MATERNAL HEALTH AND THE BABY BOOM

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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, and with a decline in fertility for women born in 1941-1950. These findings are consistent with a theory of fertility featuring a trade-off between the quality and quantity of children. The analysis provides new insights on the determinants of fertility in the U.S. and other countries that experienced similar improvements in maternal health.

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1 Introduction

The United States experienced very big swings in fertility between the late 1930s and the early 1970s. The cohort total fertility rate[1] rose from a low of 2.27 children for women born in 1908 to a peak of 3.21 children for women born in 1932. 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, as in Becker and Barro (1988). 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. 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 across cohorts of women who were differentially exposed. The year 1936 marks the start of the rapid decline in maternal mortality for the U.S., and we use this date to identify the treated cohorts. Panel estimates suggest that for every standard deviation reduction in maternal mortality, completed fertility rises by 0.41

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[1]The Cohort Total Fertility Rate (CTFR) is a measure of the total lifetime fertility of the average woman born in a given year. Formally, let $f_{a,t}$ be the number of children born to women of age a in period t divided by the number of those women. Then, $CTFR_t = \sum_{a=15}^{a=49} f_{a,t+a}$. 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.
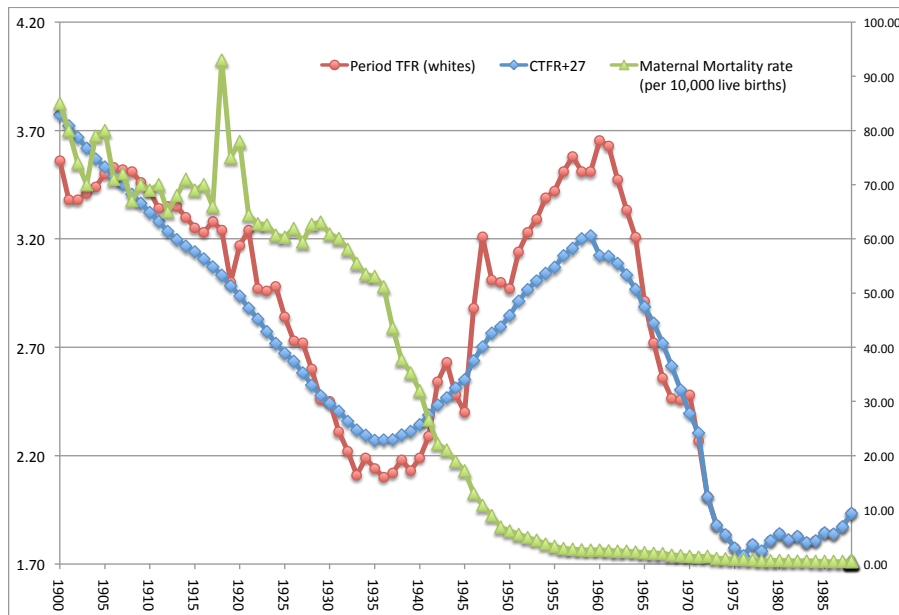
FIGURE 1: Maternal mortality, Period Total Fertility Rate and Cohort Total Fertility Rate (+27) in the U.S. 1900-1990
Source: U.S. Cohort Fertility Tables, CTFR 1917-1980, TFR 1900-1988. Produced by the National Institute of Child Health, compiled in Heuser (1976). U.S. Period Fertility Rate in Haines (2006). Maternal mortality from Vital Statistics of the United States.

children for women born in 1921-1940 relative to a control group of those born in 1913-1920, about 29% of the actual rise. The differential decline in maternal mortality accounts for over 45% of the cross-state variation in the change in fertility between cohorts.

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. For every standard deviation drop in maternal mortality, the female-male differential in graduation rates rises by 0.017 for college and by 0.046 for high school for the 1933-1950 birth cohorts. This accounts for 40% and 56%, respectively, of the actual rise relative to the control group. These findings are robust to the inclusion of economic and demographic controls, including early access to oral contraception for the treated 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 the decline in maternal mortality played a significant role in the baby bust. A standard deviation reduction in maternal mortality is associated with a decline in completed fertility by 0.47 children, or 77% of the actual decline across these groups of cohorts. Our estimates are robust to the inclusion of controls for early access to oral contraception.

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 oral contraception (Goldin and Katz, 2002, and Bailey, 2006). We interpret the rapid adoption of oral

contraception by young women in the late 1960s as spurred by a desire to reduce and postpone fertility, which originated at least in part from the improvement in maternal health and its effect on the returns to human capital investment.

This paper's main contribution is to the macroeconomic literature on the baby boom. Greenwood, Seshadri and Vanderbroucke (2005) 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[2]. 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[3].

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.[4] 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.[5] Their estimates suggest a strong positive effect, which they interpret as consistent with a rise in parental investments in the education of daughters. Our results confirm the strong impact of falling maternal mortality on women's education for the 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 demographic 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)[6]. We show that a decline in maternal mortality induces a temporary rise in fertility, which points to medical progress as an integrated explanation for both the secular trend and the medium run fluctuations in fertility.

Our findings provide new insights on the determinants of fertility in the United States and other countries that experienced similar advances in maternal health, and offer a new perspective on demographic policies in developing countries. 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, generating a rise in income per capita of over 50% via this channel. These results suggest that improving maternal health, typically a very severe problem in developing countries, could improve standards of living substantially even without a

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[2]See Albanesi (2008) for a discussion.

[3]Bailey and Collins (2009) find that differences and changes in appliance ownership and electrification in U.S. counties are negatively correlated with fertility rates from 1940 to 1960.

[4]Table 19, Goldin (1991).

[5]Bleakley (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.

[6]Doepke (2005) provides an excellent discussion.

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

2.1 Burden of Maternal Mortality and Morbidity

The maternal mortality rate (MMR),[7] 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, or just under 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, as shown in Table 1. Maternal mortality declined by only 4.5% between 1900 and 1930, whereas mortality for all causes declined by 37% for females and 32% for males. Mortality for tuberculosis dropped by over 60% in this period. The decline of maternal deaths as a fraction of all female deaths from 3.1% to 1.6% between 1900 and 1930 is mostly accounted for by the decline in births in this period.[8] In 1930, maternal mortality still accounted for 10.6% of female deaths at age 15-44 and was the second biggest cause of death for women in this age group after tuberculosis.[9]

2.2 Advances in Maternal Health

The systematic decline in maternal mortality did not start until 1936 but was precipitous in the two subsequent decades. The maternal mortality rate dropped from 51.16 per 10,000 live births in 1936 to to 2.87 in 1956, a 94% drop over a span of just twenty years. This corresponds to a -13.23% average yearly change

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

[8]The pregnancy-related mortality risk depends on age and parity. The maternal death rate has a U-shaped relation with both age and parity (Berry, 1977). The parity adjustment factors over average maternal mortality risk are 1.14, 0.62, 0.64, 0.77, 0.99, 1.12, 1.14, 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. Changes in the age and parity distribution between 1936 and the mid 1950s do not influence the pregnancy-related mortality risk.

[9]Maternal mortality exhibits a large spike during the 1918-1919 influenza epidemic, which also causes a temporary decline in the male-female mortality rate and the female-male differential in life expectancy at age 20 between 1915 and 1920. Noymer and Garenne (2000) show that this drop resulted from the effects of the influenza epidemic on female-male mortality differentials for tuberculosis. Influenza increased mortality associated with tuberculosis. Though in general tuberculosis mortality rates were higher for men, they increased for women during the influenza outbreak of 1918, temporarily closing the gender gap.

		Mortality Rates per 100,000 population, All ages			Percentage changes	
		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%
Tubercolosis	men	201	76.2	8.9	-62.1%	-88.3%
	women	187.8	65.9	3.3	-64.9%	-95.0%
Puerperal Causes		26.9	25.7	1.7	-4.5%	-93.4%
Female-Male Diff in Life Expectancy at Age 20		2	2.5	6.1	25.0%	144.0%
Deaths by cause as a percentage of all deaths						
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%		
Tubercolosis as a percentage of all deaths		11.3%	6.3%	0.7%		

Source: Vital Statistics of the United States

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 beginning in the mid-1930s and stabilized in the late 1950s.[10] The most striking decline occurs for deaths due to sepsis, which dropped from 27.5 in 1923 to 0.55 per 10,000 live births in 1955.

The decline in maternal mortality was associated with a sizable rise in the female-male differential in adult life expectancy,[11] 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.[12]

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 (Kerr, 1933). Unfortunately, there are no systematic time series data on the evolution of maternal morbidity.[13] Franks et al. (1992), the only comprehensive nationwide assessment of pregnancy related morbidity, report 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

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

[11]The 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.

[12]Rethereford (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.

