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JEL Classification: J13, I18

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

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We investigate women's fertility, labor and marriage market responses to large declines in child mortality. We find delayed childbearing, with lower intensive and extensive margin fertility, a decline in the chances of ever having married, increased labor force participation and an improvement in occupational status. This constitutes the first evidence that improvements in child survival allow women to start fertility later and invest more in the labor market. We present a new theory of fertility that incorporates dynamic choices and reconciles our findings with existing models of behavior.

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1 Introduction

This paper investigates fertility, labor market and marriage market responses to sharp declines in child mortality and morbidity occasioned by a medical innovation. We find that a sharp drop in pneumonia mortality risk in infancy created by the introduction of antibiotics in 1937 in the United States is associated with lower fertility at both the intensive *and* the extensive margins. We outline a model that can explain this, augmenting the classical quality-quantity model of fertility with a dynamic process for childbearing that takes account of women's labor market opportunities. Women in the model choose the timing of fertility, but must leave the labor market after their first pregnancy, consistent with the cultural norms of the 1930s. We characterize how the trade off between early and delayed fertility depends on the rate of child mortality. Consistent with the predictions of this model, we find that women delay childbearing, are less likely to have ever married, more likely to work and to have higher occupational scores (a measure of the skill intensity of employment).

Ours is the first attempt to analyze, within a single framework, how child mortality may modify the timing of birth, childlessness, the number of children, labor force participation and marriage. In contrast to previous studies that focus on one or the other, we take into account both the child quality versus quantity trade-off, and the family versus career trade-off. In particular, relative to Becker and Lewis (1973) and models that build on it (e.g. Galor 2012, Soares 2005, Aaronson, Lange, and Mazumder 2014, Kalemli-Ozcan 2003), we add a third dimension to the quantity-quality trade-off, namely fertility timing, the other side of which is labor market participation.

Our key insight is that innovations that affect the relative prices of child quantity and quality may also affect the disposable time of women, leading them to delay fertility and participate in the labor market. Child mortality decline implies that women need fewer pregnancies in order to achieve their target number of children, and that the target number is likely to decline due to a reduction in the price of having a higher quality child. This encourages fertility delay, which makes it more attractive to invest in the labor market. This can have knock on effects: it becomes unnecessary to marry early and any of positive shocks to wage earnings, learning about the benefits of work, or declines in the biological ability to have children with age can shift the opportunity cost of childbearing and lead to permanent childlessness.[1]

Contribution to the literature Although the transition to low fertility is central in theories of economic growth (Galor and Weil 1996), the drivers of the fertility transition remain hotly

[1] Four forces are likely to be at play here. First, the direct effect of more births surviving will lower replacement behavior in fertility. Second, it will tend to reduce hoarding behaviour that arises because mortality is uncertain. Third, because declines in mortality are typically associated with declines in morbidity (and thus an improved child health endowment), there is an increase in the incentive to invest in child quality, which will tend to reduce the target (desired) number of children as long as the effect of a reduction in the price of child quantity does not dominate. Fourth, a decline in sickness rates and hence the need to care for sick children will also tend to release the time of women for labor market engagement. Evidence of replacement and hoarding effects is provided in Bhalotra and van Soest (2008) using data from India from a period with broadly similar exposure to infectious diseases including pneumonia.

debated (Galor 2012). In a seminal paper, Aaronson, Lange, and Mazumder (2014) propose that distinguishing extensive and intensive margin fertility responses permits discrimination between competing theories of the demographic transition. Their main insight is that, in the Becker and Lewis (1973) quantity-quality model of fertility, child quality and child quantity are substitutes on the intensive margin, but complements on the extensive margin. In particular, their model implies that when a change in the opportunity cost of women's time is the dominant driver, the two margins will move together. In contrast, when changes in the price of child quality (proxied by a school building program) are the dominant driver, the two margins will move in opposite directions. They demonstrate this by estimating impacts of school construction on fertility.[2]

We provide the first analysis of extensive vs intensive margin fertility responses to declines in mortality and morbidity. In contrast to the predictions of Aaronson, Lange, and Mazumder (2014), we find lower fertility on both margins. Our explanation for this difference is that changes in the price of child quality can directly influence the opportunity cost of women's time. We capture our hypothesis in a model that extends Aaronson, Lange, and Mazumder (2014) and Becker and Lewis (1973) to allow fertility to be a dynamic choice variable determined jointly with labor force participation. By allowing changes in the price of child quality to directly influence the opportunity cost of women's time, we consider together the two forces often posited as competing explanations of the demographic transition: increases in the opportunity cost of women's time and improvements in child survival.

By drawing a new, causal link between child mortality decline and increases in women's labor force participation and childlessness, we provide a new theory of drivers of childlessness that contributes to an active literature in this area (Baudin, de la Croix, and Gobbi 2019, Baudin, de la Croix, and Gobbi 2015, Gobbi 2013, Currie and Schwandt 2014, Ananat, Gruber, and Levine 2007).[3] Our key finding that child mortality decline encourages fertility delay and higher rates of women's labor force participation augments research on the interplay between fertility and women's careers (Lundborg, Plug, and Rasmussen 2017, Adda, Dustmann, and Stevens 2017, Jensen 2012, Albanesi and Olivetti 2014, Albanesi and Olivetti 2016, Goldin and Katz 2002, Goldin 1997), and on fertility timing (de la Croix and Pommeret 2018, Herr 2016, Choi 2017, Ananat and Hungerman 2012). Our contribution here is to identify child mortality decline as a factor driving both fertility delay and women's labor force participation.

Previous work has linked fertility delay to marriage and labor market incentives (Caucutt, Guner, and Knowles 2002), but not to falling child mortality. A related literature has documented the liberating influences of the expansion of women's education and the introduction of the birth control pill, which enabled fertility delay, later marriage, and labor force participation (Goldin and Katz 2002, Goldin 2006, Bailey 2006); we show that child mortality decline had similar liberating

[2] They use a triple difference approach, modeling fertility of black relative to white women in response to the Rosenwald programme that created schools for blacks in the 1920s.

[3] Recent models of childlessness suggest that involuntary childlessness at the bottom of the education distribution arises from poverty, while childlessness at the upper end of the education distribution is voluntary, being driven by the opportunity cost of childbearing (Baudin, de la Croix, and Gobbi 2015, also see Aaronson, Lange, and Mazumder 2014 in relation to the opportunity cost mechanism).

effects.[4] There were dramatic increases in women's labor force participation in the mid-20th century, which have been associated with the high school movement and the emergence of more woman-friendly jobs (Goldin 2006); we propose child mortality decline as another contributing channel. In fact, we also find that sulfa drugs increased high school completion.

Model of fertility timing and labor market choices Our model extends the classical quantity-quality model of fertility choices. As in the existing literature following Becker and Lewis (1973), we assume that women have preferences over the quality of children, quantity of children and other consumption. We extend the classical model to a dynamic environment, which introduces two novel features relative to the existing framework, which are realistic to our study period.

First, the quantity of children is not perfectly controllable, but is subject to the risk of child mortality. Second, there is an inherent trade off between the quantity of children and the woman's ability to earn income. At each date in her period of fertility, a woman chooses whether to work for a wage or to get pregnant.

The key trade off in this model is as follows: Leaving the labor market early increases the number of pregnancies the woman can attempt. With more attempts, she is more likely to achieve the number of children that maximizes her utility, despite the risk of child mortality. On the other hand, leaving the labor market early reduces the woman's expected lifetime income and, therefore, the resources available for consumption and child quality.

We characterize women's optimal choices as a function of the rate λ of child mortality. Our model predicts that, for a realistic range of parameters, a decline in λ encourages women to delay their fertility and remain in the labor market for longer. This feature leads to a novel prediction: if enough women in the population switch from early to delayed fertility, then the total probability of childlessness in the population increases after a beneficial health shock that decreases λ, and the number of children on the intensive margin decreases. Importantly, the prediction of increased childlessness stands in contrast to the prediction of a classical quantity-quality model. It can be used to directly test for the importance of dynamic effects. We establish these results both for an illustrative model with three periods and for a general dynamic environment. We also demonstrate that these predictions are robust across a range of extensions to the baseline model.

Empirical strategy and effect sizes Identification exploits the fact that the introduction of the first antibiotics, sulfa drugs, led to a trend break in pneumonia mortality in 1937. Mortality declined more in states with higher baseline rates. We leverage these cross-cohort and cross-state patterns in a difference-in-difference strategy similar to that used in Acemoglu and Johnson (2007), Ager, Hansen, and Jensen (2017), Bleakley (2007), Bhalotra and Venkataramani (2015) and Gollin, Hansen, and Wingender (2021).

[4] In contrast to our findings, the pill had no impact on completed fertility at either margin (Ananat and Hungerman 2012). A possible explanation for this difference is that women in the antibiotic era were more likely to have to choose between career and family, while women in the pill era could more easily have both (Goldin 2004, Coles and Francesconi 2018).

Modelling the fertility timing of women giving birth in a window around the 1937 event, we find that a representative interquartile decline in pneumonia mortality led to a 0.6 percentage point (6.9%) reduction in the annual probability of birth, and a 0.3 percentage point (5.9%) reduction in the annual probability of becoming a mother (extensive margin). We then track mothers of the sulfa era in later census files to study total fertility, with a view to identifying tempo effects and effects on the distribution of the number of children per woman. Plugging in the same reduction in pneumonia mortality and considering women who are still in their childbearing years at the time of census interview, we identify a 4.6 percentage point increase in the probability of childlessness (12.8% of baseline), and 0.18 fewer (net) children conditional on at least one (7% of the baseline mean of 2.61). Considering the same cohorts of women at age 40-50, by when they will have completed their fertility, we find that the impact of mortality decline on childlessness is reduced by two thirds, indicating significant delay effects in the childbearing sample. Analysing changes in the distribution of fertility, we observe statistically significant responses at the two ends, with women being more likely to be childless and less likely to have three or more children.

Turning to labor market outcomes, we find that, in response to pneumonia mortality decline, the probability of women's labor force participation increased by 2.6 percentage points (7%), occupational scores increased by 6.6 percentage points, and women worked more hours (1.15 hours more per week). Importantly, we find that child mortality decline increased the joint probability that a woman was both childless and in the labor force by 13% of the baseline probability (20.5%), and this increase was driven entirely by a decline in the number of women who were stay-at-home mothers. The estimated impacts on marriage rates are smaller, with the chances of being ever-married declining by 1.5 percentage points (1.7%), indicating that marriage was one, but not a key, pathway.

Identification challenges and specification tests We demonstrate the robustness of our findings to several coherence and specification checks. The main threat to identification is differential pre-trends in outcomes between states with higher versus lower disease burdens in the pre-sulfa era. We scrutinize trends in an event study-style specification, and investigate stability of the results to controls for relevant time-varying covariates and unobservable trends. Following Pei, Pischke, and Schwandt (2018), we also conduct a balance test using these covariates and, similar to Aaronson, Lange, and Mazumder (2014), we estimate impacts of a placebo intervention. Identification is aided by the fact that sulfa drugs were only effective for certain antibacterial infections - for example, they were effective in treating pneumonia but not diseases such as tuberculosis, which had similar risk factors. We show that our findings are not driven by potentially confounding events, including the Second World War which encouraged women's labor force participation (Acemoglu, Autor, and Lyle 2004, Goldin and Olivetti 2013), New Deal spending and the Dust Bowl. We also show that the results are not explained by mean reversion, the introduction of prescription charges, measurement error in pneumonia mortality, survivorship bias, sample selection or endogenous migration, and we discuss potential concerns over measurement of fertility and measurement of pneumonia. We present evidence that women did have access to fertility control in this early era, so that they could alter the timing of their births and, to allow for selection into uptake, we estimate a specification conditional on woman fixed effects.

Broader relevance We confirm that the relationships between child mortality decline and childlessness, total fertility and women's labor force participation that we identify are also evident as stylized facts in contemporary data for developing countries. This underlines the broad scope of our theoretical model and empirical findings.

Pneumonia is the leading cause of death among children today, and 6 million under-5 children continue to die every year (Liu et al. 2016). 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 et al. 2014). Our findings suggest that a benefit of policies that reduce child mortality is that they can "liberate" women from early childbearing and multiple pregnancies and the disempowerment that often accompanies this practice. There is considerable variation in women's rates of labour force participation across countries, conditional upon income. While uneven access to childcare is a potential explanation in OECD countries (Olivetti and Petrongelo 2017), our results suggest that uneven child survival rates may be a potential explanation in developing countries.

The remainder of the paper proceeds as follows. Section 2 describes the disease environment in the 1930s and the event of introduction of the first antibiotics. Section 3 describes the empirical strategy, data, results and robustness checks for fertility during the sulfa era. Motivated by the extensive margin result, Section 4 outlines a theoretical model that rationalizes the link between child mortality decline, fertility timing and labor force participation, and drives predictions for completed fertility, childlessness and labor market outcomes. Section 5 presents the empirical strategy and results for these later life outcomes. Section 6 discusses external validity and Section 7 concludes.

2 Context

2.1 Mortality Rates and Sulfa Drugs

The United States in the 20th century was characterized by high levels of mortality (Britten 1942, Linder and Grove 1947). The arrival of the first antibiotics - sulphonamides, or sulfa drugs - in 1937 drastically altered the standard of medical care, creating large and sharp changes in morbidity and mortality from several bacterial infections. Sulfa drugs were discovered by German chemists experimenting with textile dyes in 1932 and they became available in the United States starting in 1937, achieving wide penetration in the consumer pharmaceuticals market (Lesch 2007). This was enabled by low costs (especially for a life-saving drug), the lack of prescription requirements to purchase the drugs (which were established only in 1939), and the lack of a binding patent.[5] By all accounts, there was a "sulfa craze", with adoption being widespread and quick (Lerner 1991).

Sulfa drugs were particularly effective in treating pneumonia among children, which was previously managed by supportive care (Jayachandran, Lleras-Muney, and Smith 2010).[6] Pneumonia was the leading infectious cause of child morbidity and mortality, and the third leading cause overall (after death from premature birth and congenital defects), accounting for 17% of infant deaths in the United States in the 1930s. Mortality rates from pneumonia are U-shaped in age. Child mortality from pneumonia (under-5s) in the United States stood at an average of 11.8 per 1000 population between 1930-36 (pre-sulfa), and most deaths occurred among infants. The under-1 pneumonia mortality rate was 8.2 per 1000 live births, while the adult rate for the same period was 0.4 per 1000 people (also see Figure 1 which plots mortality rates by age per 1000 population). Both the level and the trend in the all-age pneumonia mortality rate was dominated by the infant rate.

In addition to reducing mortality, the arrival of sulfa drugs led to significant reductions in morbidity (Greengard et al. 1943, Hodes et al. 1939, Moody and Knouf 1940). Prior to the introduction of antibiotics, pneumonia was a debilitating disease with typical spells often lasting 39 days on average, and children tending to have recurrent spells (Britten 1942). Sulfa drugs reduced the severity and length of these episodes (Connolly et al. 2012), and this was documented in clinical trials among hospital inpatients (Greengard et al. 1943, Moody and Knouf 1940). A reduction in pneumonia morbidity among children thus considerably increased the disposable time of women, who were the main care-givers for children and the sick. By strengthening the infant health endowment, it will have also led parents to move along the quantity-quality indifference curve, trading up quantity in favour of quality. Consistent with this (and despite negative selection into survival following the positive shock of antibiotic access), children born after 1937 who survived to adulthood had significantly better indicators of educational attainment, employment, income, and lower disability (Bhalotra and Venkataramani 2015). This is a marker of the substantive importance of pneumonia mortality decline among children born in the sulfa era and a result that is consistent with our finding of a decline in intensive margin fertility.[7]

2.2 Fertility and Fertility Control in Early Twentieth Century America

Between 1910 and the mid 1920s, the average number of children per woman stood between 3 and 3.5. Fertility thereafter fell during the Great Depression (to 2.5 children per woman) but rose during

[5] The chemical structure of sulfonamide had previously been documented as part of a PhD thesis in the early 1900s. By the time it was established in the 1930s that it had antibacterial properties, the patent had expired and anyone was allowed to produce sulfonamide for commercial purposes.

[6] 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 mortality rates. For example, the all-age mortality rate from skin diseases in the U.S. was 1.8 per 1000 in 1930, and from meningitis 2.1, compared to an all-cause infant mortality rate of 69 (Vital Statistics).

[7] 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, both of which improved due to the arrival of sulfa drugs. In this sense, mortality decline also captures improvements in the health of survivors.

World War II and the post-War (Baby Boom) period (1945-late 1950s). Over this period, there is evidence that women born in the early 20th century were able and willing to practise fertility control (Morgan 1991).[8] 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.[9]

2.3 Trends in Women's Labor Force Participation and Marriage

In the 1930s, there was a substantial increase in female labor force participation; for example, there was an increase of 15.5 percentage points, from 10% to 25%, on the extensive margin among married women (Goldin 2006). Marriage and fertility choices were closely related in this period, with only 8.5% of births being out of wedlock (Bachu 1999). [10] Previous work has argued that important drivers of the increase in labor force participation in this period included higher rates of high school completion, the arrival of "nice" jobs, such as secretarial work in offices that reduced the stigma associated with married women working, and the virtual elimination of marriage bars by the early 1940s.[11] Regardless of education, most working women were engaged in typing-oriented jobs and teaching (Goldin 2006).

3 Birth Timing

This Section presents the data, empirical specification, results and an extensive series of robustness checks for analysis of impacts of antibiotic exposure on birth timing among women who were of reproductive age when the antibiotics first became available. As indicated earlier, we find that the antibiotic-led decline in pneumonia mortality resulted in a decline in fertility on both the intensive and the extensive margins. As the extensive margin result is contrary to the predictions of Aaronson, Lange, and Mazumder 2014, we develop an extension of their model in Section 4 which is consistent with our result. Our model generates predictions for the impact of mortality decline on childlessness, the labour market and marriage outcomes, which we investigate in Section 5.

[8] 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 the importance of birth control. The first birth control clinic was opened in 1916, but shut down, followed by a clinic in 1923, which was not shut down. These clinics were the precursor to Planned Parenthood.

[9] 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 analyze this in the context of the United States demographic transition in the 19th century.

[10] 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 permanently childless couples that they knew. This shows that 22% of couples were believed to be childless due to the wife's career, and 16% childless due to economic pressure, suggesting that fertility and labor market choices were linked during this period, even for married women.

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

3.1 Data

Data on individual outcomes are taken from the United States Census Microdata (Ruggles et al. 2010). Appendix A describes the data in more detail, including sources and variable definitions. Below we discuss the birth and mortality data.

Birth timing data To model fertility during the sulfa era we use pooled census microdata using the 1940 and 1950 1% samples.[12] We select women who were of childbearing 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. 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. Thus, the cohorts in the "birth hazard sample" were born in the years 1890-1928.

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.[13] Using a variable that links child records to mothers, we constructed a complete history of live births for each woman, restricting births to biological births (95% of children in the household) that occurred in the U.S. As is standard, this measure excludes any pregnancies that did not result in live births, and any deaths that occurred after birth but before the census. 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. In Section 3.4, we discuss estimates that measure fertility using only information on children under the age of 10 at the time of the census.

Mortality rate data We gathered data on diseases treatable with sulfa drugs and also for diseases that were not treatable, and that thus act as placebos. These data are from several volumes of the U.S. Vital Statistics (Grove and Hetzel 1968, Linder and Grove 1947, Ruggles et al. 2010, Bureau 1943). We create the pre-intervention or baseline levels of cause-specific mortality rates as the state-level average over the years 1930-36 of the mortality rate, which allows us to smooth over fluctuations in the rate created by influenza epidemics. There are no annual time series data in this era for pneumonia mortality. Instead, the vital statistics data contain the mortality rate from pneumonia and influenza combined. This is because the two diseases shared symptoms such as cough and fever, making them difficult to distinguish. Moreover, they were intrinsically related since pneumonia often followed as the more serious development of an initial influenza infection. Importantly for our purposes, because influenza is viral, it was not treatable with sulfa drugs but pneumonia, which includes a bacterial strain, was. Thus a sulfa-driven decline in the combined

[12] In all datasets, we restrict the sample to 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; see Section 3.4 for robustness checks on migration.

[13] 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).

influenza-pneumonia mortality rate reflects a decline in pneumonia mortality. Henceforth, for ease of exposition, we refer to this combined rate as pneumonia mortality.

We found decadal data that provide separate series for pneumonia and influenza to strengthen faith in this approach. First, these data show that pneumonia dominated the combined mortality rate, being responsible for 8.9 deaths per 1000 in 1930, compared to 1.3 deaths per 1000 live births from influenza. Second, the decline in the combined mortality rate between 1930 and 1940 was *entirely* on account of a decline in the pneumonia mortality rate: influenza rates fluctuated considerably with epidemics and seasons, but the average influenza rate was steady across the decade.

In a similar vein, we use the all-age pneumonia and influenza mortality rate on the grounds that both the level and the trend are driven by the infant rate. Infant births and deaths, the two components required to calculate infant and child mortality rates, were known to be under-reported in this era (Linder and Grove 1947, Eriksson, Niemesh, and Thomasson 2017), making the child rate noisier than the all-age rate. In addition, underreporting may have been more prominent in the Southeastern U.S., where rates of pneumonia were higher on average, and this could result in systematic measurement error (Ewbank 1987). We will show that infant and child pneumonia mortality was the overwhelming source of variation in the all-age rate. In Section 3.4, we also show that our estimates are robust to using the all-age rate as an instrumental variable for the more noisily measured child rate.

Summary statistics Tables A.1 and A.2 in the Appendix provide descriptive statistics. 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.

3.2 Empirical Strategy

We begin by documenting the "first stage" impacts of the introduction of sulfa antibiotics on pneumonia mortality, demonstrating a nationwide trend break in 1937, and that this was larger in states with higher baseline rates of pneumonia mortality. As a result, the introduction of sulfa drugs drove convergence in pneumonia mortality across states. We then describe an empirical strategy that identifies the impact of sulfa drugs on fertility timing.

Trend break in pneumonia mortality The timing of the introduction of antibiotics created sharp variation across cohorts in exposure to pneumonia, a disease treatable with sulfa drugs. Figure 2 shows trend breaks in pneumonia mortality in 1937. Pneumonia mortality declined on average 11.1% per year from 1937: see Table A.4 that shows the coefficients in regressions of changes in level and log pneumonia pre- and post-sulfa. This per-year decline in pneumonia mortality was driven entirely by the infant and child rate, while the adult and elderly rates exhibited a modest annual *increase* between 1937 and 1943. Most deaths from pneumonia occur in infancy, when the immune system is still fragile, and the steepest post-sulfa decline in pneumonia mortality was amongst infants and young children.[14]

State convergence in pneumonia mortality There was considerable geographic dispersion in pre-intervention levels of pneumonia mortality (Figure A.2 in Appendix C.1). Because sulfa drugs led to larger declines in pneumonia in states where the pre-intervention burden was higher, we see post-sulfa convergence in pneumonia mortality rates across the U.S. states, and that this convergence was most marked for children and infants (Figure 3).[15]

Estimating equation for birth timing Identification exploits these two features of the antibiotic revolution - the post-1937 decline in mortality rates and the large variation across U.S. states in pre-intervention levels of the disease burden, in a difference-in-difference design similar to that in Acemoglu and Johnson (2007), Bleakley (2007), Ager, Hansen, and Jensen (2017) and Bhalotra and Venkataramani (2015). The event of the nationwide introduction of sulfa drugs in 1937 was plausibly exogenous, being the direct result of scientific innovation. However, the identifying assumption of parallel counterfactual trends in fertility in states with higher vs lower pre-1937 levels of pneumonia merits scrutiny. We elaborate our approach to this below.

We analyse births in a short window around 1937, namely 1930-1943. This limits the role of confounding events, such as the influenza epidemic of 1928-9 and the increasingly widespread use of penicillin after 1943. We restrict the sample to women of reproductive age (and hence at risk of birth) during this period. Using longitudinal birth history data for these women, we estimate impacts of the discrete event of antibiotic availability on birth timing (the probability that a given woman gives birth in a given year). The estimation equation is:

$$\begin{aligned}\Pr(Y_{jsctr} &= 1) = \alpha_0 + \alpha_1 * prePneumonia_s * post1937_t \\ &+\alpha_2 * preMMR_s * post1937_t + statecontrols_s * post1937_t \\ &+birthorder_{jt} + timesincelastbirth_{jt} + race_j + educ_j \\ &+\theta_t \times region_r + \kappa_s + \phi_c + \varepsilon_{jsctr},\end{aligned} \tag{1}$$

where Y_{jsctr} is a binary indicator that equals 1 if woman j born in state s, census region r and cohort c, gave birth in year t and zero otherwise. The variable $post1937_t$ equals one if the potential birth year of the child is 1937 or after and zero otherwise, as 1937 marks the nation-

[14]The uptick in the combined influenza plus pneumonia mortality rate on account of an influenza epidemic in 1936-1937 results in the decline after 1937 being sharper, but some of this reflects reversion to the mean. Comparison of the post-1937 rates with the rates in 1930-1934 in the plot makes even clearer that mortality decline after 1937 was driven by the child mortality rate. In the analysis, we address the uptick in 1936-1937 by defining baseline rates as an average over 1930-1936 and we also provide a test of whether our estimates are driven by mean reversion.

