Fertility Responses to Reductions in Mortality: Quasi-Experimental Evidence from 20th Century America*

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Abstract

The introduction of the first antibiotics in the United States in 1937 led to sharp drops in child mortality from pneumonia and maternal mortality from puerperal sepsis. We leverage this plausibly exogenous medical innovation to examine fertility responses among women giving birth in this era. The decline in child mortality led women to delay childbearing and have fewer children overall, with fewer women having three or more children, and a larger share remaining childless. We present a new theory of the extensive margin response, where reductions in child mortality reduce the time women need to achieve their target number of children. This prompts fertility delay and labor market entry which, coupled with wage or fecundity shocks, can result in childlessness. We show that reductions in child mortality increased women's labor force participation, improved their occupational status and reduced their chances of ever having married. Maternal mortality decline had opposing effects on all of these outcomes.

Keywords: fertility timing, childlessness, child mortality, maternal mortality, women's labor force participation, medical innovation

JEL Classification: J13, I18

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1 Introduction

This paper investigates fertility responses to one of the most dramatic health innovations in modern times, the introduction of the first antibiotics in the United States in 1937, which led to sharp drops in both mortality and morbidity from a range of treatable bacterial infections. Among these, two of the most prevalent were pneumonia, the leading cause of child mortality, and puerperal sepsis, the main cause of maternal mortality (Jayachandran, Lleras-Muney, and Smith 2010, Thomasson and Treber 2008).[1] Although many studies have argued that mortality decline generates fertility decline in the context of the demographic transition (e.g. Soares 2005), the evidence is mixed and a recent survey questions the role of mortality (Galor 2012). More generally, the drivers of the fertility transition remain hotly debated.

We depart from previous work on the relationship between mortality and fertility in three substantive ways, which together contribute to illuminating why the evidence is so mixed. We provide the first analysis distinguishing extensive from intensive margin fertility responses to declines in morbidity and mortality.[2] In a key paper, Aaronson, Lange, and Mazumder (2014) highlight the broader relevance of this distinction to discriminating between competing theories of the demographic transition, and argue that in a Becker and Lewis (1973) quantity-quality model of fertility, a decline in the cost of child quality (e.g. due to a decline in the cost of education) increases the value of having at least one child without changing the value of childlessness, and hence should lead to an increase in fertility at the extensive margin. We find that, in contrast, the decline in child mortality and morbidity precipitated by the antibiotic revolution, which presumably improved child health and thus child quality, resulted in more women remaining childless.

This key finding motivates our second innovation. We develop a new theory of the extensive margin response in which child mortality decline leads to fertility delay, increased labor force participation of women and, thereby, to an increase in the fraction of women who are childless. Essentially, we allow for a third margin in child production in addition to quantity and quality, namely timing. We empirically confirm the main predictions of this model, showing that the decline in child mortality occasioned by the arrival of antibiotics led women to delay fertility and to increase their labor force participation. The third distinctive feature of our analysis is that it distinguishes fertility responses to a woman's own mortality risk around birth from the risk of child mortality. This is important because child and maternal mortality decline often occur simultaneously, and can have opposing effects on fertility. Thus, omission of maternal mortality may bias estimated effects of child mortality.

Our findings contribute substantially to a growing body of evidence on the causal effects of child and maternal mortality decline on fertility (Galor 2012, Albanesi and Olivetti 2014), and an active literature on childlessness (Baudin, de la Croix, and Gobbi 2015, Currie and Schwandt 2014, Ananat, Gruber, and Levine 2007). We also provide the first systematic evidence that child

[1] Puerperal sepsis is an infection of the reproductive tract after childbirth.

[2] The extensive margin of fertility is whether to have zero or at least one child, while the intensive margin of fertility is the number of children conditional on having at least one.

mortality decline encourages women's labor force participation, contributing to research on fertility timing (Herr 2016, Choi 2017, Ananat and Hungerman 2012), and on the interplay between fertility, labor and marriage market choices (Lundborg, Plug, and Rasmussen 2017, Jensen 2012, Goldin and Katz 2002, Goldin 1997, Caucutt, Guner, and Knowles 2002). The rest of this section elaborates on the three innovations, describes the quasi-experimental variation in mortality that we exploit, summarizes the main results and remarks further upon their contributions to the literature and to contemporary policy making.

First, we document that the decline in child mortality due to antibiotics led to a decline in fertility, with a decrease in the number of women with three or more children and, importantly, an increase in the number of childless women. In the context of classical quantity-quality theory, the decline in child mortality and morbidity following the introduction of antibiotics acted to lower the price of child quality and potentially also quantity. The main insight of Aaronson, Lange, and Mazumder (2014) is that in a Becker and Lewis (1973) style model, a trade-off between child quality and child quantity can only occur on the intensive margin of fertility for women having at least one child, where quantity and quality are substitute goods.[3] For women considering the extensive margin decision of whether to have any children, a decrease in the price of child quality unambiguously raises the value of having at least one child relative to remaining childless, and so reduces childlessness. Thus, the model predicts an ambiguous response to child mortality decline on the intensive margin of fertility but less childlessness, whereas we find that child mortality decline acted to increase the number of childless women. We argue that this is due to the change in fertility timing and labor market choices in response to child mortality decline.

To formalize this argument, we develop a new theory of the extensive margin response, which is our second main contribution. It is premised on the notion that a decline in child mortality acts to reduce the total number of pregnancies that a woman needs to have in order to achieve her target number of children.[4] As a result, she can initiate childbearing later. This makes it more attractive to invest in the labor market, and unnecessary to marry early.[5] Once fertility is delayed and women are in the labor market, positive shocks to wage earnings, learning about the benefits of work (Fernández 2013), decline in fecundity with age, or indeed inertia, can lead to childlessness. We thus propose a model that allows fertility to be part of a dynamic choice-set that includes labor decisions and fertility timing, extending the static quantity-quality framework (Becker and Lewis 1973, Aaronson, Lange, and Mazumder 2014).[6]

[3] Specifically, the shadow price of child quality is increasing in quantity, and the shadow price of child quantity is increasing in quality. Hence, when the price of child quality falls, parents substitute out of quantity and into quality, which further raises the price of child quantity and generates additional substitution out of quantity and into quality.

[4] This may be because of the direct effect of more births surviving or because declines in mortality increase the quality of children, thus reducing the desired number of children. The intensive margin response is consistent with a reduction in the desired number of children; see also Bhalotra and Venkataramani (2012), who show that children born after the introduction of sulfa antibiotics had higher human capital as adults.

[5] The sharp decline in mortality that we trace the effects of was accompanied, as is typically the case, by a sharp decline in morbidity. As women in this era spent a significant share of their time caring for sick children (an episode of pneumonia in our pre-reform era lasted 39 days on average), improvements in the health of surviving children will also have facilitated women's labor market engagement.

[6] The 1930s was a period of growing labor force participation of women (Goldin 2006), but the cohorts of women

Consistent with our theoretical framework, we find that the sharp fall in child mortality rates raised women's labor force participation, working hours and occupational scores (a measure of the skill intensity of employment), and decreased the probability that women ever married. These findings line up with the observed increase in childlessness. Recent models of childlessness suggest that (voluntary) childlessness is driven by the opportunity cost of childbearing at the upper end of the education distribution (Baudin, de la Croix, and Gobbi 2015, Aaronson, Lange, and Mazumder 2014).[7] Ours is a fundamentally different proposition, as it relies on the intuitive idea that falls in child mortality give women more time to engage in other activities, which applies to women of any level of education. Similarly, previous work has linked fertility delay to marriage and labor market incentives (Caucutt, Guner, and Knowles 2002), but no previous work seems to have linked fertility delay to falling child mortality.

Importantly, we confirm that the identified marginal responses of fertility and women's labor force participation to quasi-experimental changes in mortality are also evident as clean stylized facts in historical cross-state data (United States) and in contemporary cross-country data. This underlines the broad scope of our findings. They have important ramifications, especially in today's low-income countries where mortality rates remain high, suggesting that a benefit of policies that reduce child mortality is that they can "liberate" women from early childbearing and multiple pregnancies. Previous work has documented the liberating influences of the expansion of women's education, the introduction of the contraceptive pill, and the legalization of abortion, which enabled fertility delay, later marriage, and labor force participation (Goldin and Katz 2002, Bailey 2006, Ananat and Hungerman 2012, Ananat, Gruber, and Levine 2007). We offer a new hypothesis in arguing that child mortality decline may trigger these same patterns.[8]

A third feature of our analysis is that it distinguishes fertility responses to child and maternal mortality. While theory suggests that, on balance, fertility tends to decline in response to declines in child mortality, declines in maternal mortality risk should encourage fertility because they lower the cost of birth. Often, major innovations such as in infectious disease therapy or policy regime changes will lead to reductions in child mortality that are contemporaneous with reductions in maternal mortality. Then, if child and maternal mortality decline do have opposing effects, we may see limited change in fertility even when the two underlying causal relationships are strong. There is limited evidence on the size or nature of fertility responses to maternal mortality decline, with the exception of Albanesi and Olivetti (2014) and Albanesi and Olivetti (2016), who estimate

in our sample were those that typically had to choose between career and family (Goldin 2004). Aaronson, Dehejia, Jordan, Pop-Eleches, Samii, and Schulze (2017) show that a negative relationship between fertility and labor supply first emerges in the United States in the 1940 census, which is consistent with our findings.

[7] Baudin, de la Croix, and Gobbi (2015) also observe that (involuntary) childlessness is driven by poverty at the lower end of the education distribution.

[8] It is striking that the suite of outcomes that we show respond when potential mothers are exposed to child mortality decline is similar to the suite of outcomes that Aaronson, Lange, and Mazumder (2014) show respond when women are exposed to higher education as children. Among the second generation women in their sample, net fertility is lower, childlessness is higher, marriage is delayed and women have higher occupational scores. Next, the contraceptive pill is estimated to have delayed fertility and increased labor market participation but with no impacts on completed fertility at either margin. Indeed, women in the sulfa era had to choose between career and family while women in the pill era could have both (Goldin 2004, Coles and Francesconi 2017).

that a decline in maternal mortality of one death per 1000 live births is associated with a rise in completed fertility of 0.27 children per married woman and a decline in childlessness among the college-educated, and that medical progress through maternal mortality decline and the invention of infant formula explains around half of the increase in female labor force participation between 1930-1960. We find that the decline in maternal mortality increased fertility (at the extensive margin) and raised the fraction of women who married, but in contrast with Albanesi and Olivetti (2016), that it also discouraged women's labor force participation.

Having profiled our main findings in relation to the existing literature, we now discuss our identification approach, the magnitudes of the estimated parameters, and competing interpretations of our results. When introduced, sulfonamide (sulfa) drugs were available nationwide, at pharmacies, and at an affordable cost (Lesch 2007). However, there was considerable geographic dispersion in the pre-intervention levels of pneumonia and maternal mortality, and we exploit the fact that states initially most burdened by pneumonia and maternal mortality experienced the largest declines in these mortality rates following the introduction of sulfa drugs, in a difference-in-difference approach. Our empirical strategy is similar to that in Acemoglu and Johnson (2007) and Bleakley (2007).[9] Effectively, we investigate whether the post-sulfa convergence in levels of pneumonia and maternal mortality across the U.S. states after 1937 is mirrored in the fertility behavior of women giving birth during this time.[10]

The main threat to identification arises if birth outcomes exhibited non-parallel trends in the pre-sulfa era in states with higher versus lower disease burdens. We investigate this and find no evidence of diverging pre-trends. We nevertheless control for a rich set of placebo diseases (diseases not treatable with sulfa drugs), measures of health care access and socioeconomic conditions at the state-year level. We also include region-year fixed effects to control more flexibly for convergence in unobservable factors. We show that our estimates are robust to a number of specification checks, including controlling for confounding events, such as the role of the Second World War, New Deal spending and the Dust Bowl, and that they stand up to checks that account for measurement error, survivorship bias, sample selection and migration.

Many previous studies analyze gross fertility (number of births) rather than net fertility (surviving births), while theoretical predictions tend to refer to the latter (Galor 2012). Measures of gross fertility will tend to overestimate fertility targets because some children do not survive, while measures of net fertility will tend to overestimate voluntary childlessness since they will not record births that did not survive. We use both net and gross measures of fertility and show that, in response to sulfa drugs, they moved in the same direction.

Using the United States decennial population censuses for 1940-1970, we identify women of

[9] These are amongst the small set of studies that attempt to account for the endogeneity of mortality. Bleakley (2007) is the only other paper analysing fertility responses to a reduction in child morbidity, but in a context where there was no concomitant reduction in either child or maternal mortality. Acemoglu and Johnson (2007) analyse the impacts of historical health innovations, including antibiotics, on economic growth per capita; fertility is considered as it influences the population size (the denominator).

[10] Although there was only one medical innovation, the pre-intervention levels of pneumonia mortality and maternal mortality exhibit a different distribution across the U.S. states - see Section 2.

reproductive age (15-40) in a window around the introduction of antibiotics in 1937. First, we estimate the hazard of birth of a woman in a given year in 1930-1943 as a function of exposure to sulfa drugs. This model estimates differences in birth timing and birth probability before compared to after 1937 in states with higher versus lower burden from sulfa-treatable pneumonia mortality (the leading cause of child mortality) and maternal mortality. Second, to complement this analysis and estimate the effect of antibiotics on childlessness and completed fertility, we find these women in later censuses and estimate the "stock" of children as a function of exposure to sulfa drugs, defined as the number of fertile years a woman was exposed to antibiotics interacted with pre-sulfa mortality rates at the state level. The hazard model captures the dynamics of fertility, but the stock model allows us to distinguish the extensive and intensive margins of fertility, as well as gross and net fertility.

Our main findings are as follows. If we use a decline from the 75th to the 25th percentile of the pre-intervention pneumonia mortality distribution to scale our estimates, they imply that the time to first birth increased when pneumonia mortality declined, and the average woman was 0.3 percentage points less likely to become a mother for every additional year of exposure to the decline in child mortality (relative to a pre-intervention per-year mean of 5.1%). As the average length of sulfa exposure in this sample was 16 years, this effect is economically significant. We find no significant impact of maternal mortality decline on the time to the first birth. Estimates from the stock model imply that this same decline led to a 4.6 percentage point increase in the probability of childlessness for women who had not yet completed their fertility at the time of census enumeration, and to 0.18 fewer (net) children conditional on at least one (which is 7% of the baseline mean of 2.61). By the time women completed their fertility, the impact on childlessness is estimated to have been an increase in 1.4 percentage points, suggesting that two thirds of the response for the childbearing sample was due to delay, and one third due to permanent childlessness.

At the same time, the decline in maternal mortality resulted in the average woman in the childbearing sample being 3.3 percentage points (or 9% of baseline) less likely to be childless. However, this effect is estimated to be zero for the sample of women with completed fertility, suggesting that the decline in maternal mortality altered the timing of births but not the desired number. Note that the impacts of maternal and child mortality are not comparable because they are on different scales: an inter-quartile shift in pneumonia mortality (-0.26) is of very different magnitude to an inter-quartile shift in maternal mortality (-1.84). Moreover, the compliers responding to declines in pneumonia and maternal mortality are likely different; for instance, estimates by education suggest that the average response of women's employment to maternal mortality was driven by women with less than high school education (and larger among black women, though evident for both races).[11] In contrast, women's employment responses to child mortality are evident at both ends of the education distribution (although restricted to white women). Overall, to the extent that different types

[11] This is presumably because women with low levels of education had higher baseline risks and so were more treatable by sulfa drugs. Bhalotra and Clarke (2016) document negative causal estimates of women's education on maternal mortality. In contrast, we find similar coefficients on child mortality decline for women with high (college) and low education (high school dropouts).

of women are at the margin for responses to child and maternal mortality, this suggests a possible polarization among women, with some increasing labor supply and decreasing their fertility, and others doing the opposite.

Turning to mechanisms, and consistent with the predictions of our fertility delay model, we find that the decline in pneumonia mortality led to increases in the probabilities that women were in the labor force and employed, increased hours of work, and higher occupational scores, while also leading to a reduction in the probability that they ever married. We estimate that the mean duration of exposure (during the reproductive years) to sulfa drugs led to an increase in the probability of labor force participation and employment of 2.6 and 2.8 percentage points respectively, which is 7% and 8% of the baseline means respectively. Over the decade of the 1930s, labor force participation among married women increased by 15.5 percentage points. Previous work has attributed this to the rise in high school completion (Goldin 2006) but our results suggest that child mortality decline may have contributed a substantial share of this rise.[12] We also estimate an increase of 6.6 percentage points in the average woman's occupational score, and that she worked 1.15 hours more per week. Our estimated impacts on marriage rates are smaller, with the chances of being ever-married declining by 1.5 percentage points (or 1.7%).

As is common, we do not have an instrument for each potential channel, but we estimate that child mortality decline increased the joint probability of childlessness and work by 19% of baseline, and that this increase was driven entirely by the decline in the number of women who were both not working and not childless. The descriptive associations are consistent with labor force participation and marriage being mechanisms driving the link between child (pneumonia) mortality and childlessness. For instance, comparing means for childless women versus mothers in our sample, we observe that 53% of childless women were employed compared to 37% of mothers, and that 89.5% of childless women were ever-married compared with 99.6% of mothers.

The magnitudes of the estimated effects of child mortality on fertility, labor and marriage market choices in this study are broadly comparable to estimates in other studies of the impacts of improvements in own education (on fertility and occupational scores) (Aaronson, Lange, and Mazumder 2014), and to impacts of the birth control pill on fertility delay and labor market participation (Goldin and Katz 2002, Bailey 2006, Ananat and Hungerman 2012).[13]

We consider competing interpretations of our findings, focusing upon the result that child mortality decline increased women's labor force participation. One is that it was sulfa-related improvements in maternal health and women's life expectancy that led to increases in their labor market attachment (as in Albanesi and Olivetti 2016), rather than the decline in child mortality. Another candidate is the role of income change (as in Baudin, de la Croix, and Gobbi (2015) and Baudin, de la Croix, and Gobbi (2017)). A third possibility is that the increases in women's labor

[12] If every additional childless woman worked, the increase in employment would be 12.8%, which is the increase in childlessness we estimate using the net fertility measure. Thus, 8% underlines the plausibility of the link between changes in childlessness and employment of women.

[13] Bailey (2006) estimates that contraceptive pill access by age 21 increased labor force participation among 26-30 year olds by 18%.

force participation that we attribute to child mortality can instead be attributed to the Second World War, given previous work showing that women joined the labor force in response to men's deployment (Acemoglu, Autor, and Lyle 2004, Goldin and Olivetti 2013). We investigate these alternatives and show that they do not drive our findings.

In contrast to recent models of childlessness (Aaronson, Lange, and Mazumder 2014, DeCicca and Krashinsky 2017), our findings are not driven by changes in women's acquisition of education (although we do estimate a small increase in high school completion rates in response to child mortality decline). Moreover, in contrast to the results in Baudin, de la Croix, and Gobbi (2017) and DeCicca and Krashinsky (2017), marriage does not seem to be a necessary pathway. Similar to Baudin, de la Croix, and Gobbi (2017), we find that child mortality decline is associated with a later age at marriage and lower chances of having ever married, alongside an increase in childlessness. However, the coefficients indicate relatively small effects and, more importantly, results by education and race for marriage outcomes do not mirror the results for fertility and employment, suggesting that marriage was one important margin of response but not the main mediator of impacts on childlessness. Although the vast majority of births in this era were in-wedlock so that marriage and fertility co-varied, marriage bars that prevented married women from working were virtually eliminated by 1940 (Goldin 2006), affording a de-linking of marriage and the labor force participation of women.[14] Importantly, we underline that voluntary fertility timing choices generate childlessness, while in the model in Baudin, de la Croix, and Gobbi (2015), childlessness is largely involuntary. We assume that all women can control their fertility, in line with Morgan (1991) and Engelman (2011).[15]

Our findings are of contemporary relevance. Pneumonia is the leading cause of death among children today, and puerperal sepsis continues to be a major cause of maternal mortality. Child and maternal mortality rates in developing countries remain high: 6 million under-5 children continue to die every year (Liu, Oza, Hogan, Chu, Perin, Zhu, Lawn, Cousens, Mathers, and Black 2016), while maternal mortality still remains unnecessarily high, at around 800 deaths each day (Ceschia and Horton 2016). Fertility also remains high at 4.7 births per woman on average in Africa, and 2.2 births per woman in Asia and Latin America (United Nations 2015). While eighty years have elapsed since the innovation of antibiotics, the average consumption of antibiotics in West Africa is approximately 90% lower than in the United States despite much higher rates of infectious disease, marking poor access (Hogberg, Muller, Zorzet, Monnet, and Car 2014). Although there have been marked declines in child and maternal mortality in the last 25 years in response to worldwide mobilization and increasing investments in public health, there is limited causal evidence of fertility responses to these investments. Our findings suggest that investments in global health will lead to fertility decline, with possible knock-on effects on childlessness, women's labor market

[14] In DeCicca and Krashinsky (2017), education raises the probability of being married, and marriage lowers the price of child quality as fathers provide inputs and insurance, so women have more children on both the extensive and intensive margins. Although their earnings increase, their labor force participation does not.

[15] In the simulations presented in Baudin, de la Croix, and Gobbi (2017), the ability to control fertility increases marriage rates and overall fertility (these women become more attractive on the marriage market), whereas in our case, the ability to control fertility gives women the option to delay, which can result in childlessness.

participation and marriage market choices. These knock-on effects rely on the availability of labor market opportunities for women, which will vary across developing countries. Furthermore, women being economically active and financially autonomous is linked to increased investments in children (Lundberg, Pollak, and Wales 1997, Baranov, Bhalotra, Biroli, and Maselko 2017) and reductions in domestic violence (Aizer 2010), underlining the broad scope of our findings.

The remainder of the paper evolves as follows. Section 2 explains the context and documents the decline in child and maternal mortality rates occasioned by the sulfa drug revolution. Section 3 describes the empirical strategy and Section 4 discusses the data. Section 5 discusses the estimated effect of sulfa exposure on fertility. Section 6 presents the dynamic model of fertility and labor choices and the estimated effect of sulfa exposure on labor and marriage market outcomes. Section 7 concludes.

2 Mortality Rates and the Sulfa Drug Revolution

The United States in the 20th century was characterized by high levels of maternal and child mortality (Britten 1942, Linder and Grove 1947, Thomasson and Treber 2008, Jayachandran, Lleras-Muney, and Smith 2010). Pneumonia was the leading infectious cause of child morbidity and mortality, most often occurring as a complication of influenza, and the leading cause in general after death from premature birth and congenital defects. Child mortality (among the under-5s) from pneumonia in the United States stood at an average of 11.8 per 1000 population between 1930-36, of which the majority occurred among infants (under-1s; 8.2 per 1000). Pneumonia mortality rates are U-shaped in age, being around ten times higher among infants than among adults and rising among the elderly (see Figure 1). Hence, the reduction in pneumonia mortality will have had a small if any impact on the health of women.[16] A major cause of maternal mortality, accounting for 40% of maternal deaths, was puerperal fever, an ascending bacterial infection of the reproductive tract that can occur soon after birth. The rate of maternal mortality per 1000 live births was 6.35 in 1930 (Vital Statistics). Maternal mortality is the tip of an iceberg, the mass of which represented high rates of maternal morbidity (Albanesi and Olivetti 2014). Also, as we will discuss in more detail, puerperal sepsis could cause uterine scarring and infertility among those who survived, thus influencing future births. For pneumonia and post-partum fever among new mothers, the mainstay of treatment up until the introduction of sulfonamide antibiotics in 1937 was supportive care. The arrival of antibiotics drastically altered the standard of medical care.

Sulfa drugs were discovered by German chemists experimenting with textile dyes in 1932 and become widely available in the United States starting in 1937. By all accounts, adoption was widespread and quick, costs were relatively low for a life-saving drug, and this was reflected in sharp declines in mortality from pneumonia as well as maternal mortality, with an ensuing "sulfa

[16] Pneumonia was the most prevalent infectious disease and the leading cause of post-neonatal deaths. It accounted for 10% of all-age deaths and 17% of infant deaths in the United States in the 1930s. In terms of morbidity, estimates from the US National Health Survey of 1934-1936 show a case rate of 3 per 100 infants, with rates twice as high among the poor and those living in crowded conditions (Britten 1942).

craze" (Jayachandran, Lleras-Muney, and Smith 2010, Lesch 2007, Lerner 1991).[17]

In addition to reducing mortality, the arrival of sulfa drugs led to significant reductions in morbidity (Greengard, Raycraft, and Motel 1943, Hodes, Stifler, Walker, McCarty, and Shirley 1939, Moody and Knouf 1940). Prior to the introduction of antibiotics, pneumonia was a debilitating disease with typical spells often lasting 39 days and children tending to have recurrent spells (Britten 1942). With the arrival of sulfa drugs, the severity and length of these episodes decreased (Connolly, Golden, and Schneider 2012), and this was documented in clinical trials among hospital inpatients (Greengard, Raycraft, and Motel 1943, Moody and Knouf 1940), and in the community, as the medication was readily available without prescription until 1939, and at relatively low cost (Lesch 2007, Lerner 1991). Historical research documents the wide penetration in the consumer pharmaceuticals market (Lesch 2007). Relevant to the ensuing discussion, a reduction in pneumonia morbidity among children will have increased the disposable time of women, who were the main care-givers for children and the sick. A marker of the substantive importance of morbidity decline among children born in the sulfa era is that reduced exposure to pneumonia in infancy had large and statistically significant impacts on their educational attainment, employment, income, and disability when adults (Bhalotra and Venkataramani 2012).

Sulfa drugs arrived during a period of declining childlessness, increasing fertility and increasing labor force participation (Figure 7). Thus, any labor market impacts of sulfa drugs will have been bolstered by the fact that this was a time of increasing opportunity for women to have careers if they chose to do so (Goldin 2004).

3 Research Strategy

Our research strategy exploits the timing of the introduction of antibiotics, which created sharp variation across cohorts in exposure to disease, with systematically larger changes in states with higher pre-intervention burdens of disease. We essentially compare fertility behavior before and after the arrival of sulfa drugs for women born in states with higher and lower pre-antibiotic mortality rates. Figure 3 shows trend breaks in pneumonia and maternal mortality in 1937. The steepest post-sulfa decline in pneumonia mortality was amongst infants and young children (Figure 2). Moreover, the arrival of sulfa drugs stimulated convergence in the levels of pneumonia and maternal mortality rates across the U.S. states, the states with higher pre-1937 burdens of disease showing greater absolute declines in mortality levels; see Figure 4. Convergence in pneumonia mortality rates was most marked for child pneumonia (Figure 5). The patterns in the figures are formalized with tests of significance in Tables 14 and 15 in Online Appendix B. As we investigate the importance of two causes of death, it is important that pre-intervention levels of pneumonia and maternal mortality exhibit a different distribution across the U.S. states. Figure 6 shows that

[17] Sulfa drugs also reduced mortality and morbidity from skin and soft tissue infections and meningitis (Jayachandran, Lleras-Muney, and Smith 2010). However, the incidence of these diseases was very low and they made insignificant contributions to both infant and maternal mortality. For example, the all-age mortality rate from skin diseases in the U.S. was 1.8 in 1930, and from meningitis 2.1, compared to an all-cause infant mortality rate of 69 (Vital Statistics).

while they are positively correlated, they also exhibit independent variation. Following a tradition in the literature (Almond 2006, Bozzoli, Deaton, and Quintana-Domeque 2009), we assume that changes in mortality are a proxy for both mortality and morbidity.

The Dynamics of Birth

First, we analyze how the timing of birth reacted to child and maternal mortality decline occasioned by the discrete introduction of sulfa drugs. For this, we use the sample of women of reproductive age during 1930-43 and model the hazard of birth in a short window around 1937 using a data file expanded to the woman-year level. We estimate the impacts of exposure to post-sulfa decline in pneumonia and maternal mortality, conditional on time since the previous birth, birth order and other covariates. The estimating equation is:

$$\begin{aligned} \Pr(Y_{jst} &= 1) = \beta + \beta_1 * prePneumonia_s * post1937_t \\ &+ \beta_2 * preMMR_s * post1937_t \\ &+ \boldsymbol{\beta_3}\mathbf{hazardcontrols}_j + \boldsymbol{\beta_4}\mathbf{womanbirthyear}_j + \boldsymbol{\beta_5}\mathbf{race}_j + \boldsymbol{\beta_6}\mathbf{education}_j \\ &+ \boldsymbol{\beta_7}\boldsymbol{\lambda}_s + \boldsymbol{\beta_8}\boldsymbol{\theta}_t + \boldsymbol{\beta_9}\boldsymbol{\psi}_{t,r} + \boldsymbol{\beta_{10}} * post1937_t * \mathbf{statecontrols}_s + u_{jst}, \end{aligned} \tag{1}$$

where Y_{jst} is a binary indicator that equals 1 if woman j born in state s gave birth in year t, $post1937_t$ equals one if the birth year of the child is 1937 or after and $prePneumonia_s$ and $preMMR_s$ are the pre-1937 state-level mortality rates from pneumonia and maternal mortality (MMR) respectively, constructed as averages over 1930-1936. We estimate equation (1) as a logistic regression, yielding estimates for a discrete time proportional hazard model.

