

Maternal Health and the Baby Boom*

Stefania Albanesi[†]

Claudia Olivetti[‡]

Columbia University, NBER and CEPR Boston University and NBER

July 28, 2011

Abstract

U.S. fertility 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 U.S. and other countries that experienced similar improvements in maternal health.

JEL Classification: J11, J13, J24, N12, N3, N92

Keywords: Maternal mortality; Fertility choice; Baby boom; Human capital.

*We thank Jerome Adda, Joshua Aizenman, Alberto Alesina, Andy Atkeson, Hoyt Bleakley, Miriam Bruhn, Francisco Buera, Jesus Fernandez-Villaverde, Claudia Goldin, Jeremy Greenwood, Michael Haines, Christian Hellwig, Sok Chul Hong, Andrea Ichino, Nir Jaimovich, Larry Katz, Dirk Krueger, Adriana Lleras-Muney, Lee Ohanian, Nicola Pavoni, Valerie Ramey, Garey Ramey, Fabiano Schivardi, Michele Tertilt, Guillaume Vanderbroucke, and seminar participants at many institutions for helpful comments and suggestions. We are grateful to Maria J. Prados and Jodie C. Liu for outstanding research assistance and Junya Khan-Lang and Vighnesh Subramanyan who contributed to data collection. This work is supported by the National Science Foundation under Grant SES 0820135. Stefania Albanesi gratefully acknowledges financial support and hospitality from the Hoover Institution and EIEF while working on this project.

[†]Contact: Department of Economics, Columbia University, 420 West 118th Street, Suite 1022, New York NY 10027. Email: stefania.albanesi@columbia.edu.

[‡]Contact: Boston University, Department of Economics, 270 Bay State Road, # 302, Boston, MA, 02215. Email: olivetti@bu.edu.

1 Introduction

The United States experienced very big swings in fertility between the late 1930s and the early 1970s. The cohort total fertility rate¹ 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. 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. Pregnancy related deaths dropped from 1 for every 195 live births in 1936 to 1 for every 3,484 live births in 1956. 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 to 6 years over the same period.

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. We formalize this reasoning 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. 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 higher opportunity cost of children, they will have lower desired fertility than older women who experienced the decline having completed their education. The resulting boom-bust pattern in fertility qualitatively replicates the U.S. experience.

¹The 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, $CTFR_t = \sum_{a=15}^{a=49} f_{a,t+a}$. This measure is preferable to the more often used Period Total Fertility Rate (PTFR), defined as $PTFR_t = \sum_{a=15}^{a=49} f_{a,t}$, in time periods when total fertility changes across cohorts since it does not mix fertility behavior of different cohorts. The CTFR is shifted by 27 years to align its peak to the the PTFR. The CTFR underestimates completed fertility if maternal death risk is high. Both series are plotted in figure 1. See Jones and Tertilt (2007) for a discussion of alternative fertility measures.

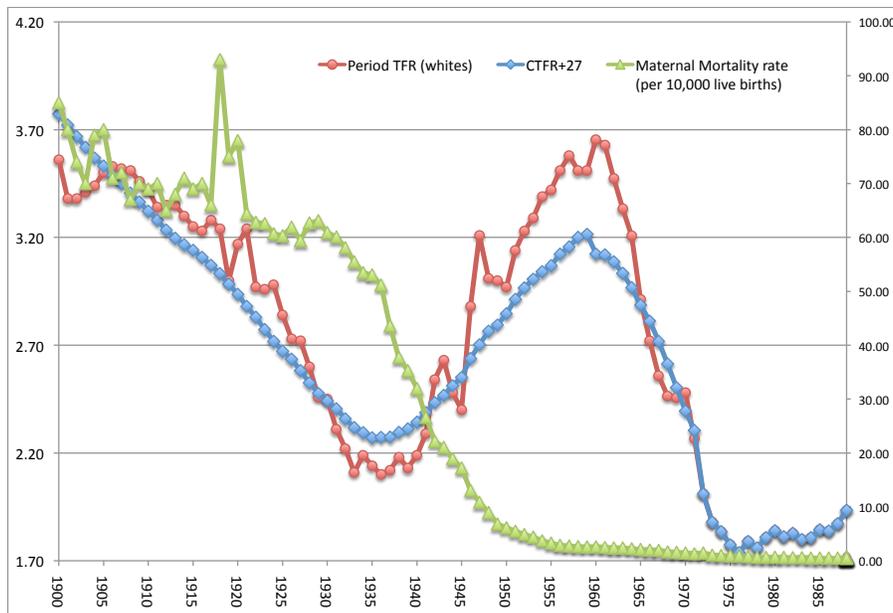


FIGURE 1: Maternal mortality, Period Total Fertility Rate and Cohort Total Fertility Rate (+27) in the U.S. 1900-1990

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. Period Fertility Rate in Haines (2006). Maternal mortality from Vital Statistics of the United States.

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 the effect of the decline on the change in completed fertility and educational attainment for different cohorts of women. Panel estimates suggest that a reduction in maternal mortality of 10 deaths per 10,000 live births is associated in a rise in completed fertility of 0.52 children for women born in 1921-1940 relative to those born in 1913-1920, about 36% 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 fully account for the baby boom.

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 the graduation rate by 0.024 for college and by 0.06 for high school for the 1933-1950 birth cohorts, from initial values of -0.0358 for college and 0.0542 for high school for the 1919-1932 cohorts. This accounts for 95% for college and 102% for high school of the actual rise in female graduation rates across these 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.

These results suggest that the decline in maternal mortality contributed significantly to the US 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, 2007), 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. In an important contribution, Greenwood, Seshadri and Vanderbroucke (2005) propose 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, 2008), may have increased the demand for home appliances.

Doepke, Hazan and Maoz (2007) argue 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), of the 80% of women who were not working in 1941, 14% were working in 1944 and only 46% of these were still in the labor force in 1951.² Moreover, Acemoglu, Autor and Lyle (2004) find 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) show that participation of mothers rose during the baby boom.

The 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 study of 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

²Table 19, Goldin (1991).

³Bleakley (2007) and Bleakley and Lange (2008) study the impact of malaria and hookworm eradication on fertility and schooling in the American South. They find a negative effect on fertility and a sizable positive effect on schooling.

the education of daughters. Our results confirm the strong impact of falling maternal mortality on women’s education for the U.S. 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. This suggests that medical progress, and the resulting decline in mortalities, can provide an integrated explanation for both the secular decline in fertility and the baby boom and bust, and 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 (2011) examines maternal mortality and fertility behavior in 25 advanced and emerging economies between 1900-2000 and finds 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) show 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 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 U.S. Section 3 presents a model of fertility choice with human capital investment to examine the impact of a decline in maternal mortality. Section 4 discusses the empirical analysis. Section 4.1 concentrates on the fertility response of women who had completed their education at the onset of the decline in maternal mortality. Section 4.2 is devoted to the response of female-male differentials in educational attainment for individuals in their formative years at the time of the decline. Section 4.3 examines the link between the baby bust and the decline in maternal mortality. Finally, Section 5 concludes.

2 Maternal Health in the US

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

2.1 Historical Background

Women were keenly conscious of the health risks associated with pregnancy and childbirth, yet only in the 1920s was maternal mortality treated as a major health problem in the U.S. (Leavitt, 1986). Even as mortality from other conditions rapidly declined in the first three decades of the twentieth century (see Table 1), 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).

In the U.S., efforts to improve maternal health were initially driven mainly by the goal of reducing infant mortality, which was very high, especially in urban areas (Meckel, 1990). At the center of those efforts was the Children’s Bureau, the first federal agency with the primary responsibility of promoting infant and child health, created by an act of Congress in 1912.⁴ The Children’s Bureau played a critical role in the decline of maternal mortality in the U.S. by raising awareness of the preventability of pregnancy-related mortality in the public and in the medical profession.

The first important milestone occurred in 1917, when the Children’s Bureau submitted a report to Congress on “Maternal Mortality from All Conditions Connected with Childbirth in the United States and Certain Other Countries” (Meigs, 1917). The main findings were 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 for maternal health among advanced nations. Following this report, the Children’s Bureau became the main sponsor and administrator of a series of key federal programs targeting maternal and infant health, which were introduced between 1921 and 1943. The most notable were the Sheppard-Towner Act of 1921-1929, which provided federal funding to the states for educational activities for the promotion of maternal and infant health, and the Social Security Act of 1935. Title V, Part 1 of this act provided federal funding to the states, on a grant-in-aid basis, for subsidies to obstetric and infant care. Finally, the Emergency Infant and Maternal Care program provided full coverage for obstetric and infant care for the wives and children of servicemen between 1941 and 1946. A brief description of these programs, including the criteria for appropriation, is provided in Appendix D.

While the federal programs were mainly aimed at increasing public education on maternal health and improving access to obstetric care, additional initiatives were targeted to medical professionals. Physicians began to enter the birth room in the 1850s, though due to poor training and inappropriate and excessive operative procedures, they did not initially contribute to a decline in maternal mortality (Loudon, 1992a).⁵ The iatrogenic nature of maternal mortality received widespread public attention following the publication of a number of reports suggesting that more than two-thirds of all maternal deaths were preventable and that many physicians were found to lack the most basic

⁴For a detailed account of the establishment of the Children’s Bureau, see Schmidt (1973), Parker and Carpenter (1981) and Skopcol (1992). The Children’s Bureau was modeled on New York City’s Department of Child Hygiene, the first of its kind, founded in 1908. For a full account see: <http://www.ssa.gov/history/childb1.html>

⁵Thomasson and Treber (2008) show that the hospitalization of childbirth had a positive effect on maternal mortality between 1927 and 1937 in the US. Loudon (1992a) shows that, in England, high income mothers, who more often gave birth attended by a physician, had higher pregnancy-related mortality rates than low income mothers.

obstetric knowledge (CDC, 1999).⁶

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 were set up in the 1930s to prevent hospitals from accepting unqualified specialists. Hospital and state maternal mortality review committees also were established in the 1930s and 1940s and contributed to the improvement of obstetric care in hospitals.

As can be seen from figures 1 and 2, all causes of maternal mortality started to decline sharply in 1936. This year marks the introduction of sulfonamides, the first type of antibiotic. These drugs were relatively cheap to produce and diffused very rapidly, bringing down mortality for 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) also contributed to the decline in maternal mortality due to sepsis. 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.⁷

The year 1936 was also significant as it marks 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 (Albanesi, 2010a). As the quality of obstetric care began to improve in the early 1930s, access was still severely limited due to high costs.⁸ 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).⁹ The development of the first Blue Cross hospital pre-payment plans starting in the

⁶The 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, convened by President Herbert Hoover and sponsored by the Children’s Bureau. Similar findings emerged from a study of 2,041 maternal deaths in childbirth by the New York Academy of Medicine, also published in 1933.

⁷At 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.

⁸Geographical distance was also a factor in rural areas prior to the widespread use of automobiles.

⁹The 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 ranged from \$20-30, and the patient would have to hire a nurse for all subsequent attention, typically doubling the cost. By 1930, the cost for a midwife had risen to \$25-30, and the cost for doctors to \$65. The cost for a specialist was \$75, and did not include the cost of any supplies required to provide care. See Litoff (1986) and Wertz and Wertz (1977) for a discussion.

late 1920s and 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.

2.2 Advances in Maternal Health

The maternal mortality ratio (MMR),¹⁰ 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.¹¹ Maternal mortality was the second largest cause of death for women after tuberculosis, the leading cause of death for both men and women 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 age 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 4.5% in this period.¹² Maternal deaths as a fraction of all female deaths declined from 3.1% to 1.6% between 1900 and 1930, though this change is mostly accounted for by the decline in births.¹³ In 1930, maternal mortality still accounted for 10.6% of female deaths at age 15-44.¹⁴

The year 1936 marks the start of a precipitous decline of maternal mortality in the U.S. 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 twenty 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.¹⁵ The most striking decline

¹⁰According 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.

¹¹For comparison, the mortality rate for heart disease, the first cause of death in the U.S., was 211.0 deaths per 100,000 population in 2006 (Center for Disease Control and Prevention, 2010).

¹²This 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 2.1 for further discussion.

¹³The 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, 1.58 for parities 1 to 8, respectively. Dublin (1936) estimates 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.

¹⁴Maternal 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.

¹⁵The 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%).

TABLE 1: Incidence of Maternal Mortality

| | | <i>Death Rates per 100,000 population, All ages</i> | | | <i>Percentage changes</i> | |
|---|-------|---|--------|--------|---------------------------|-----------|
| | | 1900 | 1930 | 1960 | 1930-1900 | 1960-1930 |
| All Causes | men | 1791.1 | 1225.3 | 1104.5 | -31.6% | -9.9% |
| | women | 1646.9 | 1036.7 | 809.2 | -37.1% | -21.9% |
| Tuberculosis | men | 201 | 76.2 | 8.9 | -62.1% | -88.3% |
| | women | 187.8 | 65.9 | 3.3 | -64.9% | -95.0% |
| Maternal Causes | | 26.9 | 25.7 | 1.7 | -4.5% | -93.4% |
| Female-Male Differential in Life | | | | | | |
| Expectancy at Age 20 | | 2 | 2.5 | 6.1 | 25.0% | 144.0% |
| <i>Deaths by cause as a percentage of all deaths</i> | | | | | | |
| Maternal deaths as a percentage of all deaths, Women age 15-44 | | 14.9% | 10.6% | 0.7% | | |
| Maternal deaths as a percentage of all female deaths | | 3.2% | 1.6% | 0.1% | | |
| Tuberculosis as a percentage of all deaths | | 11.3% | 6.3% | 0.7% | | |

Source: Vital Statistics of the United States

occurs for deaths due to sepsis, which dropped from 27.5 in 1923 to 0.55 per 10,000 live births in 1955.

The decline in maternal mortality was associated with a sizable rise in the female-male differential in adult life expectancy,¹⁶ which, as can be seen in Table 1, rose from 2.5 to 6.6 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 20-39.¹⁷

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 post-partum 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.¹⁸ Franks et al. (1992), the only comprehensive nationwide assessment of pregnancy related morbidity, report

¹⁶The female-male differential in life expectancy was negative until early years of the 20th century. Stolons (1956) argues 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 19th 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.

¹⁷Rethereford (1972) concludes that the gender difference in cigarette smoking is the main determinant of the evolution in the female-male differential in mortality rates and life expectancy at ages greater than 40 for this same period.

¹⁸There are still no generally accepted criteria for the measurement of maternal morbidity, as well as significant obstacles to data collection in this area (Wilcox and Marks, 1994).

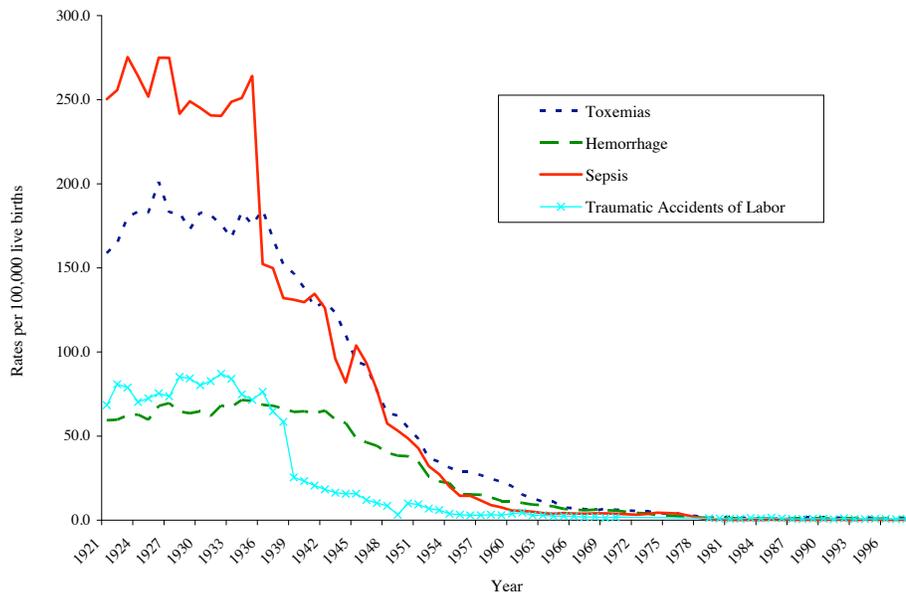


FIGURE 2: Maternal mortality by cause
Source: Vital Statistics of the United States

an annual rate of pregnancy-related post-partum morbidity requiring hospitalization of 8.1 per 1,000 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 1,000 deliveries.¹⁹ Thus, post-partum 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.²⁰

3 Theory

We now examine a model of fertility choice that explicitly incorporates maternal mortality. The model predicts that a decline in maternal mortality is associated to a temporary rise in fertility and a permanent rise in women’s human capital. These predictions will provide a conceptual framework for the empirical analysis.

We begin to illustrate the effect of pregnancy-related mortality risk on fertility choice and human capital investment in a simple model inhabited only by women, that is, all adults are female and all offspring are female. This framework, though clearly not realistic, captures the essential forces shaping fertility decisions, and the incentives to invest in daughters’ human capital. Appendix B.2

¹⁹This 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.

²⁰Weil (2004) offers an excellent discussion of this approach.

analyzes a version of the model with adults and children of both genders, which delivers the same results. Since we are interested in fertility and human capital investment decisions, income and the returns to human capital are exogenous in the model. Thus, the model does not consider the equilibrium effects of changes in fertility on the returns to human capital.

Adult women are endowed with a given level of human capital and live for one period. They derive utility from consumption and from the quantity and quality of their daughters. The daughters' quality depends on their own human capital, which is chosen by the mother. Mothers may die in childbirth. Their mortality risk is a function of the maternal mortality rate, which they take as given, and of the number of births.

Mothers and daughters face the same decision problem, given their endowment of human capital, though they may experience different values of the maternal mortality probability. Mothers have perfect foresight on the value of all exogenous variables entering their daughters' decision problem, and they take as given their daughters' decisions. Thus, women's decision problem can be formulated recursively with human capital serving as a state variable.

The decision problem is represented by the following Bellman equation:

$$U(e; \mu) = \max_{e' \geq 0, b \geq 0, c \leq w(1+\varepsilon)e} \{-v(b, e') + (1 - \mu b)u(c) + \kappa(sb)U(e'; \mu')\},$$

where $e \geq 0$ represents the mother's endowment of human capital and e' is her investment in her daughters' human capital, b is the number of births, and $U(e; \mu)$ is the mothers' value function, which is parametrized by $\mu \in [0, 1]$, the probability of maternal death associated with each birth.

The function $v(\cdot)$ is the utility cost of parental investment in children's human capital, which depends on the number of children. The function $v(\cdot)$ is twice continuously differentiable, strictly increasing in both arguments and convex.²¹

The function $u(\cdot)$ is the utility from mothers' consumption, c , which, through the budget constraint, depends on baseline income, w , and their endowment of human capital e . The parameter $\varepsilon \geq 0$ is the return to human capital investment. Mothers with higher human capital enjoy higher utility from consumption if they survive childbirth. We assume $u(\cdot)$ is twice continuously differentiable, with $u(\cdot) > 0$, $u'(\cdot) > 0$ and $u''(\cdot) \leq 0$. The term $(1 - \mu b)$ corresponds to the probability that a woman will survive childbearing. Implicitly, we are assuming that mothers do not obtain any utility from consumption if they die in childbirth.

