Fertility Responses to Infant and Maternal Mortality: Quasi-Experimental Evidence from 20th Century America

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Abstract: The introduction of the first antibiotics in the United States in the late 1930s led simultaneously to a sharp fall in infant and maternal mortality. We study the fertility response to these changes. Consistent with theoretical predictions, we find that the fall in maternal mortality led to increased fertility. The fall in infant mortality increased fertility on the extensive margin but decreased it on the intensive margin. Our results contribute to a small empirical literature that provides well-identified estimates of the quantity-quality tradeoff and they support the contention of "essential complementarity" posited in a recent extension to the canonical model (Aaronson, et al, 2012)

Keywords: fertility, quantity-quality tradeoff, maternal mortality, infant mortality endowments, essential complementarity, medical innovation, pneumonia, demographic transition

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1. Introduction

The transition to low fertility is central in theories of economic growth (Galor and Weil 1996) but the causes of the historical fertility transition remain hotly debated in contemporary economic research. Theoretical models that emphasize the role of adult or child mortality tend to posit one of two mechanisms. One involves increases in life expectancy extending the time horizon over which returns to human capital investments can be reaped, which encourages investments and simultaneously lowers fertility (Galor and David Weil, 1996, though see Marco Cervellatti and Uwe Sunde, 2005). The other rests upon parental uncertainty around child survival creating a precautionary demand for children, so that mortality decline leads to a decline in births and the number of surviving children (Sebnem Kalemli-Ozcan, 2003, Rodrigo Soares, 2005). However a decline in child mortality also reduces the price of child quantity and this will tend to stimulate fertility, particularly if the precautionary demand for children is modest (Oded Galor, 2012).

Empirical examinations of the relationship between mortality and fertility face several challenges. First, the endogeneity of mortality remains a major challenge in terms of establishing causality in the empirical sphere as well as the theoretical (Timothy W Guinnane, 2011). Second, the comparability of available empirical studies to theoretical work is complicated by the factthat many analyze total fertility (number of births) rather than net fertility (surviving births), while theoretical predictions tend to refer to the latter (Oded Galor, 2012). Analysis of the dynamics of mortality and fertility within mother suggests that mortality decline stimulates a decline in gross fertility as it lowers "replacement births" but as for every one child that dies, 0.37 additional children are born, the change in net live births is positive (Sonia Bhalotra and Arthur van Soest, 2008). A third issue is that few studies distinguish fertility responses on the extensive and intensive margins, although recent theoretical advances predict competing responses on the two margins (Aaronson et al. *forthcoming*). Finally, the literature focuses upon declines in child mortality (or gender-neutral increases in life expectancy) but often if not always the factors driving a decline in child mortality will also lead to a decline in maternal mortality. Failing to account for this may bias the estimated impacts on fertility, a concern that appears to be altogether absent in the literature (although see Albanesi, 2011). Addressing this concern requires dealing with the endogeneity of both child and maternal mortality. In view of these manifold issues, it is perhaps not surprising that the empirical literature provides a mixed picture (see Section 2c).

This paper investigates causal effects of child and maternal mortality decline on (gross and net) fertility at the extensive and intensive margins. It uses the plausibly exogenous arrival of the first antibiotics (sulfonamide drugs) in the United States in 1937 as a source of identification. Sulfa drugs were responsible for sharp declines in infant and child mortality, particularly from pneumonia and, by virtue of controlling puerperal fever, they simultaneously led to a sharp drop in maternal mortality (Seema Jayachandran et al., 2010, John E Lesch, 2007, Thomasson and Treber. 2008). We introduce state variation in treatment effects by exploiting the fact that states most burdened by pneumonia and maternal mortality in the pre-sulfa era experienced the largest declines upon the introduction of sulfa drugs, using an approach similar to that in Acemoglu and Johnson (2007) and Bleakley (2007). In particular, we investigate whether the post-sulfa convergence in birth year levels of pneumonia and maternal mortality levels across the states after 1937 is mirrored in the fertility behavior of women giving birth in the 1930s and early 1940s. This approach allows us to generate two instruments from the one innovation since the pre-intervention levels of infant pneumonia mortality and maternal mortality exhibit a different distribution across the US states.

A recent survey argues that an ideal test of the presence of a quantity-quality trade-off requires a change in the *relative price of quantity* or a change in the *return to quality*" (Galor 2012: section 4). The introduction of sulfa drugs did both, with improvements in maternal mortality directly influencing the relative price of quantity and improvements in child mortality and, importantly, also child morbidity, raising the return to quality. We expect that reduced maternal mortality rates, by lowering the cost of childbirth, will have led to increased fertility. *A priori,* the fertility response to child mortality is ambiguous. In the setting of a quantity-quality (Q-Q) fertility model (Gary S Becker and H Gregg Lewis, 1973), we posit that the innovation-led decline in childhood pneumonia will have reduced both the price of child quantity and the shadow price of child quality. It will have also reduced the number of children needed to achieve the propagation of parental genes. The fall in the price of child quality operates primarily through improvements in morbidity rates, which positively impact the formation of robust child health and cognitive endowments and increase the rate of return to subsequent human capital investments. Complementarities across inputs in the production of human capital enhance the productivity of any initial improvement, effectively lowering the price further.

Improvements in mortality are often used to proxy improvements in morbidity (Carlos Bozzoli et al, 2009) because morbidity data are harder to gather on a consistent time series basis but the function mapping morbidity to mortality is essentially unknown. Since mortality and morbidity improvements may have opposing effects on fertility it is important to identify morbidity improvements and, in particular, the extent to which they stimulate investments in quality. We do this in recent work using the sample of births analyzed in this paper (Sonia Bhalotra and Atheendar Venkataramani, 2012). We document that children born after the introduction of sulfa drugs received subsequent larger human capital investments and that this contributed to their exhibiting substantially higher levels of education, employment and income and lower levels of work-related disability. An advantage of using a historical intervention is that we actually observe rather than simply expect higher investments in the human capital of treated cohorts. This allows us to posit with greater confidence that the parents of the treated cohorts (parents giving birth after 1937) will have lowered their net fertility at the intensive margin. At the same time, it seems plausible that the increase in the survival chances and the (health and cognitive) endowments of births (their quality) will have encouraged prospective parents to shift from zero to at least one birth. This is an insight developed and tested in Daniel Aaronson et al, (2012).

We use historical census data to estimate two sets of models. One models the probability of birth in 1930-1943 as a function of the timing of the introduction of sulfa drugs. For this we use the 5% samples of the census micro-data files for 1940 and 1950 and a measure of net fertility. We complement this analysis with estimates of the stock of births recorded by the same cohorts of women (women of reproductive age in 1930-1943) at a later stage, when they have completed their fertility. We model the birth stock as a function of the woman's duration of exposure to sulfa drugs during her reproductive years and for this we avail of a measure of gross total fertility using census files for 1940-1970. The flow model captures the dynamics of fertility at the time and as discussed earlier it is an advantage to have a measure of net fertility. The stock model allows us to distinguish the extensive and intensive margins of fertility.

Our findings are consistent across the two models and they line up nicely with theoretical predictions. Essentially, we identify that the drop in maternal mortality from 1937, conditional upon the drop in pneumonia mortality amongst infants, leads to significantly higher fertility on the intensive margin while it has no significant impact at the

extensive margin. Conditional upon maternal mortality, the drop in infant pneumonia mortality leads to significantly lower fertility on the intensive margin and significantly higher fertility on the extensive margin, the undifferentiated impact being negative but weakly determined. The absence of an extensive margin response to maternal mortality decline is consistent with this representing a decline in the price of quantity and in particular with their being no complementarity of this with a change in the price of quality. Probing education gradients in fertility responses to infant pneumonia decline, we find that the increase in fertility at the extensive margin is concentrated amongst younger and more educated women while the decrease in fertility at the intensive margin comes primarily from prime-aged (19-35) women with less than secondary education.

Since the estimated impacts are heterogeneous across the states as a function of the pre-1937 levels of pneumonia and maternal mortality, the impacts of the two changes are on different scales and cannot be readily compared. Using the stock model coefficients, we simulated the impacts of a decline in mortality corresponding to an interquartile shift (a movement from the 75^{th} to the 25^{th} percentile) in the pre-intervention distribution of each mortality rate. Our estimates imply that the sulfa-induced decline in pneumonia mortality produced a 9% point increase in the probability of at least one birth while at the same causing 0.15 fewer births (4.7% of the mean) conditional upon at least one. The decline in maternal mortality resulted in an additional 0.13 births (4% of the mean). Estimates from the hazard model describing the probability of birth in the sample period reveal the same pattern of results. Since the two sets of estimates use different variables to indicate fertility, the way that they line up strengthens confidence in the results. The estimates are in general robust to the inclusion of fixed effects for the age of the mother, controls for maternal education, wealth, state and year varying macroeconomic factors, other communicable and non-communicable diseases and state specific time trends.