[15]This is formalized with tests of significance in Table A.4. The estimated convergence coefficient for pneumonia mortality is -0.29, implying that a sulfa-led decline in pneumonia mortality equivalent to an interquartile shift in the pre-intervention pneumonia mortality distribution (-0.26) was associated with a 7.5% reduction in pneumonia mortality.

wide introduction of antibiotics. $prePneumonia_s$ is the 1930-1936 (baseline) average state level pneumonia mortality rate. The coefficient of interest is α_1, which captures the causal effect of the antibiotic-led pneumonia mortality decline on the probability of birth. We estimate equation (1) as a logistic regression, yielding estimates for a discrete time proportional hazard model. Standard errors are clustered at the birth state level to account for serial correlation in outcomes within states (Bertrand, Duflo, and Mullainathan 2004).

The equation includes indicator variables for years since last birth with the count starting at age 15 and restarting after every birth ($timesincelastbirth_{jt}$), and indicator variables for the birth order of the next birth ($birthorder_{jt}$). We include fixed effects for the woman's race ($race_j$), education ($education_j$), birth state (κ_s) and birth year (ϕ_c). The other, state-level control variables are discussed below. Education will in part capture potential wages and fertility preferences. In a robustness check, we also allow for woman fixed effects, which will capture any unobservables that determine compliance and are potentially correlated with fertility preferences.

Extensive and intensive margins of fertility In order to distinguish between the extensive and intensive margins of fertility timing, we estimate Equation (1) on subsets of the sample where we only include woman-year observations in which the woman is at risk of giving birth to her first child (extensive margin), and then to observations where the woman is at risk of giving birth to her second or higher-order child (intensive margin).

Identifying assumptions The main threat to identification is that the variable $prePneumonia_s$ captures not just baseline pneumonia but also other state-level variation in pre-sulfa conditions that are correlated with both pneumonia and the outcome (fertility). The likely candidate confounders are the disease epidemiology of the state and economic development. For the first, we leverage the fact that sulfa drugs were able to treat some diseases and not others to identify a set of placebo diseases which we control for. For the second, we assimilated data on indicators of income, health and education spending, infrastructure and women's labour market position and condition on these. Each of these variables is included as its pre-intervention value interacted with an indicator for post-intervention cohorts ($statecontrols_s * post1937_t$). This sharpens the test as it allows structural breaks in the controls to coincide with the structural break in pneumonia in 1937. The baseline model includes these controls. In extensions shown as specification checks, we (a) drop all of these controls to assess sensitivity of the estimates to them and, (b) run a far more demanding specification in which each state variable is interacted with a full set of year fixed effects rather than with the dummy variable $post1937_t$.

Tuberculosis (TB) and diarrhea were highly prevalent infectious diseases that act as natural placebos. TB is a respiratory disease similar to pneumonia in causes but not treatable with sulfa drugs. Diarrhea was also not treatable with sulfa, and it stood alongside pneumonia as a leading cause of death of infants.[16] We also control for malaria, another communicable disease as, in this

[16] For diarrhea we use the mortality rate for children under the age of two, given both data availability considerations and the fact that adults typically do not die of diarrheal infections. For other control diseases we use the all-age rate.

era, there were state-specific interventions including sanitation, housing and public health programs targeting malaria. We control for mortality rates from cancer and heart disease, the main non-communicable diseases, in order to capture changes in health care quality and access, allowing for state-specific expansion of hospital care. Together, these five placebo diseases control for the state level disease environment.

To distinguish sulfa-driven convergence in pneumonia mortality rates from an underlying process of economic convergence we control for the logarithms of per capita state income, public health spending, education spending, and the numbers of schools, hospitals and physicians, women's literacy and women's labor force participation.[17]

We address common concerns with controls for observables. First, to allow for the possibility that the underlying confounders are poorly measured (e.g. state income may not fully capture economic development), we follow Pei, Pischke, and Schwandt (2018) and perform a version of a balance test, which involves regressing each of the state-level economic and disease variables on the sulfa exposure variables. Second, we additionally allow for unobservable trends by including census region * year fixed effects ($\theta_t \times region_r$) in the baseline model. These control flexibly, for example, for regional differences in impacts of the recession of 1937-38. In alternative specifications, we include finer-grained census division * year fixed effects and state-specific linear trends. As a specification check, we also directly estimate pre-sulfa trends in high vs low pneumonia states and show that they are not statistically significantly different. Third, we estimate impacts of a placebo intervention using an approach similar to that in Aaronson, Lange, and Mazumder (2014). Fourth, to address the possibility of mean reversion, we control for the state-level pre-sulfa average of the outcome variable. Finally, we scrutinize pre-trends and dynamic impacts in event study style models. The event study models show pre-event trends, the dynamic evolution of post-event impacts, and they naturally provide validation of the timing of the event.

Coincident events Even if the identifying assumption of parallel counterfactual trends holds, we need to be sure that we do not capture impacts of other events that happened to coincide with the introduction of sulfa drugs. We already address this in allowing impacts of the thirteen state-time varying controls to break in 1937. If there were a policy or other event that occurred in 1937 - for instance a sharp change in income or in a placebo disease - and if this change impacted fertility, our estimates are conditional upon this (though to explain our findings this change would need to vary systematically with the pre-1937 level of pneumonia mortality).

Our baseline model also controls for a trend break in maternal mortality in 1937 that was the result of sulfa drugs being able to treat puerperal sepsis (variable $preMMR_s$ in equation (1) above). This is an ascending bacterial infection of the reproductive tract that can occur soon after birth (Thomasson and Treber 2008) and that accounted for 40% of the 6.4 maternal deaths per 1000 live births in 1930 (Vital Statistics). The maternal mortality rate declined by around 10% per year

[17]For female labor force participation and literacy, we use the value in 1930 as values for other years are unavailable. In a robustness check we also control for baseline occupational structure at the state level, to allow that women's employment opportunities were restricted by the availability of occupation.

after 1937 and, in principle, this may have directly influenced the outcomes we study.[18]

In additional specification checks, we account for impacts of the main historical events of the time including World War 2, the Great Depression and New Deal spending, and the Dust Bowl, demonstrating that our results continue to hold.

Measurement issues In the first half of the twentieth century the states joined the U.S. Vital Statistics registration system in different years, resulting in variation in the quality of birth and death data (Eriksson, Niemesh, and Thomasson 2017). To address this, we consistently include in the baseline model the years that each state entered the U.S. Vital Statistics birth registration and death registration systems, interacted with the measure of sulfa exposure.

As indicated earlier, in line with IV measurement error models (Ashenfelter and Krueger 1994), we also investigate replacing the all-age pneumonia rate with the child rate, using the all-age rate as an instrument.

In assigning pre-intervention mortality rates and state-level controls to women, we use their birth state because migration decisions after birth are potentially endogenous. We also omit migrants in the main specification. As a result, we may underestimate treatment effects for women who moved into areas with the largest health gains. To investigate this further, we re-estimate the equation assigning all state-level variables to the resident state of the woman at the time of census enumeration, and also instrumenting state of residence mortality decline with birth state mortality decline - see the discussion in Section 3.4, where we also discuss modelling migration as an outcome and find no evidence of endogenous migration. Among other robustness checks we investigate sensitivity to age at census sample, and to removing outlier states. We show that the significance of the estimates is robust to a multiple hypothesis testing correction. In Section 3.4, we also discuss and assess our measure of fertility, checking for any bias induced by counting only children at home.

Woman fixed effects and other specification checks As we created longitudinal data on births within women, we can assess changes in the probability of birth for a given woman post- vs pre-sulfa, conditional on her age. We do this by including woman fixed effects in the model in a robustness check. This takes care of any concerns over unobservables that (endogenously) determine selective uptake of sulfa drugs for pneumonia and are correlated with fertility preferences. While the baseline model is estimated as a logistic regression, we show that estimating equation (1) as an OLS regression produces broadly similar estimates.

3.3 Birth Timing Results

We find that the conditional probability of birth declined following sulfa-led reductions in pneumonia mortality (Panel A, Table 1). Notably, fertility declined on the intensive *and* extensive

[18] Although pre-intervention levels of pneumonia and maternal mortality are positively correlated, they also exhibit substantial independent variation across the U.S. states (see Figure A.1 in Appendix C), and Appendix Figure A.2. The parameter α_2 will reflect causal effects of maternal mortality decline on the birth probability.

margins (see columns (2) and (3)).[19] We scale our estimates using an interquartile shift in (baseline) pneumonia mortality (-0.26), which is close to the actual nationwide decline that took place after 1937.

This 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% (column (1)). The estimates in column (2) for the extensive margin response 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 intensive margin result shows that the same interquartile shift implies a 0.25 percentage point reduction in the probability of transitioning to a higher order birth after 1937, which is 1.7% of the pre-intervention mean of 14.9%.[20] Thus, the decline in pneumonia mortality delayed the transition to first and higher order births. The delay to the first birth was about three times the delay to higher order births, which is consistent with the impact of sulfa exposure on childlessness that we document in Section 5 below.

Event study for birth timing We estimate an event study specification for probability of birth similar to column (1) of Table 1, where we interact baseline pneumonia mortality with indicators for every year in the sample period (1930-1943, with 1937 as the base year). Marginal effects calculated from the log odds coefficients in the logit regression are plotted in Figure 4. While this exercise is challenged by statistical power and not all coefficients are statistically significant, the plot suggests a discrete change in 1937, with a decline in birth probability for women who experienced larger declines in pneumonia mortality. This plot also provides 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, insofar as the coefficients show no trend before 1937. Overall, our findings show that women delayed childbearing in response to improvements in child survival and child health.

3.4 Robustness Checks

We now discuss results of robustness checks motivated in the empirical strategy section.

Omitted trends While we have a clean policy experiment in the introduction of the drug, we need to be sure that baseline levels of pneumonia mortality are not capturing other baseline conditions. The event study mitigates this concern. We neverthess use pre-sulfa data to directly

[19] Column (1) in Table 1 pools a woman's births, and in so doing assumes independence across recurrent events. Standard errors are clustered at the state level, which allows for non-independence within mothers as, by construction, they do not move state. The extensive margin result in column (2) is robust to this concern, as the extensive margin has one event (the first birth).

[20] 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 (2) is similar to the effect on the overall birth probability in column (1), and both are approximately three times the size of the effect on the intensive margin in column (3).

model trends. We regress 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 Appendix Table A.27, show no evidence of differential pre-trends across high and low mortality states in the hazard sample.

The most likely candidate confounders in our setting are economic development and the disease epidemiology of the state. Our baseline specification controls for trends in 15 indicators of state-level disease prevalence, income, infrastructure, women's labour force participation, and an indicator for changes in the quality of birth and death surveillance. Here we first assess sensitivity of the coefficient of interest to dropping these controls. The coefficient remains statistically significant but is smaller in absolute terms (column (1), Panel B, Table 1). We then enrich the baseline specification by interacting all state-level control variables with a full set of year dummies (instead of with just the indicator for post-1937). Although this adds 165 control variables to the specification, and renders the coefficient of interest smaller, it remains statistically significant and negative (column (2), Table 1). We next enrich the model by controlling for state occupational structure in 1930 interacted with $post1937$ (column (6), Table 2).[21] The estimates are robust to this check.

We allow for the possibility that the underlying confounders are poorly measured (e.g. state income may not fully capture economic development), following Pei, Pischke, and Schwandt (2018) and perform a version of a balance test. We regress all of the state-level variables on the sulfa exposure variable.[22] Of the numerous tests, only one is statistically significant, which is the coefficient on pneumonia exposure in the equation for tuberculosis mortality. Investigating this by estimating an event study model for tuberculosis mortality decline, we see a *secular* decline with no break in 1937.

We residualise pneumonia mortality by all our state control variables, and show the spatial dispersion of residualised pneumonia mortality across the United States in Figure 5. This illustrates that residualised pneumonia mortality is well dispersed. To provide evidence that our results do not capture differences in trends between regions of the U.S., we show estimates of the hazard model where we omit the mountain west and deep south states in turn (Appendix Table A.19, columns (3) and (4)).[23] This does not substantially change the estimates.

Recognizing that however rich the set of observables is, there are potential unobservables, the baseline specification in the hazard model includes census region-year fixed effects. The estimates are similar in magnitude and not statistically different when we instead include state linear trends or the more fine-grained census division-year fixed effects (columns (4) and (5), Panel B, Table 1).

[21]Occupational structure is calculated as the share of women employed in primary, secondary or tertiary industries in the 1930 census, yielding three variables.

[22]Specifically, we estimate variants of the regression

$$controlvar_{s,t} = \alpha + \beta_1 post1937 * prePnemonia_s + \beta_2 post1937 * preMMR_s + \beta_3 prePneumonia_s + \beta_4 preMMR_s + \boldsymbol{\beta}_5 \boldsymbol{\theta}_t,$$

where *controlvar* are all the state-time varying controls in our main estimates. For compactness we do not report these results but they are available on request from the authors.

[23]The mountain states are Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, and Wyoming. The deep south states are Louisiana, Mississippi, Alabama, Georgia, and South Carolina.

We assess mean reversion by controlling for the state-level pre-sulfa average value of the outcome variableand find it does not change the coefficient of interest; see Column (5) in Table 2.

Coincident events 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 widely 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). Appendix Figure A.3 shows extracts from two articles that appeared in the New York Times in 1936. The first stage trend break plot for pneumonia and the event study for fertility both confirm that the structural break in trend was in 1937.

Still, we may be concerned that these changes were driven by a coincident event rather than by the arrival of sulfa drugs. The United States entered the Second World War in December 1941. To assess the relevance of this, we restrict the sample to births occurring before 1942. Our findings are essentially unchanged (column (1), Table 2). To capture potential impacts of the Great Depression and subsequent recovery, we accessed data on state-year data on New Deal spending (Fishback, Kantor, and Wallis 2003) and included this as a control, interacted with sulfa exposure ($post1937$ in the hazard model). The coefficients of interest are robust to this (column (2) in Table 2). The Dust Bowl refers to a period of drought and dust storms during the 1930s that damaged agriculture in several southern U.S. states and resulted in large out-migration from those states.[24] The results are, if anything, strengthened by the omission of the Dust Bowl states (column (3), Table 2).

Sulfa drugs were available without prescription until 1939. Our findings are similar when we exclude all births after prescription was introduced (column (4), Table 2). We also verify that the coefficients on pneumonia mortality decline are not significantly different when omitting maternal mortality decline (columns (2) and (3) in Appendix Table A.32). Finally, the 1930s and 1940s did experience some increases in childcare provision, particularly during WW2, in order to encourage female employment. However, the Lanham Act was not implemented until 1943, and provided only 130,000 spaces for children (far short of the intended two million), and these child care centres were quickly closed down after WW2.

Measurement issues If we replace the all-age baseline pneumonia mortality rate with the under-5 rate then the coefficient is correctly signed, but small and imprecisely estimated, consistent with known measurement error in this measure. Once we instrument this with the all-age rate to correct for measurement error, it is statistically and substantively significant (column (3), Table 1).

So as to investigate the related concern that the pneumonia mortality measure is masking impacts of decline in adult mortality from other diseases, we include additional variables measuring adult mortality rates for the placebo diseases (in the baseline specification, these are included as all-age rates with the exception of diarrhea, which was for under-2s given that adults typically do not die of diarrhea). Column (7), Table 2 shows that the coefficient of interest is stable to this

[24]These were Nebraska, Kansas, Colorado, New Mexico, Oklahoma and Texas.

check.

We now consider potential concerns with the way that we measure fertility. First, we measure fertility using information on children living at home in census data. As a check on this, we only include potential births from the 1950 census that would have occurred in the years 1940-43: this way, the oldest child would have been ten (Appendix Table A.19, column (1)); the results are similar). Second, the conception date is a more accurate measure of a woman's fertility choice than the birth date of the child. We proxy a child's date of conception as one year before the date of birth, and re-estimate the hazard model using the date of conception as the outcome rather than the date of birth (Table A.19, column (2)) and again the estimates are similar.[25]

Endogenous migration If prospective mothers migrated in response to disease, then the introduction of sulfa drugs may have influenced migration patterns. In the baseline sample, we omit migrants, defined as women whose birth state is not the same as their state of residence at enumeration. If we instead include these women, and instrument for mortality decline exposure in their state of residence using mortality decline exposure in their birth state, the results are unchanged (Appendix Table A.26, columns (1)-(2)). We modeled migration as a function of post-sulfa declines in pneumonia and maternal mortality using two different indicators for migration. 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 information from the 1940 census. The estimates in Appendix Table A.26 show no evidence that sulfa-induced changes in mortality rates influenced migration.

Other checks Appendix C discusses a number of other specification checks. It shows that our estimates are robust to using alternative age-at-census sample definitions, to adjustment of standard errors for multiple hypothesis testing, exclusion of outlier states and to including woman fixed effects.

4 Theoretical Model: Child Mortality, Fertility and Labor Market Participation

The preceding section shows that women delayed fertility in response to child mortality decline. The intensive margin result is consistent with the predictions of the classical quantity-quality model, and recent innovations which predict that as health improves and mortality declines, women have fewer children and invest more in each (Becker and Lewis 1973). Aaronson, Lange, and Mazumder

[25]When child mortality is high, gross fertility (children ever born to a woman) will overestimate surviving births. Measuring children living with the mother 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 pre-intervention pneumonia mortality, which is part of our exposure variable. Pneumonia mortality rates decline exponentially from birth to age five, after which they flatten out. Therefore, the fact that we find similar estimates when restricting our measure of fertility to children aged below 10 and when including all surviving children, suggests that any survivorship bias is likely to play a limited role in our estimates.

(2014) argue that factors like child mortality decline should increase extensive margin fertility by increasing the value of having at least one child. We, however, find a decrease in extensive margin fertility. We hypothesise that this seeming contradiction can be resolved by taking a wider lens, and considering that child health improvements may affect fertility timing and women's labor market participation. Essentially, when child mortality declines, women can afford to start fertility later to achieve a given fertility target, in addition to which they may lower their target fertility. Fertility delay enables labor market participation and can eventually result in childlessness.[26]

In this section we present a new model incorporating dynamic fertility and labor supply choices. We present here the central economic insights from a simple model, and we discuss extensions to this framework in Appendix D, where we also provide the formal proofs. The model yields predictions not only for extensive margin fertility but also for later life outcomes, including childlessness and labor market participation, which we investigate in Section 5.

4.1 Model Environment

There is a population of women indexed by $i \in [0,1]$ whose life cycle consists of three periods $t \in \{1,2,3\}$. Women have utility $U = A \cdot u(n,e) + c$, where $n \in \mathbb{Z}$ is the (integer) number of their surviving children at the end of the life cycle, $e \in \mathbb{R}_+$ is her choice of child quality, and $c \in \mathbb{R}_+$ is her consumption. We assume that $u(n,e)$ is strictly concave and twice differentiable, with $u(0,e) = 0$ for all e.

We allow for heterogeneity in the preference parameter A, which measures women's preference for children relative to other consumption. We assume that A has cumulative distribution $F(A)$ across women, with density $f(A) = F'(A) > 0$ for all $A \geq 0$.

The timing of events is as follows: At both dates $t = 1$ and $t = 2$, a woman decides whether to get pregnant. If she gets pregnant, denoted $a_t = 1$, she gives birth to a child, who survives with probability $1 - \lambda$, where λ is the rate of child mortality. If she does not get pregnant, denoted $a_t = 0$, she can work for a wage y_t. Her potential wage follows a simple stochastic process that captures the "job then family" pattern of the sulfa drug era (Goldin 2004) and is initialized at $y_1 = 0$. If the does not get pregnant at $t = 1$, she is promoted with probability p, in which case her potential wage rises to $y_2 = Y > 0$. Otherwise, her wage remains at $y_2 = 0$. We also analyze the robustness of our results in the presence of richer income processes, which we discuss below.

At date 3, the woman's fertility is complete, and she takes as given the final number of her surviving children $n \in \{0,1,2\}$. She chooses her consumption c and child quality e so as to

[26] Adda, Dustmann, and Stevens (2017) also study a model in which women choose fertility over the life cycle, which they use to quantify the career costs of fertility in an environment where child mortality and health is held constant. Our model makes a complementary contribution by providing a clear analysis of the effects of health shocks.

maximize utility subject to her budget constraint:

$$\max_{e,c} A \cdot u(n,e) + c \text{ subject to}$$
$$c + n(\tau_q + \tau_e e) \leq \sum_{t=1}^{2} (1 - a_t) y_t + \omega. \tag{2}$$

The left-hand side of the budget constraint (2) measures her spending on consumption and children. As in the classical quantity-quality model (Becker and Lewis 1973, Aaronson, Lange, and Mazumder 2014), her expenditure on children is $n(\tau_q + \tau_e e)$, where τ_q is the price of child quantity, and τ_e is the price of quality per child. Her total wealth, on the right-hand side of the budget constraint, is given by the sum of wages in all periods in which she worked, and an exogenous endowment ω. In contrast to Becker and Lewis (1973) and Aaronson, Lange, and Mazumder (2014), where income is exogenous, we endogenize it.

Substituting the budget constraint (2) into the woman's objective, we find that the woman's choice of child quality e at date 3 must solve the simplified maximization problem

$$V_n(A) = \max_{e} \{A \cdot u(n,e) - n(\tau_q + \tau_e e)\}. \tag{3}$$

The function $V_n(A)$ denotes the woman's *surplus*, i.e., the utility that the woman enjoys at date $t = 3$ over and above consuming her wealth. Note that, for $n = 0$, surplus is $V_0(A) = 0$ for all A, while the optimal e is indeterminate. For $n > 0$, suplus maximization is a well-behaved, concave problem in e, and the surplus is increasing in the preference parameter A. With the dynamic decision process we have specified, our model therefore circumvents the potential non-convexity of the classical quantity-quality model, in which n and e are chosen simultaneously.

In this section, we present a simple case that isolates the effects of child mortality on fertility on the extensive margin. We assume that a woman's surplus is maximized when having at most one child, that is,

$$V_1(A) < V_2(A), \text{ for all } A \geq 0. \tag{4}$$

For example, if the women's preferences have the Cobb-Douglas form $u(n,e) = e^{\alpha} n^{1-\alpha}$, then (4) holds if and only if $\alpha > \frac{1}{2}$. In the Appendix, we extend our results in an environment without this assumption.

4.2 Optimal Fertility and Labor Supply choices

We begin by characterizing a woman's optimal fertility and labor supply choices as a function of the preference parameter A, which measures the strength of her preference for having children. We show that the optimal fertility choice is fully characterized by three scenarios, which we refer to as no fertility, delayed fertility and early fertility:

Proposition 1 *The woman's optimal fertility choice is as follows:*

1. ***No Fertility:*** *If $A < \underline{A}$, where $\underline{A}$ is defined by $V_1(\underline{A}) = 0$, the woman works at $t = 1$ and at $t = 2$ with probability 1.*

2. ***Delayed Fertility:*** *If $\underline{A} \leq A < \bar{A}(\lambda)$, where $\bar{A}(\lambda)$ is defined by $V_1\left(\bar{A}(\lambda)\right) = \frac{pY}{(p+\lambda)(1-\lambda)}$, then the woman works at $t = 1$ and gets pregnant at $t = 2$ if and only if she is not promoted.*

3. ***Early Fertility:*** *If $\bar{A}(\lambda) \leq A$, then the woman gets pregnant at $t = 1$, and gets pregnant again at $t = 2$ if and only if she does not have a surviving child yet.*

The first point in the proposition is clear: if the woman's preference A for children is so weak that the surplus $V_1(A)$ from having a child is negative, then it is never optimal to get pregnant. The second and third points explain the optimal timing of fertility. The proposition establishes that it is optimal for the woman to delay her fertility until $t = 2$ whenever her preference A is strong enough to satisfy the following inequality:

$$(p + \lambda)(1 - \lambda) V_1(A) \geq pY \tag{5}$$

This expression has an intuitive interpretation. The right-hand side of (5) is the *marginal benefit of delay* in terms of income. By delaying, the woman has a probability p of being promoted, which leads to additional earnings Y. The left-hand side is the *marginal cost of delay.* To interpret this term, consider the following two effects. On the one hand, a woman who delays will (optimally) remain childless if she is promoted. If not promoted, she would have had a child with probability $1 - \lambda$. Hence, the possibility of promotion reduces the probability of having a child by $p(1 - \lambda)$. On the other hand, a woman who delays has one fewer attempt at fertility, which reduces the probability of having a surviving child by $\lambda(1 - \lambda)$. Combining these two effects, delaying reduces the probability of having a child by a total of $(p + \lambda)(1 - \lambda)$. Scaling this probability by the surplus $V_1(A)$ associated with having a child yields our expression for the marginal cost. The formal proof of Proposition 1 also verifies the conjecture that it is never optimal for a woman to get pregnant after being promoted. Intuitively, this behavior would be optimal only for women with strong preferences for children, for whom early fertility is a dominant strategy to begin with.[27]

We can gain additional intuition by re-writing Equation (5) as

$$V_1(A)\lambda(1 - \lambda) \geq p\left[Y - V_1(A)(1 - \lambda)\right] \tag{6}$$

The right-hand side can be interpreted as the *option value* of delay, which measures the expected utility gain from getting promoted, which arises with probability p. The left-hand side is the *insurance value* of early pregnancy. 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 $V_1(A)$ with

[27] Different patterns of behavior are possible in models with a richer income process or dynamics beyond three periods. We show in the Appendix that these extensions have more complex solutions, but do not alter our main qualitative results.

probability $\lambda(1-\lambda)$. It is optimal to get pregnant early when the insurance value exceeds the option value of delay.

