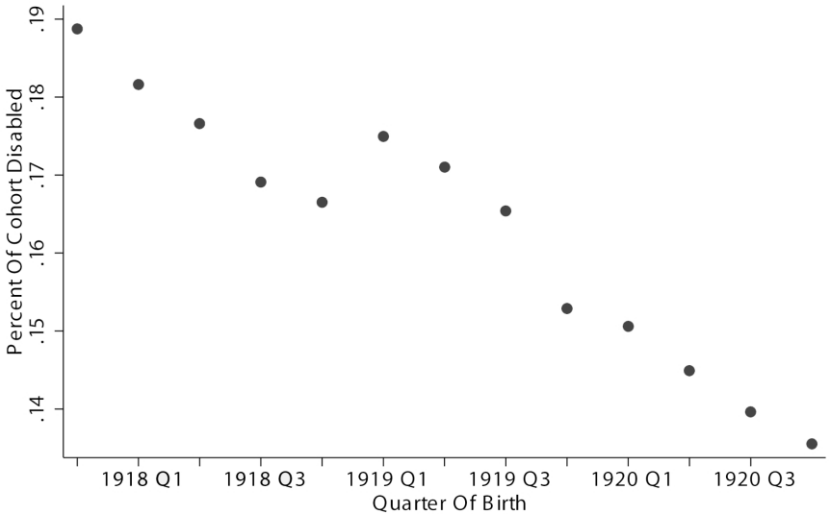

FIG. 2.—1980 male disability rates by quarter of birth: prevented from work by a physical disability.

percent compared with other cohorts. Public entitlement spending was also increased: those in their first trimester of gestation during the pandemic's peak had the highest average welfare payments in the 56 birth quarters from April 1911 to April 1925. Male disability rates in 1980 provide a stark example. Figure 2 plots by quarter of birth the share of men prevented from working by a physical disability. While a downward trend is observed—younger men are less likely to be disabled—a clear departure from this trend is evident for men born between January and September of 1919. These birth cohorts were in utero at the height of the pandemic and are estimated to have 20 percent higher disability rates at age 61 as a result of fetal influenza exposure.

This broad range of socioeconomic impacts is found among men, women, whites, and nonwhites alike. The estimated effects are qualitatively similar across the two estimation approaches, and the fact that damage is found for both workers and nonworkers suggests a *continuum* of fetal origins impairment rather than a threshold at which debilitation occurs.

Despite high infection rates, the U.S. pandemic mortality rate of 0.5 percent was low when compared with famine episodes (see Sec. I*B*). This restricts the scope for sample selection bias through pandemic mortality. Moreover, to the extent that influenza mortality was selective, it is most plausible that subsequent cohort outcomes improved through

culling of the weakest.[3] For this reason, estimates of pandemic damage may underestimate the true effect.

The responsiveness of labor market outcomes to fetal health has significant implications for health economics and human capital research. First, the strong correlation between health and socioeconomic status may be driven by variation in fetal health. Second, investments targeting fetal health may have higher rates of return than more traditional investments, such as schooling. Public policies that improve fetal health may therefore have additional "multiplier" benefits that are not accounted for in conventional cost-benefit calculations.

The paper is organized as follows. Sections I*A* and I*B* review previous research on fetal origins. Section II summarizes research on health aftereffects (or "sequelae") of influenza infection and describes the influenza pandemic in the United States. Section III develops a simple conceptual framework for understanding fetal origins effects in the context of early-life mortality rates. Section IV describes the 1960–80 census micro data, as well as the vital statistics data available for the pandemic period. Section V presents results by birth cohort for all persons born in the United States and Section VI the within-cohort (i.e., fixed-effect) results identified by geographic variation in influenza exposures. Section VII discusses the robustness of the estimated effects, and Section VIII presents conclusions.

A. Literature Review: Economics

Evaluation of the fetal origins hypothesis has been hampered by the dearth of suitable natural experiments in countries with reliable long-term longitudinal data.

Economists who studied fetal origins were keenly aware of the strong cross-sectional association between adult health and educational attainment, employment, and wages. Case, Lubotsky, and Paxson (2002) documented that this gradient unfolds during childhood, at a time when pathways from parental income to health are arguably stronger than the reverse. Case, Fertig, and Paxson (2005) found that maternal smoking during pregnancy and low birth weight predicted poor academic performance and adult health in a British cohort born in 1958. These relationships persisted despite adjustment for differences in family socioeconomic status and health in childhood. However, in the absence of an explicit source of variation in fetal health, concern exists that measures of fetal health may be correlated with unobserved determinants of adult outcomes (e.g., genetic endowments), thereby biasing estimates. Several recent studies have sought to reduce the scope for

[3] See Mamelund (2006) for a recent survey and analysis.

such omitted variables bias by studying twin differences in early-life health, thereby holding constant both observed and unobserved differences across families (see Royer [2005] for California; Black, Devereux, and Salvanes [2005] for Norway; and Oreopoulos et al. [2006] for Canada). These studies found that twin differences in birth weight were positively associated with subsequent educational attainment.[4] Black et al. also found a positive effect of birth weight on IQ, whereas Oreopoulos et al. found limited cognitive effects.[5] Both the Black et al. and the Oreopoulos et al. studies found that adult labor market outcomes were positively associated with differences in twin birth weights.

Parental investments in human capital can bias estimates if they vary systematically with endowments among siblings (Becker and Tomes 1976, 157). Recent research suggests that such a bias may exist when twin endowment differences are used for identification. In the "first large-scale survey" of *child* twins, Rosenzweig and Zhang (2006) found that parental expenditures on schooling were reinforcing in China, that is, positively correlated with twin differences in birth weight.[6] In the absence of measures of parental investment, Royer (2005) and Black et al. (2005) stratified their samples by measures of socioeconomic status but did not find statistically significant differences in the effects of birth weight on adult outcomes.[7]

Finally, seasonal or cyclical differences in early-life conditions have been found to affect adult mortality. U.S. death rates by birth quarter exhibit seasonality consistent with fetal origins; poor nutritional intake and "maternal infection from respiratory disease during the winter months" may have compromised birth outcomes among those born in springtime (Costa and Lahey 2005, 491).[8] Dutch men and women born

[4] The effect of birth weight on high school completion by age 17 in Oreopoulos et al. (2006) was positive but not statistically significant within twin pairs.

[5] The administrative data Royer (2005) analyzed did not include cognitive measures or labor market outcomes.

[6] Postnatal investments that reinforce identifiable differences within a brood (i.e., twins and higher-order multiple births) can confer an evolutionary advantage: "If at the beginning of a season, the parents cannot predict whether it will be a good or a bad year, and if newborn offspring are relatively cheap, then it may pay to have as many offspring as could survive in the best year possible, then reduce the number of offspring to the actual resource levels encountered, either by neglecting some offspring, by letting the offspring fight with each other, or by directly killing or eating the weakest. . . . Unpredictable resource levels select for increased brood size and flexible brood reduction" (Stearns and Hoekstra 2000, 160). Such an advantage in natural selection may pertain to human evolution since "exposure to endemic famines has been the fate of the majority of humankind for most of history" (Clair et al. 2005, 561).

[7] Partitioning a select sample—the 2 percent of the population who are twins and for whom birth and adult administrative records could be matched—limits the statistical power of this approach (Black et al. 2005, 30).

[8] The "amplitude" in seasonality has decreased over time (Costa and Lahey 2005). Doblhammer and Vaupel (2001) noted a similar seasonality for Austria and Denmark and a "reversed" pattern for Australia.

during the nineteenth-century recessions died several years earlier than those born during economic expansions (van den Berg, Lindeboom, and Portrait 2006).

B. Literature Review: Epidemiology

The best epidemiological evidence on fetal origins comes from studies of famine episodes. Researchers have focused on three major famines in developed countries: the Finnish famine of 1866–68 and two famines in continental Europe during World War II. These studies have looked exclusively at health outcomes with particular interest in the timing and causes of adult mortality.

The Finnish famine killed some 8 percent of the population, but no long-term mortality effects were found for survivors in utero during the famine (Kannisto, Christensen, and Vaupel 1997). Similarly, the Nazis' seige of Leningrad caused a third of the city's 2.5 million residents to die, mostly of starvation, and survivors exhibited limited long-term effects (Stanner et al. 1997).[9] High mortality rates during these famines imply substantial scope for culling (Rasmussen 2001). Moreover, because both famines were several years long, selection into fertility during the famine may also confound the estimated effects.[10]

The Dutch famine, by contrast, was of comparatively short duration, in a country with one of the best vital statistics and population registrations then in existence (Stein et al. 1975). During this "unhappy cadenza" to German occupation of the Netherlands, the average daily ration fell below 800 calories from late November 1944 to April 1945, according to Stein et al. (p. 4). Nevertheless, famine-induced mortality was four times lower in the Netherlands than in either the Finnish or Leningrad famine, implying limited selective attrition.[11] Their initial follow-up study of cohorts in utero during the Dutch famine found effects for just one outcome—disorders of the central nervous system. Subsequent studies following these cohorts to middle age have documented various additional health impairments, among them self-reported health (Roseboom et al. 2001), coronary heart disease morbidity (Roseboom et al. 2000; Bleker et al. 2005), and adult antisocial personality disorders (Neugebauer, Hoek, and Susser 1999). The development of such chronic conditions in middle age (as opposed to

[9] Sparén et al. (2004) found that adolescents exposed to the Leningrad famine had higher rates of heart disease and stroke in 1975–99.

[10] In the Leningrad seige in particular, the cumulative effect of the famine made infertility "virtually total" (Stein et al. 1975).