We contribute to the literature in the following respects. First, we provide some of the scarce causal evidence of a positive relationship child mortality and fertility. In particular, our results suggest that mortality declines can lead to reductions in fertility at the intensive margin if there are concomitant changes in morbidity that bolster child endowments and thereby lower the price of quality. In this vein, our results add to the still small empirical literature demonstrating the validity of the Q-Q model (Daniel Aaronson et al., 2012, Sandra Black et al., 2005, Hoyt Bleakley and Fabian Lange, 2009, Hongbin Li et al., 2008, Mark R Rosenzweig and Kenneth Wolpin, 1980, Mark R Rosenzweig and Junsen Zhang, 2009).

Our results also contribute to understanding why the empirical literature surrounding the mortality-fertility link is so mixed. In particular, the sources of mortality decline likely impact a number of factors that simultaneously change the prices of quantity and quality. In our example, the arrival of antibiotic technology led to reductions in both child mortality and morbidity, the different impacts of which express as oppositely signed intensive and extensive margin elasticities. They also led to a reduced probability of maternal death, which made childbirth more attractive. Summing across these responses, the arrival of this new technology contributed a relatively small net change in fertility in this period (see Tertilt and Jones, 2008, Albanesi and Olivetti, 2010 for a documentation of longer trends including this period). However what is important for economic outcomes including investments in human capital, women's labor supply and growth is that the distribution of births across households changed, becoming less polarized and, importantly, with relatively educated women contributing a larger share of births.

Another contribution of this paper lies in its garnering empirical support for the theory of "essential complementarity" put forth by Daniel Aaronson, et al (2012). The only

previous tests of this hypothesis would appear to be theirs and our results for infant pneumonia are arguably stronger than those offered in the original paper. Their results flow from an education intervention that increased access to schools while our results flow from a health intervention that improved infant health which, in turn, raised the return to subsequent health and education investments in treated individuals. Our work also differs in several other ways including its investigation of fertility responses to maternal mortality and its modeling of the dynamics of birth alongside total fertility.

Our findings are of wider contemporary relevance. Although infant mortality rates have been declining in developing countries, they remain unnecessarily high (Black et al. 2003, Angus Deaton, 2004). Surviving children are permanently disadvantaged as a result of exposure to infectious disease, despite the availability of mitigating interventions such as the public provision of clean water or affordable medicines (Janet Currie and Thomas Vogl, 2012, Bhalotra and Venkataramani 2012a,b). Fertility remains high in many countries especially in Africa and human capital investments are low. Many developing countries have made the fertility transition but we have at best a limited understanding of the process. Aaronson, et al, argue that since historical fertility transitions have been characterized by reductions in fertility on both the intensive and extensive margins, their findings – and ours- of increasing fertility on the extensive margin undermine the role of mortality declines driven by processes that reduce morbidity and the price of quality in engineering the transition. This said, the relevance of these results from twentieth century America to today's poorer countries remains to be established. The proportion of women who remain childless in today's developing countries is small, so the extensive margin may not be moot.

The remainder of the paper evolves as follows. Section II develops the context. It documents the "first stage" decline in pneumonia and maternal mortality rates occasioned by the sulfa drug revolution; presents the theoretical background with reference to both the standard Q-Q model and the extension incorporating essential complementarity; and briefly reviews the findings of relevant previous studies. Section III discusses the data and Section IV sketches the empirical strategy. Section V presents the main results and Section VI concludes.

2. Background

2a. Mortality Rates and the Sulfa Drug Revolution

Early 20th century America was characterized by high levels of maternal and infant mortality (Rollo H Britten, 1942, Forrest E. Linder and Robert D. Grove, 1947, WHO, 2011, Melissa A Thomasson and Janet Treber, 2008, Seema Jayachandran, Adriana Lleras-Muney and Kimberly Smith, 2010)[1]. Pneumonia was the leading cause of infant morbidity and mortality after death from premature birth and congenital defects and it is similarly the leading cause of infant death in developing countries today[2]. A major cause of maternal mortality, accounting for 40% of maternal deaths was puerperal fever, an ascending bacterial infection of the reproductive tract that occurs soon after birth. Such infections remain the

[1] The rates of maternal and infant mortality per 1000 live births were 6.35 and 1.06 respectively (US Vital Statistics).

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

leading cause of maternal mortality in the developing world and, among those who survive, can cause uterine scarring and infertility.

For pneumonia and post-partum fever amongst new mothers, the mainstay of treatment up until the introduction of sulfonamide antibiotics in 1937 was supportive care. The arrival of antibiotics drastically altered the standard of medical care. Sulfa drugs were discovered by German chemists experimenting with textile dyes in 1932 and become widely available in the United States starting in 1937 at relatively low cost (Barron H Lerner, 1991, John E Lesch, 2007). By all accounts adoption was widespread and quick and this was reflected in sharp declines in maternal mortality as well as mortality from pneumonia (Jayachandran, et al (2010).[3] *Figures 1 and 3* illustrate a trend break in both causes of death in 1937 and *Figure 1b* shows that the steepest decline in pneumonia mortality was amongst infants. *Figures 2a, b* demonstrate that the arrival of sulfa drugs stimulated convergence in the levels of both mortality rates across the US states, the states with higher pre-1937 burdens of disease showing greater absolute declines in mortality levels. As we discuss in Section III, we use the cohort breaks interacted with these sharp convergence patterns towards identification of causal effects of declines in both causes of mortality.

In addition to reducing mortality, the arrival of sulfa drugs led to significant reductions in morbidity (Joseph Greengard et al., 1943, Horace L Hodes et al., 1939, E Earl Moody and E.G. Knouf, 1940). This was established in clinical trials that we cite in Bhalotra and Venkataramani (2012a) but it seems likely that the antibiotics led to reductions in the duration and severity of disease in the wider community given that they were available without prescription until 1939 and at relatively low cost. Historical research documents their wide penetration in the consumer pharmaceuticals market (Lesch, 2007). That morbidity declines were substantively important is demonstrated in a companion paper where we show that reductions in pneumonia morbidity in infancy driven by the arrival of sulfa drugs had significant long-run impacts on educational attainment, employment, income, and disability (Bhalotra and Venkataramani, 2012a).

2b. Predicted Impacts on Fertility

This discussion is framed with reference to the theoretical framework in Galor (2012) and the extension of this in Aaronson et al. (*forthcoming*). Households possess a utility function which holds as arguments consumption goods, the number of children and the quality of children. They maximize this subject to a budget constraint which involves expenditures on consumption goods and child rearing. The cost of rearing a given child is a function of the fixed (opportunity) cost of rearing a child irrespective of quality plus the total cost of child quality (the unit cost multiplied by the desired level of quality). For a household with multiple (planned) children, the total cost of child rearing is the number of children multiplied by the total cost. Solving the model gives the shadow prices of quantity and quality. The price of quantity is increasing in the quality of children and the price of quality is increasing in the quantity of children. Thus, the model incorporates a quantity-quality tradeoff.

In the setting of this model, maternal mortality decline reduces the price of child quantity because the risk of post-partum death and/or infertility represents a (high) cost of producing an additional child. There may be some impacts on the price of quality, as well -

[3] Sulfa drugs also reduced mortality and morbidity from skin and soft tissue infections and meningitis (Jayachandran, et al, 2010). However the incidence of these diseases was very low and they made fairly insignificant contributions to both infant and maternal mortality.

for example, forward looking parents may perceive a lower probability of maternal death as an decrease in the price of child quality because maternal inputs are important in producing child health and cognition (evidence of which is in Jerome Adda et al, 2011). Available empirical results on the relationship between fertility and maternal mortality suggest that changes in the shadow cost of quantity may dominate (Albanesi and Olivetti, 2010, Albanesi 2011)[4].

The impact of reductions in pneumonia morbidity and mortality is also *a priori* ambiguous. On the one hand, reductions in infant pneumonia morbidity decrease the price of child quality as they result in improved infant health and cognitive endowments and, as a result, a higher rate of return to subsequent human capital investments. The results in Bhalotra and Venkataramani (2012) underscore the significance of this channel. On the other, reduced infant mortality risk reduces the price of child quantity. This will encourage fertility, but fertility may be reduced on account of falling mortality if parents hold a precautionary demand for children (Sebnem Kalemli-Ozcan, 2003) or because they need to have fewer births to ensure that they propogate their genes (Rodrigo Soares, 2005).

Sharpening the underlying theoretical model can help tease apart these countervailing effects. Aaronson, et al (2012) augment the model by introducing complementarity between quantity and quality on the extensive margin, to account for the fact that substitution of quality for quantity cannot happen at a corner solution. The effect of reduced pneumonia morbidity and mortality is unambiguously positive on the extensive margin as reductions in the prices of quantity and quality both increase the probability of a (first) child. On the intensive margin, the net impact on fertility remains ambiguous but the *a priori* expectation is that the quality effect will dominate. First, even if it contributed the largest share of infectious disease mortality the actual level of pneumonia mortality was low, at roughly 1.06%. Second, the results in our earlier paper on the long-run impacts of the arrival of sulfa drugs demonstrate that scarring vastly outweighed selection in this context, aided by dynamic complementarities across endowments and subsequent inputs in the formation of human capital.