The parameter $s \in (0, 1]$ denotes the youth survival probability, thus, sb is the number of children surviving to adulthood. The function $\kappa(\cdot)$ corresponds to the Barro-Becker dynastic discount factor. By assumption, $\kappa(\cdot)$ is twice continuously differentiable, with $\kappa(\cdot) \in [0, 1]$, $\kappa'(\cdot) > 0$ and $\kappa''(\cdot) \leq 0$ and $\lim_{x \rightarrow 0} \kappa'(x) = +\infty$. The functions $v(\cdot)$, $u(\cdot)$ and $\kappa(\cdot)$.

The value function for daughters' problem, $U(e'; \mu')$, corresponds to child quality in this model. It depends on the daughter's human capital, and we also index it by the maternal death probability,

²¹The cost of investment in children's human capital is modeled as a component of utility for analytical simplicity. The results derived below also apply for a version of the model in which this cost is monetary.

μ' , which may vary over time.²² Under the stated assumptions, $U'(e; \mu) > 0$ and $U''(e; \mu) \leq 0$. We further restrict $u(\cdot)$ and $v(\cdot)$ to ensure $-v(b, e') + (1 - \mu b)u(c) \geq 0$ for all $e, b \geq 0$ and $\mu \in [0, 1]$, which implies $U(e; \mu) > 0$. In addition, following Alvarez (1999), we will impose the following:

Assumption 1 *Let $V(b, e') := -v(b, e') + \kappa(sb)U(e'; \mu')$ is strictly concave in $\{b, e'\}$.*

Assumption 1²³, jointly with the assumptions on $v(\cdot)$, $\kappa(\cdot)$ ²⁴ and the resulting properties of $U(\cdot; \mu)$ implies that the Hessian of $V(b, e')$ is negative definite. This restriction is crucial for the response of fertility and human capital to changes in maternal mortality.

The first order necessary conditions for the mothers' problem are:

$$-v_b(b, e') + \kappa(sb)U'(e'; \mu') \leq 0, \quad (1)$$

with equality for $e > 0$, and

$$-v_{e'}(b, e') - \mu u((1 + \varepsilon e)w) + \kappa'(sb)sU'(e'; \mu') = 0, \quad (2)$$

since Inada condition on the utility from children implies that $b > 0$ at the optimum. The envelope condition is:

$$U'(e; \mu) = (1 - \mu b)u'(w(1 + \varepsilon e))w\varepsilon. \quad (3)$$

These optimality conditions implicitly define the policy functions for desired fertility, $b(e; \mu)$, and investment in the daughters' human capital, $e'(e; \mu)$. Equation (1) clearly implies that parental investment in daughters' human capital, e' , is increasing in youth survival probability, s , and in the daughters' baseline income, w' , return to human capital investment, ε' , and decreasing in the daughters' pregnancy-related mortality risk, for given b . Also, if the returns to human capital investment and the baseline wage for the daughters are low enough and their maternal mortality risk is high enough, the solution is $e' = 0$.

Equation (2) lays out the trade-off associated with an additional birth for given e' . The first term corresponds to the marginal increase in the utility cost of human capital investment. The second term is the loss in expected utility due to the fact that the pregnancy related death risk rises with each birth, while the last term is the expected marginal value of an additional child for the mother. Clearly, a higher maternal mortality risk, μ , reduces the optimal number of births for given e by this equation. A higher value of human capital, e , the returns to human capital investment, ε , or baseline income, w , for the mother also reduces the optimal number of births, other things

²²The youth survival probability, s , baseline income and the returns to human capital also potentially vary across cohorts, but since our focus is on maternal mortality, we omit indexing the value function on these parameters to simplify the notation.

²³The value function $U(e; \mu)$ is not a primitive of the decision problem. Assumption 1 can be stated in terms of primitives with additional restrictions on the functional forms of $v(\cdot)$, $u(\cdot)$, and $\kappa(\cdot)$. Since we are interested in qualitative predictions, we maintain a general specification for these functions.

²⁴Since the expected utility from consumption is linear in births, b drops out of all second order derivatives relevant for the predictions derived below.

equal. Finally, higher child quality, which in the model corresponds to a higher value of $U(e'; \mu')$, and higher youth survival probability, s , also increase desired fertility.

We now derive two properties of the model that give rise to predictions for the effect of a decline in maternal mortality on fertility and human capital of women at different stages of the life cycle. The first is the negative relation between desired fertility and maternal mortality. This property is intuitive, given that higher pregnancy-related mortality probability, μ , increases the loss in the expected utility from consumption associated with a rise in the number of births. The second property is the negative relation between desired fertility and mothers' endowment of human capital. This property stems from the fact that, as long as maternal mortality risk is positive, a rise in the number of births reduces the probability of enjoying consumption, and the resulting loss in welfare is greater for mothers endowed with higher human capital. 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. Fertility rises for the women that experience the decline in childbearing years, after their endowment of human capital has been chosen. Younger women, who experience the maternal mortality decline in their formative years, will be endowed with higher human capital, which increases their opportunity cost of children. Their desired fertility will thus be lower than for the initially exposed women.

We present these results in two propositions. Proposition 1 derives the effect of a permanent decline in pregnancy-related mortality.

Proposition 1 *Assume that pregnancy-related mortality risk is the same for mothers and daughters, so that $\mu = \mu'$, and that it changes permanently starting with the mother's generation. Then, under Assumption 1, the optimal response of births and parental investment in human capital satisfies:*

$$\frac{\partial b}{\partial \mu} \leq 0, \quad (4)$$

$$\frac{\partial e'}{\partial \mu} \leq 0, \quad (5)$$

if and only if:

$$[-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu')] \geq 0. \quad (6)$$

Proof: In Appendix B.1. ■

Proposition 1 states that fertility and mothers' investment in daughters' human capital rise in response to a reduction in the maternal mortality probability when condition 6 holds. This condition states that the cross-partial derivative of a mother's lifetime utility with respect to b and e' is non-negative, implying that the increase in welfare resulting from a marginal rise in investment in daughters' human capital grows with the number of births. To interpret this condition, note that the marginal benefit of an additional birth is always increasing in daughters' human capital, that is $\kappa'(sb)sU'(e'; \mu') > 0$, under the baseline assumptions. Thus, condition (6) restricts the sign of the term $v_{be'}(b, e')$, which is not pinned down by the convexity of $v(\cdot)$. If $v_{be'}(b, e') \leq 0$, condition

(6) is automatically verified. However, this assumption is unrealistic, since e' represents the level of human capital of all daughters, and presumably, the costs of attaining that level are increasing in the number of children. If $v_{be'}(b, e') > 0$, there is a trade-off between *quality* (that is human capital) and *quantity* of children. Condition (6) restricts the severity of this trade-off. Given that the dynastic discount factor $\kappa(\cdot)$ is concave and satisfies the Inada conditions, this restriction will be always satisfied if initial fertility is low enough. Condition 6 is also more likely to be satisfied for a high value of s .

To summarize, desired fertility and daughters' human capital investment rise in response to a permanent reduction in maternal mortality, as long as the marginal value of parental investment in children's human capital is not decreasing in the number of children.

We now consider the sensitivity of desired fertility and investment in daughters' human capital to the mothers' endowment of human capital for given pregnancy-related mortality.

Proposition 2 *Assumption (1) implies:*

$$\frac{\partial b(e; \mu)}{\partial e} \leq 0. \quad (7)$$

If, in addition, condition (6) holds, then:

$$\frac{\partial e'(e; \mu)}{\partial e} \geq 0. \quad (8)$$

Proof: In Appendix B.1. ■

Proposition 2 establishes that desired fertility falls with a mother's endowment of human capital, just by the joint concavity of $V(b, e')$. The inequality in (7) is strict provided the pregnancy-related mortality probability is strictly above zero. This property derives from the fact that, for given μ , an increase in the number of births reduces the probability that the mother will enjoy utility from consumption. The corresponding loss in welfare is increasing in the mother's human capital. It is straightforward to show that desired fertility is also decreasing in baseline income, w , and the returns to human capital investment, ε . These results imply that the model replicates the negative empirical relation between mother's income and fertility (Jones and Tertilt, 2007).

Investment in daughters' human capital can grow or fall with a mother's human capital in general, since higher maternal human capital generates an increase in the demand for child quality but produces a negative income effect on maternal investment in daughters' education.²⁵ Condition (6) is necessary and sufficient for investment in daughters' human capital to increase with mothers' human capital endowment.

²⁵This property would also hold if investment in daughters' human capital entailed a monetary cost, instead of a utility cost.

3.1 Discussion

Propositions 1 and 2 taken together deliver a set of predictions for the response of fertility and women’s human capital to a permanent decline in maternal mortality under condition (6). By Proposition 1, women who experience a permanent decline in pregnancy-related mortality in childbearing years increase their desired fertility and their investment in children’s human capital. Younger women who experience this decline in formative years will benefit from greater parental investments in human capital and, by Proposition 2, they 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. Proposition 2 also implies that a decline in maternal mortality causes a permanent rise in women’s human capital. The effect on fertility once the advances in maternal health are exhausted may well be negative, if the returns to human capital are high enough.

Condition (6) is more likely to hold if initial fertility is low, since $\kappa'(sb)$ is decreasing in the number of births. This was the case in the US, where in the early 1930s fertility reached a historical low. High values of the youth survival probability, s , also contribute to Condition (6) being satisfied, since they increase the marginal benefit of investment in children’s human capital. Finally, higher values of baseline income, w , and greater returns to human capital, ε , increase the value of $U'(e'; \mu')$ and make it more likely that Condition (6) will hold. Thus, Propositions 1 and 2 imply that the drop in pregnancy-related mortality will more easily generate a rise in fertility on impact and a rise in women’s human capital in economies that have experienced a fertility transition, and have low fertility and high youth survival probability, and have attained sufficiently high levels of income and returns to human capital.²⁶

Agents have perfect foresight in the model. In practice, there may have been considerable delays in the diffusion of information on improvements in pregnancy-related outcomes, as well as uncertainty on whether these developments were indeed permanent. In Appendix B.1.2, we also derive the response of desired fertility and human capital investment to a temporary decline in the pregnancy-related mortality probability. We show that desired fertility and human capital investment rise in response to a decline in pregnancy-related mortality limited to either the mothers’ or the daughters’ generation, though in this case the rise in women’s human capital is not permanent. The second case captures the response of women who experience the decline in maternal mortality in their formative years, which allows their parents to adjust their investment in human capital. These results suggest that the qualitative predictions of the theory do not hinge on the perfect foresight assumption. The model can also be adapted to allow for delays in the diffusion of information, without consequence for the qualitative predictions derived above.

Fertility also responds to changes in the youth survival probability, s . In Appendix B.1.3, we show that under Condition (6) fertility declines if youth survival probability increases, provided initial fertility is high enough, and that if fertility declines, then investment in children’s human capital rises. This property implies that the model is consistent with the historical experience of

²⁶Albanesi (2010b) assesses these predictions with international panel data on maternal mortality, infant mortality and fertility in the period 1900-2000 and finds that they are consistent with the historical experience.

the U.S. and other advanced economies, where a reduction in youth mortalities starting in the mid 1850s was associated with a decline in fertility (Preston, 1978, Haynes and Preston, 1991). Infant mortality continued to decline gradually in the U.S. during the course of the twentieth century, and we will examine its relation with fertility in our empirical analysis.

The simple model discussed in this section only features mothers and daughters. Appendix B.2 presents a general version of the model in which households are comprised of mothers and fathers. Couples choose the number of births and have daughters and sons in equal number. The dynastic discount factor is defined over the total number of surviving children. As in the basic model, mothers enjoy utility from consumption only if they survive childbirth. Parents can choose different levels of human capital for daughter's and sons. Thus, the state variable for the household problem is given by the endowment of human capital of the mother and the father, $\{e_f, e_m\}$, where f stands for female and m for male, and the vector of controls by $\{b, e'_f, e'_m\}$. As for the basic model, we show that concavity of the household welfare function in $\{b, e'_f, e'_m\}$ and a version of condition (6) guarantee that fertility increases in response to a permanent decline in maternal mortality for the initially exposed cohort, and daughter's human capital rises. Moreover, concavity of household welfare in $\{b, e'_f, e'_m\}$ guarantees that desired fertility is lower for households with higher endowment of maternal human capital. These properties imply that a permanent decline in pregnancy-related mortality risk generates a boom-bust response in fertility and a permanent rise in women's human capital.²⁷

This framework abstracts from the health burden on pregnancy-related morbidity conditional on survival, which, as discussed in Albanesi and Olivetti (2009), took a very significant toll on women's ability to participate in market work, as well as their quality of life. The model can easily be extended to accommodate this feature. If the utility cost of pregnancy-related maternal morbidity is separable from the utility from consumption, the predictions of the model remain intact²⁸.

Improved maternal health also influences the demand for children via additional channels. For example, the children's utility may be higher if the mother survives. Extending the model to allow for this feature would preserve the qualitative predictions discussed above. An additional effect of improved maternal health is to extend the length of the fecund period and to allow for decreased spacing of births. These effects on the timing of fertility cannot be analyzed in the current model given that there is only one stage in life.

²⁷A decline in youth mortality causes a decline in fertility and a rise in investment in human capital for both sons and daughters.

²⁸The women's problem with a pregnancy-related health burden conditional on survival can be represented as follows:

$$U(e; v) = \max_{e' \geq 0, b \geq 0} \{-v(be') - h(\phi b) + (1 - vb)u(w(1 + \varepsilon e)) + \beta \kappa(sb)U(e'; v')\},$$

where the parameter ϕ represents the health burden per birth, and $h(\cdot)$ is a strictly increasing and weakly convex function. With this formulation, it is straightforward to show that a permanent decline in the health burden increases desired fertility.

4 Empirical Analysis

We now proceed to examine the empirical link between the decline in maternal mortality, fertility and women’s human capital.

As discussed in Section 2.2, maternal mortality did not decline substantially until the mid 1930s, when it started to drop sharply, 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, the maternal mortality ratio had converged to uniformly low levels in all states, as can be seen in figure 3. 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.

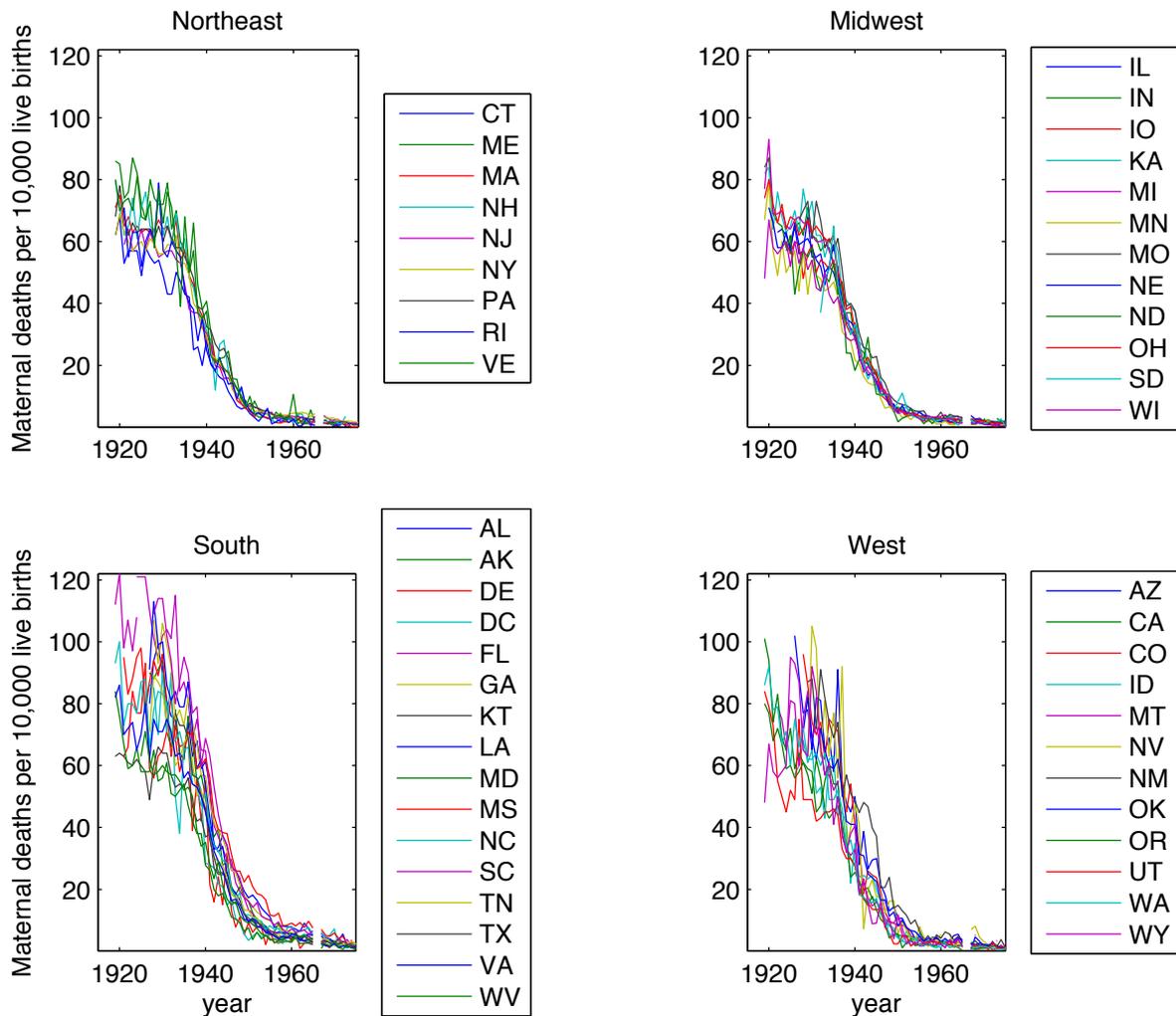


FIGURE 3: Evolution of maternal mortality in the U.S.

Source: Vital Statistics of the United States

The cross state variation in initial maternal mortality is indeed sizable. As can be seen in figure 3, the Southern and Western states display considerably higher maternal mortality rates between 1900 and 1930, 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 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.