[11] Roseboom et al. (2001) noted that mortality "more than doubled" in Amsterdam, one of the most affected cities. Using 2.5 as the factor by which mortality increased in the *whole* of the Netherlands and the baseline mortality rate of 0.87 percent reported from Stein et al. (1975, 40) would imply a 2.2 percent mortality rate due to the famine.

congenital manifestation or onset during childhood) is consistent with the predictions of Barker (1992).[12] Moreover these effects arise despite *positive* selection into fertility.[13] Long-term effects were most apparent for the cohort exposed to the famine early in gestation, although no decrease in the birth weights of this cohort was observed.[14]

II. Influenza Sequelae and the 1918 Influenza Pandemic

A. *Sequelae of Influenza Infection*

The idea that influenza infection might cause lingering damage is not a new one. In 400 BC, Hippocrates described ear complications that may have resulted from a putative influenza outbreak.[15] Following the 1918 pandemic, it was commonly speculated that influenza infection had increased rates of "sleeping sickness" (*encephalitis lethargica*) and parkinsonism (see Collier 1974; Ravenholt and Foege 1982), but more recent work using archival tissue samples has cast doubt on a link (McHall et al. 2001). Studies have looked at the adult mortality outcomes of those who were adolescents and young adults during the 1918 pandemic (see Azambuja 2004; Mamelund 2004). Conclusions from studies of postnatal influenza exposures, however, are generally subject to concerns regarding (i) the somewhat arbitrary designation of neighboring cohorts as treated and untreated and (ii) alternative hypotheses consistent with (broadly defined) cohort effects.[16]

The hypothesized effect of in utero influenza exposure offers more precise predictions for long-term effects and has precedent in the (albeit controversial) fetal origins hypothesis. The most commonly cited (and

[12] Barker reasoned that from an evolutionary perspective, onset past childbearing age would be favored.

[13] The share of children born to fathers in nonmanual occupations increased markedly in the winter of 1944–45 (Stein et al. 1975).

[14] The cohort exposed to famine in the third trimester had a lower average birth weight than other Dutch cohorts but also more limited long-term effects. The disconnect between famine-induced changes in birth weights and long-term effects is at odds with the predictions of Barker (1992).

[15] Jordan (1927) referred to the work of Hippocrates. Many 1918 survivors were "stone deaf for fully a year" (Collier 1974, 291).

[16] A notable exception is an ingenious 1934 study by Fritz Heider, who used the discontinuity in population health during the 1918 influenza pandemic to study the incidence of deafness. Heider collected enrollment data from 16 U.S. schools for the deaf and found a spike in the number of students born around September 1918. He concluded that the effect of influenza on hearing "occurred only with children who were less than four months old at the time of the epidemic" (1934, 757).

In addition to studies of morbidity effects, Brainerd and Siegler (2003) consider the effect of pandemic mortality, particularly mortality among those in prime working ages. They estimate macro models that indicate that economic growth (as measured by income per capita) was higher during the 1920s as a result of the negative shock to the labor force.

debated) pathophysiologic sequela of maternal influenza infection is schizophrenia.[17] Evidence supporting a link has been buttressed by a recent analysis of preserved serum samples drawn from pregnant women between 1959 and 1966 in Oakland, California, where influenza antibodies predicted schizophrenia in adult offspring (Brown et al. 2004), as well as controlled experiments with a human influenza strain adapted to mice (Shi et al. 2003). In addition to schizophrenia, Almond and Mazumder (2005) identified diabetes and stroke as health outcomes of the 1919 birth cohort that appear to respond to fetal exposure to the 1918 virus.

Taken as a whole, while an etiology underlying compromised adult health for birth cohorts exposed prenatally to the 1918 pandemic exists, it remains an active and controversial area of research.

B. Spread of Pandemic Influenza in the United States

The 1918 pandemic diffused nationwide in about one month (Sydenstricker 1918, 2311). By its end a few months later, the "Spanish flu" had killed more Americans than all combat deaths of the twentieth century (Crosby 1989, 207).

How the 1918 virus diffused within the United States is an "abiding puzzle" (Kolata 1999, 62). The states with the highest excess mortality rates—Pennsylvania, Montana, Maryland, and Colorado—"had little indeed in common economically or demographically, climatically or geographically" (Crosby 1989, 66). A study by economists concluded that mortality rates "appear to be randomly distributed and do not seem to be related to the level of economic development, climate or geography" (Brainerd and Siegler 2003, 11). Within states as well, the pandemic seemed to vary arbitrarily. For example, St. Paul's death rate was 70 percent higher than that in neighboring Minneapolis, and Dayton, Ohio's, death rate was 80 percent higher than that in Columbus (Huntington 1923, table 7). The National Research Council reported that population density, geographic location, minority share of the population, and so forth could not explain the geographic variation of the pandemic. It concluded that among the factors investigated, "only weather appears to have had any fundamental significance in causing the destructiveness of the epidemic to vary from city to city" (Huntington 1923, 27).

C. Maternal and Fetal Health

A distinguishing feature of the 1918 pandemic was the age profile of victims. While previous influenza outbreaks were most deadly for the

[17] Koenig, Kirkpatrick, and Lee (2002) summarized ecological studies on both sides.

relatively weak (the very old and the very young), the 1918 pandemic had its largest proportionate effect on those in the prime ages of 25–35. This resulted in an unusual "W-shaped" age distribution of influenza deaths, where the very young, those around age 30, and the elderly were most likely to die (Noymer and Garenne 2000).

While mortality from the 1918 virus was unprecedented, the vast majority of those infected survived. The U.S. Public Health Service surveyed 130,248 people shortly after the pandemic in 15 urban and rural communities and found that 28 percent reported being infected during the pandemic (Jordan 1927, 189). Pyle (1986, 52) refers to the "temporary flattening or indisposition and mandatory bedrest" of one-quarter of the U.S. population, with "repeated instances of lethargy" often following bouts of influenza (41).

Influenza was "especially prevalent among women of the child-bearing age" (Harris 1919, 978). Winn and Hobbins (2000, 281–82) noted that influenza outbreaks were associated with higher "morbidity and mortality in the pregnant patient than in the non-pregnant population." The "most vulnerable of all to influenza, those most likely to die, were pregnant women" (Barry 2004, 239).

Data from women in Maryland show that approximately one-third of women of childbearing age contracted influenza versus 28 percent of the general population (Jordan 1927, 202).[18] The large deterioration in maternal health during the pandemic led to a corresponding decline in fetal health. Appendix figure A1 shows the trend in average stillbirth rates by month during 1918. The regular trend is interrupted in October 1918, when stillbirth rates increased by 60 percent, or approximately 40 percent for October–December of 1918.

III. A Conceptual Framework for "Fetal Origins"

One hypothesized effect of the pandemic was to cause a negative shift in the (unobserved) distribution of maternal health among those infected. Another is that influenza infection of pregnant mothers caused the health of the cohort in utero to deteriorate as well. For example, the oxygen supply to the fetus may have been diminished by influenza or a secondary pneumonia infection. Such a shift in the unobserved distributions of maternal and infant health would also generate increases in mortality rates, in particular, the maternal and infant mortality rates.

This is not the only means by which mortality rates could increase. If medical care deteriorated so that for a fixed level of unobserved health

[18] Baltimore in particular was noted for having a representative experience with the 1918 pandemic (Sydenstricker 1918, 2308).

more people perished than prior to the pandemic, maternal and infant mortality rates would also increase.[19]

The means by which mortality rates increased during the pandemic has polar implications for the subsequent health of cohort survivors. If mortality rates increased because of reduced survival odds conditional on health, then fewer persons in "marginal" health survived the pandemic (culling). Observed later, pandemic survivors would have better average health ceteris paribus. If instead mortality rates increased because of a negative shift in the health distribution, the average health of survivors will depend in part on the permanence of the shift. Ecological studies using mortality rates as a proxy for fetal health implicitly assumed that (*a*) mortality rates were driven by distributional shifts and (*b*) a substantial portion of the distribution shift was permanent.

The tension between selective attrition and changes in the underlying health distribution can be considered more formally in a stylized latent variable model. Let h_i^* be the unobserved health of individual i, which is fixed from birth. Higher h_i^* implies better health. If h_i^* falls below a survival threshold d_0, then the individual will die prior to adulthood. Adults will be in poor physical health (henceforth, physically disabled) as adults if $d_0 < h_i^* \leq d_1$.

Given these health thresholds, the early-life mortality rate (for convenience, the infant mortality rate [IMR]) may be defined using the cumulative distribution function $F(h_i^*)$ as

$$\text{IMR} \equiv F(d_0). \tag{1}$$

The adult disability rate (ADR) is given by the share of persons surviving infancy that have initial health below d_1:

$$\text{ADR} \equiv \frac{F(d_1) - F(d_0)}{1 - F(d_0)}. \tag{2}$$

Deterioration in the probability distribution for health at birth (for

[19] The impotence of medicine in the face of the 1918 pandemic has been widely noted. For example, it was unknown that influenza was a virus until the 1930s. Brainerd and Siegler (2003) referred to the public health measures adopted as "completely ineffective." Insofar as maternal mortality is concerned, more mothers could have died in childbirth because physicians and nurses were unavailable while they cared for influenza victims or were sick with influenza themselves. However, the mixed success of medical care in assisting with childbirth is described by Thomasson and Treber (2005), who (*a*) note that maternal mortality rates were higher in urban areas than in rural areas throughout the 1915–40 interval and (*b*) find that the expansion of U.S. medical care between 1927 and 1937 (the earliest period analyzed by Thomasson and Treber) actually *increased* maternal mortality, albeit slightly.

convenience, a decrease in μ) generates increases in both the early-life mortality rate and the adult disability rate. In this case,

$$\text{signum}\left(\frac{\delta \text{ADR}}{\delta \mu}\right) = \text{signum}\left(\frac{\delta \text{IMR}}{\delta \mu}\right).$$

If instead early-life mortality rates rise because of rightward shifts in d_0 (for a fixed d_1), then

$$\text{signum}\left(\frac{\delta \text{ADR}}{\delta d_0}\right) \neq \text{signum}\left(\frac{\delta \text{IMR}}{\delta d_0}\right),$$

which will cause the expected value of health for adults at the follow-up period to increase when the infant mortality rate rises.

To the extent that early-life mortality during the 1918 pandemic occurred among those in the very weakest initial health, adult disability should decrease as a result.[20] Thus estimates of the effect of the distribution shift would be biased downward.

IV. Data

A. *1960–80 Census Micro Data*

U.S. Census micro data are suited to analysis of influenza birth cohorts because they identify the state of birth. This permits (*a*) restriction of the analysis sample to respondents born in the United States, thereby leveraging the discrete timing of the U.S. pandemic, and (*b*) analysis of differences in the severity of the pandemic within the United States. Moreover, large sample sizes permit comparisons of people born within a narrow birth interval yet with differing exposures to the pandemic.

