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Maternal health and the baby boom

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Fertility in the United States rose from a low of 2.27 children for women born in 1908 to a peak of 3.21 children for women born in 1932. It dropped to a new low of 1.74 children for women born in 1949, before stabilizing for subsequent cohorts. We propose a novel explanation for this boom–bust pattern, linking it to the huge improvements in maternal health that started in the mid-1930s. Our hypothesis is that the improvements in maternal health contributed to the mid-twentieth century baby boom and generated a rise in women's human capital, ultimately leading to a decline in desired fertility for subsequent cohorts. To examine this link empirically, we exploit the large cross-state variation in the magnitude of the decline in pregnancy-related mortality and the differential exposure by cohort. We find that the decline in maternal mortality is associated with a rise in fertility for women born between 1921 and 1940, with a rise in college and high school graduation rates for women born in 1933–1950 relative to previous cohorts, and with a decline in fertility for women born in 1941–1950 relative to those born in 1921–1940. The analysis provides new insights on the determinants of fertility in the United States and other countries that experienced similar improvements in maternal health.

Keywords. Maternal mortality, fertility choice, baby boom, human capital.

JEL classification. J11, J13, J24, N3, N12, N92.

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1. Introduction

The United States experienced very big swings in fertility between the late 1930s and the early 1970s. The cohort total fertility rate[1] rose from a low of 2.27 children for women born in 1908 to a peak of 3.21 children for women born in 1932, as shown in Figure 1. After dropping to a new historical low of 1.74 children for women born in 1949, the rate stabilized at around 2 children per woman in the 1980s. Despite the remarkable magnitude of these fluctuations in fertility and their clear economic and social relevance, their origins are still poorly understood. Perhaps the best known theory is Easterlin's (1961) "relative income" hypothesis, based on the notion that particularly favorable labor market conditions tend to increase desired fertility. Thus, the recovery from the Great Depression and World War II can provide an explanation for the baby boom. This hypothesis, however, runs counter to the very strong negative empirical correlation between income and fertility (Jones and Tertilt (2007)).

We propose a novel explanation for the boom and bust in U.S. fertility that links these phenomena to the dramatic improvements in maternal health that occurred starting in the mid-1930s. In 1900, one mother died for every 118 live births, and pregnancy-related causes accounted for over 15% of all deaths of women 15–44 between 1900 and 1930, the second largest cause of death after tuberculosis. In 1936, maternal mortality started to fall sharply, reaching modern levels by the late 1950s. The virtual elimination of maternal mortality risk was accompanied by a similar reduction in the incidence of pregnancy-related conditions and by a rise in the female–male differential in adult life expectancy from 2.5 years in 1936 to 6 years in 1956.

Our hypothesis is that the improvement in maternal health contributed to the mid-twentieth century baby boom and generated a rise in women's human capital, ultimately leading to a decline in desired fertility for subsequent cohorts. This reasoning can be formalized with a stylized model of fertility choice and costly parental human capital investment, which incorporates pregnancy-related death risk and a quality/quantity trade-off in the demand for children.[2] The model predicts that both fertility and parental investments in daughters' human capital will rise in response to a permanent decline in pregnancy-related mortality, as the health cost of childbearing declines and women's productive life-span expands. While the rise in women's human capital is permanent, the increase in fertility is only temporary. Given that women who experienced the decline in maternal mortality in their formative years have higher education and a higher opportunity cost of children, they will have lower desired fertility than older women who

[1] The cohort total fertility rate (CTFR) is a measure of the total lifetime fertility of the average woman born in a given year. Formally, let $f_{a,t}$ be the number of children born to women of age a in period t divided by the number of those women. Then $\text{CTFR}_t = \sum_{a=15}^{a=49} f_{a,t+a}$. During periods of substantial total fertility changes across cohorts, this measure is preferable to the more often used period total fertility rate (PTFR), defined as $\text{PTFR}_t = \sum_{a=15}^{a=49} f_{a,t}$. This is because it does not mix the fertility behavior of different cohorts. The CTFR is shifted by 27 years to align its peak to the PTFR. The CTFR underestimates completed fertility if maternal death risk is high. The peaks and troughs of the two series are well aligned for the United States, as shown in Albanesi and Olivetti (2010). See Jones and Tertilt (2007) for a discussion of alternative fertility measures.

[2] See Albanesi and Olivetti (2010) for a formal description of the model.

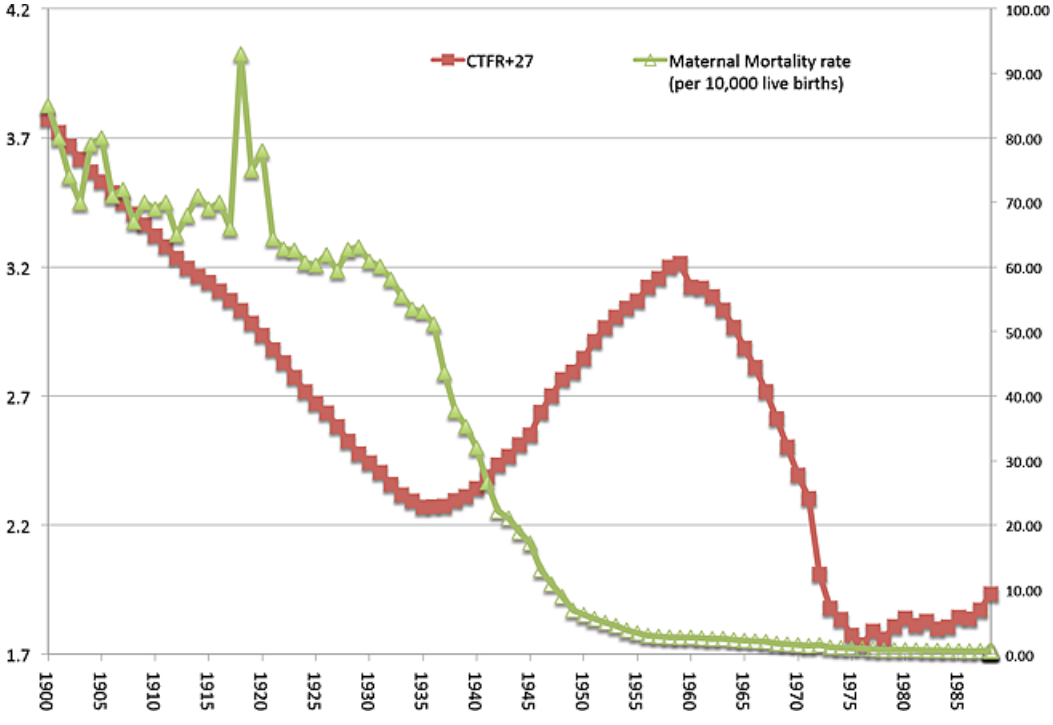

FIGURE 1. Maternal mortality and cohort total fertility rate (+27) in the United States, 1900–1990. *Source*: U.S. Cohort Fertility Tables, CTFR 1917–1980, TFR 1900–1988. Produced by the National Institute of Child Health, compiled in Heuser (1976). U.S. Maternal Mortality: Vital Statistics of the United States.

experienced the decline after completing their education. The resulting boom–bust pattern in fertility qualitatively replicates the U.S. experience. Since the effects on women's human capital are permanent, the long run effect on fertility may well be negative if the returns to human capital are high enough.

Our empirical strategy exploits the large cross-state variation in the magnitude of the maternal mortality decline to estimate its effect on the change in completed fertility and educational attainment for different cohorts of women.

Our estimates suggest that a reduction in maternal mortality of 10 deaths per 10,000 live births is associated with a rise in completed fertility of 0.27 children for married women born in 1921–1940 relative to those born in 1913–1920, about 10% of the actual rise. Given that maternal mortality declined by 40 deaths per 10,000 live births while completed fertility rose by 1.45 children across these two groups of cohorts, the decline in maternal mortality can account for approximately 74% of the rise in fertility during the baby boom. The response of fertility to maternal mortality might seem too large relative to the prevalence of maternal mortality. However, it is important to keep in mind that maternal mortality represents only a fraction of the overall health costs associated with having children. Maternal morbidity was a pervasive phenomenon in the mid-1930s, and the high frequency of stillbirths and miscarriages further increased the health

burden of motherhood. As discussed in Albanesi and Olivetti (2009), the decline in maternal mortality was accompanied by an equally large reduction in pregnancy-related morbidity and in the number of pregnancies (for given fertility). It follows that the decline in maternal mortality in our empirical analysis should be interpreted as a proxy for this, much larger, decline in the overall health cost of having children.

The estimates suggest that the maternal mortality decline also had a very strong effect on the growth in women's educational attainment relative to men. A decline in maternal mortality of 10 deaths per 10,000 live births is associated with a rise in the female–male differential in college graduation rates by 0.05 for the 1933–1950 birth cohorts. Thus, had it not been for the improvements in maternal health, we would have observed an even larger increase in the male advantage in college graduation rates (that widened from 3.6 to 6.8 percentage points between the 1919–1932 and the 1933–1950 cohorts).

Finally, we examine whether the decline in maternal mortality contributed to the decline in fertility that occurred between the early 1960s and the mid-1970s. We compare fertility outcomes of cohorts of women born in 1941–1950, whose education rose in response to the decline in maternal mortality, to outcomes of women who had completed their education when maternal mortality started to decline and only responded with fertility. Our estimates suggest that the decline in maternal mortality played a significant role in the baby bust. A reduction in maternal mortality by 10 deaths per 10,000 live births is associated with a decline in completed fertility of 0.52 children, or 79% of the actual decline across these groups of cohorts.

Taken together, these results suggest that the decline in maternal mortality contributed significantly to the U.S. baby boom and subsequent baby bust, providing a novel, integrated explanation for these important demographic phenomena. Moreover, we show that the decline in pregnancy-related mortality had a sizable impact on the rise in the female–male differential in college graduation. This trend, which began with individuals born in the mid-1930s (Goldin, Katz, and Kuziemko (2006)), has been explained mainly in terms of the introduction of oral contraception (Goldin and Katz (2002) and Bailey (2006)). We interpret the rapid adoption of oral contraception by young women in the late 1960s as spurred by a desire to reduce and postpone fertility, which originated at least in part from the improvement in maternal health and its effect on the returns to human capital investment.

This paper's main contribution is to the macroeconomic literature on the baby boom. Greenwood, Seshadri, and Vanderbroucke (2005) proposed that the diffusion of home appliances was a key determinant of the baby boom, as it reduced the time cost of children. This explanation is not fully consistent with the timing of the baby boom, as fertility started to rise prior to World War II, while the diffusion of home appliances took off in the 1950s and 1960s. It also leaves open the possibility that the rise in fertility and the resulting increase in the number of children per household, a key determinant of the demand for home hours (Ramey (2009)), may have increased the demand for home appliances.

Doepke, Hazan, and Maoz (2007) argued that World War II was an important factor for the baby boom. The rise in labor force participation of married women during the

war crowded out younger women after the war, causing them to opt for marriage and childbearing. This explanation is inconsistent with the fact that fertility began to rise before the war and with the limited direct impact of wartime female participation on labor market conditions. According to Goldin (1991), women who entered the workforce during the WWII years accounted for just 20% of the married women 27–51 years old who were employed in 1951. Goldin and Olivetti (2013) showed that the labor supply response to the war was experienced almost entirely by women in the top half of the education distribution. Moreover, Acemoglu, Autor, and Lyle (2004) found that the impact of wartime female participation on wages was largely exhausted by 1950. Finally, this hypothesis is based on the premise that women who became mothers during the baby boom were not in the workforce, while Albanesi and Olivetti (2009) showed that participation of mothers rose during the baby boom.

This paper also contributes to the literature on the effect of disease eradication on human capital. The most closely related paper in this area is Jayachandran and Lleras-Muney (2009), who studied the impact of maternal mortality decline on female literacy in Sri Lanka.[3] Their estimates suggest a strong positive effect, which they interpret as consistent with a rise in parental investments in the education of daughters. Our results confirm the strong impact of falling maternal mortality on women's education for the United States. Our analysis differs though, since our main goal is to trace out the joint response of both fertility and women's human capital for successive generations of women, not only the short run response in girls' education.

Finally, we suggest a new mechanism through which mortality reductions can influence fertility. Following the pioneering work of Preston (1978), the literature has concentrated on the impact of the reduction in youth mortality on the secular decline in fertility (Preston and Haines (1991) and Haines (1997)) and the joint rise in human capital (Becker, Murphy, and Tamura (1990), Kalemli-Ozcan, Ryder, and Weil (2000), and Soares (2005)). We show that a decline in maternal mortality induces a temporary rise in fertility and a permanent rise in women's human capital. Taken together, these findings suggest that medical progress, and the resulting decline in maternal and infant mortalities, can provide an integrated explanation for the secular decline in fertility—the baby boom and bust—for the overall rise in human capital and the gains in women's human capital relative to men in the post-war period.

Our findings provide new insights on the determinants of fertility and offer a new perspective on demographic policies in developing countries. Albanesi (2012) examined maternal mortality and fertility behavior in 25 advanced and emerging economies between 1900–2000 and found that large maternal mortality reductions were associated with a boom–bust pattern in fertility and a permanent rise in women's human capital, and that only the countries in which such reductions occurred experienced a mid-century baby boom. Albanesi and Olivetti (2009) showed that improved maternal health was critical for the rise in labor force participation of married women during the twentieth century and especially during the baby boom, generating a rise in income per capita

[3] Bleakley (2007) and Bleakley and Lange (2009) studied the impact of malaria and hookworm eradication on fertility and schooling in the American South. They found a negative effect on fertility and a sizable positive effect on schooling.

of over 50% via this channel. These results suggest that improving maternal health can improve standards of living substantially, even without a decline in fertility.

The paper is organized as follows. Section 2 discusses the historical background for the reduction in maternal mortality in the United States. Section 3 briefly presents the theoretical underpinning of our empirical analysis. Section 4 discusses the empirical strategy and presents our results for fertility (Section 4.2) and for the gender differential in educational attainment (Section 4.3). Section 5 examines the link between the baby bust and the decline in maternal mortality. Finally, Section 6 concludes.

2. Maternal health in the United States

This section documents the incidence of pregnancy-related deaths in the early years of the twentieth century and briefly discusses the main developments that led to the remarkable improvements in maternal health that began in the mid-1930s.