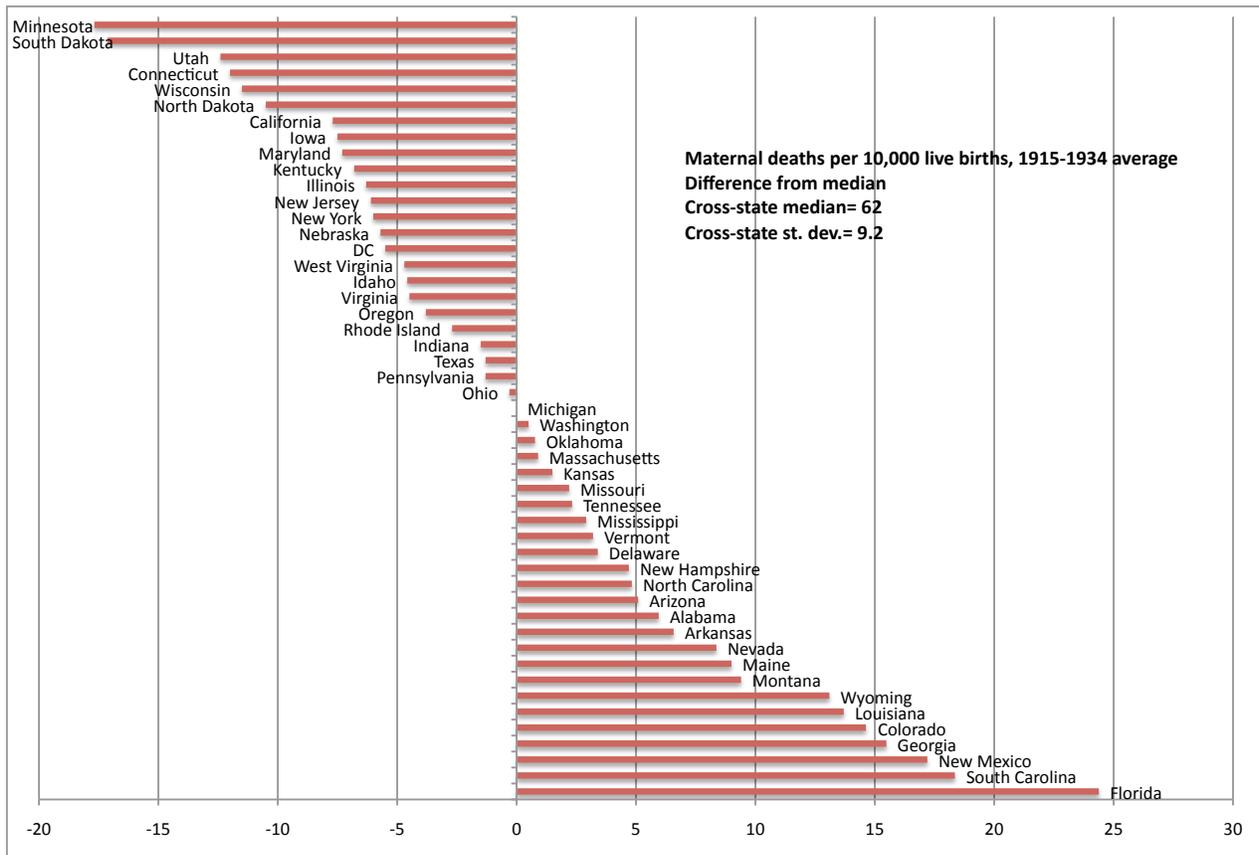


FIGURE 4: Cross-state variation in maternal mortality 1915-1934
 Source: Vital Statistics of the United States

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, the time variation in fertility is similar across states. All states experience a decline in fertility

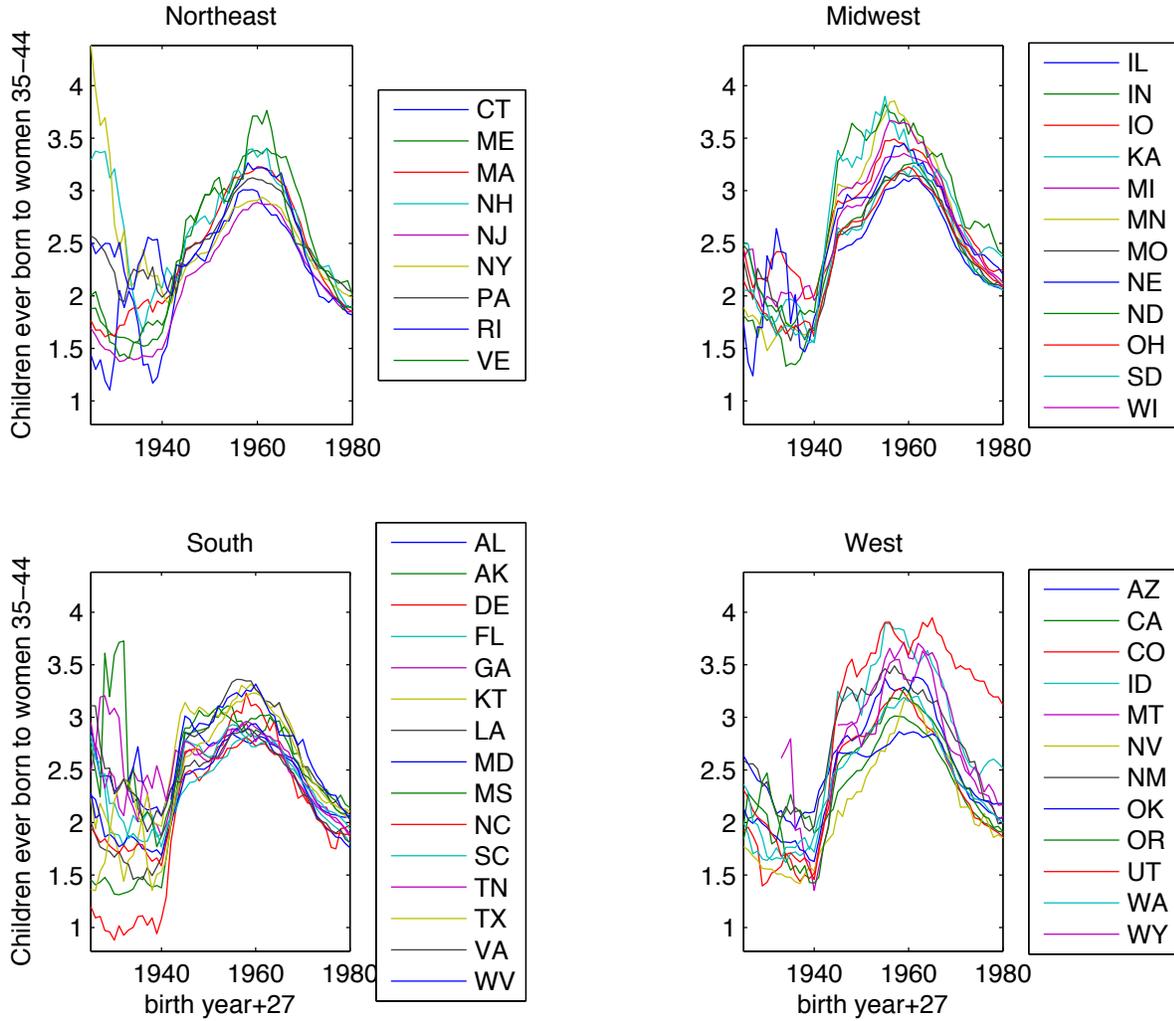


FIGURE 5: Evolution of completed fertility in the U.S.
 Children Ever Born at age 35-44 by birth cohort, shifted by 27 years. Source: IPUMS

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

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. We treat the drop in maternal mortality as a quasi-experiment and we interpret the cross-state variation in initial maternal mortality as exogenous.²⁹ The estimation is based on a panel approach, where the depend 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

²⁹We discuss and assess the validity of this assumption in the sensitivity analysis.

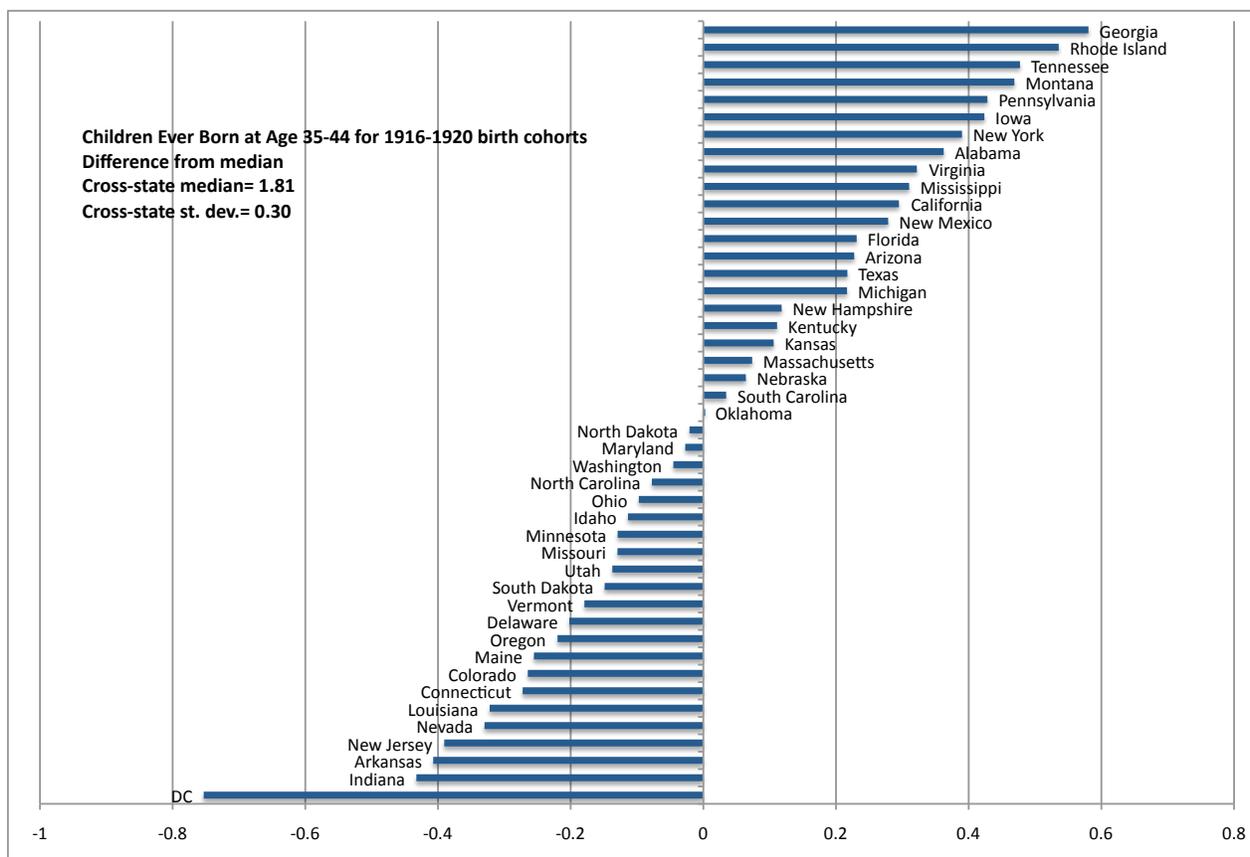


FIGURE 6: Cross-state variation in completed fertility 1932-1937
 Children Ever Born at age 35-44 by birth cohort, shifted by 27 years. Source: IPUMS

identifies the impact of the maternal mortality decline on the outcomes of interest. In order to attain a homogeneous sample, we restrict attention to white women living in non-farm households.³⁰

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 twenty years, its effect will be identified from a comparison of fertility and education for different birth years, rather than rely on a strict definition of treatment

³⁰We 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. Fertility for farm-households was initially greater than for non-farm households, though this differential declined over time. The percentage of farm households varied across states and declined for later cohorts. By contrast, there were no systematic differences in maternal mortality for urban and rural women (Loudon, 1992b).

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 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.

4.1 Fertility

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

$$Y_{st} = \alpha_0 + \alpha_1 ZZ_{st} + \mu_s + \delta_t + \beta X_{st} + \epsilon_{st}, \quad (9)$$

where Y_{st} denotes the fertility outcome for birth year t and state s . Only females are included in the analysis. The variable ZZ_{st} is the measure of the treatment, the variable X_{st} denotes a set of controls, while μ_s and δ_t correspond to state and cohort effects.

The baseline specification adopts *reference maternal mortality*, defined as the average maternal mortality rate in the state at age 15-20 for each cohort, as a measure of the treatment:

$$ZZ_{st} = MMR_{st}^{ref}, \quad (10)$$

where MMR_{st}^{ref} is reference maternal mortality for state s and cohort t . The choice of age range for reference maternal mortality is motivated by the fact that the average age of first birth was well above 20 for the cohorts we are interested in³¹, thus ensuring that the fertility behavior of the women included in the estimation does not affect their reference maternal mortality.

The baseline specification includes women born in 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, though 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 years in the analysis.³²

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, $X_{st} = IMR_{st}^{ref}$,

³¹Specifically, the average age of first birth 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.

³²Data 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.

where we define reference infant mortality as the mean infant mortality rate in the state at age 15-20 for each cohort. Progressively, we include a set of state level controls for possibly cohort specific economic, demographic, health, political and cultural indicators, which we describe in Section 4.1.2.

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 years $t' > t$ cohorts:

$$Y_{st'} - Y_{st} = \alpha_1 (ZZ_{st'} - ZZ_{st}) + \delta_{t'} - \delta_t + \beta (X_{st'} - X_{st}).$$

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. We adopt the statistic children ever born (CHBORN) at age 35-44 from the US Census³³ as the main fertility measure, since the median age of last birth for the cohorts included was 29. For robustness, we also consider number of children under 13 living in the household (NCHILD) at age 35-44, though this measure may be biased downwards due to grown children having left the household. For some specifications, we also consider the number of children under 5 living in the household (NCHLT5) at age 23-32.

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

Table 2 presents summary statistics for the variables included in the baseline specification, for women born in 1913-1920 and women born in 1921-1940. 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. CHBORN for married women rises from 2.56 for those born in 1913-1920 to 4.02 for those born in 1921-1940.

³³Appendix A provides a detailed description of the data.

TABLE 2: Fertility: Summary Statistics

| Birth Years 1913-1920 | | | | | | | | | |
|------------------------------|---------------------|---|---|---------------------|---------------------|---|---------------------|---------------------|---|
| | | Reference Maternal Mortality (Age 15-20) | | | | Reference Infant Mortality (Age 15-20) | | | |
| | | Mean | | St. Dev. | | Mean | | St. Dev. | |
| | | 53.941 | | 7.9609 | | 54.923 | | 13.1061 | |
| Fertility Outcome Age | CHBORN 35-44 | CHBORN 35-44 | CHBORN 35-44 Married with Children | NCHILD 35-44 | NCHILD 35-44 | NCHILD 35-44 Married with Children | NCHLT5 23-32 | NCHLT5 23-32 | NCHLT5 23-32 Married with Children |
| Sample Mean | 2.5584 | 2.4085 | 2.8232 | 1.9988 | 1.8258 | 2.4517 | 0.7344 | 0.5701 | 1.022 |
| St. Dev. | 0.2044 | 0.2469 | 0.1966 | 0.2603 | 0.2562 | 0.2777 | 0.2052 | 0.1681 | 0.2715 |
| Birth Years 1921-1940 | | | | | | | | | |
| | | Reference Maternal Mortality (Age 15-20) | | | | Reference Infant Mortality (Age 15-20) | | | |
| | | Mean | | St. Dev. | | Mean | | St. Dev. | |
| | | 13.944 | | 2.8745 | | 32.7648 | | 7.1215 | |
| Fertility Outcome Age | CHBORN 35-44 | CHBORN 35-44 | CHBORN 35-44 Married with Children | NCHILD 35-44 | NCHILD 35-44 | NCHILD 35-44 Married with Children | NCHLT5 23-32 | NCHLT5 23-32 | NCHLT5 23-32 Married with Children |
| Sample Mean | 4.0172 | 3.8439 | 4.0688 | 2.4144 | 2.2378 | 2.736 | 0.9892 | 0.8629 | 1.1903 |
| St. Dev. | 0.2862 | 0.352 | 0.2373 | 0.2459 | 0.2778 | 0.1966 | 0.1087 | 0.1009 | 0.1277 |

4.1.1 Baseline Results

Table 3 presents the estimation results for the baseline specification in the top panel (heading “Panel”). The baseline estimates suggest that the decline in maternal mortality had a strong positive effect on fertility. For CHBORN at age 35-44 for the sample of married women, the estimated coefficient, which is significant at the 1%, suggests that a decline in reference maternal mortality equal to 10 deaths per 10,000 live births is associated with a rise in CHBORN of 0.52 or 20%. The coefficient on infant mortality is positive, consistent with a negative relation 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 1,000 live births is associated with a decline in fertility of 0.20 children per woman.³⁴

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 the estimates is 2.09 children per woman. Reference infant mortality declined by 22.16 deaths per 1,000 live births for the 1921-1940 cohorts relative to the 1913-1920 cohorts, with an associated decline in fertility of -0.47 children per woman based on the estimates. Thus, the predicted change

³⁴The inclusion of infant mortality does not affect the estimated coefficient for maternal mortality.

in fertility between the 1921-1940 and the 1913-1920 cohorts resulting from the combined decline of maternal and infant mortality is 1.62 children per woman. The actual change in the cross-state average of CHBORN between the 1921-1940 and the 1913-1920 birth years was 1.46 children per woman. These results suggest that the change in maternal mortality and in infant mortality fully account for the change in fertility across these cohorts.

The results for all and married with children women are consistent with those for the married sample. For married women with children, the estimated coefficient drops in magnitude though it is still highly significant. A decline in reference maternal mortality of 10 deaths per 10,000 live births is associated with a rise in fertility of 0.29 or 10%. Thus, the overall decline in maternal mortality accounts for 94% of the rise in fertility between the 1921-1940 and 1913-1920 birth cohorts.

Results are similar for the other measures of fertility. For NCHILD at age 35-44 for the sample married women, based on the estimates a decline in maternal mortality of 10 deaths per 10,000 live births is associated with a 0.26 or 13% rise in the number of children in the household, which accounts for 65% of the actual rise in this statistic between the 1921-1940 and the 1913-1920 cohorts, with the coefficient is significant at the 1% level. For NCHLT5 at age 23-32, a 10 death decline in maternal mortality is associated with a 0.09 or 13% rise for married women, which accounts for 34% of the change in this variable between treated and untreated cohorts. Similar results obtain for the sample of all and married with children women.