[13]There 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).
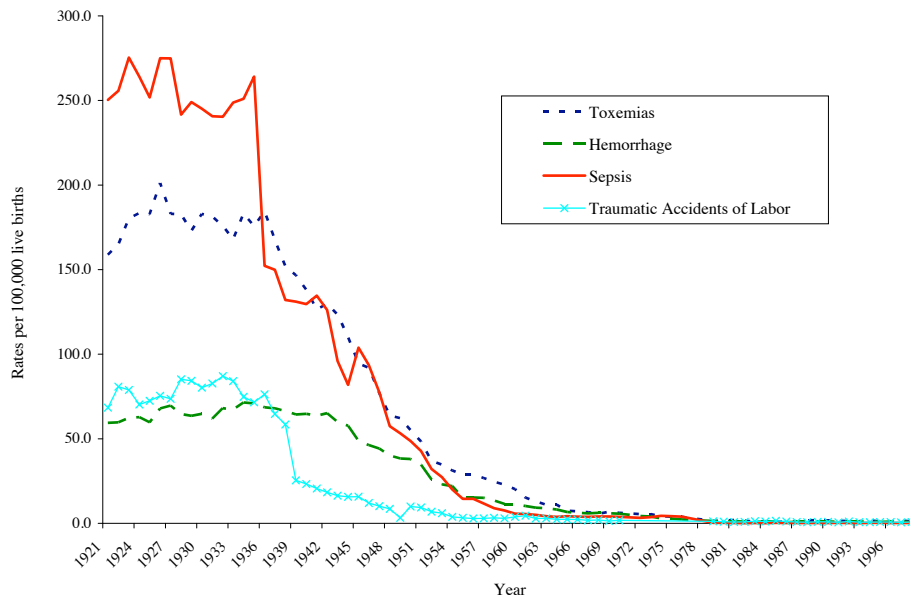
FIGURE 2: Maternal mortality by cause
Source: Vital Statistics of the United States

the United States. The corresponding statistic for the late 1920s reported in Kerr (1933) is 114.4 per 1,000 deliveries.[14] 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.[15]

2.3 Historical Background

Women were keenly conscious of the health risks associated with pregnancy and childbirth, yet it wasn't until the 1920s that maternal mortality started to be considered a major health problem in the U.S. (Leavitt, 1986).

Early efforts to improve maternal health were driven mainly by the goal of reducing infant mortality, which was very high, especially in urban areas (Meckel, 1990). The Children's Bureau, created by an act of Congress in 1912, was the first federal agency with the primary responsibility of promoting infant and child health[16]. One of its main activities was to conduct detailed statistical studies of maternal and child health outcomes.

In 1917, 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

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

[15]Weil (2004) offers an excellent discussion of this approach.

[16]For 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 modelled 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

tuberculosis in 1913, 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, explicitly targeting maternal and infant health, 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 direct subsidies for 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 the these programs, including the criteria for appropriation, is provided in Appendix B.

The Children's Bureau was also instrumental in raising awareness of the preventability of pregnancy-related mortality in the medical profession. While physicians systematically started to enter the birth room in 1850, their intervention did not contribute initially to a reduction in maternal mortality.[17] Inappropriate and excessive operative procedures were common and increased in the 1920s, leading to high rates of birth injuries for both newborns and mothers (Loudon, 1992).

The iatrogenic nature of obstetric complications and pregnancy related mortality in the early phases of the medicalization of childbirth received widespread public attention following the publication of the proceedings from the 1930 White House Conference on Child Health and Protection, sponsored by the Children's Bureau.[18] More than two-thirds of all maternal deaths were found to be preventable for a nationally representative sample, and many physicians were found to lack the most basic obstetric knowledge (CDC, 1999). [19]

These reports precipitated efforts to standardize obstetric practices and train physicians. Alongside a number of scientific discoveries and advances in general medicine that took place in the 1930s, these developments led to a fast decline in maternal mortality.

As can be seen from figures 1 and 2, the year 1936 brought a clear break in pregnancy related mortality. In 1936, sulphonamides, the first type of antibiotic, were introduced. These drugs were relatively cheap to produce and diffused very rapidly, bringing down mortality for several 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.

There was also a widespread improvement in obstetric care in hospitals, following the establishment of the American Board of Obstetrics and Gynecology in 1930 (Dannreuther, 1931). 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.

Even as the quality of obstetric care was improving in many hospitals starting in the 1930s, access to

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[17]Thomasson and Treber (2008) analyze the consequences of the hospitalization of childbirth on maternal mortality in the US.

[18]Convened in 1930 by President Herbert Hoover, the White House Conference on Child Health and Protection was called "to study the present status of the health and well-being of the children of the United States and its possessions, to report what was being done, to recommend what ought to be done and how to do it." The resulting "Child Health Protection, Fetal Newborn, and Maternal Mortality and Morbidity Report," published in 1933, and the committee reports laid the groundwork for the Fair Labor Standard Act of 1938 and for inclusion in the Social Security Act of 1935 of the federal-state programs for aid to dependent children, crippled children, and maternal and child-health/welfare services.

[19]Similar findings emerged from a study of 2,041 maternal deaths in childbirth by the New York Academy of Medicine, published in 1933.

such care was still severely limited. Geographical distance was a factor in rural areas prior to the widespread use of automobiles. The other major obstacle was cost. 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).[20] The development of the first Blue Cross hospital pre-payment plans starting in the late 1920s and other forms of employer provided health insurance in the 1930s helped to alleviate these costs for a very small number of households (Starr, 1982). The federal subsidies for obstetric care introduced by the Social Security Act were instrumental in increasing access to high quality obstetric services. Appropriations under this program started in 1936, which, as can be seen in figure 2, corresponds the start of a steep decline in pregnancy-related mortality from toxemia and traumatic accidents of labor, in addition to septicemia, which can be clearly connected to the sulfa drugs and blood banking.

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 pregnancy-related death, and blood banking, along with 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.[21]

TABLE 2: Live Births by Attendant, U.S. States

	1940		1946		1954	
Percentage of total	Median	Min,Max	Median	Min,Max	Median	Min,Max
In hospital	61	13.9, 91.4	89.6	38.6, 98.9	98	60, 100
At home with physician	36.6	8.6, 76.7	9	1, 42.6	2	0,11
At home without physician	0.71	0.01, 49.29	0.6	0, 36.9	0	0,30
	Correlation with maternal mortality					
Percentage of total	1940		1946		1954	
In hospital	-0.31		-0.42***		-0.73***	
At home with physician	0.09		0.16		0.59***	
At home without physician	0.47***		0.68***		0.74***	

Source: Children's Bureau. *** Significant at 1% level. Statistics refer to aggregate state population.
Alaska and Hawaii are excluded from the sample.

The impact of hospitalization on the drop in maternal mortality can be seen clearly by looking at state level statistics on live births by attendant, reported in Table 2. There is a sharp rise in the minimum and median percentage of births in hospitals, accompanied by a decline in both the percentage of births at home attended by a physician and the percentage of home births unattended by a physician. There is a strong negative correlation between maternal mortality and percentage of births in hospital for all years in the analysis, and a strong positive correlation between the percentage of births unattended by a physician and maternal mortality for all years of the analysis. The correlation between maternal mortality and births at

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

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

home attended by a physician is also positive for all years, but it becomes sizable and significant at the 1% level only for 1954.

3 Theory

We now examine a version of Becker and Barro's (1988) 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 C.2 analyzes a general version of the model, which features individuals and offspring of both genders, delivering the same results.

Women derive utility from consumption and from the quantity and quality of their children. The latter simply corresponds to the children's endowment of human capital, which can be raised via a costly maternal investment and raises the children's lifetime utility. Mothers may die in childbirth. Their mortality risk is a function of the pregnancy-related mortality rate, which they take as given, and of the number of births.

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

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

where $e \geq 0$ represents mother's human capital and e' is a mother's investment in the daughters' human capital, b is the number of births.

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

The parameter $\mu \in [0, 1]$ corresponds to the pregnancy-related mortality probability associated with each birth, and $(1 - \mu b)$ is the probability that a woman will survive childbearing. The function $u(\cdot)$ is the utility from mothers' consumption, which 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 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 for the utility from children, with $\kappa(\cdot) \in [0, 1)$ $\kappa'(\cdot) > 0$ and $\kappa''(\cdot) \leq 0$ and $lim_{x \to 0} \kappa'(x) = +\infty$. The functions $v(\cdot)$, $u(\cdot)$ and $\kappa(\cdot)$, are twice continuously differentiable.

The function $U(e; \mu)$ is the value function for a cohort's problem, and can be interpreted to correspond to the "quality" of a given cohort. We index this value function by the pregnancy-related mortality risk, which is allowed to vary across cohorts. All other parameters also potentially vary across cohorts, but since our focus is on maternal mortality, we omit indexing on other parameters to simplify the notation. We assume that mothers have perfect foresight on the value of parameters entering their children's utility function, and denote the daughters' utility and health parameters with a prime.

Under the stated assumptions, $U'(e; \mu) > 0$ and $U''(e; \mu) \leq 0$. We further restrict $u(\cdot)$ and $v(\cdot)$ to ensure

$U(e; \mu) > 0$ for $e \geq 0$ and $\mu \in [0, 1]$. 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 1, jointly with the assumptions on $v(\cdot)$, $\kappa(\cdot)$ 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:

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

with equality for $e > 0$, and

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

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

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

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 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, which in the model corresponds to parameter μ. This property is intuitive, given that higher pregnancy-related mortality 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, increasing 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 cohorts that experience the decline in childbearing years, after their endowment of human capital has been chosen. Successive cohorts of women, who experience the

decline in their formative years, will have higher human capital endowment and greater opportunity cost of children. Their desired fertility will thus be lower than for the initially exposed cohorts.

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:*

$$\frac{\partial b}{\partial \mu} \leq 0, \tag{4}$$

$$\frac{\partial e'}{\partial \mu} \leq 0, \tag{5}$$

if and only if:

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

Proof: In Appendix C.1. ∎

Proposition 1 states that fertility and mothers' investment in daughters' human capital rise in response to a reduction in maternal mortality 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, a standard assumption in the class of fertility choice models in the Barro-Becker tradition. Condition (6) restricts the severity of this trade-off. Given that the dynastic discount factor $\kappa(\cdot)$ is concave and satisfy the Inada conditions, this restriction will be always satisfied if initial fertility is low enough.