The vector $\mathbf{hazardcontrols}_j$ consists of indicator variables for woman j's time since her last birth (count starts at age 15) and indicator variables for the birth order of woman j's next birth.[18] We also include the following fixed effects: the woman's birth year ($\mathbf{womanbirthyear}_j$), her race ($\mathbf{race}_j$), education ($\mathbf{education}_j$), and birth state ($\boldsymbol{\lambda}_s$), and the calendar year of the potential birth ($\boldsymbol{\theta}_t$). Education is related to potential wages and fertility preferences. We control for a vector of state-year varying measures (detailed below), the evolution of which may be correlated with birth outcomes and sulfa exposure. Importantly, we include the pre-intervention level of these variables interacted with the indicator $post1937_t$, using the same formulation as that used to capture the pneumonia and maternal mortality shocks. To further allow for unobservable trends, we include census region-year fixed effects ($\boldsymbol{\psi}_{t,r}$).[19] In a robustness check in Online Appendix D, we also investigate controls for woman fixed effects, which will capture any unobservables that determine compliance and are also correlated with fertility preferences.

In order to distinguish between the extensive and intensive margins of fertility timing, we extend this analysis to estimate separately the effect of mortality decline on the probability of a first birth

[18] In pooling multiple births, we are assuming that conditional on covariates, these represent independent events. We cluster standard errors at the state level, and control for the birth order and other woman-specific factors such as education and race, which alleviates concerns over unobserved heterogeneity.

[19] There are four census regions. The estimates are similar when using state linear trends in place of census region-year fixed effects, as well as when not including census region-year fixed effects, indicating that they are not required for satisfying the common trends assumption.

and the probability of higher order births. In particular, we estimate equation (1) on the subset of the sample where we only include woman-year observations where the woman would be giving birth to her first child (the extensive margin), and only including observations where the woman would be giving birth to her second or higher-order child (the intensive margin).

In assigning pre-intervention mortality rates and state-level controls to women, we use their birth state. This is because migration decisions after birth are potentially endogenous.[20] The coefficients of interest are β_1 and β_2, which capture the causal effects of pneumonia and maternal mortality decline on the probability of birth. Standard errors are clustered at the birth state level to account for the increased propensity to falsely reject the null hypothesis in difference-in-differences models that do not account for serial correlation across years and within states (Bertrand, Duflo, and Mullainathan 2004).

The Stock of Births

Changes in the timing of births may be adjusted for later in the lifecourse; indeed, women delayed fertility following the introduction of the contraceptive pill but with no impact on completed fertility (Ananat and Hungerman 2012). So, in order to assess whether there was catch up or compensating variation in fertility in later years, we estimate the stock of births for women of the same birth cohorts as in the hazard sample, but observed in later census years. We create two such samples. One is of women who have plausibly completed fertility, being aged at least 40 at the time of census enumeration; the second pools women aged 18-40 at enumeration to provide further evidence on fertility timing. In both cases, the samples are of women who were of reproductive age either at the time of the antibiotics, or shortly before or after.[21] We now model exposure as the number of fertile years spent in the post-antibiotic era, interacting this with pre-sulfa mortality rates as in the hazard model.

Using the stock model, we give up the ability to identify responses from a discontinuity in exposure in a dynamic setting, but we gain an estimate of whether fertility responses simply reflect the advancing or deferral of overall fertility. The estimating equation is:

$$\begin{aligned} B_{jsc} &= \alpha + \alpha_1 * prePneumonia_s * sulfayears_j \\ &\quad + \alpha_2 * preMMR_s * sulfayears_j \\ &\quad + \boldsymbol{\alpha_3} * \mathbf{birthyear}_c + \boldsymbol{\alpha_4}\mathbf{race}_j + \boldsymbol{\alpha_5}\mathbf{education}_j + \boldsymbol{\alpha_6}\boldsymbol{\lambda}_s \\ &\quad + \boldsymbol{\alpha_7} * sulfayears_j * \mathbf{statecontrols}_s + e_{jsc}, \end{aligned} \tag{2}$$

where B_{jsc} denotes the total number of births to woman j born in state s in cohort c at the time of the census and $sulfayears_j$ is the number of fertile years of woman j during which she is

[20] As a result, we may underestimate treatment effects for women who moved into areas with the largest health gains. However, as a check, we re-estimated the equation assigning all state-level variables to the resident state of the woman at the time of census enumeration, and the results were qualitatively similar.

[21] In particular, the oldest woman in the stock sample at the time of antibiotics introduction was 44 in 1937, so that she was reproductive until 1933, and the youngest woman was six in 1937, so that she was reproductive from 1946 onwards; see Section 4 for details.

exposed to sulfa drugs. We assume women are fertile between the ages of 15 and 40. In the case of uncompleted fertility, $sulfayears_j$ may include future years: for example, a woman aged 30 at the time of the census who was 20 in 1937 would have 20 fertile sulfa years, of which 10 are in the future. If women make dynamic fertility choices, they care about their total exposure to sulfa drugs, and not only their past exposure. We control for birth cohort, race, education and birth state fixed effects, and for state-year varying variables interacted with $sulfayears_j$, similar to the hazard model. Standard errors are again clustered at the state level. To identify the extensive margin response, we redefine the dependent variable as a binary indicator for non-zero versus zero births. To identify the intensive margin response, we re-estimate equation (2) restricting the sample of women to those with at least one birth. When considering labor and marriage market outcomes in Section 6, we replace the left-hand side variable with several measures of labor and marriage market outcomes.

Threats to Inference

Threat to inference with the models outlined above arises if convergence would have occurred even without the arrival of sulfa drugs, and if there are unobservables that correlate with baseline mortality rates that predict different trends in outcomes.[22] In the hazard model, we control flexibly for underlying trends by including census region-year fixed effects. In both models, we allow for state-specific trends in a range of variables that reflect baseline economic and infrastructural conditions that may have been correlated with baseline pneumonia and maternal mortality, or influenced the diffusion of sulfa drugs. These are the logarithms in per capita terms of state income, public health spending, education spending, and the numbers of schools, hospitals and physicians per capita. We similarly allow for differential trends by women's labor force participation and female literacy. Income per capita, in particular, captures underlying convergence in economic development across states, which may affect mortality and fertility. However, we note that income per capita itself is not the most important predictor of variation in pre-sulfa mortality rates across states. For example, it explains only 10.7% of the variation in pneumonia mortality and 17.4% of the variation in maternal mortality. In contrast, racial structure, for example, explains more variation than income for both sources of mortality, and is particularly important for maternal mortality. Hence, we also control for individual-level race in all regressions. We return to the question of underlying trends in economic conditions on presentation of the results below; the fact that we find opposing effects of child and maternal mortality on fertility and labor market outcomes is another reason why economic development is an unlikely confounder of our findings, as it would need to be correlated in opposite directions with convergence in child and maternal mortality, which is not the case in the raw data.

We control for six cause-specific mortality rates from diseases that were not influenced by sulfa drugs (placebo diseases). This ensures that we are not simply capturing the effects of secular changes in the overall disease environment. Including deaths from communicable diseases (tuberculosis,

[22] Candidate omitted variables include pre-intervention trends in other factors that influence fertility, such as income or skill-biased technological change that differentially increased the returns to quality (i.e. human capital investment) or that produced increased opportunities for women.

diarrhea, malaria) helps control for state-specific changes in, for instance, sanitation, public health programs and housing, that may have coincided with the arrival of sulfa drugs and influenced fertility decisions. We also include non-communicable diseases (cancer, heart disease) to control for factors such as health care quality and access. For all of these variables, we use a pre-sulfa state-level measure, which is their average over 1930-1936 and, in the hazard model, we interact this with $post1937_t$ and, in the stock model, with $sulfayears_j$.[23] Since this was an era in which the states were joining the U.S. Vital Statistics registration system, there was some variation in the quality of birth and mortality data. To address this, we include the years that each state entered the U.S. Vital Statistics birth registration and death registration systems, interacted with $post1937_t$ (hazard) or $sulfayears_j$ (stock). As discussed later, we also test for differential pre-trends in birth outcomes for states with above versus below median pneumonia and maternal mortality rates, on a pre-sulfa sample of 1930-1936.

4 Data

Data on fertility outcomes are taken from the United States Census Microdata (Ruggles, Alexander, Genadek, Goeken, Schroeder, and Sobek 2010). Online Appendix A describes each dataset in greater detail. We focus our attention on women born in the years 1893-1931 and thus aged 6-44 in 1937: we exclude women aged five and under in 1937 as they were potentially directly treated by antibiotics as children, and include women aged 40-44 as a comparison group who were unexposed to sulfa drugs during their fertile period. We find these women in different censuses, depending on the age at which we are interested in their fertility and labor market outcomes.

The hazard dataset is constructed from pooled census microdata using the 1940 and 1950 1% samples, and we restrict our analysis to births that occurred in a short window around 1937, namely 1930-1943, to limit the role of confounding events, such as the influenza epidemic of 1928-9 and the increasingly widespread use of penicillin after 1943. We select women who were of child-bearing age (15-40) at any time between 1930 and 1943 and expand the data to create a woman-level panel, with observations for every year in which a woman was at risk of giving birth. Thus, we only include woman-year observations in which a woman was aged 15-40 in that year, so that we capture choices during the fertile period. This implies that the cohorts in the hazard sample were born in the years 1893-1928, as cohorts born between 1928-1931 did not become fertile until 1944, which is outside our hazard study window. In theories of population increase and economic growth, the focus is on the number of surviving children a woman has, or net fertility (Acemoglu and Johnson 2007, Brueckner and Schwandt 2015). Women looking to achieve a target number of births will also set the target in net terms (Galor 2012), although the costs to the woman stem from the total (gross) number of births. In the hazard model, we use a measure of net fertility that derives from a record of the children living in the mother's household at the time of enumeration. Using a variable that links child records to mothers, we constructed a complete history of live births for each woman.

[23] For female labor force participation and literacy, we use the value in 1930 as annual series are unavailable.

We restrict births to biological births (95% of children in the household) that occurred in the U.S. As is standard, this measure necessarily excludes any pregnancies that did not result in live births, and any deaths that occurred after birth but before the census (the child pneumonia mortality rate between the ages of one and five was approximately 3.6 per 1000 population, less than half of the under-one mortality rate, so that most child deaths occurred during infancy). It also excludes any children that had left home. The latest census we use is the 1950 census, where the oldest child born during the estimation period 1930-1943 would have been 20, which minimises concerns about missing older children.

The stock dataset is compiled from pooled census microdata containing the 1% samples of the 1940, 1950, 1960 and 1970 censuses.[24] In this sample, we include not only women aged 15-40 in the period 1930-1943 (as in the hazard sample) but also fully treated women (aged <=15 in 1937), and untreated women (aged 40-44 in 1937). All women aged 15-40 in 1937 were partially treated.[25]

We are primarily interested here in measuring completed fertility. To this end, we identify women aged 40 or more at the time of census enumeration. For example, the 1917 cohort will have turned 40 in 1957, so we find them in the 1960 census. This allows us to estimate whether any change in birth timing that we observe among women giving birth in the sulfa era reflects intertemporal substitution, or a change in the overall number of children. We have two measures of completed fertility. First, we use a gross measure, being the response to a question that asks about the total number of live births a woman has ever had.[26] Second, we also estimate the stock model using net fertility (defined in the same way as in the hazard sample, based on children resident in the household). Net fertility will tend to underestimate total fertility and overestimate childlessness due to children moving out, hence when assessing the impact on net completed fertility, we restrict the sample to women aged at most 50 at census enumeration. Gross fertility will tend to overestimate total fertility and underestimate childlessness (as some women with a positive number of live births will end up childless due to children dying). As we estimate the effect of sulfa on both measures of fertility, we are able to redress the weaknesses of each.

We also estimate the effect of sulfa exposure on fertility timing in the stock model, by using a sample of childbearing women aged 18 to 40 at the time of census enumeration. As these women have not completed their fertility, this analysis provides a bridge between the hazard model estimates (where net fertility is estimated as a flow during the reproductive years) and the stock model estimates of completed fertility.[27] Given that we are restricted to observing younger women, the

[24] Although the 1960 and later censuses were flat samples, the 1940 and 1950 censuses oversampled some groups. We reestimated our main results with survey weights and they were very similar to the reported results in Section 5. For compactness we do not report these estimates.

[25] Recall that in the stock model, we define treatment as the number of fertile years that a woman was exposed to sulfa drugs, while in the hazard model we use a binary indicator for whether the potential birth year is after 1937. In both cases we interact these with state averages of the pre-intervention mortality rates from the sulfa-treatable diseases of interest.

[26] The live births question was asked only to ever-married women in the 1940 and 1950 censuses (and all women subsequently). In our sample, 92% of women are ever-married in the 1940 and 1950 censuses. We have checked that our results are robust to considering only ever-married women in the 1960 and 1970 censuses.

[27] A threat to inference with net fertility arises if the age at which children leave home is correlated with state level baseline pneumonia or maternal mortality. Although this should be absorbed by state fixed effects, the results

cohorts in this sample were born between 1900-1931: women born between 1893-1900 would have been at least 40 by the time of the 1940 census, and hence have completed their fertility.[28]

Data on baseline rates of diseases were taken from several volumes of the U.S. Vital Statistics (Grove and Hetzel 1968, Linder and Grove 1947, Ruggles, Alexander, Genadek, Goeken, Schroeder, and Sobek 2010, Bureau 1943). For the pre-intervention levels of maternal and pneumonia mortality we use the state-level average over the years 1930-36 of the mortality rate. For maternal mortality, this is defined per 1000 live births and for pneumonia it is the all-age pneumonia-influenza mortality rate per 1000 population. We use an all-age rate in place of the child rate, and the combined measure of pneumonia-influenza instead of pneumonia only, in order to reduce measurement error. Infant deaths were known to be under-reported during this time (Linder and Grove 1947), with under-reporting varying by state, making the child rate noisier than the all-age rate. However, much of the variation in the all-age rate is from child deaths, and a steep decline in child deaths after 1937 contributed significantly to the all-age decline: see Figure 2. Still, in Section 5.3, we show that our estimates are robust to using the all-age rate as an instrumental variable for the infant rate. As for using the combined pneumonia-influenza measure in place of pneumonia, the diseases shared symptoms, such as cough and fever, and so were difficult to distinguish in the 1930s. Moreover, they were intrinsically related since pneumonia often developed from an initial influenza infection. A reflection of the problem of isolating pneumonia from influenza deaths is that annual time series data are only available for the combined measure. However, using decadal data that provide separate series for pneumonia and influenza, we note that (a) pneumonia dominated the combined mortality rate, with 8.9 deaths per 1000 in 1930, compared to 1.3 deaths per 1000 live births from influenza, and (b) the decline in the combined mortality rate between 1930 and 1940 was entirely on account of a decline in the pneumonia mortality rate: while influenza rates fluctuated considerably with epidemics and seasons, the average rate was steady across the decade.

We collated data on under-2 diarrhea, heart disease, cancer, malaria, and tuberculosis mortality from U.S. Vital Statistics as control variables, creating state-specific and cause-specific pre-sulfa era mortality rates between 1930 and 1936. We compiled time series data on the socioeconomic and infrastructure variables described in Section 3 from several sources.[29]

Tables 1, 2 and 3 provide descriptive statistics for the hazard and stock samples, as well as for

are similar when considering net fertility among different age groups at census, focusing on younger women where children are less likely to have moved out; see Online Appendix D.

[28] We have intentionally opted to select samples based on age at census enumeration, in order to compare outcomes during childbearing and after childbearing years. This means that there is a small difference between the cohorts analyzed across the childbearing and completed fertility samples. We have verified that our main results (fertility, labor and marriage market) are very similar when we restrict the sample of each estimate to have exactly the same cohorts.

[29] State time series data on logged state per capita income were downloaded from the Bureau of Economic Analysis website (http://www.bea.gov/regional/spi/). Data on the number of schools, doctors, hospitals, and educational expenditures per capita were taken from Adriana Lleras-Muney's website (http://www.econ.ucla.edu/alleras/research/data.html). These data were originally collected from various volumes of the Biennial Survey of Education (schools and expenditures) and the American Medical Association's American Medical Directory (doctors and hospitals). For state per capita health expenditures, we used data collected from various reports from the US Census Bureau. (See http://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/6304?archive=ICPSR&q=6304)

the state-specific disease environment measures and controls. In the hazard model, the woman-year observations are balanced before and after the intervention, with an annual mean probability of birth of 8.7%. All-age pneumonia and influenza mortality before the intervention was on average 1.09 per 1000 population, while maternal mortality was on average 6.26 per 1000 live births. In the stock model sample, using completed gross fertility, the average woman was exposed to sulfa for 14.9 years, 19% of women were childless at the end of their reproductive years, average total fertility was 2.6 (unconditional on at least one birth) and 3.2 conditional on at least one. In the childbearing net fertility sample, the same figures are 20 years, 36% childless, 1.7 births unconditional, and 2.6 conditional on at least one. The mean age at first birth in the stock sample was 24.1, and 26.7 at second birth.

5 Results

5.1 Hazard Model: Birth Timing

Main Results

Estimates of the hazard model show that the conditional probability of birth in a given year declined with sulfa-led reductions in pneumonia mortality but tended to increase with reductions in maternal mortality (Table 4).[30] These results are consistent with standard quantity-quality theory: the decline in pneumonia mortality and morbidity generated sharp improvements in the child health endowment and in child survival, both of which will have motivated reductions in fertility.[31] The drop in maternal mortality will have made birth less costly for women and hence more attractive, other things equal. The coefficient on pneumonia mortality is robust to the series of controls that are added as we move from left to right in the table. The coefficient on maternal mortality is statistically significant with basic controls and census region-year fixed effects, but is sensitive to the inclusion of controls for state-year variables. Columns (4) and (5) in the same table show the estimated effects of sulfa introduction on the extensive and intensive margins of fertility respectively; the signs are consistent with the average effect on all birth orders.

An interquartile shift in pneumonia mortality (-0.26) implies a 0.6 percentage point reduction in the annual probability of birth, which relative to the annual mean of 8.6% before 1937 is a reduction of 6.9%. The estimates in column (4) imply a 0.3 percentage point reduction in the annual probability of transitioning to motherhood after 1937, which is 5.9% of the pre-intervention mean of 5.1%. For a woman exposed to sulfa drugs for ten years of her fertile period, this is a 3 percentage point increase in the probability of being childless. The reduction in birth probability in response to pneumonia mortality decline implies an increase in the time to first birth, or fertility

[30] Columns (1)-(3) in Table 4 pool a woman's births, and in so doing assumes independence across recurrent events. Any concern over this assumption is alleviated by the fact that standard errors are clustered at the state level. Further, the separation of the extensive and intensive margins in Columns (4) and (5) is robust to this concern, as the extensive margin in particular only has one event (the first birth).

[31] The increase in the child survival rate will have also caused a decline in the per-child price of quantity; however, the overall decline in fertility in response to child mortality decline suggests that the decline in the price of quality dominated, causing a reduction in desired fertility (see also the model in Section 6).

delay. In the same table, column (5) presents estimates of a survival model for higher order births, which captures fertility timing on the intensive margin. The same interquartile shift implies a 0.25 percentage point reduction in the probability of transitioning to a higher order birth after 1937, or 1.7% relative to a pre-intervention mean of 14.9%. Thus, the decline in pneumonia mortality encouraged fertility delay particularly on the extensive margin.[32] We find no statistically significant impact of the reduction in maternal mortality on birth probability on the extensive or intensive margins.

In Online Appendix D, we show that when either pneumonia or maternal mortality are not included in the regressions, the coefficients on the included treatment variable are attenuated, showing the importance of not ignoring one source of mortality when considering the other.

Heterogeneity in Birth Timing by Race and Education

We explore heterogeneity of these effects by race and education in Online Appendix C. We find no statistically significant difference in fertility delay in the survival model between the college educated and high school dropouts (Table 16). We find stronger impacts for blacks than for whites, which can be explained by blacks being exposed to higher mortality rates (typically twice as high) before the introduction of sulfa drugs and thus experiencing larger declines in mortality rates (see Bhalotra and Venkataramani 2012).

Event Study

We estimated an event study specification for probability of birth similar to column (3) of Table 4 with the difference that rather than interact exposures to pneumonia and maternal mortality with the indicator for post-1937, we interact them with every year in the sample period (1930-1943). The resulting coefficients are plotted in Figures 8 and 9. The figures show a clear jump in the coefficient in 1937, especially for pneumonia. This exercise is challenged by statistical power and not all coefficients are statistically significant, but the plot suggests a discrete change. These plots also provide a test of the identifying assumption that pre-trends in birth outcomes in states with high versus low pre-intervention disease burdens were not different. In both plots, the coefficients show no trend before 1937. The figures particularly negate the existence of underlying trends in income or economic conditions that could be driving our findings, as convergence in these economic conditions was more gradual, while we find sharp changes in birth probability after 1937. We conduct further checks on pre-trends in Section 5.3 below.

Overall, our findings show that women delayed childbearing in response to improvements in child survival and child health.

5.2 Stock Model

The hazard model describes how the flow of births changed in response to the introduction of sulfa drugs. To assess whether the response was only an intertemporal substitution or whether

[32] Standardizing the logistic coefficients by the variance in the outcome variable yields coefficients that are directly comparable across samples, and shows that the negative effect of the reduction in pneumonia mortality on the extensive margin birth probability in column (4) is similar to the effect on the overall birth probability in column (3), and both are approximately three times the size as the effect on the intensive margin in column (5).

there was a change in total fertility, we estimate the effect of the intervention on the stock of births. While this is best described by completed fertility, for reasons discussed above, we start by "bridging" estimates of fertility measured as a birth stock for women who are still childbearing. These estimates capture a combination of fertility delay and changes in fertility targets. We then report results for women who have completed childbearing.

5.2.1 Net Fertility Among Women of Childbearing Age

Net fertility is measured the same way as in the hazard model (using information on children living with the mother). We select women aged 18-40 at the time of the census and aged 5-44 in 1937 from the 1940-1970 censuses.

The estimates indicate that the reduction in pneumonia mortality had a significant, negative effect on the total net fertility of women that is robust to inclusion of additional controls (Table 5). The evidence suggests that the reduction in maternal mortality led women to have more children, but this coefficient is unstable in size and significance.

Using the same controls as in column (3) in Table 5, the richest specification, we provide estimates for the intensive and extensive margins separately in columns (4) and (5). In response to pneumonia mortality decline, women had fewer children on both margins. They were more likely to remain childless and, conditional on having at least one child, women had fewer children overall. The intensive margin response to the decline in child mortality suggests that, interpreted in light of the quantity-quality tradeoff, there was a reduction in the price of child quality that dominated any reduction in the price of child quantity. Reductions in maternal mortality reduced the likelihood that women were childless. For an interquartile shift in pneumonia mortality (-0.26), there were 0.013 fewer births for the average woman for every additional year of sulfa; the average number of years of sulfa in this sample is 20, so that the average woman had 0.25 fewer births overall, which is 15% of baseline. Conditional upon having at least one birth, women had 0.18 fewer births for mean levels of exposure (which is 7% of the conditional mean, which was 2.6). A similar calculation for childlessness implies a 0.23 percentage point increase in the probability of childlessness for an additional year of sulfa exposure, and a 4.6 percentage point increase in the probability of childlessness for the mean duration of exposure (13% of the baseline rate of childlessness, which was 36%).

For an interquartile shift in maternal mortality (-1.84), the average woman had 0.12 more births overall, 0.03 more births on the intensive margin and a 3.3 percentage point reduction in the probability of being childless (the latter being 9% of the baseline mean). The impact on total fertility is somewhat smaller than that estimated in Albanesi and Olivetti (2014), who find that a decline in maternal mortality of 1 death per 1000 live births (roughly half of the 1.84 drop we use) is associated with a rise in completed fertility of 0.27 children per married woman (roughly double that of the 0.12 increase that we estimate).

Heterogeneity in Impacts by Race and Education

Fertility responses to pneumonia mortality are statistically significant for white and black women

and the coefficients are not significantly different from each other (Table 17 in Online Appendix C). Responses to maternal mortality are significantly larger for black women, for whom we see both intensive and extensive margin increases in fertility. For white women, we see only an extensive margin increase, and of a smaller magnitude. Indeed, blacks were exposed to higher maternal mortality rates pre-sulfa, and may have benefited more (in terms of reduced maternal mortality) from sulfa drugs in combination with medical intervention in childbirth, than whites (Thomasson and Treber 2008).

Turning to education gradients in impacts of the sulfa innovation, the total fertility response to pneumonia decline for women with college is not significantly different from the response for women who dropped out of high school. However, the intensive margin decline in fertility is larger for women with some college, and the extensive margin decline (the increase in childlessness) is larger, and in fact only statistically significant, for high school dropouts (Table 18). The latter result is consistent with the predictions in Baudin, de la Croix, and Gobbi (2017), where a decline in infant mortality reduces the attractiveness of low educated women on the marriage market because their expected fertility conditional on marriage increases, which results in increasing childlessness in this group. However, when we estimate impacts of the sulfa revolution on marital status in Section 6 below, we are unable to identify a difference in marriage responses by the education level of the woman, suggesting that the mechanism driving this relationship in our study is different.

Impacts on the Distribution of Fertility

When the probability of childlessness changes, this will by definition change intensive margin fertility. For instance, if women who would otherwise have had one child switch to having zero, then an increase in childlessness will be mirrored in a decrease in the share of women with one child. So as to estimate changes in the distribution of fertility, we define indicator variables for the number of children a woman had: 0, 1, 2, 3 and 4+. Figures 10 and 11 plot the coefficients on the pneumonia and maternal mortality exposure terms from these five separate regressions. The pattern of results indicates a leftward shift of the fertility distribution in response to reductions in pneumonia mortality, with statistically significant responses at the two ends of the distribution: women were more likely to be childless and less likely to have three, four or more children. There was no significant change in the share of women with either one or two children. In contrast, the reduction in maternal mortality resulted in a rightward shift of the fertility distribution. Although only the coefficient on childlessness is statistically significant, the other coefficients indicate that families of zero or one children became less common, while families of two or more became more common.

5.2.2 Completed Net Fertility

Retaining the net fertility measure, we re-estimate the model on a sample of women who had completed their fertility at the time of census enumeration. The purpose of doing this is to identify impacts on the overall number of children at the end of women's reproductive careers, purging any effects of delay. We select women aged 40-50 at census, imposing an upper age limit to minimise any

bias due to the omission of children who have left home.[33] Table 6 shows these results, where the estimated coefficients are smaller than those for the younger sample, but still statistically significant. Assuming that the coefficients now capture changes in fertility targets only, a comparison of these estimates with those for childbearing women suggests that two thirds of the estimated impact of pneumonia mortality on childlessness in the young sample was due to delay, and one third due to an actual rise in childlessness, which we estimate as 1.4 percentage points for an interquartile shift in pneumonia mortality. Similarly, the completed fertility estimates indicate that women had 0.11 fewer children on average in response to the reduction in pneumonia mortality, which is also approximately one third of the corresponding 0.25 fewer children that we estimated using the childbearing sample.

5.2.3 Completed Gross Fertility

We estimate the impact of sulfa exposure on gross fertility, using a different variable in the census that asks women to report the total number of live births. This measure is not sensitive to the potential underestimation of net fertility from children who have moved out. We report the estimated effect on completed gross fertility: the estimated effect of mortality reductions (Table 7) is very similar to the estimates for completed net fertility (Table 6), bolstering confidence in the results. An interquartile shift in pneumonia mortality (0.26), evaluated at the mean number of sulfa years in this sample (14.9), led to 0.08 fewer total births for the average woman, which is 3% of the baseline mean, and a 0.8 percentage point increase in the probability of being childless, which is 4.3% of the baseline mean. The coefficients for maternal mortality are similar to those for completed net fertility but are imprecisely determined.

Heterogeneity of Impacts by Race and Education

Gross fertility is responsive to pneumonia mortality decline in both education groups (Table 18). For total, intensive and extensive margin fertility, the coefficients are larger for high school dropouts but not significantly different to those for women with some college.[34] Although the average impact of maternal mortality is imprecisely estimated, maternal mortality decline had a statistically significant negative effect on childlessness among the college educated. This is consistent with women with college having a higher opportunity cost of childbearing (Baudin, de la Croix, and Gobbi 2015), and similar to the estimates in Albanesi and Olivetti (2014), who also find that a decline in maternal mortality only impacted childlessness among the college educated.

5.2.4 Discussion

Overall, we find that women delayed childbearing and reduced fertility in response to the decline in child mortality, with an increase in the share of childless women and a decrease in the share of

[33] We will underestimate fertility because among this older sample of women, children will be older and some will have left home. These effects are lower bounds.

[34] The increase in childlessness is only significant for high school dropouts, but the coefficient for college women is almost identical.

women with three or more children. Thus, there was a change in timing as well as a change in the desired number of children. The main results are similar irrespective of whether we use net or gross fertility, and are not sensitive to the age of the woman at the date at which fertility is assessed (also see the robustness checks on age at census enumeration in Online Appendix D).

Taking stock, consider first responses to maternal mortality decline. In general, the evidence is of increases in fertility at both the extensive and intensive margins, consistent with maternal mortality decline lowering the cost of birth. Although the responses are statistically significant at both margins in certain population groups, the average coefficient is only significant for the extensive margin and when we consider the net fertility measure. Pneumonia mortality decline had opposing effects, leading to lower fertility on both margins. The decline in intensive margin fertility when we use the gross measure may in part be mechanical, as gross fertility is increasing in the survival rate of children. However, the intensive margin response when we use the net fertility measure is necessarily behavioral, reflecting a quantity-quality tradeoff response to reduced child morbidity, or replacement or hoarding responses to reduced child mortality.