TABLE 3: Fertility: Regression Results

| Specification | | Panel, Birth Years 1913-1940 (1) | | | | | | | | |
|---------------------------|--------|----------------------------------|-----------------|---|-----------------|-----------------|---|-----------------|-----------------|---|
| Fertility Outcome | Age | CHBORN 35-44 | CHBORN 35-44 | CHBORN 35-44 Married with children | NCHILD 35-44 | NCHILD 35-44 | NCHILD 35-44 Married with children | NCHLT5 23-32 | NCHLT5 23-32 | NCHLT5 23-32 Married with children |
| Sample | | Married | All | | Married | All | | Married | All | |
| Constant | | 40.1315 | 31.9211 | -54.1319 | 82.0808 | 84.8013 | 88.7148 | 22.5779 | 25.461 | 20.3772 |
| | t-stat | 2.1925 | 1.8691 | -2.0509 | 8.0239 | 8.9277 | 9.2827 | 3.571 | 4.667 | 2.591 |
| $MMR_{st}^{ref} (2)$ | | -0.0523 | -0.049 | -0.0291 | -0.0257 | -0.0254 | -0.025 | -0.0089 | -0.011 | -0.0052 |
| | t-stat | -12.9721 | -13.0167 | -4.9969 | -11.4064 | -12.1148 | -11.8614 | -6.3752 | -9.1696 | -2.9991 |
| $IMR_{st}^{ref} (3)$ | | 0.0197 | 0.017 | 0.0146 | 0.0052 | 0.0038 | 0.0069 | -0.0013 | -0.0003 | -0.0029 |
| | t-stat | 3.7436 | 3.4659 | 1.9254 | 1.7851 | 1.3798 | 2.523 | -0.734 | -0.2007 | -1.3059 |
| Adj R-squared | | 0.4682 | 0.5151 | 0.252 | 0.2737 | 0.3405 | 0.2215 | 0.2649 | 0.3444 | 0.1857 |
| Specification | | Panel, Birth Years 1921-1940 (1) | | | | | | | | |
| Fertility Outcome | Age | CHBORN 35-44 | CHBORN 35-44 | CHBORN 35-44 Married with children | NCHILD 35-44 | NCHILD 35-44 | NCHILD 35-44 Married with children | NCHLT5 23-32 | NCHLT5 23-32 | NCHLT5 23-32 Married with children |
| Sample | | Married | All | | Married | All | | Married | All | |
| Constant | | 50.8226 | 42.359 | -48.4887 | 97.8496 | 96.4153 | 112.5026 | 49.6386 | 45.8712 | 49.9368 |
| | t-stat | 6.1342 | 5.4384 | -1.814 | 7.7728 | 8.3717 | 10.1103 | 5.7799 | 6.132 | 4.7567 |
| $MMR_{st}^{ref} (2)$ | | -0.03 | -0.029 | -0.0007 | -0.0256 | -0.0237 | -0.0299 | -0.0172 | -0.0159 | -0.0155 |
| | t-stat | -10.73 | -11.0503 | -0.0732 | -6.0418 | -6.1169 | -7.9615 | -5.9514 | -6.3102 | -4.3697 |
| $IMR_{st}^{ref} (3)$ | | 0.0118 | 0.0102 | -0.0035 | 0.0068 | 0.0049 | 0.0104 | 0.001 | 0.0002 | 0.0007 |
| | t-stat | 3.9821 | 3.6742 | -0.364 | 1.5132 | 1.1869 | 2.6245 | 0.3305 | 0.0743 | 0.1771 |
| Adj R-squared | | 0.5365 | 0.6623 | 0.054 | 0.2534 | 0.3408 | 0.2579 | 0.1269 | 0.1521 | 0.0933 |
| Specification | | IV, Birth Years 1913-1940 (1) | | | | | | | | |
| Fertility Outcome | Age | CHBORN 35-44 | CHBORN 35-44 | CHBORN 35-44 Married with children | NCHILD 35-44 | NCHILD 35-44 | NCHILD 35-44 Married with children | NCHLT5 23-32 | NCHLT5 23-32 | NCHLT5 23-32 Married with children |
| Sample | | Married | All | | Married | All | | Married | All | |
| Constant | | -86.7468 | -87.2234 | -129.3648 | 23.0772 | 26.7125 | 30.0107 | 1.6488 | -0.9456 | 9.2941 |
| | t-stat | -5.9081 | -6.359 | -6.2309 | 2.8773 | 3.5876 | 3.9705 | 0.3326 | -0.2194 | 1.5155 |
| $MMR_{s}^{pre} * I_t (4)$ | | 0.014 | 0.013 | 0.0051 | 0.0088 | 0.0087 | 0.0078 | 0.0027 | 0.0031 | 0.0022 |
| | t-stat | 9.5399 | 9.4177 | 2.4668 | 10.9151 | 11.6713 | 10.2307 | 5.4834 | 7.2697 | 3.6547 |
| $IMR_{st}^{ref} (3)$ | | -0.0012 | -0.0027 | 0.0005 | -0.0032 | -0.0045 | -0.002 | -0.0045 | -0.0045 | -0.0042 |
| | t-stat | -0.241 | -0.5953 | 0.0713 | -1.2129 | -1.8361 | -0.813 | -2.7634 | -3.1693 | -2.0697 |
| Adj R-squared | | 0.4391 | 0.4873 | 0.2414 | 0.2682 | 0.3357 | 0.2019 | 0.2591 | 0.3295 | 0.1884 |

(1) All regressions include state and cohort effects.

(2) Reference maternal mortality is the average maternal mortality in the state at age 15-20 for each cohort.

(3) Reference infant mortality is the average infant mortality in the state at age 15-20.

(4) The instrument for reference MMR in each state is the average reference MMR for the 1913-1920 cohorts in each state.

4.1.2 Sensitivity

To assess the robustness of the baseline findings, we perform a variety of robustness checks.

Controls We first 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 relation 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, 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.

For each mortality rate we consider the reference value for the white population for each cohort, that is the average in the state at age 15-20, with the age range equated to the one for 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. For all fertility measures, the effect of the decline in maternal mortality on fertility is robust to the inclusion of the health controls, both in terms of the magnitude and significance of the estimated coefficient. We also find that the estimates for all fertility outcomes are robust to the joint inclusion of all mortality indicators in the regression.

We next consider a set of economic and demographic controls. 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 for CHBORN at age 35-44, which measures completed fertility, are robust to the inclusion of these controls. The estimates are also robust for NCHILD and NCHLT5 for Groups 2-4, though controlling for the share of employment in the health sector reduces the significance of the estimates. The estimated coefficient on maternal mortality switches sign for NCHILD and NCHLT5 when controlling for economic conditions (Group 1), and loose significance when controlling jointly for all economic indicators (Group 5). This suggests that economic conditions may affect the timing of fertility.

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. 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 finds 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 towards 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 promoting 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 D. 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.3.

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) find 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 interpret as a consequence of high participation of low skill women during the war years. Doepke, Hazan and Maoz (2007) argue 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).³⁵ 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 Burns and Turner, 2002).

The results are displayed in Table 4 (right panel). Indicators in groups 1-3 are cohort invariant, and thus, 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, though the magnitude of this effect is small.³⁶ We also control for all these state characteristics

³⁵See footnote 39 for more details.

³⁶Mobilization 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 one standard deviation decline in maternal

jointly (5), and find that the estimated coefficients on reference maternal mortality are robust.

Alternative Specifications We estimate equation (9) including only cohorts born in 1921-1940. Since all included cohorts are exposed to the decline in maternal mortality in this specification, only the cross-state variation in maternal mortality is used to identify the impact of its decline on fertility for the treated cohorts. The states with lower maternal mortality can be interpreted as having experienced a larger treatment.

The results are presented in Table 3 (middle panel, heading “Panel, Birth Years 1921-1940”) and confirm those for the baseline specification. For the married sample, the estimates imply that CHBORN at age 35-44 rises by 0.30 for a 10 deaths per 10,000 live births drop in maternal mortality, with the coefficient significant at the 1% level. The coefficient on infant mortality is positive and significant, and the coefficient for maternal mortality is robust to the inclusion of infant mortality in the regression. Results are similar for all and married with children women. The estimation results for the other fertility measures also confirm the findings for the baseline specification.

Instrumental Variables As a second robustness check, we estimate an instrumental variable version of equation (9) where we use the average reference maternal mortality for birth years 1913-1920³⁷, which we denote with MMR_s^{pre} , as an instrument for the magnitude of the overall drop in maternal mortality. This approach is based on the strong convergence of maternal mortality values across states between the mid 1930s and the mid 1950s. In this case, ZZ_{st} is defined as:

$$ZZ_{st} = MMR_s^{pre} \times I_t^{post}, \tag{11}$$

where the variable I_t^{post} indicates whether a birth cohort t belongs to the treatment group³⁸. 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 estimation results are presented in Table 3 (bottom panel, heading “IV, Birth Years 1913-1940”). The criteria for inclusion in the treatment and control groups are the same as for the baseline specification. The statistic MMR_s^{pre} is a very strong instrument, as the correlation between MMR_s^{pre} and the average decline in reference maternal mortality, between the 1921-1940 and 1913-1920 cohorts, is 0.94 with a p-value of 0.00.

The estimates strongly confirm the panel estimates for all fertility outcomes and all samples. The estimated coefficient for the married sample suggests that a one standard deviation decline in the instrument is associated with an increase in CHBORN between the 1921-1940 and 1913-1920 cohorts of 1.29 or 50%, which accounts for 89% of the actual change.

mortality is associated with a rise in the outcome of 0.24, in the specification that controls for mobilization rates.

³⁷Formally, $MMR_s^{pre} = \sum_{t \in Control} \frac{MMR_{st}^{ref}}{\#Control}$, where *Control* is simply the set of cohorts in the control group.

³⁸Bleakley (2007) follows a similar approach to assess the effects of malaria eradication on fertility and educational attainment in the American South.

TABLE 4: Fertility: Regression Results with Controls

| Panel Specification (1), (2) | | | | | | | | | | | |
|-----------------------------------|----------|----------|---------|-------------------------------------|---------|---------|---------|---|----------|----------|---------|
| Health (3) | | | | Economic and Demographic | | | | State Characteristics (5) | | | |
| Dependent variable | CHBORN | NCHILD | NCHLTS | Dependent variable | CHBORN | NCHILD | NCHLTS | Dependent variable | CHBORN | NCHILD | NCHLTS |
| Age | 35-44 | 35-44 | 23-32 | Age | 35-44 | 35-44 | 23-32 | Age | 35-44 | 35-44 | 23-32 |
| 1: Male Mortality | | | | 1: Personal Income, Unemp. | | | | 1: Literacy 1930 | | | |
| $MMR_{st}^{ref} (4)$ | -0.0513 | -0.0255 | -0.0087 | $MMR_{st}^{ref} (4)$ | -0.0249 | 0.0116 | 0.0024 | $MMR_{st}^{ref} (4)$ | -0.0437 | -0.026 | -0.0082 |
| t-stat | -12.7238 | -11.28 | -6.234 | t-stat | -5.0038 | 4.9885 | 1.4132 | t-stat | -12.3629 | -12.6301 | -6.3881 |
| Adj R-squared | 0.4716 | 0.2739 | 0.2657 | Adj R-squared | 0.4978 | 0.5183 | 0.3262 | Adj R-squared | 0.4309 | 0.1604 | 0.1302 |
| 2: Sulfa Related Mortality | | | | 2: White, Foreign Born, Farm | | | | 2: Acceptance of Women's Suffrage | | | |
| $MMR_{st}^{ref} (4)$ | -0.0486 | -0.0259 | -0.0091 | $MMR_{st}^{ref} (4)$ | -0.0442 | -0.0106 | -0.0071 | $MMR_{st}^{ref} (4)$ | -0.0453 | -0.0275 | -0.0082 |
| t-stat | -12.1345 | -11.3924 | -6.4356 | t-stat | -9.8117 | -4.7059 | -4.5222 | t-stat | -13.123 | -13.7082 | -6.4618 |
| Adj R-squared | 0.4857 | 0.2734 | 0.2648 | Adj R-squared | 0.501 | 0.4539 | 0.3004 | Adj R-squared | 0.4443 | 0.1813 | 0.1375 |
| 3: TB Mortality | | | | 3: Share Public | | | | 3: Sheppard-Towner & Social Security Act | | | |
| $MMR_{st}^{ref} (4)$ | -0.0485 | -0.0259 | -0.009 | $MMR_{st}^{ref} (4)$ | -0.0471 | -0.0206 | -0.0074 | $MMR_{st}^{ref} (4)$ | -0.0437 | -0.0264 | -0.0077 |
| t-stat | -12.1351 | -11.3623 | -6.4034 | t-stat | -11.507 | -9.2254 | -5.1906 | t-stat | -12.5337 | -13.1043 | -6.0721 |
| Adj R-squared | 0.4866 | 0.2733 | 0.2646 | Adj R-squared | 0.48 | 0.3252 | 0.2763 | Adj R-squared | 0.431 | 0.1678 | 0.1339 |
| 4: Malaria | | | | 4: Share Health | | | | 4: Mob. Rates applied to 1922-1928 cohorts | | | |
| $MMR_{st}^{ref} (4)$ | -0.0563 | -0.0279 | -0.0098 | $MMR_{st}^{ref} (4)$ | -0.0337 | -0.0035 | -0.0022 | $MMR_{st}^{ref} (4)$ | -0.0325 | -0.0272 | -0.0085 |
| t-stat | -13.9627 | -12.3733 | -7.0213 | t-stat | -7.4441 | -1.5489 | -1.4165 | t-stat | -8.559 | -12.0909 | -6.0353 |
| Adj R-squared | 0.4826 | 0.2926 | 0.2745 | Adj R-squared | 0.4943 | 0.4374 | 0.3037 | Adj R-squared | 0.4498 | 0.1591 | 0.1288 |
| 5: 1+2+3+4 | | | | 5: 1+2+3+4 | | | | 5: 1+2+3+4 | | | |
| $MMR_{st}^{ref} (4)$ | -0.0515 | -0.029 | -0.0103 | $MMR_{st}^{ref} (4)$ | -0.0304 | 0.0079 | 0.0001 | $MMR_{st}^{ref} (4)$ | -0.0445 | -0.0258 | -0.0082 |
| t-stat | -13.0663 | -12.8208 | -7.4223 | t-stat | -6.0572 | 3.4657 | 0.0749 | t-stat | -12.6664 | -12.8551 | -6.4363 |
| Adj R-squared | 0.5269 | 0.3197 | 0.3137 | Adj R-squared | 0.5168 | 0.5639 | 0.3487 | Adj R-squared | 0.4495 | 0.2156 | 0.1539 |

(1) Baseline specification. All regressions include state and cohort effects. Included cohorts: 1913-1940.

(2) Estimates shown for Married sample.

(3) All mortalities are average in the state at age 15-20.

(4) Reference maternal mortality is the average maternal mortality in the state at age 15-20 for each cohort.

(5) Only includes time effects.

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, $SulfaMR_s^{ref}$, 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 define $Sulfa_s^{pre}$ to be the average of this indicator for all cohorts in the control group and use it as our instrument a_s in equation (11).

The estimates, reported in Appendix C, Table 11, suggest a strong positive relation between the instrument for sulfa related mortalities and the change in fertility across cohorts, which implies that a larger decline in sulfa mortalities is associated with a larger rise in fertility across cohorts. The estimates are significant at the 1% level for all fertility measures and all samples.

Pre-Existing Trends To check for pre-existing trends, we also estimate equation (9) including only cohorts that were not exposed to the decline in maternal mortality. The results are displayed in Table 12 (top panel, heading “Fertility”) in Appendix C. Specifically, we consider women born in 1905 – 1915 (Control Group I) and in 1910 – 1915 (Control Group II). These women had mostly completed their fertility by 1938.

Due to the fact that the state level maternal mortality data start in 1915 and are available for all the states in the sample only starting in 1933, we extend the age range for reference maternal mortality. The minimum age is set at 15 and the maximum age is the age in 1933 of the oldest included cohort (in the baseline specification, the age range is 15-20). This implies that the included cohorts may be contributing with their own fertility to reference maternal mortality. Given the systematic relation between parity and maternal mortality risk, this could bias the estimates. The sign of the bias depends on average fertility, given that maternal mortality risk varies with parity (see footnote 13). Given the low fertility of cohorts in this sample, an increase in fertility would be associated with a decline in the maternal mortality risk and lead to a negative bias in the coefficient on maternal mortality in the regressions, potentially pointing to a negative relation between maternal mortality and fertility, even though it is not present. So the bias works against the falsification exercise.

We limit attention to CHBORN at age 35-44 and find that the estimated coefficient on reference maternal mortality is not significant in either control group. This suggests that there is no relation between maternal mortality and fertility across states for the untreated cohorts. To gauge whether the age range for the calculation of reference maternal mortality influences this finding, we repeat the estimation for the 1921-1940 cohorts, using the same definition of reference maternal mortality as in the falsification exercise (Treatment Group in Table 12, top panel). We find that the baseline results are completely confirmed.

Included Cohorts and Reference Maternal Mortality We evaluate the sensitivity of the estimates to the assumptions on reference maternal mortality and on the included cohorts. Results for CHBORN at age 35-44 for Married women are presented in Table 13 reported in Appendix C.

Columns 1-4 present estimates of equation (9) for different assumptions on reference maternal mortality. Column 1 simply repeats the baseline estimates, for which reference maternal mortality is the average in the state at age 15-20. The alternative age ranges we consider are 10-15 (column 2), 10-20 (column 3) and 5-15 (column 4). Even if reference maternal mortality is sensitive to the age range, both the magnitude and the significance of the estimated coefficients are very robust to the definition of the age range, and the explanatory power of the regression essentially is unchanged.

Columns 5-8 present estimates for alternative assumptions on the included cohorts. The baseline regression equation is estimated for the 1913-1935 birth cohorts (column 6), for the 1913-1945 cohorts (column 7), and for the 1920-1940 cohorts (column 8). Column 5 reproduces the baseline results. Once again, the estimation results confirm our baseline specification.

4.1.3 Marriage Rates and Childlessness

This section examines the impact of the maternal mortality drop on marriage rates and childlessness.

For the cohorts included in the analysis, extra-marital fertility was very small, and 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 (9) using the percentage of women that are married (MARRIED) at 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 0.03 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 women with CHBORN=0 at age 35-44, which we denote with CHBORN_0. We perform the estimation on the samples All and Married. The results are displayed in Table 5. The panel estimates suggest no significant relation with CHBORN_0 for either sample, despite the fact that childlessness for married women drops from 10% in for the 1913-1920 cohorts to 7% for the 1921-1940 cohorts.

TABLE 5: Marriage and Childlessness: Regression Results

| Specification (1) | Marriage Rates | | | Childlessness | |
|---|----------------|--------------|----------------|---------------|----------------|
| | Panel | Panel | | Panel | |
| | MARRIED | CHBORN=0 | CHBORN=0 | NCHILD=0 | NCHILD=0 |
| Dependent Variable | 23 | 35-44 | 35-44 | 23-32 | 23-32 |
| Age | All | All | Married | All | Married |
| Constant | -5.8043 | -0.7827 | 2.583 | -12.7715 | -4.7401 |
| t-stat | -1.9671 | -0.7041 | 2.6075 | -12.0277 | -3.5764 |
| MMR^{ref}_{st} (2) | -0.0031 | -0.0004 | 0.0002 | 0.0018 | 0.0011 |
| t-stat | -4.7509 | -1.6074 | 0.7492 | 7.7157 | 3.7205 |
| IMR^{ref}_{st} (3) | 0.0029 | 0.0006 | 0.0001 | -0.0001 | -0.0006 |
| t-stat | 3.3705 | 1.911 | 0.5022 | -0.3732 | -1.4618 |
| Adj R-squared | 0.4617 | 0.3767 | 0.2498 | 0.303 | 0.1654 |

Summary Statistics

| Dependent Variable | Birth Years 1913-1920 | | | | |
|--------------------|-----------------------|--------------|--------------|--------------|--------------|
| | MARRIED | CHBORN=0 | CHBORN=0 | NCHILD=0 | NCHILD=0 |
| | All | All | Married | All | Married |
| Age | 23 | 35-44 | 35-44 | 23-32 | 23-32 |
| Mean | 0.9362 | 0.0999 | 0.107 | 0.0807 | 0.1033 |
| St. Dev. | 0.2422 | 0.0213 | 0.0491 | 0.0266 | 0.0339 |
| Mean | Birth Years 1921-1940 | | | | |
| | 0.9765 | 0.107 | 0.074 | 0.1014 | 0.1027 |
| | St. Dev. | 0.093 | 0.0491 | 0.0219 | 0.026 |

(1) All regressions include state and cohort effects. Included cohorts: 1913-1940.

(2) Reference maternal mortality is the average maternal mortality in the state at age 15-20 for each cohort.

(3) Reference infant mortality is the average infant mortality in the state at age 15-20.

4.1.4 Fertility by Education

We also estimate the effect of the decline in maternal mortality on fertility by education. We consider the following fertility outcomes: CHBORN, NCHILD, and CHBORN_0 at age 35-44. We run separate regressions for women with college (COLL) and with high school (HS), for marriage status All and Married.