To summarize, desired fertility and daughters' human capital investment rise in response to a permanent reduction in pregnancy-related mortality risk, 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 risk.

Proposition 2 *Assumption (1) implies:*

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

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

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

Proof: In Appendix C.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 maternal mortality risk 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. Condition (6) is necessary and sufficient for investment in daughters' human capital to increase with mothers' human capital endowment.

Taken together, these results 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. Since successive cohorts benefit from greater parental investments in human capital, they will have a higher opportunity cost of having children 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. Since the effects on women's human capital are permanent, the rise in women's human capital may well generate a permanent reduction in fertility once the advances in maternal health are exhausted, if the returns to human capital are high enough.

Condition (6) is more likely to hold if initial fertility is low, as was the case in the US, where fertility reached a historical low in the early 1930s. More in general, 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.

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 C.1.2, we also derive the response of desired fertility and human capital investment to a temporary decline in pregnancy-related mortality risk. 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. 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.

The simple model discussed in this section only features mothers and daughters. Appendix C.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 numbers. The dynastic discount factor is defined over the total number of children surviving infancy. 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, $\{e_f, e_m\}$, 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.

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. However, if the utility cost of pregnancy-related maternal morbidity is separable from the utility from consumption, the predictions of the model remain intact[22].

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, which may affect the timing of fertility. This effect cannot be analyzed in the current model given that there is only one stage in life.

4 Empirical Analysis

We now proceed to examine the empirical links among the decline in maternal mortality, fertility and women's human capital.

Two features of the decline in maternal mortality stand out clearly. First, maternal mortality did not decline substantially until 1936, when it started to drop sharply, reaching modern levels by the late 1950s.[23] This pattern allows us to identify quite precisely the cohorts of women who experienced the improvements in maternal health at different stages of their life cycle. The second feature is the substantial cross-state variation in the magnitude of the drop in maternal mortality. As shown in Table 3, the cross-state average maternal mortality in 1930 was 71 deaths per 10,000 live births, with a minimum of 49 (Utah) and a maximum of 114 (South Carolina). Maternal mortality dropped in all states in subsequent years, yet a significant cross-state dispersion in maternal mortality continued to prevail due to the variation in the size and the timing of its decline. The drop in maternal mortality ranged between 10 and 62 deaths between 1940 and 1930, between 17 and 53 deaths between 1950 and 1940, between 5 and 25 deaths between 1960 and 1950, and between 1 and 9 deaths between 1970 and 1960.

There is also substantial cross-state dispersion in fertility. As can be seen from Table 3, the cross-state average for the crude birth rate is 19.84 in 1930, with a minimum of 14.1 (Washington) and maximum of 28.4 (Colorado). The crude birth rate declined by an average 3.4% between 1930 and 1940, though the cross-state dispersion is sizable. The mean crude birth rate rose by 30.4% between 1940 and 1960, with a minimum change of -2.46% and a maximum of 51.23%. It then started to decline.

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

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

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.

[23]This time pattern in the evolution of maternal mortality prevails in all states. Specifically, 30 states experience the start of the maternal mortality drop between 1930 and 1935, 4 states experience the start of the maternal mortality drop in or after 1936, and the other states experience the start of the drop in 1920 or 1929.

TABLE 3: Cross-state variation

	MMR*			CBR**		
	(per 10,000 live births)			(per 1,000 population)		
	Levels					
	Mean	Min, Max	$Coeff.\,of$ $Variation$	Mean	Min, Max	$Coeff.\,of$ $Variation$
1930	70.68	49, 114	0.24	19.84	14.1, 28.4	0.16
1940	38.06	18.3, 68.8	0.32	18.98	14.4, 27.8	0.16
1950	8.81	1.8, 26.9	0.58	24.59	20.2, 32.4	0.11
1960	3.68	1.2, 10.6	0.56	24.44	21.2, 32.26	0.09
1970	2.14	0.4, 7.2	0.55	18.57	16.3 25.3	0.09
	Percentage Change					
	Mean	Min, Max		Mean	Min, Max	
1940-1930	-46	-68.45, -15.54		-3.4	-34.05, 30.14	
1960-1940	-131.5	-160.08,-56.83		30.4	-2.46, 51.23	
1970-1960	-35	-83.33, 14.29		-23.9	-33.14, -14.29	

* Aggregate mortality rates. ** Crude birth rate: Number of births per 1,000 population. Sources: See Appendix A.

The goal of our empirical analysis is to identify 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 and in the magnitude of its drop as exogenous. We estimate the impact of the drop in maternal mortality on the change in fertility by adopting a difference in difference approach, where one difference is across cohorts and the other is across states. In order to attain a homogeneous sample, we restrict attention to white women only.[24] 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.

There are two components of the estimation design. The first is the measure of maternal mortality rate to be used as a treatment. Such a measure must be relevant for subjects' fertility decisions or for parental investments in daughters' education. At the same time, the subjects' fertility behavior or education should not influence our measure of the treatment, to avoid concerns of joint endogeneity or reverse causation. We introduce the notion of *reference maternal mortality* as the average maternal mortality in the state in a given age range chosen to alleviate these two concerns.

The second component of the estimation design is the choice of the treatment and control groups. Our criteria for inclusion in the treatment group is that birth cohorts must be young enough for their fertility decisions or educational attainment to respond. The decline in maternal mortality starts in 1936, and the first public reports in standard media outlets, such as daily newspapers, date to late 1937. Thus, we take 1938 as the turning point. For the fertility analysis, we classify as treated the cohorts who were in childbearing age in 1938. For the educational analysis, we consider treated cohorts who in 1938 were young enough for their parental investments in education to respond.

The choice of control group is driven by two considerations. The first is data availability. 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. The second consideration is to minimize other factors that might be varying across cohorts and that are hard to control for.

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[24]The reduction in maternal mortality also occurs for the non-white population, though later than for whites. Non-whites also experience a baby boom, which is slightly smaller in magnitude in percentage terms than whites.

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:

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

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:

$$ZZ_{st} = MMR_{st}^{ref}, \tag{10}$$

where MMM_{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 in[25], thus ensuring that the fertility behavior of the women included in the estimation does not affect their reference maternal mortality.

The baseline specification assumes women born in 1921-1940 were treated. Thus, the youngest treated cohort was 17 in 1938. Women born between 1913 and 1921 are included in the control group. The sensitivity analysis explores alternative definitions of reference maternal mortality and criteria for inclusion in the untreated and treated groups.

All specifications include a control for infant mortality, which has been found to be related importantly to fertility (Preston, 1978, Haynes and Preston, 1991, Doepke, 2005). Thus, for the baseline specification, $X_{st} = IMR_{st}^{ref}$, 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 treated (t') and untreated (t) cohorts:

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

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

We are interested in the effects of the maternal mortality decline on completed fertility. We adopt the statistic children ever born (CHBORN) at age 35-44 from the US Census [26], as the main fertility outcome, as the median age of last birth for the cohorts included was 29. We also consider number of children under 13 living in the household (NCHILD) at age 35-44 for robustness, though this measure may be biased downwards due to grown children having left the household. For some specification, 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 in a given age group: all women (All), married

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[25]Specifically, 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.

[26]Fertility and education data are from the US Census. Appendix A provides a detailed description of the data.

women (Married), and married women with children (Married 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 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 4 presents summary statistics for the sample we include in the baseline specification, including the mean and standard deviation of the fertility outcomes in the treated and the control groups, the mean and standard deviation of reference maternal mortality in the treatment and control groups and similar statistics for reference infant mortality. CHBORN is the fertility outcome that experiences the largest rise across cohorts, from 2.56 in the control group to 4.02 in the treatment group for married women.

TABLE 4: Fertility_ Baseline Specification

Summary Statistics

Control Group: Birth Years 1913-1920									
		Reference Maternal Mortality (Age 15-20)				**Reference Infant Mortality (Age 15-20)**			
	Mean	53.941				Mean	54.923		
	St. Dev.	7.9609				St. Dev.	13.1061		
Fertility Outcome	CHBORN	CHBORN	CHBORN	NCHILD	NCHILD	NCHILD	NCHLT5	NCHLT5	NCHLT5
Age	35-44	35-44	35-44	35-44	35-44	35-44	23-32	23-32	23-32
Sample	Married	All	Married with Children	Married	All	Married with Children	Married	All	Married with Children
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
Treated Group: Birth Years 1921-1940									
		Reference Maternal Mortality (Age 15-20)				**Reference Infant Mortality (Age 15-20)**			
	Mean	13.944				Mean	32.7648		
	St. Dev.	2.8745				St. Dev.	7.1215		
Fertility Outcome	CHBORN	CHBORN	CHBORN	NCHILD	NCHILD	NCHILD	NCHLT5	NCHLT5	NCHLT5
Age	35-44	35-44	35-44	35-44	35-44	35-44	23-32	23-32	23-32
Sample	Married	All	Married with Children	Married	All	Married with Children	Married	All	Married with Children
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 5 presents the estimation results for the baseline specification (top panel, heading "Panel"). The baseline estimates suggest that the decline in maternal mortality had a strong positive effect on the rise in fertility of treated cohorts relative to untreated cohorts. For CHBORN at age 35-44 in the specification that includes married women, the estimated coefficient suggests that a decline in maternal mortality equal to

one standard deviation of pre-treatment maternal mortality is associated with a rise in CHBORN of 0.41 or 16%. The cross-state average change in CHBORN between treated and untreated cohorts was 1.46, or 57%, thus a one standard deviation decline in maternal mortality can account for 28% of the change in fertility. The estimated coefficient is significant at the 1% level and the specification explains 47% of the cross-state variation in fertility.