2.1 *Advances in maternal health*

The maternal mortality ratio (MMR),[4] which can be interpreted as a measure of the average probability of a maternal death for each live birth, was equal in 1900 to 85 maternal deaths per 10,000 live births, that is, one mother died for every 117 live births. Maternal causes, at 55 deaths per 100,000 female population in 1900, were the second largest cause of death for women after tuberculosis, which was the leading cause of death for both genders at the time. As shown in Table 1, maternal deaths accounted for 3.2% of all female deaths and for 14.9% of all female deaths at ages 15–44 in 1900. Between 1900 and 1930, mortality for all causes declined by 37% for females and 32% for males, and mortality for tuberculosis dropped by over 60%, while maternal mortality declined by only 5.4% in this period.[5] Maternal deaths as a fraction of all female deaths dropped to 1.6% in 1930, though this change is mostly accounted for by the decline in births,[6]

[4]According to the World Health Organization, a maternal death is the death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or from its management, but not from accidental and incidental causes. Maternal deaths are divided into two groups: direct obstetric deaths, which result from obstetric complications of the pregnant state, or from omissions, interventions, or incorrect treatment of that state; indirect obstetric deaths, which result from previous existing diseases that were aggravated by the pregnancy. This distinction was not made for early maternal mortality data; thus the statistics we use throughout the paper count both direct and indirect obstetric deaths.

[5]This pattern was common to other advanced countries and was due mainly to the low standards of maternal care provided by birth attendants (Loudon (1992b)). See Section 2.2 for further discussion on this point.

[6]The pregnancy-related mortality risk depends on age and parity. The maternal death rate has a U-shaped relation with both age and parity (Berry (1977)). The parity adjustment factors over average maternal mortality risk are 1.14, 0.62, 0.64, 0.77, 0.99, 1.12, 1.14, and 1.58 for parities 1–8, respectively. Dublin (1936) estimated that the parity and age distribution was particularly favorable for the 1905–1915 birth cohorts relative to earlier cohorts due to their low fertility, which can account for most of the reduction in maternal mortality between 1900 and 1930. By contrast, the changes in the age and parity distribution between 1936 and the mid-1950s tended to increase the pregnancy-related mortality risk, due to the rise in the number of high parities.

TABLE 1. Incidence of maternal mortality.

	1900	1930	1960	1930–1900	1960–1930
	Death Rates (100,000 Population)			Percentage Change	
All causes					
Men	1791.1	1225.3	1104.5	−31.60%	−9.90%
Women	1646.9	1036.7	809.2	−37.10%	−21.90%
Tuberculosis					
Men	201	76.2	8.9	−62.10%	−88.30%
Women	187.8	65.9	3.3	−64.90%	−95%
Maternal causes					
Women	55	52	3.4	−5.40%	−93.60%
	Deaths by Cause (Percentages)			Percentage Change	
Maternal deaths as a percentage of					
Female aged 15–44 deaths	14.90%	10.60%	7%	−28.90%	−34%
All female deaths	3.20%	1.60%	0.10%	−50.00%	−93.80%
Tuberculosis as a percentage of					
All deaths	11.30%	6.30%	0.70%	−44.20%	−88.90%
	Life Expectancy at Age 20 (Years)			Percentage Change	
Female–male differential	2.0	2.5	6.1	25%	144%

Source: Vital Statistics of the United States.

with maternal mortality still accounting for 10.6% of female deaths at ages 15–44.[7] As a consequence, by 1930, maternal causes accounted for 52 deaths per 100,000 female population, just slightly below tuberculosis, the first cause of death, which registered 65.9 deaths per 100,000 female population.

While maternal mortality began to decline slowly in 1930, its precipitous drop did not start until the year 1936. The maternal mortality rate dropped from 51.16 per 10,000 live births in 1936 to 2.87 in 1956, a 94% drop over a span of just 20 years. This corresponds to a −13.23% average yearly change and accounts for 80% of the decline in maternal mortality between 1930 and 1995; further improvements in maternal mortality in later years were modest. As shown in Figure 2, all causes of maternal death diminished starting in 1936 and reached modern levels by the late 1950s.[8] The most striking decline occurs for deaths due to sepsis, which dropped from 27.5 in 1923 to 0.55 per 10,000 live births in 1955.

[7]Maternal mortality exhibits a large spike during the 1918–1919 influenza epidemic, which also causes a temporary decline in the male–female mortality rate and the female–male differential in life expectancy at age 20 between 1915 and 1920. This is due to the fact that pregnant women are more likely to die of influenza than prime age adults of both genders.

[8]The main causes of maternal death, shown in Figure 2, were septicemia (40% of all maternal deaths in 1921), toxemia (27%), obstructed labor (10%), and hemorrhages (10%).

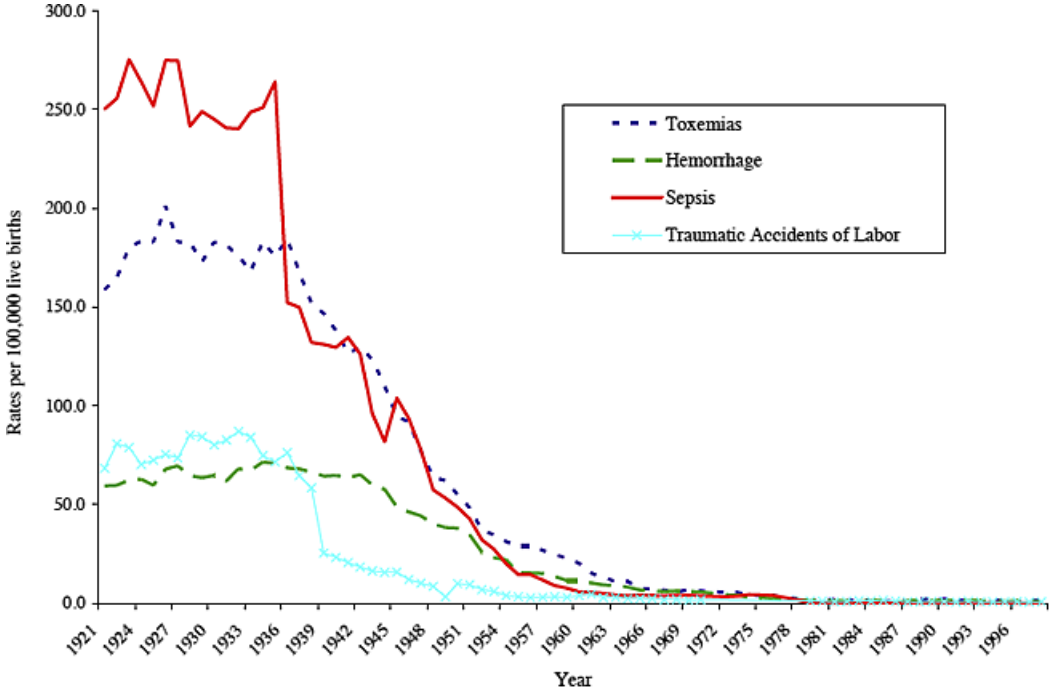

FIGURE 2. Maternal mortality by cause. *Source*: Vital Statistics of the United States.

The decline in maternal mortality was associated with a sizable rise in the female–male differential in adult life expectancy,[9] which, as can be seen in the last panel of Table 1, rose from 2.5 to 6.1 years between 1930 and 1960. Between 1930 and 1960, mortality rates fell by 22% for females but only 10% for males, whereas between 1900–1930, both genders experienced similar declines in mortality. Based on estimates from Rethereford (1972), using a broad set of death causes, the drop in maternal mortality accounts for 14% of the rise in the female–male differential in life expectancy at birth between 1910 and 1965, and for 100% of the change in female–male differentials in mortality rates at age 20–39.

Pregnancy-related morbidity also took a severe toll on women's health. A variety of conditions, such as puerperal fever, obstetric fistulas, hypertensive disorders, and chronic anaemia, could lead to protracted or permanent disability (Albanesi and Olivetti (2009)). Based on postpartum readmission data, 12% of all live births generated some form of maternal morbidity in the late 1920s (Kerr (1933)). No systematic time series data on the evolution of maternal morbidity are available.[10] Franks, Kendrick, Olson, Atrash,

[9]The female–male differential in life expectancy was negative until the early years of the 20th century. Stolnitz (1956) argued that its initial sign reversal may be due to the change in the age and parity distribution of births resulting from the fertility transition in the second half of the nineteenth century, in particular the reduction in the number of births of parity 4 and up, and the resulting decline in maternal mortality rates. The eradication of malaria also played a role, as pregnant women tend to die of malaria at higher rates than other subjects.

[10]Still today, there are significant obstacles to data collection in this area and no generally accepted criteria for the measurement of maternal morbidity (Wilcox and Marks (1994)).

Saftlas, and Moien (1992), the only comprehensive nationwide assessment of this issue, reported an annual rate of pregnancy-related postpartum morbidity requiring hospitalization of 8.1 per 1000 deliveries for 1986–1987, based on hospital discharge records for the United States. The corresponding statistic for the late 1920s, reported in Kerr (1933), is 114.4 per 1000 deliveries.[11] Thus, postpartum pregnancy-related conditions requiring hospitalization dropped by 93% between the late 1920s and the mid-1980s, a magnitude similar to the drop in maternal mortality over the same period (1930–1987). On this basis, the analysis will maintain the assumption that the decline in maternal mortality is accompanied by a similar reduction in pregnancy-related morbidity. This assumption is standard in the literature on the economic impact of disease eradication.[12]

2.2 *Historical background*

Women were keenly conscious of the health risks associated with pregnancy and childbirth, yet even as mortality from other conditions rapidly declined in the first three decades of the twentieth century, maternal mortality remained high until the mid-1930s. This pattern was common to other advanced countries and was due mainly to the low standards of maternal care provided by birth attendants (Loudon (1992b)).[13]

What led to this remarkable improvement in maternal health starting in the 1930s? Efforts to improve maternal health centered around the Children's Bureau, the first federal agency that had the primary responsibility of promoting infant and child health, created by an act of Congress in 1912.[14] The Children's Bureau played a critical role in the decline of maternal mortality in the United States by raising awareness of the preventability of pregnancy-related mortality in the public and in the medical profession. In 1917, the Children's Bureau submitted a milestone report to Congress, documenting that maternal mortality was the second largest cause of death for women age 15–44 after tuberculosis and that the United States was the worst in terms of maternal health among advanced nations (Meigs (1917)). Following this report, maternal mortality was treated as a major health problem (Leavitt (1988)), leading to a series of key federal programs that targeted maternal and infant health, which were in place between 1921 and 1948. A brief description of these programs is provided in Appendix C.

While the federal programs were mainly aimed at increasing public education and improving access to obstetric care, additional initiatives were targeted to medical professionals. Due to poor training and inappropriate and excessive operative procedures, physicians, who started to replace midwives in the 1850s, did not initially contribute to a decline in maternal mortality (Loudon (1992a)).[15] A number of studies found that more

[11] This statistic is based on 1.0646 deliveries per live birth in 1930, using the infant mortality rate for that year, and the maternal mortality rate in 1930, equal to 60.90 maternal deaths per 10,000 live births.

[12] Weil (2007) offered an excellent discussion of this approach.

[13] See Albanesi (2012) for an international account of the decline of maternal mortality in the twentieth century.

[14] For a detailed account of the establishment of the Children's Bureau, see Schmidt (1973), Parker and Carpenter (1981), and Skopcol (1992). Further information can be found at http://www.ssa.gov/history/childb1.html.

[15] Thomasson and Treber (2008) showed that the hospitalization of childbirth had a positive effect on maternal mortality between 1927 and 1937 in the United States. Loudon (1992a) showed that in England,

than two-thirds of all maternal deaths were preventable and that many physicians were found to lack the most basic obstetric knowledge (CDC (1999)).[16] These reports precipitated efforts to standardize obstetric practices and train physicians. The establishment of the American Board of Obstetrics and Gynecology in 1930 (Dannreuther (1931)) led to a widespread improvement in obstetric care in hospitals. Residency training programs and hospital and state maternal mortality review committees, established starting in the 1930s, contributed to the improvement of obstetric care in hospitals.

As can be seen from Figure 2, traumatic accidents of labor started to decline in 1930, and all causes of maternal mortality declined sharply in 1936. This year marked the introduction of sulfonamides, the first type of antibiotic. These drugs were relatively cheap to produce and diffused very rapidly, bringing down mortality from several bacterial diseases, such as pneumonia, influenza, and scarlet fever, in a span of just a few years (Jayachandran, Lleras-Muney, and Smith (2009)). Given that puerperal sepsis accounted for approximately 40% of all maternal deaths in 1936, the introduction of sulfa drugs had a very large impact on maternal mortality. Later, the discovery of the antibiotic effects of penicillin (1939–1942) further contributed to this decline. Another crucial development was the establishment in 1937 of the first hospital blood bank in the United States, at the Cook County Hospital in Chicago. Hemorrhage was the second largest cause of maternal death, and blood banking, along with other innovations in transfusion medicine, eventually also had a large impact on maternal deaths. The decline in maternal deaths from hemorrhage was more gradual, reflecting the slow rise in hospital capacity prior to World War II.[17]

The year 1936 was also significant as it marked the start of the federal subsidies for obstetric care introduced by the Social Security Act, which were critical in increasing access to trained obstetric attendants. As the quality of obstetric care began to improve in the early 1930s, access was still severely limited due to high costs.[18] The expense for a hospital birth varied from $50 to $300 in the 1920s, averaging to approximately 30% of median yearly male labor earnings (Wertz and Wertz (1977)). Fees for an obstetric specialist could significantly increase the financial outlay (Baker (1923)).[19] The development of the first Blue Cross hospital prepayment plans starting in the late 1920s and

high income mothers, who more often gave birth attended by a physician, had higher pregnancy-related mortality rates than low income mothers.

[16]The most significant was the "Child Health Protection, Fetal Newborn, and Maternal Mortality and Morbidity Report," published in 1933, which was based on a nationally representative sample and collected the proceedings from the White House Conference on Child Health and Protection (1933), convened by President Herbert Hoover and sponsored by the Children's Bureau. Similar findings emerged from a study of 2041 maternal deaths in childbirth by the New York Academy of Medicine, also published in 1933.

[17]At the end World War II, the scarcity of hospital capacity throughout the United States emerged as a major public health problem. This led to the Hill–Burton Act, passed in 1946 to improve the infrastructure of the nation's hospitals.