The results are displayed in Table 6. The estimated coefficients on maternal mortality for the two fertility measures are highly significant and have the same sign as the baseline specification for all education groups. The absolute and percentage rises in CHBORN and NCHILD 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, as proxied by CHBORN_0 at age 35-44, 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 relative to the control group, but it is not associated with the decline in maternal mortality. (Childlessness is approximately constant across the treated and 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.

TABLE 6: Fertility by Education: Regression Results

| Specification (1) | | Panel | | | | | | | |
|--|--------|----------------|----------------|------------|---------------|----------------|----------------|--------------|---------------|
| Fertility Outcome | | CHBORN | NCHILD | CHBORN=0 | NCHILD=0 | CHBORN | NCHILD | CHBORN=0 | NCHILD=0 |
| Age | | 35-44 | 35-44 | 35-44 | 23-32 | 35-44 | 35-44 | 35-44 | 23-32 |
| Sample | | All | All | All | All | Married | Married | Married | Married |
| Education | | COLL | COLL | COLL | COLL | COLL | COLL | COLL | COLL |
| Constant | | 76.0632 | 82.7137 | -5.5398 | 20.2722 | 73.565 | 95.5935 | -4.447 | 19.4051 |
| | t-stat | 3.8264 | 4.639 | -1.4437 | 5.118 | 3.4857 | 4.8955 | -1.1949 | 5.2536 |
| MMR^{ref}_{st} (2) | | -0.025 | -0.0302 | 0.0003 | 0.0029 | -0.03 | -0.0307 | 0.003 | 0.0009 |
| | t-stat | -5.7012 | -7.6975 | 0.3065 | 3.2776 | -6.4583 | -7.146 | 3.7125 | 1.1511 |
| IMR^{ref}_{st} (3) | | 0.0005 | 0.0018 | 0.0001 | -0.0007 | 0.0033 | 0.0004 | -0.0014 | 0.0003 |
| | t-stat | 0.091 | 0.347 | 0.0804 | -0.6363 | 0.5429 | 0.0681 | -1.3117 | 0.267 |
| Adj R-squared | | 0.0988 | 0.1674 | 0.0264 | 0.42 | 0.1382 | 0.1537 | 0.0329 | 0.3229 |
| Model p-value | | 0 | 0 | 0.0013 | 0 | 0 | 0 | 0.0001 | 0 |
| Summary Statistics | | | | | | | | | |
| Birth Years 1913-1920 | | | | | | | | | |
| Mean | | 1.7944 | 1.3982 | 0.143 | 0.494 | 1.8551 | 1.7267 | 0.1576 | 0.3513 |
| St. Dev. | | 0.3343 | 0.3078 | 0.0701 | 0.0887 | 0.3691 | 0.3782 | 0.0777 | 0.0788 |
| Birth Years 1921-1940 | | | | | | | | | |
| Mean | | 2.2739 | 2.038 | 0.161 | 0.2737 | 2.4751 | 2.3119 | 0.0972 | 0.1926 |
| St. Dev. | | 0.2545 | 0.2558 | 0.0473 | 0.0655 | 0.2732 | 0.2829 | 0.0269 | 0.0449 |
| Specification (1) | | Panel | | | | | | | |
| Fertility Outcome | | CHBORN | NCHILD | CHBORN=0 | NCHILD=0 | CHBORN | NCHILD | CHBORN=0 | NCHILD=0 |
| Age | | 35-44 | 35-44 | 35-44 | 23-32 | 35-44 | 35-44 | 35-44 | 23-32 |
| Sample | | All | All | All | All | Married | Married | Married | Married |
| Education | | HS | HS | HS | HS | HS | HS | HS | HS |
| Constant | | 20.0028 | 93.6221 | -1.7209 | 5.1478 | 13.9662 | 94.3862 | 3.4676 | 5.6692 |
| | t-stat | 2.2595 | 10.3468 | -1.3575 | 1.9333 | 1.5158 | 9.5737 | 3.3163 | 2.7945 |
| MMR^{ref}_{st} (2) | | -0.0231 | -0.0253 | -0.0002 | 0.004 | -0.0248 | -0.0257 | 0 | 0.0025 |
| | t-stat | -11.8395 | -12.6754 | -0.6898 | 6.9026 | -12.2026 | -11.8523 | -0.063 | 5.6061 |
| IMR^{ref}_{st} (3) | | 0.0078 | 0.0023 | 0.0009 | -0.0026 | 0.009 | 0.0031 | 0.0003 | -0.0019 |
| | t-stat | 3.0864 | 0.8826 | 2.4241 | -3.455 | 3.4048 | 1.0939 | 0.9737 | -3.2074 |
| Adj R-squared | | 0.5798 | 0.3986 | 0.3636 | 0.4086 | 0.568 | 0.3194 | 0.2718 | 0.3482 |
| Model p-value | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Predicted change in outcome for one st. dev. drop in reference maternal mortality | | 0.18389679 | 0.20141077 | 0.00159218 | -0.0318436 | 0.19743032 | 0.20459513 | 0 | -0.01990225 |
| Summary Statistics | | | | | | | | | |
| Birth Years 1913-1920 | | | | | | | | | |
| Mean | | 2.3074 | 1.8789 | 0.1002 | 0.3876 | 2.3366 | 2.0423 | 0.1008 | 0.2483 |
| St. Dev. | | 0.2832 | 0.2477 | 0.0228 | 0.0636 | 0.2821 | 0.2299 | 0.0237 | 0.043 |
| Birth Years 1921-1940 | | | | | | | | | |
| Mean | | 2.9498 | 2.2493 | 0.1014 | 0.2487 | 3.0625 | 2.4105 | 0.0714 | 0.1514 |
| St. Dev. | | 0.3673 | 0.3043 | 0.0548 | 0.0714 | 0.3152 | 0.2756 | 0.0249 | 0.0463 |

(1) All regressions include state and cohort effects. Included cohorts: 1913-1940.
(2) Reference maternal mortality is the average maternal mortality in the state at age 15-20 for each cohort.
(3) Reference infant mortality is the average infant mortality in the state at age 15-20.

4.2 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 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 substantially faster rate for about 25 years. Goldin, Katz and Kuziemko (2006) argue that 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,³⁹ 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.

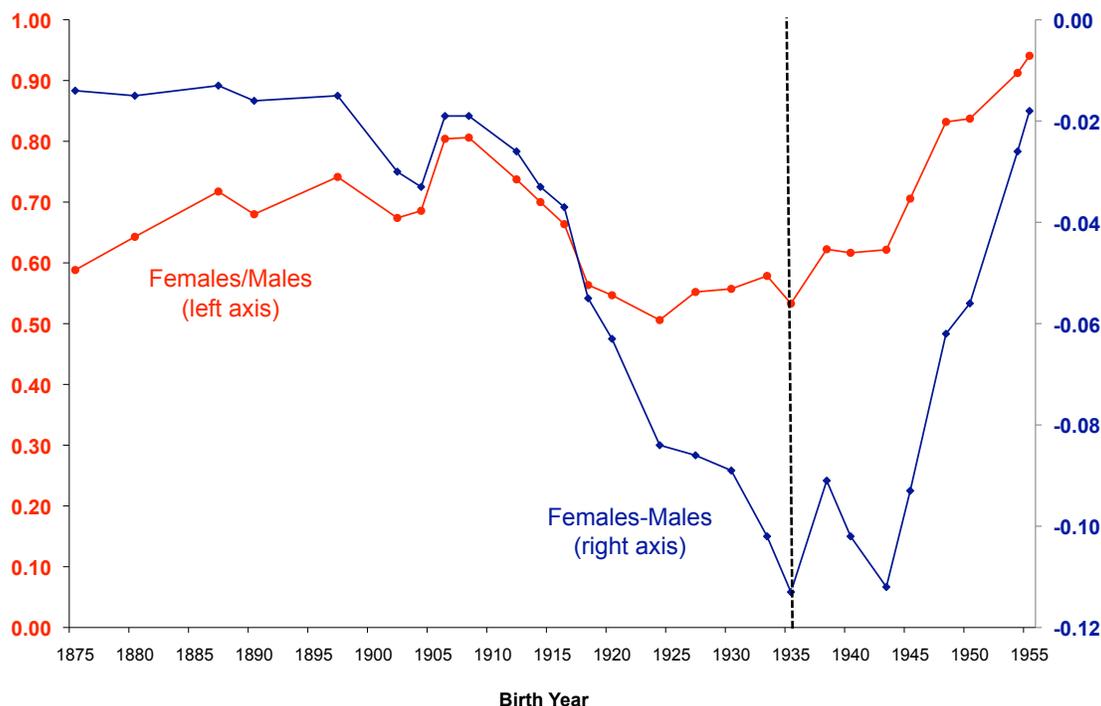


FIGURE 7: Gender differentials in college graduation rates

Source: Goldin (1997). Based on responses of individual aged 45-54 or 55-64. College graduation defined as college attendance for 4 or more years.

The main measures of education attainment used in the analysis are the fraction of individuals with a high school degree and the fraction 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. Educational outcomes are measured at age 23-32⁴⁰ and the estimation is conducted separately for All, Married and Married with children individuals. The estimation strategy is similar to the one employed for fertility, except here we include males in the analysis. Thus, in addition to

³⁹ 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's of arts using GI benefits (Burns and Turner, 2002).

⁴⁰We consider age 23-32 since our focus is on parental investments in education, and parental involvement in educational decisions can be thought to cease at the age of majority. We also consider educational outcomes at age 23, at age 35 and at age 35-44 as a robustness exercise (not reported). The findings are consistent with those for the baseline specification.

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 following equation:

$$Y_{sgt} = \alpha_0 + \alpha_1 ZZ_{st} \times F_g + \mu_{st} + \nu_{gt} + \delta_{gs} + \beta X_{st} + \epsilon_{gdt}, \quad (12)$$

where $g = f, m$ stands for gender, and $\mu_{st}, \nu_{gt}, \delta_{gs}$ correspond to state-cohort, gender-cohort and gender-state interactions⁴¹. As for fertility, the baseline specification is a panel regression that includes cohorts with varying degrees of exposure to the decline in maternal mortality, with $ZZ_{st} = MMR_{st}^{ref}$, where MMR_{st}^{ref} corresponds to the average maternal mortality in state, s , for cohort, t . 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 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 age 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, high school graduation rates were higher for females than for males, while the converse is true for college graduation rates. 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.

⁴¹This specification follows Jayachandran and Lleras-Muney (2008).

TABLE 7: Education: Summary Statistics

| Birth Years 1919-1932 | | | | | | | | | | | | |
|---|----------|---------|----------------------------|--------|---------|--------------------------|--------|---------|----------------------------|--------|---------|----------------------------|
| Reference Maternal Mortality (Age 5-10) | | | | | | | | | | | | |
| | Mean | | 48.5588 | | | | | | | | | |
| | St. Dev. | | 7.3353 | | | | | | | | | |
| Educational Attainment at age 23-32 | COLL | COLL | COLL Married with children | HS | HS | HS Married with children | HG16 | HG16 | HG16 Married with children | HG13 | HG13 | HG13 Married with children |
| | All | Married | children | All | Married | children | All | Married | children | All | Married | children |
| Sample | | | | | | | | | | | | |
| Mean Female | 0.0423 | 0.0359 | 0.0303 | 0.2268 | 0.2287 | 0.2169 | 0.0733 | 0.0605 | 0.0502 | 0.1968 | 0.1777 | 0.1588 |
| St. Dev. Female | 0.0147 | 0.0119 | 0.0088 | 0.0322 | 0.0329 | 0.0286 | 0.0236 | 0.0188 | 0.0155 | 0.0512 | 0.0452 | 0.0407 |
| Mean Male | 0.0781 | 0.0725 | 0.0643 | 0.1726 | 0.1763 | 0.1648 | 0.1195 | 0.111 | 0.0974 | 0.2463 | 0.2238 | 0.1992 |
| St. Dev. Male | 0.0218 | 0.0174 | 0.015 | 0.025 | 0.0258 | 0.0235 | 0.0345 | 0.0311 | 0.0258 | 0.0571 | 0.0524 | 0.0513 |
| Birth Years 1933-1950 | | | | | | | | | | | | |
| Reference Maternal Mortality (Age 5-10) | | | | | | | | | | | | |
| | Mean | | 9.1113 | | | | | | | | | |
| | St. Dev. | | 2.0244 | | | | | | | | | |
| Educational Attainment at age 23-32 | COLL | COLL | COLL Married with children | HS | HS | HS Married with children | HG16 | HG16 | HG16 Married with children | HG13 | HG13 | HG13 Married with children |
| | All | Married | children | All | Married | children | All | Married | children | All | Married | children |
| Sample | | | | | | | | | | | | |
| Mean Female | 0.1373 | 0.1264 | 0.1033 | 0.4538 | 0.4679 | 0.4745 | 0.1373 | 0.1264 | 0.1033 | 0.2952 | 0.2826 | 0.2533 |
| St. Dev. Female | 0.0619 | 0.0595 | 0.0355 | 0.0538 | 0.0558 | 0.0493 | 0.0619 | 0.0595 | 0.0355 | 0.077 | 0.0773 | 0.0588 |
| Mean Male | 0.2045 | 0.196 | 0.1567 | 0.3668 | 0.3815 | 0.4069 | 0.2045 | 0.196 | 0.1567 | 0.3811 | 0.3656 | 0.3161 |
| St. Dev. Male | 0.0644 | 0.0629 | 0.05 | 0.0513 | 0.0531 | 0.0593 | 0.0644 | 0.0629 | 0.05 | 0.0758 | 0.0731 | 0.0714 |

4.2.1 Baseline Results

The results for the baseline specification are presented in Table 8 (top panel, heading “Panel, Birth Years 1919-1950”). The estimates suggest that the decline in maternal mortality had a strong positive effect on the female-male differential in educational attainment for all educational outcomes and all samples. The estimated coefficients are all significant at the 1% level and imply sizable effects. The estimates predict that a decline in reference maternal mortality of 10 deaths per 10,000 live births is associated with a 0.024 rise in the female-male differential in college graduation. This corresponds to a 64% rise from the average differential for the 1919-1932 cohorts, which was -0.0358.

The results for high school graduation are similarly strong. A decline in reference maternal mortality of 10 deaths per 10,000 live births is associated with a 0.06 rise in the female-male differential in high school graduation. The sample of married and married with children individuals delivers similar results.

4.2.2 Sensitivity

To assess the robustness of these findings, we conduct a sensitivity analysis which parallels the one for the fertility estimates.

Controls We begin with the same controls used in the fertility analysis, described in Section 4.1.2. 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 the fertility analysis, we also control for infant mortality (5)⁴². 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 (Murphy, Simon and Tamura, 2005), 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 39). Interestingly, we find that mobilization rates had a strong negative effect on the female-male differential in both high school and college graduation rates. An increase in mobilization rates of 0.05 is associated with a decline of the female-male differential in graduate rates by 0.02 for high school and 0.01 for college.

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.

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

Alternative Specifications and Instrumental Variables We consider a specification that only includes the 1933-1950 birth years, which we interpret as being treated. In addition, we estimate an instrumental variable specification including the 1919-1950 birth years, where, as for fertility, we define $ZZ_{st} = MMR_s^{pre} \times I_t^{post}$, where MMR_s^{pre} is average reference maternal mortality for the 1919-1932 birth years and $I_t^{post} = 1$ if $t = 1933, ..1950$. For the IV specification, a *positive* estimated value of α_1 is consistent with a positive effect of the drop in maternal mortality on female educational attainment (relative to males).

The results are displayed in Table 8 and strongly confirm the findings for the baseline specification. The panel estimates for the 1933-1950 birth years suggest that a one standard deviation decline in maternal mortality in the treatment group is associated with 0.012 rise in the female-male differential in college graduation rates (middle panel, heading “Panel, Birth Years 1933-1950”). The

⁴²The baseline specification for fertility already controls for infant mortality.

TABLE 9: Education: Regression Results with Controls

| Panel Specification (1) (3) | | | | | | | |
|---|------------|------------------------------|--------------|--|------------|--|------------|
| I: Health (4) | | II: Economic and Demographic | | III: State Characteristics | | IV: Controls for Treated Cohorts | |
| Educational Attainment at age 23-32 | HS Married | HS Married | COLL Married | Educational Attainment at age 23-32 | HS Married | Educational Attainment at age 23-32 | HS Married |
| Sample | | | | | | | |
| 1: Male Mortality | | | | | | | |
| MMR _{st} ^{ref} * F _a (2) | -0.0055 | -0.0054 | -0.0009 | MMR _{st} ^{ref} * F _a (2) | -0.0036 | MMR _{st} ^{ref} * F _a (2) | -0.0070 |
| t-stat | -36.9122 | -33.9725 | -9.2148 | t-stat | -22.9653 | t-stat | -23.7031 |
| Adj R-squared | 0.465 | 0.4679 | 0.4811 | Adj R-squared | 0.5639 | Adj R-squared | 0.4489 |
| 2: Sulfia related | | | | 1: Literacy 1930 | | 2: Acceptance of Women's Suffrage | |
| MMR _{st} ^{ref} * F _a (2) | -0.0021 | -0.0038 | -0.0003 | MMR _{st} ^{ref} * F _a (2) | -0.0063 | MMR _{st} ^{ref} * F _a (2) | -0.0021 |
| t-stat | -13.8879 | -25.0768 | -3.0102 | t-stat | -44.5071 | t-stat | -23.1226 |
| Sulfia related | -0.0052 | 0.5833 | 0.5447 | Adj R-squared | 0.4387 | Adj R-squared | 0.4256 |
| t-stat | -41.4333 | | | 3: Sheppard-Towner & Social Security Act | | 3: Sheppard-Towner & Social Security Act | |
| Adj R-squared | 0.6397 | | | MMR _{st} ^{ref} * F _a (2) | -0.0063 | MMR _{st} ^{ref} * F _a (2) | -0.0022 |
| 3: Malaria | | | | t-stat | -43.7195 | t-stat | -21.9833 |
| MMR _{st} ^{ref} * F _a (2) | -0.0058 | -0.0048 | -0.0005 | Adj R-squared | 0.441 | Adj R-squared | 0.3356 |
| t-stat | -39.3949 | -29.7395 | -5.1892 | 4: Mob. Rates applied to 1922-1928 cohorts, interacted with gender | | 4: Mob. Rates applied to 1922-1928 cohorts, interacted with gender | |
| Adj R-squared | 0.4556 | 0.4881 | 0.4961 | MMR _{st} ^{ref} * F _a (2) | -0.0054 | MMR _{st} ^{ref} * F _a (2) | -0.0024 |
| 4: TB | | | | t-stat | -30.704 | t-stat | -19.1384 |
| MMR _{st} ^{ref} * F _a (2) | -0.0021 | -0.0047 | -0.0002 | Mob. Rate * F _a (2) | -0.1752 | Mob. Rate * F _a (2) | 0.0073 |
| t-stat | -14.0974 | -28.5397 | -1.5366 | Adj R-squared | -8.8041 | Adj R-squared | 0.5182 |
| Adj R-squared | 0.6366 | 0.485 | 0.5419 | 5: 1+2+3+4 | 0.4501 | 5: 1+2+3+4 | 0.3103 |
| 5: Infant Mortality | | | | MMR _{st} ^{ref} * F _a (2) | -0.0034 | MMR _{st} ^{ref} * F _a (2) | 0.0001 |
| MMR _{st} ^{ref} * F _a (2) | -0.0045 | -0.0029 | 0.0002 | t-stat | -23.162 | t-stat | 1.1282 |
| t-stat | -27.1509 | -20.5867 | 2.2022 | Adj R-squared | 0.5864 | Adj R-squared | 0.703 |
| IMR _t | -0.0037 | 0.6303 | 0.6837 | | | | |
| t-stat | -17.9178 | | | | | | |
| Adj R-squared | 0.494 | | | | | | |
| 6: 1+2+3+4+5 | | | | | | | |
| MMR _{st} ^{ref} * F _a (2) | -0.0036 | -0.0003 | -0.0003 | | | | |
| t-stat | -24.6109 | -3.0412 | -3.0412 | | | | |
| Adj R-squared | 0.5707 | 0.6287 | 0.6287 | | | | |

(1) Includes state/time, female/time, female/state interactions. Included cohorts: 1919-1950.
(2) Reference maternal mortality is the average maternal mortality in the state at age 5-10 for each cohort.
(3) Estimates shown for Married sample.
(4) All mortalities are average in the state at age 5-10.

mean value of this differential in the treatment group is -0.069. The estimates are not as big for high school graduation, but are still highly significant with considerable explanatory power. The IV estimates (bottom panel, heading “IV, Birth Years 1919-1950”) are consistent with the other specifications.

