Is The Captain of the Men of Death Still At Play? Long-Run Impacts of Early Life Pneumonia Exposure and the Sulfa Drug Revolution

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Abstract - We exploit the introduction of sulfa drugs in 1937 to identify the impact of exposure to pneumonia in infancy on later life well-being and productivity in the United States. Using census data from 1980-2000, we find that cohorts born after the introduction of sulfa experienced increases in schooling, income, and the probability of employment, and reductions in disability rates. These improvements were larger for those born in states with higher pre-intervention pneumonia mortality rates, the areas that benefited most from the availability of sulfa drugs. Men and women show similar improvements on most indicators but the estimates for men are more persistently robust to the inclusion of birth state specific time trends. With the exception of cognitive disabilities for men and, in some specifications, work disability for men and family income, estimates for African Americans tend to be smaller and less precisely estimated than those for whites. Since African Americans exhibit larger absolute reductions in pneumonia mortality after the arrival of sulfa, we suggest that the absence of consistent discernible long run benefits may reflect barriers they encountered in translating improved endowments into gains in education and employment in the pre-Civil Rights Era.

Keywords: early childhood, infectious diseases, pneumonia, medical innovation, antibiotics, schooling, income, disability, mortality trends
JEL codes: I18, H41

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1. Introduction

Infections are the leading cause of child mortality in developing countries, as they were in developed nations until the middle of the 20th century (Black, Cousens, Johnson, Lawn, Rudan, Bassani et al., 2010; Cohen, 2000). In addition to causing disability and death in the short-run, infectious diseases in early childhood may also lead to worse health and socioeconomic outcomes later in life. Immune responses to infections claim nutritional resources which, in the young, tend to be diverted away from physical and mental development. In this way, severe or repeated infections could lead to long-term "scarring" expressed as poorer health and lower cognitive development in adulthood (Crimmins & Finch, 2006; Gluckman & Hanson, 2005). In addition, if there are dynamic complementarities in early age inputs, any particular damage in infancy may translate into multiplicative deficits later in childhood and into adulthood (Heckman, 2007). These processes suggest that wellbeing and productivity in today's population may, to a degree, have their roots in disease and treatment conditions that prevailed a generation or more ago.

This paper focuses upon pneumonia in early 20th century America, investigating whether birth year exposure had impacts on indicators of human capital that persist into adulthood and are discernible at the population level. In the 1930s, the disease accounted for 1 of every 10 deaths in the United States and was the leading cause of infant mortality in infancy (Linder & Grove, 1947; Wegman, 2001). In today's developing countries, pneumonia is similarly the dominant cause of child mortality (Bhutta, 2007; Black et al., 2010), accounting for 20 to 30 per cent of childhood deaths, two thirds of which occur in infancy (Bang et al. 1990, Kirkwood et al. 1995). We focus on birth year exposure in view of evidence that morbidity and mortality from pneumonia were both far more severe in infancy than at any other age (section 2 below). Moreover, infancy is a time when growth is rapid, nutritional demands are high, and developmental plasticity is greatest. For these reasons, infections in the birth year are most likely to create irreversible damage.

Identification of causal effects of early life infections on later life attainments is challenged by likely selectivity into infection. We overcome this problem by investigating exogenously determined declines in infectious disease exposure generated by the sudden and plausibly exogenous arrival of sulfonamide antibiotics (sulfa drugs) in the United States in 1937. These agents were the first clinically available antibiotics and they were quickly utilized

to treat a variety of infections leading to sharp decreases in both morbidity and mortality from pneumonia (Greengard, Raycraft, & Motel, 1943; Jayachandran, Lleras-Muney, & Smith, 2010; Lesch, 2007). Our study investigates whether this short run pattern is mirrored in longer run outcomes including education, income, employment and disability recorded in Unites States census data for 1980-2000. Our research strategy exploits variation across states in the pre-intervention level of pneumonia, across diseases in their treatability with sulfa drugs and also across age of exposure.