2c. Related Literature

A recent review of studies of the demographic transition is in Galor (2012). We do not attempt a comprehensive survey of relevant studies but instead discuss a few related recent studies. In the Introduction we argued that there was no consensus on the direction in which mortality decline pushed fertility because of identification issues, differences in the forces driving mortality decline and aggregation over the margins of response. This is illustrated by recent studies that attempt to utilize exogenous variation in mortality: Fortson (2009) finds no relationship between mortality from HIV/AIDS and fertility while Turan (2011) finds that reductions in child mortality flowing from an immunization program in India (phased in over a long period of time) led to a reduction in the number of births. By virtue of studying eradication of a disease (hookworm) that did not cause mortality, Hoyt Bleakley and Fabian Lange (2010) are able to shut down the quantity challenge and they identify a decline in fertility flowing from a decline in the price of quality. In an analysis of the effects of malaria eradication on fertility, Adrienne Lucas (*forthcoming*) finds an increase in fertility which is traced to an earlier age at first birth, there being inconclusive impacts on

[4] Their estimates are set in the same context as ours but are not strictly comparable since they look at different cohorts, over a longer time span, modelling fertility trends as a function of first and second generation responses. These studies are discussed in the next section.

higher order births. A suggested explanation of these findings is that malaria has biological effects on fecundity and the survival of first births, and it is difficult with the evidence to discern a role here the prices of quantity or quality. Becker et al. (2012) show that the expansion of educational enrollment of children lowered fertility in early nineteenth century Prussia. In a similar vein but distinguishing the margins at which fertility responds, Aaronson, et al (2012) show that women facing improved schooling opportunities for their children following the building of Rosenwald Schools for Southern blacks in the US in 1913-1932 became more likely to have at least one child but had smaller families overall. We confirm their insight, but work off a health intervention incident in 1937 which acted to raise the returns to schooling for children of the index mothers.

A few recent studies show that reductions in maternal mortality that differentially improve the life expectancy of women lead to increased human capital investments in young girls, consistent with returns to these investments flowing over a longer period (Seema Jayachandran and Adriana Lleras-Muney, 2009; Albanesi and Olivetti, 2010; Alabanesi, 2011). The latter two studies additionally study whether the fertility of the young girls receiving the enhanced investments is altered. So while we look at a decline in maternal mortality as lowering the cost of childbirth, they look at it as enhancing investments in the potential birth. As a result the important difference with regard to this paper is that these studies analyze the fertility of women born in an era in which maternal mortality while we analyze the fertility of their mothers, or women giving birth in this era. Albanesi and Olivetti (2010) and Albanesi are motivated to explain booms and busts in fertility over a range of some four decades and they distinguish women in broad cohort bands, while we estimate short run elasticities focusing attention upon the discontinuity in 1937 created by the introduction of sulfa drugs. Albanesi and Olivetti (2010) find that the decline in maternal mortality is associated with a rise in fertility for women born between 1921 and 1940 in the US. Using cross-country data for 25 advanced and emerging economies between 1900 and 2000, Albanesi (2011) find that fertility peaked between 10 and 15 years after the start of the maternal mortality decline.[5] In work in progress we analyze the fertility of second generation women (women who were born in the sulfa drug era) but for the questions pertaining to human capital investment in children that drive this paper, the relevant responses are of first generation women (women giving birth in the sulfa drug era).

3. Research Strategy

Our research strategy exploits the timing of the introduction of antibiotics which created sharp variation across cohorts in exposure to disease, with systematically larger changes in disease levels in states with higher pre-intervention burdens of disease. The cross-cohort trend break and the cross-state convergence in pneumonia and maternal mortality rates created by the arrival of antibiotics were illustrated in Figure 4[6]. The corresponding equations, which establish statistical significance of these patterns, are in Table 1[7], and they establish a healthy "first stage" of the process of interest. We essentially compare fertility behavior before and after the arrival of sulfa drugs for mothers born in states with higher

[5] Our findings are broadly consistent with the Albanesi model insofar as it predicts that a permanent improvement in maternal health initially increases fertility and that declines in infant mortality contribute to a negative trend in fertility.

[6] Figure 4 currently shows convergence for pneumonia; the corresponding graphs for maternal mortality will be in the next draft.

[7] Table 1 presents tests of the trend breaks. Tests of convergence will be in the next draft.

and lower pre-antibiotic mortality rates. Although the drop in infant pneumonia and maternal mortality rates coincides in 1937, the cross-state distribution of infant pneumonia and maternal mortality rates prior to 1937 is different, and this allows us to identify the impacts of both changes.

Using the sample of women of reproductive age in 1930-43 and a data file expanded to the woman*year level, we estimate a hazard model which allows us to delineate exposure to sulfa drugs in each potential birth year and alongside we estimate a "stock model" in which we model exposure as the number of fertile years spent in the post-antibiotic era. This is the form of the hazard model:

$$(1)\ P(Y_{jsc} = 1) = \beta + \beta_1 * post\text{-}1937_c * base\_pneumonia_s + \beta_2 * post\text{-}1937_c * base\_MMR_s + f(timesince)_j + \beta_3 * age\ of\ mother_j + \theta_{jsc} + \delta_c + \lambda_s + u_{jsc}$$

where Y_{jsc} is a binary indicator = 1 if woman j in state s reports a birth in year c, $post\text{-}1937_c = 1$ if the year is 1937 or after, $base\_pneumonia_s$ and $base\_MMR_s$ are the pre-sulfa revolution average mortality rates from pneumonia and maternal mortality in state s, $f(timesince)_j$ is a flexible function for the time since the last birth, and θ_{jsc}, δ_c, λ_s are fixed effects for birth order, year, and state of residence. The model is estimating using OLS. In extensions of this model, we include interactions of birth order with the polynomial *timesince*, and we introduce mother fixed effects. The coefficients of interest are β_1 and β_2 which reflect the percentage point changes in the probability of given birth following the antibiotic driven declines in morbidity and mortality from infant pneumonia and maternal mortality respectively. Following a tradition in the literature (Douglas Almond, 2006; Bozzoli et al, 2009), we are assuming that for infant pneumonia changes in morbidity are proxied by changes in mortality but this assumption is upheld by the results of our companion study (Bhalotra and Venkataramani 2012).

We also estimate a model designed to capture total fertility. We give up the ability to identify responses from a discontinuity in exposure in a dynamic setting but we gain an estimate of whether fertility responses simply reflect the advancing or the deferral of fertility or they influence the stock of births. The estimated equation is

$$(2)\ B_{jsc} = \alpha + \alpha_1 * years\ of\ exposure\ to\ sulfa_j * base\_pneumonia_s + \alpha_2 * years\ of\ exposure\ to\ sulfa_j * base\_MMR_s + \alpha_3 * age\ of\ mother_j + \delta_c + \lambda_s + e_{sc}$$

Here, B_{jsc} represents the total number of births reported by women aged between 40 and 50 at enumeration who were 15-44 years old in 1937, the time of the arrival of sulfa drugs. The variable *years of exposure* is the number of fertile years after 1937 which is a function of the woman's birth cohort and the coefficients of interest are α_1 and α_2.

Importantly, we extend the analysis to identify the potentially different elasticities at the extensive and intensive margins. To identify the extensive margin response, we redefine the dependent variable as a binary indicator for zero versus a non-zero number of lifetime births. To identify the intensive margin response, we re-estimate equation (2) restricting the sample of women to those with at least one birth.

We consistently cluster standard errors at the state level to account for the increased propensity to falsely reject the null hypothesis in difference-in-differences models that do not account for serial correlation across years within states (Marianne Bertrand, et al, 2004).

A threat to inference with the models outlined above is that of omitted variables that exhibit cohort and state variation, the impacts of which may load on to the variables of interest indicating exposure to infant and maternal mortality. Candidate omitted variables include pre-intervention trends in other factors that influences fertility such as income or skill-biased technological change that differentially increases the returns to quality (i.e. human capital investment) or that produces increased opportunities for women. We therefore include individual level controls for mother's education and expect this to contribute to controlling for fertility preferences as well as potential wages for women (T Paul Schultz, 1985). We also control for state and year varying socioeconomic variables including the logarithm of state income per capita, state per capita public health spending, and the numbers of schools, hospitals and physicians per capita.