[18]Geographical distance was also a factor in rural areas prior to the widespread use of automobiles.

[19]The high costs of medically trained birth attendants is probably the main explanation for the persistence of the use of midwives, even in geographical areas with easily accessible hospital care or in states, such as Massachusetts, in which the practice of midwifery was banned by law. Midwives charged much lower fees and their services included daily home visits, lasting typically for a week, as well as housekeeping services. For example, in Detroit in 1917, the fee for a midwife was $7–10, while the fee for a doctor was $20–30. The patient would also have to hire a nurse for all subsequent care, typically doubling the cost. By

other forms of employer provided health insurance in the 1930s helped to alleviate these costs only for a very small number of households (Starr (1982)). The Social Security Act spread the benefits of health insurance more broadly for maternal conditions.

3. Conceptual framework

We base our understanding of the impact of the decline in maternal mortality on fertility and women's educational attainment on a theoretical framework described in detail in Albanesi and Olivetti (2010) and Albanesi (2012). Specifically, the theory predicts that a permanent decline in maternal mortality is associated to a *temporary* rise in fertility and a *permanent* rise in women's human capital.

The negative relationship between desired fertility and maternal mortality is intuitive. A higher pregnancy-related mortality decreases the marginal value of an additional birth. The negative relationship between desired fertility and mothers' endowment of human capital stems from the fact that as long as the maternal mortality probability is positive, a rise in the number of births reduces the expected utility from consumption. The resulting loss in welfare is greater for mothers endowed with higher human capital, who enjoy higher consumption if they survive pregnancy and childbirth. Finally, a decline in maternal mortality increases parental incentives to invest in a daughter's human capital, as it increases the returns to that investment by increasing the daughters' expected utility from consumption in adulthood.

Taken together these properties lead to the prediction that a permanent decline in maternal mortality causes a temporary increase in desired fertility and a permanent rise in women's human capital. This pattern is driven by the differential responses of the affected cohorts to the maternal mortality decline.

Women who experience the decline in pregnancy-related mortality in childbearing years increase their desired fertility as well as their investment in their daughters' human capital. Younger women who experience this decline in their formative years will benefit from greater parental investments in human capital. Thus, when they reach childbearing age, they will have a higher opportunity cost of bearing children than the initially exposed women and will choose a lower number of births. This property leads to a boom–bust pattern in the response of fertility to a permanent decline in maternal mortality. While the rise in women's human capital is permanent, the long run effect on fertility once the advances in maternal health are exhausted may well be negative if the returns to human capital are high enough.

Fertility also responds to changes in the youth survival probability. Specifically, fertility declines if youth survival probability increases, provided initial fertility is high enough. This property implies that the model is consistent with the historical experience of the United States and other advanced economies, where a reduction in youth mortalities starting in the mid-1850s was associated with a decline in fertility (Preston (1978), Preston and Haines (1991)). Infant mortality continued to decline gradually in

1930, the cost for a midwife had risen to \$25–30, and the cost for doctors to \$65. See Litoff (1986) and Wertz and Wertz (1977) for a discussion.

the United States during the course of the twentieth century, and we will examine its relation with fertility in our empirical analysis.

These predictions provide a conceptual framework for the empirical analysis.

4. Empirical analysis

We now proceed to examine the empirical link between the decline in maternal mortality, fertility, and women's human capital. In our analysis, we treat the drop in maternal mortality as a quasi-experiment and we interpret the cross-state variation in initial maternal mortality as exogenous.[20] Before delving into the analysis, we provide descriptive evidence of the extent of variation of maternal mortality and fertility across U.S. states.

As discussed in Section 2.1, maternal mortality did not begin to decline systematically until 1930 and started to drop sharply in 1936, reaching modern levels by the late 1950s. The timing of the maternal mortality decline was similar in all states, as can be seen in Figure 3, which displays maternal mortality for the white population in the states, grouped by Census region. By the late 1950s, maternal mortality had converged to uniformly low levels in all states. This implies that the magnitude of the overall drop in maternal mortality in each state between the mid-1930s and the late 1950s is highly correlated to the initial level of maternal mortality.

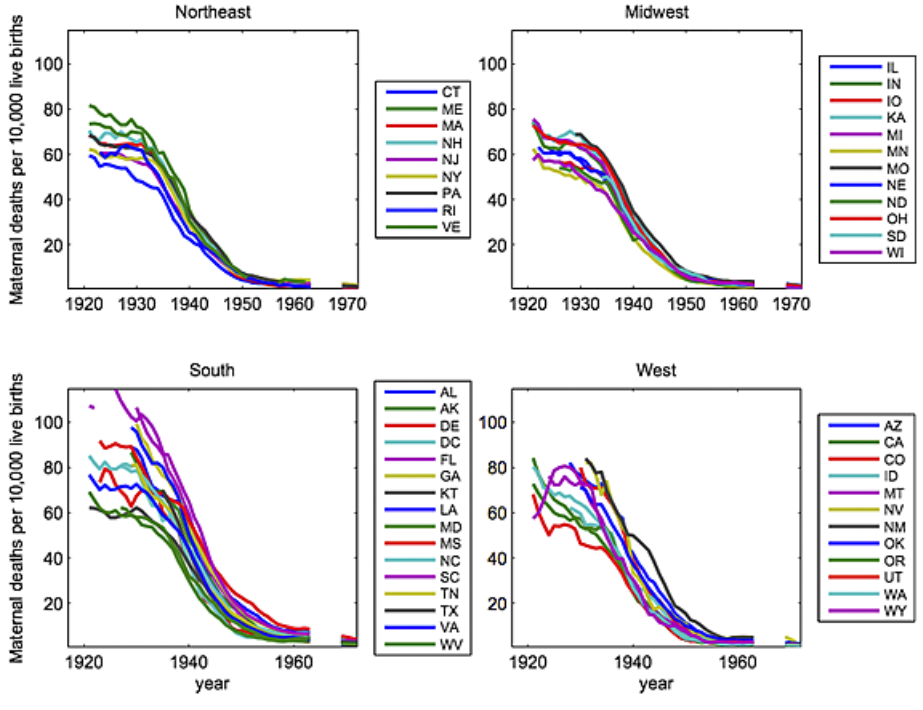

Figure 3. Evolution of maternal mortality in the United States. *Source*: Vital Statistics of the United States.

[20]We discuss and assess the validity of this assumption in the sensitivity analysis.

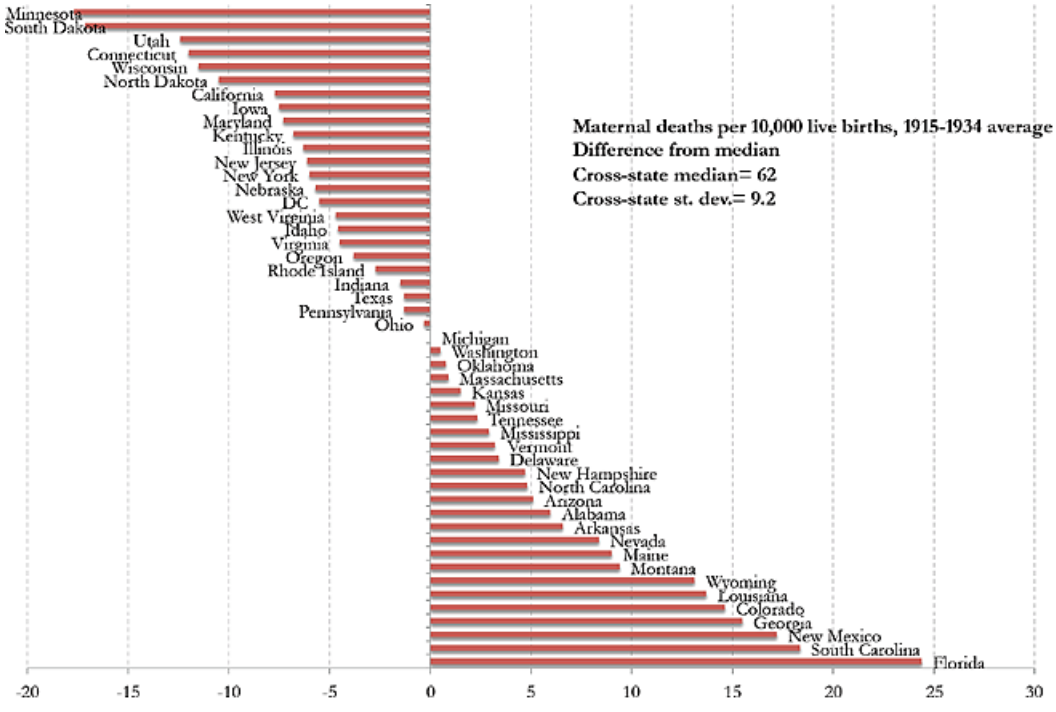

FIGURE 4. Cross-state variation in maternal mortality, 1915–1934. *Source*: Vital Statistics of the United States.

The cross-state variation in initial maternal mortality was indeed sizable. As can be seen in Figure 3, between 1900 and 1930, the southern and western states display considerably higher maternal mortality rates than the northeastern and midwestern states. To examine the pattern of cross-state variation in initial maternal mortality more in detail, Figure 4 displays the difference between the average maternal mortality rate for the white population in years 1915–1934 in each state and its cross-state median, which was equal to 62 deaths per 10,000 live births. Minnesota displays the lowest maternal mortality rate for this time period, at 44 deaths per 10,000 live births, and Florida displays the highest, at 86 deaths per 10,000 live births. The cross-state standard deviation of average maternal mortality in 1915–1934 is 9.2.

To summarize, two features of the decline in maternal mortality stand out clearly. Maternal mortality started to decline in the mid-1930s in all states. This pattern allows us to identify the cohorts of women who experienced the improvements in maternal health at different stages of their life cycle. Additionally, there is a sizable cross-state variation in the magnitude of the drop in maternal mortality, which, based on the theory, should give rise to a differential response of fertility.

The cross-state dispersion in fertility is also notable for the 1900–1935 period, as shown in Figure 5, although the time variation in fertility is similar across states. All states experienced a decline in fertility until the late 1930s, followed by a sizable baby boom, which protracts into the late 1950s, and a subsequent pronounced bust. Figure 6 examines the cross-state variation in fertility in the 5 years prior to the start of the baby

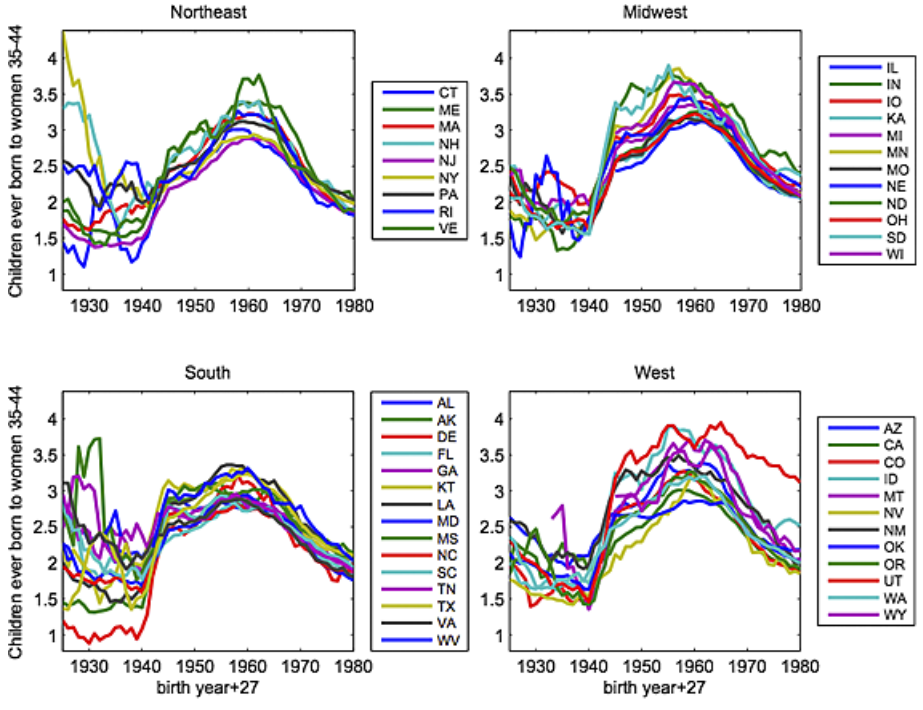

FIGURE 5. Evolution of completed fertility in the United States: children ever born at ages 35–44 by birth cohort, shifted by 27 years. *Source*: IPUMS.

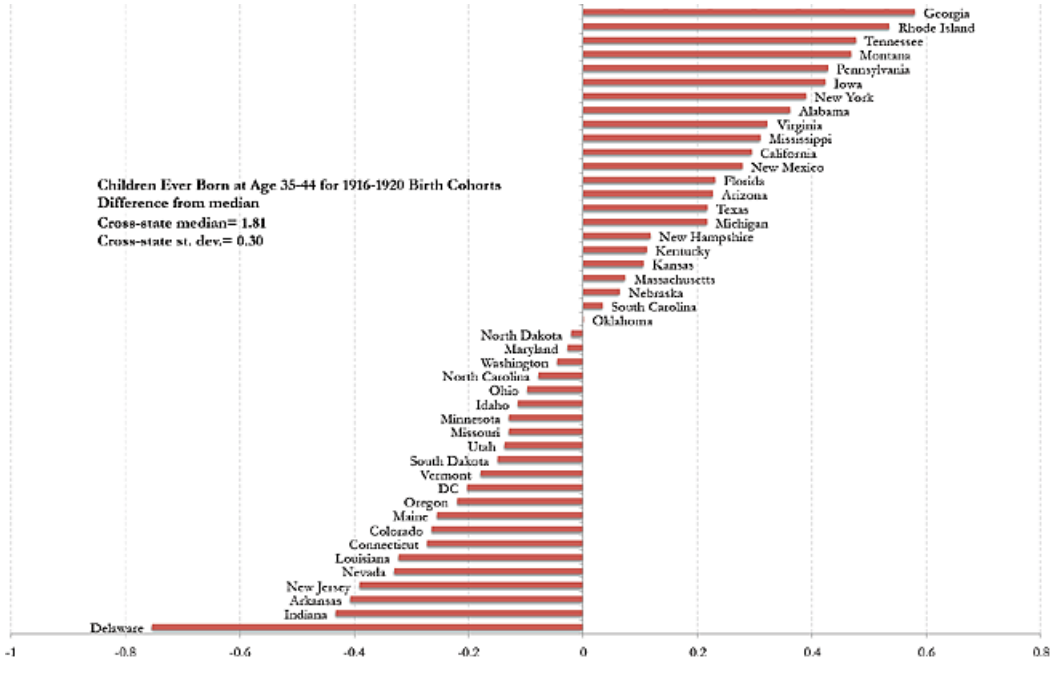

FIGURE 6. Cross-state variation in completed fertility, 1932–1937: children ever born at age 35–44 by birth cohort, shifted by 27 years. *Source*: IPUMS.

boom (1932–1937), as measured by children ever born to women born in 1916–1920 by age 35–44. The cross-state median for completed fertility in this time period is 1.81 children per woman, with a minimum of 1.05 (District of Columbia) and maximum of 2.39 (Georgia).