Next, we derive the overall probability of childlessness in the population of women. This probability is a key object of our empirical analysis in the next section, where we study the impact of sulfa drugs on childlessness.

Proposition 2 *The probability that a woman in the population is childless at date* 3 *is*

$$\begin{aligned} Pr\left[n=0|\,\lambda\right] = {} & F\left(\underline{A}\right) \\ & + \left[F\left(\bar{A}\left(\lambda\right)\right) - F\left(\underline{A}\right)\right]\left(p+\left(1-p\right)\lambda\right) \\ & + \left[1-F\left(\bar{A}\left(\lambda\right)\right)\right]\lambda^2 \end{aligned} \tag{7}$$

This proposition evaluates the probability of childlessness in each of the three cases that govern the optimal choice, weighted by the mass of women that fall into each case. The top line in Equation (7) is simply the fraction of women with no fertility. The middle line is the fraction of women with delayed fertility times their probability of childlessness. This probability is the sum of p, since they are childless if promoted, and $(1-p)\,\lambda$, since they are childless if they are not promoted but suffer an unsuccessful pregnancy. Finally, the bottom line is based on the fraction of women who choose early fertility. Since these women have two attempts at having a child, their probability of childlessness is lower and equal to λ^2.

4.3 The Effect of Shocks to Child Mortality

We now characterize the effects of a change in the rate λ of child mortality on women's incentives.

Proposition 3 *The optimal choice, as characterized in Proposition 1, responds to changes in the child mortality rate λ as follows:*

1. *The threshold $\underline{A}$, at which a woman switches from no fertility to delayed fertility, is independent of λ.*

2. *The threshold $\bar{A}\left(\lambda\right)$, at which a woman switches from delayed to early fertility, satisfies $\frac{\partial\bar{A}}{\partial\lambda}<0$ if and only if λ satisfies*
$$\lambda < \frac{1-p}{2}. \tag{8}$$

The first point in this Proposition is that the boundary between no fertility and early fertility does not change in response to a change in λ. This is because no fertility is chosen only by women who perceive negative surplus from having a child. The boundary is therefore at the point where surplus (as defined in (15)) is zero, which is independent of λ.

The second point shows that incentives to delay are decreasing in λ, as long as λ is not too large. This is because an increase in λ raises the marginal cost of childlessness by reducing the likelihood of success for a woman who has only one attempt at having a child.[28]

Figure 6 provides a numerical illustration of these effects. The figure considers a range of values for λ along the horizontal axis and a range of preference parameters A on the vertical axis. Motivated by the empirical setting, we focus on values for the child mortality rate $\lambda \leq 15\%$. We plot the regions in which women choose no fertility, delayed fertility and early fertility. Consistent with Proposition 3, Panel (a) shows that the region of delay widens when λ declines, as women switch their behavior away from early fertility. For the same preference parameter, a woman is more likely to delay her fertility as child mortality declines. Panel (b) shows that the pattern also holds, and indeed becomes more pronounced, if the woman is fertile for a greater number of time periods (see our discussion of the general dynamic model below and in Appendix D.3).

The interesting and hitherto little recognized implication is that a *decline* in child mortality can lead to *increased* incentives to delay fertility. Several extensions to our model, which we discuss in detail below, show that this finding is robust to enriching the specification. For example, we show that it goes through in environments that feature a richer income process, general dynamic decisions, increasing risks of infertility with age, as well as optimal marriage and career choices.

Before discussing the detailed empirical implications of this result, we characterize its implications for the rate of childlessness in the population as a whole.

Proposition 4 *The probability of childlessness, as characterized in Proposition 2, responds to a change in the child mortality rate λ as follows:*

$$
\begin{aligned}
\frac{\partial Pr\left[n=0|\,\lambda\right]}{\partial\lambda} &= \underbrace{\left[F\left(\bar{A}\left(\lambda\right)\right)-F\left(\underline{A}\right)\right]\left(1-p\right)+\left[1-F\left(\bar{A}\left(\lambda\right)\right)\right]2\lambda}_{\textit{mechanical effect}} \\
&\quad+\underbrace{f\left(\bar{A}\left(\lambda\right)\right)\frac{\partial\bar{A}\left(\lambda\right)}{\partial\lambda}\left(p+\lambda\right)\left(1-\lambda\right)}_{\textit{behavioral effect: switch early}\,\rightarrow\,\textit{delay}} \qquad (9)
\end{aligned}
$$

This result decomposes the effect of a change in λ on the probability of childlessness into two terms. The first line in Equation (9) is the mechanical effect of a change in λ, holding women's behavior constant. This effect is always positive. Any increase in λ mechanically leads to fewer surviving children and, hence, a higher incidence of childlessness. This force affects women who choose either delayed or early fertility. The second line is the behavioral effect that arises from changes in women's optimal choices. Women with $A=\bar{A}\left(\lambda\right)$, who are indifferent between early and delayed fertility, switch their optimal strategy in response to a marginal change in λ.[29] This effect has the sign of $\frac{\partial\bar{A}(\lambda)}{\partial\lambda}$ and scales with the density $f\left(\bar{A}\left(\lambda\right)\right)$ of "switchers".

Concretely, consider the case where λ is small in the sense of Condition (8). Suppose that there is a beneficial health shock that decreases λ. The mechanical effect is a decrease in childlessness.

[28] By contrast, the marginal cost $\left(p+\lambda\right)\left(1-\lambda\right)V_1\left(A\right)$ of delay is decreasing in λ for large values of λ. Intuitively, as $\lambda\rightarrow1$, both pregnancies of a woman who chooses early fertility are likely to fail, so that the "insurance" value of starting early declines to zero.

[29] For a discrete change in λ, this implies that there is an interval of women who will switch their behavior between delayed and early fertility.

The behavioral effect, however, is that some women switch from early to delayed fertility, which reduces their overall probability of having children. If the density of switchers is large enough, then the behavioral effect dominates and the overall effect on childlessness is positive.

In a model with a more general income process, there is an offsetting effect whereby a decline in λ can encourage women to switch from no fertility into delayed fertility.[30] However, when the density of switchers from early to delayed fertility is sufficiently large, this additional effect is dominated by the effects of increased delay.

4.4 Empirical Predictions

In our empirical analysis, we are interested in the causal effects of a beneficial shock, namely, the introduction of sulfa drugs, which led to declines in both child mortality and child morbidity. As a benchmark, recall the predictions of the classical quantity-quality model of fertility (Becker and Lewis 1973). In this model, women only solve a static surplus maximization problem similar to (15). Beneficial health shocks in this framework are typically modeled as declines in the prices (τ_n, τ_q) of child quantity and quality. The empirical implication of the quantity-quality model is that beneficial health shocks always decrease the likelihood of childlessness, because they increase the surplus associated with having at least one child (Aaronson, Lange, and Mazumder 2014).

In contrast to the basic quantity-quality model, our model predicts that a *beneficial health shock increases the likelihood of childlessnes*s, as long as it enocourages enough women to delay their fertility. As we have discussed above, a necessary condition for this prediction is that the baseline rate λ of child mortality is not too large, that is, that Condition (8) holds. In our empirical setting, a back-of-the-envelope calculation suggests that this condition is plausible. A population average child mortality rate of 47 per 1000 live births in 1939 (Dowell, Kupronis, Zell, and Shay 2000) implies that $\lambda = \frac{47}{1000}$, so that Condition (8) is satisfied if the probability of promotion is less than 90.6%.[31]

The effects of health shocks through prices, which are the focus of the quantity-quality literature, can also be incorporated in our dynamic model. If a health shock decreases the rate of child mortality but also decreases prices, then the effects of lower prices and of decreased mortality push women's optimal fertility timing decisions in opposite directions. In this case, one needs to impose a tighter upper bound than (8) on λ to ensure that a beneficial health shock encourages delayed fertility (see Appendix D.2).

In our baseline model, we have focused on extensive margin fertility decisions. In Appendix D we further consider higher birth orders. This leads to an additional prediction, namely, that *a beneficial health shock leads to lower numbers of children on the intensive margin*, again as long as it leads a sufficient measure of women to delay their fertility.[32]

[30] For example, consider a model where the pre-promotion wage, which we set to zero for simplicity, is $y > 0$. No fertility in this model is optimal whenever $A \leq \underline{A}(\lambda)$, which is implicitly defined by $y = (1-\lambda) V_1 (\underline{A}(\lambda))$. Moreover, $\underline{A}(\lambda)$ is increasing in λ.

[31] Women in this era primarily worked in teaching and typing jobs, where the probability of promotion was low (Goldin 2004).

[32] Unlike the predictions of our model on the extensive margin, this prediction is also a feature of the standard

The key mechanism in our model is that women have stronger incentives to enter the labor market, and have children later, after a positive health shock. When they do, they benefit from the possibility of being promoted and, if promoted, they remain in the labor market instead of having children. Thus, the model delivers several auxiliary predictions that allow us to test the mechanism behind changes in fertility. Concretely, our model predicts that a *beneficial health shock*:

1. Leads to later fertility, even among women that eventually have children, also increasing marriage delay and the risk of never marrying (see Appendix D.4.4),

2. Increases female labor force participation, and

3. Increases the number of women in more prestigious, highly paid employment.

In the next section we take these predictions to the data.

4.5 Extensions and Robustness

The main novelty in our model is the result, stated in Proposition 3, that declines in child mortality can lead to increased incentives to delay fertility. This is true for parameter values delineated by Condition (8), which is likely to be satisfied in our empirical context. We now discuss a series of extensions to the model. These extensions are analyzed formally in Appendix D. In each case, we analyze the equivalents of Proposition 3 and Condition (8) and discuss how they deviate from the baseline model.

1. *General dynamics (Appendix* D.3). We extend the baseline model to T periods. We derive a closed form solution to the Bellman equation associated with optimal fertility timing. We show that, if Condition (8) holds, a decline in child mortality λ strengthens incentives to delay fertility across the board. For example, the optimal date of the first pregnancy switches from $t = 1$ to $t = 2$ for a range of preference parameters A, from $t = 2$ to $t = 3$ for another range, and so forth. Thus, the qualitative effects in a general dynamic model are the same as in the simple three-period model we have presented above.

2. *Income effects (Appendix* D.4.1). We consider more general preferences and a richer income process to study the potential implications of income effects. Income effects become relevant when child quality (e.g., education) accounts for a large fraction of households' expenditure. In this setting, a woman who has worked for one period spends more on child quality and enjoys greater surplus from having a child. Therefore, changes in λ have a stronger effect on the value of delay than on the value of early fertility, which reinforces the mechanism by which health shocks encourage delay. Accordingly, with some natural restrictions on preferences, we show that income effects strengthen our argument in the sense that we now obtain our key results under a weaker condition than (8).

quantity-quality model, in which women tend to substitute out of quantity and into quality after a beneficial health shock.

3. *Higher birth orders (Appendix* D.4.2). We relax Assumption (4) so as to incorporate women who wish to have more than one child. Intuitively, the prospect of having several children weakens women's incentives to delay their fertility. We therefore derive a stronger condition (8), which guarantee that declines in child mortality encourage fertility delay.

4. *Increasing risk of infertility (Appendix* D.4.3). We consider a model in which the woman risks becoming infertile each period, so that the overall risk of infertility is increasing over time. We show that a slightly stronger condition than Condition (8) is needed to recover our main results in this case. Intuitively, declining fecundity makes delayed fertility less attractive, and also reduces the incentive to switch to delayed fertility after a health shock. Figure A.6 shows that the pattern of increased delay in the numerical example also holds when we allow for increasing risk of infertility.

5. *Marriage decisions (Appendix D.4.4).* For the sample of women in our empirical analysis, the social norm was to leave the labor force and get married before having children. We explore the implications of our model for marriage decisions by assuming that a woman's first pregnancy is contingent on finding a partner and getting married. We further assume that finding a partner is probabilistic. This feature makes delayed fertility a more risky prospect than early fertility. The economic effects of this force are similar to the case with an increasing risk of infertility, which we have discussed above.

6. *Career choices (Appendix D.4.5).* We analyze the behavior of women who can choose between a risky career with large income upon promotion, such as we have modeled above, and a safe career with a flatter income trajectory. We show that women who plan to delay fertility self-select into risky careers. We also demonstrate that the presence of career choices does not alter the qualitative effects of changes in child mortality.

5 Impacts on Later Life Outcomes: Fertility, Labor Market and Marriage

In this Section, we bring the data to bear on the theoretical predictions of the model in Section 4. Having already established that sulfa exposure caused women to delay fertility at both the intensive and extensive margins, we test whether it also led to more childlessness, lower completed fertility, higher labor market participation, better careers, and less marriage. We find evidence in support of these predictions.

5.1 Data

As before, we analyse the sample of women who were of reproductive age in a window around the time of the introduction of sulfa drugs. Earlier we studied their birth outcomes during 1930 to 1943. We now track the same cohorts of women into their later life, identifying them in later census years using the 1% microdata samples of the 1940 to 1970 censuses. To model completed fertility, we analyse women who were age 40-50 at the time of census enumeration.[33]

We also show results for women of childbearing age (18-40) at census enumeration, to corroborate any impacts on delay that we estimate in the hazard model. The sample includes birth cohorts 1893-1931 who were aged 6-44 in 1937. We exclude women aged five and under in 1937 as they were potentially directly treated by antibiotics as children. We include not only women aged 15-40 in the period 1930-1943 (as in the birth timing sample) but also women who would have been exposed to the sulfa drug era throughout their reproductive years (aged 6-15 in 1937) and women who would not have been exposed at all (aged 40-44 in 1937).[34] As in the birth timing sample, we focus on net fertility (children resident in the household), but in a robustness check we also use data on gross fertility (total number of live births) to show that both measures yield comparable estimates. Data on labor market and marriage outcomes are from the same census files, and we model outcomes for women aged 18 to 50 at the time of the census.

Table A.2 in the Appendix provide descriptive statistics. Using net fertility (based on surviving children) among women of childbearing age at the time of the census, the average woman was exposed to sulfa drugs for 20 years, and 36% of women were childless, with 1.7 births in total, and 2.6 conditional on at least one. Among women who had completed their fertility at the time of the census, the average woman was exposed to sulfa for 14.6 years, 28% of women were childless at the end of their reproductive years, average total fertility was 1.9 (unconditional on at least one birth) and 2.7 conditional on at least one. The mean age at first birth in the stock sample was 24.1, and 26.7 at second birth. In the labor maket sample, 37% of women were in the labor force, and 35% were working, with average working hours of 13 per week.[35] 73% of women reported being currently married, and 85% had been married at least once previous to the date of census enumeration.

5.2 Empirical Strategy

As we consider cumulative fertility and marital and labor market outcomes later in life, the relevant measure of exposure is the number of fertile years that occur in the post-1937 period. We again leverage variation in pre-1937 pneumonia as a proxy for treatment intensity in a double difference specification, interacting sulfa exposure years with pre-sulfa mortality rates at the birth state level. The estimating equation for outcomes measured later in life is:

[33] The oldest woman in this was 44 in 1937, and she was reproductive until 1933, and the youngest woman was six in 1937, so that she was reproductive from 1946 onwards.For example, the 1917 cohort will have turned 40 in 1957, so we find them in the 1960 census when looking at their completed fertility.

[34] 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 are very similar when we restrict the sample of each estimate to have exactly the same cohorts.

[35] This includes women reporting zero hours of work; focusing only on positive working hours, the average was 37.9.

$$
\begin{aligned}
Y_{jsc} &= \beta + \beta_1 * prePneumonia_s * sulfayears_j \\
&+\beta_2 * preMMR_s * sulfayears_j + statecontrols_s * sulfayears_j \\
&+race_j + educ_j + \kappa_s + \phi_c + \omega_{jsc},
\end{aligned} \tag{10}
$$

where Y_{jsc} is the outcome of woman j born in state s in cohort c recorded at the time of the census. The coefficient of interest is β_1. The variable $sulfayears_j$ is the number of fertile years of woman j during which she was exposed to sulfa drugs. We assume women are fertile between the ages of 15 and 40.[36] Similar to the main model for birth timing, this model includes fixed effects for the woman's birth cohort and birth state, her race and education, and the same set of state-time varying controls detailed in Section 3.2 interacted with $sulfayears_j$. Standard errors are clustered at the birth state level.

Since we model cumulative exposure and not an event, this specification is not suitable for an event study, but we provide a descriptive analogue, depicting a dose-response relationship between fertility and years exposed to sulfa drugs. As $sulfayears_j$ is defined on year of birth, we also show results replacing this linearly evolving exposure measure with a binary measure that compares women who were fully exposed with women who were unexposed, removing women who were partially exposed.[37]

To identify the extensive margin response (childlessness), we redefine the dependent variable as a binary indicator for non-zero versus zero children. To identify the intensive margin response, we re-estimate equation (10) for total number of children but restricting the sample to women with at least one birth. We estimate impacts on the following labor market outcomes: whether in the labor force; whether working; the hours worked in the past week; the Hauser-Warren occupational score (see Appendix A for a precise definition), and own income in the last year. We estimate impacts on the following marriage market outcomes: whether currently married, ever married and the age at first marriage conditional on ever having married.

5.3 Results: Impacts on Childlessness and Total Fertility

We have identified that women delayed the transition to first birth and to higher order births in response to the introduction of sulfa drugs. To assess whether the response was only an intertemporal substitution, we estimate impacts of the intervention on total fertility of the same cohorts of women tracked through to a later stage of their lifecycle. We first discuss estimates for women who

[36] Notice that when we use the sample of women of reproductive age at the time of the census in these later census files, $sulfayears_j$ may include future years: for example, a woman aged 33 at the time of the 1950 census who was 20 in 1937, would have 20 fertile sulfa years in total, of which 7 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.

[37] A number of recent studies suggest that including unit-specific trends can either exacerbate biases inappropriately or washout treatment effects when the evolution of these effects increases over time (or as in our case, with increasing cohort exposure); see, for example, Young and Gary (2011); Bhuller, Havnes, Leuven, and Mogstad (2013); Goodman-Bacon (2021).

were still childbearing at the time of the later census. These estimates will capture a combination of fertility delay and changes in fertility targets, which is useful to corroborate the birth timing estimates. We then report results for women who have completed childbearing.

An increase in childlessness will mechanically decrease intensive margin fertility. For instance, if women who would otherwise have had one child switch to having none, then the increase in childlessness will be mirrored in a decrease in the share of women with one child. We therefore also estimate the impact of pneumonia mortality decline on the distribution of fertility.

Number of children among women of childbearing age Using data on women aged 18-40 at census interview and modelling the number of children they have had at the time of interview confirms the birth timing results. There is a significant decline in fertility at both the intensive and extensive margins (Panel A, Table 3).[38] The specification in column (1) implies that an interquartile decline in pneumonia mortality (0.26) was associated with 0.013 fewer births for an additional year of exposure to sulfa drugs. Relative to the mean of 1.66 children in the estimating sample, a woman with the average years of exposure in this sample (20) had 0.25 fewer births, or 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 of 2.6 births). A similar calculation 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 is 36% in this still-childbearing sample).

To estimate impacts on the distribution, we define indicator variables for the number of children a woman has at census being 0, 1, 2, 3 and 4+ and estimate equation (10) separately for each of these outcomes, see Figure 7. We see 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: in response to pneumonia (child) mortality decline, women were more likely to be childless and less likely to have three or more children. Notably, although the coefficients are negative, there was no significant change in the share of women with either one or two children.

Completed fertility We now discuss fertility impacts on women who were age 40-50 at census and had thus plausibly completed their fertility. We present results using the same net fertility measure as used in the hazard model, which relies on counts of children living at home, for which we impose an upper limit of age at census of 50 to reduce the omission of children who have left home.[39]

The pattern of results is similar to that documented so far: pneumonia mortality decline is associated with a significant decline in fertility on both the extensive and intensive margins. In

[38] For some of these women, a disproportionate share of whom are younger and hence sulfa-treated during their reproductive years, fertility is right-truncated. This will be captured by fixed effects for the woman's age, which we consistently include.

[39] We will underestimate fertility counts because some of the older children will have left home. Since older women in the sample will have had fewer years of sulfa exposure, if we selectively under-estimate their fertility, we will tend to under-estimate the coefficient of interest. The estimates we show are, by this criterion, conservative.

particular, an interquartile decline in pneumonia mortality (0.26), evaluated at the average number of reproductive years of exposure to sulfa drugs (14.9), led to 0.11 fewer children for the average woman, which is 5.7% of the pre-sulfa baseline mean, and a 1.4 percentage point increase in the probability of being childless, a 5.0% increase from baseline (Panel B, Table 3).

Comparing estimates for net fertility among women age 40-50 at enumeration with the estimates for fertility of women of childbearing age (18-40) from the preceding section (comparing Panels A and B in Table 3), the coefficients for women who have completed fertility are consistently smaller than the coefficients for women who are of childbearing age. This is also evident when comparing changes in the distribution of incomplete and completed fertility (Figure 7). The estimates suggest that two thirds of the estimated impact of pneumonia mortality on childlessness in the childbearing-age sample is compensated delay, and that one third is a rise in permanent childlessness; the latter will likely incorporate both a decline in target fertility and uncompensated fertility delay that may arise from biological decline in fecundity or from failure to find a marital match, as modelled in Section 4.

5.4 Results: Labor Market and Education Outcomes

Panel A of Table 5 shows that reductions in pneumonia mortality led to improved labor market outcomes for women by every indicator used, other than income.[40] A decline in pneumonia mortality corresponding to an interquartile shift in the distribution, for a woman with the mean exposure to sulfa drugs in the estimating sample (18.3 years), is estimated to have increased the probability of being in the labor force by 2.6 percentage points and the probability of being employed of 2.8 percentage points. The average labor force participation rate (employment rate) in this sample is 37.1% (35.1%), so this increase is 7.0% (8.0%) of the baseline rate. This is of broadly similar magnitude to our estimate of the increase in the share of childless women.[41]

The same decline in pneumonia mortality led to an increase in the occupation-based socioeconomic index of 6.6% relative to the baseline index score of 14.4, suggesting that women exposed to sulfa drugs had better careers, consistent with the mechanism in Section 4. We obtain similar results when considering other occupational scores, including *occscore* and the Duncan socioeconomic index. Pneumonia mortality decline increased weekly working hours by 1.15 hours, or 9% of the baseline mean, which implies an annual increase of 13.8 hours for the sample average of 12 working weeks per year.[42] Although the coefficient on pneumonia mortality decline is positive for personal income, it is imprecisely estimated.

[40] As the census asks women about their current labor market status, we include all women aged 18-50 at the time of the census, pooling both childbearing women and women aged 40-50, for whom we estimated effects on completed fertility. 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 Appendix C.

[41] If every additional childless woman worked (4.6pp from the net fertility measure), the increase in labor force participation would be 12.4% of baseline labor force participation. Thus, 7% underlines the plausibility of the link between changes in childlessness and employment of women.

[42] To put this in perspective, Bailey (2006) estimates an annual increase of 68 hours among cohorts with access to the birth control pill.

The joint probability of childlessness and labor force participation Thus far we have demonstrated, using independent reduced form equations, that the sulfa-led reduction in pneumonia mortality led to lower total fertility, higher childlessness and higher rates of labor force participation, employment, working hours and occupational quality among women. Tabulating labor market outcomes by childlessness status (Table A.3 in the Appendix) shows that childless women were 34 percentage points more likely to be in the labor force, worked 12 additional hours per week, earned over $1000 more per year and had higher occupational scores than women who were mothers. In order to provide robust evidence that these choices are linked and, in particular, that the same women that respond with higher childlessness respond with higher labor force participation, we estimate the impact of sulfa-led mortality declines on the joint probability of being childless and being in the labor force (Table 6).