We find discontinuous improvements across the named outcomes for cohorts born post-sulfa. These improvements are increasing in the pre-intervention level of the pneumonia mortality rate, consistent with states with an initially higher burden of pneumonia benefiting more from the new antibiotics in terms of absolute reductions in pneumonia mortality (see Bleakley 2010). The estimates for both men and women are of similar size and, in each case, robust to including a vector of controls that allow a similar discontinuity in 1937 for a range of communicable and non- communicable diseases that were not responsive to sulfa. They similarly stand up to controls for sulfa-induced changes in maternal mortality that arose from control of puerpal sepsis, illustrated in Jayachandran and Lleras-Muney (2009). Adding in controls for income, education and health infrastructure in the birth state and birth year renders schooling and disability improvements for women insignificant and adding state specific trends to this already rich specification diminishes all of the coefficients for women, driving them to insignificance. The results for men are remarkably robust. In an extension we estimate a triple difference that exploits the steep gradient in infection risk between infancy and age fifteen, leading us to expect larger long run benefits for individuals who were infants when sulfa arrived than for individuals who were fifteen at the transition. Interestingly, while the estimates for men continue to hold steady, the estimates for women are strengthened. The coefficients for education, employment and income are now robust to state trends and cohort-region fixed effects.

The estimates for African-Americans are more imprecisely estimated and more sensitive to specification than for whites. There is nevertheless some evidence that disability reductions were larger amongst blacks. For black men, the coefficients for cognitive disability in the basic model and for work disability in the triple difference model are larger than for white men. In the models with controls for time-varying unobservables, the socioeconomic outcome coefficients are smaller for blacks. This is despite the fact that

blacks experienced larger absolute reductions in pneumonia mortality rates with the arrival of sulfa drugs. We propose that these findings may reflect more limited opportunities for blacks to translate improved health endowments into human capital and socioeconomic returns in the pre-Civil Rights Era. Pursuing this hypothesis further is an interesting area for future research.

We investigate and in general establish robustness of the reported findings to lifecycle effects on outcomes, mean reversion in outcomes, choice of sample window and race-specific rather than race-averaged state mortality series. We non-parametrically examine variation in age of exposure and this confirms that long run outcomes are most sensitive to pneumonia exposure in infancy. We argue that the estimated impacts are large (sections 5.1, 5.4).

Our research relates to a wider literature concerned with the origins of socio-economic inequality in early childhood. Much of this literature is focused upon parental attitudes and resources and their impact on the formation of cognitive and non-cognitive skills that ultimately determine education, employment and income (Flavio Cunha and James Heckman, 2009, James Heckman, 2007, Sandra Black and Paul Devereux, 2010). Our study contributes to an emerging strand that focuses upon the importance of early life health as a potential determinant of human capital development (Currie, 2007, Almond and Currie 2010). A few previous studies in this strand examine the long run impact of infectious diseases. (Almond, 2006) (Kelly, 2009) use the influenza pandemics of 1918 in the US and 1957 in the UK as natural experiments to identify long run human capital effects of *in utero* exposure.[1] (Bleakley, 2010; Cutler, Fung, Kremer, Singhal, & Vogl, 2010; Lucas, 2010; Venkataramani, 2010) utilize the introduction of large scale eradication campaigns to examine the long-run effects of childhood malaria exposure on earnings, schooling and cognition later in life.