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 one standard deviation decline in infant mortality is associated with a change in fertility of −0.22. The inclusion of infant mortality does not affect the estimated coefficient for maternal mortality.

The results for the All and Married with children samples are consistent with those for the sample including all women. For all women, the estimated coefficient, significant at the 1% level, implies that a one standard deviation decline in maternal mortality is associated with a 0.39 rise in fertility or 16%, which accounts for 27% of the actual rise in fertility. For the sample of married women with children, the estimated coefficient drops in magnitude though it is still highly significant. A one standard deviation decline in maternal mortality is associated with a rise in fertility of 0.23 or 8% and accounts for 18% of the rise in fertility between the treatment and control groups.

Results are similar for the other measures of fertility. For NCHILD at age 35-44 for the sample married women, the estimated coefficient suggests that one standard deviation decline in maternal mortality is associated with a 0.20 or 10% rise in the number of children in the household, which accounts for 47% of the actual rise in this statistic between treated and untreated cohorts. The coefficient is significant at the 1% level and the specification accounts for 27% of the variation in fertility across cohorts and across states. For NCHLT5 at age 23-32, a one standard deviation decline in maternal mortality is associated with a 0.07 or 9% rise for married women, which accounts for 28% of the change in this variable between treated and untreated cohorts. Similar results obtain for the sample of all women and the sample of married women with children.

Regression Results (1)

Specification		Panel								
Fertility Outcome		CHBORN	CHBORN	CHBORN	NCHILD	NCHILD	NCHILD	NCHLT5	NCHLT6	NCHLT7
Age		35-44	35-44	35-44	35-44	35-44	35-44	23-32	23-32	23-32
Sample		Married	All	Married with children	Married	All	Married with children	Married	All	Married with children
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$^{ref}_{st}$ (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$^{ref}_{st}$ (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
R-squared		0.488	0.5331	0.2799	0.3007	0.365	0.2505	0.2922	0.3688	0.216
Predicted change in fertility outcome for one st. dev. change in pre-treatment reference maternal mortality		0.41635507	0.3900841	0.23166219	0.20459513	0.20220686	0.1990225	0.07085201	0.0875699	0.04139668

Specification		Panel, treated only								
Fertility Outcome		CHBORN	CHBORN	CHBORN	NCHILD	NCHILD	NCHILD	NCHLT5	NCHLT6	NCHLT7
Age		35-44	35-44	35-44	35-44	35-44	35-44	23-32	23-32	23-32
Sample		Married	All	Married with children	Married	All	Married with children	Married	All	Married with children
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$^{ref}_{st}$ (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$^{ref}_{st}$ (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
R-squared		0.5607	0.6799	0.1033	0.2923	0.3751	0.2966	0.1724	0.1963	0.1405

Specification		IV								
Fertility Outcome		CHBORN	CHBORN	CHBORN	NCHILD	NCHILD	NCHILD	NCHLT5	NCHLT6	NCHLT7
Age		35-44	35-44	35-44	35-44	35-44	35-44	23-32	23-32	23-32
Sample		Married	All	Married with children	Married	All	Married with children	Married	All	Married with children
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$^{pre}_s$*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$^{ref}_{st}$ (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
R-squared		0.46	0.5064	0.2696	0.2954	0.3604	0.2315	0.2867	0.3544	0.2186

(1) Baseline specification. All regressions include state and cohort effects. Control group: 1913-1920. Treated group: 1921-1940.
(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) The instrument for reference MMR in each state is the average reference MMR for the control cohorts in each state.

4.1.2 Sensitivity

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

Alternative Specifications We estimate equation (9) including only cohorts in the treatment group. 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 5 (middle panel, heading "Panel, treated only") and confirm those for the baseline specification. For the married sample, the estimates imply that CHBORN at age 35-44 rises by 0.21 for a one standard deviation drop in maternal mortality. The coefficient is significant at the 1% level, and the adjusted R-squared coefficient is at 0.53, suggesting that this specification has considerable explanatory power. 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 the Married and Married with children samples. 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 in the control group[27], which we denote with MMR_s^{pre}, as an instrument for the magnitude of the treatment. 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 group[28]. Given that a larger initial value of maternal mortality corresponds to a larger decline, a *positive* value of the coefficient α_1 indicates that the decline in maternal mortality is associated with a rise in fertility between treated and untreated cohorts.

The estimation results are presented in Table 5 (bottom panel, heading "IV"). 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^{pre} and the average decline in reference maternal mortality, between the treated and control 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 treated and control cohorts of 1.29 or 50%, which accounts for 89% of the actual change.

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 in equation (11).

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

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

The estimates, reported in Appendix D, Table 15, 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, and explain close to 50% of the cross-state and cross-cohort variation.

Controls We now control progressively for several state level indicators to assess the potential for omitted variable bias. The details on the definition and data sources for each indicator are reported in Appendix A.

We first consider a set of health indicators, including the male mortality rate (number of male deaths per 100,000 population), the tuberculosis mortality rate (number of tuberculosis deaths per 100,000 population), the malaria mortality rate (male deaths per 100,000 population). The male mortality rate is an indicator of general health conditions in the state[29], while tuberculosis was the top cause of death for both men and women in the control group. 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 (number of deaths per 100,000).

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 6 (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 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 are basic demographic indicators, intended to capture some of the cross state variation in fertility behavior. 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 proxie 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. The results are displayed in Table 6 (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). This suggests that economic conditions may affect the timing of fertility.

Finally, we control for a set of indicators intended to proxy for state level political and cultural preferences. Group 1 simply includes the literacy rate in 1930. This indicator is potentially linked to the ability to absorb medical knowledge in the control group. Moreover, literacy is linked to the diffusion of basic schooling, which was related strongly to progressive values, including sensitivity regarding maternal health (Skopcol, 1992). Group 2 includes an indicator of the acceptance of women's suffrage, which can be linked to maternal

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[29]We exclude female deaths from this measure since it would be affected by maternal mortality.

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 can be found in 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.

Group 3 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 Sheppard-Towner Act was a federal grant-in-aid program to incentivize educational activities promoting maternal and infant health. Part 1, Title V of the Social Security Act provided federal funds, also on a grant-in-aid basis, to directly subsidize obstetric and infant care. The legislation is described in more detail in Appendix B. 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 last indicator we consider, Group 4, 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 were the most generous and popular program, and were enjoyed directly only by men (Altshuler and Blumin, 2009)[30]. Housing benefits were also substantial, and jointly with the education benefits 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 6 (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. However, the magnitude of this effect is small as a rise in mobilization rates of 0.05 raises CHBORN at age 35-44 by 0.04[31]. By contrast, a one standard deviation decline in maternal mortality is associated with a rise in the outcome of 0.24, in the specification that controls for mobilization rates.

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 16 (top panel, heading "Fertility") in Appendix D. Specifically, we consider women born in 1905 − 1915 (Control

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[30]See footnote 32 for more details.
[31]Mobilization ranged between 0.41 and 0.54, with a standard deviation of 0.034.

TABLE 6: Fertility_ Baseline Specification

Regression Results with Controls

Panel Specification (1), (2)

Health (3)

Dependent variable		CHBORN 35-44	NCHILD 35-44	NCHLT5 23-32
Age				
1: Male Mortality				
MMR^{ref}_{st} (4)		-0.0513	-0.0255	-0.0087
	t-stat	-12.7238	-11.28	-6.234
Adj R-squared		0.4716	0.2739	0.2657
2: Sulfa Related Mortality				
MMR^{ref}_{st} (4)		-0.0486	-0.0259	-0.0091
	t-stat	-12.1345	-11.3924	-6.4356
Adj R-squared		0.4857	0.2734	0.2648
3: TB Mortality				
MMR^{ref}_{st} (4)		-0.0485	-0.0259	-0.009
	t-stat	-12.1351	-11.3623	-6.4034
Adj R-squared		0.4866	0.2733	0.2646
4: Malaria				
MMR^{ref}_{st} (4)		-0.0563	-0.0279	-0.0098
	t-stat	-13.9627	-12.3733	-7.0213
Adj R-squared		0.4826	0.2926	0.2745

Economic and Demographic

Dependent variable		CHBORN 35-44	NCHILD 35-44	NCHLT5 23-32
Age				
1: Personal Income, Unemp.				
MMR^{ref}_{st} (4)		-0.0249	0.0116	0.0024
	t-stat	-5.0038	4.9885	1.4132
Adj R-squared		0.4978	0.5183	0.3262
2: White, Foreign Born, Farm				
MMR^{ref}_{st} (4)		-0.0442	-0.0106	-0.0071
	t-stat	-9.8117	-4.7059	-4.5222
Adj R-squared		0.501	0.4539	0.3004
3: Share Public				
MMR^{ref}_{st} (4)		-0.0471	-0.0206	-0.0074
	t-stat	-11.507	-9.2254	-5.1906
Adj R-squared		0.48	0.3252	0.2763
4: Share Health				
MMR^{ref}_{st} (4)		-0.0337	-0.0035	-0.0022
	t-stat	-7.4441	-1.5489	-1.4165
Adj R-squared		0.4943	0.4374	0.3037

State Characteristics (5)

Dependent variable		CHBORN 35-44	NCHILD 35-44	NCHLT5 23-32
Age				
1: Literacy 1930				
MMR^{ref}_{st} (4)		-0.0437	-0.026	-0.0082
	t-stat	-12.3629	-12.6301	-6.3881
Adj R-squared		0.4309	0.1604	0.1302
2: Acceptance of Women's Suffrage				
MMR^{ref}_{st} (4)		-0.0453	-0.0275	-0.0082
	t-stat	-13.123	-13.7082	-6.4618
Adj R-squared		0.4443	0.1813	0.1375
3: Sheppard-Towner & Social Security Act				
MMR^{ref}_{st} (4)		-0.0437	-0.0264	-0.0077
	t-stat	-12.5337	-13.1043	-6.0721
Adj R-squared		0.431	0.1678	0.1339
4: Mob. Rates applied to 1922-1928 cohorts				
MMR^{ref}_{st} (4)		-0.0325	-0.0272	-0.0085
	t-stat	-8.559	-12.0909	-6.0353
Mob. Rate		0.8208	-0.0453	-0.0498
	t-stat	6.8513	-0.6396	-1.1235
Adj R-squared		0.4498	0.1591	0.1288

(1) Baseline specification. All regressions include state and cohort effects. Control group: 1913-1920. Treated group: 1921-1940.
(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.