Pre-Existing Trends To gauge the presence of pre-existing trends, we estimate equation (12) for a sample of untreated cohorts. The results are displayed in Appendix C, Table 12 (bottom panel, heading “Education”). We limit attention to HS and COLL at age 23-32. We consider two samples of untreated cohorts, comprising birth years 1910-1917 (Control Group I, age 21 in 1931-1938) and 1918-1925 (Control Group II, age 21 in 1939-1946).

As for fertility, we extend the definition of reference maternal mortality, since the state level maternal mortality data start in 1915, and are available for all the states in the sample only starting in 1933. While in the baseline specification, the age range for reference maternal mortality is 5-10, here the minimum age is 5 and the maximum is the age in 1933 for the oldest cohort included in the regression (age 23 for Control Group I and age 15 for Control Group II). The change in the reference maternal mortality implies that the women included in the analysis potentially contribute to the measure of reference maternal mortality with their childbearing behavior. Specifically, if higher female education is associated with lower maternal mortality, this would lead us to find a negative relation between these two variables even in the control group.⁴³

We find that the estimated coefficient on reference maternal mortality is insignificant or of the wrong sign for COLL for both control groups. This suggests that there is no relation between maternal mortality and COLL across states for the untreated cohorts. For HS we find a negative and significant coefficient for both control groups and both samples. This finding may be due to reverse causation, as discussed above.

Reference Maternal Mortality and Included Cohorts We now investigate the sensitivity of our findings to the assumptions on the age range for reference maternal mortality and the treatment and control groups, concentrating on college graduation rates. The results are reported in Table 13 in Appendix C.

Columns 1-4 explore the sensitivity to the definition of reference maternal mortality. We do so to allow different time lags between the observation of maternal mortality and the influence of maternal mortality on parental investments in daughters’ education. For the baseline specification, reference maternal mortality is the average in the state at age 5-14 (column 1). Here, we also consider: age 0 to 15 (column 2), 5 to 10 (column 3) and -5 to 5 (column 4). Since the alternative assumptions mainly affect the value of reference maternal mortality for the treated cohorts, we include only birth years 1933-1950 in the estimation. We find that the alternative assumptions confirm our baseline results.

Columns 5-9 examine the sensitivity to the included cohorts. Column 5 replicates the baseline

⁴³For consistency, we also repeat the estimation for the 1933-1950 birth years using the age range for reference maternal mortality used in the falsification exercise. The estimates confirm the results in the main analysis.

results. Column 6 includes individuals born in 1919-1940, to explore whether the female-male differential in college graduation rates rose immediately as maternal mortality declined. We then run three specifications including only potentially treated cohorts, comprising birth years 1919-1955 (column 7), and 1929-1960 (column 8). The estimation results confirm the findings for the baseline specification. The absolute value of the estimated coefficient on reference maternal mortality is largest for the the 1929-1960 cohorts and smallest for the 1919-1940 cohorts.

4.3 Baby Bust

We now examine the relation 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 analyzed in Section 3 predicts a negative relation 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 (9) 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.⁴⁴ For robustness, we also consider a specification in which the control group is comprised of 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 in the treatment group.

The findings are displayed in Table 10. We focus on CHBORN at age 35-44, and consider all, married and married with children women. 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 (9) for the IV specification implies that a decline in maternal mortality is associated with a reduction in fertility between the treated and control cohorts. The estimated coefficient suggests 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

⁴⁴The 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.

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 CHBORN 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.⁴⁵ 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 to 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 CHBORN at age 35-44 for all and married women.⁴⁶

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 rises 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, *coeteris 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.

⁴⁵We also estimate specifications that incorporate the health indicators, and the economic and demographic 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.

⁴⁶We treat these controls as invariant state characteristic, even though on 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.

TABLE 10: Baby Bust

| Regression Results ⁽¹⁾ | | | | | | | | | | | |
|--|----------------|------------------|---------------------------------------|----------------|------------------|---------------------------------------|----------------|------------------|---------------------------------------|--------------------|------------------|
| Specification | IV | | | | | | | | | | |
| Included Cohorts | 1921-1950 | | | 1921-1950 | | | 1933-1950 | | | 1921-1950 | |
| Treated Cohorts | 1941-1950 | | | 1946-1950 | | | 1941-1950 | | | 1941-1950 | |
| Fertility Outcome | CHBORN | CHBORN | CHBORN Married with children | CHBORN | CHBORN | CHBORN Married with children | CHBORN | CHBORN | CHBORN Married with children | CHBORN | CHBORN |
| Sample Age | All 35-44 | Married 35-44 | Married with children 35-44 | All 35-44 | Married 35-44 | Married with children 35-44 | All 35-44 | Married 35-44 | Married with children 35-44 | COLL, All 35-44 | HS, All 35-44 |
| Constant | 2.9701 | 3.1825 | 4.6994 | 2.5002 | 2.689 | 4.2155 | 125.6098 | 121.3597 | 104.5456 | 43.4075 | 40.56 |
| t-stat | 47.2883 | 46.7532 | 27.4982 | 36.994 | 36.7368 | 25.4913 | 12.9634 | 13.7484 | 12.4743 | 4.5881 | 8.7435 |
| $MMR^{ref}_s * I_t$ (2) | -0.0504 | -0.0515 | -0.0484 | -0.0574 | -0.0571 | -0.0515 | -0.0026 | -0.003 | -0.0039 | -0.0234 | -0.0356 |
| t-stat | -37.6121 | -35.4614 | -13.2776 | -27.706 | -25.4794 | -10.1638 | -3.5652 | -4.3947 | -6.1193 | -5.5877 | -17.351 |
| IMR^{ref}_{at} (3) | -0.0045 | -0.0086 | -0.0182 | 0.0055 | 0.0019 | -0.0078 | -0.0076 | -0.0081 | -0.0062 | -0.0088 | -0.0175 |
| t-stat | -4.4688 | -7.9179 | -6.6559 | 5.3584 | 1.7046 | -3.1253 | -1.8337 | -2.1565 | -1.7208 | -2.536 | -10.2935 |
| Adj R-squared | 0.7162 | 0.6425 | 0.1495 | 0.6322 | 0.5374 | 0.1088 | 0.522 | 0.5642 | 0.5684 | 0.2348 | 0.6688 |
| Regression Results with State Level Controls | | | | | | | | | | | |
| Fertility Outcome | IV | | | | | | | | | | |
| Sample Age | All 35-44 | Married 35-44 | Married with children 35-44 | All 35-44 | Married 35-44 | Married with children 35-44 | All 35-44 | Married 35-44 | Married with children 35-44 | COLL, All 35-44 | HS, All 35-44 |
| 1: Early Access to Oral Contraception | | | | | | | | | | | |
| Year_Pill | -0.0187 | -0.0152 | -0.0163 | -0.0185 | -0.015 | -0.0161 | -0.0208 | -0.0171 | -0.0177 | -0.0194 | -0.0178 |
| t-stat | -5.3822 | -4.5105 | -2.4699 | -5.1776 | -4.2614 | -2.3865 | -5.2284 | -4.7079 | -5.1403 | -3.5121 | -4.7297 |
| $MMR^{ref}_s * I_t$ (2) | -0.0098 | -0.0106 | -0.0125 | -0.0074 | -0.0075 | -0.0082 | -0.0026 | -0.003 | -0.0039 | -0.0224 | -0.0384 |
| t-stat | -15.374 | -17.2385 | -10.3221 | -11.6862 | -12.1633 | -6.8978 | -3.6282 | -4.4562 | -6.2169 | -5.1722 | -13.0168 |
| Adj R-squared | 0.3859 | 0.3841 | 0.1013 | 0.3476 | 0.3271 | 0.0663 | 0.5359 | 0.5744 | 0.5806 | 0.1497 | 0.2899 |
| 2: Early Access to Abortion | | | | | | | | | | | |
| Year_Abortion | 0.0803 | 0.0719 | 0.0611 | 0.0804 | 0.0722 | 0.0618 | 0.0682 | 0.0537 | 0.0489 | 0.0784 | 0.0753 |
| t-stat | 5.4481 | 5.0499 | 2.1793 | 5.2925 | 4.8525 | 2.1618 | 4.0074 | 3.4557 | 3.3111 | 3.3517 | 4.7277 |
| $MMR^{ref}_s * I_t$ (2) | -0.0097 | -0.0105 | -0.0124 | -0.0073 | -0.0074 | -0.0081 | -0.0027 | -0.003 | -0.004 | -0.0221 | -0.0382 |
| t-stat | -15.2508 | -17.1542 | -10.2637 | -11.5559 | -12.0644 | -6.8382 | -3.6625 | -4.4794 | -6.2087 | -5.1069 | -12.9368 |
| Adj R-squared | 0.3862 | 0.3862 | 0.1005 | 0.3481 | 0.3296 | 0.0656 | 0.5301 | 0.5695 | 0.5733 | 0.149 | 0.2899 |
| Summary Statistics | | | | | | | | | | | |
| Fertility Outcome | CHBORN | CHBORN | CHBORN Married with children | CHBORN | CHBORN | CHBORN Married with children | CHBORN | CHBORN | CHBORN Married with children | CHBORN | CHBORN |
| Sample Age | All 35-44 | Married 35-44 | Married with children 35-44 | All 35-44 | Married 35-44 | Married with children 35-44 | All 35-44 | Married 35-44 | Married with children 35-44 | COLL, All 35-44 | HS, All 35-44 |
| Control Group | | | | | | | | | | | |
| Mean | 2.8921 | 3.0172 | 4.0688 | 2.7893 | 2.9178 | 3.9932 | 2.9177 | 3.0656 | 4.2583 | 2.2739 | 2.9498 |
| St. Dev. | 0.3368 | 0.2862 | 0.2373 | 0.3323 | 0.2797 | 0.2278 | 0.3436 | 0.278 | 0.2645 | 0.2545 | 0.3673 |
| Treated Group | | | | | | | | | | | |
| Mean | 2.2052 | 2.3595 | 3.569 | 2.032 | 2.1989 | 3.4475 | 2.2052 | 2.3595 | 3.569 | 1.7148 | 2.3397 |
| St. Dev. | 0.3038 | 0.2792 | 0.2494 | 0.2802 | 0.2893 | 0.2857 | 0.3038 | 0.2792 | 0.2494 | 0.2886 | 0.2587 |

(1) Baseline specification. All regressions include state and cohort effects.
(2) The instrument is average reference maternal mortality (average maternal mortality in state at age 15-20) for birth cohorts 1913-1920.
(3) Reference infant mortality is the average infant mortality in the state at age 15-20.

5 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 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 U.S.

The link between the decline in pregnancy-related mortality and fertility in the U.S. 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 U.S. Albanesi (2011) examines 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 the ones in which maternal mortality declined sharply between 1935 and 1955 experienced a baby boom, and that the magnitude of the boom is positively related to magnitude of the drop

in maternal mortality. The U.S., with the highest rate of maternal mortality among the advanced economies in the 1930s, experienced the largest boom in fertility. Emerging economies experience repeated episodes of temporary maternal mortality decline, followed by a rise in fertility relative to trend. 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.

References

- [1] Acemoglu, Daron, David Autor and David Lyle. 2004. Women, War and Wages: The Effect of Female Labor Supply on the Wage Structure at Mid century. *Journal of Political Economy* 112(3): 497-551.
- [2] Albanesi, Stefania. 2011. Maternal Health and Fertility: An International Perspective. Manuscript, Columbia University. Available here: <http://www.columbia.edu/~sa2310/Papers/InternationalMatHealthandFertility/IntMatHealthandFertility.htm>
- [3] Albanesi, Stefania. 2008. Comment on: “Marriage and Divorce since WWII: Analyzing the Role of Technological Progress on the Formation of Households,” by Jeremy Greenwood and Nezih Guner. Forthcoming, NBER Macroannual 2008, Volume 23, edited by Daron Acemoglu, Kenneth Rogoff, Michael Woodford. MIT Press.
- [4] Albanesi, Stefania and Claudia Olivetti. 2009. Gender Roles and Medical Progress. NBER WP 14873.
- [5] Albanesi, Stefania, Jodie C. Liu, and Grant Graziani. In progress. The Historical Determinants of Maternal Mortality Reduction in the United States. Manuscript, Columbia University.
- [6] Altshuler, Glenn C. and Sturat M. Blumin. 2009. The G.I. Bill: A New Deal for Veterans. Oxford University Press.
- [7] Alvarez, Fernando. 1999. Social Mobility: The Barro-Becker children meet the Laitner-Loury dynasties. *Review of Economic Dynamics* 2 (1): 65-103.
- [8] Angrist, Joshua D., and William N. Evans. 1999. Schooling and Labor Market Consequences of the 1970 State Abortion Reforms. *Research in Labor Economics XVIII: 75-113*.
- [9] Bailey, Martha. 2006. More Power to the Pill. *The Quarterly Journal of Economics*.
- [10] Bailey, Martha and William J. Collins. 2009. Did Improvements in Household Technology Cause the Baby Boom? Evidence from Electrification, Appliance Diffusion, and the Amish. NBER WP 14641.

- [11] Baker, S. Josephine. 1923. Why do our mothers and babies die? *Ladies' Home Journal* 40: 212-213.
- [12] Becker, Gary S. and Robert J. Barro. 1988. A Reformulation of the Economic Theory of Fertility. *The Quarterly Journal of Economics* 103(1): 1-25.
- [13] Becker, Gary S., Murphy, Kevin M., and Tamura, Robert. 1990. Human Capital, Fertility, and Economic Growth. *Journal of Political Economy* 98(5): 12-37.
- [14] Berry, Linda G. 1977. Age and Parity Influences on Maternal Mortality: United States, 1919-1969. *Demography* 14(3): 297-310.
- [15] Bleakley, Hoyt. 2007. Disease and Development: Evidence from Hookworm Eradication in the American South. *The Quarterly Journal of Economics* 122: 73-117.
- [16] Bleakley, Hoyt, and Fabian Lange. 2008. Chronic Disease Burden and the Interaction of Education, Fertility and Growth. *Review of Economics and Statistics*.
- [17] Bound, John and Sara Turner. 202. Going to war and going to college: Did WWII and the GI bill increase educational attainment for returning veterans?" *Journal of Labor Economics* XX: 784-815.
- [18] Bromley, Dorothy D. 1929. What Risk Motherhood? *Harper's* 159: 20.
- [19] Center for Disease Control. 1999. Infant and Maternal Mortality in the United States: 1900-99. *Population and Development Review* 125(4): 821-826.
- [20] Center for Disease Control and Prevention. 2010. Deaths: Leading Causes for 2006. *National Vital Statistics Reports* 58 (14). Available at: http://www.cdc.gov/nchs/data/nvsr/nvsr58/nvsr58_14.pdf.
- [21] Children's Bureau. 1931. The Promotion of the Welfare and Hygiene of Maternity and Infancy. Children's Bureau Publication 203, US Dept. of Labor, Washington DC.
- [22] Cornwall, Marie, State-level Suffrage Legislation Database (Provo, Utah: Brigham Young University, 2003).
- [23] Cornwall, Marie, Eric Dahlin, Brayden King, and Kendra Schiffman, "Moving Mountains: An Institutionalist Analysis of State-Level Woman Suffrage Legislative Success," Unpublished manuscript presented and distributed at the Social Science History Association annual meeting in Chicago, IL, 2004.
- [24] Cutler, David, Angus deaton, and Adriana Lleras-Muney. 2006. The Determinants of Mortality. *The Journal of Economic Perspectives* 20(3): 97-120.

- [25] Dannreuther, Walter T. 1931. The American Board of Obstetrics and Gynecology: Its Organization, Function and Objectives. *Journal of the American Medical Association* 96: 797-798.
- [26] Doepke, Matthias. 2005. Child mortality and the fertility decline: Does the Barro-Becker model fit the facts? *Journal of Population Economics* 18: 337-366.
- [27] Doepke, Matthias, Moshe Hazan and Yishay Maoz. 2007. The Baby Boom and World War II: A Macroeconomic Analysis. Manuscript, Northwestern University.
- [28] Dublin, Mary. 1936. Maternal Mortality and the Decline in the Birth Rate. *Annals of the American Academy of Political and Social Science* 188 (The American People: Studies in Population): 107-116.
- [29] Franks AL, Kendrick, KS, Olson, DR, Atrash HK, Saftlas, AF and Moien M. 1992 Hospitalization for pregnancy complications, United States, 1986 and 1987. *Am J Obstet Gyn.* 166(5):1339-44.
- [30] Geddes, Rick and Dean Lueck. 2002. The Gains from Self-Ownership and the Expansion of Women's Rights. *The American Economic Review* 92(4): 1079-1092.
- [31] Goldin, Claudia. 1991. The Role of World War II in the Rise of Women's Employment. *The American Economic Review* 81(4): 741-756.
- [32] Goldin, Claudia. 1997. Career and Family: College Women Look to the Past. In R. Ehrenberg and F. Blau, eds., *Gender and Family Issues in the Workplace*, New York: Russell Sage Foundation Press: 20-58.
- [33] Goldin, Claudia, Lawrence F. Katz. 2002. The Power of the Pill: Oral Contraceptives and Women's Career and Marriage Decisions. *The Journal of Political Economy* 110: 730 -770.
- [34] Goldin, Claudia, Lawrence F. Katz, and Ilyana Kuziemko. 2006. The Homecoming of American Women: The Reversal of the College Gender Gap. *Journal of Economic Perspectives* 20(4): 133-156.
- [35] Greenwood, Jeremy, Ananth Seshadri, and Guillaume Vanderbroucke. 2005. The Baby Boom and the Baby Bust. *American Economic Review* 95(1): 183-207.
- [36] Haines, Michael R. 1997. The Relationship Between Infant and Child Mortality and Fertility: Some Historical and Contemporary Evidence for the United States. In *From Death to Birth: Mortality Decline and Reproductive Change*, Mark R. Montgomery and Barney Cohen, editors. National Academy Press.