This paper makes the following contributions to the literature. We provide the first estimates for pneumonia, which despite being the leading cause of childhood mortality in the developing world has drawn relatively little attention from researchers and policy makers. In contrast to diarrhea, which has shown substantial declines, there has been limited progress in

[1] In these pandemics, unusually, infection rates peaked amongst women of childbearing age. Influenza (and pneumonia) infections do not transmit across the placenta, so the identified impacts flow from the mother's contracting the infection and the consequent impoverishment of her nutritional reserves.

controlling pneumonia in the last two decades (Bhutta, 2007). The pandemic conditions and (near)-total eradication campaigns examined in previous studies are dramatic events that may have had different long-term effects than more subtle changes in the disease environment.[2] It is therefore unclear whether these findings can be applied to other policies or contexts. We focus on reductions in infectious disease morbidity and mortality rates arising from therapeutic measures rather than preventive measures. The long run evidence we offer is hence unique in complementing short run evidence of benefits of treatment of childhood pneumonia with antibiotics (Bang et al. 1990). Failure to recognize longer run benefits will tend to lead to sub-optimal levels of investment in drug treatments. More specifically, we provide what appear to be the first estimates of the long run returns to biomedical innovation. Hence our findings motivate investment in medical research and changes in extant patent laws and marketing structures to help promote the distribution of pharmaceuticals, such as antibiotics, at affordable prices in the developing world. On the prevention side, the long-run scarring effects noted in this study should increase interest in large scale roll-outs of pneumococcal vaccines, which have shown to be highly efficacious in reducing pneumonia incidence and mortality in developed and developing countries alike (Cutts, Zaman, Enwere, Jaffar, Levine, Okoko et al., 2005).

The rest of the paper is laid out as follows. Section 2 discusses the sulfa drug revolution and its effects on pneumonia mortality and morbidity in the United States. Section 3 sets out our research strategy, and Section 4 describes the data. Section 5 presents the results and Section 6 concludes.

2. Pneumonia and the Sulfa Drug Revolution

Pneumonia is an inflammatory disease of the lung that is most often caused by infectious agents such bacteria and viruses. In the 1930s, pneumonia accounted for 10% of all-age deaths in the United States and about 44% of neonatal deaths.[3] The underlying

[2] Influenza mortality increased four-fold during the flu epidemics analysed while the sulfa-induced decline in (infant) pneumonia mortality rates was 30%. So epidemic infection rates are some orders of magnitude larger than endemic rates and it is plausible that there are no effects on long run outcomes below some threshold level of infection.

[3] Young children tend to be more vulnerable to contracting and sustaining infections such as pneumonia because their immune systems are underdeveloped. The all cause neonatal mortality rate per 1000 births in 1930 was 3.6 and mortality from pneumonia was 1.6. By 1940 these rates had dropped to 2.4 and 1.1 respectively (US vital statistics).

incidence of pneumonia was high as well though this is more difficult to pin down because of patchy and unreliable data. Estimates from the US National Health Survey conducted in 1934-1936 show a nationwide morbidity rate of 3 cases per 100 infants, with rates potentially twice as high among the poor and those living in crowded conditions (Britten, 1942). However, it is likely that this represents a significant (at least two-fold) underestimate of the true pneumonia burden (Klugman & Feldman, 2009). Data from an outpatient population in North Carolina, a state that lay in the middle of the pneumonia mortality distribution in the 1930s, gathered during the mid-1980s suggests an incidence rate of nearly 10 per 100 children under the age of 1 year (Denny & Clyde, 1986). Overall the infection rate was probably about 6% on average and as much as 12% in more affected states and more affected groups. In today's developing countries it is estimated that there are 28 pneumonia cases per 100 children under the age of 4 each year (Rudan, Tomaskovic, Boschi-Pinto, & Campbell, 2004).

Prior to the arrival of sulfa drugs, pneumonia was primarily treated with supportive care. In the early 1930s, a small but growing number of clinicians began to use intravenous serum therapy to combat bacterial pneumonia, which was often more severe and conferred a higher risk of death than its viral counterpart (Lesch, 2007).[4] Serum therapy was an expensive and time-intensive process restricted to hospitalized patients. There was nevertheless some clinical evidence of its effectiveness in reducing pneumonia mortality rates (Finland, 1960).