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 8). 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 treatment group, using the same definition of reference maternal mortality as in the falsification exercise (Treatment Group in Table 16, 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 control and treated groups. Results for CHBORN at age 35-44 for Married women are presented in Table 7.

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 control and treatment groups. Column 5 repeats the baseline results. For column 6, the control group is comprised by the 1910-1917 birth cohorts, while the treatment group corresponds to the 1918-1935 cohorts. For column 7, the 1910-1915 cohorts are the control group, while the 1916-1930 cohorts are in the treatment group. Finally, for column 8, the control group corresponds to the 1920-1929 birth years, and the treatment group to the 1930-1940 birth years. Once again, the estimation results confirm our baseline specification. The magnitude of the estimated coefficient (in absolute value) on maternal mortality is mostly very close to the one for the baseline specification and significant at the 1% level.

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

Table 7: Included Cohorts and Reference Maternal Mortality

Specification		Panel, treated only (1)				Panel (2)			
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
Column		1	2	3	4	5	6	7	8
Control Cohorts						1913-1920	1910-1917	1910-1915	1920-1929
Treated Cohorts		1921-1940	1921-1940	1921-1940	1921-1940	1921-1940	1918 - 1935	1916-1930	1930-1940
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
Cross-state Average for Treated Cohorts		13.944	25.6366	19.8484	32.4996	13.944	22.3488	31.97	15.34
Constant		0.0145	0.0172	0.0186	0.0126	0.0125	-0.0041	-0.0144	0.0118
	t-stat	4.9044	6.3743	6.0394	3.9457	4.6696	-1.3434	-3.5734	4.4387
MMR^{ref}_{st} (3)		2.6344	2.5373	2.5284	2.6635	2.9486	3.6398	4.1507	2.7017
	t-stat	28.4758	27.1616	26.0407	25.7019	28.8387	30.4086	27.6723	30.9591
IMR^{ref}_{st} (4)		-0.0203	-0.0157	-0.0192	-0.0121	-0.0248	-0.0204	-0.018	-0.0177
	t-stat	-9.002	-9.7359	-9.4732	-6.5817	-15.028	-11.0093	-7.8296	-9.1321
Adj R-squared		0.5208	0.5184	0.5207	0.4913	0.5159	0.5669	0.5458	0.5287
R-squared		0.5453	0.543	0.5452	0.5173	0.5335	0.584	0.568	0.5516
Model p-value		0	0	0	0	0	0	0	0

(1) Panel estimates, including only treated cohorts. All regressions include state and cohort effects. Treated group: 1921-1940.
(2) Baseline specification. All regressions include state and cohort effects. Control group: 1913-1920. Treated group: 1921-1940.
(3) Reference maternal mortality is the average maternal mortality in the state at age 15-20 for each cohort.
(4) Reference infant mortality is the average infant mortality in the state at age 15-20.

results, displayed in Table 8, suggest that the decline in maternal mortality was associated with a significant rise in marriage rates. A one standard deviation decline in pre-treatment maternal mortality is associated with a 4% rise in the marriage rate in the baseline specification. The estimates for the IV specification are not significant.

We also investigate the effect of the decline in maternal mortality on childlessness. Childlessness 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 8. 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 the control group to 7% in the treatment group. The IV estimates confirm these results.

TABLE 8: Marriage and Childlessness

Regression Results (1)

Specification	Marriage Rates		Childlessness							
	Panel	IV	Panel	IV	Panel	IV	Panel	IV	Panel	IV
Dependent Variable	MARRIED	MARRIED	CHBORN=0	CHBORN=0	CHBORN=0	CHBORN=0	NCHILD=0	NCHILD=0	NCHILD=0	NCHILD=0
Age	23	23	35-44	35-44	35-44	35-44	23-32	23-32	23-32	23-32
Sample	All	All	All	All	Married	Married	All	All	Married	Married
Constant	-5.8043	-10.4791	-0.7827	0.1126	2.583	0.044	-12.7715	0.178	-4.7401	0.1536
t-stat	-1.9671	-4.64	-0.7041	8.3932	2.6075	3.6701	-12.0277	13.3136	-3.5764	9.6462
MMR^{ref}_{st} (2)	-0.0031	-0.0002	-0.0004	0	0.0002	-0.0001	0.0018	-0.0004	0.0011	-0.0005
t-stat	-4.7509	-0.8125	-1.6074	-0.1607	0.7492	-0.8055	7.7157	-4.3838	3.7205	-4.5012
IMR^{ref}_{st} (3)	0.0029	0.0016	0.0006	-0.0003	0.0001	0.0011	-0.0001	-0.0018	-0.0006	-0.001
t-stat	3.3705	2.1705	1.911	-1.7026	0.5022	7.229	-0.3732	-11.0171	-1.4618	-5.363
Adj R-squared	0.4617	0.5364	0.3767	0.3733	0.2498	0.2415	0.303	0.2367	0.1654	0.1683
R-squared	0.4817	0.5536	0.3999	0.3962	0.2777	0.2692	0.329	0.2645	0.1964	0.1986
Predicted change in fertility outcome for one st. dev. change in pre-treatment reference maternal mortality	0.04062891	0.00262122	0.00318436	0	-0.00159218	-0.00079609	-0.01432962	-0.00318436	-0.00875699	-0.00398045

Summary Statistics

Dependent Variable	MARRIED	CHBORN=0	CHBORN=0	NCHILD=0	NCHILD=0
Sample	All	All	Married	All	Married
Age	23	35-44	35-44	23-32	23-32
Control Group: Birth Years 1913-1920					
Mean	0.9362	0.0999	0.107	0.0807	0.1033
St. Dev.	0.2422	0.0213	0.0491	0.0266	0.0339
Treated Group: Birth Years 1921-1940					
Mean	0.9765	0.107	0.074	0.1014	0.1027
St. Dev.	0.093	0.0491	0.0219	0.026	0.0292

(1) Baseline specification. All regressions include state and cohort effects. Control group: 1913-1920. Treated group: 1921-1940.
(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 9. 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 one standard deviation decline in maternal mortality is associated with a 4% 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. For robustness we run also an IV version of all specifications. The IV estimates completely confirm the baseline results.

Regression Results (1)

Specification		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
R-squared		0.1323	0.1983	0.0626	0.4415	0.1703	0.1852	0.0688	0.3481
Model p-value		0	0	0.0013	0	0	0	0.0001	0
Predicted change in outcome for one st. dev. drop in reference maternal mortality		0.1990225	0.24041918	-0.00238827	-0.02308661	0.238827	0.24439963	-0.0238827	-0.00716481

Summary Statistics

Fertility Outcome		Control Group: 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
		Treated Group: 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		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
R-squared		0.5955	0.4209	0.3873	0.4306	0.5841	0.3447	0.2988	0.3725
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

Fertility Outcome		Control Group: 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
		Treated Group: 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) Baseline specification. All regressions include state and cohort effects. Control group: 1913-1920. Treated group: 1921-1940.
(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 3, 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. As discussed in Goldin, Katz and Kuziemko (2006), the scarcity of job opportunities during the Great Depression may have provided an incentive to attend college for men. Moreover, men were the exclusive recipients of the substantial educational benefit program comprised in the GI Bill (Altshuler and Blumin, 2009)[32]. More than 10% of veterans born between 1922 and 1928 achieved a bachelor's of arts using GI benefits (Burns and Turner, 2002), and the gender disparity in access probably contributed to the clear widening of the gender differential in college graduation rates for those cohorts.

Starting with the 1935 birth year, both the female/male ratio and the female-male difference in college graduation rate started rising sharply, inverting the trend prevailing for the 1910-1930 cohorts. Women born in 1935 would mostly have started their schooling by 1941 and they would have graduated from high school by the mid 1950s. Their parents would have been aware of the progress in maternal health, and according to the theory, they would have responded by increasing their investment in daughter's education. Our empirical analysis seeks to examine the link between the decline in maternal mortality and various measures of women's educational attainment.