Reductions in pneumonia mortality increased the probability that women were both working and childless by 2.7 percentage points (13% of the baseline probability of 20.5%); see column (1). The probability of not being in the labor force and having children declined by 3.2 percentage points, or 6.5% (column (4)), while the probability of not working and being childless, or working and having children, did not change. We cannot reject that the coefficients in columns (1) and (4) are of the same magnitude but opposite sign, which indicates that among women experiencing greater declines in pneumonia mortality, stay-at-home mothers became less common, while working women without children became more common.

Education outcomes To complement the results on labor market outcomes, in Appendix Table A.15, we report estimates of the effect of sulfa-induced mortality decline on education choices. Note that the fact that we consider women who were already of childbearing age in 1937 in our main analysis means that many of these women will have already completed their education (these were 74% in the birth timing sample and 68% in the completed fertility sample). We nevertheless investigated education acquisition 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, as college completion typically occurred in the early 20s. We estimate a 5.4 percentage point increase in the probability of high school completion, relative to a baseline mean of 19.2% for this subsample of women. We find no effect on the rate of college completion, which is consistent with Goldin, Katz, and Kuziemko (2006), who discuss the timing of the expansion of college education, and that this occurred later, among the children (rather than the mothers) of the sulfa cohorts.

5.5 Results: Marriage Market Outcomes

Fertility delay and investment in the labor market is likely to have repercussions for the marriage market, and this mechanism is also discussed in an extension to the model in Section 4. We explore 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, and ever married status for women aged 18 to 50 at census.[43] The results are in Panel B of Table 5: reductions in pneumonia mortality led to both postponement of marriage and a decline in marriage entry. A reduction in pneumonia mortality of the size of an interquartile shift is estimated to have reduced the probability of women ever having married by 1.4 percentage points (1.65% of baseline mean), and, among still childbearing women, to have reduced the probability of being married at the time of the census by 1 percentage point (1.5%).

Baudin, de la Croix, and Gobbi (2019) propose a model of childlessness in which marriage is a key pathway.[44] In line with their predictions, we find that child mortality decline is associated with lower chances of having ever married, alongside an increase in childlessness. However, the coefficients indicate small effects relative to the labor market responses, suggesting that marriage was one important margin of response, but that labor force participation, rather than marriage, was the main mediator of impacts of child mortality decline on childlessness in our setting.

5.6 Robustness checks

This section discussed specification checks on the later life outcomes. Many of the checks presented here are motivated and described in Section 3, so we discuss them briefly and point to the results. We conduct all robustness checks on the net and gross fertility measures, to provide firm evidence that our results are not driven by our choice of fertility measure.

Nonparametric patterns To corroborate our results, we show that similar patterns emerge in a nonparametric specification that compares average outcomes over time in states that had above versus below median pneumonia mortality in the pre-sulfa era. Figure A.5 in the Appendix shows that in the 1950, 1960 and 1970 censuses, women in above-median mortality states who experienced larger declines in child mortality exhibited increases in childlessness alongside lower intensive margin fertility, relative to women in below-median states. In the pre-sulfa years (1930 census and before), the trends in fertility outcomes look similar across these two groups of states. Similarly, while labor force participation rates converge until 1940, they diverge after that, with women in above-median mortality states participating in the labor force in greater numbers. Finally, marriage rates evolve in a parallel fashion up until 1940, after which the marriage rates of women in above-median mortality states fall and remain lower than those of women in below-median mortality states. These patterns are consistent with the causal estimates.

Omitted trends The birth timing results are identified on the assumption that the event of the introduction of sulfa drugs disrupts women's birth profiles. In contrast to most studies of fertility, we provide estimates both for birth probabilities around the event and for the eventual number

[43] Estimates using alternative age-at-census samples are in Appendix C.

[44] An important assumption in their model is that women have imperfect control over fertility. Child mortality acts to limit the fertility of women of low socioeconomic status. Thus, they argue, a reduction in child mortality will make less educated women less attractive on the marriage market, leading to increased childlessness and lower fertility. We thank David de la Croix for this insight.

of children. By their nature, models of end-line fertility rest on stronger identifying assumptions than models of fertility behaviour on either side of an event. Nevertheless, the results stand up to a number of checks. There is no natural event study but we provide a visual depiction of changes in the outcome against variation in exposure. As exposure is a function of birth cohort, we interact birth cohort "bins" with baseline pneumonia mortality, and plot the coefficients on the interaction terms in Appendix Figure A.4. The omitted case is zero years of exposure. Panels A and B confirm the main results that childlessness was more likely with higher years of exposure, and that the total number of children was smaller. Panel C confirms hat women with higher years of exposure were more likely to be in work.

Panel B in Appendix Tables A.5, A.6 and A.7 adds controls for a full set of cohort * census region fixed effects, and the coefficients are stable even in this more demanding specification. We display sensitivity of the coefficients to varying the controls included in Table A.8. We augment the rich set of state-level controls for disease environment, economic conditions and surveillance quality with occupation structure. We include the state level occupational distribution of women in 1930 interacted with $sulfayears$ (Panel A, Appendix Tables A.16, A.17 and A.18). Finally, we include as a control adult mortality rates for placebo diseases interacted with $sulfayears$ (Panel B, Appendix Tables A.16, A.17 and A.18).

We address the possible role of mean reversion by controlling for the state-level pre-sulfa average value of the outcome variable (Panel D, Tables A.9-A.11). We estimate impacts of a "placebo" intervention using an approach similar to that in Aaronson, Lange, and Mazumder (2014). This directly addresses concerns over omitted long-run trends in the stock model. We use data on individual outcomes in the 1910-1930 censuses, selected so that the outcomes were realized before the invention of sulfa drugs, and we create a placebo intervention forty years previous to the true year (i.e. in 1897 rather than 1937), so that we have sufficient variation in placebo sulfa exposure in the census data. We then estimate the baseline specification of the stock model. The results in Panel A of Appendix Tables A.20-A.22 show that the coefficients on pneumonia mortality are small and insignificant.

Since individual years of exposure evolve linearly as a function of birth cohort, we redefine the exposure variables to be binary, comparing the fully exposed with the unexposed, and omitting partially exposed women from the sample. The results are consistent with the main findings (Appendix Table A.28).

To provide evidence that our results do not capture differences in trends between regions of the U.S., we show estimates omitting the mountain west and deep south states in turn (Appendix Tables A.20-A.22, Panels B and C). We also check that the coefficients on pneumonia mortality decline are not sensitive to omitting maternal mortality decline (Panel B in Appendix Tables A.33-A.35).

Coincident events As with the birth timing model, we account for events occurring around the time of sulfa drug intervention: WW2, the Great Depression and New Deal program and the Dust Bowl. To account for the Second World War, in the stock model, we control for state-level troop deployment, obtained from Goldin and Olivetti (2013), interacted with individual exposure to the war, measured as the number of fertile years of the woman from 1942 onwards. Our findings are essentially unchanged (Panel B, Tables A.9-A.11). We interact New Deal spending (Fishback, Kantor, and Wallis 2003) with sulfa exposure ($sulfayears$ in the stock model). The coefficients of interest are robust to this (Panel A, Appendix Table A.9 for fertility outcomes, and Panel A, Appendix Tables A.10 and A.11, for labor and marriage market outcomes). Finally, we estimate a specification where the Dust Bowl states are omitted (Panel C, Tables A.9-A.11).

Measurement issues To address the concern that the net fertility measure misses children who have left home, we show similar estimates in three variants. First, we estimate results using gross fertility, defined by a census question on total live births. These estimates, in Table 4, show 0.081 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 estimates are very similar to those for net fertility.

Second, we follow Aaronson, Lange, and Mazumder (2014) and in our measures of net fertility only include children aged below 10 (Panel B of Appendix Table A.20). A further 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 are similar when considering net fertility among different age groups of women at census, focusing on younger women where children are less likely to have moved out; see Appendix C. We also show estimates that use under-5 pneumonia mortality in place of the all-age rate, instrumented with the all-age rate. The broad pattern of results holds, see Appendix Tables A.5, A.6 and A.7.

Our argument is that the decline in child mortality releases women's time away from childbearing and caring, allowing them to avail of labor market opportunities. We considered the alternative explanation that labor market outcomes of women improved because the women themselves became healthier. This is undermined by the low rates of morbidity and mortality from pneumonia among prime-age adults. Nevertheless, to investigate this, we re-estimated the model including baseline pneumonia mortality rates of 25-35 year-old adults alongside rates for children under five. This is shown for labor market outcomes in Appendix Table A.13, and for completeness similar specifications for fertility and marriage outcomes in Tables A.12 and A.14. The coefficients on the adult rate are mostly insignificant and of the opposite sign, while the coefficients on the child rate are consistent with the main estimates.

In the stock model, we also check that our conclusions are unchanged by including migrating women and instrumenting their mortality decline exposure in their residence state with their birth state (Appendix Tables A.23-A.25).

Other checks Appendix C discusses and shows that our estimates are robust to the following further checks: alternative age-at-census sample definitions; adjustment of standard errors for multiple hypothesis testing; including woman fixed effects; and exclusion of outlier states.

6 Broader Relevance: Long-Run Patterns in the Data

We have shown 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. To complement these findings from the context of sulfa drugs, we demonstrate that these patterns are evident more broadly, in the raw data for the United States in 1930, as well as in contemporary data across African countries.

Figure 8 plots the cross-sectional correlation across U.S. states in 1930 of pneumonia mortality with each of childlessness and total fertility. Childlessness is negatively correlated with pneumonia mortality, while total fertility is positively correlated with it. The Figure also shows scatter plots of labor force participation and marital status against child mortality. Labor force participation is inversely correlated with child mortality (though less pronounced than childlessness), while marriage is positively correlated, consistent with our causal estimates.

Turning to contemporary data, Figure 9 plots the cross-country relationship between fertility, labor supply and marriage and infant mortality (from all causes), across African countries in 2015. Similar to the early 20th century data from the U.S., these contemporary data show a positive correlation of infant mortality with both total fertility and ever married rates, and a negative correlation of infant mortality with childlessness and labor force participation. These figures illustrate that the effects of child mortality decline that we document using the invention of sulfa drugs can be seen as broad correlational patterns in different time periods and different settings.

7 Conclusions

The analysis produces two striking 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 the two findings are linked, namely, that child mortality decline made it rational to delay fertility, and that this led to the changes in labor market and marriage outcomes that we document.

Reductions in child mortality allowed women to delay the start of fertility, reduced the total time that women had to spend childbearing, and also reduced their target number of children due to the effect of improvements in child health and child quality. They additionally reduced the time that women, being main caregivers, spent caring for sick children. Together, this will have allowed women more time for productive activities. For 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.

We outline a dynamic model of fertility and labor market choices that shows a greater propensity for fertility delay and labor force participation in response to a decline in child mortality, when the joint probability of promotion at work and child mortality is low. The estimated patterns are consistent with this theory, fairly large, and robustly determined. We also document these patterns in other time periods and contexts, namely in correlational plots in contemporary African data, and US data before the invention of sulfa drugs.

The estimates are relevant to debates concerning the trade-off between career and family, the rise of female labor force participation, and to models of the demographic transition and economic growth. We provide new evidence on the drivers of childlessness and female labor force participation, and the need to consider fertility, labor market and marriage market choices in conjunction. No previous work appears to have proposed and tested the idea that child mortality decline may influence labor force participation and marriage decisions of women, by triggering fertility delay.

Our findings are relevant for contemporary development policy. Although there have been marked declines in child mortality in the last 25 years in response to worldwide mobilization and increasing investments in public health, there is limited causal evidence of fertility and labor market responses to these investments. Our findings suggest that investments in child mortality decline can contribute to the economic independence of women, where labor market opportunities are available. Women's labor force participation and associated economic independence can lead to increased investments in children (Lundberg, Pollak, and Wales 1997, Baranov, Bhalotra, Biroli, and Maselko 2017) and a reduction in domestic violence (Aizer 2010, Bhalotra, Britto, Pinotti, and Sampaio 2021).

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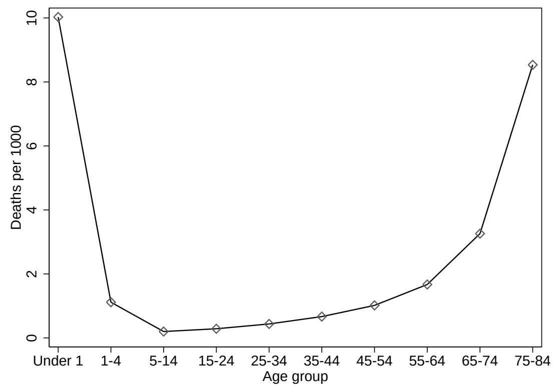
Tables and Figures

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

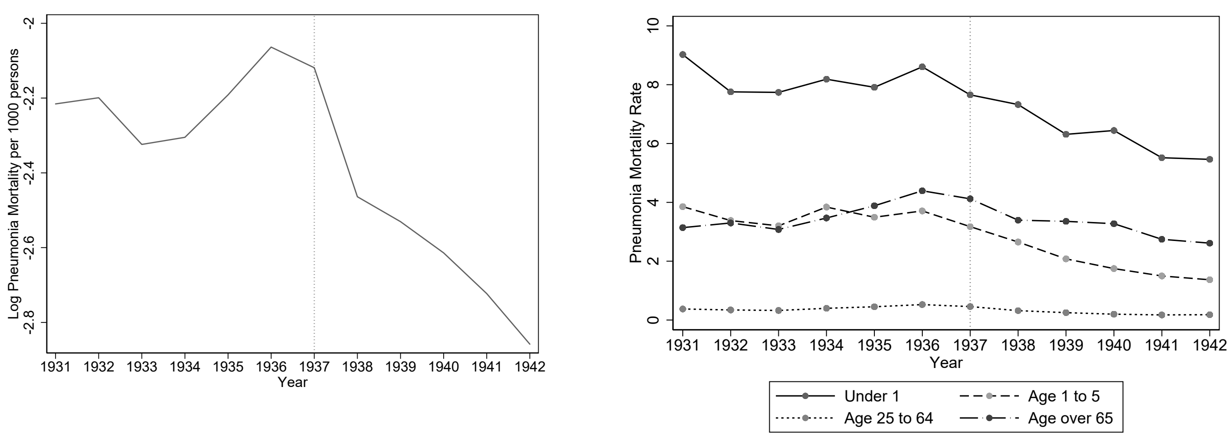


This figure shows the average pneumonia mortality rate by age group in 1935 in the United States. Source: Britten (1942).

Figure 2: Pneumonia Mortality, United States

(a) All age groups

(b) By age group

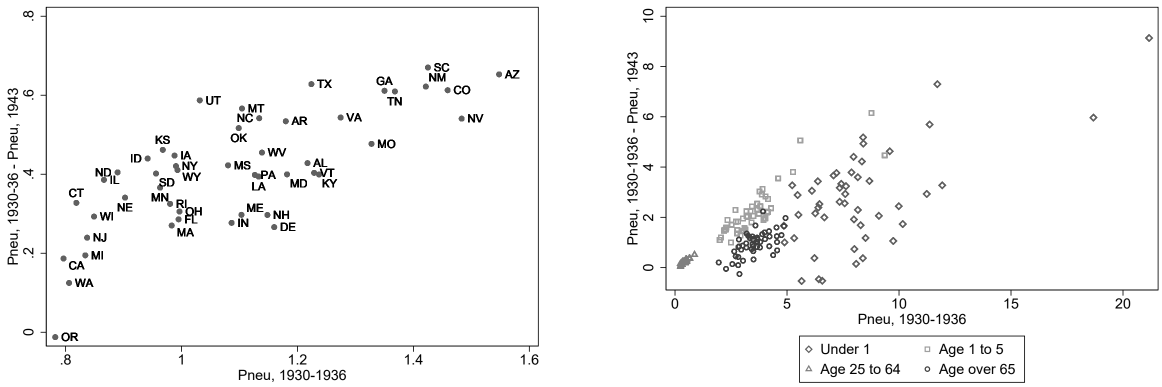


These figures show the log average pneumonia mortality rate for all ages (left) and by age group (right) in the United States over time. Source: Vital Statistics.

Figure 3: Pneumonia Mortality Convergence Post-1937, United States

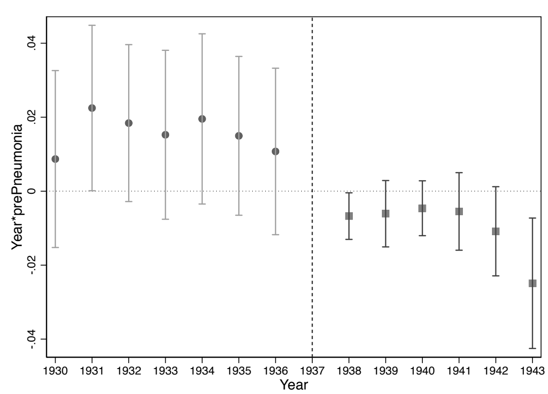
(a) All age groups

(b) By age group



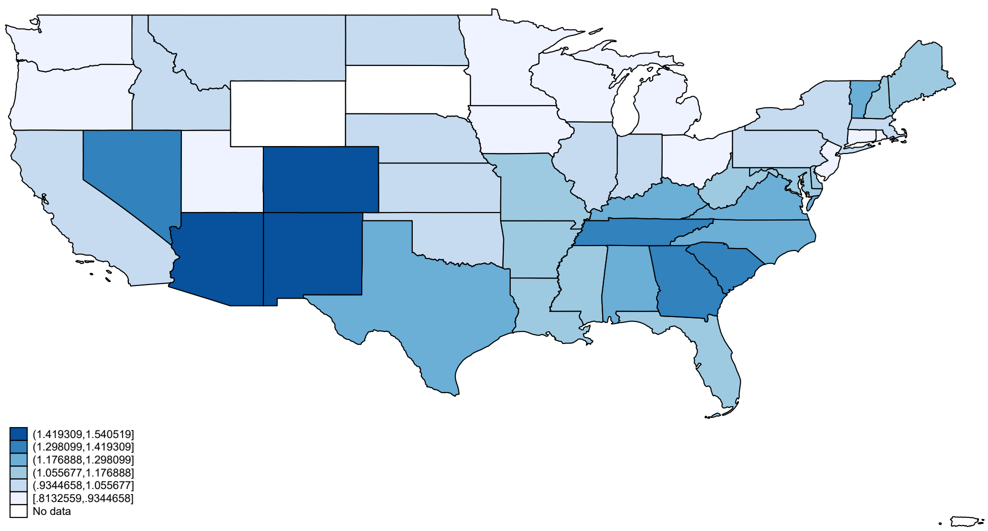
These figures show the relationship between the 1937-1943 absolute change and the 1930-1936 average level of pneumonia mortality for all age groups (left) and by age group (right) across the states of the United States. Source: Vital Statistics.

Figure 4: Event Study: Probability of giving birth over time as a function of pneumonia mortality decline



This figure displays the marginal effects from logit coefficients and 95% confidence intervals around these effects 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.

Figure 5: Values of residualised pneumonia mortality across the United States.

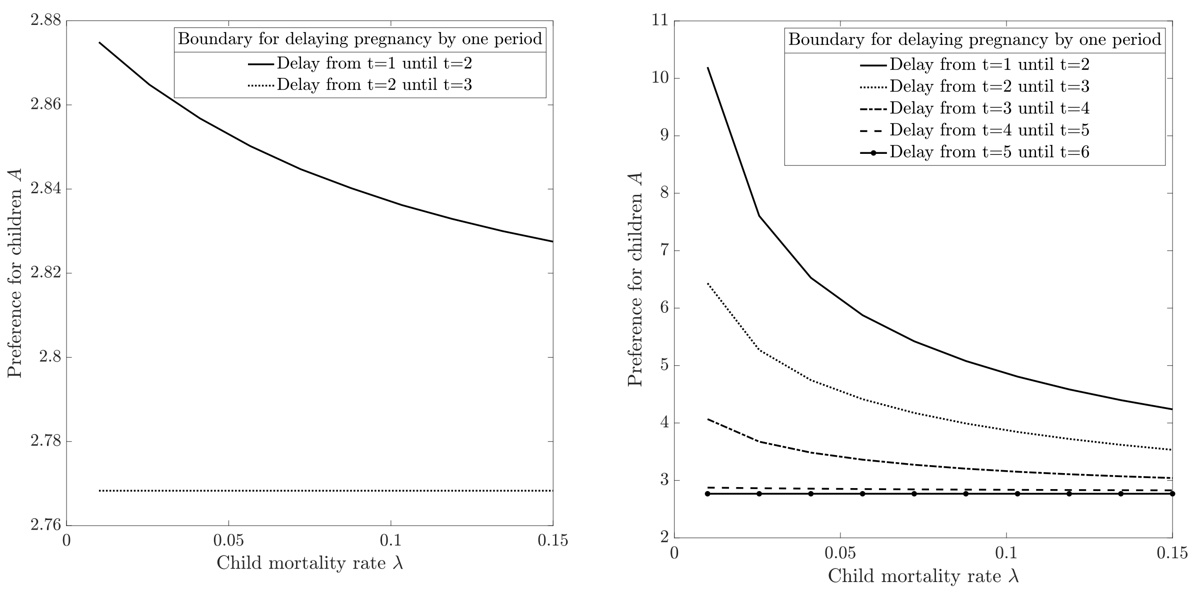


This figure shows values of pneumonia mortality across the United States that have been residualised in a state-level regression by all the control variables included in the main regressions.

Figure 6: Numerical example: The effect of a reduction in child mortality on fertility delay

(a) Model with 3 periods

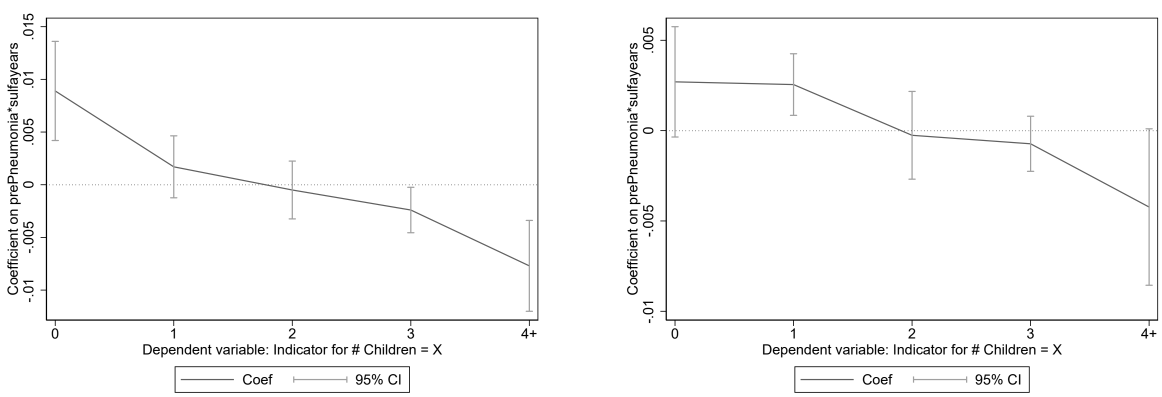
(b) Model with 6 periods



The figure plots the critical values of the preference parameter A, which measures the strength of the preference for children, as a function of the rate λ of child mortality. If A is above the top line in panel (a), it is optimal to get pregnant early at $t = 1$. If A is between the top and the bottom line, it is optimal to delay fertility $t = 2$. If A is below the bottom line, then no fertility (or, equivalently, delay until $t = 3$) is optimal. The region of delay becomes wider as λ declines. Similarly, panel (b) shows the optimal regions of pregnancy timing for a model with 6 periods. The parameter values are: $\tau_q = \tau_e = 2$, $u(n, e) = Ae^{\alpha}n^{1-\alpha}$, $\alpha = 0.9$, $Y = 1$, $p = 0.1$.