- [37] [32] Haines, Michael R. 2006a. Fetal death ratio, neonatal mortality rate, infant mortality rate, and maternal mortality rate, by race: 1850-1998. Table Ab912-927 in *Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition*, edited by Susan B. Carter, Scott Sigmund Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch, and Gavin Wright. New York: Cambridge University Press.
- [38] Haines, Michael R. 2006b. Live births, deaths, infant deaths, marriages, and divorces, by race: 1909—1998. Table Ab11-30 in *Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition*, edited by Susan B. Carter, Scott Sigmund Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch, and Gavin Wright. New York: Cambridge University Press.
- [39] Haines, Michael R., and Richard Sutch. 2006. Population, by marital status, sex, and race: 1880—1990. Table Aa614-683 in *Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition*, edited by Susan B. Carter, Scott Sigmund Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch, and Gavin Wright. New York: Cambridge University Press.
- [40] Hauser, Robert L. 1976. *Fertility Tables for Birth Cohorts by Color: United States 1901-1973*. Rockville, MD: National Center for Health Statistics.
- [41] Jayachandran, Seema and Adriana Lleras-Muney. 2009. Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines. *The Quarterly Journal of Economics* 124 (1): 349-397.
- [42] Jayachandran, Seema, Adriana Lleras-Muney and Kimberly V. Smith. 2009. Modern Medicine and the 20th-Century Decline in Mortality: Evidence on the Impact of Sulfa Drugs. *American Economic Journal: Applied Economics*, forthcoming.
- [43] Jones, Larry E. and Alice Schoonbroodt. 2007. Complements versus Substitutes and Trends in Fertility Choice in Dynastic Models. NBER WP 13680.
- [44] Jones, Larry E. and Michele Tertilt. 2007. An Economic History of Fertility in the U.S.: 1826-1960. *Forthcoming in "Frontiers of Family Economics," edited by Peter Rupert, Elsevier*.
- [45] Kerr, J.M. Munro. 1933. *Maternal Mortality and Morbidity*. Edinburgh.
- [46] Lesser, Arthur J. 1985. The Origin and Development of Maternal and Child Health Programs in the United States. *American Journal of Public Health* 75(6): 590-598.
- [47] Litoff, Judy Barrett. 1986. *The American Midwife Debate*. New York: Greenwood Press.

- [48] Lott, John R. Jr, and Lawrence W. Kenney. 1999. Did Women's Suffrage Change the Size and Scope of Government? *The Journal of Political Economy* 107(6): 1163-1198.
- [49] Loudon, Irvine. 1992a. Death in Childbirth: An International Study of Maternal Care and Maternal Mortality 1800-1950. Clarendon Press, Oxford.
- [50] Loudon, Irvine. 1992b. The Transformation of Maternal Mortality. *British Medical Journal* 305(6868): 1557-1560.
- [51] Meckel, Richard A. 1990. Save the Babies: American Public Health Reform and the Prevention of Infant Mortality 1850-1929.
- [52] Meigs, Grace H. 1917. Maternal Mortality from all conditions connected with childbirth in the United States and certain other countries. Children's Bureau Publication No. 19, Miscellaneous Series 6. US Department of Labor, Washington DC.
- [53] Miller, Grant. 2008. WOMEN'S SUFFRAGE, POLITICAL RESPONSIVENESS, AND CHILD SURVIVAL IN AMERICAN HISTORY. *Quarterly Journal of Economics* 123(3): 1287-1327.
- [54] Moehling, Carolyn and Melissa Thomasson. 2009. The Political Economy of Saving Mothers and Babies: The Politics of State Participation in the Sheppard-Towner Program. Manuscript. Rutgers University.
- [55] Mount, Steve. "Ratification of Constitutional Amendments". <http://www.usconstitution.net/constamrat.html>. Retrieved February 24 2007.
- [56] Parker, JK, and EM Carpenter. 1981. Julia Lathrop and the Children's Bureau: The emergence of an institution. *Social Science Review* 55: 60-77.
- [57] Preston, Samuel H. and Michael R. Haines. 1991. Fatal Years: Child Mortality in Late Nineteenth-Century America. *Princeton, NJ: Princeton University Press*.
- [58] Rethereford, Robert D. 1972. Tobacco Smoking and the Sex Mortality Differential. *Demography* 9(2): 203-216.
- [59] Rosen, G. 1958. A history of public health. New York: MD Publications.
- [60] Schmidt, W M. 1973. The development of health services for mothers and children in the United States. *American Journal of Public Health* 63: 419 - 427.
- [61] Sinai, Nathan and Odin W. Anderson. 1947. The Emergency Maternal and Infant Care Program 1943-1946. Bureau of Public Health Economics, School of Public Health. University of Michigan, Ann Arbor.
- [62] Soares Rodrigo. 2005. Mortality Reductions, Educational Attainment, and Fertility Choice. *The American Economic Review* 95(3): 580-601.

- [63] Stanley, Marcus. 2003. College Education and the Midcentury GI Bills. *The Quarterly Journal of Economics* 118(2): 671-708.
- [64] Starr, Paul. 1982. *The Social Transformation of American Medicine*. Basic Books.
- [65] Stolnitz, George J.. 1956. A Century of International Mortality Trends. *Population Studies* 10(1): 17-42
- [66] Thomasson, Melissa A. and Treber, Janet. 2008. From home to hospital: The evolution of childbirth in the United States, 1928-1940. *Explorations in Economic History* 45(1): 76-99.
- [67] Weil, David N. 2007. Accounting for the Effects of Health on Economic Growth. *The Quarterly Journal of Economics* 122(3): 1265-1306.
- [68] Wertz, Richard W. and Dorothy C. Wertz. 1977. *Lying-In. A History of Childbirth in America*. Expanded Edition. Yale University Press. New Haven and London.
- [69] White House Conference on Child Health and Protection. *Fetal, Newborn and Maternal Morbidity and Mortality*. New York and London: D. Appleton-Century Company, 1933.
- [70] Wilcox LS, Marks JS. 1994. *From Data to Action: CDC's Public Health Surveillance for Women, Infants, and Children*. CDC maternal and child health monograph. Atlanta, GA: Centers for Disease Control and Prevention. 10.
- [71] Wolpin, Kenneth. 1993. Determinants and consequences of the mortality and health of infants and children. *Handbook of Population and Family Economics*, M. R. Rosenzweig & Stark, O. (ed.). Edition 1, volume 1, chapter 10, pages 483-557. Elsevier.

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 here: <http://www.columbia.edu/~sa2310/Papers/MaternalHealth/abstract.htm>

A.1 Fertility and Education Data

Most of our demographic and economic state-level data are from the Integrated Public Use Micro Sample (IPUMS) of the decennial Census of the United States (from 1930 to 2000). 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 through 64. In both samples we exclude individuals living in farms, as well as those living in group quarters (e.g. prisons, and other

group living arrangements such as rooming houses and military barracks).⁴⁷ We use the following variables:

Fertility variables: CHBORN: Number of children ever born to each woman. (Women were to report all live births by all fathers, whether or not the children were still living; they were to exclude stillbirths, adopted children, and stepchildren.) NCHILD: Number of children below 13 living in the household. NCHLT5: Number of children below 5 living in the household. MARRIED: Equals 1 if married, with spouse present or absent (if IPUMS variable `marst` is either 1 or 2). Available years: 1880-2000. MARRIED WITH CHILDREN: Equals 1 if married with children (if `married=1`, and IPUMS variable `nchild>=1`). Available years 1880-2000

Education variables: For 1940 to 1980 we use the IPUMS variable HIGRADE which records the highest grade of school attended or completed by the respondent. This variable 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 1st 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 4th year of college (if IPUMS variable `'higraded'>=190`). Equals 0 if not, and is set to " " if missing. Available years: 1940-1980. HS: Equals 1 if high school degree (if IPUMS variable `educrec=7`, which corresponds to grade 12 being the highest grade attained). Available years: 1940-2000. 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/cmfc-icd9-archive1998.html>.

Infant Mortality

Death Rates: 1925-1940: VSUS 1900-1940, Table 28; 1941-1960: VSUS, 1940-1960, Table

⁴⁷That is, we further restrict the sample to observations with group quarters status equal 1, "Households under 1970 definition."

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 on-line at <http://wonder.cdc.gov/cmfi-icd9-archive1998.html>.

Live Births

Birth, Stillbirth, and Infant Mortality Statistics 1931-36, Table 2; VSUS 1937-38, Table 2; VSUS 1939-41, Table 3; VSUS 1942-43, Table 9; VSUS 1944, Table 5; VSUS 1945, Table 6; VSUS 1946, Table 4; VSUS 1947-48, Table 3; VSUS 1949, Table 9; VSUS 1950, Table 17; VSUS 1951-54, Table 21; VSUS 1955, Table 30; VSUS 1956, Table 34; VSUS 1957, Table 33; VSUS 1959, Table 31; VSUS 1960-61, Table 2-8; VSUS 1962, Table 1-36; VSUS 1963-65, Table 1-41; VSUS 1966, Table 2-1; VSUS 1967-68, Table 1-42; VSUS 1969, Table 1-72; VSUS 1970, Table 1-73; VSUS 1971-75, Table 2-1. 1979-1998: “1979-1998 Archive” accessible on-line at <http://wonder.cdc.gov/cmfi-icd9-archive1998.html>.

Male, Tuberculosis, Scarlet Fever Pneumonia, Influenza, Malaria Mortality Rates

Death Rates: 1900-1936 Miller (2008), available online at <http://www.stanford.edu/~ngmiller/>

Death Rates: 1937-1940: Vital Statistics in the United States 1937-1958, Various Tables. See online appendix for details. All data available at <http://www.cdc.gov/nchs/products/vsus.htm#historical>.

Population

1915-02 Statistical Abstract of the US Census Bureau: Chart Title Missing; 1916-02 Statistical Abstract of the US Census Bureau: No. 23 - Population of the United States at each Census: 1790 to 1910, With Estimates for July 1, 1916; 1917-02 Statistical Abstract of the US Census Bureau: No. 23 - Population of the United States at each Census: 1790 to 1910, With Estimates for July 1, 1917; 1919-02 Statistical Abstract of the US Census Bureau: No. 23 - Population of the United States at each Census: 1790 to 1910, With Estimates for July 1, 1918; 1920: 1920-02 Statistical Abstract of the US Census Bureau: No. 21 - Population of the United States at Each Census, 1790 to 1920: By States and Geographic Divisions; 1920 (White): 1924-02 Statistical Abstract of the US Census Bureau: No. 10 - Population: Race, By States; VSUS 1925-1929 Mortality Statistics, Table 1 A; 1930-02 Statistical Abstract of the US Census Bureau: No. 7 - Population by states: 1930, 1930 (White): 1941-02 Statistical Abstract of the US Census Bureau: No. 15 - Population, by Race, by States: 1890 to 1940; 1931 - 1940: VSUS, 1900-1940; 1941 - 1960: VSUS, 1940-1960; VSUS 1961, Vol. I, Natality: Table 5-4; VSUS 1962-1963, Vol. I, Natality: Table 4-5; VSUS 1964. Volume I, Natality: Table 4-4; VSUS 1965. Volume I, Natality: Table 4-3; VSUS 1966. Volume I, Natality: Table 4-4; VSUS 1967-1969, Volume I, Natality: Table 4-3. 1970 - 1998: CDC Wonder Census Estimates, 1970-1998 accessible on-line at <http://wonder.cdc.gov/cmfi-icd9-archive1998.html>.

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 state-wide measure, i.e., 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 Kenney (1999). For date of ratification of 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 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 to 44 years old registered males in a state who were drafted for war,⁴⁸ based on published tables from the Selective Service System (1956). The average mobilization rate was .474 with a standard deviation of .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 base on Acemoglu, Autor and Lyle (2004), intended to serve as a proxy for the number of eligible recipients.

Based on findings in Stanley (2003) and Burns and Turner (2002), suggesting that the 1922-1928 birth cohorts displayed the largest take up of WWII GI benefits. Specifically, based on in Table 2 in Bound and Turner (2002), more than 10% of veterans achieved a bachelor’s of arts using GI benefits for these cohorts. Thus, we set $I_t^G = 1$ for $t = 1922, \dots, 1928$. 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.

⁴⁸Since 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 D.C. are not available (the former because it saw large population changes during this time period).

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 N. 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 N. 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 which 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 is coded as 1970 for Alaska, California, Hawaii, New York, and Washington, and 1972 for Vermont and New Jersey. All other states permitted early legal access with Roe v. Wade in 1973.

B Theory

B.1 Basic Model

B.1.1 Proofs

Proof of Proposition 1:

Totally differentiating the system of first order necessary conditions (1)-(2) with respect to μ for $\mu = \mu'$, and simplifying yields:

$$\begin{aligned} & [-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)] \frac{\partial e'}{\partial \mu} + [-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu)] \frac{\partial b}{\partial \mu} = -\kappa(sb) \frac{\partial U'(e'; \mu)}{\partial \mu}, \\ & [-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu)] \frac{\partial e'}{\partial \mu} + [-v_{bb}(b, e') + \kappa''(sb)s^2U'(e'; \mu)] \frac{\partial b}{\partial \mu} = u((1+\varepsilon)e)w - \kappa'(sb)s \frac{\partial U(e'; \mu)}{\partial \mu}. \end{aligned}$$

Solving:

$$\begin{aligned} \frac{\partial e'}{\partial \mu} &= \frac{-[-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu)] \frac{\partial b}{\partial \mu} - \frac{\kappa(sb) \frac{\partial U'(e'; \mu)}{\partial \mu}}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]}}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]}, \\ &= \left\{ \frac{\det H_V}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]} \right\} \frac{\partial b}{\partial \mu} \\ &= \frac{[-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu)] \kappa(sb) \frac{\partial U'(e'; \mu)}{\partial \mu}}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]} + u((1+\varepsilon)e)w - \kappa'(sb)s \frac{\partial U(e'; \mu)}{\partial \mu}. \end{aligned}$$

By (3):

$$\frac{\partial U'(e; \mu)}{\partial \mu} = -bu'(w(1+\varepsilon))w\varepsilon < 0, \quad (13)$$

$$\frac{\partial U(e; \mu)}{\partial \mu} = -bu(w(1 + \varepsilon e)) < 0. \quad (14)$$

By Assumption 1, $\det H_V > 0$ and $[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)] < 0$. Thus, condition (6) guarantees $\frac{\partial b}{\partial \mu} < 0$ and $\frac{\partial e'}{\partial \mu} < 0$. ■

Proof of Proposition 2:

Totally differentiating the system of first order necessary conditions (1)-(2) with respect to e and simplifying yields:

$$[-v_{e'e'} + \kappa(sb)U''(e'; \mu')] \frac{\partial e'}{\partial e} + [-v_{e'b} + \kappa'(sb)sU'(e'; \mu')] \frac{\partial b}{\partial e} = 0,$$

$$[-v_{e'b} + \kappa'(sb)sU'(e'; \mu')] \frac{\partial e'}{\partial e} + [-v_{bb} + \kappa''(sb)s^2U(e'; \mu)] \frac{\partial b}{\partial e} = \mu u'(w(1 + \varepsilon e))\varepsilon w.$$

Substituting for $\frac{\partial b}{\partial e}$ and simplifying the second equation:

$$\frac{\partial e'}{\partial e} = - \frac{[-v_{e'b} + \kappa'(sb)sU'(e'; \mu')]}{[-v_{e'e'} + \kappa(sb)U''(e'; \mu')]} \frac{\partial b}{\partial e}, \quad (15)$$

$$\frac{\partial b}{\partial e} = \left\{ \frac{\det H_V}{[-v_{e'e'} + \kappa(sb)U''(e'; \mu')]} \right\}^{-1} \mu u'(w(1 + \varepsilon e))\varepsilon w, \quad (16)$$

By Assumption 1, $\det H_V > 0$ and $[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)] < 0$. Thus, $\frac{\partial b}{\partial e} < 0$. In addition, condition (6), equation (15) implies $\frac{\partial e'}{\partial e} > 0$. ■

B.1.2 Response to a Temporary Decline in Maternal Mortality

Proposition 3 derives the response of desired fertility and investment in daughters' human capital to a temporary decline in pregnancy-related mortality rate for the mothers' generation.

Proposition 3 *Assume the pregnancy-related mortality risk changes for the mothers' generation. Then, by Assumption 1:*

$$\frac{\partial b}{\partial \mu} \leq 0. \quad (17)$$

In addition:

$$\frac{\partial e'}{\partial \mu} \leq 0, \quad (18)$$

if condition (6) holds.

Proof:

Differentiating the system of first order necessary conditions (1)-(2) with respect to μ for given μ' and simplifying yields:

$$[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')] \frac{\partial e'}{\partial \mu} + [-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu')] \frac{\partial b}{\partial \mu} = 0,$$

$$[-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu')] \frac{\partial e'}{\partial \mu} + [-v_{bb}(b, e') + \kappa''(sb)s^2U(e'; \mu')] \frac{\partial b}{\partial \mu} = u((1 + \varepsilon e)w).$$

Solving:

$$\frac{\partial e'}{\partial \mu} = \frac{-[-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu')]}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')]} \frac{\partial b}{\partial \mu},$$

$$\left\{ \frac{\det H_V}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')]} \right\} \frac{\partial b}{\partial \mu} = u((1 + \varepsilon e)w).$$

Thus, by assumption 1, $\frac{\partial b}{\partial \mu} < 0$. In addition, condition (6) guarantees $\frac{\partial e'}{\partial \mu} < 0$. ■

Proposition 4 derives the sign of the response to a decline in daughters' pregnancy-related mortality risk.

Proposition 4 *Assume the pregnancy-related mortality risk changes for the daughters' generation. Then, the optimal response of births and parental investment in human capital satisfies:*

$$\frac{\partial b}{\partial \mu'} \leq 0 \quad , \quad (19)$$

$$\frac{\partial e'}{\partial \mu'} \leq 0, \quad (20)$$

if and only if condition (6) holds.

Proof:

Totally differentiating the first order necessary conditions (1)-(2) with respect to μ' for given μ obtains:

$$[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')] \frac{\partial e'}{\partial \mu'} + [-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu')] \frac{\partial b}{\partial \mu'} = -\kappa(sb) \frac{\partial U'(e'; \mu')}{\partial \mu'},$$

$$[-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu)] \frac{\partial e'}{\partial \mu'} + [-v_{bb}(b, e') + \kappa''(sb)s^2U(e'; \mu)] \frac{\partial b}{\partial \mu'} = -\kappa'(sb)s \frac{\partial U(e'; \mu)}{\partial \mu'}.$$

Solving:

$$\frac{\partial e'}{\partial \mu'} = \frac{-[-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu)] \frac{\partial b}{\partial \mu'}}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]} - \frac{\kappa(sb) \frac{\partial U'(e'; \mu')}{\partial \mu'}}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')]} ,$$

$$\left\{ \frac{\det H_V}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')]} \right\} \frac{\partial b}{\partial \mu'} = \frac{[-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu')]}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu')]} \kappa(sb) \frac{\partial U'(e'; \mu')}{\partial \mu'} - \kappa'(sb)s \frac{\partial U(e'; \mu)}{\partial \mu'}.$$

By (13) and (14) and assumption 1, condition (6) guarantees $\frac{\partial b}{\partial \mu'} < 0$ and $\frac{\partial e'}{\partial \mu'} < 0$. ■

Proposition 4 then implies that both desired fertility and mothers' investment in daughters' human capital rise when pregnancy-related mortality risk falls for daughters when condition (6) holds.

