Maternal Health and the Baby Boom*

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Abstract

In 1900, one mother died for every 118 live births in the United States. Approximately 15,000 women died of childbirth each year between 1900 and 1930, and pregnancy related causes accounted for over 15% of all female deaths at age 15-44. For every death, twenty more mothers suffered obstetric complications leading to severe and long term disability. Between 1936 and 1956, maternal deaths dropped by 94%, reaching modern levels by the late 1950s. The incidence of pregnancy-related conditions also underwent a similar reduction. We examine the link between the decline in the maternal health burden and the mid-twentieth century baby boom, exploiting the large cross-state variation in the magnitude of this drop and the differential exposure of women by cohort. We find that for every 10 unit drop in maternal mortality, completed fertility rises by 0.6-1.1 children for women born between 1931 and 1938. The drop in maternal mortality is also associated with a rise in women's educational attainment for these and later cohorts. These findings provide new insights on the determinants of fertility in the U.S. and other countries that experienced similar improvements in maternal health.

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

Keywords: Maternal mortality; Fertility choice; Human capital; Infant Mortality; Medical progress.

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1 Introduction

The United States experienced a very dramatic rise in fertility between the late 1930s and the early 1960s. 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 low of 1.74 children for women born in 1949, it stabilized around 2 children per woman in the 1980s. Despite the remarkable magnitude of the mid-twentieth century baby boom and its clear economic and social relevance, its origins are still poorly understood. Perhaps the best known explanation is Easterlin's (1961) "relative income" hypothesis, based on the notion that fertility is high for cohorts that experience particularly favorable labor market conditions when young, leading to relatively high projected lifetime income. Thus, the recovery from the Great Depression and World War II can provide an explanation for the baby boom. This hypothesis is not fully consistent with the timing of the baby boom, and also runs counter the very strong negative empirical correlation between income and fertility (Jones and Tertilt, 2007).

Greenwood, Seshadri and Vanderbroucke (2005) propose that the diffusion of home appliances reduced the time cost of children and was a key determinant of the baby boom. This explanation is also not fully consistent with the timing of the baby boom. Fertility started to rise prior to World War II, while the diffusion of home appliances was limited by falling incomes during the Great Depression and their production was suspended during the war.[2] The rapid diffusion of home appliances in the 1950s and 1960s may well have been driven by the rise in fertility in those years and the resulting increase in the number of children per household, a key determinant of the demand for home hours (Ramey, 2008).

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 anticipate marriage and increase their fertility. This explanation is inconsistent with the fact that fertility began to rise before the war. Moreover, the direct impact of wartime female participation on labor market conditions appears to be limited. Evidence on female employment based on the US Census presented in Goldin (1991) suggests that 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.[3] According to Acemoglu, Autor and Lyle (2004), the impact of wartime female participation on wages was largely exhausted by 1950. Finally, this hypothesis is based on the premise that the women who became mothers during the baby boom were not in the workforce. While married women with small children exhibit lower labor force participation than married women without children and unmarried women, Albanesi and Olivetti (2009) show that participation of married women with young children rose during the baby boom.

We propose a novel explanation of the baby boom that links the evolution of fertility and women's human capital investment to the dramatic improvements in maternal health. In 1900, one mother died for every 118 live births. Approximately 15,000 women died of childbirth each year between 1900 and 1930, and pregnancy related causes accounted for over 15% of all deaths of women 15-44, the second largest cause of death for women in this age group after tuberculosis. For every death, twenty more mothers

[1] The Cohort Total Fertility Rate (TFR) 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, 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.

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

[3] Table 19, Goldin (1991).

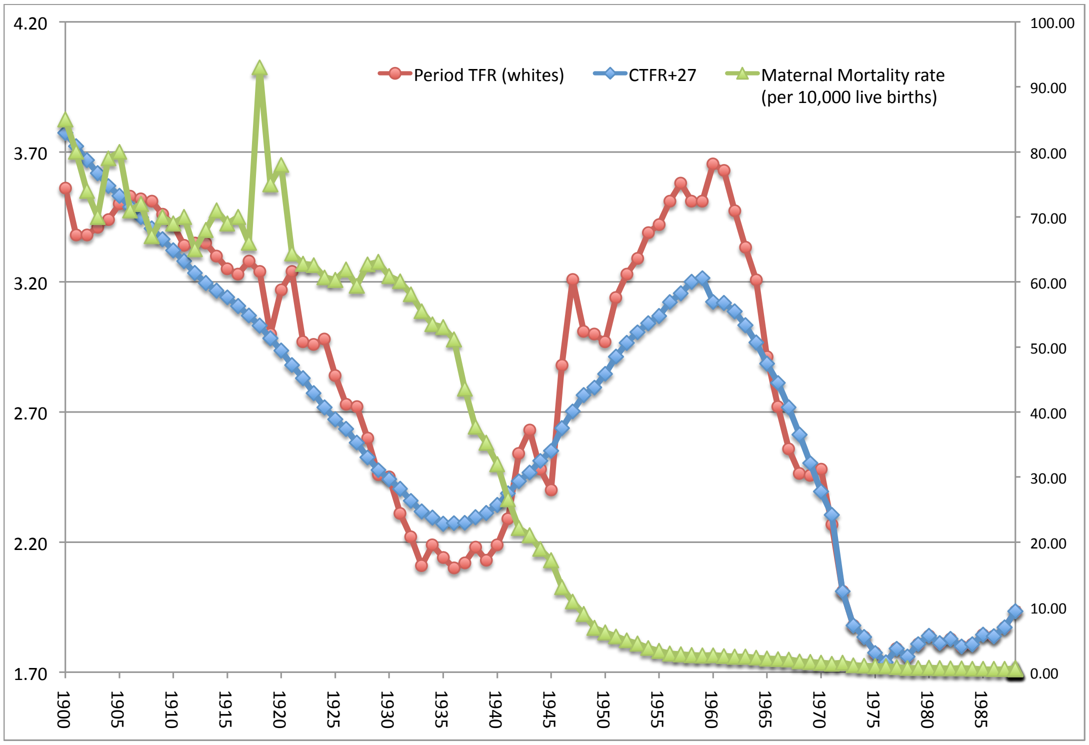

Figure 1: Maternal mortality, Period Total Fertility Rate and Cohort Total Fertility Rate (+27) in the U.S. 1900-1990
Source: U.S. Cohort Fertility Tables, CTFR 1917-1980, TFR 1900-1988. Produced by the National Institute of Child Health, compiled in Heuser (1976). U.S. Period Fertility Rate in Haines (2006). Maternal mortality from Vital Statistics of the United States.

suffered obstetric complications leading to severe and long term disability (Kerr, 1933). Starting in 1936, the maternal mortality rate dropped dramatically, reaching modern levels by the late 1950s. The maternal mortality rate was 51.16 per 10,000 live births in 1936 and reached 2.87 per 10,000 live births by 1956, a 94% drop in twenty years. The virtual elimination of maternal mortality risk was accompanied by a rise in the female-male differential in adult life expectancy from 2.5 to 6 years over the same period. There was a similar reduction in the incidence of maternal conditions, leading to a sizable decline in the health burden associated with pregnancy.

We examine the contribution of the improvement in women's health to the mid-twentieth century baby boom and the rise in women's educational attainment. Fertility theory predicts that a reduction in the health burden associated with pregnancy can increase the demand for children, while the corresponding rise in women's productive life span raises the returns from human capital. We formalize this reasoning with a stylized model of fertility choice and human capital investment, which incorporates two dimensions of maternal health, pregnancy related death risk and the health burden of pregnancy conditional on survival. The model predicts that if initial maternal mortality risk is high enough, both fertility and human capital will rise in response to a reduction in pregnancy-related mortality. A coeteris paribus decline in the health burden of conditional on survival may reduce human capital investment if fertility rises, since an increase in births increases maternal mortality risk and lowers the return to human capital for given maternal mortality rate. Thus, a positive fertility response could be accompanied by a positive or negative response of human capital, depending on the channel that prevails. Moreover, once the maternal mortality risk is sufficiently low, additional advances in maternal health do not generate a rise in fertility.

Our empirical strategy exploits the large cross-state variation in the magnitude of the maternal mortality decline to estimate its effect on the change in fertility across cohorts of women who were differentially exposed to the improvement in maternal health, using a difference-in-difference approach. Comparing women born in 1931-1938, whose maternal mortality rates are close to modern levels, to those born in 1911-1918, who were the last to experience high maternal mortality, we estimate that for every 10 unit reduction in maternal deaths (per 10,000 live births), completed fertility rises $0.6 - 1.1$ children or 25-43%. These estimates are robust to the inclusion of demographic and economic controls, as well as indicators for state level expenditures on maternal and infant health services. For women born in 1921-1922, who experience only a partial drop in maternal mortality, every 10 unit reduction in maternal mortality relative to the 1911-1918 birth cohorts is associated with a rise in completed fertility of $0.20 - 0.51$ children, or 8-19%. However, these estimates are not robust to the inclusion of demographic and economic controls. Our estimates suggest that the drop in maternal mortality was also associated with a rise in women's human capital. For every 10 unit drop in maternal mortality, the male-female differential in college graduation drops by 20% for women born in 1931-1938. Even if this effect is sizable, the estimates have low significance and are not robust.

Infant mortality also declined substantially during the same time period. The infant mortality rate[4] dropped from 124.46 infant deaths per 1,000 live births in 1900 to 9.2 infant deaths per 1,000 live births in 1990, with most of the reduction concentrated between 1900 and 1940. The decline in youth mortality has been linked to the secular decline in fertility starting in the 1850s (Preston, and Haines, 1991, and Haines, 1997). A decline in infant mortality eliminates the hoarding and replacement motives in the demand for children. There is also a strong evidence of "reverse causation," as maternal depletion resulting from high fertility may lead to high infant mortality. Since the rise in infant survival probability and the drop in maternal mortality have opposing effects on fertility, we also examine the relation between the drop in infant mortality and the change in fertility. We find that the reduction in infant mortality is strongly associated with a reduction in fertility for the 1921-1928 birth cohorts in comparison to the 1911-1918 birth cohorts. For every 10 unit drop in infant mortality (per 1,000 live births), completed fertility drops by 0.50 children or 18%. This is an important finding, as the below trend fertility for these cohorts is hard to reconcile with standard explanations.[5] For later birth cohorts, we do not find that the drop in infant mortality is significantly related to fertility.

Taken together, these results suggest that medical progress, the ultimate factor in the reduction of both maternal and infant mortality, significantly contributed to the sharp decline in fertility of the 1920s and early 1930s and the subsequent baby boom and may provide an integrated explanation of these phenomena. This is one of the main contributions of the paper, since the both the demographic and the economic literature on the determinants of fertility have concentrated on the impact of the reduction in youth mortality on the secular decline in fertility,[6] treating the medium run fluctuations, such as the U.S. baby boom, as separate

[4]Infant mortality is defined as the death of a child below one year of age. Child mortality is the death of a child within its first five years of life.

[5]See Jones and Tertilt (2007) for a discussion.

[6]See Doepke (2005) for an excellent discussion of the economic literature. Murphy, Simon and Tamura (2008) focus on the

phenomena.[7]

The paper also makes a contribution to the literature on the effects of advances in health on human capital. The most closely related paper in this literature is Jayachandran and Lleras-Muney's (2009) study of the impact of maternal mortality decline on female years of schooling in Sri Lanka.[8] Their estimates suggest a strong positive effect, which they interpret as consistent with a rise in parental investments in the education of daughters resulting from the decline in pregnancy-related mortality. Our hypothesis emphasizes adult women's decisions on their own human capital and fertility. Additionally, we focus on college graduation, since the U.S. had already achieved high rates of basic schooling by the mid-1930s. Our findings suggest that the maternal mortality decline is only weakly related to the growth in women's college graduation relative to men. While this finding is consistent with our theoretical analysis, for the birth years we consider it could also be driven by crowding out of female students by the sharp rise in male college attendance resulting from the GI Bills for World War II and the Korean war.[9]

Our findings provide new insights on the determinants of fertility in the United States and other countries that experienced similar advances in maternal health. Our analysis also has broader implications. Albanesi and Olivetti (2009) show that improved maternal health plays critical role for the rise in married women's labor force participation during the twentieth century. In particular, it can provide an explanation for the joint rise in fertility and participation during the baby boom. Numerical simulations suggest that the advances in maternal health alone can generate a rise in income per capita of over 50% through its positive effect on women's participation. These findings offer a new perspective on demographic policies in developing countries, often based on the premise that fertility decline is a necessary condition for the rise in income per capita. Our results suggest that improving maternal health, typically a very severe problem in developing countries, could improve standards of living even without a decline in fertility.

The paper is organized as follows. Section 2 discusses the historical background for the reduction in maternal mortality in the U.S. Section 3 presents a simple model of fertility choice to examine the impact of maternal health on fertility. Section 4 discusses the empirical analysis, and Section 5 concludes.

2 Maternal Health in the US

This section documents the incidence of pregnancy related deaths and disabilities 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.

historical link between child mortality, fertility and human capital investment in the US.

[7]A notable exception is Greenwood, Seshadri and Vanderbroucke (2005) who provide an integrated theory of the secular decline in fertility and the baby boom in the US. The secular rise in labor productivity drives the downward trend in fertility, while the diffusion of home appliances gives rise to a baby boom.

[8]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.

[9]Altshuler and Blumin (2009) provide a detailed account of college admission policies during this period.

2.1 Burden of Maternal Mortality and Morbidity

The maternal mortality rate (MMR),[10] which can be interpreted as a measure of the average probability of a maternal death for each live birth, was equal in 1900 to 85 maternal deaths per 10,000 live births, 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.[11] In 1930, maternal mortality still accounted for 10.6% of female deaths at age 15-44 and was the second biggest death cause for women in this age group after tuberculosis. [12]

Women who survived childbirth were exposed to a severe risk of suffering from a variety of conditions, such as puerperal fever, obstetric fistulas, hypertensive disorders, chronic anaemia, leading to protracted or permanent disability. To provide a quantitative assessment of the health burden of pregnancy conditional on survival, Albanesi and Olivetti (2009) construct a measure of this burden, using hospital based studies from the late 1920s that offer detailed information on incidence and duration of the most common ailments. The measure corresponds to the Years Lost to Disability (YLD) concept developed by the World Health Organization. Specifically, $YLD = I \times D \times DW$, where I is the incidence, D represents duration, and the variable DW, which stands for disability weight, is an index of the degree of disablement associated with a disease, with a value of 0 standing for perfect health and 1 for death.[13]

Based on post-partum readmission data, 12% of all live births generated some form of maternal morbidity (Kerr, 1933).[14] Perineal lacerations from obstructed labor were the most debilitating and prevalent, accounting for 67% of all cases of morbidity or 8% of all live births, with an average duration of 55.67 months and a disability weight of 0.43. By comparison, the disability weight for blindness is 0.60 and the one for AIDS is 0.505. Other common conditions gave rise to severe and chronic disablement. For example, hypertensive disorders of pregnancy are chronic and their disability weight is 0.38 in childbearing years and

[10] 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.

[11] The maternal mortality risk also depends on fertility, through the effect of age and parity on the risk of a pregnancy-related death. 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. This can account for some of the reduction in maternal mortality between 1900 and 1930. On the other hand, the age and parity distribution does not influence the decline in maternal mortality for later years.

[12] 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.

[13] This data is collected as part of the Global Burden of Disease Study Program. See HTTP://www.who.int/healthinfo/bod/en/index.html. Table A2 in the appendix reports this disability weights by maternal conditions.

[14] This measure is likely subject to measurement error. On the one hand, if women who experience more difficult pregnancies are more likely to seek a hospital delivery, they may also be more likely to experience complications. On the other, certain conditions that originate as complications of pregnancy and childbirth may later lead to symptoms treated outside of the maternity or gynecological ward. For example, Bromley (1929) reports that for every woman who died of puerperal septicemia, an estimated five more contract it and experience resulting long term disabilities, such as "inflammation of the breasts, pulmonary and cardiac complications, or ulcers and abscesses of the generative tract, which frequently cause permanent invalidism or necessitate operations later in in life."

Table 1: Incidence of Maternal Mortality

Incidence of Maternal Mortality

		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

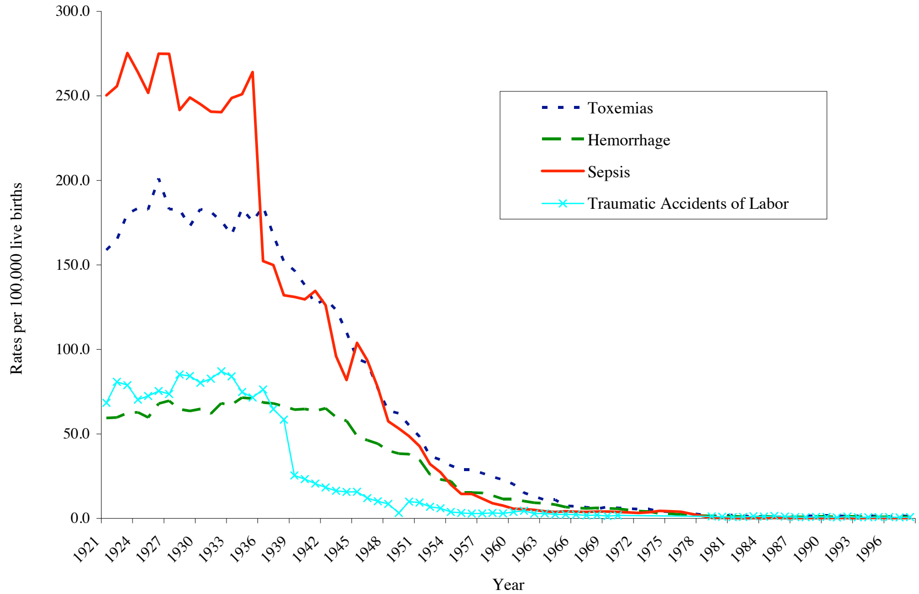

Figure 2: Maternal mortality by cause
Source: Vital Statistics of the United States

increases with age, the weight for severe anaemia resulting from a maternal hemorrhage is 0.09 until the end of life. Including the pregnancy itself, which has a disability weight of 0.22, the historical data on incidence and duration with the WHO age specific disability weights, yield an estimate of 2.48 years lost to disability per pregnancy.

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 between 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 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 dropped beginning in the mid-1930s and stabilized in the late 1950s.[15] 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,[16] which, as can be seen in Table 1, rose from 2.5 to 6.6 years between 1930 and 1960. Between 1930 and 1960, mortality rates declined by 22% for females and only 10% for males, whereas between 1900-1930 the mortality decline was similar across genders. 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

[15] 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%).

[16] 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.

female-male differential in life expectancy at birth between 1910 and 1965, and for 100% of the change in female-male differentials in mortality rates at age 20-39.[17]

There are no systematic time series data on the evolution of maternal morbidity.[18] Still, since the main causes of maternal morbidity in the 1920s and 1930s were the same as those for maternal mortality, and many of these conditions arose as a consequence of poor obstetric practices, it seems plausible to assume that the same factors that led to a decline in maternal mortality also contributed to reduce the incidence of maternal morbidity. This is confirmed by estimates in Franks et al. (1992), which is the only comprehensive nationwide assessment of serious pregnancy related morbidity following childbirth (Wilcox and Marks, 1994). Based on data from hospital discharge records for the United States, the annual rate of pregnancy-related post-partum morbidity requiring hospitalization was 8.1 per 1,000 deliveries for 1986-1987. The corresponding statistic based on the records of post-partum conditions reported in Kerr (1933) for the late 1920s is 114.4 per 1,000 deliveries. [19] Thus, post-partum pregnancy-related conditions requiring hospitalization dropped by 93% between the late 1920s and the mid 1980s. This decline is similar in magnitude to the drop in maternal mortality over the same period (1930-1987), equal to 99.16%. 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 and the resulting burden. This assumption is standard in the literature on the economic impact of health outcomes.[20]

2.2.1 Advances in infant health

Infant mortality also experienced a very large reduction in the same period. Infant mortality declined from 124.48 to 9.2 per 1,000 live births between 1900 and 1990, as shown in figure 3.

As we discuss below, many public health initiatives and government programs were aimed at reducing infant mortality, before any attention was devoted to maternal health. Not surprisingly, infant mortality started falling earlier than maternal mortality. Prenatal care, advances in obstetrics and improved maternal health reduced the incidence of prematurity and congenital malformation and "maternal depletion." Despite these developments, infant mortality declined at a slower pace than maternal mortality starting in the 1930s. Between 1936 and 1956, when maternal mortality dropped by 99%, infant mortality declined by 53%. There was no improvement in the infant mortality rate during the 1950s. The decline in infant mortality continued at a steady pace starting in the 1960s.