The antibiotic properties of sulfonamides were first noted in 1932 by German chemists conducting experiments on textile dyes. Evidence of their anti-microbial potential was first published in 1935 and confirmed in clinical trials conducted in the following two years (Gibberd, 1937; Jayachandran et al., 2010; Kiefer, 2001; Lesch, 2007; Long & Bliss, 1937). A December 1936 New York Times article lauded the potential benefits of sulfa and, by early 1937, the drugs became widely available in the United States. They were relatively inexpensive and heavily promoted, leading to a "sulfa craze" that lasted until the arrival of the first penicillins in 1942-44.

[4] This involved obtaining antibodies from animals that were infected with a specific microbe. Serum refers to the component of animal blood comprised of fluid and antibodies, which has to be separated from the cellular component. The serum was injected intravenously into human patients, where the antibodies from the animal would bind to the infectious agent and aid the endogenous immune response (Lesch, 2007).

The arrival of sulfa drugs represented a tremendous boon to clinicians treating pneumonia. The first sulfa agents, such as Prontosil, were only somewhat effective against *Streptococcus pneumonaie*, the agent responsible for the majority of bacterial pneumonias. However, in 1938, sulfapyridine (also known as M&B 693), became available for clinical use. Clinical trials, conducted soon after sulfapyridine became available showed striking reductions in pneumonia case fatality rates among inpatients (Evans & Gaisford, 1938; Gaisford, 1939; Lesch, 2007). Within months after its introduction, the use of sulfapyridine for treating pneumonia became widespread.

In addition to the benefits noted in small clinical trials, the arrival of sulfa drugs had large impacts on mortality from pneumonia at the population level. Using state and national time series data for the United States, Jayachandran, et al (2010) demonstrate structural breaks in the time series data for mortality from sulfa treatable diseases around the time the drugs first became available. They attribute 17-32% of the post-1937 decline in pneumonia to the arrival of sulfa drugs.

Figure 1, which plots national pneumonia mortality rates between the years 1930 and 1943 illustrates these findings nicely. Prior to the arrival of sulfa, pneumonia rates held steady, suggesting that serotherapy had little impact on population level pneumonia mortality rates. As shown in the figure, the death rate began to drop in 1937 (though it still was higher than in the 1930-1935 period) and then fell sharply in 1938 and thereafter. This pattern is consistent with the arrival of sulfa drugs better suited to combat pneumococcal bacteria in 1938. *Figure 2* examines the post-sulfa absolute reduction in pneumonia mortality as a function of birth state pre-sulfa pneumonia mortality rates. This shows convergence in pneumonia mortality across states after 1937, confirming our prior that states with a greater initial burden of disease from pneumonia gained most from the arrival of sulfa drugs. As discussed in the next section, the patterns in *Figures 1* and *2* form the basis of our identification strategy.

Because our goal is to examine the long-run impacts of pneumonia exposure, it is important to explicitly examine the effects sulfa drugs on pneumonia *morbidity*, as well as mortality, at the population level. If the advent of sulfa only saved the lives of those with severe cases of pneumonia, our long-run impact estimates may simply reflect the effects of reduced mortality selection owing to the therapeutic intervention rather than the effects of scarring from the underlying disease. However if sulfa therapy (additionally) reduced the

severity and incidence of pneumonia episodes across the population, we would be better placed to investigate our hypothesis that childhood exposure to the disease scarred adulthood outcomes for survivors.