The stimuli we wish to trace the impacts of are changes in diseases specific to infant and maternal mortality that were treatable with sulfa drugs. So as to ascribe the estimated impacts to these diseases and this intervention, we control for diseases that were not treatable with sulfa drugs but that may have had (a more secular) influence on fertility. This effectively controls for the broader disease environment including state specific public health interventions at the time. We enter these controls in the same form as we enter the mortality rates of interest, using the pre-1937 mortality rates for a number of communicable and non-communicable diseases interacted with the sulfa drug exposure measure. The inclusion of communicable disease rates (tuberculosis, diarrhea, malaria) helps control for state specific changes in for instance sanitation, public health programs and housing that may have coincided with the arrival of sulfa drugs and also impact fertility decisions. The inclusion of non-communicable diseases is expected to control for factors such as health care quality and access.

To allow for any remaining trends that may have driven convergence or divergence across the US states in the sample period and that may jointly influence mortality risk and fertility at the state level, we also report specifications with birth state specific time trends. After we present the main results, we discuss robustness checks including a falsification test on the timing of the introduction of sulfa drugs and checks on sensitivity of our estimates to assumptions about the state of residence of mother during the fertile years that are relevant in the stock model. We also discuss how data limitations specific to each of the hazard and stock models may bias the estimates, making it important to look for consistency in the broad pattern of results across the two specifications.

4. Data

Data on fertility outcomes are taken from the United States Census Microdata (Steven Ruggles et al., 2010). We create two different datasets, one for each empirical strategy we employ, given the complementary strengths and weakness for each (*Appendix Table 1* describes each dataset in greater detail). The first dataset (hereafter, the "hazard data set") is from pooled census microdata from the 1940 1%, and 1950 1% samples. We select women who were of child-bearing age (age 15-44) at some point during the window 1930 and 1943 and create a dataset with observations at the individual woman*year level so that for every woman we have an observation on whether or not she gave birth in every year that she was at risk in the sample and we estimate a hazard equation which models the probability of birth in a given year conditional upon the time since the previous birth. The dependent variable is a dummy variable set to unity for each woman*year cell in which a birth occurred. As in Bhalotra and Venkataramani (2012), we restrict our analysis to the time window 1930-1943 in order to reduce the possibility of confounding from other public

health events or interventions (for example, the influenza epidemic of 1928-9 and the increasingly widespread use of penicillin after 1943). Exposure to sulfa-drug driven reductions in maternal and pneumonia mortality and morbidity risk is assigned on the basis of hazard year, with fertile years 1937 and after defined as being exposed to the new antibiotics. As discussed, we enhance identification by looking for fertility to mirror the cross-state convergence in mortality (and implicitly morbidity) rates driven by sulfa-drugs. Thus, the state of residency in which the woman is at risk of fertility is important. We assign this on the basis of the child's reported birth state and birth year. The state of residence in between births is not known, but the majority of mothers reported residence in the state in which their first child was born (84%).

The indicator of fertility derives from a record of the number of children living in the mother's household at the time of enumeration (*nchild*). Using a variable that links child records to mothers, we constructed a complete history of live births for each woman. We restrict births to biological births (95% of children in the household) that occurred in the US and we glean year of birth (from age) of own children living in the household in the year of the census. We are only able to measure net fertility; our indicator of fertility excludes any pregnancies that did not result in live births (this is standard) and it is net of any child deaths that occurred after birth and before the census (these were small, the under-5 mortality rate in this period was xx). As discussed in section 2a, the relevant theoretical predictions pertain to net fertility and this is also what is relevant to theories of population growth and economic growth (Acemoglu and Johnson, 2007) so this is not a major concern. Another potential concern is that we do not know the timing of births that moved out of the household before enumeration, but for births in 1930-43, we expect that children are too young to have migrated at enumeration in 1930-1950 so, again, this is not a real concern.

Our second data set (hereafter, the "stock data set") is constructed so as to identify the completed fertility of women of age 15-44 in 1937. To measure exposure to the sulfa drug revolution we create for each woman an indicator for the number of fertile years remaining after 1937. By this measure, women 15 and under in 1937 spent the entirety of their reproductive lives in the sulfa drug era, those who had crossed the age of 35 in 1937 are defined as being unexposed and women aged 25 in 1937 are defined as having 10 fertile years exposed to sulfa drugs. The state of residence during the fertile years is somewhat tricky to assign. For women with at least one birth, we assign the state the oldest enumerated child was born in; this is supported by the fact that some 85% of women reported residence in the same state as their oldest enumerated birth. For those women with no births (33%), we assume that the current state of residence is the state where they spent their fertile years, a choice that we challenge in one of our robustness checks.

To measure completed fertility we look for women aged 15-44 in 1937 in future census files in which they are between 40 and 50 years of age[8]. We assume that they have completed fertility by age 35 (consistent with a plot of age at birth in these data) and we limit the age of observation to 50 to avoid survivor selection which would set in at older ages. This involves using census microdata for each year in 1940-1970. We measure fertility using

[8] For example, women who were 20 in 1937 will have turned 40 in 1957 so we use the 1960 census to obtain their completed fertility as reported at the age of 43.

a variable which asks all ever-married women to record the total number of births they had over their lifetime (*chborn*)[9].

For the exposure measures we utilized state level time series data on pneumonia and maternal mortality rates compiled from several volumes of the US Vital Statistics (Robert D. Grove and Alice M Hetzel, 1968, Forrest E. Linder and Robert D. Grove, 1947, Steven Ruggles, J Trent Alexander, Katie Genadek, Ronald Goeken, Matthew B Schroeder and Matthew Sobek, 2010, United States Bureau of the Census, 1930-1943)[10]. We also collected data on under-2 diarrhea, heart disease, cancer, malaria, and tuberculosis mortality from these sources as control variables. For each disease, we created state specific pre-sulfa era rates by averaging the cause-specific mortality rates between 1930 and 1936.

We also collected state time series data on a number of socioeconomic and infrastructure variables to serve as controls. 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 [11] . For state per capita health expenditures, we used data collected from various reports from the US Census bureau[12].

Table 1 provides descriptive statistics for both the hazard and stock datasets, as well as for the state-specific disease environment measures and controls.

5. Results

5a. Hazard Model

There is a significant response of the conditional probability of birth to both changes; it increases in response to the post-1937 decline in maternal mortality while decreasing in response to the post-1937 decline in infant pneumonia mortality (Table 2). Effect sizes for a change in "*base*" corresponding to an interquartile shift (from the 75th to the 25th percentile) of the pre-1937 distribution of maternal and pneumonia mortality rates respectively, using the coefficients in the final column conditional upon state trends are as follows. A change in *base_pneumonia* equivalent to a movement from the 75th to 25th percentile (0.26) implies a 0.47% point drop in the probability of a birth after 1937, which is 4.3% of the mean (0.11). A similar shift in *base_maternal mortality* (1.73) implies a 0.48% point increase in the probability of a birth after 1937, 4.4% of mean. The exact balancing of these changes is a coincidence and it should be noted that the units of change are not comparable, that is, an interquartile shift in the maternal mortality distribution is not the same as an interquartile shift in the infant mortality distribution.

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[9] We could use the variable that we used in the hazard sample, which records the number of a woman's own children living in the current household. We avoid this since for women in their forties, children may have left the household.

[10] 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).

[11] 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). We used linear interpolation for each state to calculate education and health infrastructure values for 1940-1943, as Lleras-Muney's data was only collected through to 1939.

[12] See http://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/6304?archive=ICPSR&q=6304.

5b. Stock Model

The pattern of results is very similar to that exhibited by the hazard model (Table 3). We simulate effect sizes for the interquartile shift in "*base*" as before but now since the treatment term involves not a dummy but fertile years exposed to sulfa, effect sizes are evaluated at the sample mean of this exposure variable (11.2 years). The drop in maternal mortality leads to an additional 0.403 births and the drop in infant pneumonia mortality to 0.405 fewer births. These changes constitute 16% of the mean of the dependent variable (i.e. completed fertility, 2.5 births).

Isolating the extensive from the intensive margin reveals significant differences in the fertility response at the two margins (Tables 4 and 5). The change in maternal mortality has no impact on fertility at the extensive margin. The positive effect observed overall is evident at the intensive margin but is now smaller for the same change in *base_maternal mortality*, at an additional 0.127 births, or 4.08% of the mean (the mean at the intensive margin is 3.1 births). The impacts of pneumonia mortality reduction are significant at both margins and, consistent with expectation, are of opposite sign. At the extensive margin, the interquartile change in *base_pneumonia* implies an increase in the probability of at least one birth of 9.2% points. At the intensive margin, the same change in base implies 0.145 fewer births, 4.71% of the mean.[13] Comparing Tables 4 and 5 with Table 3, it is clear that restricting the extensive and intensive margin responses to be identical (as is standard in the literature), the overall response takes the sign of the intensive margin but the "pooled" coefficient over-estimates the response at the intensive margin.