2.3 Determinants of improved maternal health

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 US (Leavitt, 1986).

[17]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.

[18]There are still no generally accepted criteria for the measurement of maternal morbidity, as well as significant obstacles to data collection in this area. In developing countries, lack of access to prenatal and to post-partum care prevents systematic reporting of complications of pregnancy. In the US and other advanced countries, privacy concerns imply that, with the exception of a few, small, voluntary studies, there is no systematic panel data in symptomatology associated with childbirth (Wilcox and Marks, 1994). Hospital discharge and ambulatory care records are the only comprehensive source of information on pregnancy-related morbidity. They report data on the number of procedures that can be associated to specific morbid states, which can then be used to infer the incidence of the corresponding conditions.

[19]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.

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

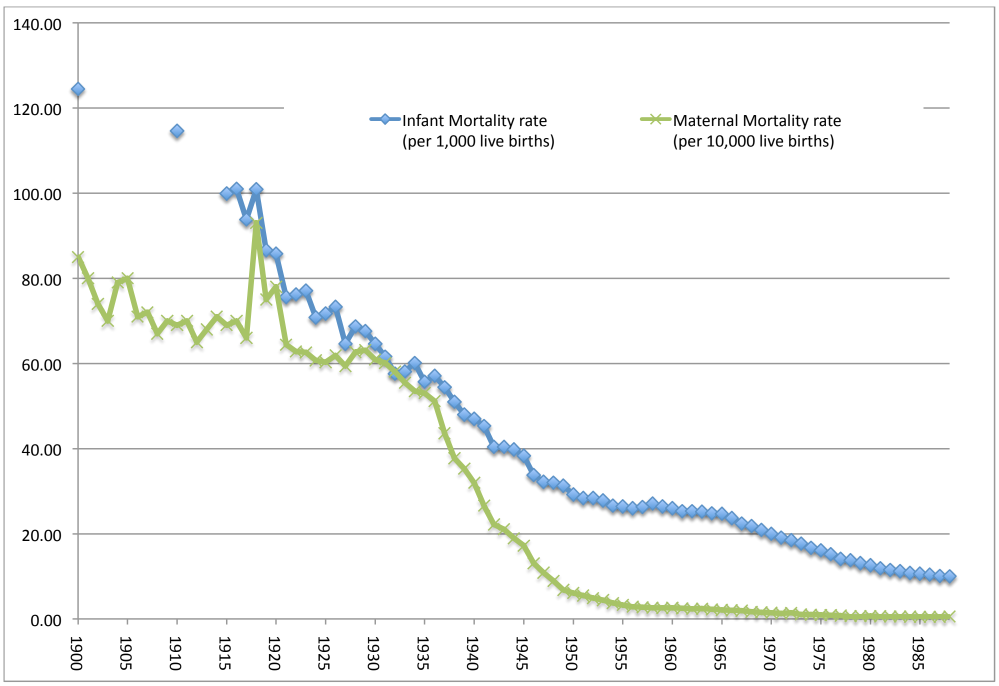

Figure 3: Maternal and infant mortality rates
Source: Vital Statistics of the United States

Early efforts to improve maternal health were mainly driven by the goal of reducing infant mortality, which was identified as major problem, especially in urban areas (Meckel, 1990), after the expansion of death and birth registration in the second half of the nineteenth century.[21] The evidence on the influence of the environment and nutrition on child health led by the 1890s to the installation of hygienic milk stations to provide safe milk for urban infants (Rosen, 1958).[22] The milk stations often also operated programs for home visits by nurses and social workers. While these visits were mainly targeted to small children, advice on "maternal hygiene" and nutrition was often provided. At the turn of the twentieth century, the availability of national census data started to shed light on the slow decline in infant mortality. In response to these statistics, Congress created the Children's Bureau in 1912, an agency of the Department of Labor with the primary responsibility of promoting infant and child health.[23]

The Children's Bureau became a forceful advocate for children's health through its public campaigns, as well as through direct lobbying. One of its main activities was to conduct detailed statistical studies of maternal and child health outcomes. The publication of the first national report on neonatal health in 1913, with premature birth, "congenital debility," and "malformations" reported as the main causes of death in the neonatal period, made health officials increasingly aware of the link between maternal and infant health. Reflecting these concerns, the Children's Bureau published the first edition of its pamphlet *Prenatal Care* in 1913.

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). One of the main findings was that maternal mortality was the second largest cause of death for women age 15-44 after tuberculosis in 1913.[24] Moreover, the report clearly identified the United States as the worst for maternal health among advanced nations. As the problem of maternal mortality gained independent status in public discourse, the Children's Bureau's efforts began to explicitly target maternal health. The Bureau became the main sponsor and administrator of a series of key federal programs targeting maternal and infant health introduced between 1921 and 1943. These programs, jointly with the contemporaneous scientific and medical advances, led to a dramatic reduction in maternal mortality.

2.3.1 Medical and scientific developments

Several medical and scientific factors contributed to the decline in maternal mortality (CDC, 1999). The main development was the medicalization and hospitalization of childbirth, a process that took place very gradually. Physicians started to systematically enter the birth room in 1850. Hospital deliveries grew very

[21] One of the best known early U.S. reports on public health data was Shattuck's Report of the Sanitary Commission of Massachusetts, 1850. This extensive study documented infant and maternal mortality in detail and recommended public programs for sanitary surveillance, immunization and well-baby clinics. Also critical and influential in highlighting the extremely high rates of death among the children of the urban poor and the foreign born was the 1857 American Medical Association Report on Infant Mortality in Large Cities, the Sources of Its Increase and the Means of Its Diminution. Authored by D.M. Reese, the report appeared in Transactions of the American Medical Association 1857(10): 102. Cited in Meckel (1990).

[22] The first milk station was opened in New York city in 1893, while the first program of school health inspectors was launched in Boston in 1894. (Schmidt, 1973)

[23] 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. The Children's Bureau was also in charge of monitoring child labor practices and expanding the birth and death registration area. The Bureau was transferred to the Social Security Administration upon its creation in 1946. Its focus then shifted to health and welfare issues. The Bureau was transferred to the Department of Health, Education and Welfare in 1962, and eventually dismembered in 1967. The Office of Child Development, and agency created in 1969, holds what remains of the statutory responsibilities of the Children's Bureau. For a full account see: http://www.ssa.gov/history/childb1.html

[24] Another key finding, reported in Woodbury (1925) that in the time period 1900-1920 there had been only a modest improvement in maternal deaths from septicemia and no improvement in maternal deaths from other causes, despite the increasing adoption of aseptic practices in childbirth.

slowly starting in the late 1800s, and increased precipitously after 1935. The intervention of physicians, however, did not initially contribute to a reduction in maternal mortality.[25] Inappropriate and excessive operative interventions, such as induction of labor, use of forceps, episiotomy and cesarean delivery, 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 came to widespread public attention following the publication by the New York Academy of Medicine in 1933 of a study of 2,041 maternal deaths in childbirth. At least two-thirds, were found to be preventable. The investigators found that many physicians lacked the most basic obstetric knowledge. The proceedings from the 1930 White House Conference on Child Health and Protection, also published in 1933, reported similar findings based on nationally representative sample (CDC, 1999). [26]

These reports precipitated the efforts to standardize obstetric practices and train physicians. The American Board of Obstetrics and Gynecology was established in 1930 to provide hospitals with criteria for evaluating obstetric skills (Dannreuther, 1931). Residency training programs began to be established in the 1930s to prevent hospitals from accepting unqualified specialists. Hospital and state maternal mortality review committees were also established in the 1930s and 1940s. These developments led to a widespread improvement in obstetric care in hospitals. The advent of electronic imaging and advanced neonatal therapies that could only be administered in a hospital setting led to a steep rise in the demand for hospital births.

The impact of the hospitalization of childbirth on the drop in maternal mortality can clearly be seen by analyzing the Children's Bureau state level statistics on live births by attendant. Despite the cross state dispersion in live births by attendant for all available years, there is a sharp rise in the minimum and median percentage of births in hospitals, accompanied by a decline in both of the percentage of births at home attended by a physician and the percentage of home births unattended by a physician, as reported in Table 2.

The table also reports the strong negative correlation between maternal mortality and percentage of births in hospital for all years in the analysis. There is also 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 home attended by a physician is also positive for all years, but it becomes sizable and significant at the 1% level only for 1954.

A number of scientific discoveries and advances in general medicine that took place in the 1930s had a particularly beneficial effect on maternal mortality. As can be seen from figures 1 and 2, the year 1936 constitutes a clear break in pregnancy related mortality. This year is notable from a scientific standpoint, since it marked the introduction of sulphonamides. As discussed in Jayachandran, Lleras-Muney and Smith (2009), these drugs were relatively cheap to produce and diffused very rapidly, contributing to a large reduction in mortality for several diseases, such as pneumonia and influenza, and scarlet fever, in a span of just a few years. 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. The discovery of the

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

[26] 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.

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.

antibiotic effects of penicillin (1939-1942) later also contributed to the decline in maternal mortality due to sepsis. Another crucial development was the establishment in 1937 of the first hospital blood bank in the United States, at the Cook County Hospital in Chicago. Within a few years, hospital and community blood banks spread across the United States. Hemorrhage was the second largest cause of maternal deaths, and blood banking, jointly with ongoing innovations in transfusion medicine, had a large impact on maternal deaths.

Even as the quality of obstetric care was improving in many hospitals starting in the 1930s, access to 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 expand the financial outlay (Baker, 1923).[27] 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 contributed to alleviate these costs for a very small number of households (Starr, 1982).

A number of federal programs promoting maternal and child health had contributed to an expansion in the number of maternity facilities since the 1920s, facilitating access. The most extensive of these programs, and the first to subsidize obstetric care was the Social Security Act, for which appropriations started in 1936. This may explain why 1936 marked the onset of a steep decline in pregnancy related mortality from *all* causes, as can be seen in figure 2, not just septicemia and hemorrhage, which can be clearly connected to the sulfa drugs and blood banking.

2.3.2 Government Intervention in the Area of Maternal and Infant Health

The United States's government enacted several programs for the promotion of maternal and infant health starting in the 1920s. We now briefly describe those programs. The criteria for appropriations, when

[27]The high costs of medically trained birth attendants is probably the main explanation for the persistence 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.

available, are reported in Appendix A.5.

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.[28] 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 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.[29] 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).

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, as well as 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 the number of live births, poorer states received more transfers. This may have contributed to a convergence in maternal health outcomes across states (Schmidt, 1973).

1943-1946 EMIC The Emergency Maternity and Infant Care Program (EMIC), passed into law in March 1943, provided funds for maternity and infant care for the wives and infants of servicemen in the four lower pay grades. Medical, nursing, and hospital services for the prenatal period as well as delivery and six weeks of postpartum care were provided for these families at *no charge*, in addition to complete care for infants. States obtained federal funds based on need, and there was no means testing for participants. Yearly appropriations to the states were made based on the number of projected cases, with the possibility of deficiency appropriations. By the end of the program in 1946, approximately 1.25 million mothers and 230 thousand children received care. It was the largest public medical care program undertaken in the United States up to that time (Schmidt, 1973).[30] The program was widely recognized for the reduction in maternal

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

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

[30] The program originated between 1941 and 1942, when following the attack on Pearl Harbor and the declaration of war against Germany, the number of men in the armed forces increased to approximately twelve million (Sinai and Anderson, 1947). Wives following their enlisted husbands to their place of deployment increased to a point that the areas around military camps became badly congested. Children were born in sub-standard conditions because of inadequate funds to procure obstetric services from the existing medical personnel and facilities. As documented in Sinai and Anderson (1947), American Red Cross

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 Hill-Burton Act was passed in 1946 to improve the infrastructure of the nation's hospitals. The objective 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. [31]

3 Theory

The main purpose of the theoretical analysis is to conceptualize the linkages between maternal health, fertility and human capital investment. The model also incorporates infant mortality, to explore the link between the infant mortality reduction and fertility. We will use these insights to inform the empirical analysis presented in Section 4.

Women derive utility from consumption and the number of children. They experience two stages in life. In the first, they choose human capital investment, and in the second they choose fertility. Human capital investment entails a utility cost at stage one but increases affordable consumption at stage two. There are two dimensions of maternal health. A mother may die in childbirth and each birth is associated with a health cost, which reduces the mother's utility conditional on survival. An improvement in maternal health generates a decline in maternal mortality risk and a reduction in the health burden associated with each birth.

We now describe the model in detail. Women's preferences are represented by the following utility function:

$$\Upsilon(e, c, b, n; \mu, \varphi, \beta, U) = -v(e) + \mu(b)\left[u(c) - h(\varphi b) + \kappa(n)\right],$$

where $e \geq 0$ represents human capital investment, $c \geq 0$ denotes consumption, $b \in [0, \bar{b}]$ is the number of births and $n \geq 0$ is the number of children. The parameter $\bar{b}$ is the upper bound on the number of births, which we impose for analytical convenience.

The function $v(\cdot)$ represents the utility cost of human capital investment, $u(\cdot)$ is the utility from consumption, and $\kappa(\cdot)$ represents the utility from children. We assume: $v'(e) > 0$ and $v''(e) \geq 0$ for all $e \geq 0$, $u'(c) > 0$ and $u''(c) \leq 0$ for all $c \leq 0$, $\kappa'(n) > 0$ and $\kappa''(n) \leq 0$ for all $n \geq 0$ with $lim_{n \to 0} \kappa'(n) = +\infty$.

The function $\mu(\cdot)$ is the probability that a woman will survive childbirth, given her number of births, b. We assume $\mu(0) = 1$ with $\mu'(b) \leq 0$ and $\mu''(b) \geq 0$ consistent with the empirical evidence on parity influence on maternal mortality.

The function $h(\cdot)$ captures the utility cost associated with the health burden of childbirth, φb, where the parameter $\varphi \geq 0$ corresponds to the burden for each birth. The function satisfies $h'(x) > 0$ and $h''(x) \geq 0$ for all $x \geq 0$, and $h(0) = 0$.

Children may die in infancy. Letting $s \in (0, 1]$ denote the infant survival probability, the number of

representatives at 240 army posts reported that "in one month between July 15 and August 15, 1942, 3,262 servicemen requested help in securing maternity care for their wives; 39% of these were for assistance in obtaining care for wives living near an Army post, and 61% for assistance in obtaining care for wives living in other states. Furthermore, 2,601 requests were received from ... wives of men in service for help in obtaining maternity care or care of their sick children during the month of August 1942. "

[31] In 1975, the Act was amended and became Title XVI of the Public Health Service Act.

children is related to the number of births as follows:

$$n = sb. \tag{1}$$

The budget constraint for consumption at stage two is given by:

$$c = (1 + \varepsilon e)w, \tag{2}$$

where the parameter $\varepsilon \geq 0$ can be interpreted as the return to human capital investment and $w \geq 0$ is baseline income when human capital investment is equal to 0.

We posit that human capital investment is chosen at stage one and this decision cannot be revised or updated at stage two, while fertility is chosen at stage two, to allow for the possibility of new information influencing fertility choice.

After substituting constraints (1) and (2) into the objective function, we can write women's decision problem as follows:

$$max_{e \geq 0, b \in [0, \bar{b}]} \{-v(e) + \mu(b) \left[u((1 + \varepsilon e)w) - h(\varphi b) + \kappa(sb)\right]\}.$$

The assumptions guarantee that utility is strictly increasing and concave in consumption and the number of children, and decreasing and convex in human capital investment, e. The Inada condition on the utility from children implies that $b > 0$ at the optimum.

We also impose the following additional assumptions.

Assumption 1 $u(w) - h(\varphi \bar{b}) + \kappa(0) > 0$

Assumption 2 $-h'(\varphi \bar{b})\varphi + \kappa'(s\bar{b})s > 0$

Assumption 1 guarantees that women's utility conditional on survival is positive and thus their welfare rises if the maternal mortality rate declines. Assumption 2 that ensures the marginal utility of an additional birth is positive for all $b \in [0, \bar{b}]$.

The first order necessary and sufficient conditions for this problem are:

$$-v'(e) + \mu(b)u'((1 + \varepsilon e)w)\varepsilon w \leq 0, \tag{3}$$

with equality for $e > 0$, and

$$\mu'(b) \left[u((1 + \varepsilon e)w) - h(\varphi b) + \kappa(sb)\right] + \mu(b)[-h'(\varphi b)\varphi + \kappa'(sb)s] \leq 0, \tag{4}$$

with equality for $b \in (0, \bar{b})$.

The first optimality condition clearly implies that human capital investment, e, is increasing in w, ε and $\mu(b)$, at an interior solution. Also, for $\mu(b)$ and ε low enough, the only solution is $e = 0$.

The second optimality condition lays out the trade-off associated with an additional birth. The first term corresponds to the loss in expected utility due to the fact that the pregnancy related death risk rises with each birth. The second term is the expected marginal value of an additional child for the mother. Clearly, lower values of $\mu'(\cdot)$ and $\mu(\cdot)$ tend to decrease the optimal number of births for given e by this equation. Assumptions 1 and 2 guarantee that the optimal number of births will always be strictly positive.

We now conduct the comparative statics analysis for the parameters of interest to evaluate the impact of advances in maternal health on the demand for children and investment in human capital.

3.1 Comparative Statics

The goal of the comparative statics analysis is to derive predictions on the human capital and fertility response of different cohorts of women who are confronted with advances in maternal and infant health at different points in the life cycle.

We specialize the function $\mu(\cdot)$ to an analytically tractable linear specification: $\mu(b) = 1 - \upsilon b$, with $\upsilon \in [0, 1/\bar{b})$. The parameter υ represents the marginal increase in maternal death risk with each additional birth and maps directly into the empirical maternal mortality rate. With this formulation, there are three health parameters in the model. The pregnancy related death risk, υ, the health burden of pregnancy conditional on survival, φ, and the infant survival probability, s. We consider the response to both unanticipated and anticipated changes in these health parameters.

We say that a change in a health parameter is *unanticipated* when it occurs after human capital investment has been chosen at stage one. Thus, for unanticipated changes, we derive the fertility response keeping human capital investment constant. *Anticipated* changes in the health parameters are assumed to occur at stage one, so that both human capital investment and fertility will respond. An unanticipated change in a health parameters occurs while women are in their childbearing years, while anticipated changes in health parameters occur during women's early adulthood, when they can still adjust schooling choices, in addition to fertility.

The response of optimal fertility and human capital to a change in health parameters can be interpreted as a comparison across different populations/cohorts of women, confronted with different health conditions. We examine these comparisons analytical for each of the health parameters in the model.

3.1.1 Decline in Maternal Mortality

A decline in maternal mortality corresponds to a reduction in the parameter υ in the model.

The response of fertility and human capital investment to a change in the value of υ at an interior solution can be obtained by totally differentiating equations (3) and (4), evaluated at equality, with respect to υ. We summarize the results in the following proposition.

Proposition 1 *Let Assumption 1 and Assumption 2 hold. Then, fertility rises in response to an unanticipated reduction in υ. Moreover, if υ, w and ε are high enough:*

$$\frac{\partial b}{\partial \upsilon} \leq 0,$$

$$\frac{\partial e}{\partial \upsilon} \leq 0.$$

Thus, fertility and human capital investment rise in response to an anticipated reduction in υ.

Proof. In Appendix B. ■

Under Assumptions 1 and 2, which guarantee that women's utility is decreasing in υ and that the marginal utility of an additional birth is positive, fertility rises in response to an unanticipated rise in maternal survival probability, that is a decline in υ at stage two.

The solution implies that an anticipated reduction in maternal mortality risk will not have a positive effect on fertility or human capital if such risk is low to start with or if women's consumption, and therefore the opportunity cost of maternal death, is low. If instead the initial mortality risk and income, which determines the opportunity cost in terms of utility of a maternal death, are high enough, an anticipated decline in maternal mortality risk increases fertility and human capital.

Assumption 2 is sufficient but not necessary for the result. Given convexity of the function $h(\cdot)$ and the concavity of $\kappa(\cdot)$, the marginal utility of an additional birth will be positive for initially low fertility, high youth survival probability and low values of the health burden of childbirth. Moreover, the analytical expression for the response of fertility to a change in υ (derived in Appendix B) implies that the magnitude of the fertility response is positively related to utility conditional on survival. Thus, it will tend to be greater for women with high income, w, for low values of the health burden φ and high values of the youth survival probability, s.