As discussed in the Introduction, the increase in childlessness in response to pneumonia mortality decline is more challenging to explain given the prior that when children become healthier, women should be more likely to have at least one child. We shall argue that fertility timing is key. Child mortality decline allowed women to delay fertility, and some of those who incurred delay ended up childless even if this was not their initial intention. Indeed, the lifecycle behavior of the cohorts of women in the samples we analyse has been described by Goldin (2004) as "job then family". Interestingly, this mechanism of delay and possibly unintended childlessness has been described to in women born in a more recent set of birth cohorts, namely those born between 1966-1979, who deferred marriage and fertility to pursue their careers, leading to increased childlessness (Goldin 2004). In addition, a survey of women entering college in 1976 indicates that while most accurately predicted their future working lives, many mis-forecasted their fertility, with 82% expecting to have children by age 37, and only 69% doing so (Goldin 2006). Thus, it is plausible that such behavior marked the sulfa cohorts that we analyse.

The mechanism is similar to that driving impacts of the birth control pill on women's choices, namely as an improved technology for fertility control, allowing women to delay fertility and pursue their careers. A reduction in child mortality acts similarly, reducing the uncertainty around achieving fertility targets, and hence reducing the number of years dedicated to childbearing. This enables women to initiate fertility later, other things equal. However, women who delay fertility and enter the labor market are potentially exposed to shocks that may eventually result in childlessness. In fact, the birth control pill increased fertility delay and labor force participation but did not have an impact on completed fertility (Bailey 2006, Ananat and Hungerman 2012). We estimate that the reduction in child mortality both increased fertility delay and had an impact on lifetime fertility. A potential explanation for this is that women in the sulfa era (who were born much earlier than women of the pill era) had to choose between family and career. It was typical for women to leave their jobs when they became pregnant with their first child (Goldin 2004). In

contrast, a non-negligible proportion (13-18%) of women who grew up during the spread of the birth control pill achieved what Goldin terms as "career and family" by age forty. We model these ideas and test their predictions for labor force participation and marriage in Section 6 below.

5.3 Robustness checks

We have already noted that the estimates for pneumonia mortality decline are robust to the inclusion of fixed effects for the age and birth state of the mother, maternal education and race, state and year varying macroeconomic factors, communicable and non-communicable diseases that were not treatable with sulfa drugs, and census division-year fixed effects. The estimates for maternal mortality decline are more sensitive to specification but are consistently positive, and we estimate a significant positive coefficient on the extensive margin response. The pattern of results is consistent across the hazard and stock models, is similar irrespective of whether we use net or gross measures of fertility, and is not sensitive to changing the precise cohorts of women included in the sample. This stability strengthens confidence in the results. In this Section, we discuss additional specification and data checks. The tables showing these estimates are found in Online Appendix D, which also shows and discusses robustness to further checks, including alternative sample definitions, adjustment of standard errors for multiple hypothesis testing, and exclusion of outlier states.

Confounding Factors

Inference with our research strategy relies on getting the timing of the introduction of antibiotics right. For this we rely on documentary evidence that the advent of sulfa drugs was publicized, for example in a *New York Times* article in December 1936 ("Conquering Streptococci"), and that historians have documented widespread uptake in 1937 (Lesch 2007). The event study plots discussed earlier are consistent with this, as they show a fairly flat profile of coefficients up until 1937, after which there is a dip associated with pneumonia decline and a jump associated with maternal mortality decline. Still, we may be concerned that these changes were driven by a coincident event rather than by the arrival of sulfa drugs. We therefore investigate the robustness of our findings to accounting for events that occurred around the time of the sulfa intervention: the New Deal program, the Second World War, and the Dust Bowl. The New Deal was a government-funded program of spending and loans that aimed to tackle the effects of the Great Depression. We accessed data on state-year variation in New Deal spending as a proxy for both the severity of the Great Depression and the subsequent inflow of funds (Fishback, Kantor, and Wallis 2003). We include this variable as a control, interacted with the post-1937 dummy in the hazard model and with years of exposure to sulfa in the stock model (Table 23 and Panel A, Table 24, respectively). The coefficients of interest are robust to this.

The United States entered the Second World War in December 1941, following which there was initially a sharp drop in fertility and then a substantial increase in birth rates in the post-war period (the Baby Boom). To account for state-level differences in the dynamics of fertility in this period, we control for state-level troop deployment, obtained from Goldin and Olivetti (2013), interacted with individual-level exposure to WW2, measured as the number of fertile years of the woman from

1942 onwards. In the hazard model, we restrict the sample to births occurring before 1942. Our substantive findings remain robust (Table 23 and Panel B, Table 24).

We also estimate our models excluding states most affected by the Dust Bowl (Nebraska, Kansas, Colorado, New Mexico, Oklahoma and Texas).[35] The results are, if anything, strengthened by the omission of the Dust Bowl states (Table 23 and Panel C, Table 24).

Sulfa drugs were available without prescription until 1939. Our findings are similar when we exclude all births after prescription was introduced (Table 39 in Online Appendix D).

Omitted Trends

A common concern with the statistical approach we adopt is that there may be omitted trends that drive the findings. In the case of the sulfa innovation, the concern would be that the outcome (birth) may have diverged between high and low disease burden states after 1937 for reasons unrelated to sulfa drugs. One contender is mean reversion, which we account for by controlling for the state-level average value of the outcome variable, calculated over 1900-1930 censuses and interacted with sulfa years in the stock model, and calculated for the years 1930-1936 and interacted with the post-1937 dummy in the hazard model. Table 23 and Panel D, Table 24 show that the estimated coefficients are similar to the main results. We also estimate a specification with a "placebo" intervention using the stock model. In particular, we assume that the invention of antibiotics happened forty years previous to the actual year (1897), so that no woman in the new sample that we create was directly treated by the real sulfa innovation during her fertile period. We then estimate the equations for fertility outcomes using the usual specification, with the fake intervention year (this is a similar approach to the "placebo" check in Aaronson, Lange, and Mazumder 2014). The results in Panel E of Table 25 show that the coefficients on pneumonia and the maternal mortality rate are small and insignificant.

Using the hazard sample, we estimate whether there were pre-trends in birth outcomes by regressing the probability of birth in the pre-sulfa era, 1930-1936, on a linear time trend interacted with a dummy variable equal to one for states with above median mortality, and zero otherwise. The results, in Table 27, show no evidence of differential pre-trends.[36]

In the hazard model, identification is relatively clean because the flow of births is analysed with reference to the discrete event of the introduction of sulfa drugs. However, in the stock model, individual years of exposure evolve linearly within a woman and there is more scope for interference by omitted trends in cohort of birth. To investigate this, we redefine the treatment variables in the stock model to be binary, comparing the fully treated with the untreated, and omitting partially treated women from the sample. The binary treatment variable is interacted with the pre-intervention mortality rates. The results are consistent with the main findings (Table 26), particularly when considering net fertility. When we use the gross measure instead, the coefficients are of the expected signs but are not precisely determined.

[35] The Dust Bowl was a period of drought and severe dust storms during the 1930s that damaged agriculture in several southern U.S. states and resulted in large out-migration from those states.

[36] In the hazard specification, Figures 8 and 9 showed a trend break in the probability of birth in 1937 and not elsewhere, which is similar to a "placebo" check.

Under-Reporting and Measurement Error

For reasons detailed in Section 4, we use the all-age pneumonia and influenza mortality rate in place of child pneumonia mortality. To assess the sensitivity of our estimates to this choice, we used the under-5 mortality rate from pneumonia and influenza instead. The results are in Table 23 and Panel F, Table 25, and they indicate that the main results are similar although the coefficients are reduced in magnitude, suggesting attenuation bias. Consistent with this, the coefficients increase in magnitude and precision when we estimate a 2SLS regression where the under-5 mortality rate is instrumented with the all-age rate used in the main estimates (Table 23 and Panel G, Table 25).

Survivorship Bias

We face the common problem that we only observe survivors; mothers who died as a result of childbirth are not observed in census enumerations, and we do not observe children who die in both measures of fertility. With regard to maternal mortality, it seems plausible to assume that these were high risk women who, on average, had higher fertility, and that they were concentrated in states with higher pre-intervention levels of maternal mortality (which we have seen are the states that showed larger increases in fertility after 1937 in response to maternal mortality decline). It follows that when these women are selected out of the fertility sample, we will tend to overestimate the increase in fertility that flows from the drop in maternal mortality.[37] This bias is mitigated by our controls for observable individual indicators of risk such as the age and education of the woman. For a sense of the extent of selection, see the mortality rate statistics in Table 1.

Now consider biases associated with child mortality. When child mortality is high, the gross measure of fertility (children ever born to a woman) will overestimate surviving births. The net measure only counts children living with the mother, so it partially addresses this problem, but not entirely as children alive at the census date may subsequently die. The extent of overestimation will be correlated with pneumonia mortality, our treatment variable. To check the sensitivity of our estimates to child survival, we exploit the fact that pneumonia mortality rates decline exponentially from birth to age five, after which they flatten out. We re-defined the net measure of fertility to include only children aged at least five (see Panel H of Table 25). The impact of mortality decline on net fertility with children surviving to at least five is similar to the impact on net fertility overall.

Migration

If individuals migrated in response to disease, then the introduction of sulfa, which led to convergence in disease burdens across states, will have influenced migration patterns. In this case, we need to be sure that our findings do not reflect compositional changes. To investigate this, we modelled migration as a function of post-sulfa declines in pneumonia and maternal mortality. First, we defined an indicator for migrants as individuals for whom the birth state is different from the census enumeration state; second, we defined an indicator for migration between 1935 and 1940 using the 1940 census, which records this information. The estimates in Table 28 show no evidence

[37] The positive correlation between fertility and maternal mortality implies that, pre-sulfa, high fertility women were more likely to die. Thus, observed fertility pre-sulfa for high mortality states is underestimated and the rise in fertility that we attribute to a reduction in maternal mortality may be partly due to high fertility women being more likely to survive post-sulfa.

that sulfa-induced changes in mortality rates influenced migration.

6 Fertility, the Labor Market and the Marriage Market

6.1 Fertility Control

The ability to control fertility is a necessary precondition for altering fertility timing. The early 20th century in the U.S. featured the birth control movement, led by political radicals Emma Goldman, Mary Dennett and Margaret Sanger, who argued for the need for birth control among low income women who were burdened by more children than they desired.[38] There is considerable evidence that women born in the early 20th century were able and willing to practise fertility control (Morgan 1991). Before the arrival of the birth control pill in the 1960s, couples used diaphragms, latex condoms, vaginal suppositories, withdrawal and douching techniques (Engelman 2011), with the invention of the diaphragm in 1882 being particularly crucial to the advent of effective fertility control by women.[39] In fact, our results imply fertility control. If we had only observed an increase in childlessness in response to pneumonia mortality decline, this could be attributed to changes in fecundity or marriage rates (as in Baudin, de la Croix, and Gobbi 2017), but we additionally observe a reduction in higher parity births, which is evidence of fertility control.

In 1936, a year before the introduction of sulfa drugs, Popenoe (1936) conducted a survey among students at the University of Southern California asking them to describe the history of all instances of permanently childless couples that they knew. Based on these histories, he categorised reasons for childlessness; see Table 8. Although not a representative survey, it gives some indication of possible reasons for childlessness during the 1930s among married women.[40] In particular, after "self-centred", the most common responses were "wife's career" and "economic pressure", suggesting that fertility and labor market choices were linked. Several women were reported to prefer working in order to keep their "freedom" (p.470).

We shall argue that the decision to delay fertility and enter (or remain in) the labor market can explain our somewhat unexpected finding that childlessness increases with reductions in child mortality. In the next Section, we outline a dynamic model of fertility and labor market choices that shows the assumptions under which reductions in child mortality can lead to childlessness.

[38] Sanger was particularly active and opened the first birth control clinic in 1916, which was shut down, followed by the second birth control clinic in 1923, which was not shut down. These birth clinics were the precursor to Planned Parenthood. She is considered the founder of the modern birth control movement.

[39] The relationship between fertility control and career choices has been discussed in sociology, for example see Wilkie (1981), Hayford (2013), Lundquist, Budig, Curtis, and Teachman (2009), Bloom and Trussell (1984). In particular, see Murray and Lagger (2001), who analyses this in the context of the United States demographic transition in the 19th century.

[40] Popenoe oversampled wealthier and more educated women, as these were more likely to be known to the students of the university.

6.2 A Model of Fertility and Labor Market Choices

6.2.1 The quality-quantity model

We begin by considering the canonical quality-quantity model of fertility, following Becker and Lewis (1973) and Galor (2012). A woman derives utility $U(c, n, e)$ from consuming c and having n children with quality e. To simplify the exposition, we assume that utility is quasilinear, with

$$U(c, n, e) = u(n, e) + c,$$

where $u(0, e) = 0$.[41] Her budget constraint is

$$n(\tau^q + \tau^e e) + c \leq I, \tag{3}$$

where I denotes her lifetime income, τ^q is the price of quantity, and τ^e is the per-child price of quality. The maximized value of having at least one child is

$$\max_{c,e,n\geq 1} U(c, n, e) \text{ subject to (3)}.$$

Aaronson, Lange, and Mazumder (2014) point out that this is a decreasing function of prices $\tau = (\tau^q, \tau^e)$. Meanwhile, the value of being childless is simply $U(I, 0, 0)$ and therefore independent of prices. A corollary of these observations is that, other things equal, the number of women who choose to remain childless is *increasing* in the prices of child quantity and quality, since higher prices make childbearing less attractive.

The introduction of sulfa drugs reduced both the mortality and morbidity of children. In terms of the current framework, it seems likely that this change *lowered* the cost of child quality (and quantity). Therefore, the quality-quantity model does not yield unambiguous predictions of changes in fertility on the intensive margin. However, our empirical results indicate that fertility on the intensive margin declined in response to reductions in child mortality, suggesting that the reduction in the price of child quality dominated the reduction in the price of child quantity.

The model also predicts that the introduction of sulfa drugs would lead to decreased childlessness (if reduced mortality and morbidity lower the prices τ of quality or quantity). In addition to price effects, a decrease in child mortality reduces the probability that a woman decides to have children ($n > 0$) but ends up childless due to the death of her child. This mechanical effect, too, leads to decreased childlessness in response to exposure to sulfa drugs.

Our results indicate, by contrast, that childlessness increased in response to the introduction of sulfa drugs. This finding does not necessarily constitute a contradiction of Aaronson et al.'s

[41] The effect of assuming quasilinear utility is to remove an income effect where a rise in income increases the demand for both quality and quantity, generating a positive relationship between income and the number of children. Becker and Lewis (1973) argue that the income elasticity of quality is much higher than the income elasticity of quantity, such that these income effects do not arise in practise. This means that we also do not have the effect in Baudin, de la Croix, and Gobbi (2015), where an increase in education (income) moves a woman down the U-shape, away from childlessness caused by poverty.

empirical findings, or of the quality-quantity trade-off. Rather, it suggests an offsetting effect of sulfa drugs, which encouraged childlessness and dominated the effects of reduced prices and the mechanical effects of higher survival rates.

6.2.2 Dynamic fertility choices

We now consider a dynamic model of fertility choices, which gives rise to offsetting effects in response to a decrease in child mortality. We consider a woman whose preferences and budget constraint are identical to the quality-quantity model above, but whose decisions about her income-earning activity and fertility evolve over time.

The woman is fertile at dates $t = 1, ..., T$. Each period she has one chance to get pregnant. Pregnancy leads to a surviving child with probability $1 - \lambda$, where λ is child mortality. The woman can work up to the date t_0 of her first pregnancy, consistent with the "job then family" lifecycle pattern of cohorts exposed to sulfa drugs (Goldin 2004). If she never gets pregnant, she can work up to T.[42] Her initial wages are y. Each period she has the potential to get promoted with probability p, and in this event her wages rise from y to $Y > y$. She can only be promoted once. This mechanism captures a positive labor market shock, which can eventually result in childlessness if a woman decides not to get pregnant after a favorable labor market outcome. Fertility delay motivated by potential benefit from returns on the labor market is similarly a feature of the model in Goldin and Katz (2002), where the birth control pill serves as an effective technology for women to delay fertility and marriage and invest in their human capital.

The woman's lifetime income is

$$I = \sum_{t=1}^{t_0-1} \tilde{y}_t,$$

where $\tilde{y}_t \in \{y, Y\}$ denotes the realized wage each period.

At time T, when fertility is completed and the number n of children is known, the woman chooses the quality e of her children and her consumption c, to solve the problem

$$\max_{c,e} U(c, n, e) \text{ subject to (3)}.$$

Let $e^\star(n; \tau)$ be the optimal choice of quality when the woman has had n children and prices are $\tau = (\tau^e, \tau^q)$. Substituting the binding budget constraint yields the woman's maximized utility, conditional on having n children, as

$$\sum_{t=1}^{t_0} \tilde{y}_t + V(n; \tau),$$

where

$$V(n; \tau) = u\left(n, e^\star(n; \tau)\right) - n\left(\tau^q + \tau^e e^\star(n; \tau)\right)$$

is the indirect utility from having n children when prices are τ. By a parallel argument to Aaronson,

[42] To save notation, we do not allow women to work after their fertile period, but this assumption does not affect our qualitative results.

Lange, and Mazumder (2014), we can show that the value $V(n;\tau)$ of having $n \geq 1$ children is strictly *decreasing* in the prices of quantity and quality.

In line with our argument above, the (negative) effect of sulfa drugs on prices is, on its own, unable to explain the increase in childlessness that we identify. Moreover, holding constant the timing of pregnancy t_0, it is clear that a decrease in child mortality (an increase in λ) mechanically increases the number of successful pregnancies, and therefore reduces the number of childless women.

To understand the added effect of endogenous fertility timing, we consider the simplest case where the woman is fertile for $T = 2$ periods. We focus on the extensive margin by assuming that $u(1, e) > u(n, e)$ for all $n > 1$. Under this condition, it is optimal to have at most one child. We write $V(\tau) = V(1, \tau)$ for simplicity. We further normalize the pre-promotion wage to $y = 0$.

To solve the model, it is useful to separate three parametric cases. First, if $V(\tau) < 0$, it is never optimal to get pregnant, and the woman remains childless by definition. Second, if $Y < (1 - \lambda)V(\tau)$, it is never optimal to enter the labor market, because even promotion cannot lead to an outcome that dominates getting pregnant. The interesting case is the intermediate region with $0 < V(\tau) < Y/(1 - \lambda)$.

6.2.3 The trade-off between early and late pregnancy

We focus on the intermediate parametric region. It is optimal to remain in the labor market, and thus to remain childless, if the woman gets promoted at $t = 2$. The key trade-off is between (i) getting pregnant at $t = 1$ and (ii) entering the labor market for one period and waiting until $t = 2$ to attempt childbearing if promotion does not occur.

Getting pregnant at date $t = 1$ gives utility

$$\begin{aligned} V(\tau) \times Pr\left[\text{one surviving child}|t = 1\right] &= V(\tau) \times \left[1 - \lambda^2\right] \\ &= V(\tau)(1 - \lambda)(1 + \lambda). \end{aligned}$$

This is because the woman earns no wages in this case, and the probability of having one successful pregnancy out of two attempts is $1 - \lambda^2$.

Waiting until $t = 2$ gives expected utility

$$pY + (1 - p)V(\tau) \times Pr\left[\text{one surviving child}|t = 2\right] = pY + (1 - p)V(\tau)(1 - \lambda).$$

This is because the woman gets promoted, and optimally remains childless, with probability p, and the probability of having one successful pregnancy now falls to $1 - \lambda$ if she does not get promoted (and gets pregnant at $t = 2$).

It pays to delay pregnancy and enter the labour market at $t = 1$, instead of getting pregnant

early, if and only if the net value of waiting, $N(\tau, \lambda)$, is greater than zero, where

$$\begin{aligned} N(\tau,\lambda) &= pY - (1-\lambda)(p+\lambda)V(\tau) \\ &= \underbrace{p\left[Y - V(\tau)(1-\lambda)\right]}_{\text{option value of delay}} - \underbrace{V(\tau)\lambda(1-\lambda)}_{\text{insurance value of early pregnancy}}. \end{aligned}$$

The option value of delay measures the expected utility gain from getting promoted. It is increasing in λ: the option of working for high wages is more valuable, relative to attempting a pregnancy, if child mortality is high. The insurance value of early pregnancy measures the expected utility gain from having a second chance: with probability λ, the first pregnancy does not survive, but the second survives with probability $1-\lambda$. The second chance thus adds value V with probability $\lambda(1-\lambda)$. The insurance value is a hump-shaped function of λ with its peak at $\lambda = 1/2$. Intuitively, when $\lambda \simeq 0$, the first pregnancy almost never fails, so the insurance value is low. When $\lambda \simeq 1$, both pregnancies almost always fail, so again the insurance value is low. The insurance value is therefore highest for intermediate levels of λ.

Similar intuition applies if we consider other sources of new information that can lead to persistence of the childless state, conditional on fertility delay. We sketch the intuition here. First, consider learning about the future benefits of work (Fernández 2013). In our framework, a simple way to model this is to suppose that with a certain probability, the woman learns at the end of the first period that her utility from work in the second period will be higher than her current utility (if this does not happen, then her utility from work in the second period stays the same). This is similar to the effect of job promotion, because the crucial factor that affects a woman's decision to delay childbearing is the relative (expected) utility from work compared to the (expected) utility from childbearing immediately. The learning effect will encourage delay, and especially so when the probability of learning is high, because a woman's utility from delay is increasing in her expected utility from work in the second period.

Second, consider a change in fertility preferences. Suppose that with probability q, V falls to $v < V$ in the second period, and $v < 0$, so that a woman never wishes to have a child if her fertility preferences fall. This has the same implications as a rise in income from y to Y, with delay leading to more childless women because some of them experience a reduction in the utility from childbearing relative to the utility from working. Third, we can model fecundity similarly to child mortality, as a success rate of pregnancy. However, the important difference to child mortality is that the success rate declines in the second period because the woman is older, either for everyone or only for some women. Let us assume that fecundity declines deterministically for all women, and the probability of a pregnancy failing due to reduced fecundity is θ. Then, the probability of a successful pregnancy in the first period is $(1-\lambda)$ while the same probability in the second period is $(1-\lambda)(1-\theta)$. This means that any woman who delays has a higher probability of a failed pregnancy in the second period, relative to the first period. This raises the proportion of childless women among those women who delay.

We have restricted the analysis to the extensive margin response. We pointed out in Section

6.2.1 that on the intensive margin, reductions in the price of child quality that dominate reductions in the price of child quantity will reduce the desired number of children, n^*. Allowing for an intensive margin choice in the model would mean that such a reduction would be an additional reason to delay, as lowering n^* reduces the insurance value of early pregnancy (fewer successful pregnancies are needed to achieve the target number of children). In the empirical results, we showed that reductions in child mortality led to reduced fertility on the intensive margin, suggesting that women reduced their fertility targets; therefore, this effect may have also contributed to increased fertility delay in response to lower child mortality.

6.2.4 Population effects of decreased child mortality

Suppose there is a population of women $i \in [0,1]$, distributed according to a probability measure μ, who differ in their potential income Y^i and their utility $V^i(\tau)$ of having a child. Both Y^i and $V^i(\tau)$ are measurable functions of i. For simplicity, we assume that the probability p of promotion is constant across the population.

The fraction of women who never wish to get pregnant is

$$\eta(\tau) = \int\limits_{i:V^i(\tau)<0} d\mu.$$

Note that this proportion does not directly depend on the child survival rate λ, because the value $V^i(\tau)$ of having one surviving child is fully determined by the prices τ of child quality and quantity.

From the previous subsection, it is clear that woman i delays pregnancy if and only if $N^i(\tau,\lambda) = pY^i - (1-\lambda)(p+\lambda)V^i(\tau) > 0$. The fraction of women for whom this is the case is equal to

$$\delta(\lambda,\tau) = \int\limits_{i:N^i(\tau)>0} d\mu$$

This proportion does depend on λ, because the probability of child survival affects the trade-off between early and late pregnancy.

For women who delay pregnancy until $t=2$, the probability of having one child is $(1-p)(1-\lambda)$. For women who get pregnant at $t=1$, the probability of having one child is $1-\lambda^2$. Integrating over the population, the proportion of women who have one child is therefore

$$\begin{aligned} C(\lambda,\tau) &= \delta(\lambda,\tau)(1-p)(1-\lambda) + (1-\delta(\lambda,\tau)-\eta(\tau))(1-\lambda^2) \\ &= 1-\lambda^2 - \delta(\lambda,\tau)(1-\lambda)(p+\lambda) - \eta(\tau)(1-\lambda^2). \end{aligned}$$

The baseline probability of having a child if not delaying is $1-\lambda^2$, and this is reduced in proportion to $\delta(\lambda,\tau)$ by the women who delay, and in proportion to $\eta(\tau)$ by the women who never get pregnant.

We now consider the impact of a shock, designed to represent the introduction of sulfa drugs, which decreases child mortality (lowers λ), and potentially decreases the prices τ of child quality and

quantity. The total population effect of this shock on fertility is $-\frac{dC(\lambda,\tau)}{d\lambda}$. This can be decomposed into the direct effect of λ and the indirect effect through prices τ:

$$-\frac{dC(\lambda,\tau)}{d\lambda} = -\frac{\partial C(\lambda,\tau)}{\partial \lambda} + \frac{\partial C(\lambda,\tau)}{\partial \tau} \cdot \left(-\frac{d\tau}{d\lambda}\right).$$

The price effect (the second term) is always positive. To see this, note that

$$\frac{\partial C(\lambda,\tau)}{\partial \tau} = \left(-\frac{\partial \delta(\lambda,\tau)}{\partial \tau}\right)(1-\lambda)(p+\lambda) + \left(-\frac{\partial \eta(\tau)}{\partial \tau}\right)(1-\lambda^2).$$

Since a fall in prices increases the value of getting pregnant, and decreases the net value of delay, it is easy to see that $-\frac{\partial \delta(\lambda,\tau)}{\partial \tau} < 0$ and $-\frac{\partial \eta(\tau)}{\partial \tau} < 0$. It follows that $\frac{\partial C(\lambda,\tau)}{\partial \tau} < 0$. Then, as long as the prices of quality and quantity are non-increasing in response to decreased mortality (we expect that they decreased), we have $-\frac{d\tau}{d\lambda} \leq 0$ and

$$\frac{\partial C(\lambda,\tau)}{\partial \tau} \cdot \left(-\frac{d\tau}{d\lambda}\right) > 0.$$

Thus, the price effect leads to higher fertility and fewer childless women. This formalizes our earlier intuition that, in line with Aaronson, Lange, and Mazumder (2014), price effects in response to sulfa drugs are unlikely to explain the increased childlessness that we find in the data.

The direct effect of decreased mortality on fertility can be further decomposed as

$$-\frac{\partial C(\lambda,\tau)}{\partial \lambda} = \underbrace{2\lambda(1-\delta(\lambda,\tau)-\eta(\tau)) + (1-p)\delta(\lambda,\tau)}_{\text{mechanical effect}} - \underbrace{(1-\lambda)(p+\lambda)\left[-\frac{d\delta(\lambda,\tau)}{d\lambda}\right]}_{\text{behavioral effect}}.$$

The mechanical effect is positive. As discussed above, more pregnancies are successful, so there are more children. However, the behavioral effect can be negative, and offset the mechanical and price effects, if more women delay in response to the change, that is if $-\frac{d\delta(\lambda,\tau)}{d\lambda} > 0$. It is easy to see that this effect has the same sign as the effect on the marginal woman's incentives:

$$-\frac{d\delta(\lambda,\tau)}{d\lambda} \overset{\text{sign}}{=} -\frac{dN(\lambda,\tau)^i}{d\lambda}\bigg|_{N^i=0} = V^i\left(1-2\lambda-p\right).$$

Then the behavioral effect is negative if and only if

$$\lambda < \frac{1-p}{2}. \tag{4}$$

Under this condition, it is possible for the behavioral effect to offset the mechanical and price effects, and if the behavioral effect is large enough, we will see more childless women when child mortality declines. The condition says that a decrease in child mortality (e.g. via sulfa drugs) encourages delay if child mortality $\lambda < \frac{1}{2}$ and the probability p of getting promoted is also low enough. How can we interpret this? Suppose that λ falls. Recall that the insurance value of early pregnancy,

$V(\tau)\lambda(1-\lambda)$, is a hump-shaped function of λ with a maximum at $\lambda = \frac{1}{2}$. If λ is higher than $\frac{1}{2}$, then the insurance value of getting pregnant increases with a marginal fall in child mortality, which discourages delay. In contrast, when λ is less than $\frac{1}{2}$, then the insurance value declines in response to this marginal fall in λ, which encourages delay. Therefore, $\lambda < \frac{1}{2}$ is required. Second, the option value of delay, $p[Y - V(1-\lambda)]$, declines by amount $-pV$ when λ declines. A small enough p ensures that this decline in the option value of delay, which reduces the net value of delay $N(\lambda, \tau)$, does not dominate the decline in the insurance value, which increases the net value of delay, so that on balance the net value of delay increases in response to the decline in λ. A population average child mortality rate of 47 per 1000 live births in 1939 (Dowell, Kupronis, Zell, and Shay 2000) implies that $\lambda = \frac{47}{1000}$, and the condition is satisfied if the probability of promotion is less than 90.6%, which seems plausible.[43]

Figure 12 shows the population effect of a decrease in λ when Equation (4) holds. The solid line delineates the two groups of women: those above the line prefer to delay fertility and enter the labor market, while those below the line prefer to start childbearing in the first period. The line is given by the condition in (4). When child mortality falls, the solid line shifts down to the dotted line. More women prefer to delay instead of childbearing in the first period. Switchers are those women who are close to indifferent between earning income Y and enjoying utility from childbearing V. The shaded area to the left of the y-axis depicts women with $V < 0$, who always prefer to work instead of having a child.