Our finding of no extensive margin response to the drop in maternal mortality is consistent with it making small if any changes to the price of child quality. The lower price of child quantity provides the same incentive to have a first birth as it does to have further births. In contrast the decline in infant pneumonia mortality changes the price of quantity and quality, and these changes interact. In particular, the lower price of quality leads to an additional stimulus to have a child at the extensive margin that bolsters the impact of a reduced price of quantity (prices are complementary), consistent with the hypothesis of Aaronson et al. (*forthcoming*).

5c. Heterogeneity by Education and Age of the Woman

We re-estimated the models, sub-sampling by an indicator for whether the woman had above or below secondary education. The stock model results (available in a subsequent draft of the paper) show that the extensive margin response derives from the sample of relatively educated women while the intensive margin response is driven by less educated women. This is consistent with the baseline rates of childlessness and total fertility being higher for the more and less educated (respectively) and the antibiotic induced health improvements stimulating convergence in fertility levels between these groups. We also re-estimated the models allowing coefficients specific to age of exposure (results available in the next draft of the paper).

[13] The simulations reported here are for the average years of exposure to sulfa in the full sample (of 11.2 years). The difference is small but these figures need to be updated for intensive margin results using the average years of exposure in the subsample of women on whom the intensive margin equation is estimated. In the next draft of the paper we will show estimates of extensive and intensive margin responses in the hazard model. The transition to the first birth in this dynamic specification will essentially show the response of age at first birth. The intensive margin response will measure changes in the probability of a birth on a sample of women with at least one birth.

5d. Robustness Checks

The estimates are in general robust to the inclusion of fixed effects for the age of the mother, controls for maternal education, wealth, state and year varying macroeconomic factors, other communicable and non-communicable diseases and state specific time trends. The fact that the pattern of results is very similar in the "flow" and "stock" models, using different variables to indicate fertility, strengthens confidence in the results. In this section we discuss additional specification and data checks. Some of the issues we address here were introduced in section 3.[14]

Inference with our research strategy relies upon getting the timing of the reform right and, equally, upon our assumption that there were no other interventions or events in the sample period, omission of which may bias the coefficients of interest. To assess these matters, we estimated the hazard specification, replacing "post-1937" with a vector of dummies for birth year (which, as before, are interacted with the pre-1937 levels of pneumonia and maternal mortality). We plot these coefficients and examine whether the pattern of the relationship between the probability of birth and changes in mortality rates is consistent with the discontinuous arrival of antibiotics in 1937 and whether any discontinuity in the probability of birth is evident in any other year. So as to allow different responses at the extensive and intensive margins, we plot these coefficients for the transition to the first birth and, separately, conditional upon at least one birth. We do indeed see a trend break in the probability of birth in 1937, and not elsewhere.

Our research strategy involves looking for post-sulfa state convergence in mortality rates to reflect in convergence of fertility rates across states with higher and lower pre-sulfa burdens of infant and maternal mortality. A plot showing that this holds is in Appendix Figure 3.

There are two potential issues raised with migration. First, endogenous migration may have created compositional changes that produce the post-sulfa state convergence in fertility that we attribute to state convergence in pneumonia mortality rates. We investigated the post-sulfa change in the state population amongst individuals of reproductive age using a specification similar to that in equation (1) but with migration replacing fertility. The estimates, reported in Appendix Table 2, show that conditional upon state income, sulfa-induced changes in mortality rates did not influence migration patterns[15]. A second concern is that, as discussed in section 3, we cannot confidently assign state for all women in the stock model. This is important as it decides the state-specific assignment of the "*base*" variables, the pre-intervention mortality rates. To assess the sensitivity of our estimates to this we re-estimated it restricting the sample of women to non-migrant women for whom we can confidently assign state in the reproductive years to be the state of residence at census. The coefficients are larger in the non-migrant sample but they take the same signs at the extensive and intensive margins and are not significantly different from the coefficients in the baseline equations using the full sample.

[14] This draft does include Tables of results for these checks although we have conducted them; the next draft will.

[15] The potential concern and the estimation are both elaborated in Bhalotra and Venkataramani (2012a).

We face the common problem that we only observe survivors; mothers who died as a result of childbirth are not observed in census enumerations.[16] It seems plausible to assume that these were high risk women who, on average, had higher fertility and that they were concentrated in states with higher pre-intervention levels of maternal mortality where the intensity of the sulfa drug "treatment" was highest. It follows that when these women are selected out of the fertility sample, we will tend to under-estimate the increase in fertility that flows from the drop in maternal mortality. This bias is mitigated by our controls for observable individual indicators of risk such as age and education of the woman but, all in all, we may under-estimate the true effects. For a sense of the extent of selection, see the mortality rate statistics in Appendix Table 1.

Our estimates are not sensitive to the choice of years over which we compute pre-intervention mortality rates. The default is an average over 1930-36 but we find similar results if we instead use 1925-1935 or subsets of the 1930-36 window. Another potential issue that we investigated is whether age-specificity in the pneumonia mortality rate matters to our estimates. Pneumonia infections are most prevalent amongst infants (age 0-1); see Appendix Figure 1 and using non-parametric (flexible coefficient) models we establish in our earlier work that it is exposure to pneumonia in infancy that influences adult education and labor market outcomes (Bhalotra and Venkataramani 2012a). For these reasons we interpret variation in the all-age pneumonia mortality rate (that is used in the analysis so far) as driving variation in exposure to pneumonia for infants. This is confirmed in Figures 3 and 4 where we plot trend breaks and state convergence for age-specific pneumonia mortality alongside that for all-age pneumonia mortality; indeed the infant rate reacts most sharply to sulfa drugs.

As discussed, the hazard model relies upon a measure of net fertility, that is, we do not observe non-live births (this is standard) and births that succumbed prior to the time of census enumeration (this is not standard). The relevant theoretical predictions pertain to net fertility and this is also the statistic of interest for theories of population transitions and economic growth (Galor, 2012). Still, if one wanted to estimate changes in gross fertility then our estimates will be biased by child mortality. This bias is likely to be downwards since the sulfa-driven changes in fertility in our model are largest in states with higher pre-intervention levels of infant pneumonia mortality. Also infant mortality from pneumonia was less than 1% of live births and the few available causal estimates suggest that replacement fertility occurs, with an additional 0.37 births for every one (neonatal) death (see Bhalotra and van Soest, 2008, and references therein).

6. Conclusions

This paper presents quasi-experimental estimates of the impact on fertility of improvements in health and survival generated by the introduction of antibiotics early in the last century. These are the two striking findings of this study. First it shows that simultaneous declines in infant mortality and maternal mortality push fertility in opposite directions, the first on average causing a decline in fertility consistent with a quantity-quality tradeoff and the second causing an increase in fertility that is in line with maternal mortality primarily constituting a decline in the shadow price of quantity. Second, it shows that fertility responses to these changes are significantly different at the extensive margin than at the intensive margin, consistent with "essential complementarity" at the extensive margin, or the

[16] Note that the maternal and pneumonia mortality rates are state level averages so the problem arises from the individual fertility records being available only for women who survive to the census date.

irrelevance of substitution at a corner. Overall, the simultaneous declines in infant and maternal mortality that we study create an increase in fertility at the extensive margin, or a decrease in childlessness. At the intensive margin, in our data, the fertility-decreasing impact of infant mortality tends to offset the fertility increasing impact of maternal mortality. The estimated patterns are consistent with theory, fairly large, and robustly determined.

These results make the following contributions to the literature. First, they contribute causal estimates of the impact of infant and maternal mortality declines, both of which are scarce, but relevant to models of the demographic transition and growth. Second, our analysis has some useful features relative to previous work in this domain. By virtue of looking at a historical reform, we are able to establish an increase in the "quality" of the children of the index women when the children are adult and, in this way, clarify a source of the estimated fertility response. This is important as this is a measure of the significance of declining morbidity which is typically not observed but thought to track mortality. We model both the dynamics of fertility at the time of exposure to the reform and completed fertility, for the same cohorts of women observed in census data in different years. We present estimates for both gross and net (net of child mortality) fertility.

A third contribution to the literature is that we model a simultaneous decline in infant and maternal mortality, allowing for differential impacts of each conditional upon the other. This is clearly of importance given that public health interventions tend to influence both. Fourth, we provide the second test of the essential complementary hypothesis of Aaranson et al. (forthcoming), second only to the test in their paper. While they model fertility in response to school construction raising school opportunities for children of the index women, we model fertility in response to a health shock that improves the infant health endowment and thereby raises the returns to schooling (and other human capital investments) for children of the index woman. We further introduce maternal mortality and investigate whether this also generates different responses at the two margins.