Note that there is no evident correlation across states between initial (i.e., pre-decline) levels of maternal mortality and initial (i.e., pre-baby boom) completed fertility. These series, plotted as deviations from the cross-state median, respectively, in Figure 4 and 6, display a correlation of 0.014 with a p-value of 0.926. This indicates that our empirical strategy is valid, as we are not capturing preexisting trends in fertility and mortality across U.S. states. Further evidence that our results are not driven by preexisting trends in these variables is provided in Table 13 in Appendix B.

4.1 *Estimation strategy*

The goal of our empirical analysis is to estimate the effect of the decline in maternal mortality on completed fertility and women's educational attainment. The estimation is based on a panel approach, where the dependent variable is given by a fertility or education outcome, the independent variable is a measure of maternal mortality, and the unit of observation is a state–cohort pair. The variation in exposure across states and birth years identifies the impact of the maternal mortality decline on the outcomes of interest.

To attain a homogeneous sample, we restrict attention to white women living in non-farm households. We exclude non-whites to eliminate the effects of the cross-state variation in black–white differentials in maternal mortality. Maternal mortality was higher for blacks; it declined by a similar magnitude as for whites, though later. Blacks also experience a baby boom, slightly smaller in magnitude than whites. We exclude women living in farm households to eliminate the effects of the cross-state variation in the share of farm households. The percentage of farm households varied across states and declined for later cohorts. Fertility for farm households was initially greater than for non-farm households, though this differential declined over time. By contrast, there were no systematic differences in maternal mortality for urban and rural women (Loudon (1992b)).[21]

There are two components of the estimation design. The first is the measure of maternal mortality rate to be used in the estimation. Such a measure must be relevant for subjects' fertility decisions or for parental investments in daughters' education. At the same time, it should not be influenced by the subjects' fertility behavior or education. For this purpose, we introduce the notion of *reference maternal mortality*, defined as the average maternal mortality in the state in a specific age range, chosen to alleviate concerns pertaining to joint endogeneity or reverse causation.

The second component of the estimation design is the choice of cohorts to include. Since the decline in maternal mortality occurs over 20 years, its effect will be identified

[21]The main regression specifications were estimated including women in farm households, with essentially identical results. The estimates are available upon request.

from a comparison of fertility and education for different birth years, rather than relying on a strict definition of treatment and control group. Women young enough for their fertility decisions or education to respond to the drop in maternal mortality are considered potentially treated. Since 1936 marks the start of the phase of most rapid decline in maternal mortality and since public media outlets, such as daily newspapers, started reporting on this phenomenon in late 1937, cohorts who reached childbearing age by 1938 can be considered potentially treated for the fertility analysis, and cohorts who, in 1938, were young enough for their parental investments in education to respond can be considered potentially treated for the education analysis. We then also include older cohorts, based on data availability, to serve as a control group.

The next sections describe the estimation strategy in detail and discuss our main findings.

4.2 *Fertility*

We adopt a simple panel estimation approach, based on the baseline regression equation

$$Y_{st} = \alpha_0 + \alpha_1 \text{MMR}_{st}^{\text{ref}} + \beta X_{st} + \mu_s + \delta_t + \varepsilon_{st}, \tag{1}$$

where Y_{st} denotes the fertility outcome for birth year t and state s. Only females are included in the analysis. The treatment variable $\text{MMR}_{st}^{\text{ref}}$ is the *reference maternal mortality* for state s and cohort t. This is defined as the average maternal mortality rate in the state of residence at age 15–20 for each cohort.[22] The variable X_{st} denotes a set of controls, while μ_s and δ_t correspond to state and cohort effects.

The baseline specification includes women born between 1913 and 1940. Women born in 1921–1940 are 17 or younger in 1938, enjoy the benefit of declining maternal mortality throughout their childbearing years, and can be classified as treated. Women born between 1913 and 1920 can be interpreted as a control group, although the youngest of these women may have also benefited from the maternal mortality decline at a later stage of the reproductive cycle. The sensitivity analysis explores alternative definitions of reference maternal mortality and criteria for inclusion of different birth cohorts.[23]

All specifications include a control for infant mortality, which, as previously discussed, has been found to be positively related to fertility. Thus, for the baseline specification, $X_{st} = \text{IMR}_{st}^{\text{ref}}$, we define reference infant mortality as the mean infant mortality rate (IMR) in the state at age 15–20 for each cohort. Progressively, we include a set of state-level controls for possible cohort specific economic, demographic, health, political, and cultural indicators, which we describe in Section 4.2.2.

[22] Average age of first birth was well above 20 for our cohorts (specifically, it was 24.6 for women born in 1911–1918, 23.7 for women born in 1921–1928, and 22.7 for women born in 1931–1938). Thus our choice of age range for the reference maternal mortality ensures that the fertility behavior of the women included in the estimation does not affect their reference maternal mortality.

[23] Data availability also poses a restriction on the older cohorts we can include in the analysis. State-level data on maternal mortality as a fraction of live births became available only in 1915 and were available for all states (excluding Alaska and Hawaii) only starting in 1929.

TABLE 2. Summary statistics on completed fertility and reference maternal mortality.

	Birth Years 1913–1920			Birth Years 1921–1940		
	Reference Maternal Mortality (Age 15–20) Mean: 53.94 St. Dev.: 7.969			Reference Maternal Mortality (Age 15–20) Mean: 13.94 St. Dev.: 13.1061		
Children Ever Born (Age 35–44):	Married	All	Married With Children	Married	All	Married With Children
Mean	2.558	2.409	2.823	4.017	3.844	4.069
St. dev.	0.204	0.247	0.197	0.286	0.352	0.237

The coefficient of interest is α_1, which captures the cross-state average impact of the change in maternal mortality on the change in fertility in a comparison of two birth year $t' > t$ cohorts:

$$Y_{st'} - Y_{st} = \alpha_1\left(\mathrm{MMR}^{\mathrm{ref}}_{st'} - \mathrm{MMR}^{\mathrm{ref}}_{st}\right) + \delta_{t'} - \delta_t + \beta(X_{st'} - X_{st}).$$

A negative value of α_1 implies that the decline in maternal mortality is associated with a rise in fertility.

Our outcome of interest is completed fertility. Since the median age of last birth for the cohorts included in our analysis was 29, we use children ever born at age 35–44 as our main fertility measure.[24]

We conduct the estimation on three different samples: all women, married women, and married women with children. Since extramarital fertility was small for the cohorts we consider, the results for all women can be seen as a robustness check for our benchmark specification that includes only married women. Separate analysis of the sample of married women with children allows us to assess the response of fertility on both the extensive and the intensive margins for married women.

Table 2 presents summary statistics for the main variables included in the baseline specification. Reference maternal mortality averages 54 deaths per 10,000 live births for the 1913–1920 birth years, and drops to 14 deaths per 10,000 live births for the 1921–1940 birth years. Fertility for married women rises from 2.56 for those born in 1913–1920 to 4.02 for those born in 1921–1940.

4.2.1 *Baseline results* Table 3 presents the estimation results for the benchmark specification. The baseline estimates suggest that the decline in maternal mortality had a strong positive effect on fertility. For the sample of married women, the estimated coefficient, which is significant at the 1% level, suggests that a decline in reference maternal mortality equal to 10 deaths per 10,000 live births is associated with a rise in completed fertility of 0.27, corresponding to 10% of the average number of children ever born by 35–44-year-old married women born between 1913 and 1920. The coefficient on infant

[24]Data are from the U.S. Census. See Appendix A for a detailed description of the data.

TABLE 3. Fertility: regression results.

Sample:	Married	All	Married With Children
MMR_{st}^{ref}	**−0.027**	**−0.024**	**−0.03**
	(−7.0978)	(−6.4142)	(−7.3895)
IMR_{st}^{ref}	0.0115	0.0093	0.0151
	(3.0821)	(2.3631)	(2.4272)
Constant	19.1918	23.1878	−51.441
	(1.7029)	(1.9787)	(−2.9244)
Adj. R-squared	0.5178	0.5049	0.2528

Notes: All regressions include state and cohort effects. Reference maternal mortality is the average maternal mortality in the state at age 15–20 for each cohort. Reference infant mortality is the average infant mortality in the state at age 15–20. Standard errors are clustered at state level. The T-stats are shown in parentheses.

mortality is positive, consistent with a negative relationship between the decline in infant mortality and the change in fertility, and significant at the 1% level. A decline in infant mortality equal to 10 deaths per 1000 live births is associated with a decline in fertility of 0.11 children per woman.[25]

The estimated effect of a decline in maternal mortality on fertility is sizable. Since reference maternal mortality declined by 40 deaths per 10,000 live births for the 1921–1940 cohorts relative to the 1913–1920 cohorts, the associated rise in fertility across these two groups of cohorts based on our benchmark estimates is 1.08 children per woman.

The response of fertility to maternal mortality may seem implausibly large relative to the prevalence of maternal mortality. However, it is important to consider that maternal mortality represents only a fraction of the overall health costs associated with having children. Maternal morbidity was also pervasive, and the high frequency of stillbirths and miscarriages further increased the health burden of motherhood. As discussed in Albanesi and Olivetti (2009), the decline in maternal mortality was accompanied by an equally large reduction in pregnancy-related morbidity and in the number of pregnancies for given fertility, due to the associated decline in the fetal death rate. The decline in maternal mortality in our empirical analysis is a proxy for the much larger reduction in the overall health cost of having children. For this reason, the large estimated response of fertility is not surprising.

Reference infant mortality declined by 22.16 deaths per 1000 live births for the 1921–1940 cohorts relative to the 1913–1920 cohorts, with an associated decline in fertility of −0.24 children per woman based on the estimates. Thus, the predicted change in fertility between the 1921–1940 and the 1913–1920 cohorts resulting from the combined decline of maternal and infant mortality is 0.84 children per woman. The actual change in the cross-state average of completed fertility between the 1921–1940 and the 1913–1920 birth years was 1.46 children per woman. These results suggest that the improvement in maternal health and the decline in infant mortality can account for 58% of the change

[25]The inclusion of infant mortality does not affect the estimated coefficient for maternal mortality.

in fertility across these cohorts. As shown in Table 3, the estimates for the sample of all women and married women with children are very similar both in size and statistical significance to those obtained for married women.[26]

To assess the robustness of our findings, we perform a variety of robustness checks. The results of this sensitivity analysis are reported in Appendix B.

Table 11 in Appendix B shows the robustness of our results when we estimate equation (1) including only cohorts born in 1921–1940, that is, cohorts who were all exposed to the decline in maternal mortality. In this case, the states with lower maternal mortality can be interpreted as having experienced a larger treatment. The point estimates obtained in this case are statistically indistinguishable from our benchmark estimates. This indicates that the main source of variation in our analysis is the intensity of the maternal mortality decline across the treated cohorts.

As a second robustness check, we estimate an instrumental variable version of equation (1). We consider two alternative instruments. In the first case, we use the average reference maternal mortality for birth years 1913–1920.[27] This instrument only exploits the initial cross-state variation in maternal mortality, and it is justified by the strong convergence of maternal mortality values across states between the mid-1930s and the mid-1950s. As shown in Table 11 in Appendix B, we obtain smaller coefficients, but our findings for the baseline specification are confirmed. Our estimate implies that a decline in maternal mortality by 40 deaths per 10,000 live births can explain 15.4% of the increase in completed fertility between the 1921–1940 and the 1913–1920 cohorts. This finding is not surprising. The estimates based on this instrumental variable strategy should be considered as a lower bound for our effect since we are not exploiting the variation in the timing of the maternal mortality decline across states and cohorts.[28] We also estimate a specification in which we instrument reference maternal mortality for the treated cohorts with the mortality rates for the diseases that were most affected by the introduction of sulfa drugs, that is, scarlet fever, pneumonia, and influenza (Jayachandran, Lleras-Muney, and Smith (2009)). We define the reference sulfa related mortality rate as the equally weighted average of the mortality rates for scarlet fever, pneumonia, and influenza at age 15–20 for each cohort t and state s. We then use the average of this indicator for all cohorts in the control group as the instrument. The estimates, reported in Table 12 in Appendix B, are similar, both in order of magnitude and significance, to those in the baseline specification. They imply that a decline of 40 deaths per 10,000 live births can explain 43% of the increased fertility across the relevant cohorts.

To conclude our sensitivity analysis, we conduct two additional robustness checks. First, to check for preexisting trends, we also estimate equation (1) including only cohorts that were not exposed to the decline in maternal mortality. Second, we evaluate

[26]We also obtain similar results using as a dependent variable the number of children under 13 living in the household at age 35–44, although this measure of fertility may be biased downward due to grown children having left the household. See Table 3 in Albanesi and Olivetti (2010).

[27]Bleakley (2007) followed a similar approach to assess the effects of malaria eradication on fertility and educational attainment in the American South.

[28]Given that a larger initial value of maternal mortality corresponds to a larger decline, a *positive* value of the coefficient α_1 indicates that the decline in maternal mortality is associated with a rise in fertility between treated and untreated cohorts.

the sensitivity of the estimates to alternative assumptions on reference maternal mortality and included cohorts. The results of these additional robustness checks, reported in Appendix B, Tables 13 and 14, respectively, confirm our findings for the baseline specification.[29]

4.2.2 *Fertility: Additional controls* We run additional regressions where we control progressively for several state-level indicators. The maintained assumption in our approach is that the cross-state variation in initial maternal mortality is exogenous. Thus, we include a broad set of controls that are possibly related to both fertility and maternal mortality to isolate the direct relationship between these two variables and to assess the potential for omitted variable bias.