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[32] Only 2% of the 16 million World War II veterans eligible for GI Bill educational benefits were female. The female beneficiaries received lower stipends than the male counterparts as their stipend did not rise with the number of dependents. See Altshuler and Blumin (2009) for more details.
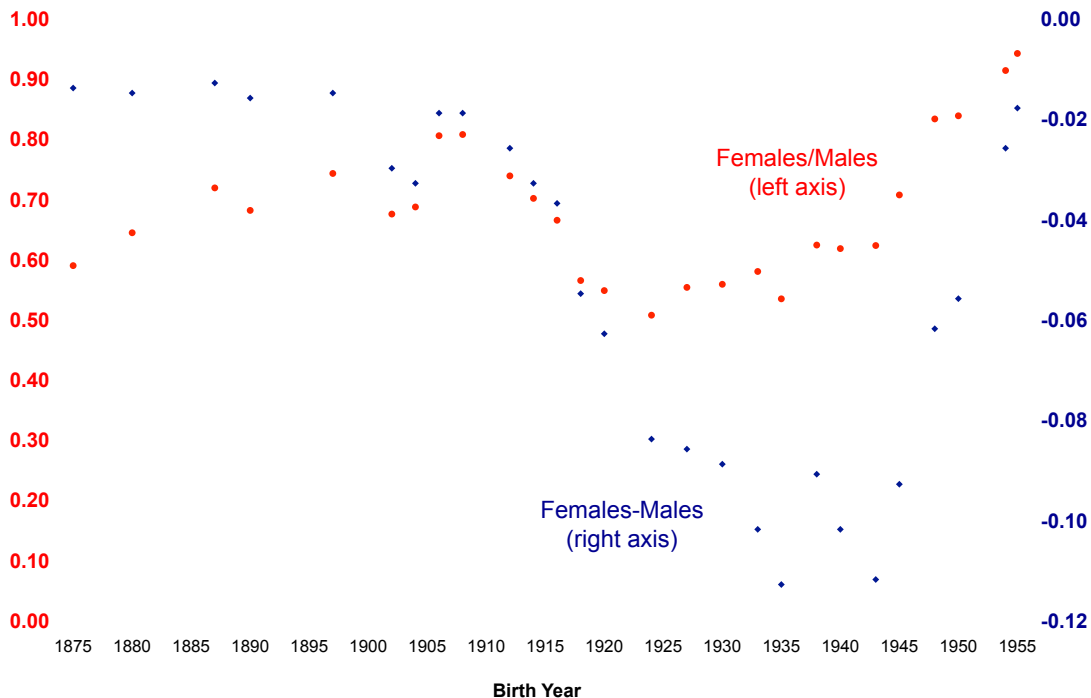
FIGURE 3: Gender differentials in college graduation rates.

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 23-32[33] 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 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:

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

where $g = f, m$ stands for gender, and μ_{st}, ν_{gt}, δ_{gs} correspond to state-cohort, gender-cohort and gender-state interactions[34]. As for fertility, the baseline specification is a panel regression that includes both treated and untreated cohorts, 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 assumptions on the age range for the calculation of reference maternal mortality and the inclusion

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[33]Given the small incidence of post-graduate and continuing education for the cohorts we consider, education was mostly completed by age 23-32. We also consider educational outcomes at age 23, at age 35 and at age 35-44 as a robustness exercise. The findings are consistent with those for the baseline specification.

[34]This specification follows Jayachandran and Lleras-Muney (2008).

in the treated group 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. The restricted age range for the calculation of reference maternal mortality ensures that the subjects' education cannot affect reference maternal mortality, thus removing the possibility of reverse causation.

For the baseline specification, individuals age -12 (born in 1950) to 5 (born in 1933) in 1938 are included in the treated group. Thus, the treated cohorts are age 5 or younger in 1938, the year in which reports on the decline in maternal mortality become available in the media. At age 5, parental investments in education at all grade levels can potentially respond. The control group comprises birth years 1919-1932. The choice of the control group is driven in part by data availability, as for the fertility analysis. We conduct sensitivity analysis on the definition of treatment and controls groups.

Table 10 provides summary statistics on the control and treatment groups in the baseline specification. For both the treatment and control 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 in the control group and 9.1 for the treatment group.

4.2.1 Baseline Results

The results for the baseline specification are presented in Table 11 (top panel, heading "Panel"). 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 one cross-state standard deviation decline in reference maternal mortality in the control group is associated with a 0.018 rise in the female-male college graduation differential, a 49% rise from the average cross-state differential for the control group, which was -0.0358. The adjusted R-squared suggests that the baseline specification explains 0.39 of the cross-state and cross-cohort variation.

The results for high school graduation are similarly strong. A decline in maternal mortality corresponding to a one standard deviation in the control group is associated with a 0.044 (or 81%) rise in the female-male differential in high school graduation rate for the sample of all individuals, with an adjusted R-squared of 0.45. 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.

Alternative Specifications and Instrumental Variables   We consider a specification that only includes treated cohorts and an instrumental variable specification, where, as for fertility, we define $ZZ_{st} = MMR_s^{pre} \times I_t^{post}$, where MMR_s^{pre} is average reference maternal mortality in the control group and the variable I_t^{post} indicates whether a birth cohort t belongs to the treatment group. 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 11 and strongly confirm the findings for the baseline specification. The panel estimates for the treated cohorts suggest that a one standard deviation decline in maternal mortality

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Summary Statistics

TABLE 10: Education _ Baseline Specification

Control Group: Birth Years 1919-1932

Reference Maternal Mortality (Age 5-10)

	Mean	48.5588
	St. Dev.	7.3353

Educational Attainment at age 23-32

Sample	COLL All	COLL Married	COLL Married with children	HS All	HS Married	HS Married with children	HG16 All	HG16 Married	HG16 Married with children	HG13 All	HG13 Married	HG13 Married with children
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

Treated Group: Birth Years 1933-1950

Reference Maternal Mortality (Age 5-10)

	Mean	9.1113
	St. Dev.	2.0244

Educational Attainment at age 23-32

Sample	COLL All	COLL Married	COLL Married with children	HS All	HS Married	HS Married with children	HG16 All	HG16 Married	HG16 Married with children	HG13 All	HG13 Married	HG13 Married with children
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

TABLE 11: Education_ Baseline Specification

Regression Results (1)

Panel

Specification		COLL	COLL	COLL	HS	HS	HS	HG16	HG16	HG16	HG13	HG13	HG13
Educational Attainment at age 23-32													
Sample		Married	All	Married with children	Married	All	Married with children	Married	All	Married with children	Married	All	Married with children
Constant		0.0894	0.0917	0.0819	0.2724	0.2613	0.262	0.1089	0.1141	0.0991	0.2287	0.2463	0.2121
	t-stat	5.8598	6.3851	5.3709	12.5099	12.7325	10.3643	7.1108	8.0123	6.3218	10.5235	11.9621	9.2842
$MMR^{ref}_{st} * F_g$ (2)		-0.0023	-0.0024	-0.0019	-0.0063	-0.006	-0.0068	-0.0017	-0.0016	-0.0014	-0.0027	-0.0025	-0.0024
	t-stat	-23.4651	-26.0324	-19.3789	-44.2502	-44.6459	-41.1853	-16.656	-17.341	-13.2257	-18.8579	-18.5846	-16.4066
Adj R-squared		0.3105	0.3586	0.2047	0.4362	0.4461	0.3852	0.2844	0.3249	0.1739	0.2499	0.2821	0.1834
R-squared		0.332	0.3787	0.2295	0.4538	0.4635	0.4044	0.3068	0.346	0.1997	0.2733	0.3046	0.2089
Predicted change in female-male differential for 1 st.dev. change in pre-treatment reference MMR		0.01687119	0.04621239	0.01247001	0.01980531	0.01760472	0.0440118	0.01173648	0.01833825	0.01393707	0.04988004	0.01026942	0.01760472

Panel, treated only

Specification		COLL	COLL	COLL	HS	HS	HS	HG16	HG16	HG16	HG13	HG13	HG13
Educational Attainment at age 23-32													
Sample		Married	All	Married with children	Married	All	Married with children	Married	All	Married with children	Married	All	Married with children
Constant		0.143	0.1458	0.1292	0.3941	0.3765	0.4	0.143	0.1458	0.1292	0.288	0.2956	0.262
	t-stat	7.7984	9.3781	6.0428	24.3242	26.7975	18.1037	7.7984	9.3781	6.0428	12.1114	13.9043	9.3125
$MMR^{ref}_{st} * F_g$ (2)		-0.0055	-0.0057	-0.0044	-0.0008	-0.0004	-0.0015	-0.0055	-0.0057	-0.0044	-0.0093	-0.0093	-0.008
	t-stat	-14.08	-17.3514	-9.6583	-2.379	-1.4894	-3.1796	-14.08	-17.3514	-9.6583	-18.3508	-20.5435	-13.4722
Adj R-squared		0.4641	0.5611	0.2356	0.4891	0.5536	0.2916	0.4641	0.5611	0.2356	0.4599	0.5276	0.2915
R-squared		0.4939	0.5855	0.2781	0.5175	0.5784	0.331	0.4939	0.5855	0.2781	0.4899	0.5538	0.3308

IV

Specification		COLL	COLL	COLL	HS	HS	HS	HG16	HG16	HG16	HG13	HG13	HG13
Educational Attainment at age 23-32													
Sample		Married	All	Married with children	Married	All	Married with children	Married	All	Married with children	Married	All	Married with children
Constant		0.0837	0.0858	0.077	0.257	0.2465	0.2451	0.1049	0.1103	0.0959	0.222	0.24	0.206
	t-stat	5.3509	5.7949	4.9571	10.8468	11.0196	8.9929	6.7742	7.6432	6.0732	10.0447	11.4672	8.9032
$MMR^{pre}_s * F_g * I_t$ (3)		0.0018	0.0019	0.0015	0.0048	0.0046	0.0052	0.0013	0.0012	0.0011	0.002	0.0019	0.0019
	t-stat	19.3412	21.3895	15.842	34.334	34.5118	32.1036	14.0687	14.5889	11.2721	15.5249	15.2867	13.5756
Adj R-squared		0.2748	0.3182	0.1745	0.332	0.3411	0.2846	0.2668	0.3067	0.1611	0.2237	0.2576	0.1619
R-squared		0.2974	0.3395	0.2003	0.3529	0.3617	0.3069	0.2897	0.3283	0.1873	0.2479	0.2808	0.1881

(1) Baseline specification. All regressions include fully interacted state, gender and cohort effects. Control group: 1919-1932. Treated group: 1933-1950.
(2) Reference maternal mortality is the average maternal mortality in the state at age 5-10 for each cohort.
(3) The instrument for reference MMR in each state is the average reference MMR for the control cohorts in each state.

in the treatment group is associated with 0.012 rise in the female-male differential in college graduation rates (middle panel, heading "Panel, treated cohorts"). The 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") are consistent with the other specifications.