3.1.2 Decline in the health burden of pregnancy

The health burden associated with pregnancy and childbirth is represented in the model by the parameter φ. The following proposition summarizes the predictions for the effect of a change in this parameter.

Proposition 2 *Let Assumption 1 and Assumption 2 hold. Then, fertility rises in response to an unanticipated reduction in φ if υ and φ are low enough. The response of fertility and human capital always have opposite sign for anticipated changes in φ. If υ, w and ε are high enough, the response to an anticipated change in φ is:*

$$\frac{\partial b}{\partial \varphi} \leq 0,$$

$$\frac{\partial e}{\partial \varphi} \geq 0.$$

Thus, fertility rises and human capital investment falls in response to an anticipated reduction in υ.

Proof. In Appendix B. ■

Assumption 2, that is the demand for children is not saturated, is not sufficient to generated a positive response to an unanticipated decline in the health burden. In addition, it must be that the maternal mortality risk is low enough and the initial health burden are low enough. That is for given human capital and therefore consumption, only if the weight on utility conditional on survival is high enough and if the health burden is low enough is it optimal to increase fertility in response to a decline in the health burden.

For an anticipated reduction in the health burden of pregnancy, the response of human capital and fertility always have opposite signs. For given maternal mortality risk, a change in the number of births affects the probability with which women will enjoy consumption, and therefore the effective expected marginal value of human capital investment. If fertility rises in response to a reduction in the health burden, this reduces the marginal value of human capital since it decreases the probability with which women will enjoy the resulting additional consumption.

Once again, only if the initial maternal mortality risk is high enough and income is high enough, the decline in the health burden causes fertility to rise. The logic behind this result is that for high values of the maternal mortality risk, the marginal returns to human capital in terms of utility are low. Therefore, it is optimal to respond to a decline in the health burden by increasing fertility and reducing human capital investment.

3.1.3 Youth Survival Probability

Finally, we examine the response to an increase in youth survival probability, s. We introduce the following additional assumption:

Assumption 3 $1 \leq -\frac{\kappa''(x)x}{\kappa'(x)}$ *for all* $x \in [0, \bar{b}]$.

The results are summarized in the following proposition.

Proposition 3 *If Assumption 3 holds, fertility declines in response to an unanticipated rise in infant survival probability. The human capital response to an anticipated change in infant survival probability always has opposite sign to the fertility response. If* υ, w *and* ε *high enough:*

$$\frac{\partial b}{\partial s} \leq 0,$$

$$\frac{\partial e}{\partial s} \geq 0.$$

Thus, fertility rises and human capital investment falls in response to an anticipated rise in s.

Proof. In Appendix B. ■

It is well known that standard fertility models have ambiguous predictions on the response of fertility to a rise in youth survival probabilities (Doepke, 2005, and Jones and Schoondroodt, 2007). This result also applies to the model we analyze. Assumption 3 implies that the coefficient of relative risk aversion in the utility from children is greater than 1 and guarantees the the response of fertility to an unanticipated rise in infant survival probability is negative. If in addition, υ, w and ε high enough, that maternal mortality risk and the opportunity cost of maternal deaths are high enough, then fertility also falls in response to an anticipated rise in youth survival probability. This condition that guarantee a negative relation between maternal mortality and fertility, and thus is the relevant parameter configuration for our analysis.

An additional result is that the response of fertility and the response of human capital always have opposing signs. The logic for this result is that if fertility rises, the marginal value of human capital falls, given that maternal survival probability is negatively related to the number of births.

3.2 Summary and Discussion

We can summarize the results on the impact of advances in maternal health on fertility and human capital investment as follows:

- An *unanticipated* reduction in maternal death risk generates a rise in fertility. The magnitude of the fertility response is positively related to income.
- An *anticipated* reduction in maternal death risk generates a rise in fertility and in human capital investment if the initial maternal death risk is high enough and if income is high enough.
- An *unanticipated* decline in the health burden of pregnancy causes a rise in fertility if the pregnancy related death risk is *low* enough.
- An *anticipated* reduction in the health burden of pregnancy generates a rise in fertility if the pregnancy related death risk is high enough and if income is high enough. If fertility rises, human capital will fall.

These results highlight the different mechanism through which the two dimensions of maternal health influence the demand for children and the incentive to invest in human capital. A decline in maternal death risk causes both fertility and human capital to rise, since it increases the marginal value of human capital investment and th emarginal value of a birth. By contrast, the response of the fertility and human capital to a reduction in the health burden of pregnancy always have opposing signs.A positive fertility response, then for given maternal death risk, reduces the marginal value of human capital investment, since it decreases $\mu(b)$.

There response of fertility and human capital to advances in maternal health depends on initial conditions. An anticipated reduction in the pregnancy related death risk causes fertility and human capital investment to rise, only if the initial mortality risk and income are high. Thus, a baby boom in response to a reduction in maternal death risk will arise in high income economies, and as maternal mortality risk declines, further improvements will not have a positive effect on fertility. Under similar conditions, a decline in the health burden will induce a rise in fertility, but a decline in human capital.

The model thus predicts that, as successive cohorts of women are exposed to an improvement in maternal health, fertility will at rise if maternal mortality risk is initially high, and if income is high enough. Then, as maternal mortality risk declines, the response of fertility to furthers improvements in maternal health will weaken and potentially reverse. Human capital investment will rises on response to a reduction in maternal mortality, but will fall in response to the decline in the health burden of pregnancy.

Improved maternal health influences the demand for children via additional channels. For example, if a dynastic utility term as in Barro and Becker (1988) is included, children's utility may be higher if the mother survives. Moreover, mothers arguably could experience a utility cost of the health burden of pregnancy even if they do not survive childbirth. Extending the model to allow for these features preserves all the qualitative predictions discussed above.

An additional effect of improved maternal health is to extend the length of the fecund period. This may affect the number of desired children, as well as the timing of fertility. This effect cannot be analyzed in the current model given that there are only two stages in life.

A novel feature of this model is that it considers women's decision to invest in their *own* human capital, as well as their fertility decisions. Typical models of human capital investment and fertility consider the parents' decision on the number of children and their human capital. Thus, the mechanism for the joint determination of human capital and fertility model differs from the standard literature.[32] Clearly, we could also endogenize human capital for children, by allowing the average child utility U, typically interpreted as child quality and related to human capital investment, to be chosen in the model. We leave this extension to future work.

We now examine empirical evidence on the response of fertility and human capital for subsequent cohorts of women exposed to the improvement in maternal health in the US.

4 Empirical Analysis

We now proceed to examine the empirical link between the improvement in maternal health and fertility and women's human capital. Due to the absence of time series data on maternal morbidity, we interpret the

[32] In Jayachandran and Lleras-Muney (2008), a decline in maternal mortality increases parents' returns to human capital investment on daughters. Soares (2005) considers a fertility choice model in which adults can make investments in their own human capital, in addition to the human capital of their offspring.

maternal mortality ratio as a proxy of maternal health.

Two features of the decline in maternal mortality clearly stand out. First, maternal mortality did not decline substantially until 1936, when it started to drop dramatically. By the late 1950s, it had reached modern levels.[33] This pattern allows us to identify quite precisely the cohorts of women who experienced the improvement in maternal health, and the stage of the life cycle at which they were exposed. 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.[34] 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 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.

Table 3: Cross-state variation

	MMR* (per 10,000 live births)			CBR** (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 strategy will be to identify the effect of the decline in maternal mortality on fertility and women's education. 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

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

[34] Maternal mortality dropped by more in levels but less in percentage terms in the states where it was initially high, as shown in Table 19 in Appendix C.

a homogeneous sample, we restrict attention to white women only.[35]

The next session describes the estimation procedure in detail, laying out the main concepts and maintained assumptions. Section 4.1.1 and 4.1.2 provide a detailed analysis of variation across cohorts and across states of the variables used in the estimation. Finally, Sections 4.2 and 4.3 discuss the estimation results in detail.

4.1 Estimation

We estimate the following equation:

$$\Delta Y_{sc} = \alpha_o + \alpha_1 \Delta MMR_{sc} + \alpha_2 \Delta X_{sc} + \alpha_3 Z_{s\bar{c}} + \alpha_4 Z_s + \varepsilon_s, \tag{5}$$

where s indexes state of residence, $\Delta Y_{sc} = Y_{sc} - Y_{s\bar{c}}$ represents the difference in outcome Y for state s between cohorts c and $\bar{c}$. The variable ΔMMR_{sc} is the drop in maternal mortality across the two cohorts, ΔX_{sc} corresponds to a set of changes of state-level controls, X, $Z_{s\bar{c}}$ includes a set of state controls observed for the older cohort in the comparison while Z_s includes a set of state-level characteristics that are invariant across cohorts. The coefficient of interest in our analysis is $\alpha_{1.}$

The model discussed in Section 3 predicts that the fertility response to a reduction in maternal mortality depends on when the treatment occurs during the life cycle. Thus, we consider maternal mortality at two different stages of the life cycle. *Expected* maternal mortality is the value of this variable in the state at age 18, which we take to correspond to the beginning of adult life, when women can still make decisions about schooling and early career paths. *Concurrent* maternal mortality is the value of this variable during childbearing years, and we interpret it as a proxy of the health risks that women face as they are having children. We compute it as the average value of maternal mortality at age 20-31, which corresponds to a two-year interval around the minimum age of first birth (age 22.7) and the maximum age of last birth (age 29.5) for the cohorts in our analysis (see Table 4). Only expected maternal mortality can affect human capital investment, while both expected and concurrent maternal mortality potentially influence the fertility response. The relative magnitude of the decline in these two measures of maternal mortality for each cohort can be used to isolate the unanticipated component of the reduction in maternal mortality.

We adopt Children Ever Born (CEB) at age 32-39 as a measure of completed fertility, and report results for this outcome in Section 4.2. To assess the impact on women's human capital, we consider the male-female differential in college graduation rate, evaluated at age 32-39, as a dependent variable. These results are reported in Section 4.3. We also examine the relation between the drop in infant mortality and the change in fertility across cohorts. Section 4.5 reports these findings.

We now describe our selection of cohorts and discuss the cross-state variation in the variables of interest for the cohort comparisons used in the estimation. We also examine a large set of potential predictors of maternal mortality, which will inform our choice of controls. Appendix A provides a detailed description of the data sources and variable definitions.

4.1.1 Cohorts

We consider women at age 22-29 in each Census year from 1930 and to 1960. This age range encompasses the childbearing years for all the women in our analysis and allows us to identify a selection of cohorts which

[35]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.

is representative of the historical variation in the magnitude and the timing in the exposure to the drop in maternal mortality.

Table 4 reports selected summary statistics for the cohorts included in the analysis.

Table 4: Cohort Definition and Summary Statistics

	Pre-Shock	*Early-Shock*	*Mid-Shock*	*Full-Shock*
Born	1901-08	1911-18	1921-28	1931-38
Age [22-29] in Year	1930	1940	1950	1960
Exposure	None	Partial drop in concurrent MMR	Partial drop in expected MMR; Concurrent MMR lower than expected	Full drop in expected MMR; Concurrent MMR close to expected
Expected MMR (per 10,000 live births)	63.19 (16.9)	56.65 (8.0)	23.78 (4.83)	4.85 (1.45)
Concurrent MMR (per 10,000 live births)	60.03 (8.94)	33.24 (5.61)	8.61 (2.10)	2.68 (0.62)
Expected IMR (per 1,000 live births)	63.52 (20.55)	55.26 (12.49)	39.99 (10.00)	25.98 (4.08)
Concurrent IMR (per 1,000 live births)	59.91 (13.42)	44.69 (10.91)	29.19 (5.47)	23.3 (2.28)
Children Ever Born	2.71 (0.42)	2.57 (0.27)	2.80 (0.21)	3.22 (0.26)
Age at First Birth	23.41 (0.67)	24.58 (0.68)	23.72 (0.67)	22.7 (0.79)
Age at Last Birth	28.45 (0.68)	29.48 (0.48)	29.51 (0.54)	28.68 (0.69)
College Grad. Rate	0.048 (0.05)	0.062 (0.02)	0.10 (0.04)	0.098 (0.04)
M-F Differential in College Grad. Rate	0.03 (0.07)	0.05 (0.04)	0.11 (0.03)	0.12 (0.04)

Standard errors in parentheses.

Notes: The mortality measures refer to the white population. The table reports state level averages for a sample of white married men and women, living in non-farm households. Completed fertility and age at first and last birth are computed for women with children.

The Pre-shock cohort is clearly untreated, while subsequent cohorts experience the advances in maternal health to varying degrees. To isolate this exposure, we focus on three "experimental comparisons." The first is between the Early-shock and the Pre-shock cohort. Expected MMR drops by 10% for this comparison, from 63 deaths per 10,000 live births for the Pre-shock cohort to 56.65 deaths for the Early-shock cohort. Thus, this cohort comparison can be treated as a placebo for expected maternal mortality. Concurrent maternal mortality drops by 45%, reaching 33.24 per 10,000 live births from 60.03 for the Pre-Shock cohort. While this drop is sizable, it occurs rather late in the childbearing years and only for the states with initially high expected maternal mortality (see Table 19).

The second experimental comparison is between the Mid-shock and the Early-shock cohorts. Mean expected and concurrent maternal mortality decline by 52% and 86% respectively for this comparison. While expected maternal mortality at 24 deaths per 10,000 live births is still sizable, concurrent maternal mortality at 8.61 deaths per 10,000 live births is close to modern levels. This difference can be used to isolate the impact of unanticipated improvement in maternal health. Since the drop in expected maternal mortality is relatively modest and very recent for this cohort, we do not expect to find a strong impact on fertility and educational attainment.

The third experimental comparison is between the Full-shock and Early-shock cohort. Mean expected and concurrent maternal mortality both drop by 92% for this comparison. Concurrent and expected maternal mortality are very similar for the Full-shock cohort. Thus, these women are the first to experience modern values of maternal mortality from early adulthood. Moreover, since the decline in maternal mortality had been ongoing for a significant period of time[36], Full-shock women should respond with both human capital and fertility. Moreover, their fertility response should mainly be driven by expected maternal mortality, since concurrent maternal mortality was very similar in value.[37]

Figure 4 plots the relation between the drop in expected maternal mortality and the change the variables of interest for the experimental cohort comparisons. There is a clear positive relation between the drop in expected maternal mortality and the change in the endogenous variables for the comparisons involving the Mid-shock and Full-shock cohort. The correlation between the percentage drop in expected maternal mortality and the percentage change in completed fertility is 0.42 for the Mid-shock vs Early-shock comparison, and 0.56 for the Full-shock vs Early-shock comparison. There is a negative relation between the drop in expected maternal mortality and the change in the male-female differential in college graduation rate for both the Mid-shock vs Early-shock and the Full-shock vs Early-shock comparison. The correlation coefficient between these two variables is -0.10 for the Mid-shock vs Early-shock comparison, and -0.23 for the Full-shock vs Early-shock comparison.

The estimation strategy is based on the maintained assumption that the cross-state variation in initial maternal (and infant) mortality, and in the magnitude of the drop in maternal mortality are exogenous. However, our analysis may be subject to omitted variable bias. To address these concerns, the next two sections analyzes possible predictors of maternal mortality for the cohorts included in our analysis.[38]

4.1.2 Predictors of Maternal Mortality

This section considers several possible predictors of maternal mortality to assess the potential for omitted variable bias and guide the choice of controls in the estimation. In addition to a standard set of demographic and economic indicators, we include additional variables based on the historical determinants of the drop in maternal mortality.

The first group comprises state-level demographic indicators, such as the share of whites, the share living on a farm and the share of foreign born in the population, and state-level economic indicators, such as the share of employment in the health sector and in public administration, in agriculture and in textiles, the unemployment rate, the share of the 18-64 population with a high school degree and the male and female hourly wage for full-time year-round workers.[39] All variables are evaluated at the corresponding Census year, unless otherwise noted.

The second group of predictors includes the literacy rate in 1930, the year of acceptance of women's suffrage, and the total per capita federal payments received by each state under the 1921-1929 Sheppard-Towner Act and the 1936-1939 Social Security Act, Title V, Part 1. This newly digitized data on state level

[36]The dramatic decline in maternal mortality was widely reported by the national press since it began in the late 1930s, owning to the high degree of awareness of maternal mortality as a major social problem in the late 1920s and early 1930s.

[37]We will also examine the comparison between the Mid-shock and Full-shock cohorts and the Pre-shock cohort. Though, we treat the Early-shock cohort as the untreated cohort for the purpose of our estimation. This is to avoid the impact of any changes in correlates across cohorts that are not accounted for by the controls variables on the estimation results.

[38]Table 4 also documents the behavior of infant mortality (IMR). We will discuss its decline and link it to the change in fertility in Section4.5.

[39]The 1930 Census does not have data on labor income and educational attainment.

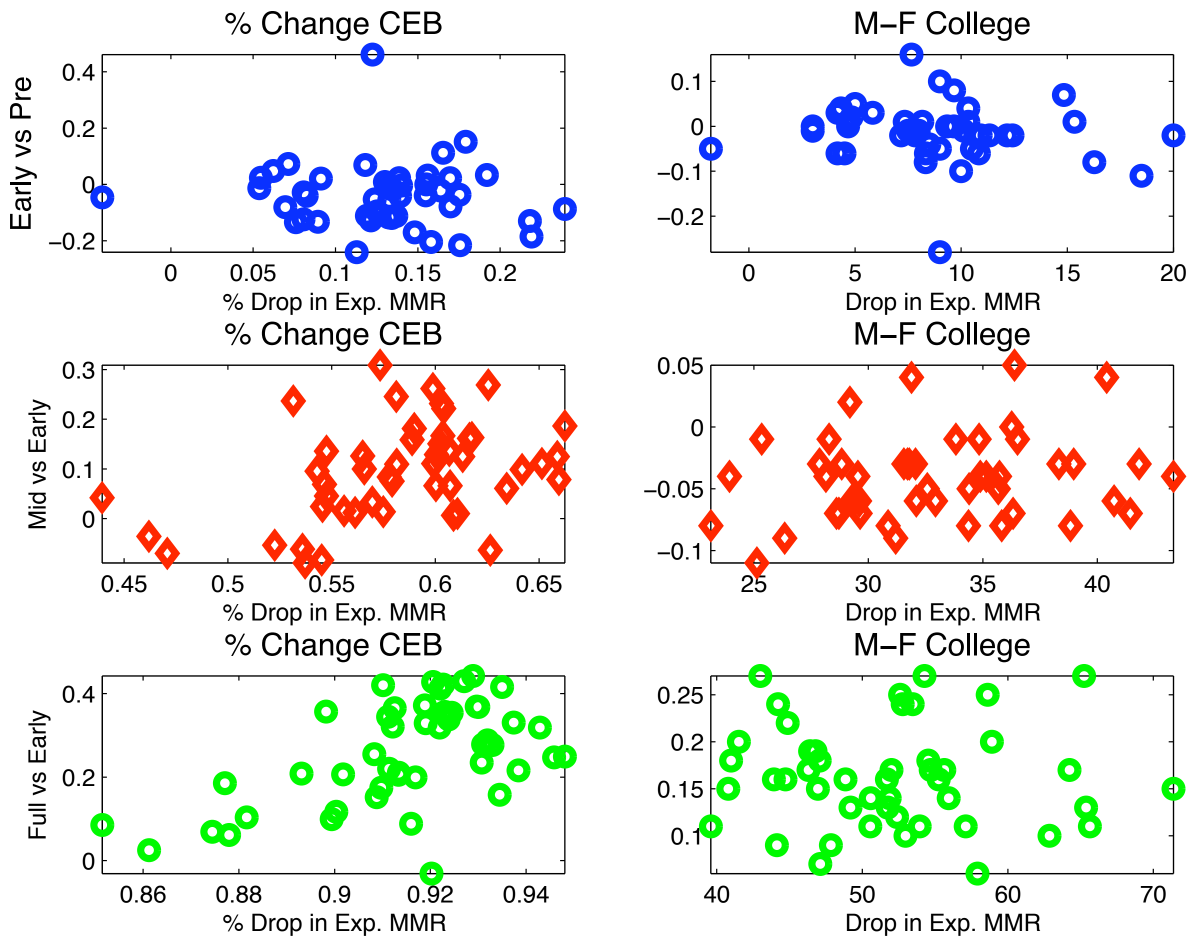

FIGURE 4: Relation with the drop in expected maternal mortality

appropriations and spending for infant and maternal health under these two important pieces of legislation is described more in detail in Appendix A.5.