An interesting question is whether switching is more likely to occur among women with high potential income or low potential income. Income is likely to be correlated with education so this gives an indication of how responses to sulfa exposure may vary with education levels. The predictions of the model in this regard are ambiguous. Indeed, it is clear from the figure that women with low Y are switchers if they have a low utility V from childbearing, and women with high Y are switchers if they have high V. Thus, the relationship between switchers and income is determined by the joint distribution of Y and V in the population. For example, we would expect increased delay for all income levels if Y and V are positively correlated; if they are strictly negatively correlated, switchers will only be observed for intermediate income levels.[44]

Childlessness in our model is *voluntary*: women make lifecycle decisions about labor force participation and childbearing based on their preferences over children and their returns on the labor market, and switchers can theoretically occur at any point of the education distribution. Baudin, de la Croix, and Gobbi (2015) argue that childlessness at the top end of the education distribution is voluntary and occurs due to a high opportunity cost of childbearing, while childlessness at the

[43] It is relevant to note that women in this era primarily worked in teaching and typing jobs, where the probability of promotion was low (Goldin 2004).

[44] We can also extend the model to take into account a non-constant probability of promotion, p. In particular, if we allow p to depend on Y, then the slope of the solid (indifference) line in the figure will depend on whether the derivative of $p(Y)$ with respect to Y is positive or negative. If the derivative is positive, then the indifference line will be flatter, and there are more women in the population who delay. This is because the return to delay has increased for a given Y, as the probability of achieving the promotion in the second period is higher. In contrast, if p is decreasing with Y (a less realistic assumption), then the indifference line will be steeper.

low end of the education distribution occurs due to a lack of resources needed to afford children, which can be interpreted as being involuntary. Our framework treats women at the two ends of the education distribution similarly, and so provides a very different perspective on childlessness at the lower end. Moreover, the new mechanism we propose is that child mortality reductions encourage fertility delay, and changes in labor market opportunities for women (promotion in our example) are just one of a number of shocks that can result in eventual childlessness (others being fecundity or fertility preferences, for instance). In contrast, in Baudin, de la Croix, and Gobbi (2015), improved labor market opportunities may directly lead women to choose career over children, similar to the mechanism in the second period of our model conditional on delay and promotion, when a woman rationally chooses to keep working because the opportunity cost is too high.

Note that the quality-quantity trade-off, and the effect of sulfa drugs through prices τ, can also be understood intuitively in terms of Figure 12. If sulfa drugs cause a decrease in τ^q and/or τ^e, the value V^i of having a child increases for each woman, and the distribution of V in the population shifts to the right (in the sense of first-order stochastic dominance). Therefore, a group of women move from the "never pregnant" to the "delay" region, and another group of women move from "delay" to "rush". Both groups are now less likely to remain childless, which translates to an overall decrease in childlessness in the population.

Our model abstracts away from education decisions. The intuition for investment in human capital is in principle similar to that for decisions about work: women may delay fertility to extend their education and then enter the labor market, similar to the mechanism in Goldin and Katz (2002). A large fraction of the women in our data had completed their education by the time sulfa arrived (74% in the hazard sample, 68% in the completed fertility stock sample and 37% in the uncompleted fertility stock sample).[45] Restricting the sample to those that had not completed their education (for example, those aged under 21 in 1937), would result in a sample of women with little variation in sulfa exposure, making it more difficult to identify the effect of sulfa drugs on education decisions. We nevertheless investigated this by creating a sample of women aged 15 to 25 in 1937, and defining exposure to sulfa drugs as being aged 20 or under in 1937. We find no evidence that exposure to sulfa drugs led to higher investment in college, though we do find a 5.4 percentage point increase in the probability of high school completion (see Online Appendix E, Table 43). In our sample, childlessness is higher among college-educated women and high school dropouts than among women with high school. Hence, human capital accumulation does not appear to be the relevant pathway here. To confirm this, we re-estimated the main equations for fertility, and the labor market and marriage outcomes to be discussed shortly, using a sample of women who were at least 21 (so that they had completed education choices) when sulfa drugs arrived, and the pattern of results is very similar (Online Appendix E, Tables 44-47).[46]

Next, we estimate the impact of sulfa exposure on women's labor and marriage market outcomes

[45] Recall that here we are focused on outcomes for women who were of reproductive age when sulfa drugs were introduced, and primarily on their fertility responses. Bhalotra and Venkataramani (2012) document that the children of these women, who were born into an environment with sulfa drugs, grew up to acquire more education.

[46] In addition, the estimates are not sensitive to the removal of woman's education fixed effects as a control variable.

in the data, and in so doing provide further evidence for the behavioral channel of fertility delay illustrated in the model.

6.3 Labor Market Choices and Sulfa Exposure

The 1930s was a period of growing labor force participation among women and the largest change occurred on the extensive margin among married women, with an increase of 15.5 percentage points, from 10% to 25% (Goldin 2006). This was accompanied by a virtual elimination of marriage bars by the early 1940s.[47] In our stock sample of married and unmarried women drawn from the 1940-1970 censuses, the average labor force participation rate was 37%. An important driver of the increase in labor force participation was the arrival of "nice" jobs in offices, which reduced the stigma associated with married women working; however, regardless of education, most working women were engaged in typing-oriented jobs and teaching (Goldin 2006).

To assess the impact of pneumonia and maternal mortality reductions on women's labor market choices, we estimate the stock model (as specified in equation (2)), with several labor market outcomes as dependent variables: whether currently working, whether in the labor force, a measure of the occupational score (the Hauser-Warren Socioeconomic Index; see Online Appendix A for information on how this is constructed), personal income in the last year in U.S. Dollars, and hours worked in the past week. As the census asks women about their current labor market status, we include all women aged 18-50 at the time of the census and 6-44 in 1937, pooling both childbearing women and women aged 40-50, for whom we estimated the effect on completed (net) fertility. Thus, all cohorts 1893-1931 are included. The results are very similar when widening the sample to include women aged up to 60 (and narrowing the sample to women aged under 40; see Online Appendix D).

The results in Table 9 show that reductions in pneumonia mortality led to a greater propensity for women to be in the labor force and working, they worked for more hours per week worked and they had higher occupational scores, a measure of the skill intensity of their occupations. The mean number of years of exposure to sulfa drugs for this sample is 18.3, which implies an increase in the probability of work of 2.8 percentage points and an increase in the probability of being in the labor force of 2.6 percentage points for the average woman. The average proportion of women who report working in this sample is 35.1%, so this increase is 8.0% of the baseline rate. In contrast, the fall in maternal mortality led to a decline in the probability of work of 2.6 percentage points and a decline in labor force participation of 3.8 percentage points. The increase in the occupation-based socioeconomic index is an improvement of 6.6% relative to the baseline index score of 14.4.[48] The reduction in pneumonia mortality is estimated to have increased weekly working hours by 1.15 hours on average for an interquartile shift in the mortality rate, or 9% of baseline; this is an annual increase of 13.8 hours for the sample average of 12 working weeks per year. To put this in

[47] Marriage bars were regulations that prevented married women from working - see Goldin (2006).

[48] We obtain similar results when considering other occupational scores available in the census data, including *occscore* and the Duncan socioeconomic index.

perspective, Bailey (2006) estimates an annual increase of 68 hours among cohorts with access to the birth control pill.

The estimated increase in the probability of work of 8% of the mean is consistent in magnitude with our estimate of the effect of sulfa exposure on childlessness, which was 4.6% of the mean for gross fertility and 12.8% of the mean for net fertility. Similarly, using a logistic survival model, we estimated an increase in the probability of childlessness of 3 percentage points for women exposed to sulfa for ten years, or 5.9% of the baseline mean. The share of women who entered the workforce and remained childless due to pneumonia decline is similar. In other words, the magnitudes are consistent with a high degree of substitution between being employed and becoming a mother.

The fall in maternal mortality had opposing effects. It led to a decline in the probability of work of 2.6 percentage points and a decline in labor force participation of 3.8 percentage points. It reduced working hours by 1.08 hours on average, but had no effect on the occupational score for working women. We find no significant effect of pneumonia or maternal mortality reductions on personal income. Together, these results show that while reductions in pneumonia mortality increased the likelihood that women had careers, reductions in maternal mortality reduced this likelihood.

The Joint Probability of Childlessness and Work

Thus far we have demonstrated, using independent reduced form equations, that the sulfa-led reduction in pneumonia mortality led to lower fertility, higher childlessness and to higher rates of labor force participation and employment among women. We have argued, with reference to a stylized model, that these two results are linked. In order to provide further evidence in support of this, we estimate the impact of mortality decline on the joint probability of being childless and working (Table 11). Reductions in child mortality increased the probability that women were both childless and working by 1.64 percentage points, or 19% of the baseline probability of 8.6% (column (1)). Interestingly, this coefficient on pnuemonia mortality decline is virtually the same as in column (2), which is the estimated effect on the probability of working and not being childless. Thus, pneumonia mortality decline increased the probability of work. Of those women who engaged in the labor market, around half ended up childless, while the other half did not, consistent with childlessness being driven by a woman's experience conditional on working. On the other hand, pneumonia mortality decline reduced the probability of not working and not being childess by 2.9 percentage points, or 5.2% of baseline, and did not change the probability of not working and being childless, indicating that work was the operative channel through which child mortality decline increased childlessness.

We also explore the descriptive relationship between childlessness and labor force participation, tabulating labor market outcomes by childlessness status (Table 12). Childless women were 17 percentage points more likely to be in the labor force, earned over $1000 more per year and worked 7 more hours per week, as well as having a higher occupational score, compared to mothers. We also estimate a descriptive regression of the relationship between labor force participation and childlessness, conditioning on birth cohort, state of birth and race (Table 13). This indicates that

labor force participation is associated with a 16.9 percentage point higher likelihood of childlessness among 18-40 year old women and a 6.7 percentage point higher likelihood among 40-50 year old women.

Heterogeneity in Impacts by Race and Education

We estimate impacts of sulfa exposure on the labor market choices of women, dividing the sample by race and education, the latter polarized into those with some college and those who had not completed high school (Online Appendix C). We find that the labor market impacts of sulfa drugs were concentrated among white women, but were not significantly different by education. This result is instructive for two reasons. First, interpreted in light of our theoretical model, it suggests a positive correlation between Y and V. Second, it suggests that the labor market impacts of sulfa drugs were not driven by WW2 mobilisation, which only affected the labor force participation of women in the upper end of the education distribution (Goldin and Olivetti 2013). On the latter, we have also verified that our results are robust to controlling for Goldin and Olivetti's measure of WW2 mobilisation interacted with WW2 exposure years (see Section 6.5).

6.4 Marriage Market Choices and Sulfa Exposure

Next, we consider the impact of sulfa exposure on marriage market outcomes. Marriage rates were relatively stable over this period (see Figure 7), and marriage and fertility choices were closely related; this was an era in which only 8.5% of births were out of wedlock (Bachu 1999). For example, women exposed to the birth control pill delayed marriage as well as childbearing (Goldin and Katz 2002). We estimate the effect of sulfa exposure on three dimensions of marriage market outcomes: current marital status, whether the woman was ever married, and age at first marriage for ever married women (Table 10). In the main estimates, for ever married status, we use the same sample as for labor market outcomes: 18 to 50 year old women at census enumeration, thus combining childbearing women and women who have completed their fertility. We estimate the impact of sulfa exposure on current marital status and age at first marriage for childbearing women (aged 18 to 40 at census) to capture any delay in marriage that may have mirrored fertility delay; we consider alternative samples with different age-at-census ranges in Online Appendix D.

Reductions in pneumonia mortality are estimated to have reduced the probability of women ever having married by 1.4 percentage points, and similarly to have reduced the probability of being married at the time of the census in the sample of women of childbearing age by 1 percentage point. Thus, pneumonia reductions led to a postponement of marriage and a reduction in marriage entry. Relative to the baseline probabilities of being currently married (72.6%) and ever married (85%), these imply declines of 1.5 and 1.7 percent.

We also estimate significant positive impacts of maternal mortality decline on marriage market choices: reductions in maternal mortality increased the probability that a woman was married at the time of the census by 2.1 percentage points, and the probability that she had ever married by 1.9 percentage points. These translate to 2.9 and 2.3 percent of the mean respectively. We estimate no significant impact of sulfa exposure on the average age at first marriage; however, in Online

Appendix C, where we estimate effects separately by race and education, we uncover a positive, significant effect of the decline in pneumonia mortality on age at first marriage among white women, suggesting that these women delayed marriage as well as childbearing. We also estimate a negative significant effect of the decline in maternal mortality on age at first marriage among white women, consistent with the positive effect of this decline in mortality on the probability of birth after 1937.

Overall, marital status did respond in a direction consistent with lower fertility and higher labor force participation of women, but more weakly than these outcomes. This suggests that many women in this era were childless despite being married.[49] Staying on in the labor market was more clearly associated with childlessness than being unmarried.

This view is supported by summary statistics for marriage outcomes by childlessness status (Table 12) and a descriptive regression of this relationship (Table 13). On average, without conditioning on covariates, childless women were 10 percentage points less likely to have married, and married 2.5 years later conditional on ever marrying. Conditional on birth year, birth state, and race, this gap widens, with unmarried women aged 18-40 being 9.7 percentage points more likely to be childless, and unmarried women aged 40-50 being 14.7 percentage points more likely to be childless. In the younger sample, labor force participation is more predictive of childlessness than marital status, while this pattern reverses in the older sample.

6.5 Robustness Checks

We conduct the same robustness checks for labor and marriage market outcomes as discussed in Section 5.3 for fertility outcomes. As these checks were discussed in detail in that section, we merely refer the reader to the tables here. The tables showing these estimates are found in Online Appendix D. Panels A-C in Tables 29 and 30 display robustness checks for *confounding events* (New Deal, Second World War and the Dust Bowl). Panel D, Tables 29 and 30 and Panel E, Tables 33 and 34 shows that our results are not driven by *omitted trends* by controlling for long-run means of the outcome variables and estimating a placebo intervention. Tables 31 and 32 show the binary difference-in-difference estimates, which compare fully treated (25 sulfa years) and untreated women (zero sulfa years) and omit women who were partially treated. To address *measurement error*, Panels F-G in Tables 33 and 34 consider the child pneumonia-influenza mortality rate. In Online Appendix D, we also show the robustness of our main effects to other checks, including consideration of alternative sample definitions and corrections for multiple hypothesis testing. In general, the estimated effects are robust to these alternative specifications.

6.6 Relevance to Contemporary Data

We have demonstrated that in response to declines in pneumonia mortality, women had fewer children and were more likely to be childless. They also had higher labor force participation and lower marriage rates. Here we demonstrate that this confluence of results is not an artefact of our sample,

[49] This ties in with the previously cited survey of Popenoe (1936), which was of married childless women.

nor driven by a particular feature of the estimated specifications. We show that these correlations are evident in the raw data for the United States in 1930, as well as in contemporary data across countries. Figure 13 plots the cross-sectional correlation across U.S. states in 1930 between pneumonia mortality and two measures of fertility, childlessness and total fertility.[50] Childlessness is negatively correlated with pneumonia mortality, while total fertility is positively correlated with it.[51]

We find a similar negative correlation between childlessness and other measures of child mortality, including under-2s diarrhea mortality and under-1s mortality from all causes - see Online Appendix F. In contrast, adult mortality rates, such as heart disease and cancer, are positively correlated with childlessness (and negatively correlated with total fertility). This is in line with our contention that the relationship of childlessness and child mortality is related to the choice to delay fertility and continue in the labor market in the reproductive ages rather than women's health; the mechanisms for the associations with adult mortality are clearly different. We show similar plots of labor force participation and marital status against child mortality in the same Appendix. We find that labor force participation is inversely correlated with child mortality, while marriage is positively correlated, consistent with our causal estimates.

Turning to contemporary data, Figure 14 plots the cross-sectional relationship between fertility and, first, infant mortality (from all causes) and, second, neonatal mortality from pneumonia and other respiratory causes, across countries in 2015.[52] Similar to the early 20th century data from the U.S., contemporary all-country data show a positive correlation of infant mortality with both margins of fertility (i.e. a negative correlation between infant mortality and childlessness). Given our finding that declines in child mortality stemming from pneumonia decline increased labor force participation and reduced marriage rates in the US, these data suggest that child mortality decline may have the potential to "liberate" women in developing countries into work and away from early marriage, and the disempowerment that often accompanies this practice.

7 Conclusion

This paper presents quasi-experimental estimates of how improvements in health and survival generated by the introduction of antibiotics early in the last century influenced fertility, with a particular focus on the timing of fertility, the behavior of the extensive margin response, and its relation to labor market careers and marriage among women. The analysis produces two striking

[50] These figures are for 25-40 year olds and fertility is net fertility, being based on information about children living in the household at the time of the survey. The upper age limit is chosen to minimise underreporting due to older children having left home. Still, to the extent that some women continue fertility after age 40, we may underestimate fertility and overestimate childlessness, as is evident from the fairly high proportion of childless women in this age group. However, this is only an issue for the cross-sectional relationship if the age at which children leave home is correlated with underlying mortality rates.

[51] New Mexico appears to be an outlier in the infant mortality data. In Online Appendix D, we show that the main results are robust to the exclusion of New Mexico from the sample.

[52] The source of the fertility data is the IPUMS International Database. Every country for which gross fertility information in the 2000s was available was included in the sample. We chose the census year closest to 2015 for each country. The mortality data are for 2015 and these data are sourced from UNESCO.

findings. First, a decline in child mortality (driven by pneumonia decline) led to fertility delay, a reduction in overall fertility (with fewer women having three or more children) and an increase in childlessness. Second, the decline in pneumonia mortality was associated with a higher propensity to work, higher occupational scores, and a lower probability of having ever married.

We argue that these findings are linked, indeed, that child mortality decline made it rational to delay fertility relative to before the introduction of antibiotics, and that this had spillovers for labor market and marriage decisions. We argue that reductions in child mortality meant that women had to spend less time childbearing to achieve a target number of children, in addition to which they will have reduced their target number of children, if the reduction in the price of child quality outweighed the reduction in the price of child quantity; the reduction in fertility on the intensive margin in response to the decline in child mortality suggests that the decline in the price of child quality dominated and women did change their fertility targets. Both mechanisms contribute to fewer years spent childbearing together with less time expected to be spent caring for sick children, allowing women more time for productive activities and the ability to remain in the labor market longer. Among women in the labor market, positive shocks to wages, negative shocks to fecundity or fertility preferences, or inertia, can result in persistence of the childless state. This suggests that fertility choices should be modelled as a dynamic process; we outline a dynamic model of fertility and labor market choices that shows a greater propensity for fertility delay in response to a decline in child mortality, when the joint probability of promotion at work and child mortality are low. The estimated patterns are consistent with this theory, fairly large, and robustly determined.

These results make the following contributions to the literature. First, they contribute causal estimates of the impact of pneumonia and maternal mortality decline on fertility, both of which are scarce but relevant to models of the demographic transition and economic growth. Second, our analysis has some useful features relative to previous work in this domain. We model both the dynamics of fertility at the time of exposure to the reform and completed fertility, for the same cohorts of women observed in census data in different years. We present estimates for both gross and net fertility, and we allow pneumonia and maternal mortality to have a different impact on fertility. Third, we provide new evidence on the causes of childlessness, and the need to consider fertility, labor market and marriage market choices in conjunction. Our findings are relevant for contemporary development policy. They suggest that child mortality decline can contribute to the economic independence of women, where labor market opportunities are available such that women can delay childbearing and enter the labor market. This labor force participation and associated economic independence can, under certain conditions, lead to increased investments in children (Lundberg, Pollak, and Wales 1997) and a reduction in domestic violence (Aizer 2010).

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Tables and Figures

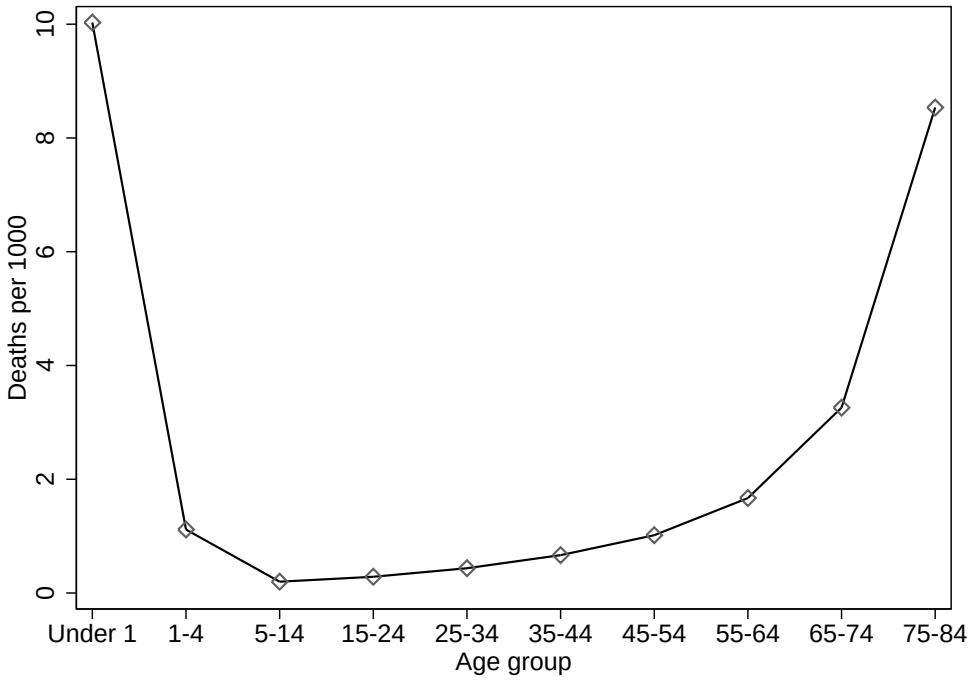


Figure 1: Pneumonia Incidence by Age, United States, 1935

This figure shows the average pneumonia mortality rate by age group in 1935 in the United States. Source: Britten (1942).

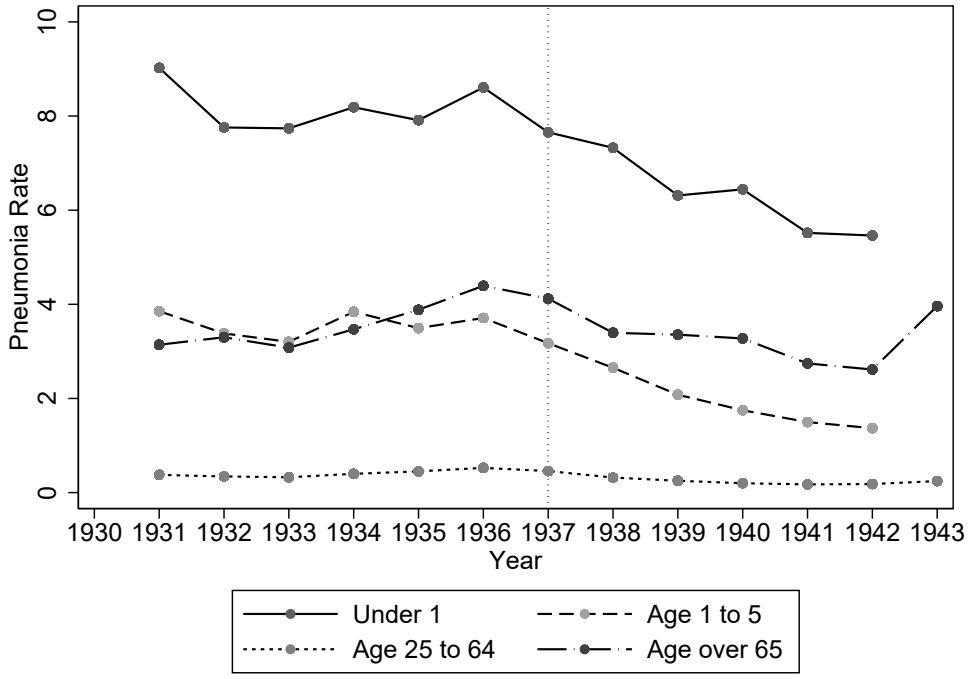
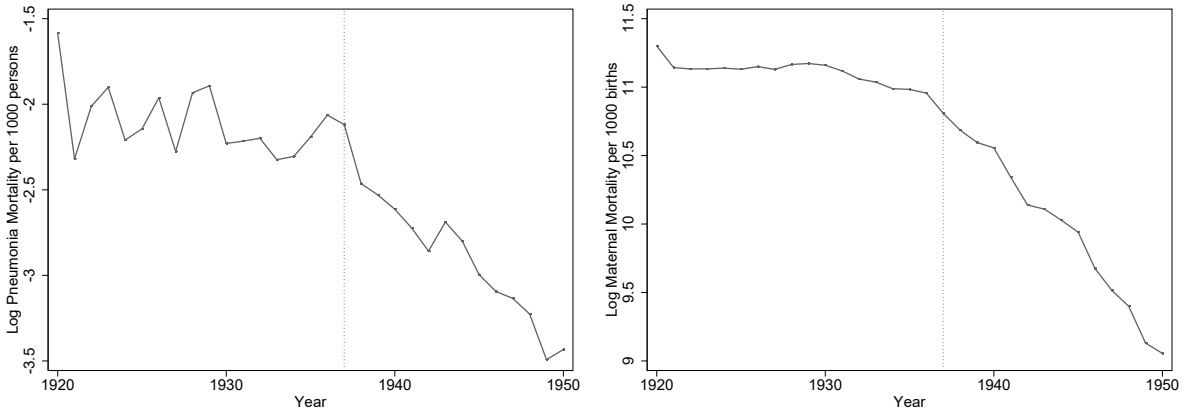


Figure 2: Pneumonia Mortality by Age, United States

This figure shows the average pneumonia mortality rate by age group and over time in the United States. Source: Vital Statistics.

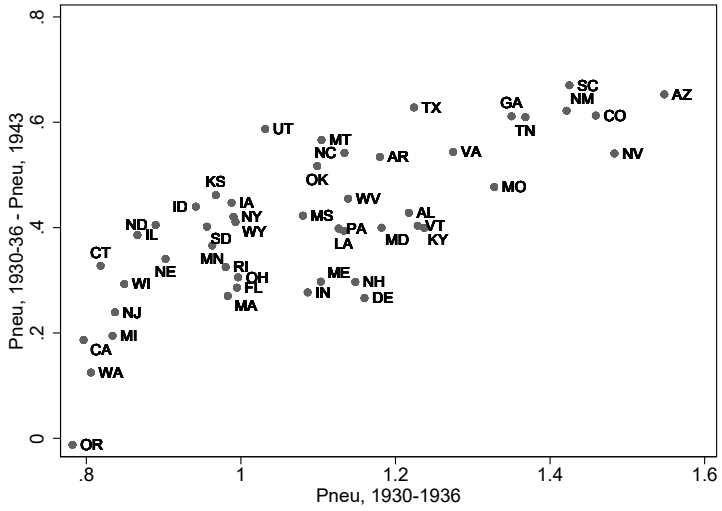


(a) Pneumonia Mortality

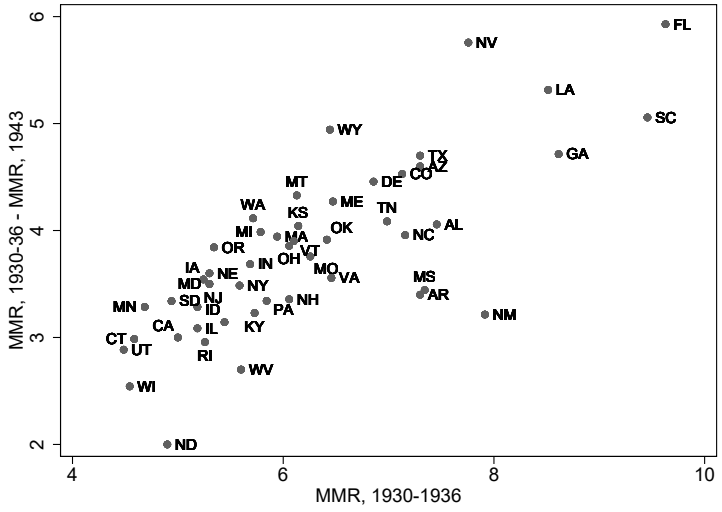
(b) Maternal Mortality

Figure 3: Pneumonia Mortality and Maternal Mortality, United States

These figures show the average pneumonia mortality rate (left) and maternal mortality rate (right) in the United States over time. Source: Vital Statistics.



(a) Pnuemonia Mortality Convergence Post-1937



(b) Maternal Mortality Convergence Post-1937

Figure 4: Pneumonia Mortality and Maternal Mortality Convergence Post-1937, United States

These figures show the relationship between the 1937-1943 change and the 1930-1936 average level of pneumonia mortality (top) and maternal mortality (bottom) for different states in the United States. Source: Vital Statistics.