Among postpandemic censuses, only the 1960, 1970, and 1980 data identify the quarter of birth. This enables analysis not only by birth quarter but also by birth year.[21] Year of birth is constructed as

$$\text{year of birth} = \begin{cases} \text{census year} - \text{age} - 1 & \text{if born April to December} \\ \text{census year} - \text{age} & \text{if born January to March.} \end{cases}$$

The Integrated Public Use Microdata Series (IPUMS) provides a 1 percent sample for 1960 (Ruggles et al. 2004). All persons born in the

[20] Neither evidence from contemporaneous sources nor the results of this analysis suggest that this threshold shift was the primary effect of the pandemic.

[21] Age in integers is reported as of March 31, which prevents identification of birth year from age alone. While the 1950 Census identifies the quarter of birth, it does so only for those under age 1. See Ruggles et al. (2004).

United States whose age was not allocated[22] are included in the cohorts analysis sample of Section V. Respondents for whom place of birth was allocated are also dropped for the analysis of geographic variation of the pandemic (Sec. VI) (IPUMS variables QAGE = 0 and QBPL ≠ 4). For 1970, Form 1 data are used since they contain disability measures, welfare payments, and so forth. The state, neighborhood, and metro samples are combined, yielding a 3 percent sample.[23] Some limited 1970 Form 2 data are also used, as elaborated below. The IPUMS provides its largest samples, 5 percent, beginning with the 1980 Census. Allocated records are dropped as above.[24]

The IPUMS variable DISABWRK combines responses from two mutually exclusive questions on the 1970 Census. The wording of the two work disability questions was changed for the 1980 questionnaire, making them no longer mutually exclusive.[25] However, IPUMS continued to treat the questions as exclusive, collapsing responses into DISABWRK again in 1980. The share of records with allocated DISABWRK values approximately doubled with the questions' rewording. For cohorts born around the pandemic (specifically, respondents aged 50–70 in the 1980 Census), more than a third of 1980 records with a work disability were allocated by IPUMS.

The Econometrics Laboratory at the University of California, Berkeley maintains a 5 percent extract of the 1980 Census micro data without the questionable IPUMS recoding (http://emlab.berkeley.edu/eml/emldata.shtml). Therefore, for the 1980 disability questions, Berkeley data are used instead of the recoded IPUMS data. This data substitution affects the results for work-limiting disabilities, but not the results for work-preventing disabilities.

[22] Allocation refers to the editing of census responses for (*a*) logical consistency; (*b*) hot-deck allocation—"searching the data file for a 'donor' record which shares key characteristics with the missing, illegible or inconsistent cases"; or (*c*) "randomly assigning a value from a pre-determined distribution, or assigning a modal value" (Ruggles et al. 2004).

[23] Not all states are sampled in the state and metro samples. Allocated records are dropped as in 1960.

[24] Additional data quality information is available for 1980 but is not used in order to make the case selection as comparable as possible with 1960 and 1970 data.

[25] Question 19a asked about a health condition that limited work that could be performed on "a job," and 19b asked about a disability that prevented work on "a job" (19c asked about a disability affecting use of public transportation). Those who answered yes to both 19a and 19b were apparently recoded with DISABWRK = 3, along with those who answered affirmatively to 19b alone. Therefore, respondents who claimed more than one disability that affected a given job in different ways or respondents with a single disability that limited work in some jobs but not others are obscured. IPUMS, when contacted on January 26, 2006, concurred that the 1980 work-disability questions were not mutually exclusive.

TABLE 1
MALE OUTCOME MEANS

	1960 Census		1970 Census		1980 Census	
	Born 1919	Surrounding Cohorts[a]	Born 1919	Surrounding Cohorts[a]	Born 1919	Surrounding Cohorts[a]
Age	40.3	40.3	50.2	50.2	60.2	60.2
	[1.08]	[.43]	[1.08]	[.43]	[1.08]	[.43]
High school graduate	.486	.509	.516	.537	.550	.564
	[.500]	[.500]	[.500]	[.499]	[.498]	[.496]
Years of education	10.6	10.7	10.8	11.0	11.0	11.2
	[3.6]	[3.5]	[3.5]	[3.5]	[3.7]	[3.7]
Total income	39,288	39,909	50,285	51,389	42,144	42,952
	[27,260]	[27,827]	[39,218]	[39,750]	[33,961]	[34,829]
Wage income	37,862	38,711	48,964	49,848	29,459	29,950
	[21,789]	[23,135]	[32,993]	[33,291]	[31,972]	[32,548]
Poor	.289	.283	.130	.124	.145	.141
	[.454]	[.450]	[.336]	[.330]	[.352]	[.348]
Neighbors' income[b]			51,359	52,310		
			[17,564]	[18,064]		
Socioeconomic status	35.0	35.6	37.3	38.1	34.5	35.0
	[24.0]	[24.3]	[24.4]	[24.5]	[26.3]	[26.4]
Disability limits work			.107	.102	.266	.256
			[.309]	[.302]	[.442]	[.436]
Disability prevents work			.048	.047	.166	.158
			[.214]	[.211]	[.372]	[.364]
Years of disability			1.4	1.3		
			[3.9]	[3.8]		
Social Security income			41	43	1,138	1,096
			[281]	[295]	[3,319]	[3,261]
Welfare income			91	82	177	162
			[930]	[850]	[1,282]	[1,206]
Observations	10,310	21,864	29,093	59,410	44,345	93,759

NOTE.—Standard errors are in brackets. All income figures are given in 2005 dollars.
[a] Born in 1918 or 1920.
[b] N = 9,587 and 19,897.

B. Census Means

Table 1 reports sample means for 12 socioeconomic measures among U.S.-born men. Birth years are divided by influenza exposure. Those born in 1918 were most likely born before the pandemic[26] and therefore were exposed as infants. Those born in 1920 were born after the pandemic and were not directly exposed. These two groups are collectively referred to as the "surrounding birth cohorts." The 1919 cohort contains those most likely to have been exposed in utero to the pandemic.

The average age in these two cohort groups is identical for each census year. Respondents born around the pandemic were slightly over 40 years old in 1960 and just over 60 at the end of the observation window in 1980. Approximately half completed high school, and mean years of schooling was approximately 11 years. Mean income peaked with the 1970 Census at just over $50,000 (2005 dollars), more than 95 percent of which came from wage income. The median neighbor's income,

[26] More than three-quarters of the 1918 cohort was born before October 1918.

available from the 1970 neighborhood sample, was slightly higher on average at approximately $52,000. The socioeconomic index, based on the occupation of the respondent, averaged slightly over 35. Disability measures are available for 1970 and 1980 only. Finally, mean Social Security and welfare payments for the 1918–20 birth cohorts are reported for 1970 and 1980. Disabled workers were eligible to receive Social Security payments prior to retirement through the Disability Insurance Program. Most of these payments, however, were recorded in the welfare field, together with Aid to Families with Dependent Children (AFDC) and General Assistance payments.[27]

Two patterns apparent in table 1 bear mention. First, educational measures improved across the census years. It is unlikely that continued education after age 40 accounts for this, but rather disproportionate mortality among those with less education (a pattern consistent with Lleras-Muney [2005]). Second, the surrounding cohorts presumably shared similar period effects (e.g., World War II) with the 1919 cohort since they were just one year "ahead" or "behind" in life. It is of note, therefore, that 27 of the 28 comparisons in table 1 reveal inferior outcomes for the 1919 birth cohort.

C. 1917–19 Vital Statistics Data

Influenza infection was not made a reportable disease in the United States until after the pandemic began. As a result, reliable data on regional differences in pandemic morbidity are unavailable. As the U.S. Public Health statistician noted, "Were there any necessity for demonstrating the utter inadequacy and lack of uniformity of morbidity reporting in the United States, such a demonstration would be fully available during the present epidemic" (Sydenstricker 1918, 2306).

The censuses of birth and death records are used to assess pandemic severity and are available at various levels of aggregation. In 1915, the federal government began collection of data on births in the United States. However, data from just 19 states are available for 1917–19. The best measure of fetal health available for this time period is the maternal mortality rate, defined as maternal deaths related to childbirth, divided by the number of births.[28] In 1917, 12,528 mothers died in childbirth (Department of Commerce 1919, 96), or 0.66 percent of live births (U.S. Public Health Service 1947).

[27] Payments for hospital or other medical care are not included. See the description of the "welfare (public assistance) income" field in Ruggles et al. (2004).

[28] Data on fetal deaths were published consistently beginning in 1922.

V. Outcome Differences by Birth Cohort

A ceteris paribus comparison of two birth cohorts is impossible. The linear dependence of period, age, and cohort implies that changing the cohort necessitates a change to either age or period (see Hall, Mairesse, and Turner [2005] for a recent survey and treatment). Furthermore, cohort effects tend to be smooth (as the figures below illustrate) and therefore especially difficult to disentangle empirically from other smooth effects (e.g., the life cycle of wages).

Two features of this study interact to help isolate the cohort effect. First, the pandemic was short. In the United States, 85 percent of pandemic mortality occurred in the four months from October 1918 to January 1919. Second, according to the fetal origins hypothesis, cohorts born before the pandemic (and therefore exposed postnatally) as well as cohorts born more than nine months after the pandemic ended should be relatively unaffected, thereby generating a discontinuous prediction for the cohort effect. As this section demonstrates, U.S. Census data bear out this prediction. Moreover, these effects are observed at different ages of the respondents (i.e., different census years), further reducing the likelihood that age or period effects account for the findings.

This section presents two types of evidence. First, the raw data are displayed in a series of figures, and various alternative hypotheses are discussed. Second, deviations of census outcomes from smooth cohort trends are estimated systematically for cohorts born between 1912 and 1922 as

$$y_i^c = \beta_0 + \beta_1 \cdot I(\text{YOB} = 1919) + \beta_2 \cdot \text{YOB} + \beta_3 \cdot \text{YOB}^2 + \epsilon_i, \quad (3)$$

where y_i^c denotes the census outcome for individual i in year c, $I(\cdot)$ denotes the indicator function, YOB denotes birth year, and β_1 measures the departure of outcomes for the 1919 birth cohort from the quadratic cohort trend. Estimates for β_1 are reported in tables 2, 3, and 4 for men, women, and nonwhites.