Pre-Existing Trends To gauge the presence of pre-existing trends, we estimate equation (12) for the cohorts in the control group. The results are displayed in Appendix D, Table 16 (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. For consistency, we also repeat the estimation for the treated cohorts in the baseline specification using the age range for reference maternal mortality used in the falsification exercise.

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. The estimates for the treatment group with reference maternal mortality defined as for Control Group I (age 5-23), confirm the results in the main analysis.

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 12.

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 report only estimates for this group. We find that the alternative assumptions confirm our baseline results.

Columns 5-9 examine the sensitivity to the definition of control and treatment group. Column 5 reports the baseline results. Column 6 maintains the assumption on the control cohorts (born in 1921-1932) and assumes individuals born in 1933-1940 are treated, to explore whether the female-male differential in college graduation rates rose immediately as maternal mortality declined. We then change both the treatment and the control groups relative to the baseline specification. Specifically, we assume that the treated cohorts

comprise birth years 1940-1950 (column 7), 1945-1955 (column 8), 1950-1960 (column 9) and take the previous 10 birth years as a control group in each case. 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 1950-1960 cohorts and smallest for the 1933-1950 cohorts.

Controls   We use the same set of controls as 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 Groups 1-4 included in the fertility analysis, we also control for infant mortality (not interacted with gender), which corresponds to Group 5[35]. The results are displayed in Table 13 (first panel) 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.

We also find that the education results are robust to the inclusion of the economic and demographic controls (second panel) and the controls for state level cultural and political preferences (third panel). For this analysis, we interact mobilization rates with a female dummy, given that GI Bill education benefits were available only for male veterans (see footnote 32). Interestingly, we find that mobilization rates had a strong negative effect on the female-male differential in 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. As shown by Goldin and Katz (2002), access to contraception for unmarried women had a positive impact on their educational attainment, which could have exerted an independent negative effect on fertility. We use Bailey's (2006) coding of legal access to oral contraception for unmarried women, 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 the control for early legal access to oral contraception does not affect the estimates on maternal mortality.

4.3   Baby Bust

We also estimate a specification intended to capture 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 (relative to men) for the birth cohorts that were young enough when the decline in maternal mortality took place. 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 investigate this hypothesis, we estimate equation (9) including the cohorts whose fertility or education responded to the decline in maternal mortality, based on our previous analysis, that is birth years 1921-1950. Recall that birth years 1921-1940 responded positively with fertility to the decline in maternal mortality (relative to the 1913-1920 cohorts), while birth years 1933-1950 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[36], and estimate an IV specification, where the

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[35]The baseline specification for fertility already controls for infant mortality.

[36]For robustness, we also repeat the analysis assuming women born in 1933-1950 and 1946-1950 are treated and find similar

Table 12: Sensitivity: Treated and Control Groups, Reference Maternal Mortality

Specification		Panel, treated only (1)				Panel (1)				
Education Outcome		COLL	COLL	COLL	COLL	COLL	COLL	COLL	COLL	COLL
Sample		All	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	23-32
Column		1	2	3	4	5	6	7	8	9
Control Cohorts						1919-1932	1919-1932	1930-1939	1935-1944	1940-1949
Treated Cohorts		1933-1950	1933-1950	1933-1950	1933-1950	1933-1950	1933-1940	1940 - 1950	1945-1955	1950-1960
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	Age 5-14
Cross-state Average for Control Cohorts						48.5588	48.5588	34.9437	18.2049	8.4337
Cross-state Average for Treated Cohorts		9.1113	13.3392	11.474	29.5263	9.1113	9.0756	2.6726	2.2117	1.6517
Constant		0.1458	0.1467	0.1461	0.1478	0.0917	0.0951	0.1736	0.1131	0.1764
	t-stat	9.3781	9.5515	9.4479	9.782	6.3851	6.3149	2.4467	2.2877	4.4615
MMR^{ref}_{st} (2)		-0.0057	-0.0045	-0.0046	-0.0028	-0.0024	-0.0024	-0.0043	-0.0077	-0.0149
	t-stat	-17.3514	-18.6772	-17.9068	-20.3782	-26.0324	-23.1403	-7.0285	-9.0317	-9.4558
Adj R-squared		0.5611	0.5715	0.5654	0.5852	0.3586	0.3244	0.2764	0.493	0.6938
R-squared		0.5855	0.5953	0.5896	0.6082	0.3787	0.3456	0.6401	0.7478	0.8477
Model p-value		0	0	0	0	0	0	0.0029	0	0

Regression Results with Controls

Panel Specification (1) (3)

Health (4)

Educational Attainment at age 23-32

Sample	HS Married	COLL Married
1: Male Mortality		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0055**	**-0.002**
t-stat	-36.9122	-18.9452
Adj R-squared	0.465	0.3226
2: Sulfa related		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0021**	**0.0003**
t-stat	-13.8879	3.0709
Sulfa related	**-0.0052**	**-0.0033**
t-stat	-41.4333	-36.2608
Adj R-squared	0.6397	0.5187
3: Malaria		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0058**	**-0.0021**
t-stat	-39.3949	-20.5294
Adj R-squared	0.4556	0.3178
4: TB		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0021**	**0.0003**
t-stat	-14.0974	3.0194
TB	**-0.0026**	**-0.0017**
t-stat	-40.9281	-36.2331
Adj R-squared	0.6366	0.5185
5: Infant Mortality		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0045**	-0.0001
t-stat	-27.1509	-1.1563
IMR_t	**-0.0037**	**-0.0048**
t-stat	-17.9178	-37.6286
Adj R-squared	0.494	0.5361

Economic and Demographic

Educational Attainment at age 23-32

	HS Married	COLL Married
1: Personal Income, Unemp.		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0054**	**-0.0009**
t-stat	-33.9725	-9.2148
Adj R-squared	0.4679	0.4811
2: White, Foreign Born, Farm		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0038**	**-0.0003**
t-stat	-25.0768	-3.0102
Adj R-squared	0.5833	0.5447
3: Share Public		
MMR_st*F_g	**-0.0048**	**-0.0005**
t-stat	-29.7395	-5.1892
Adj R-squared	0.4881	0.4961
4: Share Health		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0047**	**-0.0002**
t-stat	-28.5397	-1.5366
Adj R-squared	0.485	0.5419

State Characteristics

Educational Attainment at age 23-32

	HS Married	COLL Married
1: Literacy 1930		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0036**	0.0001
t-stat	-22.9653	1.2996
Adj R-squared	0.5639	0.5694
2: Acceptance of Women's Suffrage		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0063**	**-0.0021**
t-stat	-44.5071	-23.1226
Adj R-squared	0.4387	0.4256
3: Sheppard-Towner & Social Security Act		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0063**	**-0.0022**
t-stat	-43.7195	-21.9833
Adj R-squared	0.441	0.3356
4: Mob. Rates applied to 1922-1928 cohorts, interacted with gender		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0054**	**-0.0024**
t-stat	-30.704	-19.1384
Mob. Rate*F_g	**-0.1752**	0.0073
t-stat	-8.8041	0.5182
Adj R-squared	0.4501	0.3103
7: Mob. Rates applied to 1922-1928 cohorts		
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0045**	**-0.0016**
t-stat	-33.0156	-14.9518
Mob. Rate	**-0.3453**	**-0.1483**
t-stat	-31.5435	-17.6226
Adj R-squared	0.5752	0.3742

Controls for Treated Cohorts

Educational Attainment at age 23-32

Early Legal Access to Pill

	HS Married	COLL Married
MMR$^{ref}_{st}$ *F$_g$ (2)	**-0.0073**	**-0.0026**
t-stat	-23.3019	-11.5905
Adj R-squared	0.4387	0.3105

(1) Baseline specification. Includes state/time, female/time, female/state interactions. Control group: 1919-1932. Treated group: 1933-1950.
(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.

instrument is the average reference maternal mortality (at age 15-20) in the state for the birth years 1913-1920 (the untreated cohorts for the main fertility specification). 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. 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 14. We focus on CHBORN at age 35-44, and consider the samples of 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, in all three specifications. For this specification, a negative value of α_1 in equation (9) for the IV specification implies that a larger decline in maternal mortality is associated with a reduction in fertility between the treated and control cohorts for this specification. 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 significant at the 1% level and implies that a one standard deviation decline in the instrument is associated with a decline in CHBORN of -0.47 between the treated (1941-1950) and the control cohorts (1921-1940) for Married women. This corresponds to 77% of the actual decline. Similar results hold for the other specifications.