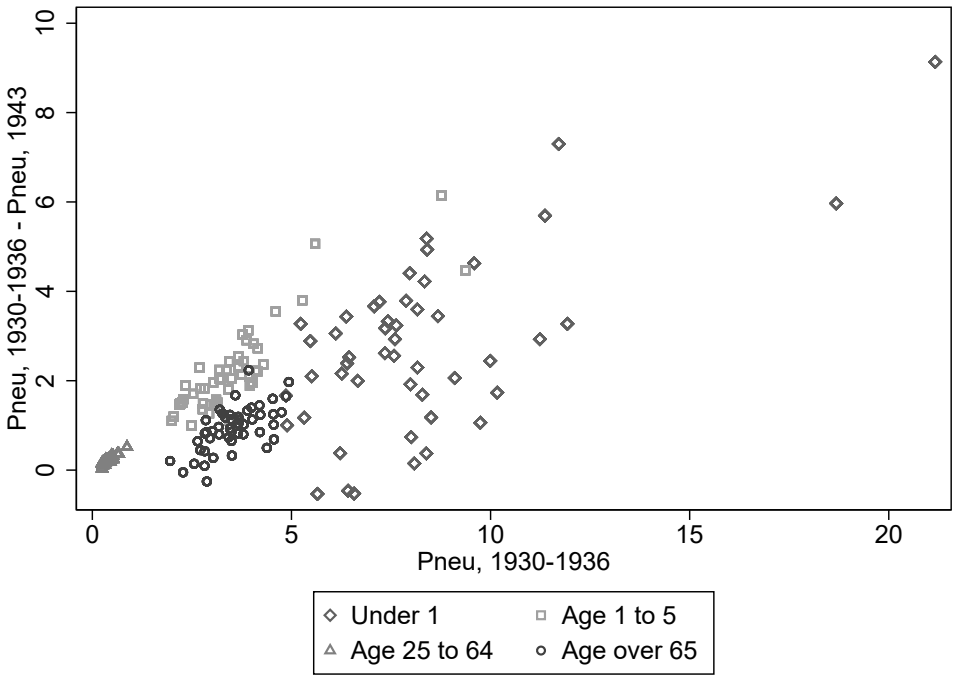


Figure 5: Pneumonia Mortality Convergence Post-1937 by Age, United States

This figure shows the relationship between the 1937-1943 change and the 1930-1936 average level of pneumonia mortality in different age groups and different states in the United States. Source: Vital Statistics.

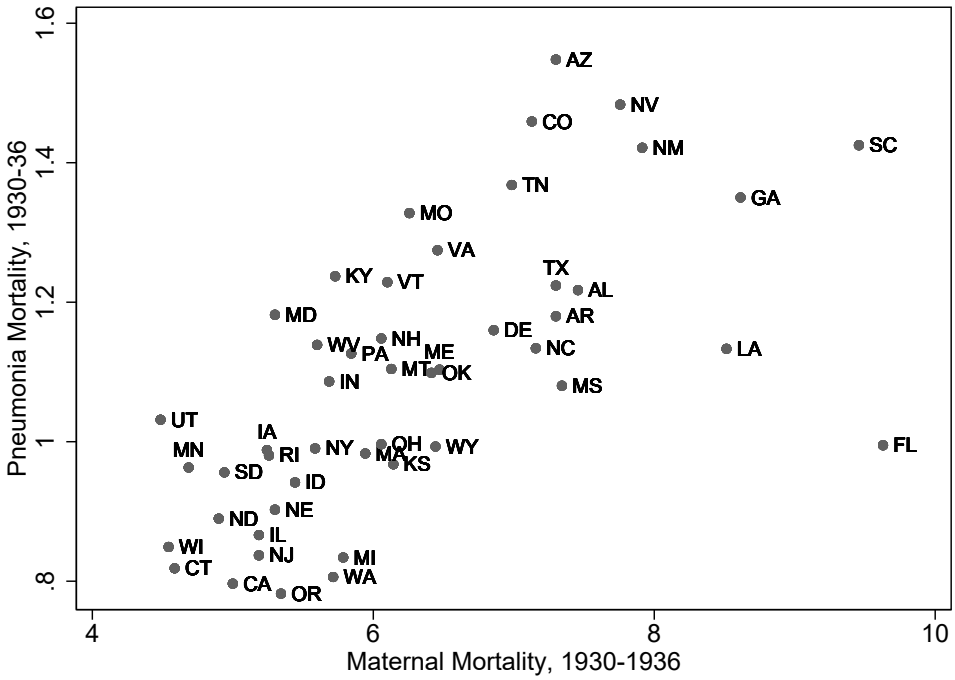


Figure 6: Pneumonia and Maternal Mortality, United States, 1930-1936

This figure shows the relationship between the average pneumonia and maternal mortality rates in 1930-1936 across different states in the United States. Source: Vital Statistics.

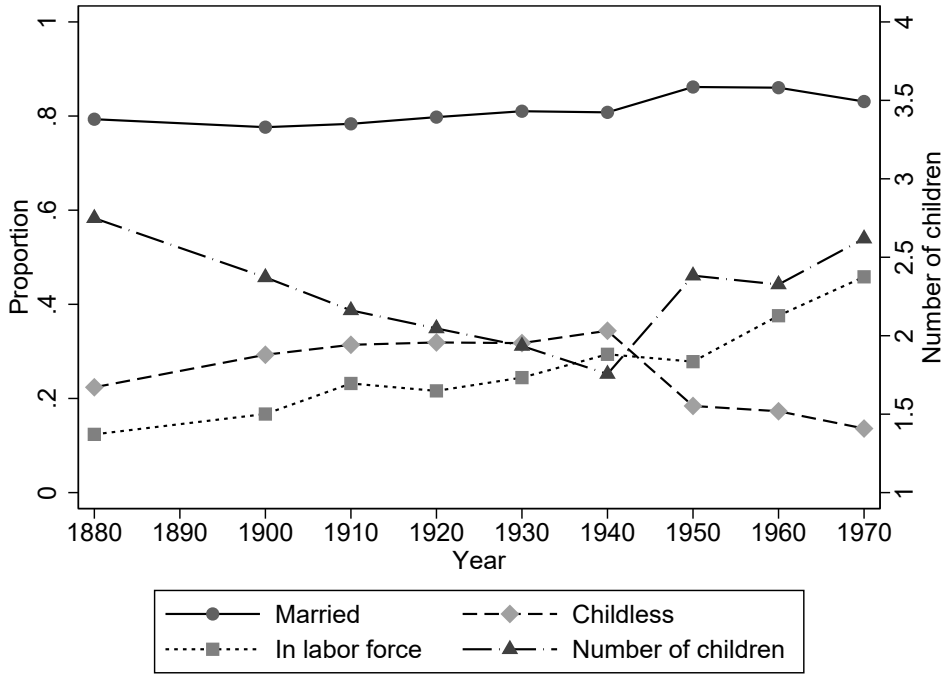


Figure 7: Trends in childlessness, fertility, labor force participation and marriage, United States, 1880-1970

This figure is constructed from the US decennial population censuses 1880-1970. The sample consists of all women aged 30 to 40 at the time of the census interview and born in the US. Childlessness and total fertility is defined based on net fertility (children living in the home), and marriage and labor force status refer to these statuses at the time of the census.

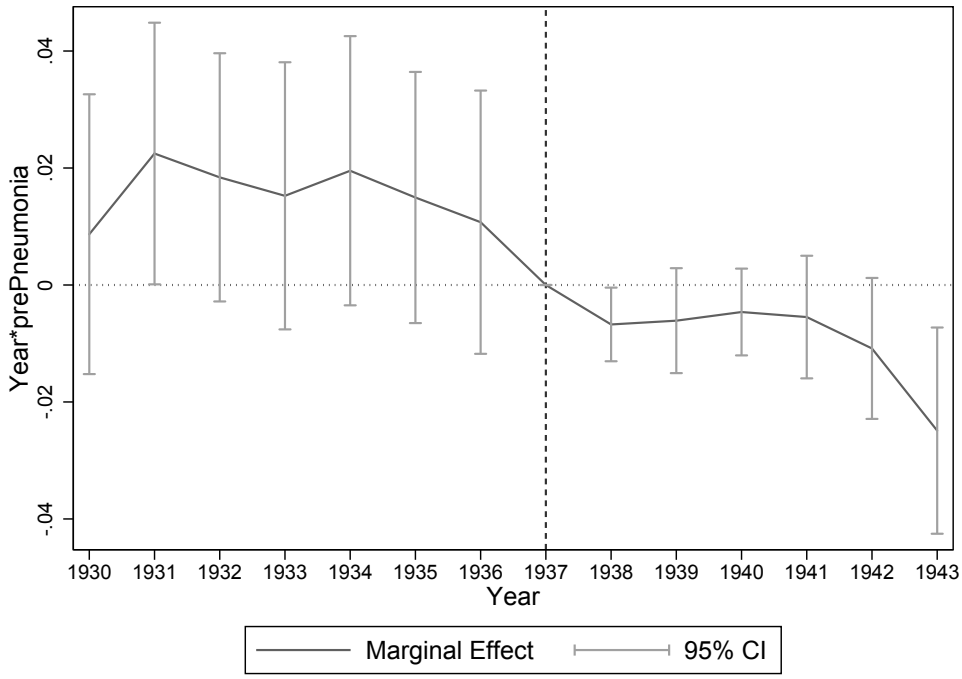


Figure 8: Event Study for Pneumonia

This figure displays the coefficients and 95% confidence intervals around these coefficients on the set of variables $prePneumonia * year$ where $year$ is a set of dummy variables for the 13 years 1930-1936 and 1938-1943 (1937 is the omitted case). The dependent variable is a dummy variable that equals one if the woman gave birth in that year, and zero otherwise. This is a Logistic regression that also contains the set of variables $preMMR * year$. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, education, birth order and time since last birth fixed effects and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$.

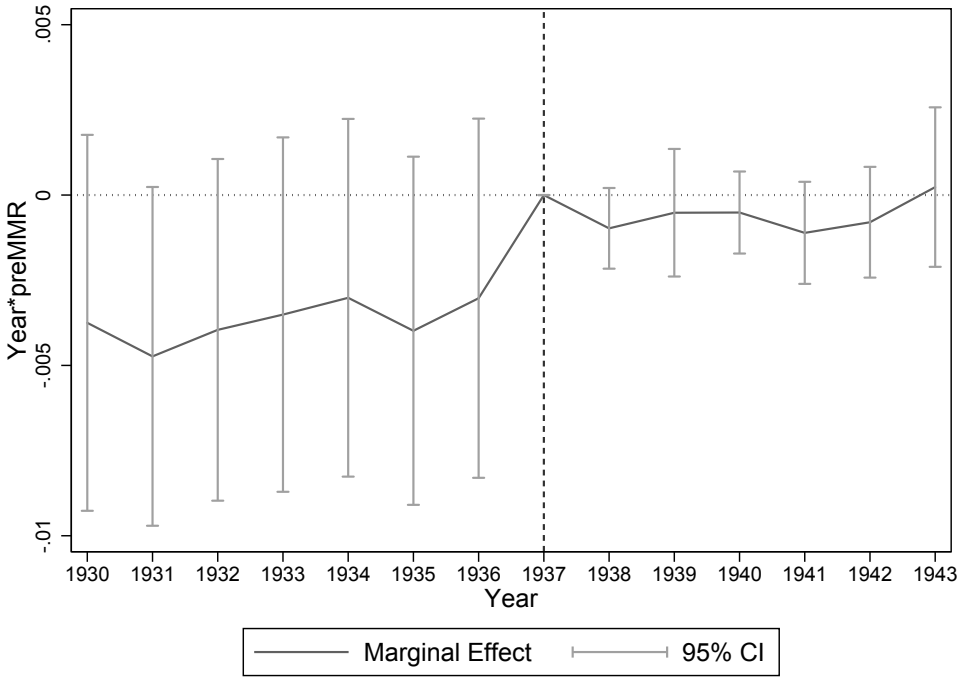


Figure 9: Event Study for Maternal Mortality

This figure displays the coefficients and 95% confidence intervals around these coefficients on the set of variables $preMMR * year$ where $year$ is a set of dummy variables for the 13 years 1930-1936 and 1938-1943 (1937 is the omitted case). The dependent variable is a dummy variable that equals one if the woman gave birth in that year, and zero otherwise. This is a Logistic regression that also contains the set of variables $prePneumonia * year$. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, education, birth order and time since last birth fixed effects and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$.

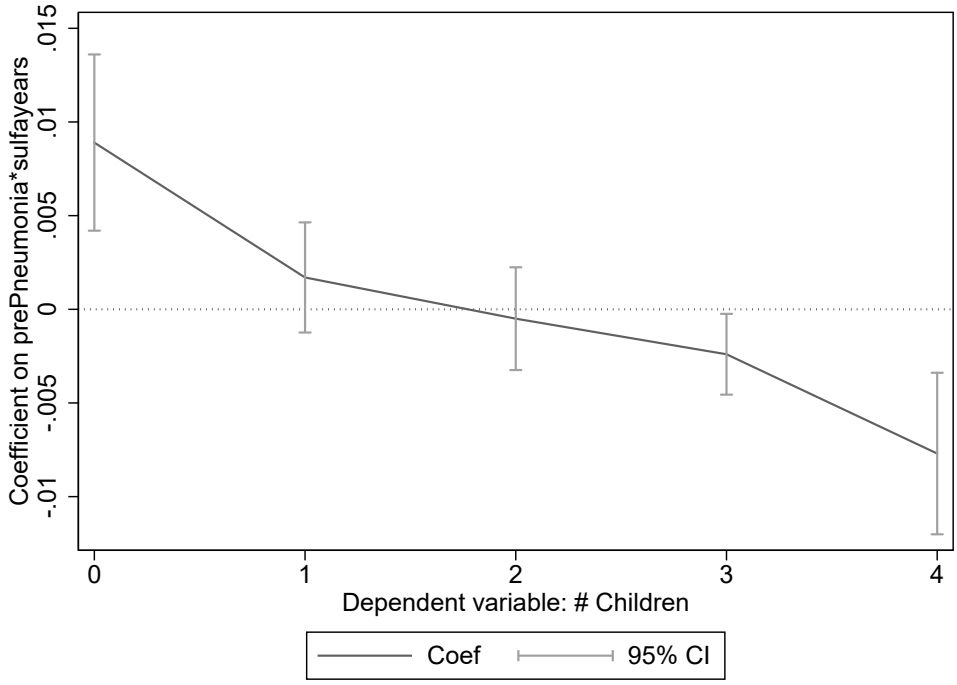


Figure 10: The Estimated Effect of Pneumonia Mortality Decline on the Fertility Distribution

This figure displays the coefficients and 95% confidence intervals around these coefficients on the variable $prePneumonia * sulfayears$ in a set of five separate OLS regressions, where the dependent variables in these regressions are dummy variables for having no children in the household, exactly one child, exactly two children, exactly three children, and four or more children (net fertility). Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for maternal mortality, malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$.

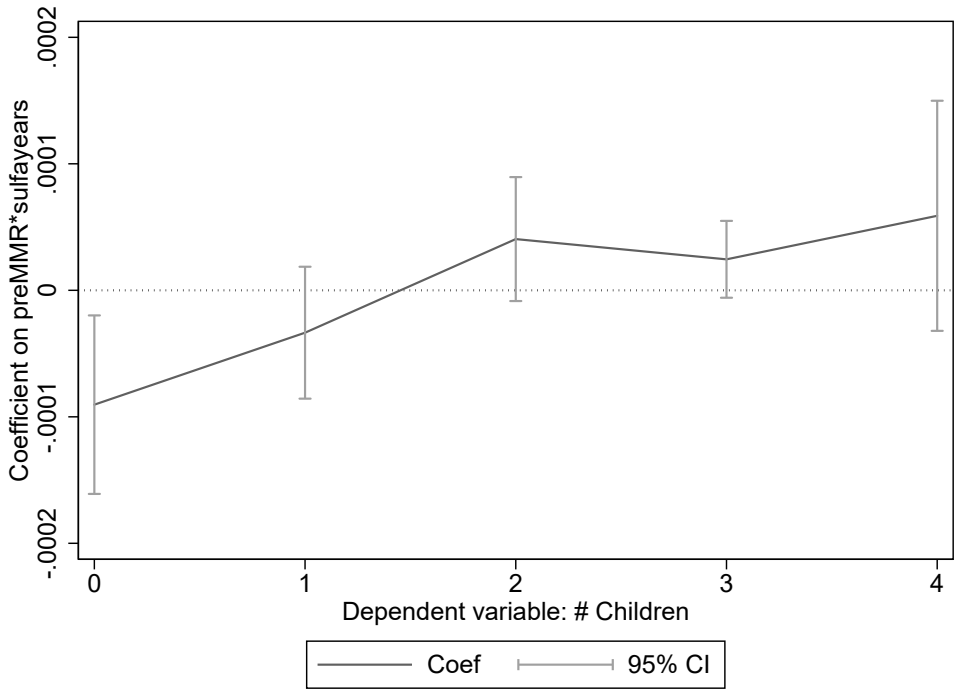


Figure 11: The Estimated Effect of Maternal Mortality Decline on the Fertility Distribution

This figure displays the coefficients and 95% confidence intervals around these coefficients on the variable $preMMR*sulfayears$ in a set of five separate OLS regressions, where the dependent variables in these regressions are dummy variables for having no children in the household, exactly one child, exactly two children, exactly three children, and four or more children (net fertility). Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for pneumonia-influenza, malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$.

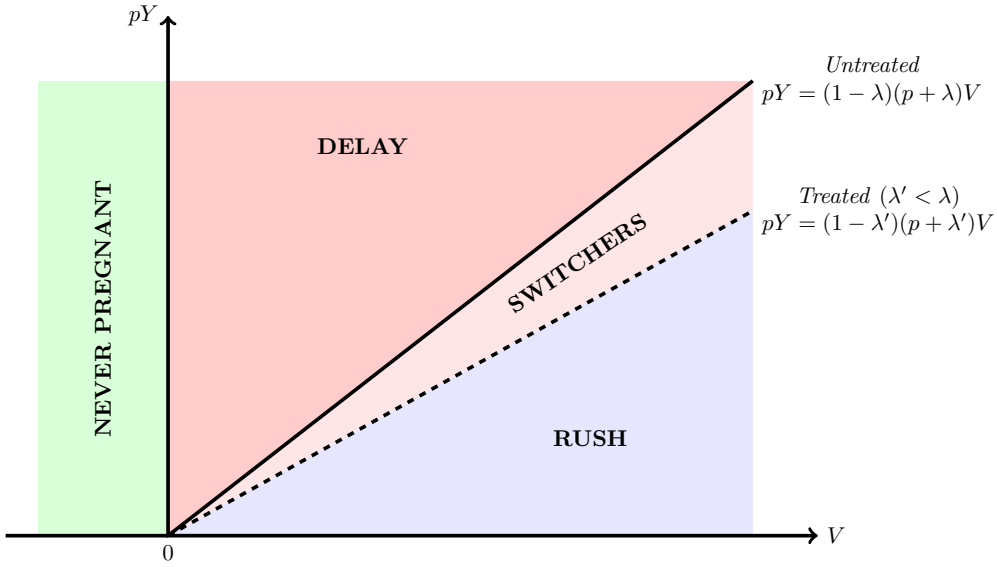
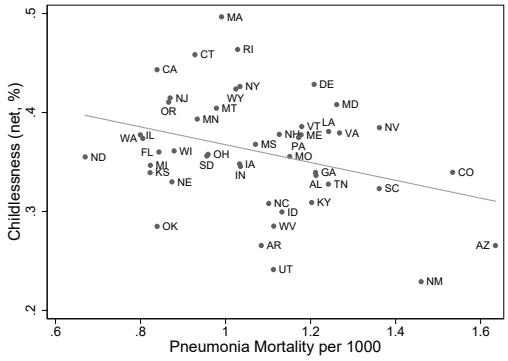


Figure 12: The effect of a reduction in child mortality λ on fertility delay in the model

The solid line delineates two groups of women: those above the line prefer to delay fertility and enter the labor market, while those below the line prefer to start childbearing in the first period. The line is given by the condition in (4). When child mortality falls, the solid line shifts down to the dotted line. More women prefer to delay instead of childbearing in the first period.

Figure 13: Pneumonia Mortality and Net Fertility in 1930 across US states

(a) Pneumonia Mortality and Net Childlessness



(b) Pneumonia Mortality and Net Fertility

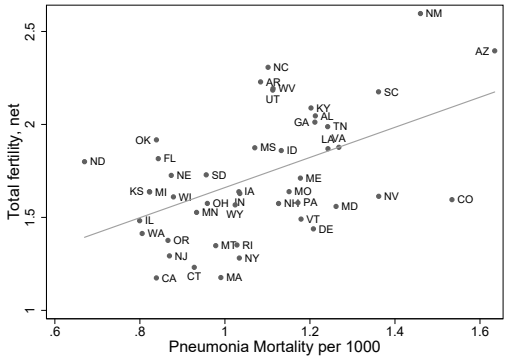
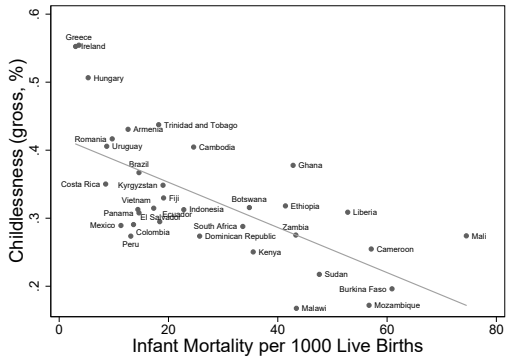
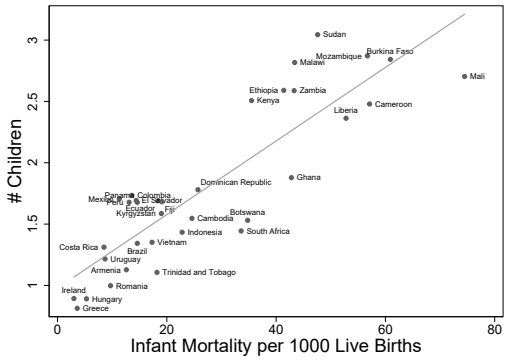


Figure 14: Infant Mortality and Gross Fertility, Worldwide, 2015

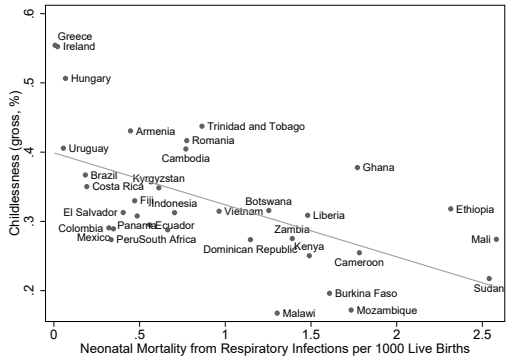
(a) Infant Mortality and Gross Childlessness



(b) Infant Mortality and Gross Fertility



(c) Neonatal Respiratory Infection Mortality and Gross Childlessness



(d) Neonatal Respiratory Infection Mortality and Gross Fertility

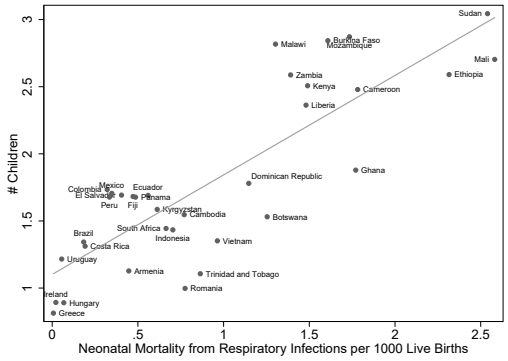


Table 1: Descriptive statistics: Control variables

Variable	Mean	Standard deviation
$prePneumonia$	1.0918	0.1989
$preMMR$	6.2610	1.2403
$preDiarrhea$	8.1358	5.7157
$preMalaria$	34.1667	70.4349
$preCancer$	0.9674	0.3109
$preHeartDisease$	2.1483	0.6439
$preTuberculosis$	0.6284	0.3616
$ln(Income\_per\_capita)$	5.9551	0.3960
$ln(Number\_of\_schools\_per\_capita)$	0.7586	0.6491
$ln(Number\_of\_hospitals\_per\_capita)$	-2.80	0.4427
$ln(Number\_of\_doctors\_per\_capita)$	0.1246	0.2291
$ln(Education\_expend\_per\_capita)$	4.6150	0.3887
$ln(Health\_expend\_per\_capita)$	-1.2317	0.6275
$Year\_of\_birth\_registration$	1921.17	5.3726
$Year\_of\_death\_registration$	1910.681	13.478
$Literacy$	0.9760	0.0374
$Female\_LFP$	0.1971	0.0596
N	48	

This table shows the mean and standard deviation of state level characteristics that are interacted with $post1937$ in the hazard sample and $sulfayears$ in the stock sample and included as control variables. The mortality rates from diseases are the average between 1930-1936, per 1000 population (or 1000 live births in the case of MMR), and all other variables are measured in 1930, except the year of entering the birth and death registration systems.

Table 2: Descriptive statistics: Hazard model

Variable	Mean	Standard deviation
Birth	0.0865	0.2811
$post1937$	0.5001	0.5
$Current\_birth\_order$	1.7182	1.2364
$Years\_since\_last\_birth$	6.8448	6.1723
$Birth\_year\_of\_woman$	1910.724	98.0187
N	4559108	

This table shows the mean and standard deviation of outcome and control variables in the hazard model. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses.

Table 3: Descriptive statistics: Stock model

Variable	Mean	Standard deviation	N
Net Fertility (childbearing sample)			
# Children	1.6590	1.8316	496783
# Children | Children>0	2.6118	1.6712	315548
Childless (0-1)	0.3648	0.4814	496783
$Sulfayears$	20.0	6.0626	496783
Net Fertility (completed fertility sample)			
# Children	1.9282	1.9473	239432
# Children | Children>0	2.6760	1.8060	172524
Childless (0-1)	0.2794	0.4487	496783
$Sulfayears$	14.6401	8.8397	239432
Gross Fertility (completed fertility sample)			
# Children	2.5750	2.2927	520591
# Children | Children>0	3.1660	2.1428	423423
Childless (0-1)	0.1866	0.3896	520591
$Sulfayears$	14.8679	8.6624	520591
Labor Market			
Working (0-1)	0.3510	0.4773	730498
In labor force (0-1)	0.3710	0.4831	730498
Hauser-Warren SEI	14.4093	17.181	519972
Personal income	1505.191	2817.12	307378
Hours worked	12.8097	19.1029	730498
$Sulfayears$	18.2949	7.5187	730498
Marriage Market			
Currently married	0.7258	0.4461	496783
Ever married	0.8499	0.3572	926552
Age at 1st marriage	21.1798	3.4153	106814
$Sulfayears$	17.5947	7.9902	926552
Age at birth			
Age at 1st birth	24.0750	4.9714	440156
Age at 2nd birth	26.7165	5.0326	316185
Age at 3rd birth	28.6299	5.0395	183840
Age at 4th birth	30.1623	4.9682	101896

This table shows the mean and standard deviation of key variables in the stock model. Our dataset is a cross-section of fertility, labor and marriage outcomes of women aged 5-44 in 1937 and 18-40 (net childbearing fertility, current marital status), at least 40 (net completed, gross fertility), 18-50 (labor, age at birth, ever married) at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 (1900-1931 for net fertility and current marriage) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses.

Table 4: Probability of birth as a function of sulfa exposure, Logit

	(1) Birth	(2) Birth	(3) Birth	(4) Extensive Margin	(5) Intensive Margin
$prePneumonia * post1937$	-0.0143***	-0.0142***	-0.0233**	-0.0121**	-0.0095*
	(0.0033)	(0.0026)	(0.0100)	(0.0060)	(0.0051)
$preMMR * post1937$	0.0009**	0.0009	0.0031	0.0021	0.0007
	(0.0004)	(0.0006)	(0.0024)	(0.0016)	(0.0010)
N	4558873	4541009	4499588	2894976	1604613
Mean	0.0865	0.0865	0.0865	0.0513	0.1491
Controls					
Baseline	Y	Y	Y	Y	Y
Control diseases	N	Y	Y	Y	Y
State characteristics	N	N	Y	Y	Y

The dependent variable is a dummy variable that equals one if the woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the woman's birth state level and the table shows marginal effects at the means of all covariates in the estimating sample. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, education, birth order and time since last birth fixed effects, year and census division*year fixed effects; column (2) adds state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s interacted with $post1937$, and column (3) adds income, public services, literacy, female labor force participation, and the year of state birth and death registration interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 5: Net fertility as a function of sulfa exposure: Childbearing women

	(1) # Children	(2) # Children	(3) # Children	(4) # Children | Children >0	(5) Childless
$prePneumonia * sulfayears$	-0.0782***	-0.0638***	-0.0483***	-0.0345***	0.0089***
	(0.0213)	(0.0140)	(0.0127)	(0.0117)	(0.0024)
$preMMR * sulfayears$	0.0008	0.0083***	0.0035	0.0009	-0.0009**
	(0.0028)	(0.0028)	(0.0024)	(0.0026)	(0.0004)
N	496783	494854	494437	313981	494437
Mean	1.6590	1.6590	1.6590	2.6118	0.3648
Controls					
Baseline	Y	Y	Y	Y	Y
Control diseases	N	Y	Y	Y	Y
State characteristics	N	N	Y	Y	Y

The dependent variable in columns (1)-(3) is the total number of own children living in the household (net fertility). In column (4) it is the total number of own children living in the household conditional on having at least one (net fertility). The dependent variable in column (5) is a dummy variable that equals one if a woman has no children living in the household, and zero otherwise (net childlessness). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects. The following variables are added after the first column: state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s interacted with $sulfayears$ (column 2), income and public services, literacy, female labor force participation, and the year of state birth and death registration interacted with $sulfayears$ (column 3). * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 6: Net fertility as a function of sulfa exposure: Completed fertility

	(1) # Children	(2) # Children | Children >0	(3) Childless
$prePneumonia * sulfayears$	-0.0212**	-0.0218**	0.0027*
	(0.0103)	(0.0098)	(0.0016)
$preMMR * sulfayears$	0.0006	-0.0001	-0.0001
	(0.0021)	(0.0018)	(0.0003)
N	237603	171166	237603
Mean	1.9282	1.6760	0.2794

The dependent variables are the total number of children (column 1), the total number of children conditional on having at least one (column 2) and a dummy variable that equals one if the woman has zero children and zero otherwise (column 3), all based on net fertility (the number of own children living in the household). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 40-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 7: Gross fertility as a function of sulfa exposure: Completed fertility

	(1) # Children	(2) # Children | Children >0	(3) Childless (0-1)
$prePneumonia * sulfayears$	-0.0209*	-0.0187*	0.0021**
	(0.0118)	(0.0106)	(0.0009)
$preMMR * sulfayears$	0.0007	0.0003	-0.0000
	(0.0023)	(0.0021)	(0.0001)
N	518933	421983	518933
Mean	2.5750	3.1660	0.1866

The dependent variable in column 1 is the total number of live births (gross fertility). The dependent variable in column 2 is the total number of live births conditional on having at least one (gross fertility). The dependent variable in column 3 is a dummy variable that equals one if a woman has no live births, and zero otherwise (gross childlessness). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and at least 40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 8: Reasons for Childlessness, excerpted from Popenoe (1936) p. 470

Reason	N	%
Self-centred	180	31
Wife's career	128	22
Economic pressure	96	16
Health	51	9
Dislike of all children	49	8
Miscellaneous	36	6
Eugenics	27	5
Marital discord	15	3

This information is excerpted from Popenoe (1936), p.470, and shows the reasons for permanent childlessness reported by university students about cases of permanently childless couples that they know. The survey was conducted in the early 1930s and the categories were constructed by Popenoe based on the descriptive histories provided by the students.