A. Educational Attainment

1. Results

Figure 3 plots the average schooling of men and women born in the United States by year of birth from the 1960 Census. The strong upward trend in educational attainment for these cohorts was driven by the high return to education, social capital assembled locally (Goldin and Katz 1999), and also more stringent compulsory schooling and child labor laws (see Lleras-Muney 2002). The 1919 birth cohort lies off this steady trend and received approximately one and a half months less schooling

TABLE 2
DEPARTURE OF 1919 MALE BIRTH COHORT OUTCOMES FROM 1912–22 TREND

	CENSUS YEAR		
OUTCOME	1960	1970	1980
High school graduate	−.021***	−.020***	−.014***
	[.005]	[.003]	[.003]
Years of education	−.150***	−.176***	−.117***
	[.038]	[.023]	[.019]
Total income	−573*	−1,236***	−1,065***
	[295]	[253]	[191]
Wage income	−812***	−875***	−688***
	[261]	[233]	[179]
Poor (below 150% of the poverty level)	.010**	.009***	.006***
	[.005]	[.002]	[.002]
Neighbors' income (N = 102,948)		−875***	
		[197]	
Socioeconomic status (Duncan's socioeconomic index)	−.640**	−.808***	−.816***
	[.259]	[.157]	[.137]
Disability limits work		.006***	.005**
		[.002]	[.002]
Disability prevents work		.004***	.001
		[.001]	[.002]
Years of disability		.092***	
		[.025]	
Social Security income		1	83***
		[2]	[19]
Welfare income		12**	17**
		[6]	[7]
Observations	114,031	308,785	471,803

NOTE.—Robust standard errors are in brackets. All income figures are given in 2005 dollars.
* Significant at 10 percent.
** Significant at 5 percent.
*** Significant at 1 percent.

than the cohort trend would predict. Since not all pregnant mothers contracted influenza prior to delivery, this deviation is accounted for by a larger treatment effect among the treated (if indeed influenza infection accounts for the departure from trend). With the estimated one-third infection rate among women of childbearing age, education falls approximately five months for those with infected mothers.

Figure 4 again plots educational attainment, using 1970 Census data and adding two pieces of information. First, the departure from trend exists for both men and women. Therefore, curtailment of education for military service is presumably not the culprit.[29] Second, a primary "node" of the education effect was whether high school was completed. The 1919 cohort was 4–5 percent less likely to complete high school than the cohort trend would predict (13–15 percent among the treated). The deterioration of educational attainment for men, women, and non-

[29] The 1925 birth cohort appears to register the brunt of the effect of military service during World War II on education attainment.

TABLE 3
1912–22 CENSUS OUTCOMES AMONG WOMEN (Census Years 1960, 1970, and 1980)

	SAMPLE MEAN			1919 COHORT DEPARTURE		
	1960	1970	1980	1960	1970	1980
High school graduate	.504 [.500]	.523 [.499]	.542 [.498]	−.028*** [.005]	−.021*** [.003]	−.015*** [.002]
Years of education	10.5 [3.0]	10.7 [3.0]	10.8 [3.2]	−.163*** [.032]	−.123*** [.019]	−.071*** [.015]
Total income	8,282 [12,718]	13,372 [18,138]	13,385 [16,952]	244* [135]	−139 [112]	−235** [93]
Wage income	15,682 [12,019]	21,212 [15,926]	6,911 [13,405]	50 [190]	−90 [138]	−62 [76]
Poor (below 150% of the poverty level)	.284 [.451]	.170 [.376]	.231 [.421]	.012** [.005]	.009*** [.002]	0 [.002]
Neighbors' income (N = 111,057)		51,007 [17,418]			−449** [189]	
Socioeconomic status (Duncan's socioeconomic index)	22.5 [24.2]	25.7 [25.1]	19.8 [25.1]	−.176 [.253]	−.461*** [.155]	−.470*** [.124]
Disability limits work		.066 [.247]	.240 [.427]		0 [.002]	.002 [.002]
Disability prevents work		.078 [.268]	.184 [.388]		.004** [.002]	.003 [.002]
Years of disability		1.2 [3.6]			.028 [.021]	
Social Security income		50 [299]	2,460 [3,583]		1 [2]	−50*** [13]
Welfare income		139 [1,087]	269 [1,360]		18** [7]	11* [7]
Observations	118,471	331,985	550,108	118,471	331,985	550,108

NOTE.—Robust standard errors are in brackets. All income figures are given in 2005 dollars.
* Significant at 10 percent.
** Significant at 5 percent.
*** Significant at 1 percent.

whites is significant at the 1 percent level (tables 2, 3, and 4, respectively). Point estimates are larger among nonwhites across census years. Furthermore, the lower levels of educational attainment among nonwhites imply that the estimated effects are twice as large for nonwhites.

Figure 5*a* plots average high school completion (measured in 1980) by the quarter of birth.[30] The upward trend in birth year/quarter is again apparent, along with seasonality (Angrist and Krueger 1991). Figure 5*b* plots the point estimates on the 20 quarters of birth from 1916 to 1920, taking out a linear trend and dummies for the four quarters of birth (estimated over the years 1912–23). Departure from trend for each of the first two quarters of 1919 is significant at the 5 percent level.

[30] The larger 1980 Census sample permits its use for the quarter of birth outcomes.

TABLE 4
1912–22 CENSUS OUTCOMES AMONG NONWHITES (Census Years 1960, 1970, and 1980)

	SAMPLE MEAN			1919 COHORT DEPARTURE[a]		
	1960	1970	1980	1960	1970	1980
High school graduate	.224	.246	.276	−.032***	−.026***	−.013***
	[.417]	[.431]	[.447]	[.010]	[.006]	[.005]
Years of education	8.1	8.5	8.5	−.241***	−.225***	−.116***
	[3.8]	[3.7]	[4.0]	[.086]	[.051]	[.043]
Total income	13,641	18,839	16,013	38	−441*	−574***
	[14,718]	[20,249]	[18,625]	[281]	[266]	[217]
Wage income	17,006	23,484	10,047	−127	−430	−500**
	[13,338]	[18,390]	[17,398]	[319]	[294]	[206]
Poor (below 150% of the poverty level)	.598	.398	.422	.024**	.01	.004
	[.490]	[.489]	[.494]	[.011]	[.007]	[.006]
Neighbors' income (N = 20,228)		0			175	
		[0]			[346]	
Socioeconomic status (Duncan's socioeconomic index)	15.7	18.4	15.3	−.415	−.866***	−.364
	[17.2]	[19.2]	[20.5]	[.384]	[.264]	[.224]
Disability limits work		.096	.358		−.003	.009**
		[.295]	[.479]		[.004]	[.005]
Disability prevents work		.120	.276		.009**	.009*
		[.325]	[.447]		[.004]	[.005]
Years of disability		1.7			.033	
		[4.2]			[.057]	
Social Security income		70	2,368		1	−2
		[356]	[3,842]		[5]	[35]
Welfare income		427	705		51*	20
		[1,937]	[2,220]		[29]	[26]
Observations	23,008	60,390	104,391	23,008	60,390	104,391

NOTE.—Robust standard errors are in brackets. All income figures are given in 2005 dollars.
[a] Regressions include a gender dummy.
* Significant at 10 percent.
** Significant at 5 percent.
*** Significant at 1 percent.

2. Discussion

The absence of effects for neighboring cohorts has implications for sample selection. If comparisons between the 1919 and 1918 birth cohorts were biased by higher early-life attrition among weaker infants born in 1918 and killed by influenza (i.e., an increase in the threshold d_0 of Sec. III), we would expect the surviving members of the 1918 birth cohort to have higher educational attainment than predicted by the cohort trend. This is not apparent (see figs. 3, 4, and 5).[31] Those born in the first half of 1919 were conceived before the pandemic arrived,

[31] Similarly, if the 1920 birth cohort had better subsequent outcomes because the set of potential parents had been culled by the influenza pandemic, outcomes for the 1920 birth cohort should also deviate from the age trend, which is not observed. The potential biases from selective attrition are explored in greater detail in Almond (2005, sec. 9).