To gauge the robustness of these results, we include controls for access to oral contraception for unmarried women and to early legal abortion. As shown by Goldin and Katz (2002), access to contraception for unmarried women had a positive impact on their educational attainment, which could have exerted an independent negative effect on fertility. Angrist and Evans (1999) show that access to legal abortion mainly reduces fertility of teenage girls. 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 to unmarried women and early legal abortion access[37]. We still find that for the treated cohorts the decline in maternal mortality is significantly negatively related to fertility for CHBORN at age 35-44 for the sample of All and Married.

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

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results.

[37]We 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.

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. The estimates conditional on education provide support for this mechanism. However, given the indirect link between the maternal mortality decline and the baby bust, one should be cautious to embrace a causal interpretation of the empirical findings.

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, as the rise in the opportunity cost of children reduces desired fertility for women who experienced the decline in maternal mortality while still in their formative years, relative to women who experienced it after having completed their education. The resulting boom-bust cycle in fertility and rise in female-male differential in educational attainment are broadly consistent with the U.S. experience.

Our empirical analysis suggests that the decline in maternal mortality had a very strong positive effect on completed fertility for women born in 1921-1940 relative to earlier cohorts, and a strong negative effect on completed fertility of women born in 1941-1950 relative to those born in 1921-1940. We also find that the decline in maternal mortality had a positive effect on the female-male differential in college and high school graduation rates for individuals born in 1933-1950 relative to younger cohorts. The empirical estimates suggest that the decline in maternal mortality can account for well over 50% of the boom and bust in completed fertility and over 40% of the rise in women's educational attainment with respect to men.

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 behavior. Many advanced economies experienced baby booms similar in timing, but smaller in magnitude, relative to the U.S. in the same historical period. The United States had the highest rates of maternal mortality among the advanced economies in the 1930s, as documented in Loudon (1992). It follows that the U.S. experienced the greatest drop in maternal mortality when sulfa drugs, blood banking and other medical innovations generated a sharp reduction in maternal mortality. Albanesi (in progress) explores whether the cross-country variation in the path of maternal mortality can account for the international variation in fertility patterns.

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TABLE 14: Baby Bust

Regression Results [1]

IV

Specification		1921-1950			1921-1950			1933-1950			1921-1950	
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	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN
		All	Married	Married with children	All	Married	Married with children	All	Married	Married with children	COLL, All	HS, All
Sample / Age		35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	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
$MMR^{pre}\_s*I\_t$		-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_st		-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
State effects		Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time effects		Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
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
R-squared		0.7259	0.6547	0.1785	0.6448	0.5531	0.1391	0.5237	0.5657	0.5699	0.2614	0.6803
Model p-value		0	0	0	0	0	0	0	0	0	0	0

Regression Results with State Level Controls [2]

IV

Fertility Outcome		CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN
		All	Married	Married with children	All	Married	Married with children	All	Married	Married with children	COLL, All	HS, All
Sample / Age		35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	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
$MMR^{pre}\_s*I\_t$		-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
$MMR^{pre}\_s*I\_t$		-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	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN	CHBORN
	All	Married	Married with children	All	Married	Married with children	All	Married	Married with children	COLL, All	HS, All
Sample / Age	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	35-44	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 Instrument is average reference maternal mortality (average maternal mortality in state at age 15-20) for birth cohorts 1913-1920.
2 The specification is the same as for the main regression results.

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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).[38] 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/cmf-icd9-archive1998.html.

Infant Mortality

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

Live Births

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

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/cmf-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/cmf-icd9-archive1998.html.

A.3   State Level Controls

Economic and Demographic Controls   UNEMPLOYMENT RATE: We use IPUMS variable EMP-STAT 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,[39] 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), inteded to serve as a proxie for the nuber 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 $t = 1922, ...1928$. We also run a specification where I_t^G corresponds to the percentage of individuals who used GI benefits, in the same year, to allow for variation in intensity across cohorts.

Federal Programs for the Promotion of Maternal and Infant Health   1921-1929 Maternity and Infancy Care (Sheppard-Towner) Act: Appropriations, Payments to the States, Activities carried out under the Act by the States, fiscal years 1921-1929: Children's Bureau Publication 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   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.

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

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.[40] 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.[41] The response of the states to the availability of the federal funding via this legislation varied greatly. Many states did not accept the benefits of the act for several years, 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 modelled 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

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

[41]The 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).

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.

C   Theory

C.1   Basic Model

C.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:

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

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

Solving:

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

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

By (3):

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

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

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:

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

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

By Assumption 1, $detH_V > 0$ 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 $\frac{\partial e'}{\partial e} > 0$.∎

C.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:*

$$\frac{\partial b}{\partial \mu} \leq 0. \tag{17}$$

In addition:

$$\frac{\partial e'}{\partial \mu} \leq 0, \tag{18}$$

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:

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

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

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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:*

$$\frac{\partial b}{\partial \mu'} \leq 0 \qquad , \tag{19}$$

$$\frac{\partial e'}{\partial \mu'} \leq 0, \tag{20}$$

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:

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

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

Solving:

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

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

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

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.

C.2   Extended Model

The model described in Section 3 can easily be extended to allow for daughters and sons. Each cohort has a representative household, comprised by 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 infant mortality risk.

The first order necessary conditions for this choice problem are:

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

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

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 suggest 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 from the parents' cohort.

Proposition 5 *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 Assuption 2, the optimal response of births and parental investment in human capital satisfies:*

$$\frac{\partial b}{\partial \mu} \leq 0, \tag{23}$$

$$\frac{\partial e'_f}{\partial \mu} \leq 0, \tag{24}$$

if and only if:

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

for $j = f, m$ *and:*

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

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:

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

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

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

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

$$\left[-v_{e'_f e'_m}(b, e') + \kappa(sb) U_{e'_f e'_m}(e'; \mu') \right] \left[-v_{be'_m}(b, e') + \kappa'(sb) s U_{e'_m}(e'; \mu') \right]$$
$$> \left[-v_{be'_f}(b, e') + \kappa'(sb) s U_{e'_f}(e'; \mu') \right] \left[-v_{e'_m e'_m}(b, e') + \kappa(sb) U_{e'_m e'_m}(e'; \mu') \right].$$

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

Proposition 6 shows that desired fertility is decreasing in the mother's endowment of human capital.

Proposition 6 *Assumption (2) implies:*

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

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, $\frac{\partial b}{\partial e_f} < 0$. ∎

D Additional Empirical Results

TABLE 15: Fertility_ Sulfa Mortalities as Instrument

Regression Results (1)

Specification		IV								
Fertility Outcome		CHBORN	CHBORN	CHBORN	NCHILD	NCHILD	NCHILD	NCHLT5	NCHLT5	NCHLT5
				Married with children			Married with children			Married with children
Sample		**Married**	**All**	**children**	**Married**	**All**	**children**	**Married**	**All**	**children**
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
R-squared		0.4444	0.4899	0.2465	0.2916	0.3549	0.2235	0.2867	0.3544	0.2175
Model p-value		0	0	0	0	0	0	0	0	0

(1) Baseline specification. All regressions include state and cohort effects. Control group: 1913-1920. Treated group: 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 tretable 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 16: Falsification Exercise

Fertility (1)

	Control Group I			Control Group II			Treatment Group		
Included Birth Cohorts	**1905-1915**			**1910-1915**			**1921-1940**		
Reference MMR	**Age 15-28 (2)**			**Age 15-28 (2)**			**Age 15-28 (3)**		
Statistics									
mean(MMR)	56.027			59.2552			9.1082		
std(MMR)	8.242			8.7857			1.9438		
Fertility Outcome	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**	**CHBORN 35-44**
Age	**All**	**Married**	**Married with children**	**All**	**Married**	**Married with children**	**All**	**Married**	**Married with children**
Sample									
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			
Included Birth Cohorts	**1910-1917**				**1918-1925**				**1933-1950**			
Reference MMR	**Age 5-23 (2)**				**Age 5-15 (2)**				**Age 5-23 (3)**			
Statistics												
mean(MMR)	59.5396				56.7425				6.189			
std(MMR)	8.5992				8.3314				1.3483			
Educational Attainment at age 23-32	**HS All**	**COLL All**	**HS Married**	**COLL Married**	**HS All**	**COLL All**	**HS Married**	**COLL Married**	**HS All**	**COLL All**	**HS Married**	**COLL Married**
Sample												
Constant	0.1639	0.0414	0.1391	0.0315	0.0759	0.0312	0.0814	0.0283	0.3765	0.1457	0.3942	0.1429
t-stat	7.8439	3.4186	5.6076	2.201	5.5659	3.4826	5.6464	3.5521	26.8056	9.3602	24.3352	7.7848
$MMR^{ref}_{st} * F_a$ (4)	**-0.0096**	0.001	**-0.0079**	**0.0022**	**-0.002**	0	**-0.0017**	-0.0001	-0.0008	**-0.0095**	**-0.0015**	**-0.0091**
t-stat	-9.7617	1.7432	-6.7639	3.214	-4.8601	-0.1367	-3.8735	-0.2922	-1.6276	-17.206	-2.5392	-13.9421
Adj R-squared	0.7242	0.4387	0.6501	0.2732	0.4752	0.2191	0.4401	0.1927	0.5537	0.5599	0.4894	0.463

(1) Baseline specification. 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.