The findings for the demographic and economic indicators are presented in Table 5. Only the share of whites and the share with high school significantly predict maternal mortality among the demographic indicators. Specifically, the share of whites is negatively related to maternal mortality, for the Pre-shock cohort and the Early-shock cohort. The share with high school is significantly negatively related to maternal mortality starting with the Mid-shock cohort.

Per capital personal income and the male full time wage are significantly negatively related to maternal mortality for all cohorts. The female full time wage displays a negative and significant relation with expected maternal mortality for the Full-shock cohort, and with concurrent maternal mortality for the Early-shock, Mid-shock and Full-shock cohorts.[40] The share of health sector employment is strongly negatively related with both expected and concurrent maternal mortality for all cohorts. The share of employment in agriculture is positively related to expected maternal mortality for the Mid-shock cohort.

The findings for the cultural and federal funding indicators are displayed in Table 6. We include two cultural indicators: literacy and the attitude towards women's suffrage. Literacy, which is only available up to 1930 from Census data, is potentially linked to the ability to absorb information about improved medical techniques and hygienic practices that importantly influence both maternal and infant health. Since diffusion of basic schooling is also linked to a "progressive era" activism, literacy can be considered a proxy for the sensitivity towards health of mothers and children, a key item in the progressive agenda (Skopcol, 1992).

The attitude towards women's suffrage could be linked to maternal health and fertility via multiple channels. In the aggregate, an early attribution of voting rights to women may increase their 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 shows that child mortality was lower and spending for public health higher in states that introduced women's suffrage early. At an individual level, greater political power or a positive attitude towards 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. The variable "Acceptance Year" is a proxy for the attitude towards women's suffrage. This variable corresponds to the date at which a state introduced 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.[41]

[40] Measures of income and wages are a natural predictor of health outcomes, since richer states may be better able to finance public health interventions and the construction of hospitals. A reverse causal relation is also likely, as better health may lead to higher income and productivity.

[41] 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.

Table 5: Economic and Demographic Predictors of Maternal Mortality

Expected Maternal Mortality

		share white	share farm	share foreign born*	share high school*	share in health	share in public	share agri-colture*	share textile*	unemploy ment rate	per capita personal income*	female full time real wage*	male full time real wage*
1930/Pre-shock	coeff	-57.82	5.11			-683.12	-33.51			-36.09			
	t-stat	-3.00	0.54			-1.75	-0.65			-0.56			
	p-val	0.00	0.59			0.09	0.52			0.58			
1940/Early-shock	coeff	-31.84	-0.77	-45.56	7.09	-356.23	-41.49	9.67	31.22	-53.86	0.00	-4.84	-6.63
	t-stat	-2.13	-0.09	-1.30	0.68	-1.52	-1.17	0.22	1.00	-1.21	-2.41	-1.45	-2.73
	p-val	0.04	0.93	0.20	0.50	0.14	0.25	0.83	0.32	0.23	0.02	0.15	0.01
1950/ Mid-shock	coeff	-14.98	5.71	-33.72	-15.92	-294.67	-12.88	50.92	0.41	-25.94	0.00	-2.53	-4.71
	t-stat	-1.80	1.15	-1.59	-2.50	-2.56	-0.84	2.16	0.03	-0.64	-3.52	-1.53	-3.59
	p-val	0.08	0.26	0.12	0.02	0.01	0.40	0.04	0.98	0.53	0.00	0.13	0.00
1960/ Full-shock	coeff	-0.01	3.95	-12.17	-11.81	-116.94	-4.26	2.74	-2.76	10.86	0.00	-0.88	-0.58
	t-stat	0.00	2.14	-1.55	-4.27	-3.90	-0.80	0.31	-0.51	0.67	-3.48	-3.23	-3.32
	p-val	1.00	0.04	0.13	0.00	0.00	0.43	0.76	0.62	0.51	0.00	0.00	0.00

Concurrent Maternal Mortality

		share white	share farm	share foreign born*	share high school*	share in health	share in public	share agri-colture*	share textile*	unemploy ment rate	per capita personal income*	female full time real wage*	male full time real wage*
1930/Pre-shock	coeff	-47.352	-1.729			-434.380	-34.781			2.750			
	t-stat	-3.046	-0.232			-1.342	-0.819			0.054			
	p-val	0.004	0.818			0.186	0.417			0.957			
1940/Early-shock	coeff	-25.522	6.782	-36.48	-2.983	-452.400	-31.655	5.74	12.17	-51.577	-0.003	-4.628	-6.750
	t-stat	-2.747	1.333	-1.69	-0.462	-2.914	-1.282	0.21	0.64	-1.672	-3.791	-2.013	-4.376
	p-val	0.009	0.189	0.10	0.647	0.005	0.206	0.83	0.53	0.101	0.000	0.050	0.000
1950/ Mid-shock	coeff	-0.856	4.726	-6.45	-9.678	-163.230	-11.756	16.91	-5.11	-9.261	-0.001	-1.486	-2.332
	t-stat	-0.245	2.273	-0.72	-3.614	-3.411	-1.822	1.71	-0.82	-0.523	-4.186	-2.119	-4.275
	p-val	0.807	0.028	0.47	0.001	0.001	0.075	0.10	0.42	0.603	0.000	0.039	0.000
1960/ Full-shock	coeff	-0.076	1.234	-0.61	-4.987	-43.223	-0.995	-2.41	-4.64	8.451	0.000	-0.272	-0.184
	t-stat	-0.080	1.515	-0.18	-4.086	-3.249	-0.439	-0.61	-1.93	1.230	-2.503	-2.240	-2.336
	p-val	0.937	0.137	0.86	0.000	0.002	0.663	0.54	0.06	0.225	0.016	0.030	0.024

* Data for these variables is not available in the 1930 U.S. Census.

Table 6: Federal Funding for Maternal and Infant Health

Cohorts		Acceptance Year	SSA per capita payments	Sheppard-Towner per capita payments	Literacy rate in 1930
Expected Maternal Mortality					
Pre-shock	coeff	0.13	19.63	85758.00	-134.66
	t-stat	1.54	2.21	2.22	-4.22
	p-val	0.13	0.03	0.03	0.00
Early-shock	coeff	0.13	13.29	52034.00	-102.88
	t-stat	2.44	2.61	3.16	-4.67
	p-val	0.02	0.01	0.00	0.00
Change in Expected Maternal Mortality					
Mid-shock	coeff	0.00	-0.02	-347.93	-0.74
vs	t-stat	2.80	-0.46	-1.54	-5.33
Early-shock	p-val	0.01	0.65	0.13	0.00
Full-shock	coeff	0.00	0.00	-50.74	-0.23
vs	t-stat	1.27	0.06	-0.51	-3.22
Early-shock	p-val	0.21	0.95	0.61	0.00

Notes: Robust standard errors used. Acceptance Year is the date at which a state introduced women's suffrage or, if later, the date at which it ratified the XIX Amendment. SSA per capita payments is the sum of the total payments to the state for the Social security Act, Title V, Part 1, for years 1936-1939 divided by the population in the state in that year. Sheppard-Towner Act total payments per capita is the same statistic for years 1921-1929.

The results are reported in Table 6. The literacy rate in 1930 is strongly negatively related to expected maternal mortality for the Pre-shock and Early-shock cohort. The acceptance of women's suffrage is a strong predictor of maternal mortality for both the Pre-shock and Early-shock cohort. The payments per capita under the Sheppard-Towner Act do not predict expected maternal mortality for the Pre-shock and Early-shock cohort. The payments per capita under the Social Security Act are positively related to expected maternal mortality for the Pre- and Early-shock cohort. This positive relation may be driven by the fact that the poorest states drew more funds per capita under the Social Security Act.

4.2 Results: Maternal Mortality and Fertility

We estimate equation 5 using the level change across cohorts in the number of children ever born at age 32-39 as a dependent variable and the level drop in either concurrent or expected maternal mortality across the same cohorts as the main explanatory variable. A positive estimate for the coefficient α_1 implies that the drop in maternal mortality is associated with a rise in fertility.

We adopt a level change specification to obtain conservative estimates of the coefficient on the drop in maternal mortality. As shown in Table 19, there is a positive correlation between maternal mortality and fertility for the Pre-shock and Early-shock cohort. Expected maternal mortality declined more in levels in the states where it was originally high, but less in percentage terms, while completed fertility rose by more in states where it was initially high. This pattern tends to generate an upward bias in α_1 for the percentage specification and a downward bias for the level specification.

The baseline specification for all regressions includes the level of maternal mortality (expected or concurrent) for the older cohort and the change in state per-capita household income. The former allows to capture a possible direct relation between the change in fertility and the initial level of maternal mortality, while the latter controls for the impact of change in general economic conditions on the fertility behavior of the young cohort in the comparison. We also include the change in concurrent infant mortality for each comparison, to capture the effect of its decline on the change in fertility.[42] We then progressively include the control variables discussed in Section 4.1.2. Finally, we include controls that potentially account for forces independently affecting fertility according to the existing explanations of the baby boom. We use the percentage of dwellings with refrigerators as a proxy for the diffusion of home appliances, based on Greenwood, Seshadri and Vanderbroucke (2005).[43] To account for the hypothesis in Doepke, Hazan and Maoz (2007) that the rise in labor force participation of married women during World War II crowed out younger women from the labor market after the war, causing them to opt for marriage and child bearing, we use state level mobilization rates as a proxy for the proportion of women who went to work during the war and the resulting impact on labor market conditions.[44] Since mobilization rates were lower for non-whites and in farming areas, we also jointly control for the share of non-white and the share of farmers in the population in 1940, to isolate the effect of mobilization rates. Given that we control for the initial level of maternal mortality, the estimates capture the independent effect of these controls on the change in fertility.

4.2.1 Early-Shock Cohort

Expected maternal mortality for the Early-shock cohort only drops by 10% relative to the Pre-shock cohort and continues to be very high, as shown in Table 4. Thus, we should not find an effect of drop in expected maternal mortality on the cross-cohort change in completed fertility. A significant estimated relation between maternal mortality and fertility for this cohort comparison could be a symptom of pre-existing trends or omitted variable bias. The 45% drop in concurrent maternal mortality is more sizable, though it occurs late in the childbearing years for Early-shock women and thus most will not be able to respond.

The results, reported in Table 7 suggest that the differences across states in the drop in maternal mortality not related to the change in completed fertility for this cohort comparison. The only significant coefficient suggests a negative effect on the change in fertility of the drop in concurrent maternal mortality. On the other hand, the drop in concurrent infant mortality appears to have a sizable negative effect on the change in completed fertility across these cohorts.[45] This is not surprising, given that the drop in concurrent infant mortality is the largest for this cohort comparison, as shown in Table 4.[46] Only per capita personal income, among the other controls, seems to be related to the change in fertility in this cohort comparison.

Based on these findings, we will assume the Early-shock cohort is untreated in the rest of the analysis.[47]

[42] We consider concurrent infant mortality since expected infant mortality is not systematically linked to the change in fertility. See Section 4.5.

[43] The information on the percentage of dwellings with refrigerators is available only 1940 and 1950. Ownership rates for other home appliances are available in later years.

[44] Acemoglu, Autor and Lyle (2004) find that post-war labor market conditions were significantly related to mobilization rates. Specifically, low skill salaries were lower in states with high mobilization rates, which they interpret as consistent high participation of low skill women during the war years.

[45] We use the drop in concurrent infant mortality as a control, since, as we will discuss in Section 16, the drop in concurrent

Table 7: Completed Fertility: Early-shock Cohort

MMR and Completed Fertility: Early-Shock vs. Pre-Shock Cohort

Dependent Variable is Level Change in Completed Fertility

	Expected MMR		*Concurrent MMR*	
	1	2	3	4
Level Drop MMR	-0.0119	-0.0198	-0.005	-0.0558***
	[0.0145]	[0.0118]	[0.0113]	[0.0195]
Level Drop IMR		-0.0263***		-0.0238***
		[0.0093]		[0.0081]
MMR Pre-Shock Cohort	0.0026	0.0113	0.0016	0.0356***
	[0.0071]	[0.0068]	[0.0082]	[0.0121]
Per Capita Personal Income		0.0001		0.0002**
		[0.0001]		[0.0001]
Level Change:				
Per Capita Personal Income	0	0	0	0.0001
	[0.0001]	[0.0001]	[0.0001]	[0.0001]
Share White		-7.7075*		-6.5734*
		[3.9030]		[3.5399]
Share Farm		-2.6969		-2.2093
		[2.1558]		[2.0528]
Share Foreign Born		-0.5697		-0.7468
		[2.7183]		[2.5502]
Share Health Sector		-7.7822		-5.4094
		[11.5515]		[11.0669]
Share Public Sector		-0.4056		-2.5017
		[2.6599]		[1.9973]
Unemployment Rate		-1.1396		0.2208
		[3.8168]		[3.1275]
Share High School Graduates		-1.6118		-0.893
		[2.7929]		[2.5172]
Female FT Hourly Wage		-0.0559		-0.0627
		[0.2181]		[0.1710]
Male FT Hourly Wage		0.2329		0.1245
		[0.2258]		[0.2280]
Constant	-0.21	-0.9223*	-0.0898	-1.3632**
	[0.4066]	[0.5428]	[0.3279]	[0.5330]
Observations	47	47	49	49
Adj R-squared	-0.0525	0.22	-0.064	0.344

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Pre-shock cohort, born in 1901-08. In all columns the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In all columns the sample excludes Alaska and Hawaii. In columns 1 to 3 the sample also excludes South Dakota and Texas because data on expected MMR are not available for the pre-shock cohort. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

4.2.2 Full-Shock Cohort

We first discuss the results for the Full-shock cohort, for whom both expected and concurrent maternal mortality reach modern levels. The estimates suggest a very strong positive impact of the drop in expected maternal mortality on the change in completed fertility for the comparison against the Early-shock cohort. The results are robust to the inclusion of the additional demographic and economic controls.

Table 8 presents the main estimation results. The point estimate for the baseline regression, displayed in column 1, implies that a 10 unit drop in expected maternal mortality is associated with a 1.1 unit increase in completed fertility, significant at the 1% level. Since actual fertility rose by 1.09 units for this cohort comparison, this effect is very sizable. The change in fertility is strongly negatively related to initial expected maternal mortality. Once again, the effect is sizable, as a 10 unit difference in expected maternal mortality for the Early-shock cohort reduces the change in fertility by 0.58 units, and highly significant. The cross-state variation in the change in fertility that can be explained by the drop in maternal mortality is 38% for this specification.

Columns 2 to 5 progressively incorporate the demographic and economic controls. The estimated coefficient on the maternal mortality drop falls for some specifications but remains significant. The share of white population displays a positive and highly significant coefficient, while the change in the share of employment in the public sector is negative and significant. All other controls are not significantly related to the change in fertility. Column 6 reports estimates for the sample of women born in state,[48] and column 7 excludes DC from the sample. The findings are robust to these restrictions.

Table 9 reports the estimates from specifications that include additional controls. Columns 1 and 2 control for the 1950 percentage of dwellings with refrigerators, treated as a proxy for the diffusion of home appliances. The estimated coefficient is positive and significant, but small in magnitude. A 10% rise in the share of dwellings with refrigerators would lead to a 0.03 unit increase in the change in fertility. Column 3 includes the drop in concurrent infant mortality, to explore whether the effect of the drop in maternal mortality is mitigated by the decline in infant mortality over the same period. The coefficient on the drop in expected maternal mortality remains significant, while the drop in concurrent IMR is negative but not significant.

Columns 4 controls for the cultural and government predictors of maternal mortality discussed in Section 4.1.2. Controlling for these variables reduces the magnitude and the significance of the estimated coefficient on the drop in expected maternal mortality. The literacy rate in 1930 has a positive and significant effect on the change in fertility, suggesting that a 10% difference in the literacy rate is associated with a 0.13 units rise in the change in fertility. The year of acceptance of women's suffrage also has a positive and significant effect on the change in fertility. Spending on maternal and infant health is not significantly related to the change in fertility.

We also estimate the effect of the drop in concurrent maternal mortality on fertility. (See Table 21 in AppendixC). We find that the estimated coefficient on the drop in concurrent maternal mortality on the

infant mortality has a stronger impact on fertility than the drop in expected infant mortality.

[46] We will analyze the impact of the drop in infant mortality on fertility in Section 4.5.

[47] We also repeated the analysis using the Pre-shock cohort as the untreated cohort, and we find similar and in some case stronger results. However, the comparison to the Early-shock cohort is more appropriate since it minimizes the confounding effects of changes in other variables over the longer time period.

[48] This is a potentially informative restriction, since the state of residence is endogenous and possibly dependent on underlying state characteristics correlated both with the drop in maternal mortality and the increase in fertility. Moreover, a woman born in a particular state may have formed her expectations about the pregnancy-related health burden based on the maternal mortality rate in that state or in her current state of residence, depending on when the move occurred.

Table 8: Completed Fertility: Full-shock Cohort

Expected MMR and Completed Fertility: Full-Shock vs. Early-Shock Cohort

	Dependent Variable is Level Change in Completed Fertility						
	1	2	3	4	5	6	7
Level Drop Expected MMR	0.1141***	0.0854***	0.0627*	0.0630**	0.0778**	0.1067**	0.0712**
	[0.0230]	[0.0307]	[0.0316]	[0.0288]	[0.0326]	[0.0477]	[0.0341]
Expected MMR	-0.0586***	-0.0444***	-0.0351**	-0.0362***	-0.0431***	-0.0623**	-0.0385**
	[0.0106]	[0.0142]	[0.0146]	[0.0132]	[0.0149]	[0.0231]	[0.0154]
Per Capita Personal Income		0	0	0	0	-0.0001	0
		[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0001]	[0.0000]
Level Change:							
Per Capita Personal Income	0	0	-0.0001	-0.0001*	0	-0.0001	-0.0001
	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]
Share White			2.1090***	1.6486***	0.9858	1.6546	0.7459
			[0.5152]	[0.5909]	[1.0266]	[1.6254]	[1.4021]
Share Farm			1.7542*	1.7371*	1.2739	2.9643*	1.4352
			[0.9251]	[1.0101]	[1.2255]	[1.5686]	[0.9110]
Share Foreign Born			-0.6303	0.6809	1.0822	-2.1805	-0.5236
			[1.2009]	[1.4613]	[1.3778]	[2.2226]	[1.2694]
Share Health Sector			0.955	0.625	0.6286	8.1202	0.0079
			[5.4093]	[5.1468]	[6.2520]	[11.3012]	[5.4461]
Share Public Sector			-4.1326*	-5.8697**	-5.7600**	1.5089	-4.2175*
			[2.1343]	[2.4826]	[2.4448]	[6.3059]	[2.1087]
Unemployment Rate				-3.4476	-3.917		
				[2.8870]	[2.8281]		
Share High School Graduates				2.2814	2.259		
				[1.5438]	[1.5457]		
Female FT Hourly Wage					-0.1912		
					[0.1575]		
Male FT Hourly Wage					0.0343		
					[0.1045]		
Constant	0.6572***	0.4697**	0.9249**	0.8081**	0.7638**	1.6972**	0.9183**
	[0.1904]	[0.1954]	[0.4152]	[0.3629]	[0.3682]	[0.6772]	[0.3964]
Observations	49	49	49	49	49	49	48
Adj R-squared	0.381	0.4	0.535	0.548	0.566	0.404	0.538

Notes: Robust standard errors in brackets. Full-shock cohort, born in 1931-38. Early-shock cohort, born in 1911-18. In columns 1-5 and 7 the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In column 6 the sample is further restricted to women born-in-state. In all columns the sample excludes Alaska and Hawaii. In column 7 DC is also excluded from the sample. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 9: Completed Fertility: Full-shock Cohort, Robustness

Expected MMR and Completed Fertility: Full-Shock vs. Early-Shock Cohort

Robustness Checks and Alternative Hypothesis

Dependent Variable is Level Change in Completed Fertility	1	2	3	4	5	6
Level Drop Expected MMR	0.0691**	0.0795**	0.0798**	0.0640*	0.0666*	0.0608*
	[0.0312]	[0.0318]	[0.0370]	[0.0347]	[0.0369]	[0.0334]
Share Dwellings w Refrigerators	0.3818*	0.3899*				
	[0.1926]	[0.2180]				
Level Drop Concurrent IMR			-0.0016			
			[0.0039]			
Literacy				1.2917**		
				[0.5763]		
Date Suffrage Accepted				0.0001***		
				[0.0000]		
Sheppard-Towner payments per capita				0.2663		
				[0.2991]		
Social Security payments per capita				-0.0344		
				[0.0803]		
WWII Mobilization Rates					0.6096	0.9909
					[0.9939]	[0.7367]
% Nonwhite 1940					-0.3534	
					[0.4046]	
% Farmers 1940					0.2455	
					[0.5097]	
Constant	0.5029	0.3261	0.4611**	-1.1426*	0.2776	0.2089
	[0.3993]	[0.4108]	[0.2013]	[0.5957]	[0.7681]	[0.6128]
Additional controls	{1}	{1,2}	{1}	{1}	{1}	{1}
Observations	49	49	49	49	47	47
Adj R-squared	0.557	0.585	0.388	0.528	0.562	0.559

Notes: Robust standard errors in brackets. Full-shock cohort, born in 1931-38. Early-shock cohort, born in 1911-18. The dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. Controls: 1= 1950's expected MMR and per-capita personal income, level change in: per-capita personal income, share white, share farm, share foreign born, share working in health and in the public sector, 2= 1 + level change in unemployment rate, share high school graduate, male and female full-time real hourly wage. In all columns the sample excludes Alaska and Hawaii. In columns 5-6 the sample also excludes Nevada and DC because of missing information on mobilization rates. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

change in fertility is smaller, significant only at the 10% level and not robust to the inclusion of the full set of economic and demographic controls. This result is not surprising since concurrent maternal mortality is very similar to expected maternal mortality for the Full-shock cohort.