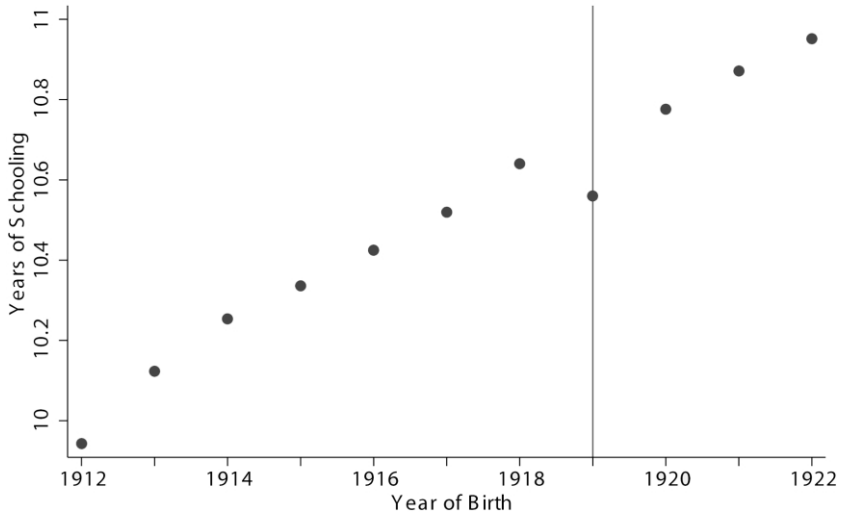

FIG. 3.—1960 average years of schooling: men and women born in the United States

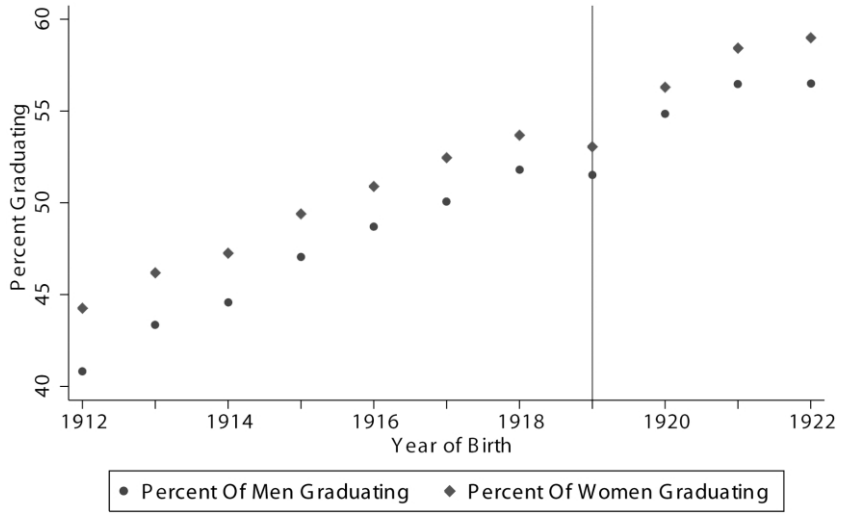

FIG. 4.—1970 high school graduation: by year of birth

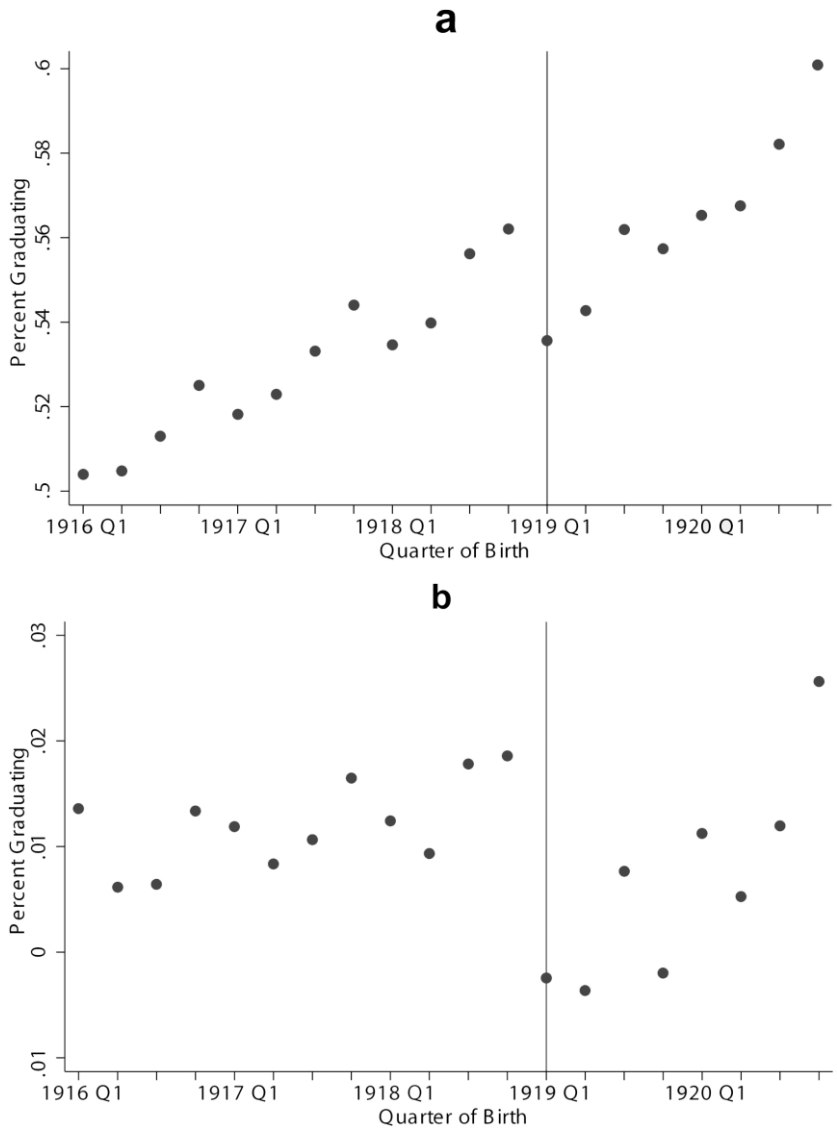

FIG. 5.—*a*, 1980 high school graduation rate by quarter of birth. *b*, Regression-adjusted 1980 high school graduation rate by quarter of birth.

were in utero during its peak, and were born after it had departed. Therefore, (negative) selection into childbearing after the arrival of the pandemic cannot account for the departure of the 1919 birth year outcomes from trend.

Instead, selection into pregnancy would have had to change radically approximately six months before the pandemic and then abruptly revert following the pandemic to account for the observed patterns. Unfortunately, there are few census measures determined prior to fetal health (and therefore not possibly affected by it). An exception is nativity of parents, which is strongly correlated with the income, educational attainment, socioeconomic status, and disability status of one's child. If selection indeed operated to change the background characteristics of the 1919 birth cohort, we might expect the share with parents born abroad (one-quarter of the U.S.-born analysis sample) to change as well. This is not observed. The *t*-statistic for the 1919 cohort dummy when we control for a quadratic trend as in table 2 is .20. Similarly in 1970, no departure from trend is observed in the annual or quarterly plots of the share of respondents with at least one foreign-born parent.[32]

The education results, nevertheless, do suggest some sample selection. As noted in Section IV*B*, average educational attainment increased between 1960 and 1980 for these (fixed) birth cohorts. While estimates of β_1 are similar for the 1960 and 1970 Censuses, they are approximately 25 percent smaller in 1980 for both men and women (tables 2 and 3). The weakening of the effect suggests selective mortality among those with large reductions in education due to the pandemic. The effect is more pronounced among nonwhites, further suggesting that blacks may have died substantially earlier (in their 50s) as a result of the pandemic.

Finally, the proximate cause of reductions in educational attainment was presumably decisions made during teenage years. It should be noted that effects at such young ages are not predicted by a narrow interpretation of the fetal origins hypothesis, which expects effects to manifest after primary reproductive ages. The timing and nature of the education results are more consistent with the "life course" models of health (e.g., Kuh and Wadsworth 1993). While effects for outcomes in which proximate causes likely operate at older ages are also observed (see below), such fetal origins effects are presumably amplified by educational choices.

[32] Data are taken from Form 2 of the 1970 U.S. Census. Income and education outcomes from Form 2 display the same 1919 cohort discontinuity for education and income measures as the Form 1 data. Nativity of parents was not queried in the 1980 Census.

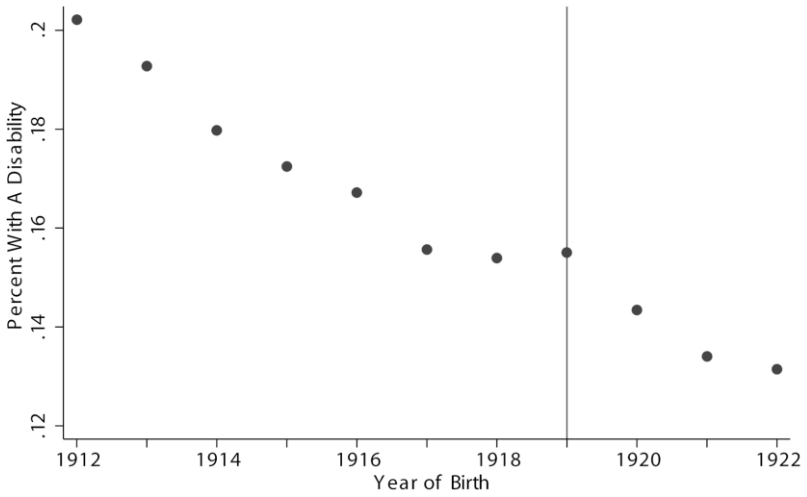

FIG. 6.—1970 male disability rate: physical disability limits or prevents work

B. Wages and Disability

Annual wage income is $700–$900 lower (2005 dollars) for men born in 1919 across the three censuses, or approximately $2,500 (5–9 percent) lower for the sons of infected mothers (tables 1 and 2). Part of the income reduction is presumably attributable to the reduction in education noted above. If it is assumed that the return to schooling for the 1919 birth cohort is comparable to that found in the literature, the decrease in wages can be decomposed into that caused by shortened schooling versus other impacts of poor fetal health. The gross effect associated with decreasing schooling one year is to reduce wage income from 10 percent to 25 percent. Therefore, as much as half of the total wage effect is apparently due to noneducation factors.

Disability status, first queried in the 1970 Census, appears to contribute to the wage effect.[33] Figure 6 plots the share of cohorts reporting a physical disability that limited or prevented work in 1970.[34] A discontinuous increase is readily apparent for the 1919 birth cohort. Men are 6 percent more likely to have a work-limiting disability and 8 percent

[33] Those with no wages are dropped, so work-preventing disabilities per se cannot account for this effect.

[34] Only disabilities lasting six months or longer are considered, so as to be consistent with wording on the 1980 Census questionnaire.

FIG. 7.—1980 male disability rate: physical disability limits work

more likely to have a work-preventing disability if born in 1919 (17 percent and 25 percent among the infected, respectively). Years of disability also increase by a similar magnitude (see tables 2 and 3). Smaller disability effects are found among women and nonwhites than men.[35]

Figure 7 plots the unadjusted Berkeley census data for the 1980 "limiting disability" question. (Records in which age is allocated are dropped, as above.) As with figures 2 and 6, it is apparent that the smooth age trend is interrupted for the cohort in utero during the pandemic's peak.[36] With this disability measure, it appears that the last quarter of birth of 1918 may also be affected, suggesting that third-trimester exposure may be more important for this milder health outcome.

C. *Entitlement Payments and Incarceration*

Disabled workers are eligible to receive Social Security payments prior to retirement through the Disability Insurance Program. Such payments (along with AFDC) are recorded as welfare payments (Ruggles et al.

[35] However, estimates for work-preventing disabilities are more similar across these groups (see tables 1–4).

[36] Disability rates before this interval appear to depart from the cohort trend in late 1916 and early 1917. This pattern is of (separate) interest because it occurs around the Social Security "notch," i.e., cohorts born in the fourth quarter of 1916 vs. those born in the first quarter of 1917. For a detailed description, see Snyder and Evans (forthcoming).