We first consider a set of health indicators, including the male mortality rate, the tuberculosis mortality rate, and the malaria mortality rate. The male mortality rate and tuberculosis, which was the top cause of death for both men and women for the 1913–1920 birth years, can be interpreted as proxies for general health conditions in the state. We control for malaria, since malaria eradication has been linked to a decline in fertility and educational attainment (Bleakley (2007)). Moreover, pregnant women are more likely to die from malaria, so variation in the incidence of malaria may account in part for the cross-state differences in maternal mortality. Finally, we control jointly for mortality rates for diseases affected by the introduction of sulfa drugs (Jayachandran, Lleras-Muney, and Smith (2009)), specifically scarlet fever, pneumonia, and influenza. We also control jointly for all the health indicators.

These controls are cohort specific; they capture changes other than reference maternal mortality across cohorts. For each mortality rate, the reference value for the white population for each cohort is calculated as the average in the state at age 15–20 (consistent with the definition of reference maternal mortality). The results are displayed in Table 4 (left panel). We report estimates only for the sample of married women for the baseline specification. The effect of the decline in maternal mortality on fertility is robust to the inclusion of the health controls, one by one or jointly, both in terms of the magnitude and the significance of the estimated coefficient.

We next control for economic and demographic changes across cohorts. Group 1 includes state-level personal disposable income per capita and unemployment, interpreted as simple measures of the level of economic activity. Group 2 includes the share of white population, the share of foreign born, and the share of population living on a farm. These indicators are often linked to fertility behavior and are included to capture some of the cross-state variation in fertility. Group 3 simply includes the share of employment in the public sector, as an indicator of the size of government in a particular state. Group 4 includes the share of employment in the health sector, as a proxy for the availability of medical services. The variable included in the regression is the average value of the control at age 15–20 for each cohort. We also control jointly for all these indicators (group 5). The results are displayed in Table 4 (middle panel). The results are robust to the inclusion of these controls. These controls are also cohort specific; for each cohort, the average of each variable at age 21–34 is used.

[29] See Section 4.1.2 in Albanesi and Olivetti (2010) for a detailed description of the sensitivity analysis.

TABLE 4. Fertility: regression results with controls.

Health		Economic and Demographic		State Characteristics	
1: Male mortality		1: Personal income, unemp.		1: Literacy 1930	
MMR_{st}^{ref}	**−0.051**	MMR_{st}^{ref}	**−0.025**	MMR_{st}^{ref}	**−0.044**
	(−12.7238)		(−5.0038)		(−12.3629)
Adj. R-squared	0.4716	Adj. R-squared	0.4978	Adj. R-squared	0.4309
2: Sulfa related mortality		2: White, foreign born, farm		2: Acceptance of women's suffrage	
MMR_{st}^{ref}	**−0.049**	MMR_{st}^{ref}	**−0.044**	MMR_{st}^{ref}	**−0.045**
	(−12.1345)		(−9.8117)		(−13.123)
Adj. R-squared	0.4857	Adj. R-squared	0.501	Adj. R-squared	0.4443
3: TB mortality		3: Share public		3: Sheppard–Towner & Social Security Act	
MMR_{st}^{ref}	**−0.049**	MMR_{st}^{ref}	**−0.047**	MMR_{st}^{ref}	**−0.044**
	(−12.1351)		(−11.507)		(−12.5337)
Adj. R-squared	0.4866	Adj. R-squared	0.48	Adj. R-squared	0.431
4: Malaria		4: Share health		4: Mob. rates applied to 1922–1928 cohorts	
MMR_{st}^{ref}	**−0.056**	MMR_{st}^{ref}	**−0.034**	MMR_{st}^{ref}	**−0.033**
	(−13.9627)		(−7.4441)		(−8.559)
Adj. R-squared	0.4826	Adj. R-squared	0.4943	Adj. R-squared	0.4498
5: 1 + 2 + 3 + 4		5: 1 + 2 + 3 + 4		5: 1 + 2 + 3 + 4	
MMR_{st}^{ref}	**−0.052**	MMR_{st}^{ref}	**−0.03**	MMR_{st}^{ref}	**−0.045**
	(−13.0663)		(−6.0572)		(−12.6664)
Adj. R-squared	0.5269	Adj. R-squared	0.5168	Adj. R-squared	0.4495

Notes: Baseline specification. All regressions include reference state and cohort effects. Included cohorts: 1913–1940. Estimates for married sample. Reference maternal mortality is the average maternal mortality in the state at age 15–20 for each cohort. All the other mortalities are defined similarly. Standard errors clustered at state level. The T-statistics are shown in parentheses.

Finally, we control for a set of indicators intended to proxy for state characteristics, including political and cultural preferences, potentially linked to both fertility behavior and maternal mortality. These characteristics are time invariant, thus, for specifications including these controls we drop state effects. The first is the literacy rate in 1930, which may be linked to the ability to absorb medical knowledge for the older cohorts. Moreover, literacy is linked to the diffusion of basic schooling, which was related strongly to progressive values, including sensitivity regarding maternal health (Skopcol (1992)).

The second is an indicator of the acceptance of women's suffrage, which can be linked to maternal health and fertility via multiple channels. In the aggregate, early access to voting rights for women may increase women's political participation and heighten legislative intervention in the area of maternal and infant health. Evidence in favor of this channel is provided by Miller (2008), who found that child mortality was lower and spending for public health higher in states that introduced women's suffrage early. Greater political representation for women may also improve women's bargaining position within the household and directly influence maternal health outcomes by increasing household expenditures on obstetric care, which, as discussed in Section 2, entailed a significant financial outlay. We control for the variable "Acceptance Year," which corresponds to the date at which a state introduced or ratified women's suffrage. A state with an earlier acceptance year is interpreted as having more openness toward women's suffrage.

The third includes indicators that capture state-level spending on maternal and infant health under the auspices of the Sheppard–Towner Act of 1921–1929 and the Social Security Act of 1935. The main goal of the Sheppard–Towner Act was to incentivize educational activities that promote maternal and infant health, while Part 1, Title V of the Social Security Act enacted subsidies for obstetric and infant care. The legislation is described in more detail in Appendix C. For both programs, funding to the states was provided on a grant-in-aid basis and state participation was voluntary. A possible concern is that high fertility states may have had greater incentives to invest in maternal health and experienced a larger decline in maternal mortality. We use newly digitized data on state-level appropriations and spending under these two programs to compute the total per capita federal payments received by each state. The data are described in detail in Appendix A.

The fourth indicator we consider is WWII mobilization rates. Mobilization rates could have influenced fertility and education through a variety of channels. Acemoglu, Autor, and Lyle (2004) found that post-war labor market conditions were related significantly to mobilization rates. Specifically, unskilled salaries were lower in states with high mobilization rates, which they interpreted as a consequence of high participation of low skill women during the war years. Doepke, Hazan, and Maoz (2007) argued that the rise in labor force participation of married women during the war crowded out younger women from the labor market after the war, causing them to opt for marriage and child bearing. Finally, mobilization rates may be linked to the presence of war veterans eligible for GI Bill benefits. The educational benefits, enjoyed directly only by men, were the most generous and popular program, and housing benefits were also substantial

(Altshuler and Blumin (2009)).[30] These subsidies may have affected household income and the demand for children. For example, higher household income may have discouraged wives' participation, thereby increasing their desired fertility. We use state-level mobilization rates from Acemoglu, Autor, and Lyle (2004), interacted with an indicator variable, equal to 1 for the 1922–1928 birth cohorts, who were the greatest recipients of GI Bill educational benefits (Stanley (2003) and Bound and Turner (2002)).

The results are displayed in Table 4 (right panel). Note that since indicators in groups 1–3 are cohort invariant, we drop the state fixed effects from the regression equation. We find that the estimates for all fertility measures are robust to the inclusion of these controls. Interestingly, we find that mobilization rates have a significant positive effect on completed fertility of the 1921–1928 cohorts, although the magnitude of this effect is small.[31] We also control for all these state characteristics jointly and find that the estimated coefficients on reference maternal mortality are robust.

4.2.3 *Marriage and childlessness* We also examine the impact of the maternal mortality drop on marriage rates and childlessness.

For the cohorts included in the analysis, extramarital fertility was very small; women who wished to have children typically married. Therefore, if the decline in maternal mortality made childbearing desirable for more women, it may have led to a rise in marriage rates. To examine this hypothesis, we estimate equation (1) using the percentage of women who are married by age 23 as the dependent variable. The results, displayed in Table 5, suggest that the decline in maternal mortality was associated with a significant rise in marriage rates. A decline in maternal mortality of 10 deaths per 10,000 live births is associated with a 0.02 rise in the marriage rate, which was equal to 0.94 for the 1913–1920 cohorts.

We also investigate the effect of the decline in maternal mortality on childlessness, which can be linked to maternal health, as the adverse health consequences of pregnancy may discourage childbearing. We measure lifetime childlessness as the percentage, by state, of 35–44-year-old women who report to have never given birth to a child. We perform the estimation on the samples "All" and "Married." The results are displayed in Table 5. The panel estimates suggest no significant relationship between women's childlessness and maternal mortality for either sample, despite the fact that childlessness for married women drops from 10% for the 1913–1920 cohorts to 7% for the 1921–1940 cohorts.

4.2.4 *Fertility by education* We also estimate the effect of the decline in maternal mortality on fertility and childlessness by education. We run separate regressions for women with college (COLL) and with high school (HS), for marriage statuses "All" and "Married."

The results are displayed in Table 6. The estimated coefficients on maternal mortality are highly significant and have the same sign as the baseline specification for all

[30] See footnote 32 for more details.

[31] Mobilization ranged between 0.41 and 0.54, with a standard deviation of 0.034. The estimated coefficient on mobilization rates for CHBORN is 0.82 and is significant at the 5% level. This implies that a rise in mobilization rates of 0.05 raises CHBORN at age 35–44 by 0.04. By contrast, a 1 standard deviation decline in maternal mortality is associated with a rise in the outcome of 0.24, in the specification that controls for mobilization rates.

TABLE 5. Marriage and childlessness.

	Marriage Rates	Childlessness	
Sample:	All	All	Married
Regression results			
MMR_{st}^{ref}	**−0.002**	−0.0004	0.0002
	(−2.6315)	(−1.2019)	(0.6279)
IMR_{st}^{ref}	0.0005	0.0006	0.0001
	(0.6925)	(0.8888)	(0.8175)
Constant	11.6204	−0.7706	2.5954
	(4.3945)	(−0.457)	(2.0673)
Adj. R-squared	0.2312	0.3767	0.2498
	Birth Years 1913–1920		
Summary statistics			
Mean	0.9362	0.0999	0.107
St. dev.	0.2422	0.0213	0.0491
	Birth Years 1921–1940		
Mean	0.9765	0.107	0.074
St. dev.	0.093	0.0491	0.0219

Notes: All regressions include state and cohort effects. Included cohorts: 1913–1940. Reference maternal (infant) mortality is the average maternal (infant) mortality in the state at age 15–20 for each cohort. Marriage rates measured at age 23. Childlessness measured at age 35–44. Standard errors clustered at the state level. The T-statistics are shown in parentheses.

education groups. The absolute and percentage rises in fertility were greater for college women, and the estimated coefficient indeed predicts a greater rise in fertility for COLL women, relative to HS women.

For childlessness, the estimated coefficient is close to zero and insignificant for all samples except for married women with college. For this group, it is positive and significant, suggesting that a decline in maternal mortality of 10 deaths per 10,000 live births is associated with a 0.03 decline in childlessness for this group. For married high school women, the percentage who are childless at age 35–44 is smaller in the treated group than in the control group, but it is not associated with the decline in maternal mortality (childlessness is approximately constant across the treated and the control groups for the sample of all women). One interpretation of this finding is that the opportunity cost of the adverse health consequences of pregnancy, including death, is greater for college educated women. Then the reduction in pregnancy-related mortality would generate a greater reduction in childlessness for these women.

4.3 *Education*

We now examine the impact of the decline in maternal mortality on female education. Educational attainment rose sharply throughout the twentieth century for both men

TABLE 6. Fertility and childlessness by education.

	Children Ever Born				Childlessness			
Sample:	All	Married	All	Married	All	Married	All	Married
Education:	COLL	COLL	HS	HS	COLL	COLL	HS	HS
Regression results								
MMR_{st}^{ref}	**−0.025**	**−0.03**	**−0.023**	**−0.025**	0.0003	**0.003**	−0.0002	0.0000
	(−5.7012)	(−6.4583)	(−11.8395)	(−12.2026)	(0.3065)	(3.7125)	(−0.6898)	(−0.063)
IMR_{st}^{ref}	0.0005	0.0033	0.0078	0.009	0.0001	−0.0014	0.0009	0.0003
	(0.091)	(0.5429)	(3.0864)	(3.4048)	(0.0804)	(−1.3117)	(2.4241)	(0.9737)
Constant	76.0632	73.565	20.0028	13.9662	−5.5398	−4.447	−1.7209	3.4676
	(3.8264)	(3.4857)	(2.2595)	(1.5158)	(−1.4437)	(−1.1949)	(−1.3575)	(3.3163)
Adj. R-squared	0.0988	0.1382	0.5798	0.5680	0.0264	0.0329	0.3636	0.2718
Model p-value	0.0000	0.0000	0.0000	0.0000	0.0013	0.0001	0.0000	0.0000
	Birth Years 1913–1920				Birth Years 1913–1920			
Summary statistics								
Mean	1.7944	1.8551	2.3074	2.3366	0.143	0.1576	0.1002	0.1008
St. dev.	0.3343	0.3691	0.2832	0.2821	0.0701	0.0777	0.0228	0.0237
	Birth Years 1921–1940				Birth Years 1921–1940			
Mean	2.2739	2.4751	2.9498	3.0625	0.161	0.0972	0.1014	0.0714
St. dev.	0.2545	0.2732	0.3673	0.3152	0.0473	0.0269	0.0548	0.0249

Notes: All regressions include state and cohort effects. Included cohorts: 1913–1940. Reference maternal (infant) mortality is the average maternal (infant) mortality in the state at age 15–20 for each cohort. Standard errors are clustered at the state level. The T-statistics are shown in parentheses.

and women. As shown in Figure 7, college graduation rates were similar for men and women born between 1885 and 1910, after which male graduation rates rose at a substantially faster rate for about 25 years. Goldin, Katz, and Kuziemko (2006) argued that the scarcity of job opportunities during the Great Depression may have provided an incentive to attend college for men. Another important factor that may have contributed to the widening of the gender gap in college graduation rates for the 1911–1930 birth cohorts may have been the GI Bill,[32] for which men were the exclusive recipients of the education benefits. Starting with the 1936 birth year, the female college graduation rate started rising sharply relative to men's. Our empirical analysis seeks to examine the link between the decline in maternal mortality and the rise in female college graduation rates and other measures of women's educational attainment.