These findings lend considerable support to the notion that the drop in maternal mortality was an important determinant for the rise in fertility of the Full-shock cohort relative to the Early-shock.

4.2.3 Mid-Shock Cohort

For this cohort, we would expect a stronger effect of the drop in concurrent maternal mortality, since it drops by 74% relative to the Early-shock coming close to modern values, whereas expected maternal mortality drops by 58%.

Table 10 reports the estimates for both expected (columns 1-7) and concurrent (columns 8-14) maternal mortality. The drop in expected maternal mortality has a positive and significant effect on the change in fertility. A 10 unit reduction in expected maternal mortality is associated with a 0.20 unit rise in fertility, while actual fertility rose by 0.23 units. Despite its notable magnitude, the estimate is not robust to the inclusion of demographic and economic controls. The drop in concurrent maternal mortality is also positively associated with fertility. A 10 unit reduction in concurrent maternal mortality is associated with a 0.50 unit rise in fertility, though the estimated coefficient is only significant at the 10% level. The inclusion of demographic and economic controls does not alter the magnitude or the significance of the estimated coefficient. However, the estimate looses significance when the sample is restricted to women born in state. If we also include the cultural and government controls, as well as World War II mobilization rates and the diffusion of home appliances, the estimated coefficient on the drop in maternal mortality is not significant and none of the additional controls are significantly related to the change in fertility.

The Mid-shock cohort and the Early-shock cohort both experienced World War II during childbearing years, but at different points in the reproductive cycle. The Early-shock cohort experienced the war during in the last phase of the childbearing years, which may affected their completed fertility, while the Mid-shock cohort was affected by the war early in their fecund years, which may have altered the timing of their fertility. This difference may exert a confounding effect in a comparison of completed fertility for these cohorts. To more precisely isolate the impact of the improvement in maternal health on fertility, we replicate the analysis for children ever born at age 22-29, a measure we refer to as *interim fertility*. This statistic is unlikely to capture completed fertility,[49] however, it is less likely to have been affected by the war for the Early-shock cohort than completed fertility. Interim fertility is also interesting in relation to Doepke, Hazan and Maoz (2007), who emphasize that earlier marriage and child bearing are the main mechanism for the rise in fertility during the baby boom. Clearly, earlier child bearing would be reflected in a greater rise of interim fertility for the Mid-shock cohort, and based on the hypothesis proposed in Doepke et al. (2007) we should expect a positive estimated coefficient on mobilization rates.

Table 11 presents the estimates for interim fertility. The coefficient on the drop in concurrent maternal mortality for the change in interim fertility is very similar to the one for completed fertility and robust across specifications. It is not significant for the specifications that include the full set of demographic and economic controls (additional controls {1,2}), but it is highly significant when the World War II and the cultural and government controls are also included. This suggest that the presence of several confounding factors for this cohort comparison. Only Sheppard-Towner funds per capita have a significant and negative effect on the

[49]The average age of last birth was 29 and 30 for the Early-shock and Mid-shock cohort (see Table 4), thought about xx? of births occurred at later ages.

Table 10: Completed Fertility: Mid-shock Cohort

MMR and Completed Fertility: Mid-Shock vs. Early-Shock Cohort

	Dependent Variable is Level Change in Completed Fertility							Dependent Variable is Level Change in Completed Fertility						
	Expected MMR							Concurrent MMR						
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Level Drop MMR	0.0241***	0.0240**	0.0067	0.0113	0.0151	0.0171	0.0074	0.0474*	0.0590*	0.0588*	0.0561*	0.0618*	0.0382	0.0597*
	[0.0088]	[0.0106]	[0.0167]	[0.0164]	[0.0171]	[0.0383]	[0.0170]	[0.0261]	[0.0317]	[0.0319]	[0.0323]	[0.0352]	[0.0364]	[0.0350]
MMR	-0.0250***	-0.0250***	-0.0127	-0.0167	-0.0176	-0.03	-0.0136	-0.0545***	-0.0655***	-0.0641**	-0.0498	-0.0421	-0.1068	-0.0499
	[0.0053]	[0.0083]	[0.0130]	[0.0127]	[0.0126]	[0.0293]	[0.0132]	[0.0169]	[0.0238]	[0.0245]	[0.0320]	[0.0321]	[0.0807]	[0.0321]
Per Capita Personal Income		0	0	0	0	-0.0001	0		0	0	0	0	-0.0001	0
		[0.0000]	[0.0001]	[0.0001]	[0.0001]	[0.0002]	[0.0001]		[0.0000]	[0.0000]	[0.0001]	[0.0001]	[0.0002]	[0.0001]
Level Change:														
Per Capita Personal Income	0	0	0	-0.0001	-0.0002	-0.0002	0	0	0	0	0	-0.0001	-0.0001	0
	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0002]	[0.0002]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0002]	[0.0002]	[0.0001]
Share White			1.655	1.5286	1.6618	-1.3802	2.4473			0.1651	0.976	1.3977	-2.7165	0.9379
			[1.5320]	[1.5444]	[1.5300]	[5.4648]	[2.4555]			[0.7205]	[1.3247]	[1.4267]	[5.4704]	[2.6059]
Share Farm			0.3828	0.4553	0.4133	1.7144	0.4962			0.2959	0.0261	-0.0541	0.8516	0.0206
			[1.1154]	[1.1577]	[1.4021]	[3.5720]	[1.2104]			[1.0215]	[1.0401]	[1.3590]	[2.9742]	[1.1444]
Share Foreign Born			-0.0556	1.0224	0.6977	-1.7642	-0.1279			-0.7605	-0.3083	0.6774	-2.0939	-0.3086
			[2.6541]	[2.7852]	[2.7650]	[4.9598]	[2.6972]			[2.7758]	[2.6564]	[2.7780]	[4.7065]	[2.6595]
Share Health Sector			10.663	8.1123	10.3563	23.5622	10.9951				7.7773	9.1332	17.2708	7.7517
			[7.6654]	[6.8874]	[6.7553]	[16.7512]	[7.7036]				[6.8963]	[5.8867]	[16.7451]	[7.1578]
Share Public Sector			-3.8042	-4.0598	-4.5038	10.2573	-3.4485				-2.5805	-3.5447	12.672	-2.5887
			[3.1378]	[4.2269]	[3.8233]	[13.4608]	[3.4830]				[3.3217]	[4.3369]	[14.1377]	[3.4279]
Unemployment Rate				-3.8214	-4.7006							-4.1244		
				[2.6606]	[2.8015]							[2.6085]		
Share High School Graduates				0.2963	0.5081							0.347		
				[1.8132]	[1.7294]							[1.7756]		
Female FT Hourly Wage					-0.07							-0.1136		
					[0.1682]							[0.1609]		
Male FT Hourly Wage					0.1142							0.11		
					[0.1055]							[0.0988]		
Constant	0.9730***	0.9707**	0.6785	1.0265	0.904	1.8501	0.6797	0.9753***	1.1759***	1.2156***	0.9075	0.9897*	2.405	0.908
	[0.2658]	[0.4061]	[0.6877]	[0.6907]	[0.6332]	[2.1035]	[0.6884]	[0.2210]	[0.3715]	[0.4390]	[0.6359]	[0.5842]	[2.1777]	[0.6401]
Observations	49	49	49	49	49	49	48	49	49	49	49	49	49	48
Adj R-squared	0.269	0.266	0.243	0.242	0.233	0.0847	0.229	0.327	0.321	0.276	0.271	0.272	0.175	0.265

Notes: Robust standard errors in brackets. Mid-shock cohort, born in 1921-28. Early-shock cohort, born in 1911-18. In columns 1-5/8-12 and 7/8 the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In column 6/12 the sample is further restricted to women born-in-state. In columns 1-6/8-13 the sample excludes Alaska and Hawaii. In column 7/14 DC is also excluded from the sample. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

change in fertility, a result that also holds for the Full-shock cohort.

The coefficient on the World War II mobilization rates is negative and highly significant. Acemoglu, Autor and Lyle (2004) argue that mobilization rates are a good proxy for the state level effects of the war on labor market conditions. Thus, these results suggests that the war had a negative effect on early fertility, and no effect on completed fertility, which is inconsistent with Doepke, Hazan and Mahoz (2007).

4.3 Results: Maternal Mortality and Education

In the model analyzed in Section 3, the response of women's human capital is driven by two opposing forces. The reduction in maternal mortality leads to a rise in women's incentive to invest in human capital, while an anticipated decline in the burden of maternal conditions reduces human capital investment, if fertility rises in response. Thus, the theory is consistent with a negative response of women's human capital investment if the response of fertility to the decline in the health burden of pregnancy is sufficiently strong.

We adopt college graduation rates as a proxy for human capital investment for the purpose of this analysis. As shown in Table 4, female college graduation rates rose from 5% for the Pre-shock cohort to 10% for the Full-shock cohort. Male college graduation rates expanded at a faster pace over the same period. As a result, the male-female differential in college graduation rates rose to 11% for the Mid-shock and 12% for the Full-shock cohorts, from the relatively low values of 5% for the Early-shock and 3% for the Pre-shock cohorts. These statistics suggest that progress in female human capital investment for this sample was slow, and in particular much slower than for men. Goldin, Katz and Kuziemko (2006) document a similar pattern for a broader sample. They attribute the emergence of a gender imbalance in college attendance with the 1910 birth cohort to lack of employment opportunities for men and marriage bars in female employment during the thirties. The GI Bills, which provided generous financial support for college education to male veterans of World War II and the Korean War, led to a peak in the gender imbalance in the forties and fifties. The gap began to close starting with the mid-1930s birth cohorts.

Despite the decline in women's human capital investment relative to males in the aggregate, there is negative correlation between the change in the male-female differential in college graduation and the drop in expected maternal mortality for the Full-shock vs. Early-shock comparison, displayed in figure 4, while there is no relation between the drop in maternal mortality and the male-female differential in college graduation for the earlier cohorts. This pattern is consistent with a positive effect of the drop in maternal mortality on the growth in women's human capital relative to males.

Estimating equation (5) for the male-female college graduation differential across cohorts mostly confirms these patterns. A negative sign of the estimated coefficient on the drop in maternal mortality is consistent with a positive effect of the improvement in maternal health on women's incentive to invest in human capital. This effect is present in the estimates both for the Mid-shock and Full-shock relative to the Early-shock cohort, as shown in Table 12.[50] For the Mid-shock cohort, given that expected maternal mortality drops on average by 33 deaths for 10,000 live births relative to the Early-shock cohort, the predicted change in the male-female differential in college graduation rates is -0.056. For the Full-shock cohort, given that expected maternal mortality drops on average by 52 deaths for 10,000 live births relative to the Early-shock cohort, the predicted change in the male-female differential in college graduation rates is -0.089. For both comparisons, despite the sizable effect of the drop in expected maternal mortality on the gender gap in college graduation rates, the estimated coefficient is only significant at the 10% level and its value drops to

[50] There is no relation between the drop in maternal mortality and the change in the male-female college graduation rates for the Early-shock vs. Pre-shock comparison. See Table 20 in Appendix C.

Table 11: Interim Fertility: Mid-shock Cohort

Concurrent MMR and Interim Fertility: Mid-Shock vs. Early-Shock Cohort

Robustness Checks and Alternative Hypothesis

	Dependent Variable is Level Change in Completed Fertility							
	1	2	3	5	6	7	8	9
Level Drop Concurrent MMR	0.0673	0.061	0.0598*	0.0613	0.0616*	0.0710*	0.0726*	0.0966**
	[0.0522]	[0.0459]	[0.0343]	[0.0366]	[0.0343]	[0.0350]	[0.0358]	[0.0440]
Level Drop Concurrent IMR	0.0005							
	[0.0134]							
Share Dwellings w Refrigerators			0.8944	0.1556			0.3795	
			[0.7765]	[0.8114]			[0.8459]	
Literacy		1.0943						-1.3709
		[1.1888]						[2.1712]
Date Suffrage Accepted		0						-0.0057
		[0.0001]						[0.0054]
Sheppard-Towner funds per capita		-1.2127**						-0.5142
		[0.4717]						[0.7856]
Social Security funds per capita		-0.0944						-0.5326
		[0.2132]						[0.6193]
WWII Mobilization Rates					-1.1493	-4.8842***	-4.9669***	-4.4383***
					[1.4894]	[1.6906]	[1.7605]	[1.5304]
% Nonwhite 1940						-1.1008*	-1.1428*	-0.8043
						[0.5715]	[0.5923]	[1.3477]
% Farmers 1940						-1.6114**	-1.4395*	-1.0315
						[0.7311]	[0.8071]	[0.7896]
Constant	0.1931	-0.8385	0.0274	0.0458	0.6676	3.4112***	3.2277**	15.2766
	[0.5625]	[1.4344]	[0.5842]	[0.5600]	[0.8380]	[1.1403]	[1.2387]	[10.4276]
Additional controls	{1}	{1}	{1}	{1,2}	{1}	{1}	{1,2}	{1,2}
Observations	49	49	49	49	47	47	47	47
Adj R-squared	0.157	0.273	0.187	0.201	0.166	0.349	0.333	0.405

Notes: Robust standard errors in brackets. Mid-shock cohort, born in 1921-28. Early-shock cohort, born in 1911-18. The dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. Controls: 1= 1950's expected MMR and per-capita personal income, percentage change in: per-capita personal income, share white, share farm, share foreign born, share working in health and in the public sector, 2= 1 + percentage change in unemployment rate, share high school graduate, male and female full-time real hourly wage. In all columns the sample excludes Alaska and Hawaii. In columns 6-9 the sample also excludes Nevada and DC because of missing information on mobilization rates. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

zero and looses significance when the full set of demographic and economic controls are included. Moreover, the estimated regression equations have very low explanatory power.

Even if these estimates are conservative,[51] these results suggest the link between the drop in maternal mortality and women's college graduation rates is tenuous for these cohorts. The next section examines the link between the maternal mortality drop, fertility and female education for later birth cohorts, to assess the long term consequences of the advances in maternal health.

4.4 Later Cohorts

The theoretical analysis allows for the possibility that further a decline in maternal mortality and the health burden of maternal conditions starting from an initially low level may reduce the demand for children, even as it continues to increase human capital investment.

For cohorts following the Full-shock, the decline in maternal mortality and its cross-state dispersion are small, so our empirical strategy is not ideal. With these limitation in mind, we conduct a similar analysis for the Post-shock cohort, corresponding to women 22-29 in 1970.

We first analyze the impact of the maternal mortality drop on the change in fertility for the Post-shock vs. Early-shock comparison. The results are reported in Table 13. The estimated coefficient on the drop in expected maternal mortality is positive and significant at the 5% level, however, this finding is not robust to the inclusion of additional controls.

The Post-shock cohort is particularly suited to gauge the impact of the advances in maternal health on educational attainment, since the male-female differential in college graduation rates starts to close for this cohort. Thus, we compare the male-female differential in college graduation rates for Post-shock cohort to the Early-shock cohort. The results of for the percentage specification are reported in Table 14. There is a significant effect of the drop in expected maternal mortality on the male-female differential in college graduation rates for this comparison. The coefficient for the baseline specification implies that a 10% drop in expected maternal mortality is associated with a 20% decline in the male-female differential in college graduation rates, which corresponds to a decline of 0.01 relative to the value of 0.05 for the Early-shock cohort. This finding is robust to the inclusion of the full set of demographic and economic controls. However, the estimated coefficient for the drop in maternal mortality is negative for the level specification, but never significant.

These findings provide weak evidence in support of a link between the maternal mortality drop and the rise in female education for the Post-shock vs. Early-shock comparison. The connection may be undermined by other factors that affecting fertility and human capital for the Post-shock cohort, such as access to legal abortion and the diffusion of oral contraception. Angrist and Evans (1999) analyze the impact of early access to legal abortion and conclude that it mainly affected teen pregnancy for black females. Goldin and Katz (2002) argue that the access to oral contraception for unmarried women delayed marriage and fertility and contributed to the entry in traditionally male careers for women born in 1945 and later years. Bailey (2002), however, shows that early access to the pill had no significant impact on completed fertility for these cohorts, though it exerted a strong impact on the timing of fertility, and that the delay in fertility was accounted for by change in education and occupational outcomes.

[51] As shown in Table 19 in Appendix C, the male-female differential in college graduation is negatively correlated with expected maternal mortality and, as shown in Table 19, drops by more in states where it is initially high. This pattern of correlation generates upward bias in the estimated coefficient for the effect of the maternal mortality, which should be negative based on the theory, further weakening the results.