FIG. 8.—Average welfare payments for women and nonwhites: by year of birth

2004). In 1980, men born in 1919 received 8 percent more in welfare (tables 1 and 2).[37]

The discontinuity in welfare payments for the 1919 birth cohort is especially evident among women and nonwhites: perhaps unsurprising given that these are groups for whom negative shocks are likely to result in a higher incidence of welfare receipt. Average payments to women and nonwhites in 1970 are plotted in figure 8. The average welfare payment was 12 percent higher for both women and nonwhites born in 1919, or approximately one-third higher for children of mothers who contracted influenza.[38] When we focus on quarter of birth, it is apparent that these increased payments are generated by high payments to those born between April and June of 1919. As noted in Section I, those born in this interval had the highest average welfare payments of *any* quarter of birth from April 1911 to April 1925.[39]

Finally, decreased schooling and wages may have lowered the opportunity cost of illegal activities for cohorts exposed prenatally to influenza.

[37] For 1980 income measures only, the cohort trend is estimated as a cubic function to attempt to account for retirement at age 62.

[38] Participation also increases from 1918 to 1919, but the the change is primarily on the intensive margin. While not apparent for other outcomes, that 1920 also appears above trend is noteworthy since the 1918 virus made a brief reappearance in January 1920.

[39] It is apparent from the quarterly plots that Social Security income (SSI) payments were modestly higher for men born in early 1919 in the 1980 data (and not yet categorically eligible for SSI at age 62). Men of the 1919 cohort received approximately 10 percent more disability insurance than a cubic trend would predict (table 2). This suggests that some disability insurance payments may have been recorded in the SSI field.

Incarcerated respondents can be identified in the census using the respondents' relationship to the household head (IPUMS variable RELATED = 1301 [Inmate]). The 1940 Census captures pandemic cohorts near their peak crime ages of 18–24 (Donohue and Levitt 2001, 382).[40] Respondents born in 1919 are significantly more likely to be in jail in 1940 than surrounding cohorts.[41]

VI. Outcome Differences by State of Birth

The brevity of the pandemic, which assists identification at the national level, circumscribes regional variation in pandemic timing. As noted above, the pandemic diffused nationwide in about a month. Because the census does not report month of birth, these data are not suited for using variation in pandemic timing per se (if we leave aside the question of statistical power). Nevertheless, this limited variation is useful when combined with an additional observation: the pandemic reportedly became less virulent as it unfolded. This section therefore begins by evaluating whether respondents born in "laggard" states exhibited less pronounced long-term effects than respondents born in areas in which the pandemic arrived earlier (Sec. VI*A*) and then utilizing differences in the virulence of the virus directly (Sec. VI*B*). The poor quality and coverage of data on pandemic onset dates (and how these dates correspond to virulence) restrict how far this analysis can be pursued.

This section's primary identification approach is to utilize geographic variation: differences in the severity of the pandemic as measured by mortality. As noted in Section II*B*, this variation was surprisingly idiosyncratic. Section VI*C*1 describes the suitability of maternal mortality as a measure of pandemic severity and resultant fetal damage. Section VI*C*2 describes how infection rates may be inferred from maternal mortality rates, and Section VI*C*3 incorporates the infection measure into an econometric framework. Sections VI*C*4 and VI*C*5 then present the results.

A. Pandemic Timing by State

In the midst of the pandemic, the U.S. Public Health Service collected morbidity and mortality information from 376 U.S. localities. The data were summarized in a map of the initial diffusion of the fall pandemic

[40] The 1940 Census does not report quarter of birth. Therefore, year of birth is correct for approximately 75 percent of respondents; see Sec. IV*A*.

[41] Significant at the 10 percent level for men, and significant at the 5 percent level for men and women when eq. (3) is estimated.

(Sydenstricker 1918).[42] This map identified the seven states in which the pandemic arrived last. These states were all in the western United States: Oregon, Idaho, Montana, North Dakota, South Dakota, Nevada, and Wyoming.

The reprieve enjoyed by these states was short. In early September 1918, the pandemic flared in isolated locations, spreading across most states in mid-September. The laggard states were affected shortly after September 28. While variation in the date of arrival of the fall pandemic itself is not sufficient for long-term analysis, the pandemic reportedly lost force as it progressed.[43]

A prediction is therefore that persons born in these seven states experienced a milder form of the virus and therefore muted long-term effects. The fact that only 3 percent of U.S. births at the time occurred in these states makes rejection of the null hypothesis (of identical damage) difficult. A consistent pattern nevertheless emerges. When a dummy for birth in one of the seven laggard states (and its interaction with a dummy for birth in 1919) is included in the estimation of cohort effects (eq. [3]), these states generally experienced more mild long-term effects than the other 43 states (according to the signs on the point estimates). Differences for the laggard states are statistically significant for four outcomes: poverty status in 1960, disability status in 1970 (preventing work), average wage income in 1980, and average SSI in 1980. In each case, respondents born in the seven states exhibit less damage than those born in 1919 in other U.S. states.[44]

While these results are encouraging, one would like to use information on timing by state in a more systematic (and continuous) fashion. Unfortunately, the data underlying the map are no longer available. As noted by Pyle (1986, 43), "these data were apparently either not sent to the National Archives . . . or they were not retained in those files over the years."

B. Pandemic Timing and Virulence by Census Division

While the state-level morbidity data have been lost, Sydenstricker (1918) reported pandemic timing (the number of localities reaching "epidemic

[42] This map has been reproduced in a number of secondary sources, including Pyle (1986, 42) and Crosby (1989, 65).

[43] Sydenstricker (1918, 2319) noted a "definite tendency for the disease to become milder as the epidemic spread." Unfortunately, the last states the pandemic struck have no mortality data for this period, and therefore this pattern is difficult to confirm. Among the states with mortality data, the pandemic appeared nearly simultaneously.

[44] Finer divisions of the pandemic's arrival do not provide any additional traction. In particular, no pattern emerges when the 10 "middle" states that first experienced the pandemic in the week of September 21–28 (Arizona, Florida, Michigan, Missouri, Mississippi, Nebraska, New Mexico, Oklahoma, Washington, and West Virginia) are compared with states that experienced the pandemic before September 21 or after September 28.

stage" by week) for the nine census divisions. The compressed timing of the pandemic is again apparent. The New England states were the first affected, peaking on average the week of September 21. The Mountain and Pacific regions were last, peaking two and a half weeks later. This timing variation alone is not sufficient to identify long-term effects. Crosby (1989, 64) noted that although the "disease tended to become less lethal with the passage of time . . . the decline was too slow for a week or two or three to make much difference." And indeed, no significant differences in long-term effects are found when these timing differences by division are used.

Fortunately, Sydenstricker (1918, table 8) also recorded the strength of the influenza virus by week. Virus strength was assessed as the tendency for influenza infection to develop into pneumonia.[45] This information was reported for Baltimore because that city was "believed to be representative of various sections of the country" (2308). The canvass indicated that the virus *gained* strength between mid- and late September and then weakened substantially in mid-October of 1918.[46]

The data on virus strength by week from Baltimore can be combined with the data on epidemic timing by census division to yield a measure of average pandemic virulence by division.[47] Accordingly, the New England and the East South Central states (Alabama, Kentucky, Mississippi, and Tennessee) experienced the most virulent stages of the pandemic, and the Mountain and Pacific states experienced the mildest.

The effect of virulence is estimated by expanding (3) to include the virulence measure (virulence_d, applied to those born in 1919), along with main effects for each census division (λ_d):

$$y_i^c = \beta_0 + \beta_1 \cdot I(\text{YOB} = 1919) + \beta_2 \cdot \text{virulence}_d + \beta_3 \cdot \text{YOB} + \beta_4 \cdot \text{YOB}^2 + \lambda_d + \epsilon_i. \quad (4)$$

Estimates of β_2 reflect the effect of exposure to a more virulent stage of the pandemic in one's division of birth. In the 1 percent sample of 1960, $\hat{\beta}_2$ generally has the predicted sign but is imprecisely estimated and is significant for just one outcome (total income, for which $\hat{\beta}_2$ has a perverse sign).[48] The 1970 results offer more consistent support. Respondents from virulent areas were more likely to be in poverty and live

[45] In particular, the share of reported pneumonia cases, divided by the sum of pneumonia, influenza, and cases causing "disability of less than three days" (Sydenstricker 1918, 2319).

[46] The initial increase in the virulence measure may have been an artifact of improved reporting early in the pandemic.

[47] As the distribution of epidemic timing within each division is skewed and has different variances (Sydenstricker 1918, table 1), the distribution of dates by division (rather than the average) is applied to the virulence data.

[48] Standard errors are clustered at the division level for the 1919 birth cohort.

in neighborhoods with lower income (significant at the 1 percent and 10 percent levels, respectively). They were more likely to have a work-limiting or a work-preventing disability and to be disabled for more years (significant at the 10 percent, 1 percent, and 1 percent levels, respectively). For 1980, $\hat{\beta}_2$ is imprecisely estimated for nearly all outcomes, except wage income, and $\hat{\beta}_2$ has the expected sign (significant at the 5 percent level).

In sum, results using differences in pandemic timing and virulence corroborate the findings of Section V. However, the coverage and quality of data on timing and virulence prevent more definitive conclusions. These data issues led Sydenstricker (1918, 2306) to conclude that "we must look to mortality reports for our main statistics of the epidemic." Therefore, this section turns to the final identification strategy based on geographic differences in pandemic mortality.

C. Pandemic Intensity

1. Why Maternal Mortality?

Despite the similarity across states in pandemic timing, state death rates were markedly different. Maternal mortality by year and state constitutes the best available measure of how geographic differences in the pandemic affected fetal health. The suitability of this measure is detailed below.

First, the W-shaped age profile of influenza victims implies that state mortality rates were a function of the age distribution of the population. Differences in the age distribution across states were large. For example, the share of the population over 60 was more than three times larger in Vermont than in Washington State (relative to the population aged 20–29). For assessing fetal origins effects, measures of pandemic intensity among those of childbearing age are relevant. Maternal mortality captures this aspect of pandemic intensity.