Figure 7: Estimated Effects of Pneumonia Mortality Decline on the Fertility Distribution

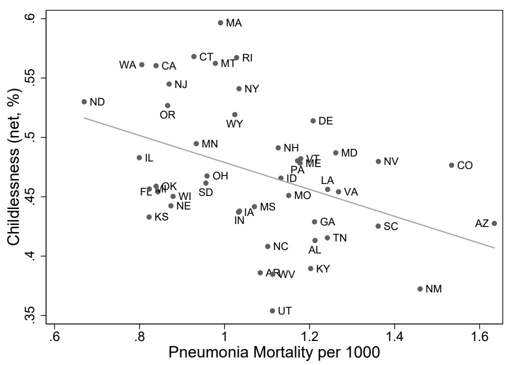
(a) Still Childbearing (b) Completed Fertility



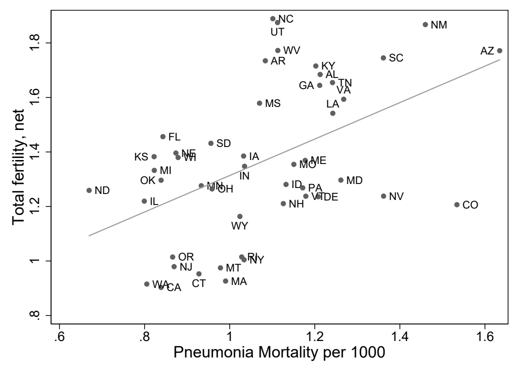
This figure displays the coefficients and 95% confidence intervals around coefficients on the variable $prePneumonia * sulfayears$ in a set of five separate OLS regressions, where the dependent variables in these regressions are indicator variables for having no children in the household, exactly one child, exactly two children, exactly three children, and four or more children, based on the net fertility measure.

Figure 8: Relationship Between Pneumonia Mortality and Fertility, Labor Supply and Marriage in 1930 across US States

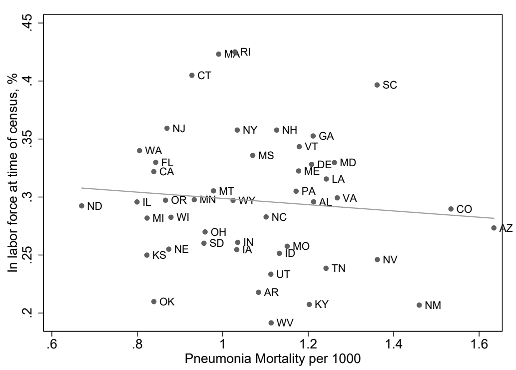
(a) Childlessness



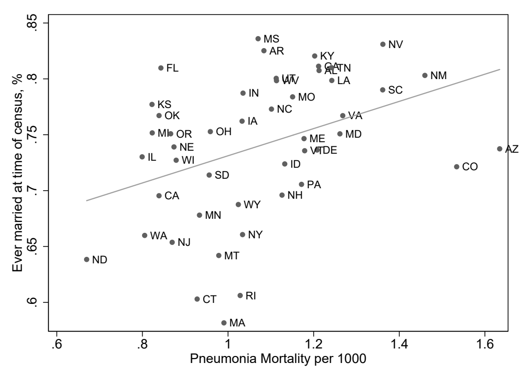
(b) Number of children



(c) Labor force participation



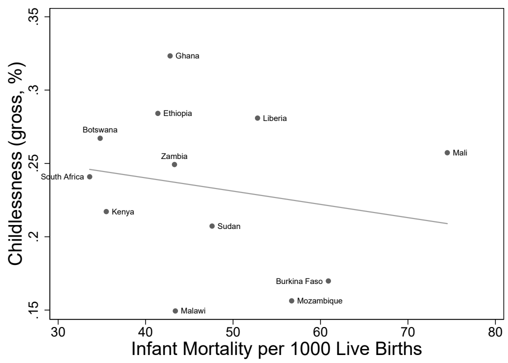
(d) Marital status



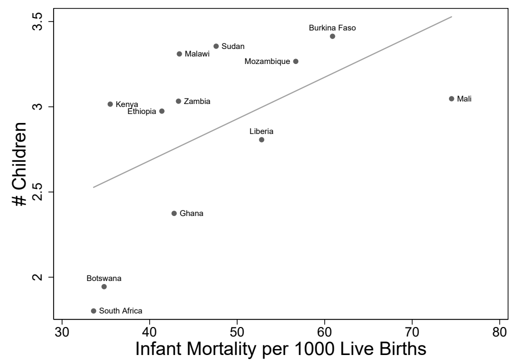
These figures show the relationship between the average state-level outcomes of women in the different US states in the 1930 census and the 1930-36 average pneumonia mortality rates in these states. Fertility outcomes are defined based on net fertility and other outcomes are as used in the main analysis. The sample includes all women aged 18-50 at the time of the census.

Figure 9: Relationship Between Infant Mortality and Fertility, Labor Supply and Marriage in 2015 across African Countries

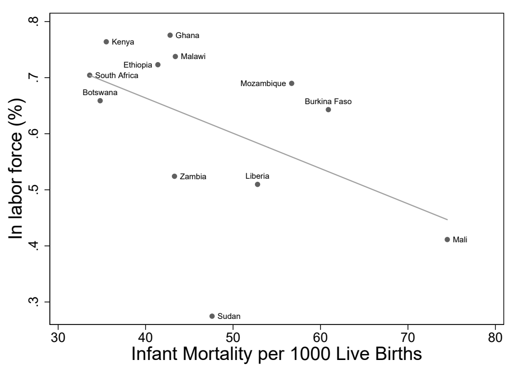
(a) Childlessness



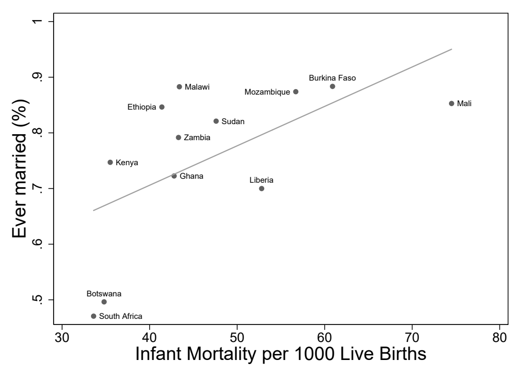
(b) Number of children



(c) Labor force participation



(d) Marital status



These figures show the relationship between the average country-level outcomes of women in different African countries and the infant mortality rate in these countries. The source of the fertility, labor market and marriage market data is the IPUMS International Database: all countries for which IPUMS data was available in 2000 or later are included, and all women aged 18-50 at the time of the census are included. We chose the census year closest to 2015 for each country. The mortality data are for 2015 and these data are sourced from UNESCO. Fertility is measured using the gross fertility measure (total births); childlessness is zero births.

Table 1: Probability of birth as a function of sulfa exposure

A: Main estimates

	(1) Birth	(2) Extensive Margin	(3) Intensive Margin
$prePneumonia * post1937$	-0.0233**	-0.0121**	-0.0095*
	(0.0100)	(0.0060)	(0.0051)
N	4499588	2894976	1604613
Mean	0.0865	0.0513	0.1491

B: Specification checks

	(1) w/out state controls	(2) w/yr interact	(3) 2SLS Under5s Birth	(4) State trend	(5) Division*year FE
$prePneumonia * post1937$	-0.0143***	-0.0100***		-0.0218*	-0.0288***
	(0.0033)	(0.0034)		(0.0127)	(0.0092)
$prePneumoniaU5 * post1937$			-0.0039*		
			(0.0023)		
N	4053834	4499588	4499792	4499588	4499588

The dependent variable is a dummy variable that equals one if the woman gave birth in that year, and zero otherwise. $prePneumonia * post1937$ is the average state-level pneumonia mortality rate between 1930-36, interacted with a dummy variable for the years 1937 and later. These are marginal effects from logistic (2SLS in Panel B, column (3)) 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. In Panel B, column (1) removes state-year varying controls, column (2) adds allows the state-year controls to have flexible coefficients by year, column (3) instruments the under 5 pneumonia mortality rate with the adult rate, column (4) replaces the census region-year fixed effects with state linear trends, and column (5) replaces them with census division*year fixed effects. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 2: Probability of birth as a function of sulfa exposure - robustness checks

	(1) WW2	(2) New Deal	(3) Dust Bowl	(4) Excl 1939+ Birth	(5) Mean Rev	(6) Occ struct	(7) Adult mort rates
$prePneumonia * post1937$	-0.0239**	-0.0247**	-0.0247**	-0.0256**	-0.0251**	-0.0235***	-0.0104
	(0.0105)	(0.0107)	(0.0106)	(0.0113)	(0.0103)	(0.0068)	(0.0074)
N	4053834	4499588	4053176	3417407	4499588	4499588	4499588

See notes to Table 1 for a description of the baseline regression. The robustness checks in each column is described in detail in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 3: Total net fertility (number of children) as a function of sulfa exposure

A: Still Childbearing at Census	(1)	(2)	(3)
	# Children	# Children | Children >0	Childless
$prePneumonia * sulfayears$	-0.0483***	-0.0345***	0.0089***
	(0.0127)	(0.0117)	(0.0024)
N	494437	313981	494437
Mean	1.6590	2.6118	0.3648

B: Completed Fertility at Census	(1)	(2)	(3)
	# Children	# Children | Children >0	Childless
$prePneumonia * sulfayears$	-0.0212**	-0.0218**	0.0027*
	(0.0103)	(0.0098)	(0.0016)
N	237603	171166	237603
Mean	1.9282	1.6760	0.2794

The dependent variables are (1) the total number of children, (2) the total number of children conditional on having at least one and (3) a dummy variable that equals one if the woman has zero children and zero otherwise. $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 4: Total gross fertility (number of children) as a function of sulfa exposure

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

These regressions are comparable to Panel B of Table 3, except that fertility outcomes are measured based on gross fertility (total number of live births). The sample contains women aged 40-50 at census. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 5: Labor market and marriage market outcomes as a function of sulfa exposure

A: Labor market outcomes

	(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)
N	727398	727398	517857	306280	727398
Mean	0.3510	0.3710	14.4093	1505.191	12.8097

B: Marriage market outcomes

	(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)
N	494437	727398	116632
Mean	0.7258	0.8499	21.1798

$prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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. Panel A: 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 (coefficient(standard error) 0.0775(0.0473) on $prePneumonia * sulfayears$), and the Duncan socioeconomic score (coefficient(standard error) 0.1512(0.0893)). Panel B: 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 women who have ever married. The cohorts in this panel 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. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table 6: The joint probability of labor force participation and childlessness as a function of sulfa exposure

	(1)	(2)	(3)	(4)
	In Labor Force		Not in Labor Force	
	Childless	Not childless	Childless	Not childless
$prePneumonia * sulfayears$	0.0056***	-0.0001	0.0013	-0.0068***
	(0.0017)	(0.0010)	(0.0012)	(0.0019)
N	727398	727398	727398	727398
Mean	0.205	0.166	0.140	0.489

The dependent variables are dummy variables for the four possible combinations of labor force participation status and childlessness status (based on net fertility) at the time of census enumeration i.e. each woman will fall into one of these four categories and will have a one for that dummy variable and a zero for the other three. These are OLS regressions with standard errors (in parentheses) clustered at the state of birth level. The sample contains women age 18-50 at census. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Appendix for Online Publication

Fertility and Labor Market Responses to Reductions in Mortality

Sonia Bhalotra

Atheendar Venkataramani

Selma Walther

Appendix

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A Data Description and Descriptive Statistics

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).

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 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.

Table A.1: Descriptive statistics: Individual characteristics in the hazard model dataset and state-level characteristics

Variable	Mean	Standard Deviation
State-level Characteristics		
$prePneumonia$	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	
Individual Characteristics in the Hazard Model Dataset		
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: In the top panel, the mean and standard deviation of state level characteristics (used as control variables in the regression estimates, where they are interacted with $post1937$ in the hazard sample and $sulfayears$ in the stock sample); In the bottom panel, outcome and control variables at the individual level in the hazard model dataset. 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 state characteristics are measured in 1930, except for the year of entering the birth and death registration systems, which is simply the year when that occurred.

Table A.2: Descriptive statistics: Individual characteristics in the stock model dataset

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 (0-1)	0.7258	0.4461	496783
Ever married (0-1)	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

Table A.3: Outcomes in Stock Model Dataset by Childlessness Status

Outcome	Childless women			Not childless women		
	Mean	St.dev.	N	Mean	St.dev.	N
Working	0.56	0.50	251652	0.24	0.43	478846
In labor force	0.59	0.49	251652	0.25	0.44	478846
H-W SEI	21.10	17.46	147691	11.76	16.33	372281
Personal income	2295.83	3328.75	88539	1185.31	2511.73	218839
Hours worked	21.06	21.05	251652	8.37	16.33	478846
Currently married	0.43	0.50	251652	0.92	0.27	478846
Ever married	0.51	0.50	251652	0.99	0.07	478846
Age at 1st marriage	21.01	5.38	50079	20.92	3.92	150534
Graduated from HS	0.25	0.44	251652	0.21	0.41	478846
Attended some college	0.12	0.32	251652	0.08	0.27	478846

This table shows the mean and standard deviation of outcomes by (net) childlessness status in the stock model dataset. All differences in means between childless and not childless women are statistically significant at the 1% level.

B Trend Breaks and Cross-State Convergence

We formally test convergence in mortality rates after the introduction of sulfa drugs in 1937. Table A.4 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, and shows that high mortality states pre-1937 had larger declines in mortality rates post-1937.

Table A.4: Trend breaks and test of convergence in pneumonia mortality rates

	(1) Δ Pneumonia	(2) Δ Log Pneumonia	(3) Pneumonia
$year * post1937$	-0.0999***	-0.1110***	
	(0.0059)	(0.0059)	
$post1937$	-0.1408***	-0.1287***	
	(0.0240)	(0.0238)	
$year$	0.0192***	0.0153***	
	(0.0042)	(0.0042)	
$prePneumonia * post1937$			-0.2940***
			(0.0459)
N	667	667	667
R^2	0.7573	0.7988	0.8603

These are OLS regressions (standard errors in parentheses) at the state-year level. The dependent variables are (1) the year-on-year change in *Pneumonia*, the state-year average mortality rate from pneumonia, (2) the year-on-year change in log *Pneumonia*, and (3) *Pneumonia*. *prePneumonia* is the 1930-36 average state-level mortality rate from pneumonia. All regressions also include state fixed effects, and regression (3) includes year 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.

C Additional Tables, Figures and Robustness Checks

C.1 Figures

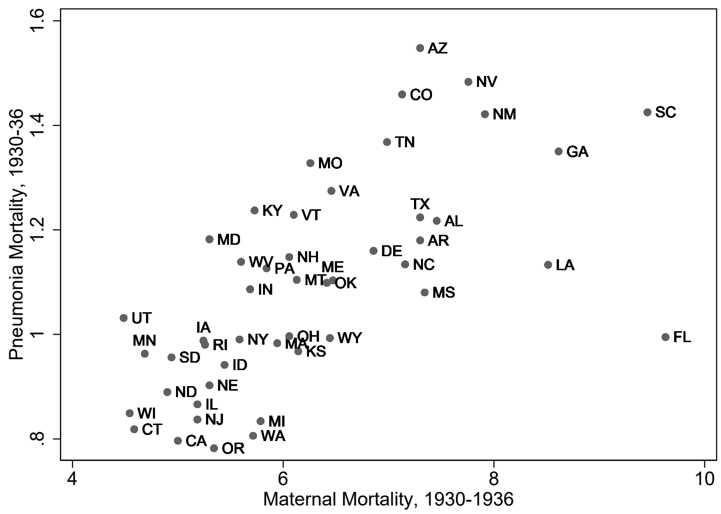
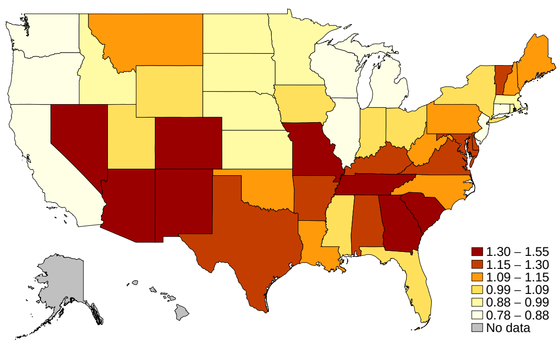
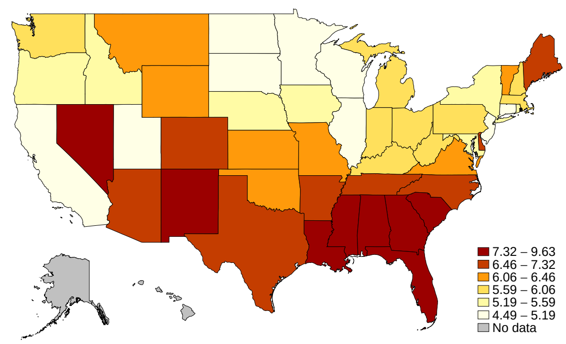


Figure A.1: Pneumonia and Maternal Mortality, United States, 1930-1936

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.



(a) Pneumonia Mortality



(b) Maternal Mortality

Figure A.2: Maps showing pneumonia and maternal mortality across the U.S.

This figure displays the average state-level mortality rates between 1930-36, with shading representing discrete categories of levels of mortality rates.

Figure A.3: Articles appearing in the New York Times when sulfa drugs arrived to the U.S.

NEW DRUG SAID TO AID IN PUERPERAL FEVER; British Doctors Report Prompt Drop in Temperature and Remission of Symptoms.

Special Cable to THE NEW YORK TIMES. ();
June 06, 1936,
, Section , Page 7, Column , words

PERMISSIONS

[DISPLAYING ABSTRACT]

LONDON, June 5. -- Experiments here with the new drug commonly called prontosil, a German aniline compound, in cases of childbed fever have given exceptional results.

YOUNG ROOSEVELT SAVED BY NEW DRUG; Doctor Used Prontylin in Fight on Streptococcus Infection of the Throat. CONDITION ONCE SERIOUS But Youth, in Boston Hospital, Gains Steadily -- Fiancee, Reassured, Leaves Bedside. YOUNG ROOSEVELT SAVED BY NEW DRUG

Special to THE NEW YORK TIMES. ();
December 17, 1936,
, Section , Page 1, Column , words

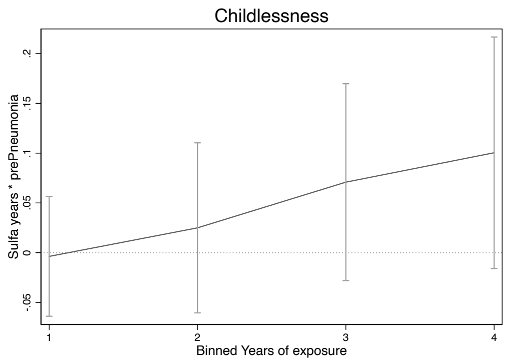
PERMISSIONS

[DISPLAYING ABSTRACT]

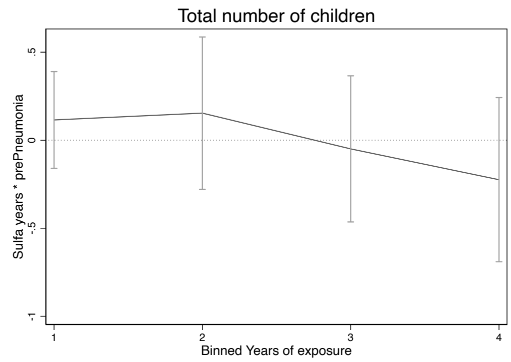
BOSTON, Dec. 16. -- Franklin D. Roosevelt Jr. faced death from a throat infection last week, it was disclosed tonight by his personal physician, Dr. George Loring Tobey Jr., at the Phillips House of the Massachusetts General Hospital, where young Roosevelt is a patient.

Figure A.4: Event Study: Stock fertility, labor market and marriage outcomes as a function of years of exposure bins

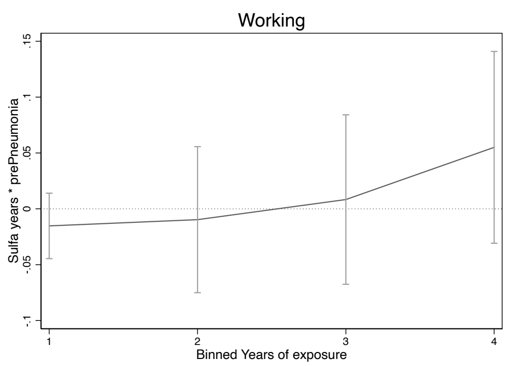
(a) Childlessness



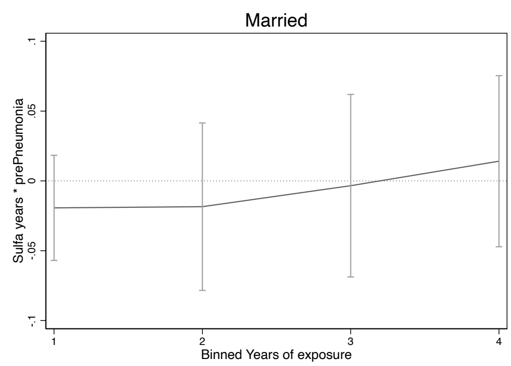
(b) Total Fertility



(c) Working



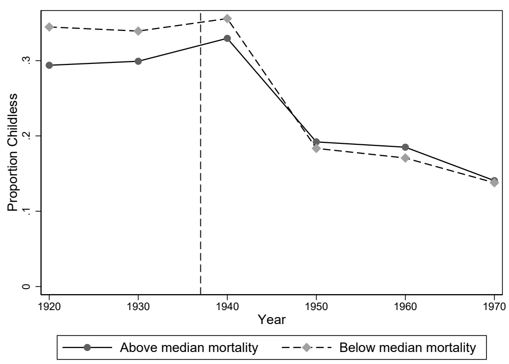
(d) Married



This figure displays the coefficients and 95% confidence intervals around these coefficients on the variable $prePneumonia * sulfayear_{b}ins$, where $sulfayear_{b}ins$ are five dummy variables for years of exposure to sulfa drugs. The bins are 0 years, 1-8 years, 9-16 years, 17-24 years and 25 years. The base (omitted) case is 0 years, the unexposed.

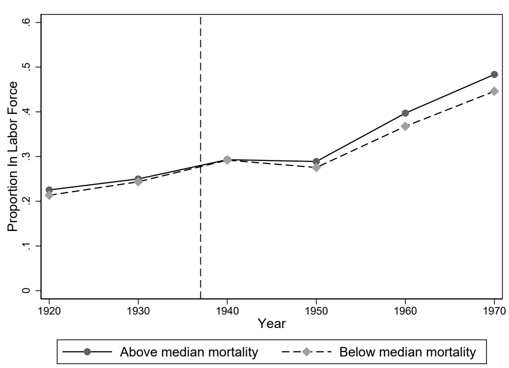
Figure A.5: Nonparametric Patterns of Outcomes by Above/Below Median Pneumonia Mortality

(a) Childlessness

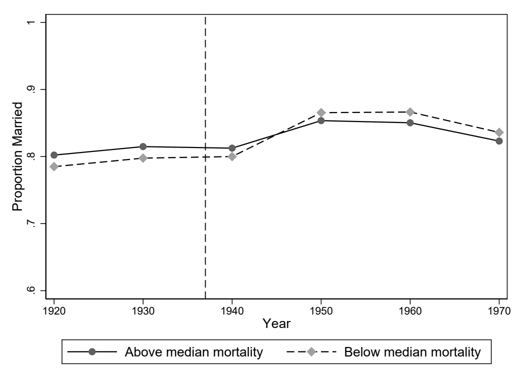


(b) Number of children

(c) Labor force participation



(d) Marital status



These figures show the average state-level outcomes of women in above and below median pneumonia mortality states in each census year, where this is defined based on average pneumonia mortality rates in 1930-36. Fertility outcomes are defined based on net fertility. The sample includes all women aged 30-40 at the time of the census. The dashed line shows the year 1937, when sulfa drugs were introduced to the US.