Table 9: Labor market outcomes as a function of sulfa exposure

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1991**	7.3736	0.2421***
	(0.0017)	(0.0018)	(0.0771)	(15.3400)	(0.0652)
$preMMR * sulfayears$	-0.0008**	-0.0008**	0.0004	-0.1344	-0.0322***
	(0.0003)	(0.0003)	(0.0123)	(2.7242)	(0.0114)
N	727398	727398	517857	306280	727398
Mean	0.3510	0.3710	14.4093	1505.191	12.8097

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation, available for the 1950+ censuses; (4) the US Dollar amount of personal earnings in the past year, available for the 1950+ censuses; (5) hours worked in the past week, converted from intervalled data to a continuous measure using the midpoint of each interval. We find similar estimated effects of sulfa exposure on other measures of occupational score, including occscore (coefficients(standard error) 0.0775(0.0473) and -0.0146(0.0079) for $prePneumonia * sulfayears$ and $preMMR * sulfayears$ respectively), and the Duncan socioeconomic score (coefficients(standard error) 0.1512(0.0893) and -0.0139(0.0129) respectively). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 5-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 10: Marriage market outcomes as a function of sulfa exposure

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0023*	-0.0032**	0.0021
	(0.0012)	(0.0012)	(0.0243)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0053
	(0.0002)	(0.0002)	(0.0055)
N	494437	727398	116632
Mean	0.7258	0.8499	21.1798

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage for woman who have ever married. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 5-44 in 1937 and 18-40 for columns 1 and 3 and 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 for columns 1 and 3 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 11: The joint probability of work and childlessness as a function of sulfa exposure

	(1)	(2)	(3)	(4)
	Working		Not working	
	Childless	Not childless	Childless	Not childless
$prePneumonia * sulfayears$	0.0031***	0.0027	-0.0004	-0.0054*
	(0.0011)	(0.0024)	(0.0013)	(0.0027)
$preMMR * sulfayears$	-0.0003*	-0.0003	0.0001	0.0006
	(0.0002)	(0.0005)	(0.0002)	(0.0005)
N	326643	326643	326643	326643
Mean	0.086	0.283	0.084	0.548

The dependent variable is the joint status of work and childlessness (based on gross fertility) at the time of census enumeration. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility, labor and marriage market outcomes of women aged 18 to 50 at the time of the census, aged 5 to 44 in 1937, and born in the United States. The cohorts in this table were born in the years 1893 to 1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s interacted with $sulfayears$, income and public services, literacy, female labor force participation, and the year of state birth and death registration interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 12: Descriptive outcomes by childlessness status

Outcome	Childless women			Not childless women		
	Mean	St.dev.	N	Mean	St.dev.	N
Working	0.54	0.50	112916	0.37	0.48	593323
In labor force	0.56	0.50	112916	0.39	0.49	593323
H-W SEI	24.28	18.44	90226	19.12	17.85	552716
Personal income	2961.29	4404.62	90159	1901.75	3781.59	552177
Hours worked	20.01	20.55	112916	12.78	18.54	593323
Currently married	0.77	0.42	112916	0.88	0.32	593323
Ever married	0.89	0.31	112916	0.996	0.06	593323
Age at 1st marriage	23.58	5.36	62622	21.14	3.67	317119
Graduated from HS	0.36	0.48	112916	0.39	0.49	593323
Attended some college	0.21	0.41	112916	0.17	0.37	593323

This table shows the mean and standard deviation of outcome variables by (gross) childlessness status in the stock model. All differences in means between childless and not childless women are statistically significant at the 1% level. Our dataset is a cross-section of fertility, labor market and marriage market outcomes for women aged 5 to 44 in 1937 and 18 to 50 at the time of the census, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses.

Table 13: Descriptive regression of relationship between childlessness and labor and marriage market outcomes

	(1)		(2)
		Childless	
	Childbearing women		Completed fertility women
In labor force	0.1727***		0.0489***
	(0.0090)		(0.0035)
Married	-0.0336***		-0.1502***
	(0.0055)		(0.0116)
N	180555		176542
Mean	0.1829		0.1528

The dependent variable equals one if the woman is childless (based on gross fertility i.e. zero live births) and zero otherwise. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility, labor and marriage market outcomes of women aged 18 to 40 (40 to 50 in column (2)) at the time of the census, aged 5 to 44 in 1937, born in the United States and resident in their birth state at census. The cohorts in this table were born in the years 1893 to 1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Online Appendix
Fertility Responses to Reductions in Mortality: Quasi-Experimental Evidence from 20th Century America

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A Data description

We use the United States Decennial Population Census Microdata 1% files for the fertility, labor market and marriage market outcomes (Ruggles, Alexander, Genadek, Goeken, Schroeder, and Sobek 2010). The **hazard dataset** is from pooled census microdata from the 1940 1%, and 1950 1% samples and includes women of childbearing age (15 to 40) between 1930-1943. The construction of births is based on the birth dates of own children living in the household at the time of the census. This information is used to construct a retrospective panel dataset at the woman-year level, which identifies whether a woman gave birth in a given year or not. Only woman-year observations where a woman is aged 15 to 40 in that year are included.

The **stock dataset** is based on pooled data from the 1940, 1950, 1960 and 1970 1% cenuses, and we include all women who were aged between 5 and 44 in 1937. Within this sample, we introduce additional restrictions on age at census depending on the outcome variable. For net fertility, we restrict the data to women aged 18-40 at the time of the census interview and separately to women aged 40-50. For gross fertility, we restrict the data to women who have completed their fertility, so aged 40 or over at the time of the census. For labor market outcomes, we restrict the data to women aged 18-50 at the time of the survey. For current marital status and age at first marriage, we consider 18-40 year old women, and for ever married status we consider women aged 18-50 at the time of the census, the same as the labor sample. We show robustness to widening and narrowing all of these samples in Online Appendix D, and our results are not sensitive to varying the age window.

In both datasets, we only include US born women not residing in group quarters who are resident in their birth state at the time of the census. The latter limits bias that could arise due to migration.

The **mortality data** is extracted from US Vital Statistics (Grove and Hetzel 1968, Linder and Grove 1947, Ruggles, Alexander, Genadek, Goeken, Schroeder, and Sobek 2010, Bureau 1943). In particular, we combined and extended the data series collected by Grant Miller (http://www.nber.org /data/vital-statistics-deaths-historical/), and by Seema Jayachandran, Adriana Lleras-Muney, and Kimberly Smith (http://www.aeaweb.org/articles.php?doi=10.1257/app.2.2.118). The control variables are from the Bureau of Economic Analysis (state variables) and the data on public services are from Adriana Lleras-Muney' website. The health expenditure data is from the US Census Bureau. The state level data is matched to individual data by women's birth state.

The **main outcome variables** are constructed as follows.

- *Net total fertility* is the total number of own children living in the household. *Net childlessness* is a variable equal to one when this is zero and equal to zero otherwise.
- *Gross total fertility* is the total number of live births the woman ever had. *Gross childlessness* is a variable equal to one when this is zero and equal to zero otherwise. The number of live births was a question asked to ever-married women in the 1940 and 1950 censuses and to all women in subsequent censuses.
- The *intensive* margin of fertility for both of these measures is defined as total fertility conditional on not being childless; hence, this variable takes a missing value for childless women.
- The variable *Working* takes a value of one if the woman reports working at the time of the census and zero otherwise.
- The variable *In Labor Force* takes a value of one if the woman reports she is in the labor force at the time of the census.
- *Personal income* is the reported own income from all sources in the last year. It is available for the 1950 census and onwards.
- The *Hauser and Warren Socioeconomic Index (H-W SEI)* is a measure of occupational status based on earnings and education. It assigns a measure of prestige to each occupation. See ipums.org for a detailed explanation of its construction. It is available for the 1950 census and onwards. We also considered *occscore* from the IPUMS data and the *Duncan socioeconomic score* as outcomes, with similar results.
- *Hours worked* is the reported number of hours worked in the past week. The original data is an intervalled variable and it is converted to a continuous variable using the midpoint of each interval.
- The variable *Currently married* takes the value one if a woman is married at the time of the census and zero otherwise.
- *Ever married* is a dummy variable equal to one if a woman has been married at some point in her life and zero otherwise.
- *Age at 1st marriage* is the age at which a woman first married, only defined for women who have ever married, and not available for the 1950 census, hence making the sample size for this variable smaller than for the other outcomes.

B Convergence regressions

These tables formally test convergence in mortality rates after the introuduction of sulfa drugs in 1937. Table 14 tests for the existence of a trend break in mortality rates in 1937, captured by a linear trend interacted with a post-1937 dummy variable. Table 15 shows that high mortality states pre-1937 had larger declines in mortality rates post-1937.

Table 14: Trend breaks in mortality rates

	(1)	(2)	(3)	(4)
	Levels		Logs	
	Pneumonia	*MMR*	*Pneumonia*	*MMR*
year ∗ *post*1937	-0.0999***	-0.2143***	-0.1110***	-0.0930***
	(0.0059)	(0.0252)	(0.0059)	(0.0058)
*post*1937	-0.1408***	-0.5128***	-0.1287***	-0.0930***
	(0.0240)	(0.1021)	(0.0238)	(0.0235)
year	0.0192***	-0.2154***	0.0153***	-0.0335***
	(0.0042)	(0.0180)	(0.0042)	(0.0041)
N	667	667	667	667
R^2	0.7573	0.8981	0.7988	0.8944

These are OLS regressions (standard errors in parentheses) at the state-year level. The dependent variables are *Pneumonia*, the state-year average mortality rate from pneumonia, and *MMR*, the state-year average maternal mortality rate. The regressions also include state fixed effects. *year* is a linear time trend and *post*1937 is a dummy variable for the years 1937 and later. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 15: Test of convergence in state mortality rates

	(1)	(2)
	Pneumonia	*MMR*
prePneumonia ∗ *post*1937	-0.2940***	
	(0.0459)	
preMMR ∗ *post*1937		-0.2234***
		(0.0396)
N	667	667
R^2	0.8603	0.9067

These are OLS regressions (standard errors in parentheses) at the state-year level. The dependent variables are *Pneumonia*, the state-year average mortality rate from pneumonia, and *MMR*, the state-year average maternal mortality rate. *prePneumonia* ∗ *post*1937 and *preMMR* ∗ *post*1937 are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. The regressions also include state and year fixed effects. *denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C Heterogeneity of main effects by race and education

In this Section we discuss the heterogeneity of our main estimated effects with respect to race and education. Bhalotra and Venkataramani (2012) find that the positive effects of sulfa drugs on the human capital of children born in the antibiotics era were focused among white children, and explain this by arguing that the returns to human capital for blacks were limited due to institutional constraints. The decision to delay fertility and enter the labor market will be affected by constraints to workforce participation and promotion, as well as the relative return of working and waiting, compared to not working and having children immediately. These constraints and relative returns are likely to be affected by education.

In the hazard model, we find a stronger estimated effect of reductions in pneumonia and maternal mortality on birth probability for blacks than for whites (Table 16), while we find no difference among the college educated compared to those who did not finish high school. Blacks experienced larger reductions in mortality rates than whites (average pre-sulfa pneumonia mortality was 1.2 among blacks and 1.1 among whites while the same figures for maternal mortality are 7.5 and 6.0 respectively), which can partly explain the larger magnitude of the estimated effect. There was no difference in the average mortality rates between the college educated and those who did not complete high school. In columns (5) and (6) of the same table, we estimate the logistic survival model to first birth with interactions between the treatment variables, race and education dummies. There is no statistically significant difference in fertility delay between blacks and whites, and between the college educated and high school dropouts.

Turning to estimates of the stock model by education and race, we find larger fertility effects among blacks than whites, consistent with the findings in the hazard model, and no differences by education, except that reductions in maternal mortality have a particularly significant, negative estimated effect on gross childlessness among the college educated. Both the college educated and those who did not complete high school have similar estimated labor market responses to reductions in mortality, but we find negative (although insignificant) effects on marriage rates for those without completed high school.

Interestingly, we find virtually zero estimated effects of mortality reductions on blacks' labor market behavior, even though a larger proportion of black women reported being in the labor force compared to white women (41% vs. 34%), suggesting that there were institutional constraints to promotion opportunities among blacks. For both groups we estimate a negative, significant estimated effect of mortality reductions on the probability of marriage. We uncover a positive, significant estimated effect of pneumonia mortality decline on the age at first marriage among white women, suggesting that they delayed marriage in response to mortality decline. We also estimate a negative, significant effect of maternal mortality decline on age at first marriage in this group. The effect of pneumonia mortality decline on age at first marriage for black women is estimated to be negative; however, this estimate is from a substantially smaller sample than the other specifications, and should be interpreted with caution.

Table 16: Hazard model: Heterogeneous effects by race and education

	(1) - White	(2) - Black	(3) - College	(4) - HS Dropout
			Birth	
$prePneumonia * post1937$	-0.0177**	-0.0551***	-0.0235***	-0.0266**
	(0.0090)	(0.0099)	(0.0063)	(0.0109)
$preMMR * post1937$	0.0023	0.0026	0.0014	0.0034
	(0.0021)	(0.0021)	(0.0015)	(0.0027)
N	4021342	461423	286975	3594323

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. The first column restricts the sample to whites, the second to blacks, the third to those with some college and the fourth to high school dropouts. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 17: Fertility in the stock model by race

	Net Fertility			Gross Fertility		
	(1) # Children	(2) # Children | Children >0	(3) Childless	(4) # Children	(5) # Children | Children >0	(6) Childless
Panel A. Whites						
$prePneumonia * Sulfayears$	-0.0403**	-0.0318**	0.0078***	-0.0132	-0.0129	0.0016
	(0.0156)	(0.0142)	(0.0028)	(0.0126)	(0.0122)	(0.0010)
$preMMR * Sulfayears$	0.0027	0.0006	-0.0007*	-0.0040	-0.0009	-0.0000
	(0.0027)	(0.0029)	(0.0004)	(0.0023)	(0.0023)	(0.0001)
N	438414	280761	438414	472851	386482	472851
Panel B. Blacks						
$prePneumonia * Sulfayears$	-0.0743***	-0.0492*	0.0128***	-0.0328**	-0.0164	0.0032
	(0.0200)	(0.0254)	(0.0035)	(0.0130)	(0.0150)	(0.0021)
$preMMR * Sulfayears$	0.0109***	0.0106***	-0.0013**	-0.0009	-0.00274	-0.0002
	(0.0030)	(0.0039)	(0.0006)	(0.0023)	(0.0028)	(0.0004)
N	53749	31714	53749	43470	33271	43470

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). Panel A restricts the sample to whites only, and Panel B restricts the sample to blacks only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 18: Fertility in the stock model by education

	Net Fertility			Gross Fertility		
	(1) # Children	(2) # Children | Children >0	(3) Childless	(4) # Children	(5) # Children | Children >0	(6) Childless
Panel A. At Least Some College						
$prePneumonia * Sulfayears$	-0.0469***	-0.0620***	0.0020	-0.0262**	-0.0241**	0.0023
	(0.0134)	(0.0130)	(0.0052)	(0.0107)	(0.0113)	(0.0019)
$preMMR * Sulfayears$	0.0063**	0.0061**	-0.0007	0.0028*	-0.0001	-0.0009**
	(0.0031)	(0.0027)	(0.0011)	(0.0015)	(0.0019)	(0.0004)
N	36204	20733	36204	74577	56158	74577
Panel B. High School Dropouts						
$prePneumonia * Sulfayears$	-0.0584***	-0.0413***	0.0102***	-0.0364**	-0.0337**	0.0024*
	(0.0136)	(0.0129)	(0.0022)	(0.0160)	(0.0150)	(0.0013)
$preMMR * Sulfayears$	0.0042	0.0011	-0.0011**	0.0006	0.0002	0.0001
	(0.0028)	(0.0027)	(0.0004)	(0.0025)	(0.0024)	(0.0002)
N	295665	217949	295665	292285	241372	292285

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). Panel A restricts the sample to those with some college only, and Panel B restricts the sample to those who did not complete high school. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 19: Labor market outcomes as a function of sulfa exposure by race

	(1) Working	(2) In Labor Force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
Panel A. Whites					
$prePneumonia * sulfayears$	0.0057***	0.0054***	0.1707*	4.8582	0.2077**
	(0.0018)	(0.0018)	(0.0853)	(16.1432)	(0.0676)
$preMMR * sulfayears$	-0.0005*	-0.0005*	0.0067	0.5119	-0.0146
	(0.0003)	(0.0003)	(0.0137)	(2.9529)	(0.0107)
N	649136	649136	462266	277065	649136
Panel B. Blacks					
$prePneumonia * sulfayears$	-0.0021	-0.0010	0.0945	52.3038**	0.1165
	(0.0031)	(0.0032)	(0.1568)	(22.0283)	(0.1411)
$preMMR * sulfayears$	-0.0013**	-0.0012**	0.0034	5.6628*	-0.0840***
	(0.0006)	(0.0006)	(0.0264)	(2.8950)	(0.0215)
N	75014	75014	53061	27762	75014

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where intervalled data is converted to a continuous measure using the midpoints of the intervals. Panel A restricts the sample to whites only, and Panel B restricts the sample to blacks only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 5-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 (1900-1931 for column 5) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 20: Labor market outcomes as a function of sulfa exposure by education

	(1) Working	(2) In Labor Force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
Panel A. At Least Some College					
$prePneumonia * sulfayears$	0.0066*	0.0070**	0.3603	41.2755	0.2387*
	(0.0033)	(0.0033)	(0.2924)	(31.0808)	(0.1133)
$preMMR * sulfayears$	-0.0000	-0.0001	0.0027	-4.0435	0.0131
	(0.0006)	(0.0007)	(0.0518)	(7.1769)	(0.0161)
N	61894	61894	43375	43343	66641
Panel B. High School Dropouts					
$prePneumonia * sulfayears$	0.0051**	0.0044**	0.1609*	14.0886	0.3073**
	(0.0021)	(0.0022)	(0.0862)	(15.3356)	(0.1004)
$preMMR * sulfayears$	-0.0006*	-0.0007*	-0.0026	-1.9909	-0.0434**
	(0.0004)	(0.0004)	(0.0151)	(2.4924)	(0.0161
N	443874	443874	330023	142950	494514

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where we convert intervalled data to a continuous measure using the midpoint of each interval. Panel A restricts the sample to those with at least some college, and Panel B restricts the sample to those who did not complete high school only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 5-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 (1900-1931 for column 5) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 21: Marriage market outcomes as a function of sulfa exposure by race

	(1) Currently Married	(2) Ever Married	(3) Age at 1st marriage
Panel A. Whites			
$prePneumonia * sulfayears$	-0.0041** (0.0018)	-0.0033** (0.0015)	0.0434* (0.0249)
$preMMR * sulfayears$	0.0011*** (0.0003)	0.0008*** (0.0002)	-0.0052 (0.0052)
N	438414	649136	104643
Panel B. Blacks			
$prePneumonia * sulfayears$	-0.0029 (0.0049)	-0.0058** (0.0026)	-0.1698*** (0.0520)
$preMMR * sulfayears$	-0.0013* (0.0007)	-0.0001 (0.0003)	-0.0044 (0.0086)
N	53749	75014	11477

The dependent variables are: (1) a dummy variable equal to one equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage, only defined for ever married women. Panel A restricts the sample to whites only, and Panel B restricts the sample to blacks only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and influenza and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 22: Marriage market outcomes as a function of sulfa exposure, by education

	(1) Currently Married	(2) Ever Married	(3) Age at 1st marriage
Panel A. At Least Some College			
$prePneumonia * sulfayears$	0.0006	0.0005	-0.0341
	(0.0039)	(0.0031)	(0.0570)
$preMMR * sulfayears$	-0.0001	0.0001	-0.0022
	(0.0009)	(0.0006)	(0.0121)
N	36204	61894	14587
Panel B. High School Dropouts			
$prePneumonia * sulfayears$	-0.0013	-0.0018*	0.0157
	(0.0014)	(0.0010)	(0.0289)
$preMMR * sulfayears$	0.0006*	0.0004**	0.0045
	(0.0003)	(0.0002)	(0.0055)
N	295665	443874	56323

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage, only defined for ever married women. Panel A restricts the sample to those with at least some college, and Panel B restricts the sample to those who did not complete high school only. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year and race fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

D Robustness checks

Results tables for robustness checks discussed in main text

Table 23: Probability of birth as a function of sulfa exposure - robustness checks

	(1) - WW2	(2) - New Deal	(3) - Dust Bowl Birth	(4) - Mean Reversion	(5) - Under5s	(6) - 2SLS
$prePneumonia * post1937$	-0.0239**	-0.0247**	-0.0247**	-0.0251**		
	(0.0105)	(0.0107)	(0.0106)	(0.0103)		
$preMMR * post1937$	0.0041	0.0031	0.0048*	0.0029	0.0020	0.0056
	(0.0026)	(0.0024)	(0.0026)	(0.0023)	(0.0021)	(0.0046)
$prePneumoniaU5 * post1937$					-0.0006	-0.0039*
					(0.0008)	(0.0023)
N	4053834	4499588	4053176	4499588	4499588	4499792

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic (columns 1-5) and OLS 2SLS (column 6) regressions with standard errors (in parentheses) clustered at the state of birth level. For comparison, the coefficient on $prePneumoniaU5 * sulfayears$ when estimating column (5) using OLS is -0.0008 (s.e. 0.0010). The robustness check in each column is described in detail in Section 5.3. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 24: Fertility outcomes as a function of sulfa exposure - robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel A (New Deal)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0417***	-0.0316**	0.0074***	-0.0210	-0.0186	0.0020*
	(0.0140)	(0.0125)	(0.0024)	(0.0134)	(0.0119)	(0.0010)
$preMMR * sulfayears$	0.0033	0.0008	-0.0009**	0.0007	0.0003	-0.0000
	(0.0024)	(0.0026)	(0.0003)	(0.0023)	(0.0021)	(0.0002)
N	494437	313981	494437	518933	421983	518933
Panel B (WW2)						
$prePneumonia * sulfayears$	-0.0442*	-0.0218	0.0113***	-0.0172	-0.0150	0.0020**
	(0.0235)	(0.0189)	(0.0039)	(0.0115)	(0.0103)	(0.0009)
$preMMR * sulfayears$	0.0003	-0.0033	-0.0013**	0.0005	0.0001	0.0000
	(0.0038)	(0.0035)	(0.0006)	(0.0021)	(0.0019)	(0.0001)
N	317789	230684	317789	518832	421898	518832
Panel C (Dust Bowl)						
$prePneumonia * sulfayears$	-0.0499***	-0.0358***	0.0092***	-0.0267**	-0.0233**	0.0025**
	(0.0127)	(0.0123)	(0.0022)	(0.0117)	(0.0105)	(0.0010)
$preMMR * sulfayears$	0.0033	0.0001	-0.0010**	0.0011	0.0006	-0.0000
	(0.0023)	(0.0024)	(0.0004)	(0.0022)	(0.0020)	(0.0001)
N	444734	280263	444734	463435	376022	463435
Panel D (Mean reversion)						
$prePneumonia * sulfayears$	-0.0570***	-0.0416***	0.0106***	-0.0234*	-0.0197	0.0021**
	(0.0123)	(0.0124)	(0.0020)	(0.0130)	(0.0118)	(0.0009)
$preMMR * sulfayears$	0.0050**	0.0019	-0.0013***	0.0010	0.0004	-0.0000
	(0.0024)	(0.0026)	(0.0004)	(0.0024)	(0.0022)	(0.0002)
N	494437	313981	494437	518933	421983	518933

See notes to Tables 5 and 7 for definitions of outcomes. The robustness checks are described in Section 5.3. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the U.S. and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940-1970 US decennial population censuses. See notes to Table 33 for further details on estimation and control variables. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 25: Fertility outcomes as a function of sulfa exposure - further robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel E (Placebo)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.2112	-0.3751	-0.0170			
	(0.2053)	(0.2319)	(0.0323)			
$preMMR * sulfayears$	-0.0080	0.0604	0.0153			
	(0.0449)	(0.0493)	(0.0063)			
N	61918	42447	61918			
Panel F (Under 5s Pneu)						
$prePneumoniaU5 * sulfayears$	-0.0025**	-0.0008	0.0006***	-0.0009	-0.0008	0.0001*
	(0.0010)	(0.0009)	(0.0002)	(0.0009)	(0.0008)	(0.0001)
$preMMR * sulfayears$ 0.0019	-0.0011	-0.0007*	-0.0001	-0.0003	0.0000	
	(0.0028)	(0.0026)	(0.0004)	(0.0024)	(0.0023)	(0.0002)
N	494437	313981	494437	518933	421983	518933
Panel G (2SLS)						
$prePneumoniaU5 * sulfayears$	-0.0071***	-0.0050**	0.0013***	-0.0032	-0.0028	0.0003*
	(0.0027)	(0.0024)	(0.0004)	(0.0022)	(0.0019)	(0.0002)
$preMMR * sulfayears$	0.0062	0.0028	-0.0014**	0.0019	0.0014	-0.0001
	(0.0046)	(0.0045)	(0.0006)	(0.0037)	(0.0032)	(0.0003)
N	494437	313981	494437	518933	421983	518933
Panel H (Excl. under 5s)						
$prePneumonia * sulfayears$	-0.0393***	-0.0309***	0.0091***			
	(0.0097)	(0.0076)	(0.0027)			
$preMMR * sulfayears$	0.0032*	0.0007	-0.0012***			
	(0.0018)	(0.0017)	(0.0004)			
N	494437	237498	494437			

See notes to Tables 5 and 7 for definitions of outcomes. The robustness checks are described in Section 5.3. For Panel E, our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1897, with outcomes drawn from the 1910-1930 censuses. For other panels, our dataset is a cross-section of outcomes of women aged 5-44 in 1937 and 18-40 at census (columns 1-3) or at least 40 (columns 4-6), born in the U.S. and resident in their birth state at census. The cohorts in this table were born in 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940-1970 US decennial population censuses. See notes to Table 33 for details on estimation and control variables. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 26: Binary difference-in-difference estimates of the effect of sulfa exposure on fertility

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * treated$	-0.8565***	-0.7925**	0.1386**	-0.4930	-0.5562	0.0325
	(0.3830)	(0.3472)	(0.0676)	(0.3667)	(0.3502)	(0.0253)
$preMMR * treated$	0.1022	0.0724	-0.0181*	-0.0164	-0.0105	0.0032
	(0.0643)	(0.0581)	(0.0102)	(0.0567)	(0.0524)	(0.0044)
N	279899	182808	279899	163036	137303	163036

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). *treated* is a variable that equals one if a woman was exposed to sulfa drugs for her entire fertile period, and equals zero if a woman was exposed for no years. Women exposed for only some years are excluded from these regressions. $prePneumonia$ and $preMMR$ are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-15 or 40-44 in 1937 and 18-50 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1897 and 1922-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *treated*. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 27: Linear trends in birth probability in above and below median mortality states, 1930-1936

	(1)	(2) Birth	(3)
$AboveMedPneu * trend$	-0.0001	-0.0001	
	(0.0002)	(0.0003)	
$AboveMedMMR * trend$	0.0003		0.0003
	(0.0003)		(0.0003)
N	2230331	2230331	2230331

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. The variable *trend* is a linear time trend. *AboveMedPneu* is a dummy variable that equals one if a state had an above median average value of *prePneumonia* in 1930-1936 and zero otherwise. The variable *AboveMedMMR* has the analogous definition for *preMMR*. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1936, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *post*1937. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 28: Migration as a function of sulfa exposure