Second, Noymer and Garenne (2000) noted that males were substantially more likely to die in the influenza pandemic because of higher tuberculosis morbidity. In order not to be confounded by excess male tuberculosis prevalence across states (and its attendant risk factors), mortality data for young women are required, which maternal mortality clearly provides. Third, putative puerperal death did not have as close cause of death substitutes as the other major pathological causes of death during the pandemic (e.g., influenza, tuberculosis, and pneumonia).

Finally, maternal mortality has previously been used as a proxy for the intrauterine health environment: Barker and Osmond (1987) found a strong relationship between local maternal mortality rates for 1911–

14 in Britain and subsequent stroke mortality of offspring. They speculated that maternal hypertension may have generated increases in both maternal deaths and stroke incidence in progeny. This finding was echoed in the United States, where cohorts in utero during the 1918 pandemic had higher rates of stroke morbidity in their 70s and 80s (Almond and Mazumder 2005).[49]

Use of maternal mortality rates restricts analysis to the roughly 50 percent of the (U.S.-born) population that was born in the 19 birth registration states. While ideally coverage would be complete, this restriction is somewhat appealing because of the relative homogeneity of the registration states. The 19 states tend to be in the northeastern United States, relatively wealthy, and predominantly white. Only 5 percent of those born in these states were nonwhite versus 15 percent in the other 31 states.[50]

The average maternal mortality rates increased from 0.66 percent in 1917 to 0.92 percent in the birth registration area.[51] As this increase was accounted for by births in the last quarter of 1918, the maternal mortality rate roughly doubled in the fall of 1918. Variation in this increase across states, like the geographic variation of the pandemic more generally, appears idiosyncratic. For example, maternal mortality rates increased by nearly identical amounts in urban and rural areas.

2. Relationship between Maternal Mortality Rates and Maternal Influenza Infection

As influenza infection was not made a reportable disease until after the pandemic began, this (direct) measure cannot be used to analyze geographic variation in the prevalence of the pandemic. This subsection explores how state-level maternal infection rates might be extracted from data available on maternal mortality rates.[52] It is shown that nonlethal influenza infection does not bear an obvious relationship to mor-

[49] The possibility that a maternal stress response might underlie physiological damage to the fetus (and not nutritional deprivation or influenza infection per se) has received support from laboratory experiments, which countered the "fetal origins" effects from undernutrition with hormonal treatments administered during pregnancy (see Langley-Evans 2001; Couzin 2002). Moreover, Stein et al. (1975) reasoned that undernutrition alone might not account for their findings from the Dutch famine, hypothesizing that the interaction of famine with an (unspecified) maternal infection prevalent in the Netherlands at the time may have caused the effects they observed (p. 230).

[50] The long-term damage estimated for these 19 states is somewhat smaller than in the nonregistration states (see Sec. VII).

[51] See Almond (2005, table 1, fig. 2) for maternal mortality rates in the 19 birth registration states by year (Source: U.S. Public Health Service 1947).

[52] In Almond (2005), maternal mortality rates were used directly as a proxy for the effect of maternal influenza infection on fetal development. Increased maternal mortality was associated with negative census outcomes, consistent with a decrease in μ in the Sec. III framework.

tality rates. In particular, while influenza infection is linear in pandemic mortality, it is nonlinear in nonpandemic mortality. This nonlinearity is important empirically since baseline maternal mortality composed the majority of maternal deaths during the pandemic.

Analogous to the conceptual framework in Section III, puerperal death is assumed to occur when (unobserved) maternal health falls below a threshold, d_0:

$$\text{MMR}_s^{1917} = \frac{\int_{-\infty}^{d_0} \phi(\mu_s^{1917},\ h)dh}{\int_{-\infty}^{\infty} \phi(\mu_s^{1917},\ h)dh} = \int_{-\infty}^{d_0} \phi(\mu_s^{1917},\ h)dh, \tag{5}$$

where MMR_s^{1917} is the 1917 maternal mortality rate in state s, h represents the (unobserved) health index, $\phi(\cdot)$ represents the probability density function for maternal health, and μ_s^{1917} is mean maternal health in state s prior to the pandemic.

The 1918 maternal mortality rate is a weighted average of the 1917 maternal mortality rate and maternal mortality rate conditional on infection (which is assumed to be constant across states[53]), where the weights are the influenza infection rates among mothers in state s, ψ_s:

$$\text{MMR}_s^{1918} = \psi_s \int_{-\infty}^{d_0} \phi(\mu^{\text{pandemic}},\ h)dh + (1 - \psi_s) \int_{-\infty}^{d_0} \phi(\mu_s^{1917},\ h)dh, \tag{6}$$

where it is assumed that $\mu_s > \mu^{\text{pandemic}}$ for all s. When we denote maternal mortality conditional on infection as κ and rearrange terms,

$$\psi_s^{1918} = \frac{\text{MMR}_s^{1918} - \text{MMR}_s^{1917}}{\kappa - \text{MMR}_s^{1917}}. \tag{7}$$

Analogously,[54]

$$\psi_s^{1919} = \frac{\text{MMR}_s^{1919} - \text{MMR}_s^{1917}}{\kappa - \text{MMR}_s^{1917}}. \tag{8}$$

As we know the pandemic began in 1918,

$$\psi_s^{1917} = 0. \tag{9}$$

[53] See n. 19 concerning the impotence of medical treatment vis-à-vis influenza, which was effectively reduced to comfort measures (Crosby 1989).

While it would be more realistic to assume that the difference $\mu_s^{1917} - \mu_s^{\text{pandemic}} = \Delta\mu^{\text{pandemic}}$ was constant across states, implementation of this assumption would require knowledge of $\phi(\cdot)$. The simplifying assumption of a common health distribution among infected mothers is arguable in view of the fact that the average increase in the maternal mortality rate was three times larger than the 1917 standard deviation in state maternal mortality rates.

[54] Two states have negative $\hat{\psi}_s^{1919}$ values. Setting $\hat{\psi}_s^{1919}$ to zero for these states does not substantially affect these results, reported in table 5 below.

3. Estimation

First, I assume no direct effect of maternal death on an infant. While this assumption is obviously false, maternal death was exceedingly rare relative to maternal influenza infection: more than 30 times as many pregnant mothers survived influenza infection as died.[55] Adult outcomes in the U.S. Census, y_i, can be estimated as a function of maternal influenza infection:

$$y_i = \beta_0 + \beta_1 \psi_s^{\text{yob}-1} + \gamma^{\text{yob}} + \lambda_s + \epsilon_i, \tag{10}$$

where s denotes the state of birth, and yob $-$ 1 denotes the year prior to the birth year. The year preceding the birth year is used in order to capture the in utero health conditions (to the extent possible with annual data). Year of birth fixed effects, γ^{yob}, remove the average outcome differences related to birth year, and λ_s accounts for the effect of fixed state-level factors. Identification of $\hat{\psi}_s$ comes from differential changes in state infection rates around the pandemic average (one-third) immediately before and after the pandemic's peak.

As attrition may occur between birth and follow-up in the census (which may be correlated with ψ_s), two attrition measures are added to (10) yielding

$$y_i = \beta_0 + \beta_1 \psi_s^{\text{yob}-1} + \gamma^{\text{yob}} + \lambda_s + \beta_2 \text{IMR}_s^{\text{yob}} + \beta_3 \text{attrition}_s^{\text{qob}} + \Lambda_{\text{sor}} + \epsilon_i, \tag{11}$$

where $\text{IMR}_s^{\text{yob}}$ is the infant mortality rate in the year and state of birth, and $\text{attrition}_s^{\text{qob}}$ represents the share of the birth cohort that does not appear in the census and is constructed at the quarter level.[56]

4. 1960 Census Results

In addition to identifying fetal origins effects using a different aspect of the 1918 pandemic, results from (10) and (11) permit separation of the pandemic exposure, ψ, from attrition, primarily through mortality, though emigration would also be registered by cohort attrition. Table

[55] Moreover, to the extent that maternal death had a socioeconomic status gradient, mothers with a lower socioeconomic status were more likely to die, implying that subsequent cohort outcomes would be better. That is, the "mechanical" effect of maternal death would tend to bias β_1 in (10) downward. Moreover, the magnitude of the bias is likely to be small. If the annual maternal mortality rate at the pandemic's peak is doubled, at 2 percent of births, mortality itself could explain only a 2 percent difference in outcomes under the most extreme assumptions on the counterfactual outcomes of the stricken birth cohort. See Almond (2005) for more discussion.

[56] The published mortality micro data begin in 1968 but do not include information on state of birth (National Centers for Health Statistics 1980).

TABLE 5
MEN BORN 1918–20: 1960 STATE OF BIRTH FIXED-EFFECTS RESULTS

	INDEPENDENT VARIABLE			
DEPENDENT VARIABLE	Maternal Infection (ψ)	Infant Mortality Rate	Attrition	State of Residence Dummies
Years of education	−.756***			No
	[.259]			
	−.793***	−.0059*		No
	[.229]	[.003]		
	−.818***	−.0553*	−.426	No
	[.228]	[.0308]	[.318]	
	−.759***	−.0329	−.392	Yes
	[.233]	[.0313]	[.314]	
High school graduate	−.101***			No
	[.070]			
	−.103***	−.0003*		No
	[.0355]	[.00005]		
	−.109***	−.0026	−.091*	No
	[.039]	[.0051]	[.050]	
	−.105***	−.00046	−.0908*	Yes
	[.0378]	[.0052]	[.0499]	
Log income	−.165***			No
	[.0719]			
	−.176***	−.0018**		No
	[.060]	[.00086]		
	−.172***	−.0181*	.0629	No
	[.062]	[.0086]	[.0557]	
	−.166***	−.0139	.0707	Yes
	[.0623]	[.0085]	[.0599]	
Poverty status (below 150% of poverty level)	.0424			No
	[.0259]			
	.0461**	.00059		No
	[.224]	[.00040]		
	.0429*	.0064*	−.0529	No
	[.0233]	[.0039]	[.0398]	
	.0386	.0041	−.0533	Yes
	[.0255]	[.0040]	[.0397]	
Socioeconomic status (Duncan's socioeconomic index)	−2.711			No
	[1.735]			
	−2.806*	−.0150		No
	[1.635]	[.03057		
	−2.863*	−.142	−.9441	No
	[1.665]	[.307]	[2.372]	
	−2.721	−.023	−1.075	Yes
	[1.764]	[.298]	[2.361]	
Observations	16,566	16,566	16,566	

NOTE.—Robust standard errors are in brackets. All income figures are given in 2005 dollars.
* Significant at 10 percent.
** Significant at 5 percent.
*** Significant at 1 percent.