C.2 Tables

C.2.1 Under 5s 2SLS and Census region x Cohort FEs check

Table A.5: Fertility outcomes - Under 5s and census region x cohort FEs

	(1)	(2)	(3)	(4)	(5)	(6)
	Net Fertility			Gross Fertility		
A: Under 5s 2SLS	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumoniaU5 * sulfayears$	-0.0071***	-0.0050**	0.0013***	-0.0032	-0.0028	0.0003*
	(0.0027)	(0.0024)	(0.0004)	(0.0022)	(0.0019)	(0.0002)
N	494437	313981	494437	518933	421983	518933
B: Census region x Cohort FEs						
$prePneumonia * sulfayears$	-0.0309**	-0.0259***	0.0047*	-0.0057	-0.0057	0.0009
	(0.0125)	(0.0091)	(0.0025)	(0.0103)	(0.0101)	(0.0008)
N	494437	313981	494437	518933	421983	518933

See the notes to Table 3 for a description of variables and sampling. The robustness checks are described in Section 3.4. Panel A are 2SLS regressions where the under 5s rate is instrumented with all-age pneumonia mortality. For comparison, the coefficients (standard errors) on uninstrumented under-5s mortality in the respective regressions are: -0.0025** (0.0010) ; -0.0008 (0.0009) ; 0.0006*** (0.0002) ; -0.0009 (0.0009) ; -0.0008 (0.0008) ; 0.0001* (0.0001) . Panel B adds census region x cohort fixed effects to the baseline regressions in Table 3. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.6: Labor market outcomes - Under 5s and census region x cohort FEs

	(1)	(2)	(3)	(4)	(5)
A: Under 5s 2SLS	Working	In labor force	H-W SEI	Personal income	Hours worked
$prePneumoniaU5 * sulfayears$	0.0009***	0.0008**	0.0290**	1.0635	0.0365***
	(0.0003)	(0.0003)	(0.0125)	(2.2722)	(0.0136)
N	727398	727398	517857	306280	727398
B: Census region x Cohort FEs					
$prePneumonia * sulfayears$	0.0038***	0.0037***	0.1190**	18.9336	0.1630***
	(0.0007)	(0.0007)	(0.0555)	(16.7793)	(0.0300)
N	727398	727398	517857	306280	727398

See the notes to Table 5 for a description of variables and sampling. The robustness checks are described in Section 3.4. Panel A are 2SLS regressions where the under 5s rate is instrumented with all-age pneumonia mortality. For comparison, the coefficients (standard errors) on uninstrumented under-5s mortality in the respective regressions are: 0.0003*** (0.0001) ; 0.0003** (0.0001) ; 0.0163*** (0.0051) ; 0.2278 (0.9473) ; 0.0131*** (0.0043) . Panel B adds census region x cohort fixed effects to the baseline regressions in Table 5. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.7: Marriage market outcomes - Under 5s and census region x cohort checks

	(1)	(2)	(3)
A: Under 5s 2SLS	Currently married	Ever married	Age at 1st marriage
$prePneumoniaU5 * sulfayears$	-0.0003*	-0.0005**	0.0003
	(0.0002)	(0.0002)	(0.0035)
N	494437	727398	116632
B: Census region x Cohort FEs			
$prePneumonia * sulfayears$	-0.0007	-0.0020*	0.0196
	(0.0011)	(0.0010)	(0.0234)
N	494437	727398	116632

See the notes to Table 5 for a description of variables and sampling. The robustness checks are described in Section 3.4. Panel A are 2SLS regressions where the under 5s rate is instrumented with all-age pneumonia mortality. For comparison, the coefficients (standard errors) on uninstrumented under-5s mortality in the respective regressions are: -0.0001* (0.0001) ; -0.0002** (0.0001) ; 0.0009 (0.0021). Panel B adds census region x cohort fixed effects to the baseline regressions in Table 5. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.2 Sensitivity to controls

Table A.8: Sensitivity of main estimates to the inclusion of controls

	(1) # Children	(2) # Children | Children>0	(3) Childless	(4) Working	(5) Currently married
			A: With basic controls only		
$prePneumonia * sulfayears$	-0.0782***	-0.0863***	0.0060**	0.0018	-0.0007
	(0.0213)	(0.0203)	(0.0029)	(0.0017)	(0.0017)
N	496783	315548	496783	730498	496783
			B: Adding state-cohort controls		
$prePneumonia * sulfayears$	-0.0483***	-0.0345***	0.0089***	0.0058***	-0.0023*
	(0.0127)	(0.0117)	(0.0024)	(0.0017)	(0.0012)
N	494437	313981	494437	727398	494437

See the notes to Tables 3 and 5 for variable definitions and sampling. Regressions in Panel A include individual birth state, birth year, race and education fixed effects, and Panel B adds 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 $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.3 New Deal, WW2, Dust Bowl and Mean reversion checks

Table A.9: Fertility outcomes - New Deal, WW2, Dust Bowl and Mean reversion checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
A: New Deal	# Children	# Children $\mid$ Children>0	Childless	# Children	# Children $\mid$ 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)
N	494437	313981	494437	518933	421983	518933
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)
N	317789	230684	317789	518832	421898	518832
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)
N	444734	280263	444734	463435	376022	463435
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)
N	494437	313981	494437	518933	421983	518933

See notes to Table 3 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.10: Labor outcomes - New Deal, WW2, Dust Bowl and Mean reversion checks

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
A: New Deal					
$prePneumonia * sulfayears$	0.0055***	0.0051***	0.3308**	29.8135***	0.2295***
	(0.0016)	(0.0017)	(0.1472)	(10.2308)	(0.0637)
N	727398	727398	247015	306280	727398
B: WW2					
$prePneumonia * sulfayears$	0.0070***	0.0066***	0.3695***	14.5503	0.2931***
	(0.0017)	(0.0017)	(0.1293)	(14.5801)	(0.0533)
N	517746	517746	246952	306209	517746
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)
N	654187	654187	223428	274954	654187
D: Mean reversion					
$prePneumonia * sulfayears$	0.0059***	0.0053***			
	(0.0017)	(0.0017)			
N	727398	727398			

See notes to Table 5 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.11: Marriage market outcomes - New Deal, WW2, Dust Bowl and Mean reversion checks

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

See notes to Table 5 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.4 Horse race model

Table A.12: Fertility outcomes - comparing child and adult pneumonia mortality

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumoniaU5 * sulfayears$	-0.0021*	-0.0004	0.0006***	-0.0005	-0.0006	0.0001
	(0.0012)	(0.0009)	(0.0002)	(0.0011)	(0.0010)	(0.0001)
$prePneumonia25to34 * sulfayears$	-0.0272	-0.0226	0.0026	-0.0264	-0.0140	0.0045*
	(0.0402)	(0.0323)	(0.0060)	(0.0334)	(0.0314)	(0.0022)
N	494437	313981	494437	518933	421983	518933

$prePneumoniaU5 * sulfayears$ and $prePneumonia25to34 * sulfayears$ are the average state-level pneumonia mortality rates between 1930-36 among under 5s and among 25-34 year olds respectively, interacted with the number of fertile years that a woman was exposed to sulfa drugs. See notes to Table 3 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.13: Labor market outcomes - comparing child and adult pneumonia mortality

	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal Income	(5) Hours worked
$prePneumoniaU5 * sulfayears$	0.0004***	0.0004***	0.0150**	0.0753	0.0142***
magnitude of effect	2.38pp	2.38pp	0.89	4.48	0.84
	(0.0001)	(0.0001)	(0.0062)	(1.0643)	(0.0046)
$prePneumonia25to34 * sulfayears$	-0.0027	-0.0032	0.0895	9.7064	-0.0741
magnitude of effect	-0.64pp	-0.76pp	0.21	23.09	-0.18
	(0.0037)	(0.0039)	(0.1567)	(32.6101)	0.1474)
N	727398	727398	517857	306280	727398
Mean	0.3510	0.3710	14.4093	1505.191	12.8097

The magnitudes of effects are calculated as the coefficient x inter-quartile range of mortality variable x average sulfa years in this sample (18.3). $prePneumoniaU5 * sulfayears$ and $prePneumonia25to34 * sulfayears$ are the average state-level pneumonia mortality rates between 1930-36 among under 5s and among 25-34 year olds respectively, interacted with the number of fertile years that a woman was exposed to sulfa drugs. See notes to Table 5 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.14: Marriage outcomes- comparing child and adult pneumonia mortality

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
$prePneumoniaU5 * sulfayears$	-0.0002	-0.0002**	0.0014
	(0.0001)	(0.0001)	(0.0021)
$prePneumonia25to34 * sulfayears$	0.0001	-0.0016	-0.0315
	(0.0031)	(0.0021)	(0.0551)
N	494437	727398	116632

$prePneumoniaU5 * sulfayears$ and $prePneumonia25to34 * sulfayears$ are the average state-level pneumonia mortality rates between 1930-36 among under 5s and among 25-34 year olds respectively, interacted with the number of fertile years that a woman was exposed to sulfa drugs. See notes to Table 5 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.5 Education outcomes

Table A.15: 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)
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* is the average state-level pneumonia mortality rate between 1930-36. 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 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 $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.6 Occupation structure and Adult mortality rates checks

Table A.16: Fertility outcomes - occupation structure and adult mortality checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
A: Occupation structure	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
$prePneumonia * sulfayears$	-0.0366**	-0.0267**	0.0065**	-0.0046	-0.0019	0.0016*
	(0.0139)	(0.0126)	(0.0026)	(0.0110)	(0.0104)	(0.0009)
N	494316	313910	494316	518832	421898	518832
B: Adult mortality rates						
$prePneumonia * sulfayears$	-0.0597***	-0.0427***	0.0104***	-0.0206*	-0.0182	0.0024**
	(0.0129)	(0.0103)	(0.0023)	(0.0113)	(0.0108)	(0.0012)
N	494316	313910	494316	518832	421898	518832

See notes to Table 3 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.17: Labor market outcomes - occupation structure and adult mortality checks

A: Occupation structure	(1) Working	(2) In labor force	(3) H-W SEI	(4) Personal income	(5) Hours worked
$prePneumonia * sulfayears$	0.0039**	0.0035**	0.1976**	2.3577	0.1673***
	(0.0015)	(0.0016)	(0.0841)	(15.2636)	(0.0570)
N	727239	727239	517746	306209	727239
B: Adult mortality rates					
$prePneumonia * sulfayears$	0.0044***	0.0039**	0.1594	0.7607	0.1906***
	(0.0014)	(0.0015)	(0.0972)	(16.7361)	(0.0625)
N	727239	727239	517746	306209	727239

See notes to Table 5 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.18: Marriage market outcomes - occupation structure and adult mortality checks

	(1)	(2)	(3)
A: Occupation structure	Currently married	Ever married	Age at 1st marriage
$prePneumonia * sulfayears$	-0.0018	-0.0032**	0.0086
	(0.0013)	(0.0014)	(0.0240)
N	494316	727239	116588
B: Adult mortality rates			
$prePneumonia * sulfayears$	-0.0029**	-0.0033**	0.0143
	(0.0013)	(0.0013)	(0.0269)
N	494316	727239	116588

See notes to Table 5 for definitions of outcomes. The robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.7 Placebo test, Age of conception and Mountain/South states checks

Table A.19: Probability of birth - Age of conception and Mountain/South states checks

	(1) Age<10	(2) Conception year	(3) Excl mountain Birth	(4) Excl deep south
$prePneumonia * post1937$	-0.0161**	-0.0263***	-0.0299**	-0.0065
	(0.0078)	(0.0101)	(0.0129)	(0.0058)
N	2810044	4035785	4414170	3941672

See notes to Table 1 for a description of the baseline regression. The robustness checks in each column is described in detail in Section 3.4. For column (1), only potential births between 1940-43 are included from the 1950 census. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.20: Fertility outcomes - Placebo test, Age of conception and Mountain/South states checks

	(1)	(2)	(3)	(4)	(5)	(6)
	Net Fertility			Gross Fertility		
A: 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)			
N	61918	42447	61918			
B: Excl. 10 and older						
$prePneumonia * sulfayears$	-0.0227**	-0.0174**	0.0074**			
	(0.0085)	(0.0078)	(0.0029)			
N	494437	236499	494437			
C: Excl Mountain West						
$prePneumonia * sulfayears$	-0.0.0417**	-0.0329**	0.0072***	-0.0225	-0.0196	0.0016
	(0.0155)	(0.0138)	(0.0026)	(0.0156)	(0.0142)	(0.0011)
N	483852	306588	483852	509656	413931	509656
D: Excl Deep South						
$prePneumonia * sulfayears$	-0.0395**	-0.0311**	0.0077**	-0.0104	-0.0107	0.0016
	(0.0157)	(0.0134)	(0.0030)	(0.0130)	(0.0121)	(0.0011)
N	435017	274490	435017	464042	376270	464042

The variables and specification are described in the notes to Table 3 and the robustness checks are described in Section 3.4. For Panel A, our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1897, with outcomes drawn from the 1910-1930 censuses. For other panels, our dataset is a cross-section of outcomes of women aged 6-44 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. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.21: Labor market outcomes - Placebo test, Age of conception and Mountain/South states checks

	(1)	(2)	(3)	(4)	(5)
A: Placebo	Working	In labor force	H-W SEI	Personal income	Hours worked
$prePneumonia * sulfayears$	0.0102	0.0211			
	(0.0388)	(0.0435)			
N	54852	54842			
B: Excl Mountain West					
$prePneumonia * sulfayears$	0.0065***	0.0062***	0.2163***	35.0206***	0.2744***
	(0.0014)	(0.0014)	(0.0538)	(12.7874)	(0.0460)
N	712693	712693	507062	300013	712693
C: Excl Deep South					
$prePneumonia * sulfayears$	0.0056***	0.0052***	0.1668	-15.0912	0.2047***
	(0.0018)	(0.0018)	(0.1032)	(16.5964)	(0.0683)
N	642475	642475	459065	274429	642475

The variables and specification are described in the notes to Table 5 and the robustness checks are described in Section 3.4. For Panel A, our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1897, with outcomes drawn from the 1910-1930 censuses. For other panels, our dataset is a cross-section of outcomes of women aged 6-44 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. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.22: Marriage market outcomes - Placebo test, Age of conception and Mountain/South states checks

A: 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)
N	61918	135524	7625
B: Excl Mountain West			
$prePneumonia * sulfayears$	-0.0022	-0.0028**	-0.0274
	(0.0014)	(0.0011)	(0.0177)
N	483852	904574	302364
C: Excl Deep South			
$prePneumonia * sulfayears$	-0.0023	-0.0022	-0.0187
	(0.0014)	(0.0013)	(0.0152)
N	435017	817174	274187

The variables and specification are described in the notes to Table 5 and the robustness checks are described in Section 3.4. For Panel A, our dataset is a cross-section of fertility outcomes of women aged 6-44 in 1897, with outcomes drawn from the 1910-1930 censuses. For other panels, our dataset is a cross-section of outcomes of women aged 6-44 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. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.8 Migration checks

Table A.23: Fertility outcomes - Migration checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
	# Children	# Children | Children>0	Childless	# Children	# Children | Children>0	Childless
A: Incl Migrants						
$prePneumonia * sulfayears$	-0.0253***	-0.0159*	0.0052***	-0.0145	-0.0145	0.0012
	(0.0083)	(0.0087)	(0.0016)	(0.0105)	(0.0095)	(0.0007)
N	828558	548080	828558	955497	780370	955497
B: 2SLS Migrants						
$prePneumonia * sulfayears$	-0.0408***	-0.0300**	0.0080***	-0.0262	-0.0255	0.0023*
	(0.0127)	(0.0141)	(0.0024)	(0.0171)	(0.0158)	(0.0012)
N	809989	533144	809989	880951	718855	880951

The dependent variables are described in the notes to Table 3 and the robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.24: Labor market outcomes - Migration checks

	(1)	(2)	(3)	(4)	(5)
A: Incl Migrants					
$prePneumonia * sulfayears$	0.0033***	0.0030**	0.1496**	-21.0175	0.1214***
	(0.0012)	(0.0012)	(0.0693)	(16.5351)	(0.0449)
N	1264807	1264807	980312	673706	1264807
B: 2SLS Migrants					
$prePneumonia * sulfayears$	0.0045**	0.0040**	0.2290**	-34.3413	0.1612**
	(0.0018)	(0.0019)	(0.1049)	(23.9145)	(0.0676)
N	1222295	1222295	939159	633897	1222295

T The dependent variables are described in the notes to Table 5 and the robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.25: Marriage market outcomes - Migration checks

	(1)	(2)	(3)
A: Incl Migrants			
$prePneumonia * sulfayears$	-0.0004	-0.0009*	-0.0132
	(0.0009)	(0.0005)	(0.0132)
N	828558	1592612	595742
B: 2SLS Migrants			
$prePneumonia * sulfayears$	-0.0008	-0.0017**	-0.0214
	(0.0014)	(0.0008)	(0.0219)
N	809989	1527667	587994

The dependent variables are described in the notes to Table 5 and the robustness checks are described in Section 3.4. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.2.9 Linear pre-trends check and Binary DiD

Table A.26: Probability of birth - Migration checks and Migration as a function of sulfa exposure

	(1) Birth - Migrants	(2) Birth - 2SLS Migrants	(3) Pr(in migrant sample)	(4) Migrated between 1935-40
$prePneumonia * post1937$	-0.0219**	-0.0386*		
	(0.0102)	(0.0211)		
$prePneumonia * sulfayears$			0.0020	-0.0045
			(0.0031)	(0.0072)
N	6349665	6319430	1122827	56722

columns 1 and 2: See notes to Table 1 for a description of the baseline regression. The robustness checks in each column is described in detail in Section 3.4. columns 3 and 4: The dependent variable in column 3 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 4 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. The cohorts in this sample were born in the years 1893-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses (column 3) and the 1940 US decennial population census (column 4). 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 $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

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

	(1)	(2)
	Birth	
$AboveMedPneu * trend$	-0.0001	-0.0001
	(0.0002)	(0.0003)
N	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. 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. Regression (1) includes while Regression (2) omits a dummy variable for above median average $preMMR$ in 1930-1936, interacted with *trend*. Both regressions include individual birth state, birth year, race, and education, child birth order, time since last birth and year and census region*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 A.28: Binary DiD estimates of the effect of sulfa exposure on fertility, labor and marriage market outcomes

A: 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)
N	279899	182808	279899	163036	137303	163036

B: Labor market	(1)	(2)	(3)	(4)	(5)
	Working	In labor force	H-W SEI	Personal inc.	Hours worked
$prePneumonia * treated$	0.1339**	2.3366***	-3303.3419***	-0.0440	4.5693
	(0.0588)	(0.0597)	(0.5128)	(64.3696)	(2.4842)
N	279899	279899	245681	168344	279899

C: Marriage market	(1)	(2)	(3)
	Curr. married	Ever marr.	Age at 1st marr.
$prePneumonia * treated$	-0.0440	-0.0998**	0.3031
	(0.0398)	(0.0376)	(0.6092)
N	279899	279899	86390

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. Panel A: The dataset is a cross-section of fertility outcomes of women aged 6-15 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. Panel B: The dataset is a cross-section of labor outcomes of women aged 6-15 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. Panel C: The dataset is a cross-section of marriage outcomes of women aged 6-15 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. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

C.3 Additional Robustness Checks

C.3.1 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 Panel A of Table A.29. All the results are statistically significant and the magnitudes are comparable to those in the main text. Panel B 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 A.30, 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.

C.3.2 Outliers

Next, we reestimate the main results but excluding New Mexico, which was shown to be an outlier state in Figure 8. The hazard model results are in column (1) of Table A.32, while the stock model results are in Panel A of Tables A.33 (fertility) and A.34-A.35 (labor and marriage markets). The exclusion of New Mexico does not change the results in a substantive way.

C.3.3 OLS and Woman Fixed Effects

In order to verify that our results are similar in a simpler estimation model, we estimate the hazard model using OLS (Table A.32). 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 1, but they are less precisely estimated.

C.3.4 Multiple hypothesis testing

Finally, in Panel C of Table A.34, we adjust the standard errors from the main results (Table 5) 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 A.29: Fertility as a function of sulfa exposure: Alternative samples

	(1) # Children	(2) # Children | Children >0	(3) Childless
A: Net fertility, 18-36 year old women			
$prePneumonia * sulfayears$	-0.0408***	-0.0297**	0.0089***
	(0.0119)	(0.0130)	(0.0029)
N	393720	234416	393720
B: Gross fertility, 18-40 year old women			
$prePneumonia * sulfayears$	-0.0288	-0.0275	0.0029
	(0.0201)	(0.0205)	(0.0029)
N	170537	138760	170537

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). $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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. 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 $sulfayears$. Panel A: Definitions based on net fertility. The dataset is a cross-section of fertility outcomes of women aged 6-44 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 were born in the years 1904-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. Panel B: Definitions based on gross fertility. The dataset is a cross-section of fertility outcomes of women aged 6-44 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 were born in the years 1900-1931 and are drawn from the 1940, 1950, 1960 and 1970 US decennial population censuses. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.30: 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
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)
N	494437	494437	317867	152468	494437
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)
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. $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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 6-44 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 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 $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.31: Marriage market outcomes as a function of sulfa exposure: Alternative samples

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
A: 18 to 50 year old women			
$prePneumonia * sulfayears$	-0.0013	-0.0032**	-0.0170
	(0.0011)	(0.0012)	(0.0152)
N	727398	727398	181562
B: 18 to 60 year old women			
$prePneumonia * sulfayears$	-0.0005	-0.0025**	-0.0294*
	(0.0011)	(0.0011)	(0.0155)
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. $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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 6-44 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 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 $sulfayears$. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.32: Probability of birth as a function of sulfa exposure: Additional robustness checks

	(1) - Excl. New Mexico	(2) - Pneu only Birth	(3) - OLS	(4) - OLS+WFE
$prePneumonia * post1937$	-0.0241**	-0.0158*	-0.0253*	-0.0185
	(0.0104)	(0.0084)	(0.0134)	(0.0209)
N	4485763	4499588	4499792	4499792

See notes to Table 1 for a description of the baseline regression. column (4) adds woman fixed effects. columns (3) and (4) are estimated using OLS. * denotes p-value<0.1, ** denotes p-value<0.05 and *** denotes p-value<0.01.

Table A.33: Net and gross fertility as a function of sulfa exposure: Additional robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
		Net Fertility			Gross Fertility	
A: 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)
N	492776	312786	492776	517651	420859	517651
B: 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

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). $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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 6-44 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 maternal mortality (not in Panel B), 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 A.34: Labor market outcomes as a function of sulfa exposure: Additional robustness checks

A: 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)
N	725118	725118	516184	305575	725118
B: 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
C: Mult. Hypothesis					
$prePneumonia * sulfayears$	0.0058***	0.0055***	0.1991**	7.7366	0.2421***
	(0.0018)	(0.0019)	(0.0857)	(54.9474)	(0.0675)
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. $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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 (and adjusted for multiple hypothesis testing in Panel C). Our dataset is a cross-section of labor outcomes of women aged 6-44 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 maternal mortality (not in Panel B), 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 A.35: Marriage market outcomes as a function of sulfa exposure: Additional robustness checks

	(1) Currently married	(2) Ever married	(3) Age at 1st marriage
A: Excl. New Mexico			
$prePneumonia * sulfayears$	-0.0023*	-0.0032**	0.0010
	(0.0012)	(0.0012)	(0.0242)
N	492776	725118	116261
B: Pneu only			
$prePneumonia * sulfayears$	-0.0008	-0.0018	0.0154
	(0.0013)	(0.0013)	(0.0203)
N	494437	727398	116632
C: Mult. Hypothesis			
$prePneumonia * sulfayears$	-0.0023	-0.0032**	0.0021
	(0.0016)	(0.0014)	(0.0652)
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. $prePneumonia * sulfayears$ is the average state-level pneumonia mortality rate between 1930-36, 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 (and adjusted for multiple hypothesis testing in Panel C). Our dataset is a cross-section of labor and marriage outcomes of women aged 6-44 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 maternal mortality (not in Panel B), 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 A Model of Fertility and Labor Market Choices

D.1 Proofs

Proof of Proposition 1

Proof. We solve the woman's problem by backward induction. At $t = 2$, if the woman has a surviving child at this date, then Assumption (4) implies that her optimal strategy is not to get pregnant ($a_2 = 0$). Her continuation utility in this case (ignoring her exogenous endowment, which does not affect optimal decisions) is simply $V_1(A)$. If she does not have a surviving child, her continuation value is

$$\max\{y_2, (1-\lambda) V_1(A)\},$$

since she can choose to either get pregnant or work for wage y_2. At $t = 1$, it is therefore optimal to get pregnant ($a_1 = 1$) if and only if

$$\lambda \max\{0, (1-\lambda) V_1(A)\} + (1-\lambda) V_1(A) \quad \geq \quad E[\max\{y_2, (1-\lambda) V_1(A)\} | a_1 = 0] \tag{11}$$

For any $A < \underline{A}$, where $\underline{A}$ is defined implicitly by $V_1(\underline{A}) = 0$, the inequality in (11) cannot hold because

$$(1-\lambda) V_1(A) < 0 \leq E[y_2 | a_1 = 0].$$

This establishes point 1 in the proposition. Next, for any $A \geq \underline{A}$, we can use the facts that $y_2 = Y$ with probability p, and $y_2 = 0$ otherwise, to reduce (11) to

$$(1+\lambda)(1-\lambda) V_1(A) \geq p \max\{Y, (1-\lambda) V_1(A)\} + (1-p)(1-\lambda) V_1(A). \tag{12}$$

As a function of A, the left-hand side has slope $(1-\lambda^2) V_1'(A)$, while the right-hand side has slope less than $(1-\lambda) V_1'(A)$. Since $V_1(0) < 0$ and $\lim_{A\to\infty} V_1(A) = \infty$, there must be a unique solution $A = \bar{A}(\lambda)$ such that (12) holds if and only if $A \geq \bar{A}(\lambda)$. We conjecture and verify that this solution satisfies $0 < (1-\lambda) V_1\left(\bar{A}(\lambda)\right) < Y$. If this conjecture is correct, then part 2 of the proposition follows, because it implies that pregnancy at date 2 after working at date 1 is optimal if and only if the woman has not been promoted. Solving (12) then yields $V_1\left(\bar{A}(\lambda)\right) = \frac{pY}{(p+\lambda)(1-\lambda)}$, which clearly satisfies our conjecture and also establishes part 3 of the proposition. ■

Proof of Proposition 2

Proof. This result follows by evaluating the probability of childlessness for each of the three cases governing the optimal policy in Proposition 1. In case 1 (no fertility), the probability is clearly 1. In case 2 (delayed fertility), the woman is childless either if i) she is promoted, which occurs with probability p, or if ii) she is not promoted, gets pregnant and fails to have a surviving child, which occurs with probability $(1-p)\lambda$. Hence, the probability of childlessness in case 2 is $p + (1-p)\lambda$. In case 3 (early fertility), the woman is childless if both she gets pregnant twice but has no surviving child, which occurs with probability λ^2. Combining these probabilities with the probability mass of women in each case, and summing across cases, yields equation (7). ■

Proof of Proposition 3

Proof. The first point in the proposition follows by observing that $\underline{A}$ is the implicit solution on $V_1(\underline{A}) = 0$. Since the definition of $V_1(A)$ in (15) does depend on λ, it follows that $\underline{A}$ is also independent of λ. aFor the second point, the definition of $\bar{A}(\lambda)$ can be alternatively written as

$$pY = (p+\lambda)(1-\lambda)V_1\left(\bar{A}(\lambda)\right).$$

By the implicit function theorem, we have

$$0 = (1-p-2\lambda)V_1\left(\bar{A}(\lambda)\right) + (p+\lambda)(1-\lambda)V_1'\left(\bar{A}(\lambda)\right)\frac{\partial\bar{A}(\lambda)}{\partial\lambda},$$

Applying the envelope theorem to (15), we obtain $V_1'\left(\bar{A}(\lambda)\right) = u(1, e_1^\star) > 0$, where $e_1^\star$ denotes the optimal choice of e in problem (15) when $n = 1$. Hence, we find that $\frac{\partial\bar{A}(\lambda)}{\partial\lambda} < 0$ if and only if $1 - p - 2\lambda > 0$, which is equivalent to (8). ■

Proof of Proposition 4

Proof. This result follows by totally differentiating Equation (7) with respect to λ. ■

D.2 Price effects

In this appendix, we consider comparative statics with respect to child health shocks that affect both the rate λ of child mortality and the prices $\boldsymbol{\tau} = (\tau_q, \tau_e)$ of child quantity and quality, respectively. In turn, prices affect the surplus associated with having n children. We make this dependence explicit:

$$V_n(A;\boldsymbol{\tau}) = \max_e \{A \cdot u(n,e) - n(\tau_q + \tau_e e)\}$$

Moreover, making the dependence of suprlus on prices and λ explicit, the value of $A = \bar{A}(\lambda;\boldsymbol{\tau})$, at which women are indifferent between early and delayer fertility is now implicitly defined by

$$(p+\lambda)(1-\lambda)V_1\left(\bar{A}(\lambda;\boldsymbol{\tau});\boldsymbol{\tau}\right) = pY \tag{13}$$

As in the paper, we focus on conditions under which $\frac{d\bar{A}}{d\lambda} < 0$, so that a positive shock (a decline in λ) encourages delay. To analyze indirect effects of health shocks through prices, we model prices as a function $\boldsymbol{\tau}(\lambda)$ and consider changes in λ. We assume that this function is twice differentiable for all $\lambda \in [0,1]$. We assume that surplus continues to satisfy our parametric assumption that $V_1(A;\boldsymbol{\tau}(\lambda)) < V_2(A;\boldsymbol{\tau}(\lambda))$ for all $\lambda \in [0,1]$.