In an extension of the analysis, we estimated the models by race. The estimates for blacks are smaller and statistically insignificantly different from zero (and the race difference in coefficients is larger on the intensive margins), so the population averaged effects that we present in this paper are driven by the behavior of white women. These results tie in with our analysis of race differences in the long run gains in human capital and labour market outcomes flowing from the introduction of sulfa drugs (Bhalotra and Venkataramani 2012a). There we show that blacks experienced reductions in pneumonia and maternal mortality upon the introduction of sulfa drugs that were no smaller than experienced by whites (see Table 1 of this paper) but that, on average, the socioeconomic gains they reaped from their improved health endowments were small. Probing this, we find that there is considerable heterogeneity amongst blacks (and not whites) in this regard. In particular, blacks born in segregated states (mostly in the South) did not complement infant health improvements with further investments in child quality because the returns to investment were capped by institutionalized segregation in schools and labour markets. We find compelling evidence that blacks in less segregated states in the North exhibited substantial long run socioeconomic gains from sulfa. Since most blacks lived in segregated states, the weak gains dominate the average. For the purposes of this paper, the upshot is that blacks did not significantly increase investments in child quality after the arrival of sulfa and so we expect that they showed more limited declines in fertility, and this is what we see. We also see more limited increases in fertility in response to maternal mortality decline which is consistent with their more limited access to hospital births. These results provide what is possibly the first illustration of the fact that fertility responses to a decline in the price of quality may not be

fully realized if there are caps on returns to quality. Even if segregation is history, children in developing countries often attend schools of poor quality, or grow up to find their skills under-rewarded on account of implicit (informal) ethnic or religious segregation in labor markets created, for instance, through networks formed along ethnic or religious lines.

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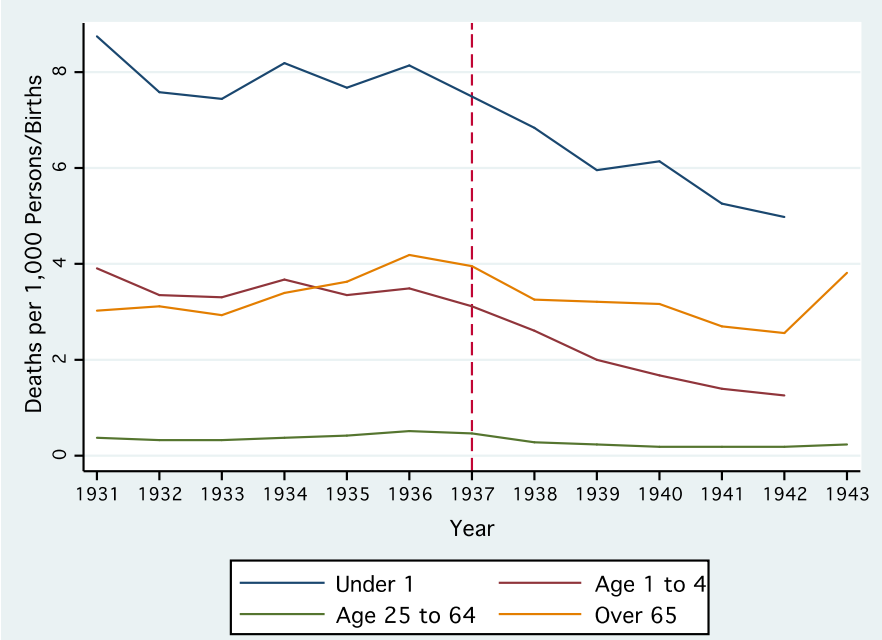
Figures

Figure 1 – National Trends in Pneumonia Mortality, 1930-1943

Panel A – All-age

Notes: Trends in all-age mortality from pneumonia plus influenza; see Data section of the paper. The pre-1937 fluctuations in the combined series are driven by influenza outbreaks (Jayachandran et al., 2010).

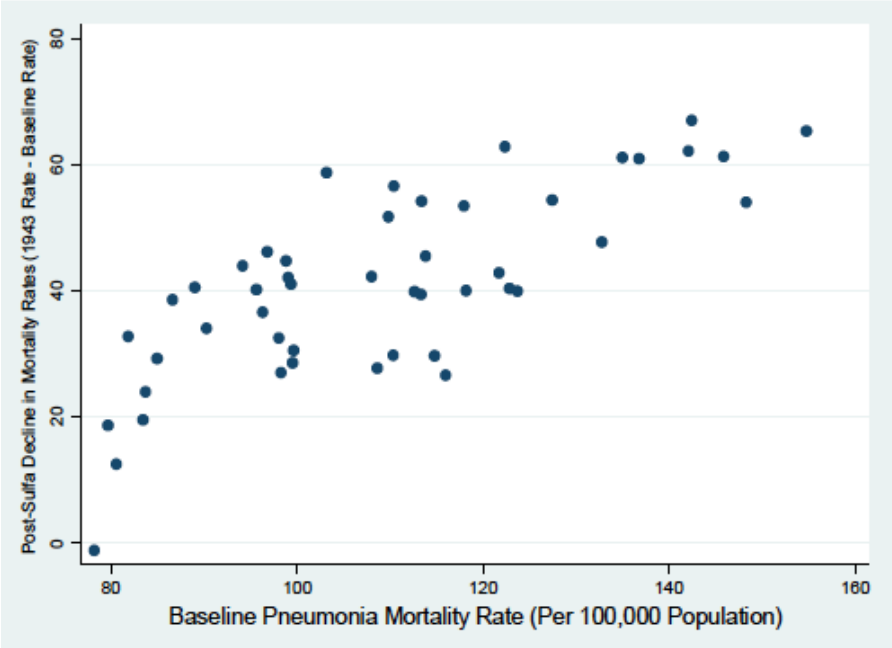
Panel B – Age specific pneumonia mortality rates



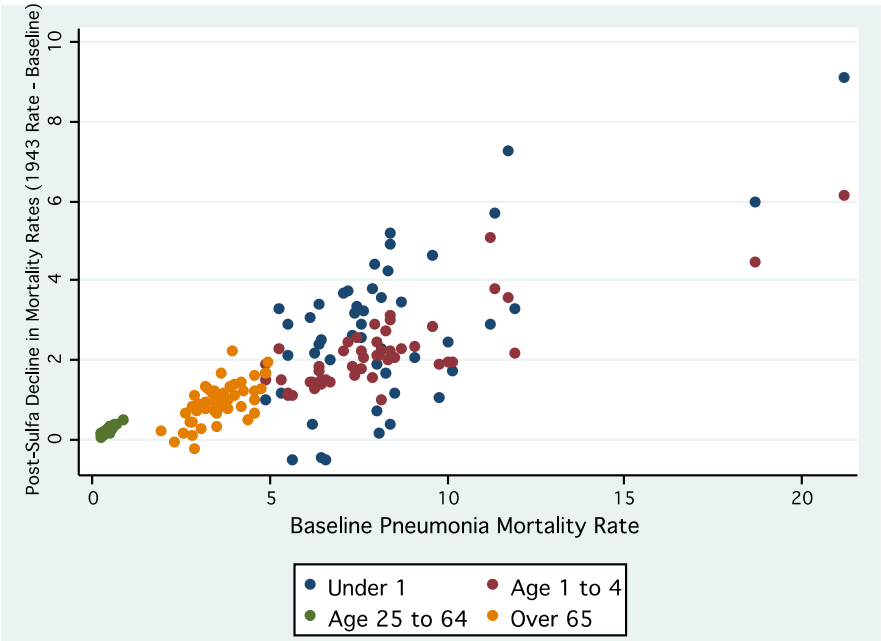
Source: US Vital Statistics.