B.1.3 Response to a Decline in Youth Mortality

We now consider the effects of a permanent rise in s .

Proposition 5 *Assumption 1 implies $\frac{\partial b}{\partial s} < 0$ if condition (6) holds and if μ and sb are small enough. In addition, if $\frac{\partial b}{\partial s} < 0$, then $\frac{\partial e'}{\partial s} > 0$.*

Proof of Proposition 5:

Totally differentiating the system of first order necessary conditions evaluated at $\mu = \mu'$ with respect to s and simplifying:

$$\frac{\partial b}{\partial s} = \frac{\frac{[-v_{be'}(b, e') + \kappa'(sb)sU'(e'; \mu)]}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]} \left[\kappa'(sb)U'(e'; \mu)b + \kappa(sb)\frac{\partial U'(e'; \mu)}{\partial s} \right] - \kappa''(sb)sU(e'; \mu)b - \kappa'(sb)s\frac{\partial U(e'; \mu)}{\partial s}}{\frac{\det H_V}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]} - \mu u((1 + \varepsilon e)w)},$$

$$\frac{\partial e'}{\partial s} = \frac{-[-v_{e'b}(b, e') + \kappa'(sb)sU'(e'; \mu)]}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]} \frac{\partial b}{\partial s} - \frac{\kappa'(sb)U'(e'; \mu)b + \kappa(sb)\frac{\partial U'(e'; \mu)}{\partial s}}{[-v_{e'e'}(b, e') + \kappa(sb)U''(e'; \mu)]}.$$

By (13) and (14) and assumption 1, condition (6) guarantees $\frac{\partial b}{\partial s} < 0$ only if μ is small enough and sb is high enough. If $\frac{\partial b}{\partial s} < 0$, then by condition (6), $\frac{\partial e'}{\partial s} > 0$. ■

B.2 Extended Model

The model described in Section 3 can easily be extended to allow for daughters and sons. The objective of this model is to assess the effects of changes in maternal mortality on household fertility and human capital investment decisions. Thus, the model does not consider the equilibrium effects of the decline in maternal mortality on the sex ratio, the marriage market or the returns to human capital.

A household is comprised of two adults of different gender, indexed by f (female) and m (male). The household decides on the number of births b , equally divided between daughters and sons and can differentially select the human capital investment for daughters, e'_f , and sons, e'_m .

The households' decision problem is captured by the following Bellman equation:

$$U(e_f, e_m; \mu) = \max_{e'_f \geq 0, e'_m \geq 0, b \geq 0} \left\{ -v(e'_f, e'_m, b) + (1 - \mu b)u(w(1 + \varepsilon_f e_f)) + u(w(1 + \varepsilon_m e_m)) + \kappa(sb)U(e'_f, e'_m; \mu') \right\}.$$

The household's lifetime utility depends on the human capital of the husband and the wife, who have separate utility from consumption. Returns to human capital are allowed to differ across genders.

For simplicity there is no adult mortality risk for men, and daughters and sons have the same youth survival probability. As for the simplified model, we restrict attention to the household decision problem and treat the health parameters (μ and s), baseline income (w) and the returns to human capital (ε_j for $j = f, m$) as given. Also, we consider the problem of a household that is already formed, and do not explore the effects of gender differentials in mortality on the marriage market.

The first order necessary conditions for the household problem are:

$$-v_b(e'_f, e'_m, b) - \mu u(w(1 + \varepsilon_f e_f)) + \kappa'(sb)sU(e'_f, e'_m; \mu') = 0, \quad (21)$$

$$-v_{e_j}(e'_f, e'_m, b) + \kappa(sb)U_{e_j}(e'_f, e'_m; \mu') \leq 0, \quad (22)$$

with equality for $e'_j > 0$ for $j = f, m$,

$$U_{e_f}(e_f, e_m; \mu) = (1 - \mu b)u'(w(1 + \varepsilon_f e_f))w\varepsilon_f,$$

$$U_{e_m}(e_f, e_m; \mu) = u'(w(1 + \varepsilon_m e_m))w\varepsilon_m.$$

The envelope conditions clearly imply that the optimal human capital investment should be lower for girls if pregnancy related mortality is different from 0.

As for the basic model, we will impose joint concavity of household welfare in the choice vector $\{b, e'_f, e'_m\}$. we will impose the following assumption:

Assumption 2 *Let: $V(e'_f, e'_j, b; \mu, \mu') := -v(e'_f, e'_m, b) + \kappa(sb)U(e'_f, e'_m; \mu')$ is strictly concave in $\{b, e'_f, e'_m\}$.*

Assumption 2 implies that the Hessian of V , denoted with $H_V(e'_f, e'_j, b; \mu, \mu')$, is negative definite.

We now derive the effect of a permanent decline in μ starting from the parents' cohort.

Proposition 6 *Assume that pregnancy-related mortality risk is the same for mothers and daughters, so that $\mu = \mu'$, and that it changes permanently starting with the mother's generation. Then, under Assumption 2, the optimal response of births and parental investment in human capital satisfies:*

$$\frac{\partial b}{\partial \mu} \leq 0, \quad (23)$$

$$\frac{\partial e'_f}{\partial \mu} \leq 0, \quad (24)$$

if and only if:

$$\left[-v_{be'_j}(e'_f, e'_m, b) + \kappa'(sb)sU_{e'_j}(e'_f, e'_m; \mu') \right] > 0, \quad (25)$$

for $j = f, m$ and:

$$\left[-v_{e'_m e'_f}(e'_f, e'_m, b) + \kappa(sb)U_{e'_m e'_f}(e'_f, e'_m; \mu') \right] \geq 0. \quad (26)$$

Proof:

Totally differentiating the system of first order necessary conditions (21)-(22) with respect to μ for $\mu = \mu'$ at an interior solution, and simplifying yields:

$$\begin{bmatrix} \frac{\partial b}{\partial \mu} \\ \frac{\partial e'_f}{\partial \mu} \\ \frac{\partial e'_m}{\partial \mu} \end{bmatrix} = H_V^{-1} \begin{bmatrix} -\kappa'(sb)s \frac{\partial U(e'_f, e'_m; \mu)}{\partial \mu} + u(w(1 + \varepsilon_f e_f)) \\ -\kappa(sb) \frac{\partial U_{e'_f}(e'_f, e'_m; \mu)}{\partial \mu} \\ 0 \end{bmatrix}.$$

Assumption 2 implies that H_V is negative definite, thus, $\frac{\partial b}{\partial \mu} < 0$ if:

$$\begin{aligned} & [-v_{be'_m}(e'_f, e'_m, b) + \kappa'(sb)sU_{e'_m}(e'_f, e'_m; \mu')] [-v_{e'_m e'_f}(e'_f, e'_m, b) + \kappa(sb)U_{e'_m e'_f}(e'_f, e'_m; \mu')] > \\ & [-v_{be'_f}(e'_f, e'_m, b) + \kappa'(sb)sU_{e'_f}(e'_f, e'_m; \mu')] [-v_{e'_m e'_m}(e'_f, e'_m, b) + \kappa(sb)U_{e'_m e'_m}(e'_f, e'_m; \mu')]. \end{aligned}$$

This restriction is satisfied under conditions (25) and (26).

Similarly, by assumption 2, $\frac{\partial e'_f}{\partial \mu} < 0$ if:

$$\begin{aligned} & [-v_{e'_f e'_m}(e'_f, e'_m, b) + \kappa(sb)U_{e'_f e'_m}(e'_f, e'_m; \mu')] [-v_{be'_m}(e'_f, e'_m, b) + \kappa'(sb)sU_{e'_m}(e'_f, e'_m; \mu')] \\ & > [-v_{be'_f}(e'_f, e'_m, b) + \kappa'(sb)sU_{e'_f}(e'_f, e'_m; \mu')] [-v_{e'_m e'_m}(e'_f, e'_m, b) + \kappa(sb)U_{e'_m e'_m}(e'_f, e'_m; \mu')]. \end{aligned}$$

Conditions (25) and (26) guarantee this restriction will hold. ■

Condition 25 is the analogue of Condition 6, and can be similarly interpreted. Condition 26 requires that the marginal value of investing in daughter's human capital is increasing in sons' human capital (and viceversa). This condition restricts the degree of negative complementarity between investment in daughters' and sons' human capital.

Proposition 7 shows that desired fertility is decreasing in the mother's endowment of human capital.

Proposition 7 *Assumption (2) implies:*

$$\frac{\partial b(e_f, e_m; \mu)}{\partial e_f} \leq 0. \quad (27)$$

Proof:

Totally differentiating the system of first order necessary conditions (21)-(22) with respect to e_f at an interior equilibrium obtains:

$$\begin{bmatrix} \frac{\partial b}{\partial e_f} \\ \frac{\partial e'_f}{\partial e_f} \\ \frac{\partial e'_m}{\partial e_f} \end{bmatrix} = H_V^{-1} \begin{bmatrix} \mu u'(w(1 + \varepsilon_f e_f)) w \varepsilon_f \\ 0 \\ 0 \end{bmatrix}.$$

Thus, by Assumption 2, $\frac{\partial b}{\partial e_f} < 0$. ■

C Additional Empirical Results

TABLE 11: Fertility: Sulfa Mortalities as Instrument

| Specification (1) | IV | | | | | | | | |
|---|---------------|---------------|-----------------------|---------------|---------------|-----------------------|---------------|---------------|-----------------------|
| | CHBORN | CHBORN | CHBORN | NCHILD | NCHILD | NCHILD | NCHLT5 | NCHLT5 | NCHLT5 |
| Fertility Outcome | Married | All | Married with children | Married | All | Married with children | Married | All | Married with children |
| Sample Age | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 23-32 | 23-32 | 23-32 |
| Constant | 3.8114 | 3.8 | 4.8701 | 1.6424 | 1.5313 | 1.9599 | 0.9457 | 0.8426 | 1.0647 |
| t-stat | 16.6202 | 17.6988 | 14.9979 | 13.2628 | 13.2984 | 16.7524 | 12.3888 | 12.6972 | 11.2666 |
| SulfaMR ^{pre} _s *I _t (2) (3) | 0.0158 | 0.0147 | 0.0077 | 0.0084 | 0.0082 | 0.0072 | 0.0027 | 0.0032 | 0.0021 |
| t-stat | 10.7525 | 10.6951 | 3.6999 | 10.5764 | 11.1904 | 9.6575 | 5.5588 | 7.4853 | 3.4625 |
| IMR ^{ref} _{st} (3) | -0.0259 | -0.0275 | -0.0361 | 0.0026 | 0.0023 | 0.0056 | -0.0044 | -0.005 | -0.002 |
| t-stat | -9.1813 | -10.4366 | -9.0519 | 1.7197 | 1.6463 | 3.9027 | -4.6435 | -6.1578 | -1.6957 |
| Adj R-squared | 0.4234 | 0.4706 | 0.2179 | 0.2648 | 0.3305 | 0.1941 | 0.2597 | 0.3299 | 0.1879 |
| Model p-value | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

- (1) All regressions include state and cohort effects. Included cohorts: 1913-1940. Treated cohorts: 1921-1940.
- (2) The instrument for reference MMR in each state is the average reference mortality rate for diseases treatable with sulfa drugs for the control cohorts in each state. The diseases treatable with sulfa drugs are pneumonia and influenza and scarlet fever.
- (3) The reference mortality rate for each disease is the average of the mortality rate for that disease in the state at age 15-20.
- (4) Reference infant mortality is the average infant mortality in the state at age 15-20.

TABLE 12: Falsification Exercise

| Fertility (1) | Control Group I | | | Control Group II | | | Treatment Group | | |
|--------------------------------------|-----------------|---------|-----------------------|------------------|---------|-----------------------|-----------------|----------------|-----------------------|
| | 1905-1915 | | | 1910-1915 | | | 1921-1940 | | |
| Included Birth Cohorts | Age 15-28 (2) | | | Age 15-23 (2) | | | Age 15-28 (3) | | |
| Reference MMR | | | | | | | | | |
| Statistics | | | | | | | | | |
| Mean Reference MMR | 56.027 | | | 59.2552 | | | 9.1082 | | |
| St. Dev. Reference MMR | 8.242 | | | 8.7857 | | | 1.9438 | | |
| Fertility Outcome | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN |
| Age | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 |
| Sample | All | Married | Married with children | All | Married | Married with children | All | Married | Married with children |
| Constant | 8.1464 | 32.9638 | 19.5245 | 28.7379 | 30.4389 | 21.3412 | 34.9224 | 42.9063 | -68.3318 |
| t-stat | 0.5641 | 2.1734 | 1.0751 | 1.2506 | 1.2801 | 0.7881 | 5.0049 | 5.7624 | -2.8425 |
| MMR ^{ref} _{st} | 0.0075 | 0.0057 | 0.0079 | -0.004 | -0.0004 | 0.0053 | -0.046 | -0.0463 | 0.0001 |
| t-stat | 1.7545 | 1.267 | 1.4702 | -0.6972 | -0.0702 | 0.7788 | -11.3903 | -10.7275 | 0.0071 |
| IMR ^{ref} _{st} (4) | -0.0066 | -0.0118 | -0.0114 | 0.0001 | -0.0041 | -0.0121 | 0.0175 | 0.0185 | 0.0048 |
| t-stat | -1.3558 | -2.3295 | -1.8663 | 0.0174 | -0.6009 | -1.572 | 4.67 | 4.6058 | 0.3679 |
| Adj R-squared | 0.5676 | 0.5616 | 0.4677 | 0.6068 | 0.6017 | 0.5327 | 0.6649 | 0.5371 | 0.054 |

| Education (5) | Control Group I | | Control Group II | | Treatment Group | |
|--|-----------------|--------|------------------|---------|-----------------|----------------|
| | 1910-1917 | | 1918-1925 | | 1933-1950 | |
| Included Birth Cohorts | Age 5-23 (2) | | Age 5-15 (2) | | Age 5-23 (3) | |
| Reference MMR | | | | | | |
| Statistics | | | | | | |
| Mean Reference MMR | 59.5396 | | 56.7425 | | 6.189 | |
| St. Dev. Reference MMR | 8.5992 | | 8.3314 | | 1.3483 | |
| Educational Attainment at age 23-32 | HS | COLL | HS | COLL | HS | COLL |
| Sample | All | All | All | All | All | All |
| Constant | 0.1639 | 0.0414 | 0.0759 | 0.0312 | 0.3765 | 0.1457 |
| t-stat | 7.8439 | 3.4186 | 5.5659 | 3.4826 | 26.8056 | 9.3602 |
| MMR ^{ref} _{st} *F ₀ (4) | -0.0096 | 0.001 | -0.002 | 0 | -0.0008 | -0.0095 |
| t-stat | -9.7617 | 1.7432 | -4.8601 | -0.1367 | -1.6276 | -17.206 |
| Adj R-squared | 0.7242 | 0.4387 | 0.4752 | 0.2191 | 0.5537 | 0.5599 |

- (1) All regressions include state and cohort effects.
- (2) 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.
- (3) Robustness exercise. Original treated cohorts, reference MMR as in main falsification exercise.
- (4) Reference infant mortality is the average infant mortality in the state at age 15-20.
- (5) Specification includes fully interacted state, cohort and gender effects.

TABLE 13: Sensitivity Analysis

| Fertility | | | | | | | | |
|---|---------------------|------------------|------------------|-----------------|----------------|------------------|------------------|------------------|
| Specification (1) | Panel, treated only | | | | Panel | | | |
| Column | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| Fertility Outcome | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN | CHBORN |
| Age | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 | 35-44 |
| Sample | Married | Married | Married | Married | Married | Married | Married | Married |
| 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 |
| Constant | 50.8226 | 86.4734 | 82.0604 | 68.0709 | 40.1315 | -111.0453 | 103.8231 | 37.9208 |
| t-stat | 6.1342 | 6.6455 | 7.4127 | 3.6453 | 2.1925 | -6.341 | 17.3483 | 4.8327 |
| MMR^{ref}_{st} (2) | -0.03 | -0.0265 | -0.0326 | -0.0211 | -0.0523 | -0.0079 | -0.0399 | -0.0241 |
| t-stat | -10.73 | -11.503 | -12.0049 | -6.7084 | -12.9721 | -2.6082 | -23.2783 | -10.0794 |
| IMR^{ref}_{st} (3) | 0.0118 | 0.0116 | 0.0142 | 0.0091 | 0.0197 | 0.012 | 0.0084 | 0.0093 |
| t-stat | 3.9821 | 4.184 | 4.6252 | 2.7382 | 3.7436 | 3.3583 | 3.5478 | 3.463 |
| Adj R-squared | 0.5365 | 0.5385 | 0.5455 | 0.4974 | 0.4682 | 0.5581 | 0.4876 | 0.5377 |

| Education | | | | | | | | |
|---|---------------------|-----------------|-----------------|-----------------|----------------|------------------|------------------|------------------|
| Specification (4) | Panel, treated only | | | | Panel | | | |
| Education Outcome | COLL | COLL | COLL | COLL | COLL | COLL | COLL | COLL |
| Sample | All | All | All | All | All | All | All | All |
| Age | 23-32 | 23-32 | 23-32 | 23-32 | 23-32 | 23-32 | 23-32 | 23-32 |
| 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 |
| Constant | 0.1458 | 0.1467 | 0.1461 | 0.1478 | 0.0917 | 0.0799 | 0.096 | 0.1507 |
| t-stat | 9.3781 | 9.5515 | 9.4479 | 9.782 | 6.3851 | 5.8849 | 6.7142 | 12.9667 |
| MMR^{ref}_{st} (5) | -0.0057 | -0.0045 | -0.0046 | -0.0028 | -0.0024 | -0.0017 | -0.0025 | -0.0049 |
| t-stat | -17.3514 | -18.6772 | -17.9068 | -20.3782 | -26.0324 | -16.8905 | -26.6245 | -29.2387 |
| Adj R-squared | 0.5611 | 0.5715 | 0.5654 | 0.5852 | 0.3586 | 0.3113 | 0.3786 | 0.593 |

- (1) All regressions include state and cohort effects.
- (2) Reference maternal mortality is the average maternal mortality in the state at age 15-20 for each cohort.
- (3) Reference infant mortality is the average infant mortality in the state at age 15-20.
- (4) Includes state/time, female/time, female/state interactions.
- (5) Reference maternal mortality is the average maternal mortality in the state at age 5-10 for each cohort.

D 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 five year program. It was extended in 1926 and finally repealed in 1929.⁴⁹ 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, though 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 the birth and death registration area. Although repealed in 1929, the Act set a pattern for state-Federal cooperation that would re-emerge for many other programs.⁵⁰ 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

⁴⁹Skopcol (1992), Moehling and Thomasson (2009) discuss the political economy of the enactment and repeal of the Sheppard-Towner Act.

⁵⁰The Sheppard-Towner Act was not the first example of federal grant-in-aid to the States, though it was the first in the area of public health. See Skopcol (1992).

several years, though 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, as discussed in a preliminary assessment of the submitted plans by Abbott (1922).

Appropriations: Each state was granted outright \$10,000 in 1922 and \$5,000 for each subsequent year. The remaining yearly apportionment of \$1,000,000 was divided between the states based on population, on condition that the states provided matching funds. A small budget was reserved also for the activities of the Children's Bureau, which was responsible for the review and approval of the state plans.

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 six 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 1,000 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.