The total derivative of interest is now given by[45]

$$\frac{d\bar{A}(\lambda;\boldsymbol{\tau}(\lambda))}{d\lambda}=\frac{\partial\bar{A}(\lambda)}{\partial\lambda}+\frac{\partial\bar{A}(\lambda)}{\partial\boldsymbol{\tau}}\cdot\frac{\partial\boldsymbol{\tau}(\lambda)}{\partial\lambda}.$$

We derive a sufficient condition under which the behavioral effect encourages delay in response to a decline in λ:

Proposition 5 *There exists a threshold $K>0$ such that $\lambda\leq\frac{K-p}{2}$ implies $\frac{d\bar{A}(\lambda;\boldsymbol{\tau}(\lambda))}{d\lambda}<0$.*

Intuitively, a decline in λ has two distinct effects on the marginal cost of delay. First, it reduces the marginal probabilityof childlessness due to delay, as long as λ is not too large. Second, there is a counterveiling effect if the decline in λ lowers prices, which raises the surplus from having children and encourages early fertility. However, the price effect affects the marginal cost of delay in proportion to the marginal probability $(p+\lambda)(1-\lambda)$, and is therefore dominated when p and λ is sufficiently small.

Proof of Proposition 5

Proof. Taking logs of Equation (13) and using the implicit function theorem, we have

$$\frac{\partial\log V_1\left(\bar{A}(\lambda;\boldsymbol{\tau}(\lambda));\boldsymbol{\tau}(\lambda)\right)}{\partial A}\frac{d\bar{A}(\lambda;\boldsymbol{\tau}(\lambda))}{d\lambda}=-\left[\frac{1-p-2\lambda}{(p+\lambda)(1-\lambda)}+J(\lambda)\right]\tag{14}$$

with

$$J(\lambda)=\frac{\partial\log V_1\left(\bar{A}(\lambda;\boldsymbol{\tau}(\lambda));\boldsymbol{\tau}(\lambda)\right)}{\partial\boldsymbol{\tau}}\cdot\frac{\partial\boldsymbol{\tau}(\lambda)}{\partial\lambda}.$$

We have assumed that women's utility and $\boldsymbol{\tau}(\lambda)$ are twice differentiable, so that the function $J(\lambda)$ is continuous in $\lambda\in[0,1]$. We can therefore define the lower bound $B=\inf_{\lambda\in[0,1]}J(\lambda)$, which is independent of p and λ. Now assume that $p+2\lambda\leq K$, so that we have

$$\frac{1-p-2\lambda}{(p+\lambda)(1-\lambda)}+J(\lambda)\geq\frac{1-K}{K}+B.$$

We can find a sufficiently small $K>0$ such that the right-hand side of this expression is strictly positive. Combining with Equation (14), and noting that $\frac{\partial\log V_1}{\partial A}>0$, we find that $\frac{d\bar{A}}{d\lambda}<0$ for sufficiently small K, which completes the proof. ■

D.3 General dynamics

This appendix presents a version of our model with general dynamics. There is a unit measure of women whose life cycles consists of T periods $t\in\{1,2,...,T\}$. Women's utility is $U=A\cdot u(n,e)+c$, as in the baseline model.

[45] We write $\frac{\partial\bar{A}(\lambda)}{\partial\boldsymbol{\tau}}$ for the derivative with respect to both prices, and $\frac{\partial\boldsymbol{\tau}(\lambda)}{\partial\lambda}$ for the vector of derivatives of both prices with respect to λ. Both are vectors with two elements, and we denote their inner product by $\frac{\partial\bar{A}(\lambda)}{\partial\boldsymbol{\tau}}\cdot\frac{\partial\boldsymbol{\tau}(\lambda)}{\partial\lambda}$.

At dates $t = 1, 2, ..., T-1$, each woman chooses whether to get pregnant, denoted $a_t = 1$, or to work, denoted $a_t = 0$. If she chooses $a_t = 1$, she has a surviving child with probability $(1-\lambda)$, where λ is the rate of child mortality. If she works, she earns wages y_t per period. Her wages are intialized at $y_1 = 0$. If she works at date t and earns $y_t = 0$, there are two possibilities. With probability p, she is promoted, in which case her wage rises to $y_s = Y > 0$ for all subsequent periods $s > t$ until her first pregnancy. With probability $1-p$, she is not promoted, and her wage remains at $y_{t+1} = 0$ for the next period. If she woman gets pregnant at t, then her wage falls to $y_s = 0$ for all periods $s \geq t$. This is a generalization of the stochastic process in our baseline model, which captures the "job then family" pattern of the sulfa drug era.

At the final date $t = T$, the woman's fertility is complete, and she takes as given the final number of her surviving children $n \in \{0, 1, ..., T-1\}$. As in the baseline model, we define the surplus she obtains at this date as

$$V_n = \max_e \{A \cdot u(n, e) - n(\tau_q + \tau_e e)\}. \tag{15}$$

We assume that this surplus is concave in n. We write $n^\star = \arg\max_{n \geq 0} V_n$ for the surplus-maximizing number of children, assuming that $0 < n^\star < \infty$, and $V^\star = \max_{n \geq 0} V_n$ for the maximized surplus.

We write the woman's dynamic optimization problem in recursive form. The relevant state variables are i) the current date t, ii) an indicator $\ell \in \{0, 1\}$ for whether the woman is still in the labor market, iii) her current wage y (which is always zero when $\ell = 0$), and iv) her current number of children n (which is always zero when $\ell = 1$). Suppose the woman chooses action $a_t = a \in \{0, 1\}$ at date t and faces state variables (t, ℓ, y, n). The state variables at date $t+1$, denoted $(t+1, \ell', y', n)$, are governed by the following laws of motion:

$$\ell' = \ell(1-a)$$

$$y' = \begin{cases} \ell(1-a)\,y, & \text{w.pr. } 1-p, \\ \ell(1-a)\,Y, & \text{w.pr. } p \end{cases}$$

$$n' = \begin{cases} n + a, & \text{w.pr. } 1-\lambda, \\ n, & \text{w.pr. } \lambda \end{cases}$$

The initial values for these state variables at date 0 are given by $t = 0$, $\ell = 1$, and $y = n = 0$.

We define her continuation surplus, that is, her maximized utility in excess of consuming her current wealth, by $\mathcal{V}(t, \ell, y, n)$. This value must satisfy the Bellman equation

$$\mathcal{V}(t, \ell, y, n) = \max_{a \in \{0,1\}} E\left[(1-a)\,y + \mathcal{V}\left(t+1, \ell', y', n'\right) \middle| \, a\right] \tag{16}$$

with terminal condition

$$\mathcal{V}(T, \ell, y, n) = V_n. \tag{17}$$

We now characterize the solution. Notice that whenever $\ell = 0$ at any date, we must also have $y = 0$, because women who have left the labor market cannot earn wages. Whenever $\ell = 1$, we must have $n = 0$, because working women cannot have children. Hence, we can restrict the state space to the following three regions:

1. The woman has left the labor market, so that $\ell = 0$ and $y = 0$.

2. The woman is in the labor market with high income, so that $\ell = 1$, $y = Y$ and $n = 0$.

3. The woman is in the labor market with low income, so that $\ell = 1$, $y = 0$ and $n = 0$.

In the following proposition, we present a general closed-form solution to the Bellman equation for each region:

Proposition 6 *The solution to the Bellman equation (16) with terminal condition (17) is as follows:*

1. *For a woman who has left the labor market, we have*

$$\mathcal{V}(t,0,0,n) = \begin{cases} \sum_{k=0}^{T-t} \begin{pmatrix} T-t \\ k \end{pmatrix} (1-\lambda)^n \lambda^{T-t-n} \max\{V_{n+k}, V^\star\}, & n < n^\star, \\ V_n, & n \geq n^\star \end{cases} \tag{18}$$

2. *For a woman who is in the labor market with high income $y = Y$, we have*

$$\mathcal{V}(t,1,Y,0) = \begin{cases} (t_H^\star - t)\, Y + v_{t_H^\star}, & t < t_H^\star, \\ \mathcal{V}(t,0,0,0), & t \geq t_H^\star \end{cases} \tag{19}$$

where $t_H^\star$ is the lowest integer t that satisfies

$$\mathcal{V}(t,0,0,0) \geq Y + \mathcal{V}(t+1,0,0,0) \tag{20}$$

3. *For a woman who is in the labor market with low income $y = 0$, we have the recursion*

$$\mathcal{V}(t,1,0,0) = \begin{cases} p\mathcal{V}(t+1,1,Y,0) + (1-p)\,\mathcal{V}(t,1,0,0), & t < t_L^\star, \\ \mathcal{V}(t,0,0,0), & t \geq t_L^\star, \end{cases} \tag{21}$$

where $t_L^\star \leq t_H^\star$ is the lowest integer t that satisfies

$$\mathcal{V}(t,0,0,0) \geq p\mathcal{V}(t+1,1,Y,0) + (1-p)\,\mathcal{V}(t+1,0,0,0) \tag{22}$$

The intuition is as follows. For point 1, a woman who has left the labor market optimally gets pregnant if and only if she has not reached the surplus-maximizing number of children $n^\star$. Her continuation value is therefore is the expectation of the maximal surplus she can achieve in $T-t$ trials of pregnancy. Evaluating the associated (binomial) probabilities yields Equation (18). For point 2, we guess and verify that a woman with high income chooses a cutoff rule, and gets pregnant after a threshold date $t_H^\star$. The intuition for this cutoff strategy is that, because the surplus V_n is concave in n, the marginal cost of delaying fertility by one more period is increasing over time, while the marginal benefit is fixed at the current wage Y. For point 3, we guess and verify that a woman with low income also chooses a cutoff rule.

This proposition immediately yields the woman's optimal policy:

Corollary 1 *The woman's optimal policy is as follows:*

1. *A woman who has left the labor market chooses to get pregnant ($a = 1$) if and only if $n < n^\star$.*

2. *A woman who is in the labor market with high income chooses $a = 1$ if and only if $t \geq t_H^\star$.*

3. *A woman who is in the labor market with high income chooses $a = 1$ if and only if $t \geq t_L^\star$, where $t_L^\star \leq t_H^\star$.*

Next, we evaluate the effect of changes in the child mortality rate λ on the woman's strategy. For comparison with the baseline model, we concentrate on extensive margin effects. We characterize the effects on λ on the optimal timing of fertility among women who have not been promoted.[46]

Proposition 7 *Assume that $n^\star \leq 1$. If the rate λ of child mortality satisfies*

$$\lambda < \frac{1-p}{2}, \tag{23}$$

then the threshold $t_L^\star$ that determine the optimal timing of fertility is decreasing in λ. Conversely, a decline in λ encourages women who have not been promoted to delay their fertility. Moreover, a woman who is indifferent between starting her fertility and waiting at $t = t_L^\star$ will choose $t = t_L^\star + 1$ after a marginal decline in λ.

We conclude that a decline in λ leads to delay across the board, i.e., regardless of the initial optimal choice of fertility, under the same upper bound on λ as in the baseline model.

Proof of Proposition 6 **Proof.** We derive the expression for value function in point 1 directly by characterizing the woman's optimal behavior. Then, we verify that the conjectured solutions in points 2 and 3 satisfy the Bellman equation.

Point 1: For a woman who has left the labor market, it is clearly optimal to get pregnant if and only if $n < n^\star$. Hence, if $n \geq n^\star$ at date t, the woman does not get pregnant at any date $s \geq t$, and enjoys surpus V_n at the final date. If $n < n^\star$ at date t, we can model the woman's potential number of live births as a latent binomial random variable with $T - t$ trials and success probability

[46] An equivalent result for women who have been promoted is available, as long as these women optimally get pregnant at least once, with $t_H^\star < T$. Promoted women who never get pregnant ($t_H^\star = T$) may switch to trying once ($t_H^\star = T - 1$) after a decline in λ.

$1-\lambda$. If the number of successes k is such that $n+k \leq n^{\star}$, then she optimally gets pregnant at every date $s \geq t$ and enjoys final surplus V_{n+k}. Otherwise, she optimally gets pregnant until $n = n^{\star}$. Evaluating her expected utility under there probabilities yields the desired expression.

Point 2: Define the sequence

$$v_t = \mathcal{V}(t,0,0,0),\ 0 \leq t \leq T,$$

as the expected surplus of a woman who begins her fertility at date t. Notice point that v_t is the expected value of $\max\left\{V_{k(t)}, V^{\star}\right\}$, where $k(t)$ is a binomial random variable with $T-t$ trials and success probability $1-\lambda$. Since $\max\{V_k, V^{\star}\}$ is increasing in k, the expectation $v_t = E\left[\max\left\{V_{k(t)}, V^{\star}\right\}\right]$ is increasing in the number of trials and therefore decreasing in t.

Moreover, consider the increments $z_t \equiv v_{t-1} - v_t$. We have

$$z_t = (1-\lambda)\sum_{k=0}^{T-t}\left(\begin{array}{c} T-t \\ k \end{array}\right)(1-\lambda)^k \lambda^{T-t-k}\left(\max\{V_{k+1}, V^{\star}\} - \max\{V_k, V^{\star}\}\right)$$

Therefore z_t is the expected value of

$$u_{k(t)} = \max\left\{V_{k(T)+1}, V^{\star}\right\} - \max\left\{V_{k(t)}, V^{\star}\right\},$$

Since surplus is concave, u_k is a decreasing sequence. Therefore, the expectation $z_t = E\left[u_{k(t)}\right]$ is decreasing in the number of trials and therefore increasing in t. From this result, it follows that there exists a unique period $t_H^{\star}$ such that (20) holds if and only if $t \geq t_H^{\star}$.

We are now ready to verify that our conjectured solution $\mathcal{V}(t,1,Y,0)$ satisfies the Bellman equation. First, suppose that $t \geq t_H^{\star} \Leftrightarrow v_t \geq Y + v_{t+1}$. Then the right-hand side of the Bellman equation is

$$\begin{aligned}\max\{v_t, Y + v_{t+1}\} &= v_t \\ &= \mathcal{V}(t,1,Y,0)\end{aligned}$$

as required. Second, suppose $t < t_H^{\star} \Leftrightarrow v_t < Y + v_{t+1}$. Then the right-hand side of the Bellman equation is

$$\max\left\{v_t, Y + (t_H^{\star} - t - 1)\,Y + v_{t_H^{\star}}\right\}$$

We need to show that this equals $(t_H^{\star} - t)\,Y + v_{t_H^{\star}}$. This is true and only if

$$\begin{aligned} & v_t \leq (t_H^{\star} - t)\,Y + v_{t_H^{\star}} \\ \Leftrightarrow & \sum_{s=t}^{t_H^{\star}-1}(v_s - v_{s+1} - Y) \leq 0 \end{aligned}$$

which is true because $v_s < Y + v_{s+1}$ for all $s < t_H^{\star}$. Hence, the proposed solution in point 2 solves the Bellman equation for all dates t.

Point 3: We first show that there exists a unique period $t_L^\star$ such that (22) holds if and only if $t \geq t_L^\star$. It is sufficient to show that the following is increasing in t:

$$v_t - pv_{t+1} - (1-p)\,\mathcal{V}(t+1,1,Y,0) = v_t - v_{t+1} - (1-p)\left[\mathcal{V}(t+1,1,Y,0) - v_{t+1}\right]$$

The first term, $v_t - v_{t+1}$, is increasing in t by our argument above. The second term is zero whenever $t \geq t_H^\star$. When $t < t_H^\star$, we need to show that the expression in square brackets is decreasing in t. This is the case if and only if

$$\begin{aligned} (t_H^\star - t)\,Y + v_{t_H^\star} - v_t &\geq (t_H^\star - t - 1)\,Y + v_{t_H^\star} - v_{t+1} \\ \Leftrightarrow v_t - v_{t+1} &\leq Y, \end{aligned}$$

which follows from the definition of $t_H^\star$. This argument also implies that $t_L^\star \leq t_H^\star$.

We are now ready to verify that our conjectured solution $\mathcal{V}(t,1,y,0)$ satisfies the Bellman equation. We consider three cases. First, suppose that $t \geq t_H^\star \geq t_L^\star$. Then the right-hand side of the Bellman equation is

$$\max\left\{v_t, p\mathcal{V}(t+1,1,Y,0) + (1-p)\,\mathcal{V}(t+1,1,0,0)\right\} = \max\left\{v_t, v_{t+1}\right\} = v_t,$$

as required. Second, suppose that $t_H^\star > t \geq t_L^\star$, which implies that $v_t \geq p\mathcal{V}(t+1,1,Y,0) + (1-p)\,v_{t+1}$. Then the right-hand side of the Bellman equation is

$$\max\left\{v_t, p\mathcal{V}(t+1,1,Y,0) + (1-p)\,v_{t+1}\right\} = v_t,$$

as required. Finally, suppose that $t < t_L^\star$, which is equivalent to $v_t < p\mathcal{V}(t+1,1,Y,0) + (1-p)\,v_{t+1}$. Then the right-hand side of the Bellman equation is

$$\max\left\{v_t, p\mathcal{V}(t+1,1,Y,0) + (1-p)\,\mathcal{V}(t+1,1,0,0)\right\}.$$

We need to show that this equals the left-hand side, which is given by

$$\mathcal{V}(t,1,0,0) = p\mathcal{V}(t+1,1,Y,0) + (1-p)\,\mathcal{V}(t+1,1,0,0).$$

We are done if we can show that our conjectured solution satisfies

$$\mathcal{V}(t,1,0,0) \geq v_t.$$

We confirm this inequality by induction. It holds with equality at $t = t_L^\star$. Suppose it holds at date

$t+1 \leq t_H^{\star}$. Then

$$\begin{aligned}\mathcal{V}(t,1,0,0) &= p\mathcal{V}(t+1,1,Y,0)+(1-p)\,\mathcal{V}(t+1,1,0,0)\\ &\geq p\mathcal{V}(t+1,1,Y,0)+(1-p)\,v_{t+1} > v_t,\end{aligned}$$

where the last inequality follows from the definition of $t_L^{\star}$. This completes the proof. ■

Proof of Proposition 7 **Proof.** If $n^{\star}=1$ then $t_L^{\star} \leq T-1$ for all λ. If $t_L^{\star}=T-1$ then it must weakly decrease after any change in λ. We therefore focus on $t_L^{\star} < T-1$. To show that $t_L^{\star}$ is decreasing in λ, it is sufficient to show that the inequality in (22) is more likely to hold, for a given $t \leq T-2$, after a marginal increase in λ. Hence, we need to show that the following expression is increasing in λ:

$$v_t-(1-p)\,v_{t+1}-p\left[\left(t_H^{\star}-(t+1)\right)Y+v_{t_H^{\star}}\right]$$

If $t_H^{\star}=T$, then this is equal to

$$v_t-(1-p)\,v_{t+1} \tag{24}$$

plus a constant that does not depend on λ. With $n^{\star}=1$, we have

$$v_t=\left(1-\lambda^{T-t}\right)V_1$$

so that

$$\frac{\partial v_t}{\partial \lambda}=-\left(T-t\right)\lambda^{T-t-1}V_1$$

Hence, we obtain that $\frac{\partial[v_t-(1-p)v_{t+1}]}{\partial\lambda}>0$ if and only if

$$\lambda<(1-p)\,\frac{T-(t+1)}{T-t},$$

which holds for all $t \leq T-2$ if $\lambda < \frac{1-p}{2}$.

If, on the other hand, $t_H^{\star} < T$, then the expression of interest is equal to

$$v_t-v_{t+1}+p\left(v_{t+1}-v_{t_H^{\star}}\right) \tag{25}$$

plus a constant that does not depend on λ. The first term is strictly increasing in λ whenever $\lambda < \frac{1}{2}$. The second term is the sum of increments v_s-v_{s+1} for $s \leq t_H^{\star}-1$, all of which are also increasing in λ whenever $\lambda<\frac{1}{2}$. This establishes that $t_L^{\star}$ is decreasing in λ, as required. Since the terms of interest in (24) and (25) are *strictly* increasing in λ, it also follows that a woman who is indifferent between starting her fertility and waiting for one period at $t=t_L^{\star}$ (i.e., for whom (22) holds with equality) will now choose $t=t_L^{\star}+1$ after a marginal decline in λ. ■

D.4 Extensions

This appendix extends our baseline model in various directions. The associated economic intuitions are discussed in the paper. Our treatment of heterogeneous preferences in terms of a single parameter A in our baseline model is not illuminating in all extensions, in particular in those with general (not quasilinear) preferences and higher birth orders. For a unified treatment, in each extension, we establish a more general version of our main result: A decline in child mortality λ expands the parametric region in which delayed fertility is optimal.

In particular, in each extension, we derive the expected continuation value U_1 of getting pregnant at date 1 and the expected continuation value U_0 of working at date 1. We then derive conditions under which the following implication holds:

$$U_1 \geq U_0 \Rightarrow \frac{\partial\left[U_1 - U_0\right]}{\partial \lambda} > 0. \tag{26}$$

The intepretation of Condition (26) is as follows: If it is satisfied, then any woman who prefers early fertility for a low value of λ will also prefer it for a higher value of λ. Moreover, if a woman is indifferent between delayed and early fertility, then she strictly preferes delayed fertility after a marginal decrease in λ. Hence, establishing (26) is sufficient to argue that a decline in λ encourages a wider set of women to delay.