There is, in fact, compelling evidence to suggest that the advent of sulfa drugs led to reductions in the severity of pneumonia episodes. With respect to hospitalized patients, a number of clinical trials on infants and children from the era cite rapid improvements in fever, mental status and other physical examination findings, illustrating that the average inpatient case of pneumonia was shorter in duration and followed a less severe course as a result of sulfa chemotherapy (Greengard et al., 1943; Hodes, Stifler, Walker, McCarty, & Shirley, 1939; Moody & Knouf, 1940; Smith & Nemir, 1939). Data on industrial workers illustrate a 20-30% reduction in the number of illness days after the arrival of sulfa drugs (Ungerleider, Steinhaus, & Gubner, 1943). Along the same lines, comparisons of US Army experiences between the first and second World Wars also suggest that sulfa drugs were instrumental in drastically reducing the severity of and infirmity time from pneumonia among case soldiers (Lesch, 2007). Similar benefits were noted for outpatients, who accounted for around 70% of all pneumonia cases (Britten, 1942). Sulfa drugs were widely available to, and utilized by, laypersons and community physicians soon after their arrival in the US (Lesch, 2007). Thus, it is likely that pneumonias treated in the community would be less severe and shorter in duration than in the absence of antibiotic therapy and less likely to warrant hospitalization. This is relevant to analysis of race differences since it suggests that even if black people faced barriers to hospital access, they could access sulfa drugs.

Regarding the incidence of pneumonia, in theory it is possible that antibiotic therapy could reduce the probability of contracting pneumonia since those undergoing treatment may produce fewer respiratory secretions and therefore be less contagious. That said, incidence rates in the sample of industrial workers alluded to above did not appear to decline once sulfa-drugs became available (Ungerleider et al., 1943). However, as measures of pneumonia incidence are sensitive to improvements in surveillance and diagnosis, it is difficult to draw firm conclusions from the available data. As such, while we can state confidently that sulfa drugs led to noteworthy reductions in the length, severity and risk of death from pneumonia, we do not have reliable evidence on whether they led to reductions in the probability of contracting the infection altogether. In this regard, it is useful that we estimate population-averaged effects.

Since the arrival of sulfa drugs sits at the crux of our identification strategy to examine the long-run impacts of pneumonia, it is worth discussing why we are less concerned with the long-run impacts of other sulfa treatable conditions. Treatable diseases other than pneumonia for which consistent time series data are available and analyzed by Jayachandran et al (2010) are scarlet fever and maternal mortality from puerperal sepsis. These authors show that while the arrival of sulfa led to larger relative declines in these conditions, they accounted for only 0.2 and 1% respectively of total all-age mortality. In contrast, nearly 10% of all-age mortality was attributable to pneumonia. Moreover, in the pre-sulfa era, morbidity and mortality from pneumonia was concentrated among infants and, outside of congenital conditions, pneumonia was the leading cause of neonatal and infant death. Scarlet fever was at best a minor cause of morbidity or mortality in infants and is therefore not germane to an analysis of the long-run effects of early childhood disease.

However, the decline in maternal mortality induced by sulfa may well have influenced long-run outcomes for children born at the time, to allow for which we control for sulfa-induced reductions in maternal mortality as discussed in the next section. At the population level, parents may perceive greater returns to early life investments in girls when maternal mortality rates are lower, potentially leading to improvements in long-run outcomes for women vis-a-vis men (Jayachandran and Lleras-Muney 2009). At the individual level, changes in the risk of one's own mother dying and in family size may have influenced investments in both boys and girls. Family size impacts can arise because of changes in the probability of infertility, a potential complication of post-partum fever. Quantity-quality tradeoffs and/or sibling competition models imply reduced investments in the index birth, so diluting the impact of long-run gains from the other mechanisms. It is important to note that these effects are necessarily indirect because maternal post-partum infections are not transmitted to infants. As such, examining the effects of maternal mortality declines would provide little information on the long-term effects of infectious disease in early childhood.