The main measures of educational attainment used in the analysis are the fraction of individuals with a college degree in the state. We also consider the fraction with at least 13 years of schooling and the fraction with at least 16 years of schooling for robustness.

[32]Only 2% of the 16 million World War II veterans eligible for GI Bill educational benefits were female. The female beneficiaries received lower stipends than the male counterparts, as their stipend did not rise with the number of dependents (Altshuler and Blumin (2009)). More than 10% of veterans born between 1922 and 1928 achieved a bachelor of arts degree using GI benefits (Bound and Turner (2002)).

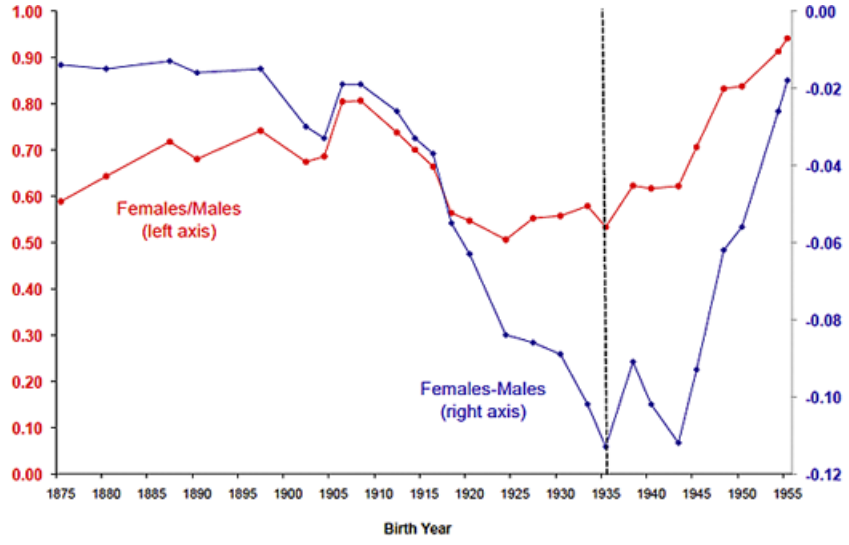

FIGURE 7. Gender differentials in college graduation rates. *Source*: Goldin (1997). Based on responses of individuals aged 45–54 or 55–64. College graduation defined as college attendance for 4 or more years.

Educational outcomes are measured at age 23–32[33] and the estimation is performed on all individuals, irrespective of their marital status. The estimation strategy is similar to the one employed for fertility, except here we include males in the analysis. Thus, in addition to the differences by state and by cohort, we also use the difference by gender to identify the effect of the maternal mortality decline on female education.

We estimate the equation

$$Y_{sgt} = \alpha_0 + \alpha_1 \text{MMR}_{st}^{\text{ref}} \times F_g + \mu_{st} + \nu_{gt} + \delta_{gs} + \beta X_{st} + \varepsilon_{gdt}, \tag{2}$$

where $g = f, m$ stands for gender, F_g is a female dummy that is equal to 1 if $g = f$ and 0 otherwise, and μ_{st}, ν_{gt}, and δ_{gs} correspond to state–cohort, gender–cohort, and gender–state interactions.[34] As for fertility, the baseline specification is a panel regression that includes cohorts with varying degrees of exposure to the decline in maternal mortality, $\text{MMR}_{st}^{\text{ref}}$. A negative value of the estimated α_1 implies a positive effect of a decline in pregnancy-related mortality on female educational attainment (relative to males) for the panel specification.

The age range for reference maternal mortality and the criteria for inclusion differ from those in the fertility analysis. Consistent with the model, we interpret the change in educational outcomes as resulting from parental investments. Thus, we set the age range for reference maternal mortality to be 5–10, reflecting the age at which parents

[33]We consider ages 23–32, since our focus is on parental investments in education, which can be thought to cease at the age of majority. We also consider educational outcomes at age 23, at age 35, and at ages 35–44 as a robustness exercise (not reported). The findings are consistent with those for the baseline specification.

[34]This specification follows Jayachandran and Lleras-Muney (2009).

TABLE 7. Education: summary statistics.

	Birth Years 1919–1932, Control			Birth Years 1933–1950, Treated		
	Reference Maternal Mortality (Age 5–10) Mean: 48.56 St. Dev.: 7.335			Reference Maternal Mortality (Age 5–10) Mean: 9.113 St. Dev.: 2.024		
Educational Attainment at Age 23–32:	COLL	HG16	HG13	COLL	HG16	HG13
Mean female	0.042	0.073	0.197	0.137	0.137	0.295
St. dev. female	0.015	0.024	0.051	0.062	0.062	0.077
Mean male	0.078	0.12	0.246	0.205	0.205	0.381
St. dev. male	0.022	0.035	0.057	0.064	0.064	0.076

typically make choices on behalf of their children that affect lifetime educational attainment. This age range ensures that the subjects' education cannot affect reference maternal mortality, thus removing the possibility of reverse causation.

For the baseline specification, we include individuals born in 1919–1950 and interpret the cohorts aged 5 or younger in 1937, the year in which reports on the decline in maternal mortality become available in the media, as treated, since parental investments in education at all grade levels can potentially respond at age 5 or less. We conduct sensitivity analysis on the included cohorts.

Table 7 provides summary statistics separately for the 1919–1932 and the 1933–1950 birth years. For both groups, college graduation rates were higher for males than for females and the differentials increases across cohorts. Reference maternal mortality was 48.6 deaths for 10,000 live births for the 1919–1932 birth years and 9.1 for the 1933–1950 birth years.

4.3.1 *Baseline results* The results for the baseline specification are presented in Table 8. The estimates suggest that the decline in maternal mortality had a strong positive effect on the female–male differential in educational attainment. The estimated coefficients for college graduation rates (COLL) and 16 and 13 years of schooling completed (HG16 and HG13, respectively) are statistically significant at the 1% level and imply sizable effects for all educational outcomes. The estimates predict that a decline in reference maternal mortality of 10 deaths per 10,000 live births is associated with a 0.05 rise in the female–male differential in college graduation, which was on average equal to −0.036 for the 1919–1932 cohorts.

The estimates also suggest a strong positive impact of the decline of maternal mortality on the female–male differential in the fraction with at least 16 and at least 13 years of schooling. A decline in maternal mortality of 10 deaths per 10,000 live births is associated with a 0.03 rise in the female–male differential in the fraction with at least 16 years of schooling for the married sample. This differential is equal to −0.05 on average for the 1919–1932 birth cohorts.

TABLE 8. Education: regression results.

Educational Attainment at Ages 23–32:	COLL	HG16	HG13
$MMR_{st}^{ref} * F_g$	**−0.005**	**−0.003**	**−0.004**
	(−15.2105)	(−12.2182)	(−19.7548)
Constant	0.1507	0.1437	0.2996
	(101.6118)	(196.6923)	(195.6218)
Adj. R-squared	0.593	0.3165	0.174

Notes: Includes state–time, female–time, and female–state interactions. Reference maternal (infant) mortality is the average maternal (infant) mortality in the state at age 5–10 for each cohort. Standard errors are clustered at state level. The T-statistics are shown in parentheses.

4.3.2 *Education: Additional controls* We use the same set of controls used in the fertility analysis, described in Section 4.2.2. The health indicators are computed as an average of the corresponding mortality rate at age 5–10 for each cohort, to ensure that the reference age is the same as for maternal mortality. In addition to the indicators included in Table 4, we also control for infant mortality.[35] The results are displayed in Table 9 (panel I) and suggest that the estimates for education are robust to the inclusion of these controls. Consistent with the literature, we find that a decline in infant mortality is associated with a rise in educational attainment.

The estimates are also robust to the inclusion of the economic and demographic controls (panel II) and the controls for state-level cultural and political preferences (panel III). The estimated coefficient on reference maternal mortality for college graduation rates loses significance when conditioning on literacy in 1930. This may be due to the positive correlation between literacy in 1930 and subsequent educational attainment for both genders. To assess the role of mobilization rates, we interact this variable with a female dummy, given that GI Bill education benefits were available only for male veterans (see footnote 32). Interestingly, we find that mobilization rates had a strong negative effect on the female–male differential in college graduation rates. An increase in mobilization rates of 0.05 is associated with a 0.01 decline of the female–male differential.

Finally, we control for access to oral contraception for unmarried women for the treated cohorts (panel IV). As shown by Goldin and Katz (2002), access to contraception before marriage had a positive impact on women's educational attainment. Access to oral contraception and legal abortion could also have affected maternal mortality directly, by reducing illegal abortions, which were associated with high rates of mortality and complications. We use Bailey's (2006) coding of legal access to oral contraception for unmarried women and access to abortion, interacted with a female dummy and an indicator equal to 1 for cohorts in the treated group, since only women in these cohorts would have had access. We find that including these control does not affect the findings on the effects of maternal mortality.

[35]Note that *all* specifications in the fertility analysis include infant mortality.

TABLE 9. Education: regression results with controls.

I: Health		II: Economic and demographic		III: State Characteristics		IV: Controls for Treated Cohorts	
1: Male mortality		1: Personal income, unemp.		1: Literacy 1930		1: Early legal access to pill and abortion	
$MMR_{st}^{ref} * F_g$	**−0.002**	$MMR_{st}^{ref} * F_g$	**−0.0009**	$MMR_{st}^{ref} * F_g$	0.0001	$MMR_{st}^{ref} * F_g$	**−0.0027**
	(−18.9452)		(−9.2148)		(1.2996)		(−12.7294)
Adj. R-squared	0.3226	Adj. R-squared	0.4811	Adj. R-squared	0.5694	$Pill_{st} * Ipost_t * F_g$	0.0020
							1.5363
2: Sulfa related		2: White, foreign born, farm		2: Acceptance of women's suffrage		$abortions_t * Ipost_t * F_g$	−0.0020
$MMR_{st}^{ref} * F_g$	**0.0003**	$MMR_{st}^{ref} * F_g$	**−0.0003**	$MMR_{st}^{ref} * F_g$	**−0.002**		−1.5405
	(3.0709)		(−3.0102)		(−23.1226)		
Sulfa related	**−0.003**	Adj. R-squared	0.5447	Adj. R-squared	0.4256	Adj. R-squared	0.3590
	(−36.2608)						
Adj. R-squared	0.5187						
3: Malaria		3: Share public		3: Sheppard–Towner & Social Security Act			
$MMR_{st}^{ref} * F_g$	**−0.002**	$MMR_{st}^{ref} * F_g$	**−0.0005**	$MMR_{st}^{ref} * F_g$	**−0.002**		
	(−20.5294)		(−5.1892)		(−21.9833)		
Adj. R-squared	0.3178	Adj. R-squared	0.4961	Adj. R-squared	0.3356		
4: TB		4: Share health		4: Mob. rates applied to 1922–1928 cohorts, interacted with gender			
$MMR_{st}^{ref} * F_g$	**0.0003**	$MMR_{st}^{ref} * F_g$	**−0.0002**	$MMR_{st}^{ref} * F_g$	**−0.002**		
	(3.0194)		(−1.5366)		(−19.1384)		
Adj. R-squared	0.5185	Adj. R-squared	0.5419	Mob. rate*F_g	0.0073		
					(0.5182)		
				Adj. R-squared	0.3103		
5: Infant mortality		5: 1 + 2 + 3 + 4		5: 1 + 2 + 3 + 4			
$MMR_{st}^{ref} * F_g$	−0.0001	$MMR_{st}^{ref} * F_g$	**0.0002**	$MMR_{st}^{ref} * F_g$	0.0001		
	(−1.1563)		(2.2022)		(1.1282)		
IMR_t	**−0.005**	Adj. R-squared	0.6837				
	(−37.6286)						
Adj. R-squared	0.5361						
6: 1 + 2 + 3 + 4 + 5							
$MMR_{st}^{ref} * F_g$	**−0.0003**						
	(−3.0412)						
Adj. R-squared	0.6287						

Notes: Include state–time, female–time, and female–state interactions. Included cohorts: 1919–1950. Reference maternal mortality is the average maternal mortality in the state at age 5–10 for each cohort. Estimates shown for married sample with educational attainment at age 23–32 equal to college. All mortalities are average in the state at age 5–10. Standard errors are clustered at state level. The T-statistics are shown in parentheses.

For each panel, we also run a specification that includes all the controls in the group and find that the estimates are robust.

To further assess the robustness of these findings, we conduct a sensitivity analysis that parallels the one for the fertility estimates. The results are reported in Table 15 in Appendix B. Specifically, we report estimates from a specification in which we include only the treated cohorts, and an instrumental variable (IV) specification, where we use initial reference maternal mortality as an instrument of the decline. This analysis confirms our main findings.[36]

5. Baby bust

We now examine the relationship between the maternal mortality decline and the baby bust. As we have shown, the decline in maternal mortality had a positive effect on educational attainment of women in formative years when the decline in maternal mortality took place (relative to men). The fertility choice model outlined in Section 3 predicts a negative relationship between mothers' education and desired fertility. This suggests that the maternal mortality decline may have contributed in part to the baby bust by increasing educational attainment for these cohorts, relative to women who had completed their education by the time maternal mortality started to decline.

To assess this prediction, we estimate equation (1) including the birth years 1921–1950. The 1921–1940 cohorts responded positively with fertility to the decline in maternal mortality (relative to the 1913–1920 cohorts), while the 1933–1950 cohorts responded positively with education (relative to the 1919–1932 cohorts). To assess whether the youngest cohorts have lower fertility than the older cohorts in this group, we designate the 1941–1950 cohorts as treated and estimate an IV specification, where the instrument is the average reference maternal mortality (at age 15–20) in the state for the birth years 1913–1920, the group of cohorts that can be considered untreated. This approach is consistent with the theory, since the instrument proxies for the magnitude of the maternal mortality decline that influenced fertility for the control cohorts and parental investments in education for the treated cohorts.[37] For robustness, we also consider a specification in which the control group comprises the 1921–1945 cohorts and the treated group of the 1946–1950 cohorts, and a specification in which the 1933–1940 cohorts are in the control group and the 1941–1950 cohorts are in the treatment group.