	(1) Pr(in migrant sample)	(2) Migrated between 1935-40
$prePneumonia * sulfayears$	0.0020	-0.0045
	(0.0031)	(0.0072)
$preMMR * sulfayears$	-0.0005	0.0001
	(0.0005)	(0.0014)
N	1122827	56722

The dependent variable in column 1 is a dummy variable that equals one if a woman's census state is different from her birth state, and zero otherwise. The dependent variable in column 2 equals one if a woman reported in the 1940 census that she has migrated in the last 5 years, and equals zero if she reported that she did not migrate. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and at least 40 at the time of the census and born in the United States. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses (column 1) and the 1940 US decennial population census (column 2). Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 29: Labor outcomes as a function of sulfa exposure - robustness checks

Panel A (New Deal)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0055***	0.0051***	0.3308**	29.8135***	0.2295***
	(0.0016)	(0.0017)	(0.1472)	(10.2308)	(0.0637)
$preMMR * sulfayears$	-0.0008**	-0.0008**	0.0218	-0.5819	-0.0320**
	(0.0003)	(0.0003)	(0.0292)	(1.6195)	(0.0114)
N	727398	727398	247015	306280	727398
Panel B (WW2)					
$prePneumonia * sulfayears$	0.0070***	0.0066***	0.3695***	14.5503	0.2931***
	(0.0017)	(0.0017)	(0.1293)	(14.5801)	(0.0533)
$preMMR * sulfayears$	-0.0003	-0.0002	0.0176	-0.6242	-0.0113
	(0.0003)	(0.0003)	(0.0271)	(2.6723)	(0.0092)
N	517746	517746	246952	306209	517746
Panel C (Dust Bowl)					
$prePneumonia * sulfayears$	0.0057***	0.0053***	0.3128*	1.7650	0.2395***
	(0.0017)	(0.0018)	(0.1627)	(14.9465)	(0.0639)
$preMMR * sulfayears$	-0.0009***	-0.0009***	0.0218	0.4506	-0.0375**
	(0.0003)	(0.0003)	(0.0313)	(2.3797)	(0.0113)
N	654187	654187	223428	274954	654187
Panel D (Mean reversion)					
$prePneumonia * sulfayears$	0.0059***	0.0053***			
	(0.0017)	(0.0017)			
$preMMR * sulfayears$	-0.0007**	-0.0007**			
	(0.0003)	(0.0003)			
N	727398	727398			

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, converted from intervalled data to a continuous variable using the midpoints of each interval. The robustness checks in the different panels are described in Section 6.5. See notes to Table 33 for further details on sampling, control variables and estimation. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 30: Marriage market outcomes as a function of sulfa exposure - robustness checks

Panel A (New Deal)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0020	-0.0024*	0.0152
	(0.0013)	(0.0012)	(0.0245)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0050
	(0.0002)	(0.0002)	(0.0050)
N	494437	727398	116632
Panel B (WW2)			
$prePneumonia * sulfayears$	-0.0051**	-0.0042***	-0.2328
	(0.0025)	(0.0014)	(0.2526)
$preMMR * sulfayears$	0.0013***	0.0005**	-0.0188
	(0.0004)	(0.0002)	(0.0372)
N	317789	517746	81854
Panel C (Dust Bowl)			
$prePneumonia * sulfayears$	-0.0023*	-0.0031**	-0.0071
	(0.0012)	(0.0012)	(0.0260)
$preMMR * sulfayears$	0.0005**	0.0005***	0.0037
	(0.0002)	(0.0002)	(0.0065)
N	444734	654187	103929
Panel D (Mean reversion)			
$prePneumonia * sulfayears$	-0.0023*	-0.0031**	0.0157
	(0.0012)	(0.0012)	(0.0233)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0039
	(0.0002)	(0.0002)	(0.0046)
N	494437	727398	116632

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage for ever married women. The robustness checks in the different panels are described in Section 6.5. See notes to Table 34 for details on sampling, control variables and estimation. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 31: Binary difference-in-difference estimates of the effect of sulfa exposure on labor market outcomes

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * treated$	0.1339**	2.3366***	-3303.3419***	-0.0440	4.5693
	(0.0588)	(0.0597)	(0.5128)	(64.3696)	(2.4842)
$preMMR * treated$	-0.0295***	-0.0335***	2.8876***	338.6701***	-1.2668**
	(0.0102)	(0.0103)	(0.0474)	(17.5327)	(0.4305)
N	279899	279899	245681	168344	279899

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the past week, where we convert the intervalled measure to a continuous measure using the midpoint of each interval. *treated* is a variable that equals one if a woman was exposed to sulfa drugs for her entire fertile period, and equals zero if a woman was exposed for no years. Women exposed for only some years are excluded from these regressions. *prePneumonia* and $preMMR$ are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 5-15 or 40-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1897 and 1922-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *treated*. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 32: Binary difference-in-difference estimates of the effect of sulfa exposure on marriage market outcomes

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * treated$	-0.0440	-0.0998**	0.3031
	(0.0398)	(0.0376)	(0.6092)
$preMMR * treated$	0.0201***	0.0175***	0.2636***
	(0.0062)	(0.0063)	(0.0986)
N	279899	279899	86390

The dependent variables are: (1) a dummy variable equal to one equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage, only defined for ever married women. *treated* is a variable that equals one if a woman was exposed to sulfa drugs for her entire fertile period, and equals zero if a woman was exposed for no years. Women exposed for only some years are excluded from these regressions. *prePneumonia* and *preMMR* are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 5-15 or 40-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1897 and 1922-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *treated*. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 33: Labor market outcomes as a function of sulfa exposure - further robustness checks

Panel E (Placebo)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0102	0.0211			
	(0.0388)	(0.0435)			
$preMMR * sulfayears$	0.0057	0.0068			
	(0.0083)	(0.0095)			
N	54852	54842			
Panel F (Under 5s Pneu)					
$prePneumoniaU5 * sulfayears$	0.0003***	0.0003**	0.0163***	0.2278	0.0131***
	(0.0001)	(0.0001)	(0.0051)	(0.9473)	(0.0043)
$preMMR * sulfayears$	-0.0006*	-0.0007**	0.0011	0.2777	-0.0250**
	(0.0003)	(0.0003)	(0.0120)	(2.5478)	(0.0119)
N	727398	727398	517857	306280	727398
Panel G (2SLS)					
$prePneumoniaU5 * sulfayears$	0.0009***	0.0008**	0.0290**	1.0635	0.0365***
	(0.0003)	(0.0003)	(0.0125)	(2.2722)	(0.0136)
$preMMR * sulfayears$	-0.0011**	-0.0011**	-0.0109	-0.4720	-0.0468**
	(0.0006)	(0.0005)	(0.0194)	(3.3553)	(0.0230)
N	727398	727398	517857	306280	727398

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the past week, converted from intervalled data to a continuous variable using the midpoint of each interval. The robustness checks in the different panels are described in Section 6.5. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. For Panel E, our dataset is a cross-section of labor outcomes of women aged 5-44 in 1897, with labor outcomes drawn from the 1910, 1920 and 1930 censuses. Our dataset for panels F-G is a cross-section of labor outcomes of women aged 5-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 34: Marriage market outcomes as a function of sulfa exposure - further robustness checks

Panel E (Placebo)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	0.0003	-0.0018	-0.2592***
	(0.0326)	(0.0024)	(0.0112)
$preMMR * sulfayears$	0.0066	-0.0052	-0.0003
	(0.0073)	(0.0005)	(0.0033)
N	61918	135524	7625
Panel F (Under 5s Pneu)			
$prePneumoniaU5 * sulfayears$	-0.0001*	-0.0002**	0.0009
	(0.0001)	(0.0001)	(0.0021)
$preMMR * sulfayears$	0.0006**	0.0005**	0.0048
	(0.0002)	(0.0002)	(0.0057)
N	494437	727398	116632
Panel G (2SLS)			
$prePneumoniaU5 * sulfayears$	-0.0003*	-0.0005**	0.0003
	(0.0002)	(0.0002)	(0.0035)
$preMMR * sulfayears$	0.0007***	0.0008**	0.0052
	(0.0003)	(0.0003)	(0.0061)
N	494437	727398	116632

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at first marriage for ever married women. The robustness checks in the different panels are described in Section 6.5. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. The sample includes women aged 18-40 at the time of the census for columns 1 and 3 and 18-50 for column 2. For Panel E, our dataset is a cross-section of marriage outcomes of women aged 5-44 in 1897, with outcomes drawn from the 1910, 1920 and 1930 censuses. For Panels F-G, our dataset is a cross-section of marriage outcomes of women aged 5-44 in 1937, born in the United States and resident in their birth state at the time of the census. The cohorts were born in the years 1900-1931 for columns 1 and 3 and 1893-1931 for column 2 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Alternative sample definitions

First, we show that our stock model results are not sensitive to sample definitions. We reestimate the net fertility results for 18-36 year olds at the time of the census (a child born to a woman aged 18 would leave home at 36, hence this measure minimises underreporting of children who have left home). These results are in Table 35. All the results are statistically significant and the magnitudes are comparable to those in the main text. Table 36 complements this analysis by presenting results for gross uncompleted fertility; that is, gross fertility for 18-40 year olds. The coefficients are comparable in magnitude to the main text, although they are not precisely estimated; this is likely driven by the fact that the gross fertility question was only asked to ever married women in the 1940 and 1950 censuses, and 95% of the sample in these regressions comes from these two censuses. As the main results suggest that fertility and marriage decisions are intertwined, restricting the sample to ever married women leads to a select sample of women.

In Table 37, we show that the labor supply results are robust to using a sample of 18-40 year olds and 18-60 year olds; as with the fertility results, the coefficients have the largest magnitudes for the youngest sample. This Table also shows robustness to widening the marriage market sample. When we widen the sample to include women up to age 60, the coefficient for current marital status is attenuated and insignificant, showing the importance of precise sample definitions.

Outliers

Next, we reestimate the main results but excluding New Mexico, which was shown to be an outlier state in Figure 13. The hazard model results are in column (1) of Table 39, while the stock model results are in Panel I of Tables 40 (fertility) and 41-42 (labor and marriage markets). The exclusion of New Mexico does not change the results in a substantive way.

Bias from considering only one source of mortality

To show the importance of considering both pneumonia mortality and maternal mortality as impacting outcomes, we exclude first one and then the other in the regressions. This is columns (2) and (3) in Table 39 and Panels J and K in Tables 40-42. Overall, the specifications perform poorly with the omission of the other treatment variable. The only estimated effects that are robust to this alteration are those on net fertility, probability of work and probability of birth; the other coefficients are mostly attenuated and insignificant. This suggests that omitting one variable biases the coefficient on the other.

Prescription check

In column (4) of Table 39, we exclude all observations from the hazard model after prescriptions were introduced for sulfa drugs, that is, all observations post-1939. The coefficient on pneumonia mortality remains statistically significant.

OLS and Woman Fixed Effects

In order to verify that our results are similar in a simpler procedure, we estimate the hazard model using OLS (Table 39). In the same table, to control for time invariant unobserved factors at the woman level that affect birth probability and potentially are also correlated with mortality rates, we estimate the hazard model with woman fixed effects. The coefficients are similar to the

main results in Table 4, but the coefficients are less precisely estimated.

Multiple hypothesis testing

Finally, in Panel L of Table 41, we adjust the standard errors from the main results (Table 9) for multiple hypothesis testing. (We do not adjust the standard errors for fertility because these variables are all defined based on one originating variable.) In particular, we implement the procedure described in Aker, Boumnijel, McClelland, and Tierney 2014, which adjusts standard errors to take into account correlation between outcomes. The formula for the adjusted p-values is

$$\begin{aligned} p^{new} &= 1-(1-p^{old})^{A} \\ A &= (1-c)^{\#outcomes}, \end{aligned}$$

where c is the average correlation between all other outcomes in the group. As we only consider two marriage market outcomes, this formula can only be implemented for the labor market outcomes. The adjusted standard errors do not change the significance of the results in a substantive way.

Table 35: Net fertility as a function of sulfa exposure: 18-36 year old women

	(1) # Children	(2) # Children | Children >0	(3) Childless
$prePneumonia * sulfayears$	-0.0408***	-0.0297**	0.0089***
	(0.0119)	(0.0130)	(0.0029)
$preMMR * sulfayears$	0.0029	0.0010	-0.0008*
	(0.0024)	(0.0028)	(0.0005)
N	393720	234416	393720

The dependent variables are the total number of children (column 1), the total number of children conditional on having at least one (column 2) and a dummy variable that equals one if the woman has zero children and zero otherwise (column 3), all based on net fertility (the number of own children living in the household). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-36 at census enumeration, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years1904-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 36: Gross fertility as a function of sulfa exposure: 18 to 40 year old women

	(1) # Children	(2) # Children | Children >0	(3) Childless
$prePneumonia * sulfayears$	-0.0288	-0.0275	0.0029
	(0.0201)	(0.0205)	(0.0029)
$preMMR * sulfayears$	0.00003	0.00002	-0.0000
	(0.00004)	(0.00005)	(0.0000)
N	170537	138760	170537
Controls	0.0700	0.0711	0.0381

The dependent variables are the total number of children (column 1), the total number of children conditional on having at least one (column 2) and a dummy variable that equals one if the woman has zero children and zero otherwise (column 3), all defined by gross fertility (the number of live births a woman ever had). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 37: Labor market outcomes as a function of sulfa exposure - alternative samples

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
Panel A			18 to 40 year old women		
$prePneumonia * sulfayears$	0.0074**	0.0064*	0.2727*	-5.3710	0.3561***
	(0.0025)	(0.0026)	(0.1351)	(12.0006)	(0.0878)
$preMMR * sulfayears$	-0.0012**	-0.0012**	-0.0291	-2.7448	-0.0525***
	(0.0004)	(0.0004)	(0.0207)	(1.7008)	(0.0145)
N	494437	494437	317867	152468	494437
Panel B			18 to 60 year old women		
$prePneumonia * sulfayears$	0.0043**	0.0040**	0.1481*	-9.0074	0.1775**
	(0.0015)	(0.0015)	(0.0570)	(11.9414)	(0.0518)
$preMMR * sulfayears$	-0.0005*	-0.0005*	0.0030	-0.1509	-0.0251**
	(0.0002)	(0.0002)	(0.0100)	(2.6067)	(0.0084)
N	922769	922769	713228	482383	922769

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where intervalled data is converted to a continuous measure using the midpoint of each interval. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 5-44 in 1937, born in the United States and resident in their birth state at the time of the census, with age at census restrictions shown above the relevant columns in the table. The cohorts in this table were born in the years 1900-1931 (Panel A) or 1893-1931 (Panel B) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 38: Marriage market outcomes as a function of sulfa exposure - alternative samples

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
Panel A		18 to 50 year old women	
$prePneumonia * sulfayears$	-0.0013	-0.0032**	-0.0170
	(0.0011)	(0.0012)	(0.0152)
$preMMR * sulfayears$	0.0006***	0.0006***	0.0033
	(0.0002)	(0.0002)	(0.0022)
N	727398	727398	181562
Panel B		18 to 60 year old women	
$prePneumonia * sulfayears$	-0.0005	-0.0025**	-0.0294*
	(0.0011)	(0.0011)	(0.0155)
$preMMR * sulfayears$	0.0004***	0.0004**	0.0045
	(0.0001)	(0.0002)	(0.0027)
N	922769	922769	309077

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) age at first marriage, only defined for ever married women. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 5-44 in 1937, born in the United States and resident in their birth state at the time of the census, with age at census restrictions shown above the relevant columns in the table. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 39: Probability of birth as a function of sulfa exposure - additional robustness checks

	(1) - Excl. New Mexico	(2) - Pneu	(3) - MMR	(4) - Excl. post 1939 Birth	(5) - OLS	(6) - OLS+WFE
$prePneumonia * post1937$	-0.0241**	-0.0158*		-0.0256**	-0.0253*	-0.0185
	(0.0104)	(0.0084)		(0.0113)	(0.0134)	(0.0209)
$preMMR * post1937$	0.0037		0.0014	0.0048***	0.0043	0.0054
	(0.0025)		(0.0023)	(0.0028)	(0.0032)	(0.0045)
N	4485763	4499588	4499588	3417407	4499792	4499792

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1928 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with *post*1937. Column (6) adds woman fixed effects. Columns (5) and (6) are estimated using OLS. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 40: Net and gross fertility as a function of sulfa exposure - additional robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
Panel I (Excl. New Mexico)	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0474***	-0.0341***	0.0088***	-0.0207*	-0.0184*	0.0021**
	(0.0124)	(0.0117)	(0.0023)	(0.0119)	(0.0107)	(0.0009)
$preMMR * sulfayears$	0.0040*	0.0011	-0.0010***	0.0011	0.0007	-0.00003
	(0.0023)	(0.0026)	(0.0003)	(0.0023)	(0.0021)	(0.0001)
N	492776	312786	492776	517651	420859	517651
Panel J (Pneu only)						
$prePneumonia * sulfayears$	-0.0397***	-0.0324***	0.0067**	-0.0192*	-0.0179*	0.0021**
	(0.0131)	(0.0097)	(0.0027)	(0.0100)	(0.0093)	(0.0009)
N	494437	313981	494437	518933	421983	518933
Panel K (MMR only)						
$preMMR * sulfayears$	-0.0005	-0.0019	-0.0002	-0.0009	-0.0011	0.0002
	(0.0030)	(0.0025)	(0.0005)	(0.0021)	(0.0020)	(0.0002)
N	494437	313981	494437	518933	421983	518933

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (columns 1-3) and 1893-1931 (columns 4-6) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 41: Labor market outcomes as a function of sulfa exposure - additional robustness checks

Panel I (Excl. New Mexico)	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1970**	7.9247	0.2419***
	(0.0017)	(0.0018)	(0.0750)	(15.3758)	(0.0634)
$preMMR * sulfayears$	-0.0008***	-0.0008***	-0.0023	-0.0013	-0.0347**
	(0.0003)	(0.0003)	(0.0120)	(2.7073)	(0.0109)
N	725118	725118	516184	305575	725118
Panel J (Pneu only)					
$prePneumonia * sulfayears$	0.0040**	0.0036*	0.2000**	7.0433	0.1642*
	(0.0017)	(0.0018)	(0.0753)	(13.2783)	(0.0662)
N	727398	727398	517857	306280	727398
Panel K (MMR only)					
$preMMR * sulfayears$	-0.0003	-0.0004	0.0166	0.4821	-0.0128
	(0.0003)	(0.0003)	(0.0140)	(2.3500)	(0.0136)
N	727398	727398	517857	306280	727398
Panel L (Mult. Hypothesis)					
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1991**	7.7366	0.2421***
	(0.0018)	(0.0019)	(0.0857)	(54.9474)	(0.0675)
$preMMR * sulfayears$	-0.0008**	-0.0008**	0.0004	-0.1243	-0.0322***
	(0.0003)	(0.0003)	(0.6441)	(234.485)	(0.0121)
N	727398	727398	517857	306451	727398

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the past week, where intervalled data is converted to a continuous measure using the midpoint of each interval. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor outcomes of women aged 5-44 in 1937 and 18-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 42: Marriage market outcomes as a function of sulfa exposure - additional robustness checks

Panel I (Excl. New Mexico)	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0023*	-0.0032**	0.0010
	(0.0012)	(0.0012)	(0.0242)
$preMMR * sulfayears$	0.0007***	0.0006***	0.0048
	(0.0002)	(0.0002)	(0.0057)
N	492776	725118	116261
Panel J (Pneu only)			
$prePneumonia * sulfayears$	-0.0008	-0.0018	0.0154
	(0.0013)	(0.0013)	(0.0203)
N	494437	727398	116632
Panel K (MMR only)			
$preMMR * sulfayears$	0.0004*	0.0003	0.0055
	(0.0002)	(0.0002)	(0.0048)
N	494437	727398	116632
Panel L (Mult. Hypothesis)			
$prePneumonia * sulfayears$	-0.0023	-0.0032**	0.0021
	(0.0016)	(0.0014)	(0.0652)
$preMMR * sulfayears$	0.0006***	0.0006***	0.00053
	(0.0002)	(0.0002)	(0.0066)
N	494437	727398	116632

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at fist marriage, only defined for ever married women. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of labor and marriage outcomes of women aged 5-44 in 1937 and 18-40 at the time of the census for columns 1 and 3, 18-50 for column 2, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1900-1931 (1893-1931 for column 2) and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

E Sulfa exposure and education

In this Section we present estimates of the effect of sulfa exposure on education outcomes (Table 43), as well as the main estimates for a subgroup of the main sample, namely women who were aged at least 21 in 1937, and hence had ostensibly already made their education choices. We estimate separately the impact of sulfa exposure on the education outcomes of two subsamples of women, corresponding to the two fertility samples: women still of childbearing age at the time of census enumeration (columns 1-3 of Table 43), and women who had completed childbearing at the time of census enumeration (columns 4-6 of Table 43). We estimate a significant, positive impact of pneumonia mortality reduction via sulfa exposure on high school completion in the sample of women of childbearing age of 5.4 percentage points, relative to a baseline mean of 19.2% for this subsample of women. In contrast, the reduction in maternal mortality reduced high school completion and increased the probability that a woman dropped out of high school, consistent with the effects of sulfa exposure on labor market outcomes.

In Tables 44-47, we estimate the main effects (with all controls) but on the subsample of women who were at least 21 in 1937. These estimates are similar to the main results, indicating that education acquisition is not the main channel through which sulfa exposure impacted labor, fertility and marriage market outcomes.

Table 43: Education as a function of sulfa exposure - women aged 15-25 in 1937

	(1) Some College	(2) High School graduate	(3) HS dropout	(4) Some College	(5) High School graduate	(6) HS dropout
	Aged 21-40 at census interview			Aged 40 or older at census interview		
$prePneumonia * treated\_educ$	-0.0113	0.0541**	-0.0428	0.0012	0.0502	-0.0514
	(0.0108)	(0.0264)	(0.0265)	(0.0168)	(0.0347)	(0.0318)
$preMMR * treated\_educ$	-0.0009	-0.0093***	0.0101**	-0.0034	-0.0013	0.0047
	(0.0016)	(0.0033)	(0.0041)	(0.0033)	(0.0032)	(0.0042)
N	199161	199161	199161	186067	186067	186067

The dependent variables are dummy variables for the highest level of education achieved by the woman. $treated\_educ$ takes the value one if a woman was 20 or under in 1937, and the value zero if she was 21 or older. The sample is further restricted to woman aged at least 21 at the time of census enumeration so that they had completed their education. $prePneumonia$ and $preMMR$ are the state-level mortality rates from pneumonia and maternal mortality respectively. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 15-25 in 1937 and 21-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1912-1922 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 44: Probability of birth as a function of sulfa exposure - women aged 21 and over in 1937

	(1) Birth
$prePneumonia * post1937$	-0.0240* (0.0125)
$preMMR * post1937$	0.0007 (0.0030)
N	3306092

The dependent variable is a dummy variable that equals one if a woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ and $preMMR * post1937$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with a dummy variable for the years 1937 and later. These are Logistic regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a panel of woman-year birth outcomes for women aged 15 to 40 in the period 1930-1943, and aged at least 21 in 1937, born in the United States, resident in their birth state at the time of the census. The cohorts in this table were born in the years 1893-1926 and are drawn from the 1940 and 1950 US decennial population censuses. Regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census division*year fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $post1937$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 45: Net and gross fertility as a function of sulfa exposure - women aged 21 and over in 1937

	(1)	(2)	(3)	(4)	(5)	(6)
	Net Fertility			Gross Fertility		
	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0572***	-0.0478***	0.0074***	-0.0314**	-0.0322**	0.0026*
	(0.0158)	(0.0168)	(0.0024)	(0.0128)	(0.0120)	(0.0016)
$preMMR * sulfayears$	0.0062*	0.0048	-0.0007	0.0026	0.0031	0.0000
	(0.0032)	(0.0033)	(0.0005)	(0.0022)	(0.0020)	(0.0004)
N	183740	124714	183740	316159	247946	316159

Net fertility is defined by the number of own children living in the household and gross fertility is defined by the number of live births. The dependent variables are the total number of children (columns 1 and 4), the total number of children conditional on having at least one (columns 2 and 5) and a dummy variable that equals one if the woman has zero children and zero otherwise (columns 3 and 6). $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 21-44 in 1937 and 24-40 at the time of the census for columns 1-3 and at least 40 for columns 4-6, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1916-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 46: Labor market outcomes as a function of sulfa exposure - women aged 21 and over in 1937

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0043*	0.0049*	-0.0500	-15.7802	0.1669
	(0.0024)	(0.0025)	(0.0992)	(21.5968)	(0.1061)
$preMMR * sulfayears$	-0.0007	-0.0009*	0.0250	-2.9891	-0.0320*
	(0.0005)	(0.0005)	(0.0242)	(5.0821)	(0.0187)
N	325467	325467	174280	77937	325467

The dependent variables are: (1) a dummy variable equal to one if the woman reports working at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman is in the labor force and zero otherwise; (3) the Hauser-Warren Socioeconomic Index, based on occupation; (4) the US Dollar amount of personal earnings in the past year; (5) hours worked in the last week, where intervalled data is converted to a continuous measure using the midpoint of each interval. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of fertility outcomes of women aged 21-44 in 1937 and 24-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1916-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 47: Marriage market outcomes as a function of sulfa exposure - women aged 21 and over in 1937

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumonia * sulfayears$	-0.0048*	-0.0026	0.0650
	(0.0025)	(0.0017)	(0.0462)
$preMMR * sulfayears$	0.0008	0.0004	-0.0127
	(0.0005)	(0.0003)	(0.0092)
N	183740	325467	27596

The dependent variables are: (1) a dummy variable equal to one if the woman is married at the time of the census and zero otherwise; (2) a dummy variable equal to one if the woman has ever married in her lifetime and zero otherwise; (3) the age at fist marriage, only defined for ever married women. $prePneumonia * sulfayears$ and $preMMR * sulfayears$ are the state-level mortality rates from pneumonia and maternal mortality respectively, interacted with the number of fertile years (aged 15-40) that a woman was exposed to sulfa drugs. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. Our dataset is a cross-section of marriage outcomes of women aged 21-44 in 1937 and 24-50 at the time of the census, born in the United States and resident in their birth state at the time of the census. The cohorts in this table were born in the years 1916-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Regressions include individual birth state, birth year, race and education fixed effects, as well as state level mortality rates for malaria, heart disease, cancer, tuberculosis and diarrhea among the under 2s, income and public services, literacy, female labor force participation, and the year of state birth and death registration, all interacted with $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

F Cross-sectional relationships between mortality, fertility, labor and marriage choices in 1930 and in 2015

In this Appendix we first explain the construction of the correlational figures in the main text, and then discuss further cross-sectional relationships between mortality rates and women's choices over fertility, the labor market and the marriage market. Figure 13 in the main text shows two plots, each of which are a measure of net fertility plotted against pneumonia mortality. Total net fertility is the weighted state-level average of the number of own children living with a woman at the time of the 1930 census, restricted to women aged 25-40 at the time of the census. Net childlessness takes the value of one when this number is zero and a value of zero otherwise. The charts utilise state-level all-age mortality from pneumonia and influenza 1930-36 per 1000 population, which is the treatment variable used in the main analysis of the paper.

Figure 14 in the main text shows the correlation between two measures of infant mortality, infant mortality from all causes and neonatal mortality from respiratory diseases, and gross fertility (live births) across countries in 2015. We use census data and only include women aged 25 to 40 at the time of the censuses. We observe a negative correlation between infant mortality and childlessness and a positive correlation between infant mortality and total fertility.

In Figure 15, we display these same correlations for two other measures of child mortality: child mortality among under-1s from all causes, and under-2s mortality due to diarrhea. In Figure 16, we plot the same cross-sectional correlations, but with mortality from diseases that did not affect infants: heart disease, diabetes and nephritis (liver disease); these measures are all for the year 1930. We observe a *positive* relationship between mortality and childlessness, and a negative relationship between mortality and fertility; this is consistent with less healthy individuals having fewer children and being more likely to have no children, for example due to sterility or poverty (as in Baudin, de la Croix, and Gobbi 2015).

Figure 17 illustrates time series plots of net fertility and gross fertility, averaged over two groups of states: high child mortality states and low child mortality states. High child mortality states are defined as those with above median pneumonia and influenza mortality in 1930-36, while low child mortality states are defined as those with below median mortality. We used this measure for years before 1930 as well, due to missing mortality data in US Vital Statistics for earlier years, which would introduce a potentially non-random selection bias. Alternative ways of defining high and low mortality states, including using the relationship between earlier and 1930 mortality data to predict earlier mortality rates for states with missing information, produces very similar charts.[53] Childlessness (total fertility) is consistenty higher (lower) among low child mortality states.

[53] In these figures, the rates of net childlessness are fairly high while the rates of gross childlessness are low; this is because net childlessness overestimates true childlessness due to children that have left home, while gross childlessness underestimates true childlessness due to children that have died during infancy. This is potentially an issue if this over or underestimation is correlated with child mortality; this is plausible for gross fertility but perhaps less plausible for net fertility, which would require either earlier age at childbearing or earlier age of children leaving home in low child mortality areas. Further, the difference-in-difference between net and gross fertility and high and low mortality states is stable between 1900-1910.