Figure 2 – Convergence in Pneumonia Mortality Rates after 1937

Panel A – All age

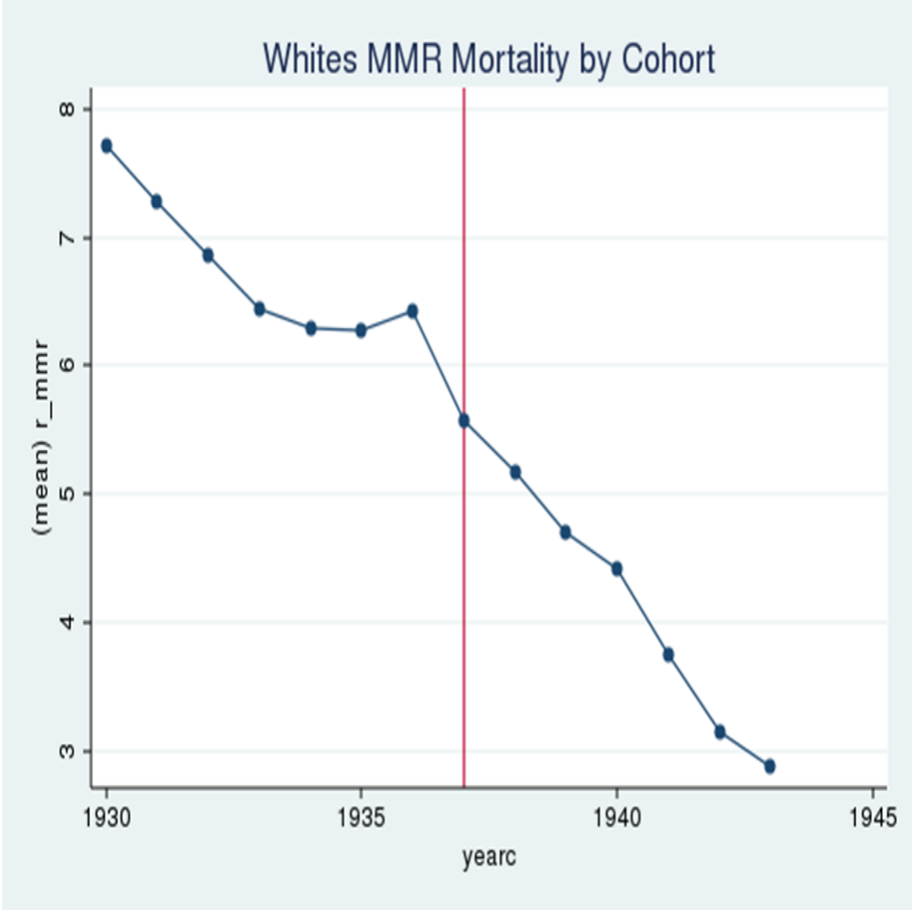


Panel B – Age Specific Rates



Notes: The base rate of pneumonia is its average over 1930-1936. Every dot is a state. Source: US Vital Statistics.

Figure 3 – National Trends in Maternal Mortality, 1930-1943



Tables

Table 1: Trend Breaks in Pneumonia and Maternal Mortality in 1937

	Levels			**Logs**		
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
	Pneumonia	TB	MMR	Pneumonia	TB	MMR
1930-1943						
Blacks	**-0.114*** **	0.0409***	-0.250***	-0.0814***	0.0172***	-0.0548***
	(0.0188)	(0.00957)	(0.0904)	(0.0101)	(0.0064)	(0.0102)
Whites	**-0.0773*** **	0.00513**	-0.235***	-0.0984***	-0.000346	-0.0963***
	(0.00962)	(0.0023)	(0.0351)	(0.00949)	(0.00507)	(0.00778)

Notes: Each dependent variable*time period*race cell represents a separate regression estimate of the coefficient on *post*year*. Each model controls for post (=1 if the year is 1937 or greater), year (1937 set to 0) and state fixed effects. Estimates for the total population are similar to the estimates for the white population as blacks constituted only about 10% of the population overall. The first column shows that the trend break for absolute pneumonia mortality is significantly larger for blacks than whites, but slightly smaller when using logged mortality. For comparison with Jayachandran et al. (2010), we show similar estimates for tuberculosis (TB, a control disease) and maternal mortality (MMR, a sulfa-treatable condition). We show these estimates in levels as well as logs and for the sample period used in Jayachandran et al., which is 1925-1943. While extending the sample period back to 1925 lowers the coefficients for blacks more than for whites (lower panel), the black advantage is preserved. The evolution of TB, included in the regression as a control disease in Jayachandran, et al (2010) differs starkly by race. For maternal mortality (MMR) we see a white advantage when log mortality is used, consistent with Jayachandran et al. For the purposes of the paper, the upshot is that there is no evidence that the sulfa-induced drop in pneumonia after 1937 was smaller for blacks.

Table 2: Probability of Birth

	1	2	3	4	5	6
post*base_pneumonia	-0.0116*	-0.0166***	-0.0283***	-0.0283***	-0.0173***	-0.0182***
	(0.006)	(0.006)	(0.007)	(0.006)	(0.006)	(0.007)
post*base_MMR			0.0027***	0.0027**	0.0028**	0.0028**
			(0.001)	(0.001)	(0.001)	(0.001)
(N = 4,104,599)						
(Mean Birth = 0.11, s.d. = 0.32)						
Controls						
Birth State and Year FE	Y	Y	Y	Y	Y	Y
Xist (time_since, order, age, race)	N	Y	Y	Y	Y	Y
Post*BaseRate(control diseases)	N	N	Y	Y	Y	Y
Birth State X Birth Year Variables	N	N	N	Y	Y	Y
Regional trends	N	N	N	N	Y	Y
Birth State trends	N	N	N	N	N	Y

Notes: *** = p<0.01, ** = p<0.05, * = p<0.10. Robust standard errors adjusted for clustering at the birth state level in parentheses. Set of controls is enlarged in moving from the first to sixth column where each estimate is from a separate regression. β_1 captures the -ve impact of sulfa-induced reductions in pneumonia exposure on probability of live birth in the hazard equation. The sample consists of US births to US-born women aged11-49 between 1930 and 1943 who reported being resident in same state at census as that of their birth. FE denotes fixed effects. *BaseRate(Control Diseases)* includes pre-sulfa birth state averages (1930-36)for maternal mortality MMR, and mortality from heart disease, cancer, under 2 diarrhea, malaria and tuberculosis. Birth State X Birth Year Variables include per capita log state income, state education expenditure, state health expenditure, school buildings, hospitals, and physicians, all by birth state and birth year. Region refers to census division region. N refers to the number of fertile-woman-years.

Table 3: Total Fertility

	(1)	(2)	(3)	(4)	(5)	(6)
*years of exposure to sulfa$_i$*base_pneumonia$_s$*	-0.0760***		-0.0637***	-0.0451***	-0.0451***	-0.139***
	(0.0164)		(0.0210)	(0.0158)	(0.0158)	(0.0500)
*years of exposure to sulfa$_i$*base_MMR$_s$*		-0.00820***	-0.00297	0.00533	0.00533	0.0208**
		(0.00227)	(0.00275)	(0.00388)	(0.00388)	(0.0102)
N=101,076						
Mean= 2.5 s.d.=2.2						
BaseMMR*age-group	N	Y	Y	Y	Y	Y
Base_other_diseae*age-group	N	N	N	Y	Y	Y
Base_SES	N	N	N	N	Y	Y
State*RaceFE, Cohort*RaceFE	Y	Y	Y	Y	Y	Y
Age-at-1937	Y	Y	Y	Y	Y	Y
State*Linear Trends	N	N	N	N	N	Y

Robust standard errors in parentheses, clustered by state
*** p<0.01, ** p<0.05, * p<0.1

Table 4: Extensive Margin for Completed Fertility

	(1)	(2)	(3)	(4)	5	6
*years of exposure to sulfa$_j$*base_pneumonia$_s$*	-0.000666		-0.00256	0.000939	0.000939	0.0317**
	(0.00207)		(0.00232)	(0.00278)	(0.00278)	(0.0131)
*years of exposure to sulfa$_j$*base_MMR$_s$*		0.000252	0.000465	-0.000506	-0.000506	-0.00312
		(0.000410)	(0.000467)	(0.000700)	(0.000700)	(0.00370)
BaseMMR*age-group	N	Y	Y	Y	Y	Y
Base_other_diseae*age-group	N	N	N	Y	Y	Y
Base_SES	N	N	N	N	Y	Y
State*RaceFE, Cohort*RaceFE	Y	Y	Y	Y	Y	Y
Age-at-1937	Y	Y	Y	Y	Y	Y
State*Linear Trends	N	N	N	N	N	Y

N=33,669, mean=0.54 s.d.=0.50

Robust standard errors in parentheses, clustered by state, *** p<0.01, ** p<0.05, *p<0.1

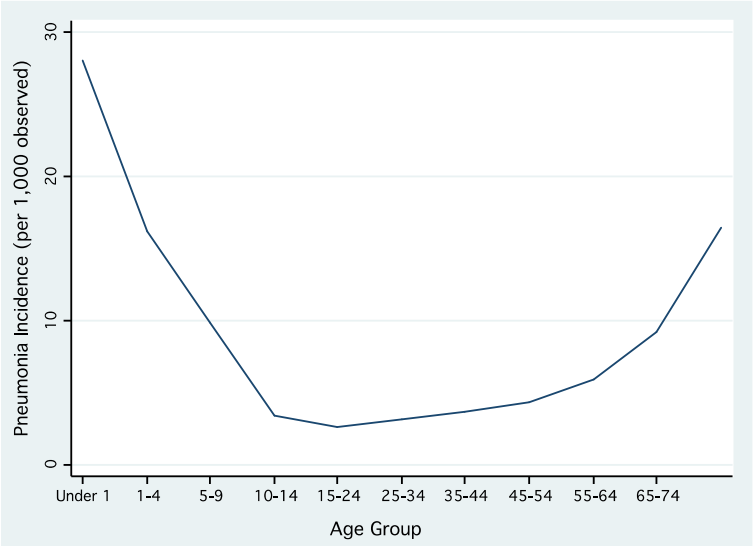
Table 5: Intensive Margin for Completed Fertility