The findings are displayed in Table 10. The dependent variable is fertility measured using children ever born by age 35–44. We consider the samples of all women, married women, and married with children. The bottom panel presents summary statistics for the fertility outcome for the control and treated cohorts in each specification. Completed fertility for married women is about 30% lower for the treated cohorts relative to the control cohorts. For the instrumental variable specification, a *negative* value of α_1 in equation (1) implies that a decline in maternal mortality is associated with a reduction in fertility between the treated and control cohorts. The estimated coefficient suggests

[36] See Albanesi and Olivetti (2010), for a detailed discussion of these results.

[37] The IV approach also removes the potential for reverse causation, as presumably women born in 1941–1950 with higher education might have experienced lower maternal mortality rates.

TABLE 10. Baby bust.

Included Cohorts:	1921–1950			1921–1950		
Treated Cohorts:	1941–1950			1946–1950		
Sample:	All	Married	Married With Children	All	Married	Married With Children
Regression results						
$MMR_{st}^{ref} * I_t$	**−0.05**	**−0.052**	**−0.048**	**−0.057**	**−0.057**	**−0.052**
	(−37.6121)	(−35.4614)	(−13.2776)	(−27.706)	(−25.4794)	(−10.1638)
IMR_{st}^{ref}	−0.0045	−0.0086	−0.0182	0.0055	0.0019	−0.0078
	(−4.4688)	(−7.9179)	(−6.6559)	(5.3584)	(1.7046)	(−3.1253)
Constant	2.9701	3.1825	4.6994	2.5002	2.689	4.2155
	(47.2883)	(46.7532)	(27.4982)	(36.994)	(36.7368)	(25.4913)
Adj. R-squared	0.7162	0.6425	0.1495	0.6322	0.5374	0.1088
Regression results with state level controls						
1: Early access to oral contraception						
Year, pill	−0.0187	−0.0152	−0.0163	−0.0185	−0.015	−0.0161
	(−5.3822)	(−4.5105)	(−2.4699)	(−5.1776)	(−4.2614)	(−2.3865)
$MMR_{st}^{ref} * I_t$	**−0.01**	**−0.011**	**−0.013**	**−0.007**	**−0.008**	**−0.008**
	(−15.374)	(−17.2385)	(−10.3221)	(−11.6862)	(−12.1633)	(−6.8978)
Adj. R-squared	0.3859	0.3841	0.1013	0.3476	0.3271	0.0663
2: Early access to abortion						
Year, abortion	0.0803	0.0719	0.0611	0.0804	0.0722	0.0618
	(5.4481)	(5.0499)	(2.1793)	(5.2925)	(4.8525)	(2.1618)
$MMR_{st}^{ref} * I_t$	**−0.01**	**−0.011**	**−0.012**	**−0.007**	**−0.007**	**−0.008**
	(−15.2508)	(−17.1542)	(−10.2637)	(−11.5559)	(−12.0644)	(−6.8382)
Adj. R-squared	0.3862	0.3862	0.1005	0.3481	0.3296	0.0656
Summary statistics						
Control group						
Mean	2.8921	3.0172	4.0688	2.7893	2.9178	3.9932
St. dev.	0.3368	0.2862	0.2373	0.3323	0.2797	0.2278
Treated group						
Mean	2.2052	2.3595	3.569	2.032	2.1989	3.4475
St. dev.	0.3038	0.2792	0.2494	0.2802	0.2893	0.2857

Notes: Instrumental variable specification. All regressions include state and cohort effects. The instrument is average reference maternal mortality (average maternal mortality in state at age 15–20) for birth cohorts 1913–1920. Reference infant mortality is the average infant mortality in the state at age 15–20. All results are for the age 35–44 sample. Standard errors are clustered at state level. The T-statistics are shown in parentheses.

that for all specifications, the drop in maternal mortality has a negative and significant effect on fertility of the treated group relative to the control cohorts, for all the samples. The estimated coefficient for maternal mortality is significant at the 1% level and implies that a decline in maternal mortality of 10 deaths per 10,000 live births is associated with a decline in fertility of 0.52 between the treated (1941–1950) and the control cohorts

(1921–1940) for married women. This corresponds to 78% of the actual decline. Similar results hold for the other samples.

To gauge the robustness of these results, we include controls for access to oral contraception for unmarried women and to early legal abortion.[38] Oral contraception may have increased the returns to female education and directly reduced fertility for the treated cohorts. Using Bailey's (2006) coding of legal access to oral contraception for unmarried women and early legal abortion access, we find that for the treated cohorts, the decline in maternal mortality is significantly negatively related to fertility for all and married women.[39]

We also repeat the estimation, separating college graduates from high school graduates. In the theory, the baby bust is generated by the rise in women's education and opportunity cost of children in response to the initial decline in maternal mortality. If the mechanism in the model is correct, the decline in maternal mortality should be associated with a bigger baby bust for all women, than for college and high school women, since part of the decline in fertility is due to the rise in the number of college women, whose fertility is lower than for high school women, ceteris paribus.

The estimates confirm this pattern in the data. The estimated value of α_1 for college graduates is less than half the size of the one for the sample of all women, and it is smaller than the coefficient for high school women. These results are preserved when controlling for early access to oral contraception and legal abortion.

These findings provide support for the theoretical prediction that the decline in maternal mortality causes an increase in fertility for the cohorts who experienced the decline in childbearing years, and a subsequent reduction in fertility for younger cohorts who experienced it in their formative years. In the model, this decline in fertility for the younger cohorts is due to their higher education generating a rise in the opportunity cost of children. While the estimates conditional on education provide support for this mechanism, given the indirect link between the maternal mortality decline and the baby bust, one should be cautious to embrace a causal interpretation of these estimates.

6. Concluding remarks

A permanent decline in pregnancy-related mortality reduces the health costs of pregnancy and increases the returns to investments in women's human capital. Fertility theory predicts a permanent increase in women's human capital and a temporary rise in

[38]We also estimate specifications that incorporate the health indicators, and the economic and demographic controls used in the fertility and education analysis. We find that the estimates are robust to the inclusion of these controls and omit them for brevity.

[39]We treat these controls as invariant state characteristic, even though only the treated cohorts would have been able to benefit from early legal access. This choice is motivated by the fact that legal early access to oral contraception and abortion is not exogenous. It may depend on unobservable state characteristics that could also drive fertility behavior or on demand for birth control coming from a highly educated female population. We also estimate a specification in which these controls are treated as invariant state characteristics and obtain similar results. Similarly, we also control jointly for both these variables, and find that the estimates for the effects of maternal mortality on the change in fertility across cohorts are robust. We omit these estimates for brevity.

desired fertility. Our empirical analysis suggests that the decline in maternal mortality can account for the boom and bust in completed fertility and over 40% of the rise in women's educational attainment with respect to men in the United States.

The link between the decline in pregnancy-related mortality and fertility in the United States opens an interesting new perspective on the cross-country variation in fertility. Many advanced economies experienced baby booms similar in timing, but smaller in magnitude, relative to the United States. Albanesi (2012) examined the link between maternal mortality decline, fertility, and women's human capital in 25 advanced and emerging economies between 1900 and 2000. Among the advanced economies, only those in which maternal mortality declined sharply between 1935 and 1955 experienced a baby boom, and the magnitude of the boom is positively related to magnitude of the drop in maternal mortality. The United States, with the highest rate of maternal mortality among the advanced economies in the 1930s, experienced the largest boom in fertility. In addition, the decline in maternal mortality is associated with a rise in the female–male differential in educational attainment in all countries in the sample.

Appendix A: Data sources and variable definitions

This section describes data definitions and basic data sources. All the data and a more extensive appendix devoted to data sources and data issues are available at http://qeconomics.org/supp/315/supplement.pdf and http://qeconomics.org/supp/315/code_and_data.zip.

A.1 *Fertility and education data*

Most of our demographic and economic state-level data (from 1930 to 2000) are from the Integrated Public Use Micro Sample (IPUMS) of the decennial Census of the United States (Ruggles, Alexander, Genadek, Goeken, Schroeder, and Sobek (2010)). Our sample includes white women and men born between 1896 and 1955. The base sample for the calculation of state-level control variables includes white men and women, aged 16–64. In both samples, we exclude individuals living on farms, as well as those living in group quarters (e.g., prisons, and other group living arrangements such as rooming houses and military barracks).[40] We use the following variables.

Fertility variables: Our main fertility measure is the number of children ever born to a woman by age 35–44. These are defined as the number of live births by all fathers, whether or not the children were still living; they exclude stillbirths, adopted children, and stepchildren. MARRIED equals 1 if married, with spouse present or absent (if IPUMS variable marst is either 1 or 2). MARRIED WITH CHILDREN equals 1 if married with children (if married=1 and IPUMS variable nchild>=1).

Education variables: For 1940–1980, we use the IPUMS variable HIGRADE, which records the highest grade of school attended or completed by the respondent. This vari-

[40]That is, we further restrict the sample to observations with group quarters status equal to 1, "households under 1970 definition."

able can be used to compute years of education as a continuous variable. For later decades (1990 and 2000) we use EDUCREC, which, although not strictly comparable, can still be used to compute comparable measures of graduation rates (high school, college, etc.). HG13 equals 1 if educational attainment is at least 1 year of college (if IPUMS variable higraded>=160), equals 0 if not, and is set to " " if missing; available years, 1940–1980. HG16 equals 1 if educational attainment is at least 4 years of college (if IPUMS variable higraded>=190), equals 0 if not, and is set to " " if missing; available years, 1940–1980. COLL equals 1 if college (if IPUMS variable educrec=9, which corresponds to 4+ years of college); available years, 1940–2000.

A.2 *Mortality data*

State-level data series on maternal mortality rates, infant mortality rates, and stillbirth rates are compiled using the information contained in several volumes of the Vital Statistics of the United States. All the mortality measures used in the analysis refer to the white population. Below we list the specific data sources for each series.

Maternal mortality *Death rates*: 1925–1940: Vital Statistics in the United States, 1900–1940, Table 37. 1940–1960: Vital Statistics in the United States, 1940–1960, Table 47. *Number of deaths from complications of pregnancy*: Vital Statistics of the United States (VSUS) 1961, Table 5-8; VSUS 1962, Table 1-24; VSUS 1963, Table 7-5; VSUS 1964, Table 7-6; VSUS 1965, Table 7-6; VSUS 1967, Table 7-6; VSUS 1968, Table 7-6; VSUS 1969, Table 7-6; VSUS 1970, Table 7-6; VSUS 1971, Table 7-6; VSUS 1972, Table 7-6; VSUS 1973, Table 7-6; VSUS 1974, Table 7-6; VSUS 1975, Table 7-6. 1979–1998: "1979–1998 Archive" accessible on-line at http://wonder.cdc.gov/cmf-icd9-archive1998.html.

Infant mortality *Death rates*: 1925–1940: VSUS 1900–1940, Table 28. 1941–1960: VSUS, 1940–1960, Table 41; VSUS 1961, Table 3-E. 1962–1966: VSUS 1966, Table 2-6. 1967–1971: VSUS 1971, Table 2-6. 1972–1975: VSUS 1975, Table 2-6. 1979–1998: "1979–1998 Archive" accessible online at http://wonder.cdc.gov/cmf-icd9-archive1998.html.

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A.3 *State-level controls*

Economic and demographic controls UNEMPLOYMENT RATE: We use IPUMS variable EMPSTAT to compute state-level unemployment rates. SHARE OF FOREIGN BORN RESIDENTS: We use IPUMS variable BPLD that contains information on place of birth. PER-CAPITA PERSONAL INCOME: We use a statewide measure, that is, across all races and genders, from the Bureau of Economic Analysis (BEA), Regional Economic Accounts. This series is converted to real values using consumer price series Cc1 from the Millennium Statistics of the United States.

Women's suffrage For date of introduction of women's suffrage, see Lott and Kenny (1999). For date of ratification of the XIX Amendment, see Mount (2007).

ACCEPTANCE YEAR: Date at which a state introduced or ratified women's suffrage, if this preceded the date of introduction of the XIX Amendment, or the date in which the XIX Amendment was ratified for those states that had no prior legislation and rejected the Amendment in 1920. There was substantial variation across the states in the timing of the introduction or ratification of the XIX Amendment, which was approved by Congress in 1920. Wyoming was the first state to introduce women's suffrage in 1869 and Mississippi was the last state to ratify it in 1984.

Mobilization rates We use the state-level mobilization rates constructed by Acemoglu, Autor, and Lyle (2004), defined as the fraction of the 18–44-year-old registered males in a state who were drafted for war,[41] based on published tables from the Selective Service System (1956). The average mobilization rate was 0.474 with a standard deviation of 0.035. Mobilization rates varied substantially across states, from less than 42% in Georgia, the Dakotas, and the Carolinas, to more than 52% in Washington, Pennsylvania, New Hampshire, Oregon, and Massachusetts. The Selective Service's guidelines for deferments were based on marital status, fatherhood, essential skills for civilian war production, and temporary medical disabilities, but also left considerable discretion to the local boards. Farm employment, in particular, was a major cause of deferment, as maintaining food supply was considered essential to the war effort. The mobilization rate is also higher in states with higher average male education and with a lower percentage of black males.

GI Bill benefits To control for access to GI Bill educational benefits, we construct an indicator variable, I_t^G, reflecting whether a cohort t substantially withdrew education benefits from the GI Bill. We then interact this variable with the percentage mobilization rate from Acemoglu, Autor, and Lyle (2004), intended to serve as a proxy for the number of eligible recipients.

Stanley (2003) and Bound and Turner (2002) found that the 1922–1928 birth cohorts displayed the largest take up of WWII GI benefits. Specifically, according to Table 2 in Bound and Turner (2002), more than 10% of veterans achieved a bachelor of arts degree using GI benefits for these cohorts. Thus, we set $I_t^G = 1$ for $t = 1922, \ldots, 1928$. We also run a specification where I_t^G corresponds to the percentage of individuals who used GI benefits in the same year to allow for variation in intensity across cohorts.