Table 12: Male-Female College Graduation: Mid-shock and Full-shock Cohorts

Expected MMR and M-F College Graduation Differential: Mid-Shock and Full-Shock vs. Early-Shock Cohort

	Dependent Variable is Level Change in M-F College Graduation Differential							
	Mid-Shock vs. Early-Shock				Full-Shock vs. Early-Shock			
	1	2	3	4	5	6	7	8
Level Drop Expected MMR	-0.0017*	-0.0017*	-0.0018*	-0.0008	-0.0017*	-0.0017*	-0.0015*	-0.0002
	[0.0010]	[0.0010]	[0.0010]	[0.0012]	[0.0010]	[0.0010]	[0.0008]	[0.0009]
Per Capita Personal Income				0				-0.0000**
				[0.0000]				[0.0000]
Level Change:								
Per Capita Personal Income		0	0	0		0	0	0
		[0.0000]	[0.0000]	[0.0000]		[0.0000]	[0.0000]	[0.0000]
Share White				-0.1853				-0.5057***
				[0.2085]				[0.1718]
Share Farm				-0.1272				0.2319
				[0.2472]				[0.3181]
Share Health Sector				1.5843**				-0.8956
				[0.7272]				[0.9990]
Share Public Sector				0.0451				0.4432
				[0.4450]				[0.5166]
Share Textile				-0.0917				-0.2955
				[0.4108]				[0.3100]
Share Agriculture				0.0819				-0.2617
				[0.2527]				[0.2543]
Unemployment Rate				-0.2329				0.1207
				[0.4946]				[0.4128]
Female FT Hourly Wage				0.0209				0.0308
				[0.0233]				[0.0270]
Male FT Hourly Wage				0.022				0.015
				[0.0167]				[0.0174]
Share of 18-64 Population with College			0.2949				0.5762	
			[0.1949]				[0.3738]	
Constant	0.1019***	0.1171***	0.0999**	0.0544	0.0784***	0.0804*	0.0469	0.0869
	[0.0318]	[0.0394]	[0.0383]	[0.0890]	[0.0258]	[0.0408]	[0.0299]	[0.0624]
Observations	49	49	49	49	49	49	49	49
Adj R-squared	0.0479	0.0516	0.0591	0.0359	0.0379	0.0171	0.17	0.468

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Mid-shock cohort, born in 1921-28. Full-shock cohort, born in 1931-38. In all columns the dependent variable is computed for the sample of white married women, not living in institutional quarters and not living in farms. In all columns the sample excludes Alaska and Hawaii. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 13: Completed Fertility: Post-shock Cohort

Expected MMR and Completed Fertility: Post-Shock vs. Early-Shock Cohort

	Dependent Variable is Level Change in Completed Fertility						
	1	2	3	4	5	6	7
Level Drop Expected MMR	0.1620***	0.1224**	0.0707	0.0668	0.0625	0.122	0.0757
	[0.0582]	[0.0530]	[0.0619]	[0.0598]	[0.0659]	[0.1165]	[0.0651]
Expected MMR	-0.0571***	-0.0433**	-0.0279	-0.0267	-0.0245	-0.0492	-0.0294
	[0.0184]	[0.0171]	[0.0204]	[0.0197]	[0.0213]	[0.0385]	[0.0214]
Per Capita Personal Income		0.0000*	0	0	0	0	0
		[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0001]	[0.0000]
Level Change:							
Per Capita Personal Income	0	0	0	0	-0.0001	-0.0001	0
	[0.0000]	[0.0000]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]
Share White			1.5471**	1.5272**	2.0090**	1.3897	1.2451
			[0.6999]	[0.7171]	[0.8705]	[2.1806]	[0.9864]
Share Farm			1.2292	1.2001	0.9318	2.4360*	1.1586
			[0.7498]	[0.7872]	[0.7942]	[1.3770]	[0.8092]
Share Foreign Born			0.6778	0.691	0.5795	0.5919	0.6867
			[0.6347]	[0.6823]	[0.6764]	[1.4962]	[0.6540]
Share Health Sector			-0.2601	-0.1348	2.1559	10.905	-0.3951
			[5.9068]	[5.8547]	[5.0931]	[9.2037]	[5.9688]
Share Public Sector			-3.9101**	-4.0345*	-4.0702*	0.0547	-4.1017*
			[1.8413]	[2.3662]	[2.2304]	[5.9005]	[2.1385]
Unemployment Rate				-0.3515	-1.4353		
				[2.0368]	[2.0510]		
Share High School Graduates				0.104	-0.5344		
				[0.9430]	[0.9398]		
Female FT Hourly Wage					-0.0954		
					[0.0961]		
Male FT Hourly Wage					0.1731		
					[0.1218]		
Constant	0.2616*	0.1157	0.4319	0.4208	0.3165	0.84	0.4336
	[0.1489]	[0.1981]	[0.4365]	[0.4421]	[0.3627]	[0.5970]	[0.4353]
Observations	49	49	49	49	49	49	48
Adj R-squared	0.291	0.337	0.413	0.382	0.394	0.238	0.411

Notes: Robust standard errors in brackets. Post-shock cohort, born in 1941-48. Early-shock cohort, born in 1911-18. In columns 1-5 and 7 the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In column 6 the sample is further restricted to women born-in-state. In all columns the sample excludes Alaska and Hawaii. In column 7 DC is also excluded from the sample. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 14: Male-Female College Graduation: Post-shock Cohort

Expected MMR and M-F College Graduation Differential: Post-Shock vs. Early-Shock Cohort

	Dependent Variable is Percentage Change in M-F College Graduation Differential			
	1	2	3	4
% Drop Expected MMR	-2.0462*	-1.724	-1.7384	-2.7138**
	[1.0664]	[1.0959]	[1.1167]	[1.1515]
Per Capita Personal Income				0
				[0.0001]
% Change:				
Per Capita Personal Income		0.603	0.6598	-0.8096
		[0.3814]	[0.4787]	[1.5046]
Share White				3.1044
				[2.3362]
Share Farm				-0.4522
				[0.3981]
Share Health Sector				0.1098
				[0.4489]
Share Public Sector				-0.5143
				[0.3441]
Share Textile				-0.0709
				[0.1623]
Share Agriculture				0.362
				[0.4806]
Unemployment Rate				0.1562
				[0.2326]
Female FT Hourly Wage				1.097
				[1.0992]
Male FT Hourly Wage				-2.4706
				[2.2360]
Share of 18-64 Population with College			0.3544	
			[1.4326]	
Constant	1.3083*	0.9394	0.9113	2.4337**
	[0.6880]	[0.7433]	[0.7470]	[0.8995]
Observations	49	49	49	49
Adj R-squared	0.055	0.0688	0.049	0.0508

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Full-shock cohort, born in 1931-38. Post-shock cohort, born in 1941-48. In all columns the dependent variable is computed for the sample of white married women, not living in institutional quarters and not living in farms. In columns 1-4 the sample excludes Alaska and Hawaii. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

4.5 Infant Mortality

We adopt the same estimation strategy to explore the relation between the drop in infant mortality and the change in fertility across cohorts. This approach is less satisfactory for infant mortality, however. While the cross-state variation in infant mortality is similar in magnitude to maternal mortality (see Table 15), the decline in infant mortality is more gradual and starts prior to 1930, as can be seen in figure 3. Since all cohorts are partially exposed, we will assume the Pre-shock cohort is untreated, and compare the Early-shock, Mid-shock and Full-shock cohorts to the Pre-shock cohort.

The gradual decline in infant mortality and its virtual constancy between 1940 and 1960, a period of very rapid advances in neonatal care, may be a symptom of a reverse causal effect from high fertility to high infant mortality due to maternal depletion, as discussed in Section 2. Supportive evidence for this channel can be found for both advanced economies and developing countries (Preston and Haines, 1991, and Haines, 1997). Since completed fertility cannot influence expected infant mortality, we can isolate the direct causal link between infant mortality and fertility. Moreover, comparing the estimates for expected and concurrent infant mortality may be useful to gauge the strength of the reverse causation channel.

There is also a strong positive correlation between infant mortality and fertility for all cohorts, shown in Table 19. Since infant mortality drops by more in states with initially high infant mortality, while fertility rises by less in states with initially high fertility, this correlation may generate a negative bias on the estimated coefficient on the drop in infant mortality in equation (5). The bias generates a tendency to find a negative relation between the drop in infant mortality and the change in fertility.

Table 23 in Appendix C examines the economic and demographic predictors of infant mortality for each cohort, discussed in Section 4.1.2. Despite the positive correlation between infant and maternal mortality, infant mortality is highly less predictable based on the demographic, economic, cultural and government controls we include in the analysis. Among the demographic indicators, only the share of high school graduates is significantly negatively related with both expected and concurrent infant mortality for all cohorts. We will still include these variables as controls in the estimation.

Table 15: Infant Mortality: Cross-state variation

IMR* (per 1,000 live births)							
	Level			Change			
	Mean	Min, Max	*Coeff. of Variation*		Mean %	Min, Max	| *Coeff. of Variation* |
1930	67.49	48.7, 145.5	0.25				
1940	48.44	33.2, 109.1	0.29	1940-1930	-28.7	-36.4, -4.5	0.33
1950	30.27	21.8, 65.1	0.24	1950-1940	-.36.5	-44, -7.9	0.44
1960	26.55	19.6, 41.6	0.17	1960-1950	-10.7	-32.2, 6	1.74
1970	20.21	14.3, 29.1	0.15	1970-1960	-23.3	-14.2, -0.5	0.47

* State averages. Sources in Appendix A

4.5.1 Estimation Results

Tables 16-18 present the estimation results for both expected and concurrent infant mortality. We only find a strong negative relation between the drop in both expected and concurrent infant mortality and the change in fertility for the Early-shock vs Pre-shock cohort comparison, though the estimates for expected infant mortality are not robust to the inclusion of demographic and economic controls.

For the Early-shock vs. Pre-shock cohort comparison, a 10 drop in expected infant mortality is associated

Table 16: Infant Mortality and Fertility: Early-shock Cohort

IMR and Completed Fertility: Early-Shock vs. Pre-Shock Cohort

	Dependent Variable is Level Change in Completed Fertility					
	Expected IMR			*Concurrent IMR*		
	1	2	3	4	5	6
Level Drop IMR	-0.0245**	-0.0224*	-0.0220**	-0.0467***	-0.0505**	-0.0462**
	[0.0117]	[0.0126]	[0.0103]	[0.0162]	[0.0210]	[0.0176]
IMR Pre-Shock Cohort	0.0045	0.0037	0.0029	0.0101**	0.0116*	0.0101*
	[0.0045]	[0.0042]	[0.0037]	[0.0049]	[0.0059]	[0.0053]
Per Capita Personal Income	0.0001***	0.0001**	0.0001	0.0001***	0.0001***	0.0001*
	[0.0000]	[0.0001]	[0.0001]	[0.0000]	[0.0000]	[0.0001]
Level Change:						
Per Capita Personal Income	0.0001	0	0	0.0001	0	0
	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]
Share White		-4.9484	-5.4358		-6.0975**	-6.5041*
		[2.9810]	[3.7313]		[2.8498]	[3.6013]
Share Farm		-4.1512*	-2.8201		-2.2592	-1.263
		[2.0657]	[2.3662]		[1.7889]	[2.1506]
Share Foreign Born		0.9885	0.1178		0.5047	0.0649
		[3.4356]	[2.6611]		[3.2957]	[2.6020]
Share Health Sector		-12.1405	-6.4414		-9.8181	-4.4981
		[12.5309]	[13.5065]		[10.5773]	[10.9410]
Share Public Sector		-2.4001	-1.1601		-3.8016*	-2.9144
		[2.1630]	[2.7195]		[1.9672]	[2.2935]
Unemployment Rate			-0.4258			0.3632
			[3.5056]			[3.4072]
Share High School Graduates			-0.7482			-1.1205
			[2.8195]			[2.6689]
Female FT Hourly Wage			-0.1307			-0.0806
			[0.2496]			[0.1984]
Male FT Hourly Wage			0.4073*			0.2673
			[0.2291]			[0.2167]
Constant	-0.7554	-0.8915*	-0.8169	-0.7833**	-0.6160*	-0.5246
	[0.4507]	[0.5099]	[0.5040]	[0.3708]	[0.3648]	[0.3496]
Observations	47	47	47	49	49	49
Adj R-squared	0.207	0.244	0.213	0.262	0.297	0.256

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Pre-shock cohort, born in 1901-08. In all columns the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In all columns the sample excludes Alaska and Hawaii. In columns 1 to 3 the sample also excludes South Dakota and Texas since expected MMR cannot be computed for the pre-shock cohort. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

with a 0.22-0.25 unit decline in completed fertility, as shown in Table16. The actual decline in fertility for this cohort comparison was 0.13. The estimated coefficients are greater in absolute value for concurrent infant mortality. A 10 unit drop in concurrent mortality is associated with a 0.47-0.51 unit decline in completed fertility. The estimates are robust to the inclusion of economic and demographic controls. However, the estimates for expected infant mortality loose significance when cultural and government controls are included, as can be seen in Table 17. This is not the case for concurrent infant mortality. This suggests that the direct causal relation between infant mortality and fertility is weaker than the reverse relation for this cohort comparison.

This finding is potentially important since it may contribute an explanation to the sharp reduction in fertility between 1900 and 1935. The low fertility rates of the 1920s and early 1930s are often considered below trend, relative to the secular path of fertility decline that started in the U.S. around 1940.[52] The strong contribution of the drop in infant mortality to the change in fertility for the Early-shock cohorts may well apply to earlier cohorts, since the decline in infant mortality was very steep in this period.

The estimates for Mid-shock and Full-shock vs. Pre-shock comparisons suggest that the link between the drop in infant mortality and the change in fertility was tenuous for these cohorts. As can be seen in Table 17, the estimated coefficient on the change in expected infant mortality is negative but never significant. The coefficient for the change in concurrent infant mortality is sizable but not robust to the variation in the set of controls.

These results suggest that reverse causation from fertility to infant mortality may be present and important for the Early-shock vs Pre-shock comparison. This implies that most existing studies of the same time period may be subject to endogeneity bias and overestimate the relationship between infant mortality and fertility.[53] The fact that this effect disappears for younger cohorts may reflect a weakening of the "maternal depletion" channel that drives the reverse causal relation. Improved maternal health, together with the rapid diffusion of safe infant formula, first introduced in the late 1920s,[54] and the high quality of water and milk supplies in most areas, may also have limited the adverse consequences on infant health of early weaning in the post-war period.

5 Concluding Remarks

The empirical findings suggest that medical progress, through its effect on infant and maternal mortality, can explain the sharp decline in fertility up until the mid 1930s and the baby boom that took place between the late 1930s and the early 1960s. The positive effect of the drop in maternal mortality on fertility is consistent with a simple theory of fertility choice. The determinants of the "baby bust" that occurred during the second half of the 1970s remain an open question. Our theoretical framework suggests that further reductions in the burden of maternal conditions starting from low values of maternal mortality may lead to a reduction in fertility and a rise in human capital. However, these effects are hard to identify with our empirical approach for later cohorts. More likely, the rise in the returns to human capital, both directly, via the rise in the skill premium, and indirectly, though the improved ability to control fertility with the diffusion of oral contraception, played a large role in the rise in women's educational attainment and the reduction in fertility during the late 1960s and 1970s. This response is consistent our model.

The link between maternal mortality reduction and the baby boom in the U.S. potentially opens an

[52] See Jones and Tertilt (2007) for a discussion.
[53] See Preston and Haines (1991) for a review.
[54] Albanesi and Olivetti (2009) provide an extensive discussion of the diffusion of infant formula in the U.S.

Table 17: Infant Mortality and Fertility: Early-shock Cohort, Robustness

IMR and Completed Fertility: Early-Shock vs. Pre-Shock Cohort
Robustness Checks and Alternative Hypothesis

	Expected Infant Mortality						*Concurrent Infant Mortality*					
	Dependent Variable is Level Change in Completed Fertility											
	1	2	3	4	5	6	1	2	3	4	5	6
Level Drop Concurrent IMR	-0.0173	-0.0141	-0.0222*	-0.0113	-0.0131	-0.0111	-0.0462**	-0.0412**	-0.0504**	-0.0491**	-0.0459***	-0.0417***
	[0.0133]	[0.0128]	[0.0130]	[0.0094]	[0.0097]	[0.0098]	[0.0173]	[0.0154]	[0.0216]	[0.0189]	[0.0166]	[0.0145]
Share Dwellings w Refrigerators	-0.9236	-1.4744					-1.2169*	-1.5494*				
	[1.0117]	[1.2800]					[0.7173]	[0.9103]				
Level Drop Concurrent MMR			-0.0009						-0.0016			
			[0.0094]						[0.0093]			
Literacy				-1.4345						-1.9687		
				[2.1535]						[1.8315]		
Date Suffrage Accepted				0.0003**						0.0004***		
				[0.0001]						[0.0001]		
Sheppard-Towner funds per capita				-1.3545*						-0.8353		
				[0.6842]						[0.7094]		
Social Security funds per capita				0.5090*						0.3283		
				[0.2708]						[0.2425]		
WWII Mobilization Rates					1.6365	0.9125					-0.0338	-0.4505
					[2.7390]	[1.6264]					[2.1348]	[1.1136]
% Nonwhite 1940					0.9071						0.9008	
					[0.8999]						[0.6799]	
% Farmers 1940					-0.6509						-0.6058	
					[0.7506]						[0.5553]	
Constant	-0.5523	-0.4155	-0.8959*	0.3225	-1.2206	-0.9538	-0.3232	-0.3088	-0.6052	0.6859	-0.3124	-0.2157
	[0.6307]	[0.6141]	[0.5149]	[2.3493]	[1.7049]	[1.1081]	[0.3744]	[0.3738]	[0.3835]	[1.8446]	[1.3351]	[0.6864]
Additional controls	{1}	{1,2}	{1}	{1}	{1}	{1}	{1}	{1,2}	{1}	{1}	{1}	{1}
Observations	47	47	47	47	45	45	49	49	49	49	47	47
Adj R-squared	0.244	0.227	0.223	0.279	0.109	0.0908	0.327	0.295	0.28	0.377	0.215	0.191

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Pre-shock cohort, born in 1901-08. The dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. Controls: 1= 1950's expected MMR and per-capita personal income, percentage change in: per-capita personal income, share white, share farm, share foreign born, share working in health and in the public sector, 2= 1 + percentage change in unemployment rate, share high school graduate, male and female full-time real hourly wage. In all columns the sample excludes Alaska and Hawaii. In columns 5-6 the sample also excludes Nevada and DC because of missing information on mobilization rates. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 18: Infant Mortality and Fertility: Mid-shock and Full-shock Cohorts

IMR and Completed Fertility

	Mid-Shock vs. Pre-Shock Cohort						Full-Shock vs. Pre-Shock Cohort					
	Dependent Variable is Level Change in Completed Fertility						*Dependent Variable is Level Change in Completed Fertility*					
	Expected IMR			*Concurrent IMR*			*Expected IMR*			*Concurrent IMR*		
	1	2	3	4	5	6	1	2	3	4	5	6
Level Drop IMR	-0.0144*	-0.0065	-0.0099	-0.0327**	-0.0191	-0.0234	-0.0268	-0.0363	-0.0393*	-0.0387	-0.0426	-0.0737**
	[0.0073]	[0.0079]	[0.0106]	[0.0122]	[0.0138]	[0.0159]	[0.0200]	[0.0238]	[0.0210]	[0.0279]	[0.0302]	[0.0307]
IMR Pre-Shock Cohort	-0.0005	-0.002	-0.0017	0.0064	0.0021	0.003	0.0052	0.0079	0.0082	0.0092	0.0107	0.0189**
	[0.0017]	[0.0019]	[0.0024]	[0.0039]	[0.0045]	[0.0051]	[0.0054]	[0.0063]	[0.0055]	[0.0082]	[0.0089]	[0.0088]
Capita	0.0001***	0.0001***	0.0001**	0.0001***	0.0001***	0.0001**	0.0001***	0	0.0000*	0.0000***	0	0
	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]
Level Change:												
Per Capita Personal Income	0.0003***	0.0003***	0.0002**	0.0002***	0.0003***	0.0002**	0.0002**	0.0002**	0.0001	0.0002**	0.0002**	0.0001
	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0001]
Share White		-1.1628	-1.2732		-0.9454	-1.0157		0.0357	-1.224		0.0851	-0.9667
		[0.8302]	[1.4792]		[0.7626]	[1.3770]		[0.9123]	[0.8434]		[0.9795]	[0.9325]
Share Farm		-1.3578*	-1.5227		-1.3642*	-1.4708		0.4652	0.0159		0.3287	-0.1299
		[0.7231]	[0.9345]		[0.7049]	[0.8720]		[1.0401]	[0.9528]		[1.0465]	[0.8878]
Share Foreign Born		0.5635	0.465		0.6507	0.5827		-1.1987	-0.7156		-0.5898	-0.0095
		[1.5921]	[2.0222]		[1.5214]	[1.8367]		[1.6093]	[1.2190]		[1.5714]	[1.2419]
Share Health Sector		-1.7139	-1.5614		-2.3504	-2.4437		-3.5275	-9.3181		-4.3365	-9.433
		[9.2125]	[9.0990]		[8.6349]	[8.7999]		[7.4518]	[5.7806]		[7.6124]	[5.9802]
Share Public Sector		-1.9439	-2.4417		-1.7164	-2.152		-2.8540*	-2.2497		-3.1807**	-2.3549
		[1.8113]	[2.4510]		[1.8980]	[2.5368]		[1.5933]	[1.5833]		[1.5112]	[1.4405]
Unemployment Rate			-1.5232			-1.3476			-0.6354			-1.8277
			[1.9987]			[1.8154]			[2.4994]			[2.1723]
Share High School Graduates			1.0697			1.0329			3.3552**			3.7117**
			[1.8743]			[1.7072]			[1.3126]			[1.4686]
Female FT Hourly Wage			-0.0134			0.0159			-0.2612			-0.3271*
			[0.3643]			[0.3410]			[0.2206]			[0.1822]
Male FT Hourly Wage			0.0394			0.0284			0.083			0.14
			[0.1975]			[0.1797]			[0.1393]			[0.1089]
Constant	-0.3704*	-0.5596**	-0.6092**	-0.4131*	-0.5924**	-0.6338**	-0.4125*	-0.2735	-0.3073	-0.4872*	-0.3477	-0.5732
	[0.2100]	[0.2694]	[0.2945]	[0.2156]	[0.2696]	[0.2936]	[0.2433]	[0.3490]	[0.3465]	[0.2610]	[0.3664]	[0.3640]
Observations	47	47	47	49	49	49	47	47	47	49	49	49
Adj R-squared	0.462	0.444	0.396	0.476	0.462	0.419	0.456	0.454	0.524	0.454	0.436	0.537

Notes: Robust standard errors in brackets. Mid-shock cohort, born in 1921-28. Pre-shock cohort, born in 1901-08. In all columns the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In all columns the sample excludes Alaska and Hawaii. In columns 1 to 3 the sample also excludes South Dakota and Texas since expected MMR cannot be computed for the pre-shock cohort. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

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 advanced economies in the 1930s, as document 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 and Olivetti (in progress) explore whether the cross-country variation in the path of maternal mortality can account for the international variation in fertility patterns.