	(1)	(2)	(3)	(4)	5
*years of exposure to sulfa$_i$*base_pneumonia$_s$*	-0.0758***		-0.0648***	-0.0498***	-0.0498***
	(0.0160)		(0.0202)	(0.0134)	(0.0134)
*years of exposure to sulfa$_i$*base_MMR$_s$*		-0.00796***	-0.00261	0.00653*	0.00653*
		(0.00250)	(0.00289)	(0.00352)	(0.00352)
controls					
BaseMMR*age-group	N	Y	Y	Y	Y
Base_other_diseae*age-group	N	N	N	Y	Y
Base_SES	N	N	N	N	Y
State*RaceFE, Cohort*RaceFE	Y	Y	Y	Y	Y
Age-at-1937	Y	Y	Y	Y	Y
State*Linear Trends	N	N	N	N	N

N=85,554, mean=3.1 s.d.=2.1
Robust standard errors in parentheses, clustered by state, *** p<0.01, ** p<0.05, * p<0.1

Appendix Figures and Tables

Figure 1 – Age Distribution of Pneumonia in the 1930s



Source: Britten (1942)

Figure 2 – Geographic Distribution of Pneumonia by Average Mortality Rates (1930-1936)

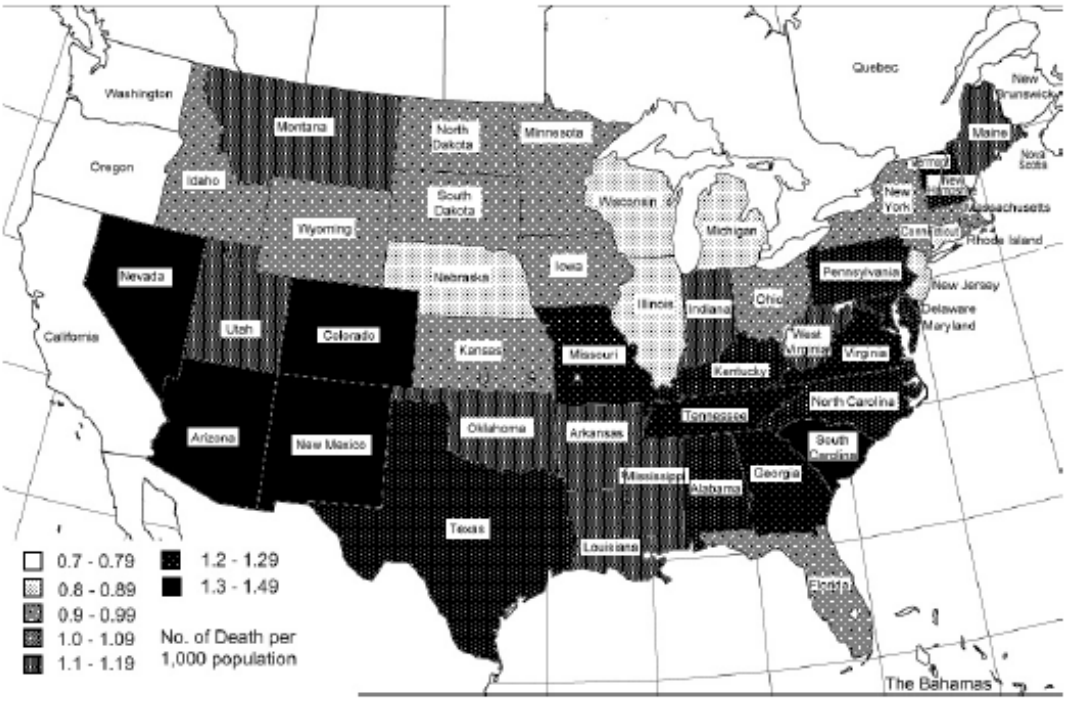
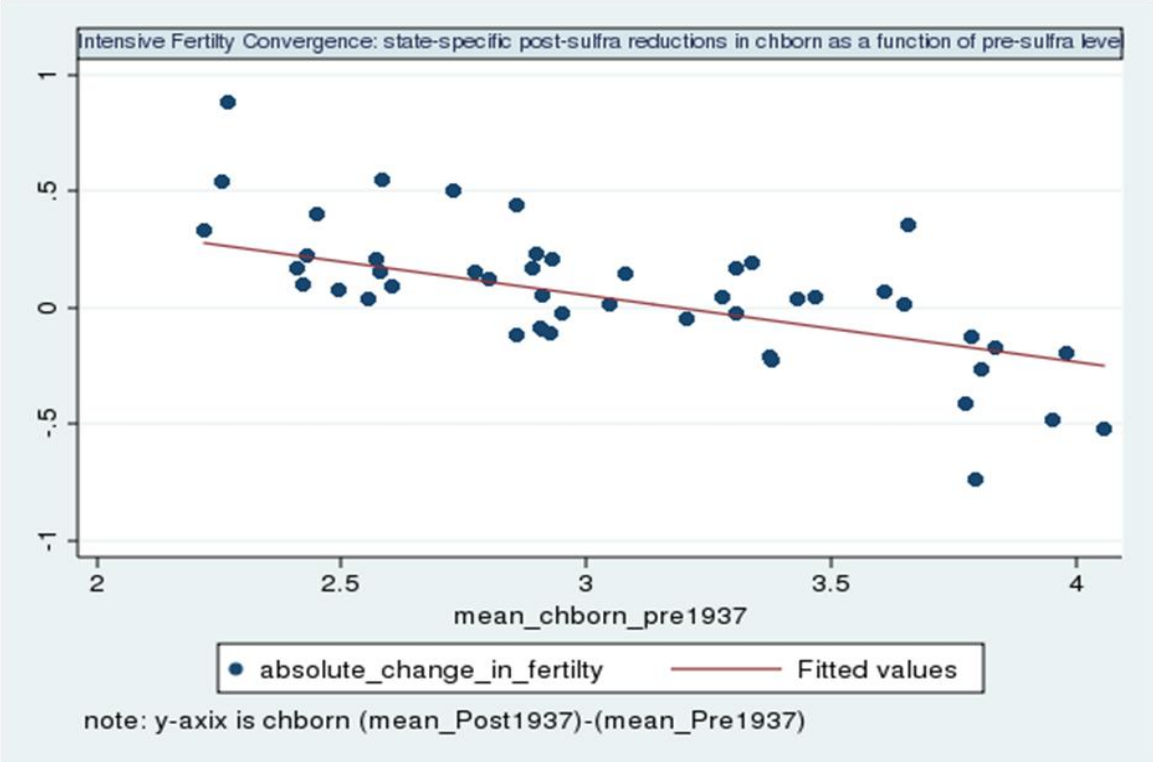


Figure 3 – Convergence in Fertility Across the US States Among Women of Reproductive Age in 1930-1943



Appendix Tables

Table 1: Descriptive Statistics

Birth State Baseline Mortality Rates (Per Thousand, N = 48 States)		Birth State X Birth Year Socioeconomic Variables (N = 669)	
Pneumonia	1.06 (0.19)	Log Income Per Capita	6.18 (0.50)
Tuberculosis	0.64 (0.37)	Log Hospitals Per 1,000	-2.83 (0.46)
Diarrhea	8.22 (5.64)	Log Physicians Per 1,000	0.14 (0.36)
Cancer	0.96 (0.31)	Log Schools Per 1,000	0.72 (0.65)
Heart Disease	2.09 (0.63)	Log Educational Spending Per Capita	4.27 (0.79)
Maternal mortality	6.35 (1.24)		
Malaria	0.032 (0.074)		

Notes: Figures provided are means with standard deviations in parentheses. The means for the census variables are based on the 2019930 men, 2137468 women, 1821471 white men, 198459 black men, 1897973 white women and 249495 black women born between 1930 and 1943 who are part of the 1980, 1990 and 2000 5% US census samples available from IPUMS-USA. Census family income figures have been converted to 2000 dollars. Baseline mortality rates reflect average mortality rates for each birth state over the period 1930-1936. Rates for pneumonia, tuberculosis, malaria, heart disease and cancer reflect deaths per 1,000 total population. Diarrhea and maternal mortality rates are per 1,000 live births.

Table 2 – Assessing Selective Migration after the Arrival of Sulfa Drugs

	(1)	(2)
Full Sample	0.100*	0.0308
	(0.0518)	(0.0713)
High School Educated Sample	-0.208	-0.127
	(0.142)	(0.201)

Notes: The dependent variable is the log of the population aged 20-40. Each cell contains the coefficient on *post*base_pneumonia* from a separate regression, holding constant *post*base_MMR*. Data are from the US Census Microdata from 1930 and 1940 and collapsed to the state*year level. Data for the year 1935 were gleaned from questions on state of residence 5 years before the enumeration in the 1940 census. There are 48 states, with N = 144. Column 1 contains only state and year fixed effects. Column 2 adds in controls for baseline income, baseline diarrheal mortality, and baseline heart disease mortality interacted with post-1937.