5 summarizes the within-cohort results. Estimates for ψ from equation (10) are reported in the first row (for each outcome variable).

The term ψ enters in the expected direction for each outcome. When one-third is used as the average maternal infection rate for the 1919 cohort, infection is estimated to reduce schooling by $.756 \times \frac{1}{3}$ year, or 0.25 year. Similarly, the likelihood of graduating from high school is estimated to fall approximately 0.03 percentage points for the 1919 cohort. Income is estimated to fall approximately 6 percent, and poverty increases approximately 1.5 percentage points. Finally, socioeconomic status falls nearly one index point for the 1919 cohort.

Inclusion of additional controls tends to strengthen the estimated impact of ψ, albeit slightly. Infant mortality enters in the same direction as ψ, suggesting that changes in infant mortality rates are again dominated by μ rather than d_0 shifts. Again, the implied μ shift for infants appears permanent; that is, health during infancy exerts persistent effects on adult outcomes. Estimates for the attrition coefficient, when significant, have the same sign as the preceding coefficients. This is somewhat surprising since the direct effect of selection (presumably among the least healthy) should mitigate impacts. However, if selection is at low levels, attrition can also be considered a fetal origins outcome: adult mortality. Thus attrition may be responding to the (otherwise unmeasured) aspects of decreased μ caused by the pandemic and therefore responding in the same direction as ψ.

Results from table 5 may be compared with the 1960 results in table 2. In general, the effects implied by estimation of (10) and (11) are approximately 50 percent larger than estimates obtained from the cohort differences approach of Section V. These differences, however, are not significant with one exception: the estimated effect of influenza infection on income obtained from (11) is nearly four times as large as the corresponding estimate from table 2.

5. 1970 and 1980 Census Results

Results with the infection measure developed below are qualitatively similar for 1960 and 1980. In general, the 1980 estimates are about half as large as the corresponding 1960 effects. As the table 2 estimates "hold up" more in 1980, the net result is that the two estimation approaches generate quite similar estimates for 1980.

Results appear different for 1970, partly as a result of the different geographic sampling in the 1970 Census. The neighborhood and metro Form 1 samples were not geographically representative; many states were not sampled and therefore have zero respondents. This would particularly affect estimation in this section, since it relies on the state of birth differences in infection rates. The Form 1 and Form 2 state samples

(IPUMS DATANUM = 1, 2) can be combined to yield a 2 percent sample for 1970 that is geographically representative. When equation (10) is estimated for the outcomes reported on both Forms 1 and 2 (income, educational attainment, socioeconomic status, etc.), estimates for ψ do not approach statistical significance.

VII. Robustness

A. *Difference by Birth Cohort*

In order to bias estimates of the pandemic's long-term effect from Section V, an omitted factor would have to shift discontinuously for the 1919 birth cohort. As mentioned above, the share of parents born abroad (a predetermined census measure that correlates with socioeconomic outcomes) does not move discontinuously for the 1919 birth cohort. This section begins by incorporating the effect of three additional factors into equation (3) that could conceivably account for the estimates of β_1 reported in tables 2–4.

First, the 1919 birth cohort may have been affected differently by World War II than other cohorts. For example, greater health exemptions may have deprived the 1919 birth cohort of the opportunities of military service (e.g., the GI Bill). Second, Costa and Lahey (2005) noted the importance of birth quarter on adult mortality rates. If pandemic mortality or subsequent cohort attrition operated so as to favor inferior birth quarters for the 1919 cohort, then inclusion of birth quarters should absorb this seasonal effect. Finally, if an inferior "mix" of birth states comprises the 1919 birth cohort, then state of birth fixed effects could account for the deviation of the 1919 cohort outcomes from trend. To allow these possible mechanisms, dummy variables for (i) whether the respondent served in World War II, (ii) each state of birth, and (iii) the four possible quarters of birth are included in equation (3). Estimates of β_1 change modestly, both increasing and decreasing with the additional covariates (results available from the author on request).

As noted in Section VI*A*, certain western states appeared to have received a milder form of the pandemic and display commensurately mild long-term effects. It can be further explored whether the estimated effects vary across the four census regions of birth. For example, we might be concerned to find that the Northeast accounted for all the long-term effects or, alternatively, displays non-long-term damage, given the severity of the pandemic in Philadelphia and the role of Massachusetts in the fall outbreak. And indeed, there is no obvious pattern among the census regions in the severity of the long-term effects estimated (apart from the tendency for certain western states to have milder ef-

fects).[57] When main effects for region of birth are added to (3), along with interactions between region of birth and the 1919 cohort dummy, no consistent pattern of regional magnitudes is apparent for 1960. For the 1970 and 1980 Censuses, however, there is some evidence that damage was larger for those born in 1919 in the South. The significance of these differences is eliminated, however, when nonwhites are dropped. Reductions in the magnitudes of the southern point estimates drive this change.[58] This raises confidence that the geographic variation of the pandemic does not operate on the obvious geographic "fault lines" of North versus South and so forth.

B. Differences by State of Birth

Permutations in the econometric specification in (10) and changes to the analysis sample have modest effects on $\hat{\psi}$. Allowing for unrestricted cohort effects by region (i.e., including 12 dummy variables for each birth year in each region of birth, as well as region main effects) strengthens the estimates of ψ. Therefore, the results are not driven by regional differences in cohort effects. Dropping states with the largest effects on $\hat{\psi}$ (Pennsylvania and Virginia) also has modest effects: $\hat{\psi}$ increases in absolute value for years of education and log income, and falls modestly for high school graduation and socioeconomic status.[59] Finally, adding an interaction of ψ with a dummy for whether the state of birth had a high influenza death rate in the *spring* of 1918 does not yield a statistically significant coefficient.

Equations (10) and (11) apply the infection rate of the year preceding birth to capture the prenatal period. This procedure is imperfect, and more so for those born at the end of the year. For those born in the fourth quarter, the preceding year's infection rate should provide the least information, given a nine-month gestation period.[60] Therefore, dropping births in the fourth quarter should increase the estimates of ψ. And indeed, the magnitudes of $\hat{\psi}$ increase when only births prior to October are retained.

The one-third maternal infection rate assumed in Section VI implies

[57] The education effects for those born in the North are weaker in some specifications. It is conceivable that more stringent compulsory school laws mitigated the reduction of education in the North.

[58] This finding suggests that prenatal influenza infection caused greater damage for African Americans. Effects for educational attainment and socioeconomic status are larger among nonwhites across census years, on the basis of $\hat{\beta}_1^r/\bar{y}^r$ in (3). More than three-quarters of those born in the South (1912–22) were white.

[59] When poverty is the dependent variable and these states are dropped, the point estimate changes the most: from .042 to .016.

[60] As most of the variation in maternal mortality rates came from the last quarter of 1918, this lagging is less problematic than if the pandemic had occurred during the summer months.

a κ of 1.4 percent. That is, 1.4 percent of infected mothers died in the pandemic, or roughly three times the influenza mortality rate for the U.S. population. Popular accounts cite evidence of very high maternal mortality rates, though these accounts usually pertain to hospital patients for whom the likelihood of negative selection is high. Increasing the mortality rate for infected mothers provides a sense of whether results from (10) depend on the low assumed value of κ. If one triples the value of κ—that is, over 4 percent of pregnant mothers died—the estimates for ψ actually increase.

VIII. Conclusion

Prenatal exposure to the 1918 influenza pandemic had large negative effects on adult economic outcomes. This study concludes that the full impact of congenital characteristics may not be immediately observed and therefore may go undetected. Like congenital characteristics, hereditary characteristics also exert latent effects on adult outcomes. Congenital characteristics, however, are mediated by the prenatal environment. This strongly suggests that economic outcomes are malleable in a way not widely recognized and therefore subject to improvement.

This finding, combined with previous positive findings on the long-term health effects of the prenatal period, helps explain the gradient between adult health and economic outcomes. That fetal health may be at the fulcrum of this relationship suffers no shortage of policy implications. The most pressing among these concerns racial disparities in the United States. Early-life health measures of blacks have stagnated since the late 1990s: a black infant is currently more than twice as likely to die before age 1 as a white infant. The results of this study indicate that a future of racial inequality is being programmed. Interventions targeting early-life health of black infants hold promise for reducing racial disparities in adult health and economic outcomes. Identifying efficient public policies to achieve this end should be a priority of future research.

Appendix

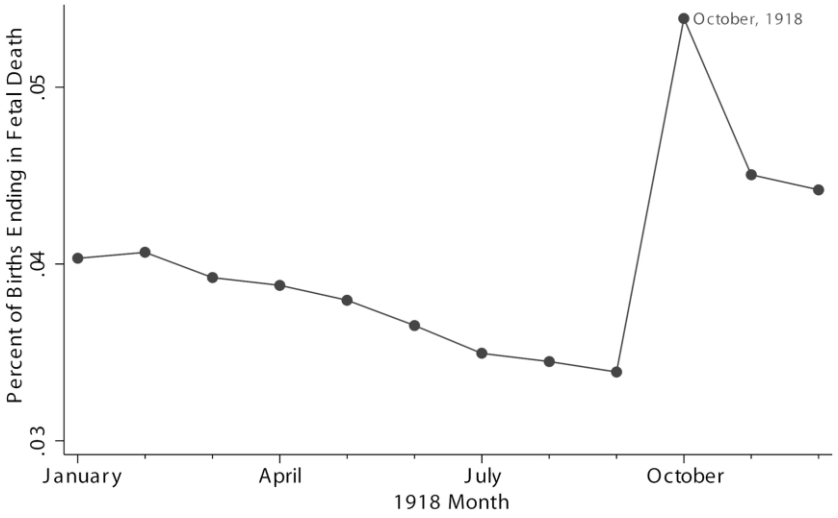

FIG. A1.—Average stillbirth rate in 17 U.S. states, by month of 1918

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