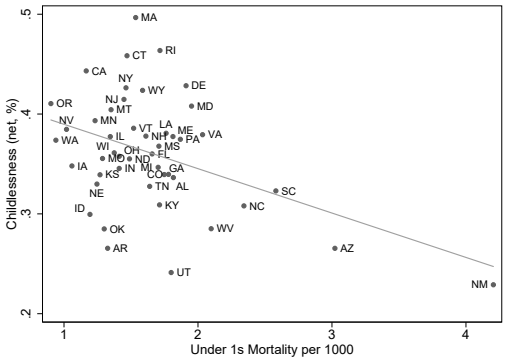
We show cross-sectional relationships between gross fertility and mortality rates in Figures 18 and 19. These are similar to Figure 15, with the issue that the question on gross fertility was only asked in the 1900 and 1910 censuses, and the 1940 and subsequent censuses. As we do not have this information for 1930, we show the data for the closest available pre-sulfa year, namely 1910. An additional complication is that mortality data from the Vital Statistics for this year is missing for more than half of states, which cannot be assumed to be a random sample. Therefore, we plot 1910 gross fertility against 1930 mortality data; this has its obvious concerns and the charts should be interpreted with caution. That being said, we find patterns that are broadly in line with those for 1930 net fertility (Figure 18). Childlessness is decreasing with child mortality, but increasing with adult mortality (Figure 19). Total gross fertility follows approximately the opposite pattern, with the exception of nephritis mortality.

Next, we consider an alternative measure of child mortality, that is measured per live births, rather than per 1000 population as in the preceding charts. This is to show that the observed relationship is not a mechanical one between childlessness and child mortality (high childlessness implies few births, implying lower child mortality). We plot net fertility in 1930 and gross fertility in 1910 against 1929 infant mortality per 1000 live births extracted from the Vital Statistics 1940 yearbook (Dunn 1943, Figure 20). The relationships are very similar to those displayed in the earlier charts.

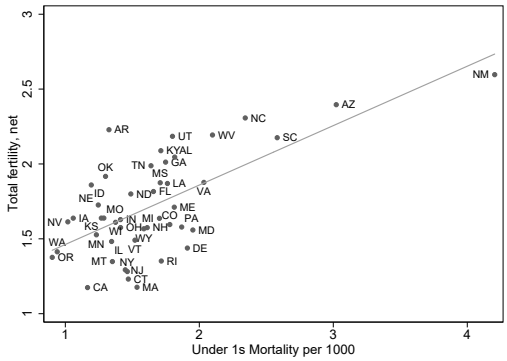
Finally, we consider the cross-sectional relationships between labor market behavior, marriage market behavior and child mortality. To measure labor market behavior, we use the variable In Labor Force, which was available in the 1930 census. For marriage market behavior, we consider the Currently Married question. For both questions, we make the same sample selections as are made in the main analysis of this paper (labor market answers were retained for 18-50 year old women at the time of the census, while currently married answers were retained for 18-40 year old women). Figure 21 shows the labor market plots for child mortality and adult mortality, Figure 22 shows these plots for the marriage market, while the comparable plots for infant mortality per 1000 live births are in Figure 20. Although these Figures will mask many unobservable factors, such as education levels, health status and income, they are surprisingly consistent with the fertility charts. On the whole, working status is negatively correlated with child mortality and positively correlated with adult mortality, although the correlation is at times weak, while marriage rates are positively correlated with child mortality and negative correlated with adult mortality.

Figure 15: Child Mortality and Net Fertility in 1930 across US states

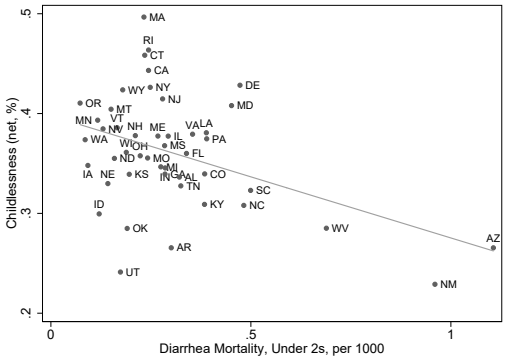
(a) Under 1s Mortality and Net Childessness



(b) Under 1s Mortality and Net Fertility



(c) Diarrhea Under 2s Mortality and Net Childessness



(d) Diarrhea Under 2s Mortality and Net Fertility

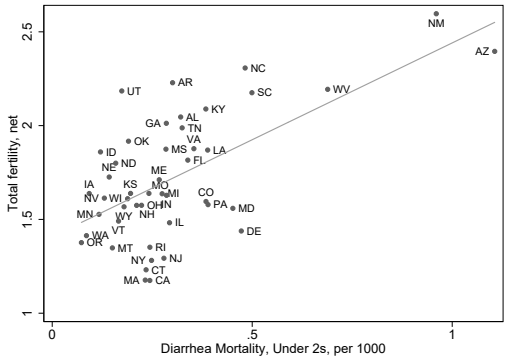
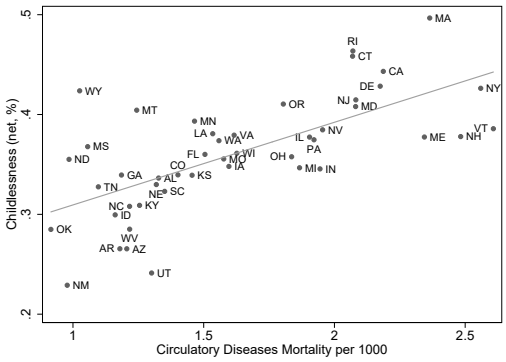
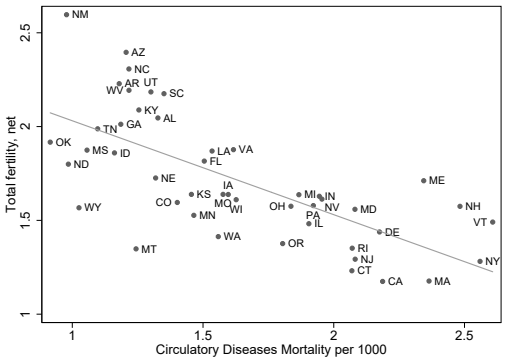


Figure 16: Mortality from Adult Diseases and Net Fertility in 1930 across US states

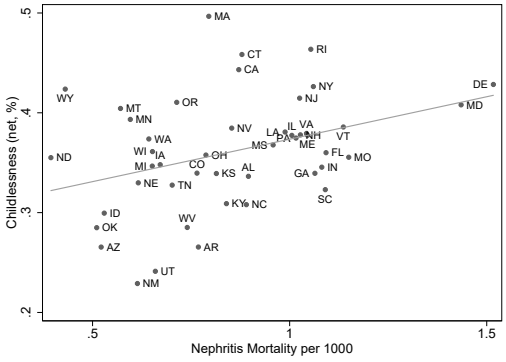
(a) Heart Disease Mortality and Net Childlessness



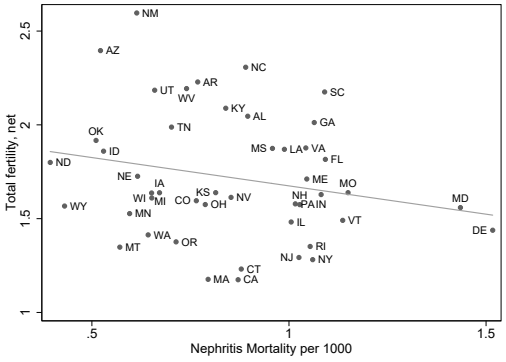
(b) Heart Disease Mortality and Net Fertility



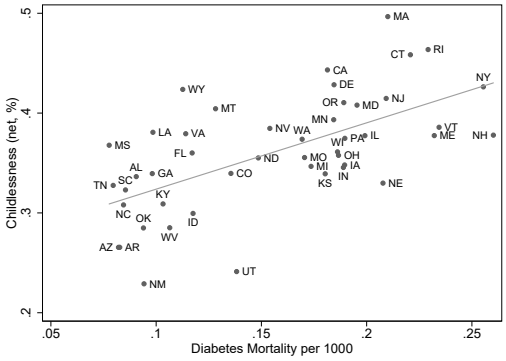
(c) Nephritis Mortality and Net Childlessness



(d) Nephritis Mortality and Net Fertility



(e) Diabetes Mortality and Net Childlessness



(f) Diabetes Mortality and Net Fertility

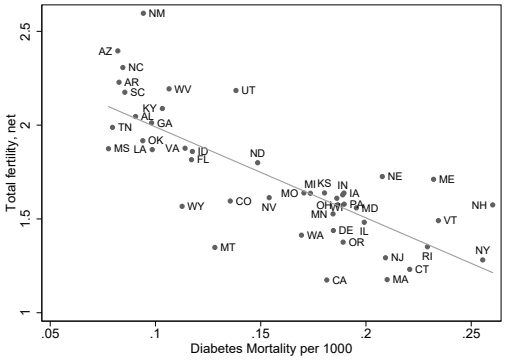
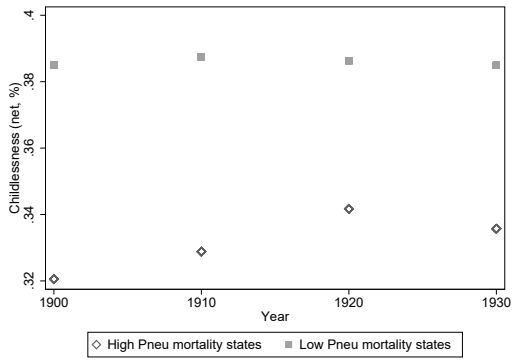


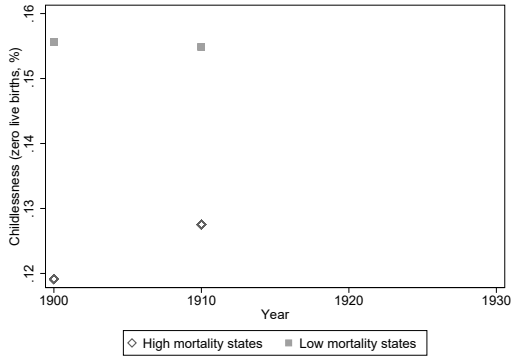
Figure 17: Pneumonia Mortality and Fertility in 1900-1930, averaged across above median mortality and below median mortality US states

(a) Pneumonia Mortality and Net Childlessness



(b) Pneumonia Mortality and Net Fertility

(c) Pneumonia Mortality and Gross Childlessness



(d) Pneumonia Mortality and Gross Fertility

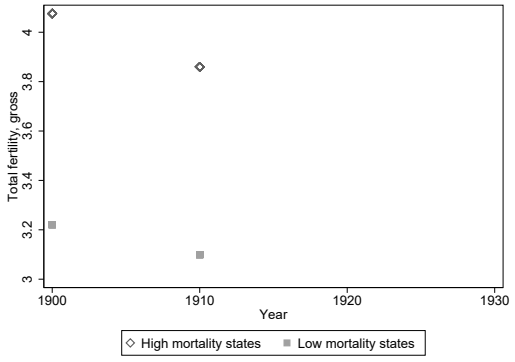
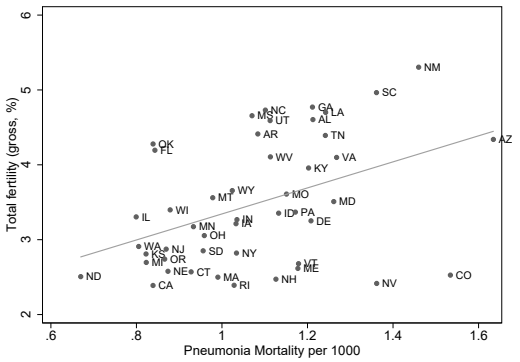
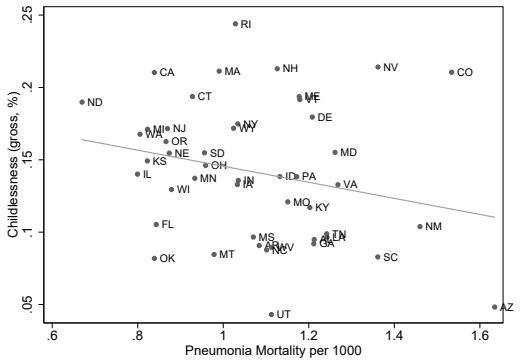


Figure 18: Cross-Sectional Correlations between Gross Fertility and Child Mortality for 1910

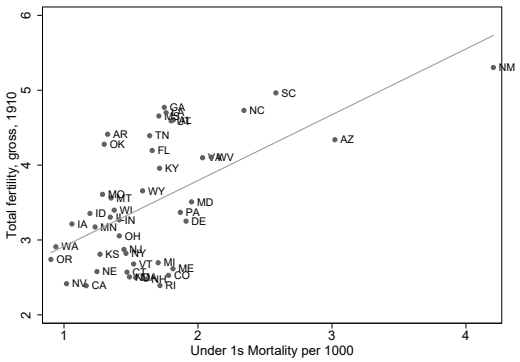
(a) Pneumonia Mortality and Gross Fertility



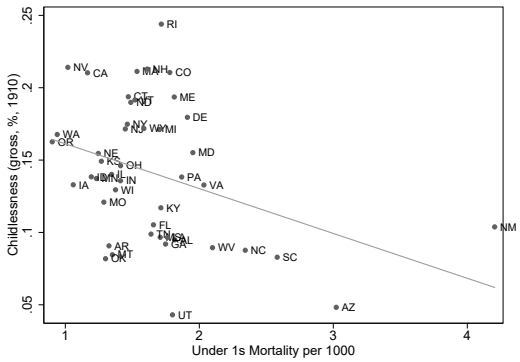
(b) Pneumonia Mortality and Gross Childlessness



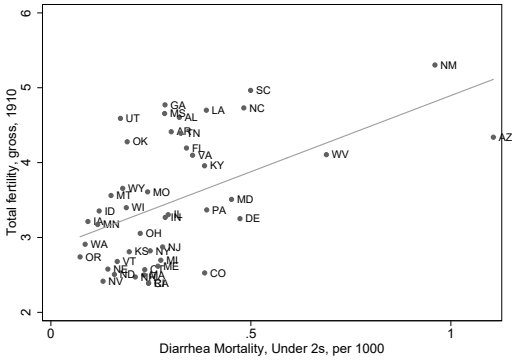
(c) Under 1s Mortality and Gross Fertility



(d) Under 1s Mortality and Gross Childlessness



(e) Under 2s Diarrhea Mortality and Gross Fertility



(f) Under 2s Diarrhea Mortality and Gross Childlessness

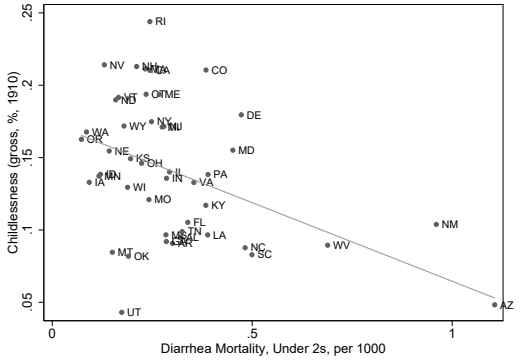
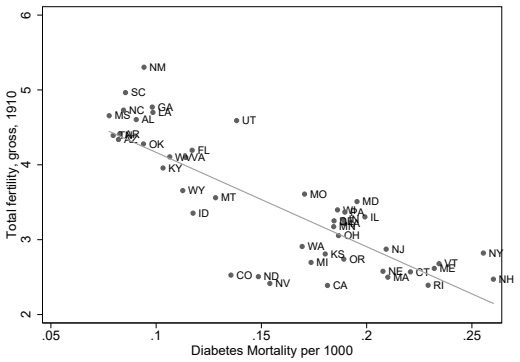
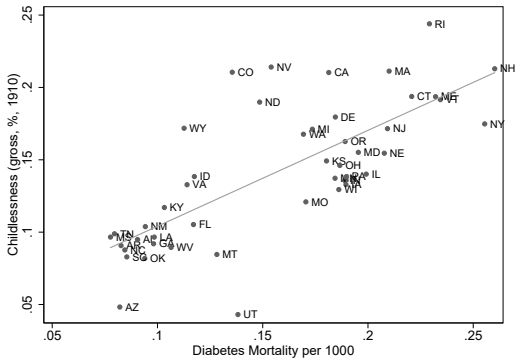


Figure 19: Cross-Sectional Correlations between Gross Fertility and Adult Mortality for 1910

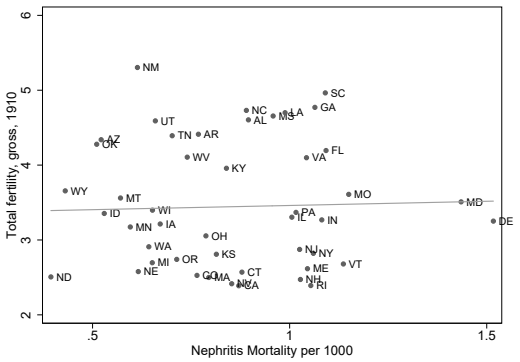
(a) Diabetes Mortality and Gross Fertility



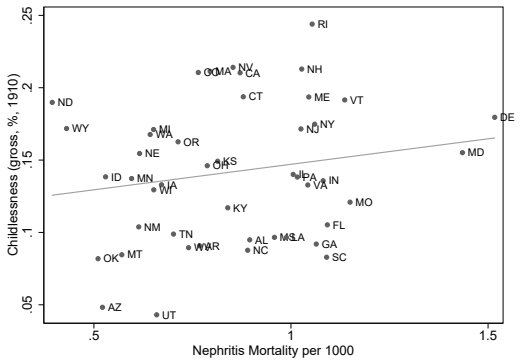
(b) Diabetes Mortality and Gross Childlessness



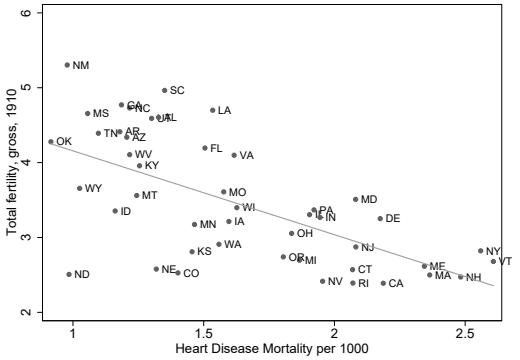
(c) Nephritis Mortality and Gross Fertility



(d) Nephritis Mortality and Gross Childlessness



(e) Heart Disease Mortality and Gross Fertility



(f) Heart Disease Mortality and Gross Childlessness

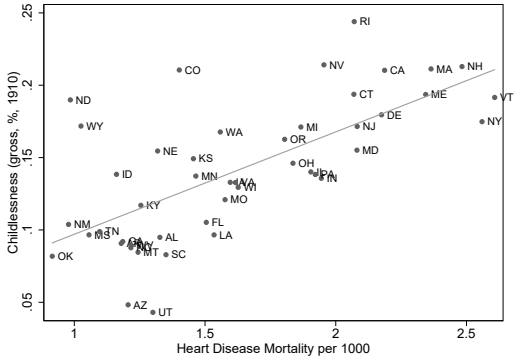
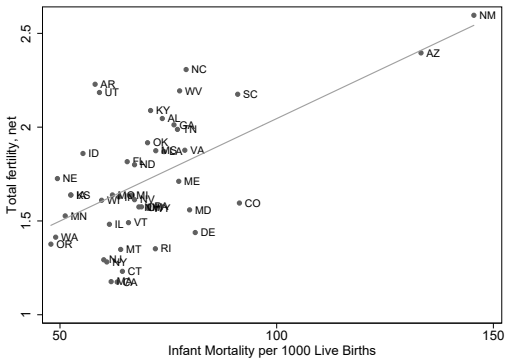
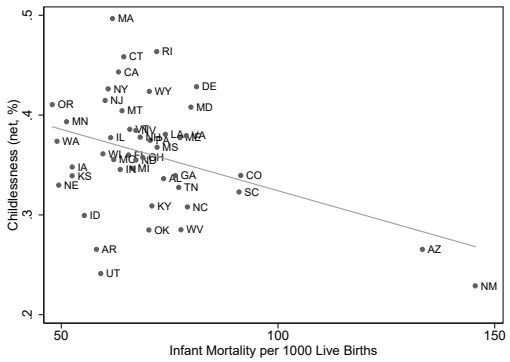


Figure 20: Cross-Sectional Correlations between Fertility, Labor and Marriage Outcomes and Infant Mortality per 1000 Live Births

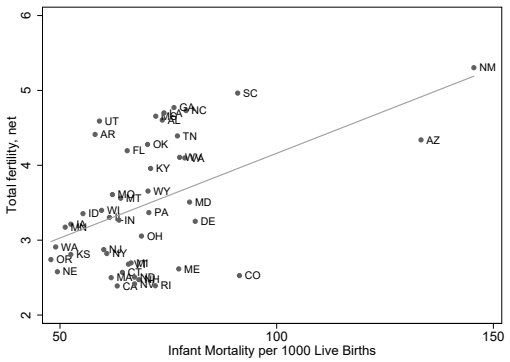
(a) Total fertility, net, 1930



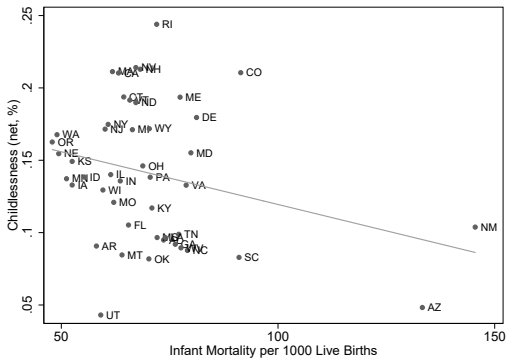
(b) Childlessness, net, 1930



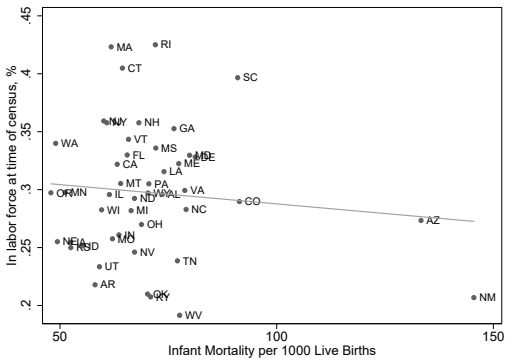
(c) Total fertility, gross, 1910



(d) Childlessness, gross, 1910



(e) Labor Force Status, 1930



(f) Marital Status, 1930

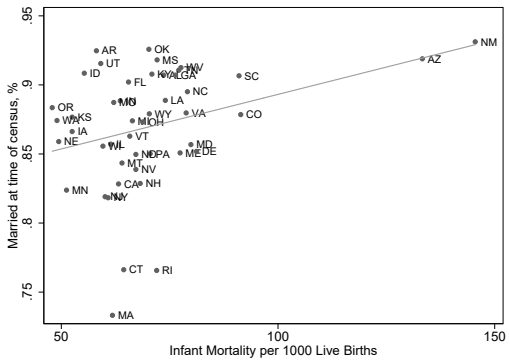
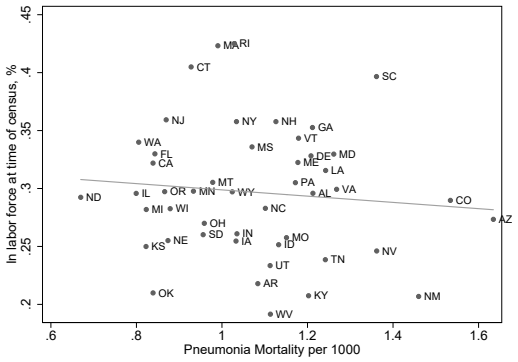


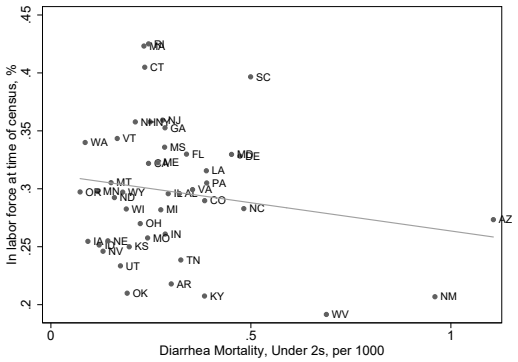
Figure 21: Cross-Sectional Correlations between Labor Force Status and Mortality Rates, 1930

(a) Pneumonia Mortality

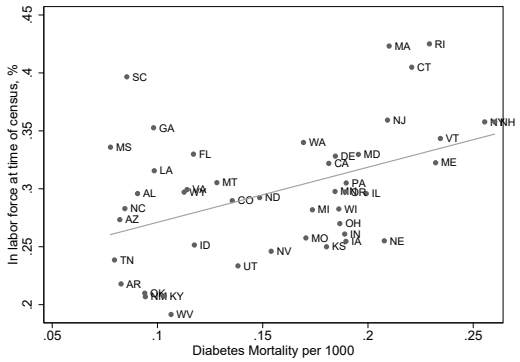


(b) Under 1s Mortality

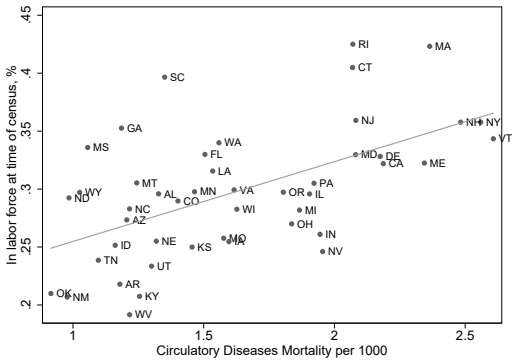
(c) Diarrhea Mortality, Under 2s



(d) Diabetes Mortality



(e) Heart Disease Mortality



(f) Nephritis Mortality

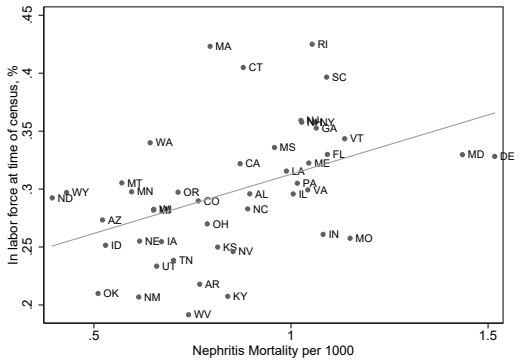
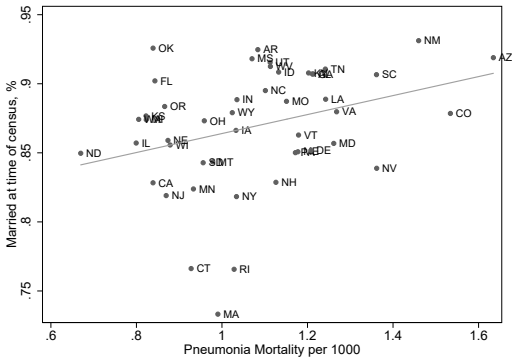
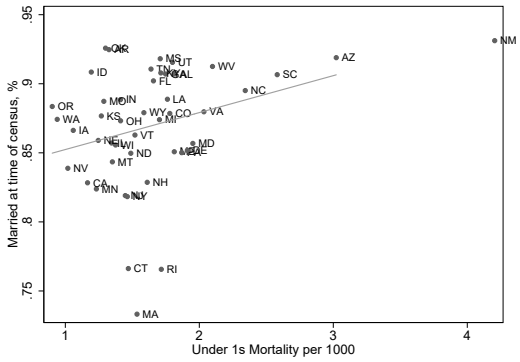


Figure 22: Cross-Sectional Correlations between Marital Status and Mortality Rates, 1930

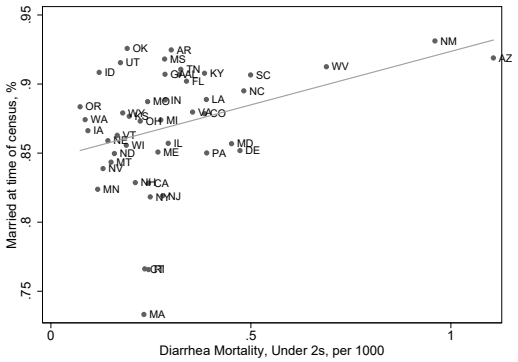
(a) Pneumonia Mortality



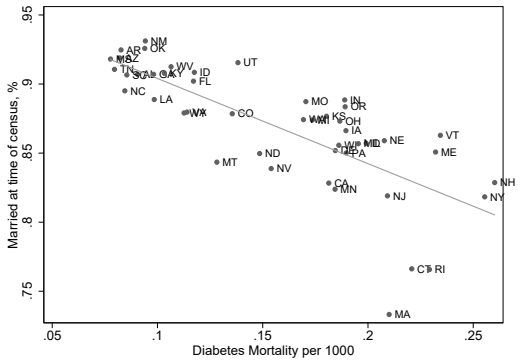
(b) Under 1s Mortality



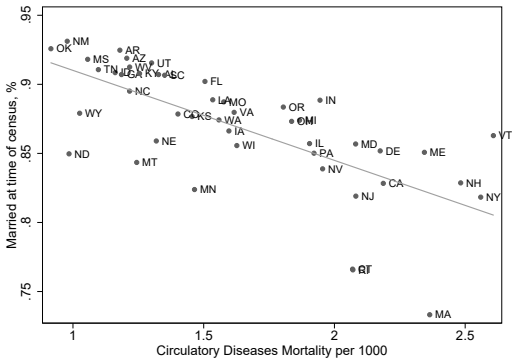
(c) Diarrhea Mortality, Under 2s



(d) Diabetes Mortality



(e) Circulatory Diseases Mortality



(f) Nephritis Mortality