References

[1] Acemoglu, Daron, David Autor and David Lyle. 2004. Women, War and Wages: The Effect of Female Labor Supply on the Wage Structure at Mid century. *Journal of Political Economy* 112(3): 497-551.

[2] Albanesi, Stefania and Claudia Olivetti. 2009. Gender Roles and Medical Progress. NBER WP 14873.

[3] Albanesi, Stefania and Claudia Olivetti. In progress. Maternal Mortality Reduction and Cross-Country Fertility Differentials in the Twentieth Century. Manuscript, Columbia University.

[4] Altshuler, Glenn C. and Sturat M. Blumin. 2009. The G.I. Bill: A New Deal for Veterans. Oxford University Press.

[5] Angrist, Joshua D., and William N. Evans. 1999. Schooling and Labor Market Consequences of the 1970 State Abortion Reforms. *Research in Labor Economics XVIII: 75–113.*

[6] Bailey, Martha More Power to the Pill. The Quarterly Journal of Economics.

[7] Baker, S. Josephine. 1923. Why do our mothers and babies die? Ladies' Home Journal 40: 212-213.

[8] Becker, Gary S. and Robert J. Barro. 1988. A Reformulation of the Economic Theory of Fertility. *The Quarterly Journal of Economics* 103(1): 1-25.

[9] Berry, Linda G. 1977. Age and Parity Influences on Maternal Mortality: United States, 1919-1969. Demography 14(3): 297-310.

[10] Bleakley, Hoyt. 2007. Disease and Development: Evidence from Hookworm Eradication in the American South. *The Quarterly Journal of Economics* 122: 73-117.

[11] Bleakley, Hoyt, and Fabian Lange. 2008. Chronic Disease Burden and the Interaction of Education, Fertility and Growth. *Review of Economics and Statistics.*

[12] Bromley, Dorothy D. 1929. What Risk Motherhood? Harper's 159: 20.

[13] Center for Disease Control. 1999. Infant and Maternal Mortality in the United States: 1900-99. Population and Development Review 125(4): 821-826.

[14] Children's Bureau. 1931. The Promotion of the Welfare and Hygiene of Maternity and Infancy. Children's Bureau Publication 203, US Dept. of Labor, Washington DC.

[15] Cornwall, Marie, State-level Suffrage Legislation Database (Provo, Utah: Brigham Young University, 2003).

[16] Cornwall, Marie, Eric Dahlin, Brayden King, and Kendra Schiffman, "Moving Mountains: An Institutionalist Analysis of State-Level Woman Suffrage Legislative Success," Unpublished manuscript presented and distributed at the Social Science History Association annual meeting in Chicago, IL, 2004.

[17] Cutler, David, Angus deaton, and Adriana Lleras-Muney. 2006. The Determinants of Mortality. *The Journal of Economic Perspectives 20(3): 97-120.*

[18] Dannreuther, Walter T. 1931. The American Board of Obstetrics and Gynecology: Its Organization, Function and Objectives. *Journal of the American Medical Association 96: 797-798.*

[19] Doepke, Matthias. 2005. Child mortality and the fertility decline: Does the Barro-Becker model fit the facts? *Journal of Population Economics* 18: 337-366.

[20] Doepke, Matthias, Moshe Hazan and Yishay Maoz. 2007. The Baby Boom and World War II: A Macroeconomic Analysis. Manuscript, Northwestern University.

[21] Dublin, Mary. 1936. Maternal Mortality and the Decline in the Birth Rate. Annals of the American Academy of Political and Social Science 188 (The American People: Studies in Population): 107-116.

[22] Fernandez, Raquel. 2009. Manuscript, New York University.

[23] Franks AL, Kendrick, KS, Olson, DR, Atrash HK, Saftlas, AF and Moien M. 1992 Hospitalization for pregnancy complications, United States, 1986 and 1987. *Am J Obstet Gyn. 166(5):1339-44.*

[24] Geddes, Rick and Dean Lueck. 2002. The Gains from Self-Ownership and the Expansion of Women's Rights. *The American Economic Review 92(4): 1079-1092.*

[25] Goldin, Claudia. 1991. The Role of World War II in the Rise of Women's Employment. *The American Economic Review 81(4): 741-756.*

[26] Goldin, Claudia, Lawrence F. Katz. 2002. The Power of the Pill: Oral Contraceptives and Women's Career and Marriage Decisions. *The Journal of Political Economy 110: 730 –770.*

[27] Goldin, Claudia, Lawrence F. Katz, and Ilyana Kuziemko. 2006. The Homecoming of American Women: The Reversal of the College Gender Gap. *Journal of Economic Perpectives 20(4): 133-156.*

[28] Greenwood, Jeremy, Ananth Seshadri, and Guillerme Vanderbroucke. 2005. The Baby Boom and the Baby Bust. *American Economic Review 95(1): 183-207.*

[29] Haines, Michael R. 1997. The Relationship Between Infant and Child Mortality and Fertility: Some Historical and Contemporary Evidence for the United States. In From Death to Birth: Mortality Decline and Reproductive Change, Mark R. Montgomery and Barney Cohen, editors. National Academy Press.

[30] [32] Haines, Michael R. 2006a. Fetal death ratio, neonatal mortality rate, infant mortality rate, and maternal mortality rate, by race: 1850-1998. Table Ab912-927 in Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition, edited by Susan B. Carter, Scott Sigmund Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch, and Gavin Wright. New York: Cambridge University Press.

[31] Haines, Michael R. 2006b. “ Live births, deaths, infant deaths, marriages, and divorces, by race: 1909—1998 .” Table Ab11-30 in Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition, edited by Susan B. Carter, Scott Sigmund Gart- ner, Michael R. Haines, Alan L. Olmstead, Richard Sutch, and Gavin Wright. New York: Cambridge University Press.

[32] Haines, Michael R., and Richard Sutch. 2006. Population, by marital status, sex, and race: 1880—1990. Table Aa614-683 in Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition, edited by Susan B. Carter, Scott Sigmund Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch, and Gavin Wright. New York: Cambridge University Press.

[33] Hauser, Robert L. 1976. Fertility Tables for Birth Cohorts by Color: United States 1901-1973. Rockville, MD: National Center for Health Statistics.

[34] Preston, Samuel H. and Michael R. Haines. 1991. Fatal Years: Child Mortality in Late Nineteenth-Century America. *Princeton, NJ: Princeton University Press*.

[35] Jayachandran, Seema and Adriana Lleras-Muney. 2009. Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines. *The Quarterly Journal of Economics 124 (1): 349-397.*

[36] Jayachandran, Seema, Adriana Lleras-Muney and Kimberly V. Smith. 2009. Modern Medicine and the 20th-Century Decline in Mortality: Evidence on the Impact of Sulfa Drugs. *American Economic Journal: Applied Economics*, forthcoming.

[37] Jones, Larry E. and Alice Schoonbroodt. 2007. COMPLEMENTS VERSUS SUBSTITUTES AND TRENDS IN FERTILITY CHOICE IN DYNASTIC MODELS. NBER WP 13680.

[38] Jones, Larry E. and Michele Tertilt. 2007. An Economic History of Fertility in the U.S.: 1826-1960. *Forthcoming in "Frontiers of Family Economics," edited by Peter Rupert, Elsevier.*

[39] Kerr, J.M. Munro. 1933. Maternal Mortality and Morbidity. Edinburgh.

[40] Lesser, Arthur J. 1985. The Origin and Development of Maternal and Child Health Programs in the United States. American Journal of Public Health 75(6): 590-598.

[41] Litoff, Judy Barrett. 1986. The American Midwife Debate. NewYork: Greenwood Press.

[42] Lott, John R. Jr, and Lawrence W. Kenney. 1999. Did Women’s Suffrage Change the Size and Scope of Government? *The Journal of Political Economy 107(6): 1163-1198.*

[43] Meckel, Richard A. 1990. Save the Babies: American Public Health Reform and the Prevention of Infant Mortality 1850-1929.

[44] Meigs, Grace H. 1917. Maternal Mortality from all conditions connected with childbirth in the United States and certain other countries. Children’s Bureau Publication No. 19, Miscellaneous Series 6. US Department of Labor, Washington DC.

[45] Miller, Grant. 2008. WOMEN’S SUFFRAGE, POLITICAL RESPONSIVENESS, AND CHILD SURVIVAL IN AMERICAN HISTORY. *Quarterly Journal of Economics 123(3): 1287-1327.*

[46] Moehling, Carolyn and Melissa Thomasson. 2009. The Political Economy of Saving Mothers and Babies: The Politics of State Participation in the Sheppard-Towner Program. Manuscript. Rutgers University.

[47] Mount, Steve. "Ratification of Constitutional Amendments". http://www.usconstitution.net/constamrat.html. Retrieved February 24 2007.

[48] Parker, JK, and EM Carpenter. 1981. Julia Lathtrop and the Children's Bureau: The emergence of an institution. Social Science Review 55: 60-77.

[49] Preston, Samuel H. and Michael Haines. 1991. Fatal Years- Child Mortality in Late Nineteenth Century America. Princeton University Press.

[50] Rethereford, Robert D. 1972. Tobacco Smoking and the Sex Mortality Differential. Demography 9(2): 203-216.

[51] Rosen, G. 1958. A history of public health. New York: MD Publications.

[52] Schmidt, W M. 1973. The development of health services for mothers and children in the United States. American Journal of Public Health 63: 419 - 427.

[53] Sinai, Nathan and Odin W. Anderson. 1947. The Emergency Maternal and Infant Care Program 1943-1946. Bureau of Public Health Economics, School of Public Health. University of Michigan, Ann Arbor.

[54] Soares Rodrigo. 2005. Mortality Reductions, Educational Attainment, and Fertility Choice. *The American Economic Review 95(3): 580-601.*

[55] Starr, Paul. 1982. The Social Transformation of American Medicine. Basic Books.

[56] Stolnitz, George J.. 1956. A Century of International Mortality Trends. Population Studies 10(1): 17-42

[57] Thomasson, Melissa A. and Treber, Janet. 2008. From home to hospital: The evolution of childbirth in the United States, 1928-1940. Explorations in Economic History 45(1): 76-99.

[58] Weil, David N. 2007. Accounting for the Effects of Health on Economic Growth. The Quar- terly Journal of Economics 122(3): 1265-1306.

[59] Wertz, Richard W. and Dorothy C. Wertz. 1977. Lying-In. A History of Childbirth in America. Expanded Edition. Yale University Press. New Haven and London.

[60] White House Conference on Child Health and Protection. Fetal, Newborn and Maternal Morbidity and Mortality. New York and London: D. Appleton-Century Company, 1933.

[61] Wilcox LS, Marks JS. 1994. From Data to Action: CDC's Public Health Surveillance for Women, Infants, and Children. CDC maternal and child health monograph. Atlanta, GA: Centers for Disease Control and Prevention. 10.

[62] Wolpin, Kenneth. 1993. Determinants and consequences of the mortality and health of infants and children. *Handbook of Population and Family Economics, M. R. Rosenzweig & Stark, O. (ed.). Edition 1, volume 1, chapter 10, pages 483-557. Elsevier.*

[63] Woodbury, Robert Morse. 1925. Causal Factors in Infant Mortality: A Statistical Study Based on Investigations in Eight Cities. U.S. Children's Bureau Publication No. 142. Washington: Government Printing Office.

A Data Sources and Variable Definitions

A.1 Economic and Demographic 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 main sample includes white married women with children belonging to the following two age groups: 22-29, 32-39. 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).[55] We use the following variables:

Children ever born: We use IPUMS variable CHBORN which reports the 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. This information is not available in the1930 Census for which we only have information on the number of children below 13 living in the household (IPUMS variable NCHILD). Hence for 1930 we estimate children ever born as follows. We compute the difference between children ever born and number of children in the household by state using 1940 to 1960 data and take its average across the three decades. We then impute children ever born for 1930 by adjusting the (state-level) number of children using the average differential thus obtained.

Age at First Birth: We define age at first birth as the difference between the mother's age and the age of the eldest child (IPUMS variable ELDCH). This information is only available if the child is younger than 13. This statistics is computed on the 22 to 29 year old sample.

Age at Last Birth: We define age at last birth as the difference between the mother's age and the age of the youngest child (IPUMS variable YNGCH). This statistics is computed on the 32 to 39 year old sample.

Education: 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.)

Industry: We use IPUMS variable IND1950 which recodes information about industry into the 1950 Census Bureau industrial classification system and thus enhances comparability of industry data across all decades.

Hourly Earnings by Gender: We use the information on annual wage and salary income (INCWAGE) from the 1940 to 2000 IPUMS Census 1% samples (for 1970, we use the 1% State sample). For all years N/A code (999999) is treated as missing data. We divide this measure by annual hours in order to obtain hourly wage rates. Annual hours are obtained by multiplying the number of weeks worked in the previous year (WKSWORK1, WKSWORK2) by the number of hours worked in the past week (HRSWORK1, HRSWORK2). In 1960 and 1970, Census information on weeks and hours worked is reported in intervals (1-13 weeks, 14-26 weeks, 27-39 weeks, 40-47 weeks, 48-49 weeks, 50-52 weeks, and 1-14 hours, 15-29 hours, 30-34 hours, 35-39 hours, 40 hours, 41-48 hours, 49-59 hours and 60+ hours, respectively). For these decades we compute our measures of weeks and hours worked by assigning the midpoint of each interval. For 1940, 1950 and 1980-2000 we use the information on actual number of weeks worked that is available in the Census.[56]

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

[56] In the 1940 Census respondents were required to report this information in terms of "equivalent full-time weeks." It was up to respondents to determine precisely what "full-time" meant, though enumerators were instructed to suggest that 40 hours

Statistics are obtained as weighted averages using sample-line weights (SLWT) for 1940 and 1950 and person weights (PERWT) for the remaining decades.

Unemployment Rate: We use IPUMS variable EMPSTAT to compute state level unemployment rates.

Share of foreign 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.

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

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.

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 -

was a good round figure. In essence, respondents were to estimate how many hours they had averaged per week, multiply this figure by 52 weeks, then divide by 40 (See Census code book).

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 Mobilization Rates

Our mobilization rate variable is the same used in Acemoglu, Autor and Lyle (2004). They use published tables from the Selective Service System (1956) and construct men's mobilization rates during WWII as the fraction of the 18 to 44 years old registered males in a state who were drafted for war.[57] 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 state differences in war mobilization reflect a variety of factors. 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, and, not surprisingly, states with a higher proportion of men who are farmers have a lower mobilization rate. The mobilization rate is also higher in states with higher average male education and with a lower percentage of black males.

To attempt to control for systematic variation in the mobilization rate, our regressions include the 1940 fraction of non-white men aged 13 to 44, the 1940 fraction of men between the ages of 13 to 44 who are not farmers and the 1940 average education of men in this same age group.[58] As shown in Acemoglu et al., after controlling for these factors and for other non-economic components (such as the age composition and the number of German-born men) there is still some thirty per cent variation of mobilization rates across states that is left unexplained and which is attributed to idiosyncratic strategies followed by local registration boards.[59]

A.4 Home Appliances

Haines, Michael R., and the Inter-university Consortium for Political and Social Research. 2004. Historical, Demographic, Economic, and Social Data: The United States, 1790-2000 [Computer file]. ICPSR02896-v2. Hamilton, NY: Colgate University/Ann Arbor: MI: Inter-university Consortium for Political and Social Research [producers]. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor].

57 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).

58 Men who are 13 in 1940 would be 18 in 1945 and therefore part of the draft target group.

59 See Table 4 in Acemoglu, Autor, and Lyle [2004].

A.5 Federal Programs for the Promotion of Maternal and Infant Health

1921-1929 Maternity and Infancy Care (Sheppard-Towner) Act 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 also reserved for the activities of the Children's Bureau, who was responsible for the review and approval of the state plans.

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, of the Social Security Act provided funding for medical care of mothers and infants. 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 were 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.

Social Security Act 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).

A.6 Women's Suffrage

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).

B Proofs

Proof of Proposition 1

Totally differentiating the first order necessary conditions with respect to the parameter υ obtains:

$$-\upsilon u'((1+\varepsilon e)w)\varepsilon w\frac{\partial b}{\partial \upsilon}+\left[(1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2-v''(e)\right]\frac{\partial e}{\partial \upsilon}=bu'((1+\varepsilon e)w)\varepsilon w, \tag{6}$$

$$\begin{aligned}&-\upsilon u'((1+\varepsilon e)w)\varepsilon w\tfrac{\partial e}{\partial \upsilon}+\left\{-2\upsilon[-h'(\varphi b)\varphi+\kappa'(sb)s]+(1-\upsilon b)[-h''(\varphi b)\varphi^2+\kappa''(sb)s^2]\right\}\tfrac{\partial b}{\partial \upsilon}\\ =\;& b[-h'(\varphi b)\varphi+\kappa'(sb)s]+[u((1+\varepsilon e)w)-h(\varphi b)+\kappa(sb)]\,.\end{aligned} \tag{7}$$

To obtain the effect of an unanticipated change in υ, we set $\frac{\partial e}{\partial \upsilon}=0$ and solve for $\frac{\partial b}{\partial \upsilon}$ in equation (7). This obtains:

$$\frac{\partial b}{\partial \upsilon}=\frac{b[-h'(\varphi b)\varphi+\kappa'(sb)s]+[u((1+\varepsilon e)w)-h(\varphi b)+\kappa(sb)]}{-2\upsilon[-h'(\varphi b)\varphi+\kappa'(sb)s]+(1-\upsilon b)[-h''(\varphi b)\varphi^2+\kappa''(sb)s^2]}. \tag{8}$$

The denominator of this expression is negative, and the numerator is positive by Assuptions 1 and 2. This implies the result.

The response to an anticipated reduction in υ is derived by solving the system comprised by equations

(6) and (7). We represent the system as: $A_\upsilon \begin{bmatrix} \frac{\partial b}{\partial \upsilon} \\ \frac{\partial e}{\partial \upsilon} \end{bmatrix} = y_\upsilon$, where:

$$A_\upsilon = \begin{bmatrix} -\upsilon u'((1+\varepsilon e)w)\varepsilon w & [(1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2 - v''(e)] \\ \{-2\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] + (1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\} & -\upsilon u'((1+\varepsilon e)w)\varepsilon w \end{bmatrix}$$

$$y_\upsilon = \begin{bmatrix} bu'((1+\varepsilon e)w)\varepsilon w \\ b[-h'(\varphi b)\varphi + \kappa'(sb)s] + [u((1+\varepsilon e)w) - h(\varphi b) + \kappa(sb)] \end{bmatrix}$$

Thus:

$$\begin{aligned} |A_\upsilon| &= [\upsilon u'((1+\varepsilon e)w)\varepsilon w]^2 - \\ &\quad \left[(1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2 - v''(e)\right]\{-2\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] \\ &\quad +(1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\}. \end{aligned} \tag{9}$$

Solving:

$$\begin{aligned} \frac{\partial b}{\partial \upsilon} &= |A_\upsilon|^{-1}\left\{-\upsilon b[u'((1+\varepsilon e)w)\varepsilon w]^2 + \left[(1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2 - v''(e)\right] * \right. \\ &\quad \left. [b[-h'(\varphi b)\varphi + \kappa'(sb)s] + [u((1+\varepsilon e)w) - h(\varphi b) + \kappa(sb)]]\right\}, \end{aligned} \tag{10}$$

$$\begin{aligned} \frac{\partial e}{\partial \upsilon} &= |A_\upsilon|^{-1}\left\{\left[-2\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] + (1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\right] bu'((1+\varepsilon e)w)\varepsilon w \right. \\ &\quad \left. -\upsilon u'((1+\varepsilon e)w)\varepsilon w \{b[-h'(\varphi b)\varphi + \kappa'(sb)s] + [u((1+\varepsilon e)w) - h(\varphi b) + \kappa(sb)]\}\right\}. \end{aligned} \tag{11}$$

The numerator of the solution for both $\frac{\partial b}{\partial \upsilon}$ and $\frac{\partial e}{\partial \upsilon}$ is negative. Since $|A_\upsilon| \geq 0$ for υ, w and ε high enough, this delivers the result.■

Proof of Proposition 2

Totally differentiating the system comprised by equations (3) and (4) with respect to the parameter φ obtains:

$$-\upsilon u'((1+\varepsilon e)w)\varepsilon w \frac{\partial b}{\partial \varphi} + \left[-v''(e) + (1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2\right]\frac{\partial e}{\partial \varphi} = 0, \tag{12}$$

$$\begin{aligned} &\left\{-\upsilon\left[-h'(\varphi b)\varphi + \kappa'(sb)s\right] + (1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\right\}\tfrac{\partial b}{\partial \varphi} \\ &\quad -\upsilon u'((1+\varepsilon e)w)\varepsilon w \tfrac{\partial e}{\partial \varphi} \\ &= -h'(\varphi b) + \upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] + (1-\upsilon b)[h'(\varphi b) + h''(\varphi b)\varphi b]. \end{aligned} \tag{13}$$

The effect of an unanticipated decline in the health burden can be derived from (13) by setting $\frac{\partial e}{\partial \varphi} = 0$. This yields:

$$\frac{\partial b}{\partial \varphi} = \frac{-\upsilon bh'(\varphi b) + (1-\upsilon b)h''(\varphi b)\varphi b + \upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s]}{\{-2\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] + (1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\}}. \tag{14}$$

The denominator of this expression is negative. Under Assumption 2, the numerator of this expression is positive for υ and φ are low enough. This guarantees that the negative first term in the numerator for equation (14) does not prevail on the other positive terms. This guarantees $\frac{\partial b}{\partial \varphi} \leq 0$.