Federal programs for the promotion of Maternal and infant health *1921–1929 Maternity and Infancy Care (Sheppard–Towner) Act*: Appropriations, Payments to the States, Activities carried out under the Act by the States, fiscal years 1921–1929: Children's Bureau Publication No. 203 (1931). *1935 Social Security Act, Title V, Part 1*: Appropriations, Payments to the States, Activities carried out under the Act by the States fiscal years 1936–1939: Children's Bureau Publication No. 259 (1941).

Early legal access to oral contraception and abortion Early legal access to oral contraception was an outcome of increased attribution of legal rights to minors, a process that started in the late 1950s with the development of the "mature minor" doctrine. We use Bailey's (2006) coding, described in Table 1, of "year law effective." Following Bailey, we code the "year of early access to abortion" as Early access to abortion. This is coded as 1970 for Alaska, California, Hawaii, New York, and Washington, and as 1972 for Vermont and New Jersey. All other states permitted early legal access with Roe v. Wade in 1973.

[41] Since all men in the age bracket 18–44 were registered, their mobilization rate variable represents the fraction of men in this age range who have served. Mobilization rates for Nevada and Washington, DC are not available (the former because it saw large population changes during this time period).

Appendix B: Sensitivity analysis

Table 11. Fertility analysis: excluding cohorts in the control group and initial mortality as instrument.

Specification:	Panel, Birth Years 1921–1940				IV, Birth Years 1913–1940		
Sample:	Married	All	Married With Children		Married	All	Married With Children
MMR_{st}^{ref}	**−0.03**	**−0.021**	**−0.00007**	$MMR_{s}^{pre} * I_t$	**0.0056**	**0.0055**	**0.0046**
	(−6.92)	(−5.17)	(−0.12)		(10.29)	(9.65)	(6.12)
IMR_{st}^{ref}	0.0118	0.0076	−0.0035	IMR_{st}^{ref}	−0.0002	−0.0006	0.0006
	(2.63)	(2.33)	(−0.52)		(−0.04)	(−0.15)	(0.09)
Constant	49.82	39.23	−48.49	Constant	−48.55	−36.09	−128.93
	(4.67)	(4.19)	(−3.98)		(−4.70)	(−3.32)	(−7.85)
Adj. R-squared	0.54	0.64	0.054	Adj. R-squared	0.49	0.48	0.24

Notes: All regressions include state and cohort effects. Treated cohorts: 1921–1940. Reference maternal mortality is the average maternal mortality in the state at age 15–20 for each cohort. Reference infant mortality is the average infant mortality in the state at age 15–20. The instrument for reference MMR in the IV specification is the average reference MMR for birth years 1913–1920. Standard errors are clustered at the state level. The T-statistics are shown in parentheses.

Table 12. Fertility analysis: sulfa mortalities as the instrument.

Sample:	IV, Birth Years 1913–1940		
	Married	All	Married With Children
$SulfaMR_{s}^{pre} * I_t$	**0.0158**	**0.0147**	**0.0077**
	(10.75)	(10.70)	(3.70)
IMR_{st}^{ref}	−0.0259	−0.0275	−0.0361
	(−9.18)	(−10.44)	(−9.05)
Constant	3.8114	3.8	4.8701
	(16.62)	(17.70)	(15.00)
Adj. R-squared	0.4234	0.4706	0.2179

Notes: All regressions include state and cohort effects. Treated cohorts: 1921–1940. The instrument for reference MMR is the average reference mortality rate for diseases treatable with sulfa drugs (pneumonia, influenza, and scarlet fever) for the control cohorts in each state. Reference infant mortality is the average infant mortality in the state at age 15–20. Standard errors are clustered at the state level. The T-statistics are shown in parentheses.

TABLE 13. Falsification exercise.

	Fertility								
	Control Group I			Control Group II			Treatment Group		
Included Birth Cohorts:	1905–1915			1910–1915			1921–1940		
Reference MMR:	Age 15–28			Age 15–23			Age 15–28		
Statistics									
Mean Ref. MMR:	56.027			59.2552			9.1082		
St. Dev. Ref. MMR:	8.242			8.7857			1.9438		
Fertility:	All	Married	Married With Children	All	Married	Married With Children	All	Married	Married With Children
MMR_{st}^{ref}	0.0075	0.0057	0.0079	−0.004	−0.0004	0.0053	**−0.046**	**−0.046**	0.0001
	(1.75)	(1.27)	(1.47)	(−0.70)	(−0.07)	(0.78)	(−11.39)	(−10.73)	(0.01)
IMR_{st}^{ref}	−0.0066	−0.0118	−0.0114	0.0001	−0.0041	−0.0121	0.0175	0.0185	0.0048
	(−1.36)	(−2.33)	(−1.87)	(0.02)	(−0.60)	(−1.57)	(4.67)	(4.61)	(0.37)
Constant	8.1464	32.9638	19.5245	28.7379	30.4389	21.3412	34.9224	42.9063	−68.332
	(0.56)	(2.17)	(1.08)	(1.25)	(1.28)	(0.79)	(5.00)	(5.76)	(−2.84)
Adj. R-squared	0.5676	0.5616	0.4677	0.6068	0.6017	0.5327	0.6649	0.5371	0.054

	Education					
	Control Group I		Control Group II		Treatment Group	
Included Birth Cohorts:	1910–1917		1918–1925		1933–1950	
Reference MMR:	Age 5–23		Age 5–15		Age 5–23	
Statistics						
Mean Ref. MMR:	59.5396		56.7425		6.189	
St. Dev. Ref. MMR:	8.5992		8.3314		1.3483	
Educational Attainment:	HS	COLL	HS	COLL	HS	COLL
$MMR_{st}^{ref} * Fg$	**−0.01**	0.001	**−0.002**	0	−0.0008	**−0.01**
	(−9.76)	(1.74)	(−4.86)	(−0.14)	(−1.63)	(−17.21)
Constant	0.1639	0.0414	0.0759	0.0312	0.3765	0.1457
	(7.84)	(3.42)	(5.57)	(3.48)	(26.81)	(9.36)
Adj. R-squared	0.7242	0.4387	0.4752	0.2191	0.5537	0.5599

Notes: Fertility regressions include state and cohort effects. Fertility is children ever born at age 35–40. Reference MMR is the average mortality rate in the state in a given age range for each cohort. Since the earliest year where MMR is available for all states is 1933, we must extend the age for reference MMR relative to the baseline specification, for which the age range is 15–20 for fertility and 5–10 for education. Reference infant mortality is the average infant mortality in the state at age 15–20. Education regressions include fully interacted state, cohort, and gender effects. Educational attainment is measured at age 25–32. Standard errors are clustered at the state level. The T-statistics are shown in parentheses.

TABLE 14. Included cohorts and reference maternal mortality.

	Fertility							
Specification:	Panel, Treated Only				Panel			
Column:	1	2	3	4	5	6	7	8
Included Cohorts:	1921–1940	1921–1940	1921–1940	1921–1940	1913–1940	**1913–1935**	**1913–1945**	**1920–1940**
Reference MMR:	Age 15–20	**Age 10–15**	**Age 10–20**	**Age 5–15**	Age 15–20	**Age 15–20**	**Age 15–20**	**Age 15–20**
MMR_{st}^{ref}	**−0.03**	**−0.03**	**−0.03**	**−0.02**	**−0.05**	**−0.01**	**−0.04**	**−0.02**
	(−10.73)	(−11.50)	(−12.00)	(−6.71)	(−12.97)	(−2.61)	(−23.28)	(−10.08)
IMR_{st}^{ref}	0.0118	0.012	0.014	0.009	0.02	0.012	0.008	0.009
	(3.98)	(4.18)	(4.63)	(2.74)	(3.74)	(3.36)	(3.55)	(3.46)
Constant	50.8226	86.47	82.06	68.07	40.13	−111	103.8	37.92
	(6.13)	(6.65)	(7.41)	(3.65)	(2.19)	(−6.34)	(17.35)	(4.83)
Adj. R-squared	0.5365	0.539	0.546	0.497	0.468	0.558	0.488	0.538
	Education: College							
Specification:	Panel, Treated Only				Panel			
Included Cohorts:	1933–1950	1933–1950	1933–1950	1933–1950	1919–1950	**1919–1940**	**1919–1955**	**1929–1960**
Reference MMR:	Age 5–14	**Age 0–15**	**Age 5–10**	**Age −5–5**	Age 5–14	Age 5–14	Age 5–14	Age 5–14
MMR_{st}^{ref}	**−0.0057**	**−0.0045**	**−0.0046**	**−0.0028**	**−0.0024**	**−0.0017**	**−0.0025**	**−0.0049**
	(−17.35)	(−18.68)	(−17.91)	(−20.38)	(−26.03)	(−16.89)	(−26.62)	(−29.24)
Constant	0.1458	0.147	0.146	0.148	0.092	0.08	0.096	0.151
	(9.38)	(9.55)	(9.45)	(9.78)	(6.39)	(5.88)	(6.71)	(12.97)
Adj. R-squared	0.5611	0.572	0.565	0.585	0.359	0.311	0.379	0.593

Notes: Fertility regressions include state and cohort effects. Reference maternal mortality is the average mortality in the state at age 15–20 for each cohort. Reference infant mortality is the average infant mortality in the state at age 15–20. Fertility outcome is children born, age 35–44. Education regressions include state–time, female–time,and female–state interactions. Reference maternal mortality is the average maternal mortality in the state at age 5–10 for each cohort. Sample: all college graduates; age: 23–32. Standard errors are clustered at the state level. The T-statistics are shown in parentheses.

TABLE 15. Education: regression results.

Specification:	Panel, Birth Years 1933–1950				IV, Birth Years 1919–1950		
Educational Attainment at Ages 23–32:	COLL	HG16	HG13		COLL	HG16	HG13
$MMR_{st}^{ref} * F_g$	**−0.006**	**−0.006**	**−0.009**	$MMR_s^{pre} * F_g * I_t$	**0.0019**	**0.0012**	**0.0019**
	(−17.35)	(−17.35)	(−20.54)		(21.39)	(14.599)	(15.29)
Constant	0.1458	0.1458	0.2956	Constant	0.0858	0.1103	0.24
	(9.38)	(9.38)	(13.908)		(5.798)	(7.648)	(11.47)
Adj. R-squared	0.5611	0.5611	0.5276	Adj. R-squared	0.3182	0.3067	0.2576

Notes: Includes state–time, female–time, and female–state interactions. Reference maternal mortality is the average maternal mortality in the state at ages 5–10 for each cohort. Standard errors are clustered at the state level. The T-statistics are shown in parentheses.

APPENDIX C: GOVERNMENT INTERVENTION IN THE AREA OF MATERNAL HEALTH

The United States government enacted several programs for the promotion of maternal and infant health starting in the 1920s.

1921–1929 Maternity and Infancy Care (Sheppard–Towner) Act

The Sheppard–Towner Act was first enacted in 1921 as a 5-year program. It was extended in 1926 and finally repealed in 1929.[42] The act provided federal grants-in-aid to the states for the promotion of infant and maternal health. The main purpose of the act was education, although its implementation resulted in the development of full-time units for maternal and child health services, and of the first standardized training programs in this area. A secondary objective was to expand birth and death registration. Although repealed in 1929, the act set a pattern for state–federal cooperation that would reemerge for many other programs.[43] The response of the states to the availability of the federal funding via this legislation varied greatly. Many states did not accept the benefits of the act for several years, although all but three states eventually accepted the act by 1928 (Skopcol (1992) and Moehling and Thomasson (2009)). For the accepting states, the nature of the programs financed under the act and their geographical extension also varied.

Appropriations: Each state was granted outright $10,000 in 1922 and $5000 for each subsequent year. The remaining yearly apportionment of $1,000,000 was divided between the states based on population, on the condition that the states provided matching funds. A small budget was also reserved for the activities of the Children's Bureau, which was responsible for the review and approval of the state plans.

[42] Skopcol (1992) and Moehling and Thomasson (2009) discussed the political economy of the enactment and the repeal of the Sheppard–Towner Act.

[43] The Sheppard–Towner Act was not the first example of federal grants-in-aid to the states, although it was the first in the area of public health. See Skopcol (1992).

1935 Social Security Act

Title V, Part 1, of the Social Security Act, signed into law in August 1935, provided funding for medical care of mothers and infants. The administration of Title V was modeled on the Sheppard–Towner Act. The main difference, in addition to a doubling of appropriations, was the provision of medical and hospital services for mothers during labor and delivery (Lesser (1985)). Participating states were mandated to make diagnostic services available free of charge without requirement of economic status or legal residence. Eligibility for medical treatment could take into account family income and size, but also the diagnosis and the estimated cost of completed care. Means testing was typically not applied. Services were provided by participating physicians and hospitals, and by public health nurses, social workers, and nutritionists. The Children's Bureau set caps on reimbursed expenses based on the average costs for a hospital bed. Since the apportionment of funds was based on the states' financial needs, as well as on the number of live births, poorer states received more transfers. This system may have contributed to a convergence in maternal health outcomes across states (Schmidt (1973)).

There were three types of yearly appropriation. A uniform yearly apportionment of $20,000 was granted outright to each state, whereas a yearly appropriation of $1,820,000 was divided among the states based on the percentage of live births. An additional yearly appropriation of $980,000 was reserved for states experiencing financial need.

1943–1946 EMIC

The Emergency Maternity and Infant Care Program (EMIC), passed into law in March 1943, provided funds for maternity and infant care for the wives and infants of servicemen in the four lower pay grades. Medical, nursing, and hospital services for the prenatal period as well as delivery and 6 weeks of postpartum care were provided for these families at *no charge*, in addition to complete care for infants. States obtained federal funds based on need, and there was no means testing for participants. Yearly appropriations to the states were made based on the number of projected cases, with the possibility of deficiency appropriations. By the end of the program in 1946, approximately 1.25 million mothers and 230 thousand children received care. It was the largest public medical care program undertaken in the United States up to that time (Schmidt (1973)). The program was widely recognized for the reduction in maternal and infant mortality and for the rise in the number of births attended by trained medical personnel.

1946 Hospital Survey and Construction (Hill–Burton) Act

The objective of this legislation was to attain a ratio of 4.5 beds per 1000 population. Federal funding was provided on a grant-in-aid basis. Facilities receiving Hill–Burton were not allowed to discriminate based on race, color, national origin, or creed, and were required to provide a "reasonable" amount of uncompensated care each year for 20 years to local residents who could not afford to pay. These restrictions limited participation in some states. In 1975, the act was amended and became Title XVI of the Public Health Service Act.

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