D.4.1 Income effects

In this section, we make two generalizations to our baseline model. First, the woman's utility takes the general shape $U(n, e, c)$, where $U(.)$ is concave in its three arguments. We assume that the marginal utility of consumption is weakly increasing in both quality and quantity of children:

$$\frac{\partial^2 U}{\partial c \partial n} \geq 0 \text{ and } \frac{\partial^2 U}{\partial c \partial e} \geq 0. \tag{27}$$

Second, to generate meaningful income effects between dates 1 and 2, we now allow the woman's wage before promotion to take a non-zero value $y > 0$.

We define the woman's total income as $m \equiv \sum_{t=1}^{2} y_t (1 - a_t)$. The indirect utility of having n children and earning total income m is

$$V_n(m) = \max_{e,c} \left\{U(n, e, c) \text{ subject to } c + n(\tau_q + \tau_e e) = m\right\}. \tag{28}$$

We further define the surplus from having n relative to having no children with income m as

$$S_n(m) = V_n(m) - V_0(m).$$

We will make use of the following intermediate results

Lemma 1 *The indirect utility function $V_n(m)$ is concave in m.*

Lemma 2 *The surplus from one child satisfies*

$$S_1(y) \geq S_1(0).$$

We characterize the conditions under which declines in λ encourage delay.

Proposition 8 *If the rate λ of child mortality satisfies*

$$\lambda < \frac{1-p}{2}\frac{S_1(y)}{S_1(0)}, \tag{29}$$

then Condition (26) is satisfied, and a decline in λ encourages a wider set of women to delay.

We can compare Condition (29), under which a decline in λ encourages delay, to the equivalent condition in the baseline model without income effects, which is

$$\lambda \leq \frac{1-p}{2}.$$

It is clear that the Condition (29) is weaker, meaning that switches to delay in response to declines in λ are (weakly) more likely when there are income effects, because $S_1(y) \geq S_1(0)$, as implied by Lemma (2).

Proof of Lemma 1 **Proof.** Fix n, and let (c, e) nnd (c', e') be the solutions to the maximization problem in (28) when income is m and m', respectively. Let $m'' = \mu m + (1-\mu) m'$ for some $\mu \in [0,1]$. Since the budget constraint is linear for a given n, the choice (c'', e''), with $c'' = \mu c + (1-\mu) c'$ and $e'' = \mu e + (1-\mu) e'$, is affordable with income m'. It follows that $V_n(.)$ is concave, because

$$\begin{aligned} V_n(m'') &\geq U(c'', n, e''), \\ &\geq \mu U(c, n, e) + (1-\mu) U(c', n, e') \\ &= \mu V_n(m) + (1-\mu) V_n(m'), \end{aligned}$$

where the second inequality follows from the concavity of $U(.)$. ■

Proof of Lemma 2 **Proof.** We need to show that $V_1(y) - V_0(y) \geq V_1(0) - V_0(0)$, or equivalently:

$$V_1(y) - V_1(0) \geq V_0(y) - V_0(0)$$

It is sufficient to show that

$$\frac{\partial V_1(m)}{\partial m} \geq \frac{\partial V_0(m)}{\partial m}, m \in [0, y] \tag{30}$$

Let c_n be optimal consumption, e_n optimal child quality, and λ_n the Lagrange multiplier on the woman's budget constraint, when the woman has n children at $t = 3$. Let $e_0 = 0$ without loss of generality. Now (30) redues to

$$\begin{aligned}
&\lambda_1 \geq \lambda_0 \\
\Leftrightarrow &\frac{\partial U\left(c_1, 1, e_1\right)}{\partial c} \geq \frac{\partial U\left(c_0, 0, 0\right)}{\partial c}
\end{aligned}$$

The woman's budget constraint yields $c_1 = c_0 - \tau_q - \tau_e e_0 < c_0$. If $U(.)$ is concave and satisfies (27), we obtain

$$\frac{\partial U\left(c_1, 1, e_1\right)}{\partial c} \geq \frac{\partial U\left(c_0, 1, e_1\right)}{\partial c} \geq \frac{\partial U\left(c_0, 0, 0\right)}{\partial c},$$

which completes the proof. ■

Proof of Proposition 8 **Proof.** To establish Condition (26), suppose that $U_0 \geq U_1$. We can write

$$\begin{aligned}
U_1 &= V_0\left(0\right) + \left(1+\lambda\right)\left(1-\lambda\right) S_1\left(0\right) \\
U_0 &= p \max\left\{V_0\left(y+Y\right), V_0\left(y\right) + \left(1-\lambda\right) S_1\left(y\right)\right\} \\
&\quad + \left(1-p\right) \max\left\{V_0\left(2y\right), V_0\left(y\right) + \left(1-\lambda\right) S_1\left(y\right)\right\}
\end{aligned} \tag{31}$$

Notice that

$$\begin{aligned}
0 &\leq U_1 - U_0 \\
&\leq V_0\left(0\right) + 2\left(1-\lambda\right) S_1\left(0\right) - V_0\left(2y\right) \\
\Rightarrow V_0\left(2y\right) - V_0\left(0\right) &\leq 2\left(1-\lambda\right) S_1\left(0\right)
\end{aligned}$$

Since $V_0\left(.\right)$ is concave we also have $V_0\left(2y\right) - V_0\left(0\right) \geq 2\left[V_0\left(2y\right) - V_0\left(y\right)\right]$. Using Lemma (2), we find that

$$\left(1-\lambda\right) S_1\left(y\right) \geq V_0\left(2y\right) - V_0\left(y\right).$$

Using this inequality to simplify U_0 in (31) and differentiating, we get

$$\frac{\partial\left[U_1 - U_0\right]}{\partial\lambda} = \left(1 - p + p\delta\right) S_1\left(y\right) - 2\lambda S_1\left(0\right),$$

where $\delta = 1\left\{V_0\left(y+Y\right) \leq V_0\left(y\right) + \left(1-\lambda\right) S_1\left(y\right)\right\} \in \left\{0,1\right\}$ is an indicator for whether the woman gets pregnant upon promotion. This is positive if

$$\lambda < \frac{1 - p + p\delta}{2\lambda} \frac{S_1\left(y\right)}{S_1\left(0\right)}.$$

Since $\delta \geq 0$, the bound in (29) is sufficient for Condition (26), which completes the proof. ■

D.4.2 Higher birth orders

In this section, we consider women for whom it may be optimal to have two children. We replace our assumption that $V_1(A) < V_2(A)$, which guaranteed that at most one child was optimal in the baseline model, with the weaker assumption that the surplus from having children is concave in the quantity of children. Concretely, in the three period model, we assume that:

$$V_1(A) - V_0(A) \geq V_2(A) - V_1(A), \forall A \geq 0. \tag{32}$$

For example, with a Cobb-Douglas utility function $u(n, e) = e^{\alpha} n^{1-\alpha}$, this is always satisfied if $\alpha \leq \frac{1}{2}$, while at most one child is always optimal when $\alpha > \frac{1}{2}$.

We characterize the conditions under which declines in λ encourage delay.

Proposition 9 *If the rate λ of child mortality satisfies*

$$\lambda < \frac{1}{1 - \Delta(A)} \left[\frac{1-p}{2} - \Delta(A) \right], \forall A \geq 0 \tag{33}$$

where

$$\Delta(A) = \max \left\{ \frac{V_2(A) - V_1(A)}{V_1(A) - V_0(A)}, 0 \right\}, \tag{34}$$

then Condition (26) is satisfied, and a decline in λ encourages a wider set of women to delay.

The upper bound for λ in Condition (33) is tighter than the equivalent in the baseline model, which is

$$\lambda \leq \frac{1-p}{2}.$$

In particular, the right-hand side of (33) is decreasing in $\Delta(A)$. This quantity, defined in (34), measures the strength of a woman's preference for a second child, relative to her preference for her first child. If a woman does not benefit from having a second child at all, then $\Delta(A) = 0$. Otherwise, $\Delta(A)$ is a number less than one, due to our assumption that surplus is concave.

Proof of Proposition 9 **Proof.** To establish Condition (26), fix a preference parameter A and suppose that $U_0 \geq U_1$, which implies that $V_1(A) \geq 0$. We now have

$$\begin{aligned} U_0 &= (1+\lambda)(1-\lambda) V_1(A) + (1-\lambda)^2 \max\{V_2(A) - V_1(A), 0\} \\ U_1 &= p \max\{Y, (1-\lambda) V_1(A)\} + (1-p)(1-\lambda) V_1(A) \end{aligned}$$

Therefore,

$$\frac{\partial [U_0 - U_1]}{\partial \lambda} = (1 - p + p\delta) V_1(A) - 2\lambda V_1(A) - 2(1-\lambda) \max\{V_2(A) - V_1(A), 0\}$$

where $\delta = 1\{Y \leq (1-\lambda) V_1(A)\} \in \{0, 1\}$ is an indicator for whether the woman gets pregnant upon promotion. To establish Condition (26), it is sufficient to show under which condition this expression is positive when $\delta = 0$. Substituting $\delta = 0$ and rearranging yields the upper bound on λ in Condition (33) and completes the proof. ■

D.4.3 Increasing risk of infertility

We assume that between dates 1 and 2 in our baseline model, the woman becomes infertile with probability ϕ, in which case her probability of childbirth at date 2 drops from $1 - \lambda$ to 0. In this environment, the risk of infertility increases when fertility is delayed.

We characterize the conditions under which declines in λ encourage delay.

Proposition 10 *If the rate λ of child mortality satisfies*

$$\lambda < \frac{1 - p - \frac{\phi}{1-\phi}}{2} \tag{35}$$

then Condition (26) is satisfied, and a decline in λ encourages a wider set of women to delay.

The upper bound for λ in Condition (35) is tighter than the equivalent in the baseline model, which is

$$\lambda \leq \frac{1-p}{2}.$$

In particular, the constraint on λ is more stringent when the likelihood ratio $\frac{\phi}{1-\phi}$ of infertility versus fertility is large.

Proof of Proposition 10 **Proof.** To establish Condition (26), fix a preference parameter A and suppose that $U_1 \geq U_0$, which implies that $V_1(A) \geq 0$. We now have

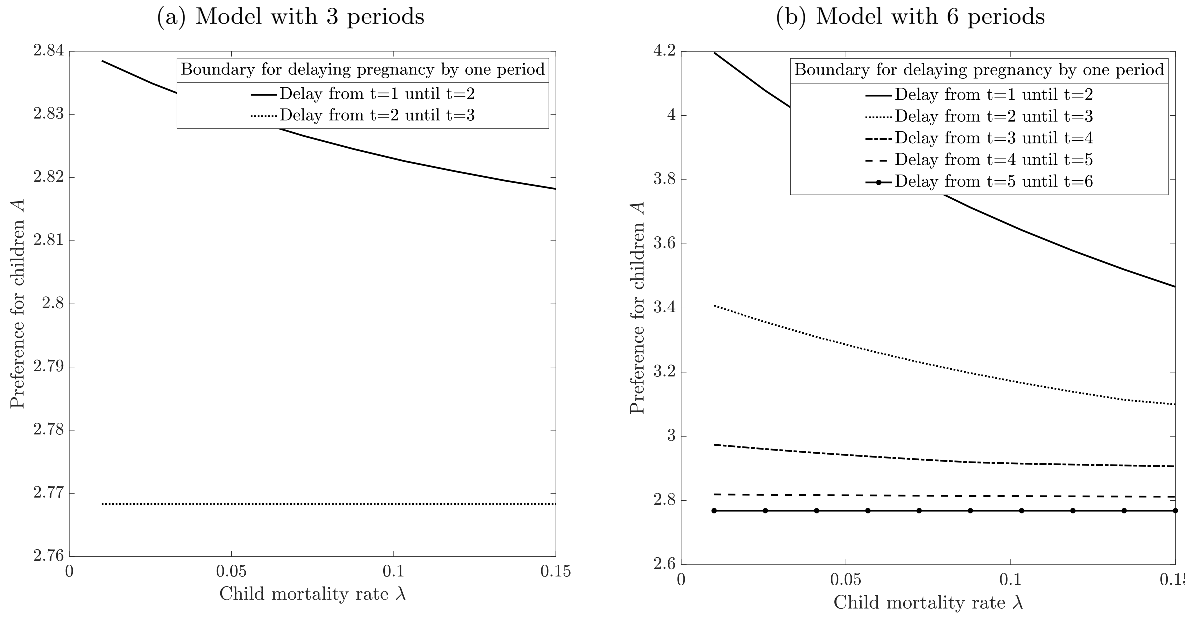
$$\begin{aligned} U_1 &= [1 - \lambda + \lambda (1-\phi)(1-\lambda)] V_1(A) \\ U_0 &= p [\phi Y + (1-\phi) \max\{Y, (1-\lambda) V_1(A)\}] \\ &\quad + (1-p)(1-\phi)(1-\lambda) V_1(A) \end{aligned}$$

Therefore,

$$\frac{\partial [U_1 - U_0]}{\partial \lambda} = V_1(A) [\delta p (1-\phi) + (1-p)(1-\phi) - \phi - (1-\phi) 2\lambda]$$

where $\delta = 1\{Y \leq (1-\lambda) V_1(A)\} \in \{0, 1\}$ is an indicator for whether the woman gets pregnant upon promotion. To establish Condition (26), it is sufficient to show under which condition this expression is positive when $\delta = 0$. Substituting $\delta = 0$ and rearranging yields the upper bound on λ in Condition (35) and completes the proof. ■

Figure A.6: Numerical example: The effect of a reduction in child mortality on fertility delay with risk of infertility



The figure plots the critical values of the preference parameter A, which measures the strength of the preference for children, as a function of the rate λ of child mortality. If A is above the top line in panel (a), it is optimal to get pregnant early at $t = 1$. If A is between the top and the bottom line, it is optimal to delay fertility $t = 2$. If A is below the bottom line, then no fertility (or, equivalently, delay until $t = 3$) is optimal. The region of delay becomes wider as λ declines. Similarly, panel (b) shows the optimal regions of pregnancy timing for a model with 6 periods. The parameter values are: $\tau_q = \tau_e = 2$, $u(n, e) = Ae^{\alpha}n^{1-\alpha}$, $\alpha = 0.9$, $Y = 1$, $p = 0.1$. We assume in this figure that the probability of a live birth at period t is $(1 - \lambda)(1 - 0.04 * t)$, which declines linearly over time for any given λ.

D.4.4 Marriage decisions

We assume that, at the beginning of each period $t = 1, 2$, the woman can search for a potential marriage partner. Her search succeeds with probability σ. If she finds a potential partner at $t = 1$, she can marry or reject him. Marriage implies that the woman leaves the labor market, potential wages drop to zero. Rejection implies that she cannot get pregnant, so that her probability of childbirth upon choosing $a_1 = 1$ drops to zero. If she does not find a partner at $t = 1$, she also cannot get pregnant. If the woman is unmarried at $t = 2$, she can conduct another (independent) search for a partner, which also succeeds with probability σ.

A woman who has not found a partner at date 1 will celarly find it optimal to work and chose $a_1 = 0$. Only a woman who has found a partner makes the crucial decision in our model, namely, whether to attempt early fertility or delay.

Consider a woman who has found a partner at date 1. If she marries him, it is optimal to get pregnant immediately and choose $a_1 = 1$. We let U_1 be the continuation payoff from this strategy. If she does not marry him, it is optimal to work and choose $a_1 = 0$. We let U_0 be the associated continuation payoff.

We characterize the conditions under which declines in λ encourage delay in both marriage and fertility. This is the case if, in terms of the continuation payoffs U_0 and U_1 that we have defined, Condition (26) holds.

Proposition 11 *If the rate λ of child mortality satisfies*

$$\lambda < \frac{1 - p - \frac{1-\sigma}{\sigma}}{2} \tag{36}$$

then Condition (26) is satisfied, and a decline in λ encourages a wider set of women to delay both marriage and fertility.

We conclude that the constraint on λ is more stringent when the likelihood ratio $\frac{1-\sigma}{\sigma}$ associated with not finding a partner is large.

Proof of Proposition 11 **Proof.** To establish Condition (26), fix a preference parameter A and suppose that $U_1 \geq U_0$, which implies that $V_1(A) \geq 0$. We now have

$$\begin{aligned} U_1 &= (1+\lambda)(1-\lambda)V_1(A) \\ U_0 &= y + p\left[(1-\sigma)Y + \sigma \max\{Y, (1-\lambda)V_1(A)\}\right] \\ &\quad + (1-p)\sigma(1-\lambda)V_1(A) \end{aligned}$$

The expression for U_0 follows from the fact that a woman who does not marry at $t = 1$ has another chance to marry and have children at date 2 with probability σ, which is the likelihood that a new potential partner is found. Clearly, these continuation values are identical to those in the previous subsection in the proof of Proposition 10, except that the probability ϕ of infertility is replaced by the probability $1 - \sigma$ of not finding a partner at date 2. The result follows immediately. ■

D.4.5 Career choices

We assume that a woman commits to a choice of careers at date 1. If she chooses a "risky" career, then the process governing her potential wages y_t is the same as in our baseline model. If she chooses a "safe" career, then she earns $y_1 = 0$ at date 1, as in the baseline model, but receives a guaranteed wage potential $y_t = \bar{y}$ at date 2 if she does not get pregnant. We assume that

$$\bar{y} = pY + w < Y$$

Hence, the safe career pays the average wage of the risky career, plus a premium w. The safe career has a flatter trajectory than the risky one, and offers no chance of promotion to a wage as high as Y. We focus on the interesting case with a positive premium $w > 0$. Indeed, for $w = 0$, the risky career is a dominant strategy because the option to leave the labor market implies that the woman's utility is a convex function of future wages. To facilitate the exposition, we further assume an upper bound on the premium w:

$$0 < w < \frac{(1-p)p}{p+\lambda} Y. \tag{37}$$

We characterize the conditions under which declines in λ encourage delay in both marriage and fertility.

Proposition 12 *If the rate λ of child mortality satisfies*

$$\lambda < \frac{1-p}{2} \tag{38}$$

then Condition 12 is satisfied, and a decline in λ encourages a wider set of women to delay both marriage and fertility.

We conclude that, under the same upper bound on λ as in the baseline model, declines in λ encourage delay in the model with career choices. An interesting auxiliary result characterizes optimal career choices:

Proposition 13 *The woman's optimal policy is as follows:*

1. ***No Fertility:*** *If $A < A^{\star}(\lambda)$, where $\underline{A}$ is defined by $V_1 (A^{\star}(\lambda)) = \frac{w}{(1-p)(1-\lambda)}$, the woman chooses a safe career, and works at $t = 1$ and at $t = 2$ with probability 1.*

2. ***Delayed Fertility:*** *If $A^{\star}(\lambda) \leq A < \bar{A}(\lambda)$, where $\bar{A}(\lambda)$ is defined by $V_1 \left(\bar{A}(\lambda)\right) = \frac{pY}{(p+\lambda)(1-\lambda)}$, then the woman chooses a risky career, works at $t = 1$, and gets pregnant at $t = 2$ if and only if she is not promoted.*

3. ***Early Fertility:*** *If $\bar{A}(\lambda) \leq A$, then the woman gets pregnant at $t = 1$, and gets pregnant again at $t = 2$ if and only if she does not have a surviving child yet. Her choice of career is indeterminate.*

Proof of Proposition 12 **Proof.** To establish Condition (26), fix a preference parameter A. In this environment, the continuation value of choosing to get pregnant at date 1, in which the woman's choice of career is irrelevant, is

$$U_1 = (1+\lambda)(1-\lambda) V_1 (A).$$

The continuation value of not getting pregnant at date 1 is evaluated under the optimal choice of career, and equals

$$U_0 = \max \{U_0^s, U_0^r\},$$

where U_0^s and U_0^r are the continuation values of working at date 1 and choosing the safe and risky career, respectively, which are formally defined by

$$U_0^s = \max\{pY + w, (1-\lambda) V_1(A)\}$$
$$U_0^r = p \max\{Y, (1-\lambda) V_1(A)\} + (1-p) \max\{0, (1-\lambda) V_1(A)\}.$$

Suppose that $U_1 \geq U_0$, which implies $V_1(A) \geq 0$. We must have $U_1 \geq U_0^r$, which yields

$$\begin{aligned} 0 &\leq U_1 - U_0^r \\ &\leq (1+\lambda)(1-\lambda) V_1(A) - pY - (1-p)(1-\lambda) V_1(A) \\ \Rightarrow V_1(A) &\geq \frac{pY}{(p+\lambda)(1-\lambda)}. \end{aligned} \tag{39}$$

We argue that we must have $U_0^r \geq U_0^s$ by considering three cases. First, suppose that $(1-\lambda) V_1(A) > Y$. Then, $U_0^r - U_0^s = 0$. Second, suppose that $(1-\lambda) V_1(A) \in [pY + w, Y]$. Then, $U_0^r - U_0^s = p(Y - (1-\lambda) V_1(A)) \geq 0$. Third, suppose that $(1-\lambda) V_1(A) < pY + w$. Then $U_0^r - U_0^s = (1-\lambda) V_1(A) - w \geq 0$, where the inequality follows from (39) and (37).

Hence, we have

$$U_1 - U_0 = U_1 - U_0^r,$$

and differentiating yields

$$\frac{\partial [U_1 - U_0]}{\partial \lambda} = [1 - p + p\delta - 2\lambda] V_1(A)$$

where $\delta = 1\{Y \leq (1-\lambda) V_1(A)\} \in \{0, 1\}$ is an indicator for whether the woman gets pregnant upon promotion. To establish Condition (26), it is sufficient to show under which condition this expression is positive when $\delta = 0$. Substituting $\delta = 0$ and rearranging yields the upper bound on λ in Condition (38) and completes the proof. ■

Proof of Proposition 13 **Proof.** In the proof of Proposition 38, we establish that $U_1 \geq U_0$ implies $U_0 = U_0^r$. Hence, the point with $U_0 = U_1$, at which the woman is indifferent between early and delayed fertility, is the same as in the baseline model. Hence, the woman gets pregnant at date 0, in which case her career choice is indeterminate, if and only if $A \geq \bar{A}(\lambda)$, where $\bar{A}(\lambda)$ is defined as in Proposition 1 in the paper. Moreover, for $A \leq \underline{A}$, where $\underline{A}$ is defined as in Proposition 1, it is optimal never to get pregnant, in which case the woman strictly prefers the safe career because it offers higher average earnings whenever $w > 0$.

For $\underline{A} < A < \bar{A}(\lambda)$, we have

$$U_0^r - U_0^s = pY + (1-p)(1-\lambda) V_1(A) - \max\{pY + w, (1-\lambda) V_1(A)\}$$

At $A = \underline{A}$, we have

$$U_0^r - U_0^s = -w < 0.$$

At $A = \bar{A}(\lambda)$, we have $U_0^r = U_1$, and

$$U_0^r - U_0^s = (1+\lambda)(1-\lambda) V_1\left(\bar{A}(\lambda)\right) - \max\left\{pY + w, (1-\lambda) V_1\left(\bar{A}(\lambda)\right)\right\}$$

We argue that at this point, we must have $U_0^r - U_0^s > 0$. This follows by considering two cases. First, if $pY + w \leq (1-\lambda) V_1\left(\bar{A}(\lambda)\right)$, then $U_0^r - U_0^s = \lambda(1-\lambda) V_1\left(\bar{A}(\lambda)\right) > 0$. Second, if $pY + w > (1-\lambda) V_1\left(\bar{A}(\lambda)\right)$, then

$$\begin{aligned} U_0^r - U_0^s &= (1+\lambda)(1-\lambda) V_1\left(\bar{A}(\lambda)\right) - pY - w \\ &= \frac{(1-p) pY}{p+\lambda} - w > 0 \end{aligned}$$

where the last line substitutes the definition of $\bar{A}(\lambda)$, and the inequality follows from our assumption in (37).

Notice that, over the interval $A \in \left[\underline{A}, \bar{A}(\lambda)\right]$, $U_0^r - U_0^s$ is a piecewise linear function of $V_1(A)$ with at most one kink, which starts strictly negative and ends strictly positive. Moreover, the function has a single crossing with zero where $A = A^\star(\lambda)$, which must be on the increasing part of the function and is obtained by solving

$$(1-p)(1-\lambda) V_1(A^\star(\lambda)) = w.$$

At this crossing point, we must have $pY + w > (1-\lambda) V_1(A^\star(\lambda))$; otherwise the function is locally decreasing, contradicting the single crossing property. Hence, we conclude women with $A < A^\star(\lambda)$ optimally choose the safe career and never get pregnant. To complete the proof, it is simple to check using our assumption in (37) guarantees that $A^\star(\lambda) < \bar{A}(\lambda)$. ■