Analyzing the effect an anticipated change in φ, by (12):

$$\frac{\partial e}{\partial \varphi} = \frac{\upsilon u'((1+\varepsilon e)w)\varepsilon w \frac{\partial b}{\partial \varphi}}{[-v''(e) + (1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2]}. \tag{15}$$

Since the numerator is negative and the marginal utility of consumption is posive, $\frac{\partial e}{\partial \varphi}$ and $\frac{\partial b}{\partial \varphi}$ always have the opposite sign.

To obtain the full solution, we represent the system in the form $A_\varphi \begin{bmatrix} \frac{\partial b}{\partial \varphi} \\ \frac{\partial e}{\partial \varphi} \end{bmatrix} = y_\varphi$, where:

$$A_\varphi = \begin{bmatrix} -\upsilon u'((1+\varepsilon e)w)\varepsilon w & \left[-v''(e) + (1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2\right] \\ \left\{-\upsilon\left[-h'(\varphi b)\varphi + \kappa'(sb)s\right] + (1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\right\} & -\upsilon u'((1+\varepsilon e)w)\varepsilon w \end{bmatrix}$$

$$y_\varphi = \begin{bmatrix} 0 \\ \upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] - \upsilon b h'(\varphi b) + (1-\upsilon b)h''(\varphi b)\varphi b \end{bmatrix}.$$

Solving, we obtain:

$$\frac{\partial b}{\partial \varphi} = \frac{\left[v''(e) - (1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2\right]\left\{\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] - \upsilon b h'(\varphi b) + (1-\upsilon b)h''(\varphi b)\varphi b]\right\}}{|A_\varphi|} \tag{16}$$

$$\frac{\partial e}{\partial \varphi} = \frac{-\upsilon u'((1+\varepsilon e)w)\varepsilon w\{\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] - \upsilon b h'(\varphi b) + (1-\upsilon b)h''(\varphi b)\varphi b\}}{|A_\varphi|} \tag{17}$$

where

$$\begin{aligned} |A_\varphi| = {} & [\upsilon u'((1+\varepsilon e)w)\varepsilon w]^2 - \\ & \left[(1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2 - v''(e)\right]\{-\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] \\ & +(1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\}. \end{aligned} \tag{18}$$

The term $|A_\varphi|$ is positive for υ and w, ε high enough. This is the relevant case, since by Proposition 1 it guarantees that fertility and human capital rise in response to a reduction in maternal death risk. For υ and φ high enough, the sign of the numerator in the expression for $\frac{\partial b}{\partial \varphi}$ is negative, while the sign of the numerator of the expression for $\frac{\partial e}{\partial \varphi}$ is positive, which delivers the result. ■

Proof of Proposition 3

We totally differentiate the system of first order necessary conditions with respect to s:

$$-\upsilon u'((1+\varepsilon e)w)\varepsilon w \frac{\partial b}{\partial s} + \left[-v''(e) + (1-\upsilon b)u''((1+\varepsilon e)w)(\varepsilon w)^2\right]\frac{\partial e}{\partial s} = 0, \tag{19}$$

$$\begin{aligned} \left\{-2\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] + (1-\upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\right\}\frac{\partial b}{\partial s} & -\upsilon u'((1+\varepsilon e)w)\varepsilon w \tfrac{\partial e}{\partial s} \\ = -(1-\upsilon b)\kappa''(sb)sb & -(1-2\upsilon b)\kappa'(sb). \end{aligned} \tag{20}$$

To derive the response to an unanticipated change in s, we set $\frac{\partial e}{\partial s} = 0$ in equation (20) and solve for $\frac{\partial b}{\partial s}$:

$$\frac{\partial b}{\partial s} = \frac{\upsilon b \kappa'(sb) - (1 - \upsilon b)\left[\kappa''(sb)sb + \kappa'(sb)\right]}{\{-2\upsilon[-h'(\varphi b)\varphi + \kappa'(sb)s] + (1 - \upsilon b)[-h''(\varphi b)\varphi^2 + \kappa''(sb)s^2]\}}. \tag{21}$$

The denominator of equation (21) is negative. The sign of the numerator depends on the elasticity of substitution in the mother's direct utility from children and in the dynastic utility from children. In particular, fertility will fall in response to an unanticipated rise in infant survival probability if: $\kappa''(sb)sb + \kappa'(sb) \leq 0$. Assuming $\kappa(x) = \frac{x^{1-\alpha}}{1-\alpha}$, then the response of fertility to an unanticipated rise in s is negative if: $-\alpha + 1 \leq 0$, that is if the coefficient of relative risk aversion in the utility from children is greater than 1.

To derive the effect of an anticipated change, represent the system of first order necessary condition in the form: $A_s \begin{bmatrix} \frac{\partial b}{\partial \varphi} \\ \frac{\partial e}{\partial \varphi} \end{bmatrix} = y_s$, where $A_s = A_\upsilon$ and:

$$y_s = \begin{bmatrix} 0 \\ \upsilon b \kappa'(sb) - (1 - \upsilon b)\left[\kappa''(sb)sb + \kappa'(sb)\right] \end{bmatrix}.$$

Solving the system:

$$\frac{\partial b}{\partial s} = |A_s|^{-1} \left\{ \left[v''(e) - (1 - \upsilon b)u''((1 + \varepsilon e)w)(\varepsilon w)^2 \right] \left[\upsilon b \kappa'(sb) - (1 - \upsilon b)\left(\kappa''(sb)sb + \kappa'(sb)\right) \right] \right\}, \tag{22}$$

$$\frac{\partial e}{\partial s} = |A_s|^{-1} \left[-\upsilon u'((1 + \varepsilon e)w)\varepsilon w \right] \left[\upsilon b \kappa'(sb) - (1 - \upsilon b)\left[\kappa''(sb)sb + \kappa'(sb)\right] \right], \tag{23}$$

where $|A_s|$ is the determinant of A_s.

Equations (22) and (23) immediately imply that the response of fertility and the response of human capital always have opposing signs. Assuming that υ, w and ε high enough, so that $|A_s| \geq 0$ implies that fertility will fall in response to an anticipated rise in s under Assumption 3. ■

C Empirical Analysis

Table 19: Cross-state Correlations

	Correlation with expected maternal mortality				Correlation with expected infant mortality			
	Pre-Shock	*Early-Shock*	*Mid-Shock*	*Full-Shock*	*Pre-Shock*	*Early-Shock*	*Mid-Shock*	*Full-Shock*
Children Ever Born	0.25*	0.32**	-0.12	-0.08	0.43***	0.57***	0.09	0.11
M-F Differential in College Graduation	-0.25*	0.06	-0.47***	-0.34***				
	Correlation with concurrent maternal mortality				Correlation with concurrent infant mortality			
Children Ever Born	0.24*	0.45***	-0.02	-0.02	0.46***	0.60***	0.10	0.05

Correlation between initial value and percentage (level) change			
		Mid-Shock vs Early-Shock	*Full-Shock vs Early-Shock*
Expected MMR		-0.19 (0.83***)	-0.14 (0.99***)
Concurrent MMR		-0.36** (0.97***)	0.03 (0.99***)
Children Ever Born		-0.72*** (-0.68)	-0.67*** (-0.54)
M-F Differential in College Grad.		(-0.74***)	(-0.70***)
	Early-Shock vs Pre-Shock	*Mid-Shock vs Pre-Shock*	*Full-Shock vs Pre-Shock*
Expected Infant Mortality	0.19 (0.85***)	0.62*** (0.98***)	0.80***(0.99***)
Concurrent Infant Mortality	0.45*** (0.99***)	0.77*** (0.98***)	0.82*** (0.99***)

* Significant at 1% level. ** Significant at 5% level. *** Significant at 1% level.

Notes: Drop is shown for maternal and infant mortality. Alaska and Hawaii are excluded from the sample.

Table 20: Male-Female Differential in College Graduation: Early-shock Cohort

Expected MMR and M-F College Graduation Differential: Early-Shock vs. Pre-Shock Cohort

	Dependent Variable is Level Change in M-F College Graduation Differential				
	1	2	3	4	5
Level Drop Expected MMR	0.0055	0.0046	0.0041	0.0046	0.0056
	[0.0038]	[0.0032]	[0.0030]	[0.0032]	[0.0044]
Per Capita Personal Income					0
					[0.0000]
Level Change:					
Per Capita Personal Income		0.0000***	0.0001***	0.0001***	0.0001*
		[0.0000]	[0.0000]	[0.0000]	[0.0000]
Share White					-0.7585
					[0.7934]
Share Farm					0.5117
					[0.8000]
Share Health Sector					1.4738
					[2.9499]
Share Public Sector					0.4713
					[0.6374]
Share Textile					0.1683
					[1.2962]
Share Agriculture					-0.2444
					[0.8650]
Unemployment Rate					0.1489
					[0.9688]
Female FT Hourly Wage					-0.0513
					[0.0504]
Male FT Hourly Wage					0.0872
					[0.0809]
Share of 18-64 Population with College			0.7977		
			[0.7556]		
Share of 18-64 Males with College				0.3579	
				[0.4509]	
Constant	-0.0367	-0.1261**	-0.1685**	-0.1532**	-0.1384
	[0.0317]	[0.0496]	[0.0670]	[0.0625]	[0.1053]
Observations	47	47	47	47	47
Adj R-squared	0.0936	0.224	0.229	0.215	0.119

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Pre-shock cohort, born in 1901-08. In all columns the dependent variable is computed for the sample of white married women, not living in institutional quarters and not living in farms. In all columns the sample excludes Alaska and Hawaii. It also excludes South Dakota and Texas because data on expected MMR are not available for the pre-shock cohort. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 21: Concurrent Maternal Mortality and Completed Fertility: Full-shock Cohort

Concurrent MMR and Completed Fertility: Full-Shock vs. Early-Shock Cohort

	Dependent Variable is Level Change in Completed Fertility						
	1	2	3	4	5	6	7
Level Drop Expected MMR	0.0474*	0.0590*	0.0588*	0.0561*	0.0618*	0.0382	0.0597*
	[0.0261]	[0.0317]	[0.0319]	[0.0323]	[0.0352]	[0.0364]	[0.0350]
Concurrent MMR	-0.0545***	-0.0655***	-0.0641**	-0.0498	-0.0421	-0.1068	-0.0499
	[0.0169]	[0.0238]	[0.0245]	[0.0320]	[0.0321]	[0.0807]	[0.0321]
Per Capita Personal Income		0	0	0	0	-0.0001	0
		[0.0000]	[0.0000]	[0.0001]	[0.0001]	[0.0002]	[0.0001]
Level Change:							
Per Capita Personal Income	0	0	0	0	-0.0001	-0.0001	0
	[0.0001]	[0.0001]	[0.0001]	[0.0001]	[0.0002]	[0.0002]	[0.0001]
Share White			0.1651	0.976	1.3977	-2.7165	0.9379
			[0.7205]	[1.3247]	[1.4267]	[5.4704]	[2.6059]
Share Farm			0.2959	0.0261	-0.0541	0.8516	0.0206
			[1.0215]	[1.0401]	[1.3590]	[2.9742]	[1.1444]
Share Foreign Born			-0.7605	-0.3083	0.6774	-2.0939	-0.3086
			[2.7758]	[2.6564]	[2.7780]	[4.7065]	[2.6595]
Share Health Sector				7.7773	9.1332	17.2708	7.7517
				[6.8963]	[5.8867]	[16.7451]	[7.1578]
Share Public Sector				-2.5805	-3.5447	12.672	-2.5887
				[3.3217]	[4.3369]	[14.1377]	[3.4279]
Unemployment Rate					-4.1244		
					[2.6085]		
Share High School Graduates					0.347		
					[1.7756]		
Female FT Hourly Wage					-0.1136		
					[0.1609]		
Male FT Hourly Wage					0.11		
					[0.0988]		
Constant	0.9753***	1.1759***	1.2156***	0.9075	0.9897*	2.405	0.908
	[0.2210]	[0.3715]	[0.4390]	[0.6359]	[0.5842]	[2.1777]	[0.6401]
Observations	49	49	49	49	49	49	48
Adj R-squared	0.327	0.321	0.276	0.271	0.272	0.175	0.265

Notes: Robust standard errors in brackets. Mid-shock cohort, born in 1921-28. Early-shock cohort, born in 1911-18. In columns 1-5 and 7 the dependent variable is computed for the sample of white married women with children, not living in institutional quarters and not living in farms. In column 6 the sample is further restricted to women born-in-state. In columns 1-6 the sample excludes Alaska and Hawaii. In column 7 DC is also excluded from the sample. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 22: Male-Female Differential in College Graduation: Post-shock Cohort

Expected MMR and M-F College Graduation Differential: Post-Shock vs. Early-Shock and Full-Shock Cohort

	Dependent Variable is Level Change in M-F College Graduation Differential							
	Post-Shock vs. Early-Shock				Post-Shock vs. Full-Shock			
	1	2	3	4	5	6	7	8
Level Drop Expected MMR	-0.0003	-0.0003	-0.0004	-0.001	0.0119	0.0118	0.0017	0.008
	[0.0008]	[0.0008]	[0.0008]	[0.0010]	[0.0117]	[0.0120]	[0.0051]	[0.0083]
Per Capita Personal Income				0				0
				[0.0000]				[0.0000]
Level Change:								
Per Capita Personal Income		0	0	0		0	0.0000***	0
		[0.0000]	[0.0000]	[0.0000]		[0.0000]	[0.0000]	[0.0000]
Share White				0.1728				-0.8478
				[0.1390]				[0.7445]
Share Farm				-0.0129				0.3032
				[0.2954]				[0.5296]
Share Health Sector				0.2059				0.7982
				[0.9735]				[1.2590]
Share Public Sector				-0.0035				-0.1632
				[0.3464]				[0.5841]
Share Textile				0.1669				0.0879
				[0.2387]				[0.6942]
Share Agriculture				0.1937				0.6522
				[0.2609]				[1.1352]
Unemployment Rate				-0.3196				-0.0967
				[0.5094]				[0.5347]
Female FT Hourly Wage				-0.0257				-0.0475
				[0.0214]				[0.0408]
Male FT Hourly Wage				0.0196				0.0935*
				[0.0244]				[0.0479]
Share of 18-64 Population with College			-0.0696				-0.8353	
			[0.1551]				[0.5909]	
Constant	0.0276*	0.0284	0.0317	0.0656	-0.0355	-0.0534*	0.0623	-0.0519
	[0.0149]	[0.0226]	[0.0219]	[0.0532]	[0.0353]	[0.0313]	[0.0603]	[0.0710]
Observations	49	49	49	49	51	51	51	51
Adj R-squared	-0.0157	-0.0377	-0.0495	-0.0112	0.0378	0.0337	0.13	0.281

Notes: Robust standard errors in brackets. Early-shock cohort, born in 1911-18. Full-shock cohort, born in 1931-38. Post-shock cohort, born in 1941-48. In all columns the dependent variable is computed for the sample of white married women, not living in institutional quarters and not living in farms. In columns 1-4 the sample excludes Alaska and Hawaii. *Significance at 10% level. **Significance at 5% level. ***Significance at 1% level.

Table 23: Predictors of Infant Mortality

Expected Infant Mortality

		share white	share farm	share of foreign born	share high school	share in health	share in public	share agri-colture	share textile	unemploy ment rate	per capita personal income	female ft real wage	male ft real wage
1930/Pre-shock	coeff	-13.88	-0.81			-98.11	-55.51			78.17			
	t-stat	-0.42	-0.05			-0.16	-0.73			0.82			
	p-val	0.68	0.96			0.87	0.47			0.42			
1940/Early-shock	coeff	-4.44	12.45	-121.45	-39.13	-328.71	-23.00	66.96	9.28	68.45	0.00	-4.89	-7.57
	t-stat	-0.18	0.93	-2.21	-2.31	-0.88	-0.41	0.98	0.19	0.98	-1.58	-0.93	-1.93
	p-val	0.86	0.36	0.03	0.03	0.38	0.68	0.33	0.85	0.33	0.12	0.36	0.06
1950/ Mid-shock	coeff	3.90	-0.91	-86.32	-38.43	-188.52	18.47	84.88	-41.55	66.26	0.00	1.08	-3.93
	t-stat	0.19	-0.08	-1.60	-2.47	-0.74	0.58	1.41	-1.10	0.79	-1.14	0.31	-1.31
	p-val	0.85	0.94	0.12	0.02	0.46	0.56	0.17	0.28	0.43	0.26	0.76	0.20
1960/ Full-shock	coeff	-1.63	2.63	-42.21	-32.47	-165.43	22.77	29.40	-24.05	67.91	0.00	-0.61	-0.60
	t-stat	-0.23	0.44	-1.90	-3.65	-1.76	1.56	1.17	-1.56	1.51	-1.03	-0.73	-1.11
	p-val	0.82	0.66	0.06	0.00	0.08	0.13	0.25	0.13	0.14	0.31	0.47	0.27

Concurrent Infant Mortality

		share white	share farm	share of foreign born	share high school	share in health	share in public	share agri-colture	share textile	unemploy ment rate	per capita personal income	female ft real wage	male ft real wage
1930/Pre-shock	coeff	-7.32	-0.42			-178.11	-54.18			44.92			
	t-stat	-0.27	-0.03			-0.36	-0.85			0.59			
	p-val	0.79	0.97			0.72	0.40			0.56			
1940/Early-shock	coeff	-10.47	14.71	-98.71	-29.75	-446.34	-18.15	57.34	-8.83	45.30	0.00	-4.39	-7.91
	t-stat	-0.50	1.27	-2.08	-2.03	-1.39	-0.37	0.97	-0.21	0.74	-1.89	-0.95	-2.35
	p-val	0.62	0.21	0.04	0.05	0.17	0.71	0.34	0.84	0.46	0.06	0.35	0.02
1950/ Mid-shock	coeff	6.24	1.27	-38.19	-17.46	-83.62	12.43	37.33	-34.19	28.00	0.00	0.68	-1.61
	t-stat	0.55	0.19	-1.29	-2.01	-0.60	0.72	1.14	-1.66	0.61	-0.83	0.35	-0.97
	p-val	0.58	0.85	0.20	0.05	0.55	0.48	0.26	0.11	0.55	0.41	0.72	0.34
1960/ Full-shock	coeff	-4.84	1.15	-18.94	-15.85	-76.86	20.46	18.99	-11.75	44.89	0.00	-0.11	-0.29
	t-stat	-1.20	0.33	-1.55	-3.07	-1.45	2.62	1.37	-1.39	1.81	-0.82	-0.23	-0.96
	p-val	0.24	0.74	0.13	0.00	0.15	0.01	0.18	0.17	0.08	0.42	0.82	0.34