3. Research Strategy

We utilize the plausibly exogenous availability of sulfa drugs, along with the fact that areas with higher mortality from pneumonia mortality rates experienced greater returns from the new therapies to identify the long-run effects of birth year exposure to pneumonia. The baseline model is

$$Y_{rstc} = a + \beta * post_t * base\_pneumonia_s + \delta_s + \zeta_t + \gamma_r + \mu_c + \theta_{rs} + \eta_{rt} + \lambda_{rc} + e_{rstc}$$

Y_{grstc} denotes the outcome of interest for individuals of gender (*g*), race (*r)*, birth state (*s*) and birth year (*t*) for whom outcomes are observed in census year (*c*). Since Jayachandran et al. (2010) test for structural breaks in the mortality series and identify 1937 as the break associated with sulfa, we define $post_t = 1$ for birth cohorts 1937 and after. The pre-sulfa pneumonia mortality rate in the birth state is denoted $base\_pneumonia_s$ and we use this to proxy for pre-sulfa pneumonia exposure. A similar strategy is employed in (Acemoglu & Johnson, 2007; Bleakley, 2007; Lucas, 2010). The Greek letters represent race-specific fixed effects for birth state, birth year and census year. The standard errors are clustered at the birth state level to account for serial correlation in the outcomes (Bertrand, Duflo, & Mullainathan, 2004) and regressions are weighted by cellsize[5].

The outcomes we initially examine are years of schooling (highest year of education completed), logged household income (total pre-tax income owned by a family unit), employment status and work limiting or preventing disability. We then extend the analysis to investigate cognitive and physical disability (and, forthcoming: personal income and poverty risk). We restrict our analysis to the time period 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.[6] Our use of multiple census years allows us to observe the same birth cohorts at different ages and so to identify cohort effects as distinct from life cycle effects. It also increases the precision of our estimates.

Unobserved heterogeneity at the birth state and birth year level

The main threat to inference with this sort of differences-in-differences model is the potential play of unobservables that vary by birth state and birth year and are correlated with both declines in pneumonia and the outcomes. To address this, we investigate sensitivity of the results to a variety of controls reflecting health and socioeconomic conditions in the birth state and birth year. For example, *post* may pick up coincident factors other than sulfa

[5] The substantive results remain unchanged even if cell-count weights are not used.

[6] Any direct impacts of World War II will be absorbed by the year dummies. We nevertheless examined and established robustness of the estimates to truncating the post-sulfa period at 1941, after which the US got involved in the war.

drug availability that led to sudden improvements in health. A powerful check against this is to use information on trends in diseases that were not treatable with sulfa drugs, so invoking what is loosely a third difference across cause of death. The premise is that the impact of other factors on mortality trends would not have discriminated between sulfa-treatable and sulfa-untreatable diseases. We interact *post* with the *base* (pre-sulfa) level of birth state specific mortality rates from tuberculosis, under-2 diarrhea, heart disease and cancer. The communicable diseases help control for state specific changes in sanitation, public health programs, housing (etc) that may have coincided with the arrival of sulfa. The inclusion of non-communicable diseases is expected to further control for factors such as health care quality and access. In exploiting information on diseases that were and were not treatable with sulfa drugs, we mirror in our state level analysis of long run outcomes the approach taken by Seema Jayachandran et al. (2010) in their aggregate analysis of short run changes in mortality rates. We add to the "untreated" diseases in their sample diarrhea mortality as this is available for the under-2 population and our focus is on early life exposure to infection. In other words, we expect diarrheal mortality rates to help capture unobserved improvements in conditions specific to young children.

Importantly, we also control for the pre-sulfa maternal mortality rate interacted with *post*. For reasons discussed in the previous section, this helps us isolate the impact of improved treatability of pneumonia. In addition to the vector of control disease mortality rates at the birth state-birth year level, we directly control for trends in birth state-birth year varying socioeconomic variables including logged state income per capita and the numbers of schools, hospitals and physicians per capita. To allow for any remaining pre-existing differences in trends we investigate specifications with birth state specific linear (and quadratic) time trends and census region* birth year fixed effects.

The richer specifications may amount to "over-controlling." For example, increased risk of infections from weakened immune systems as well as competing risks from different conditions create population level correlations in disease rates. Thus, controlling for additional diseases may capture variation in disease trajectories that are in fact driven by the use of sulfa drugs rather than by unobserved confounding factors. As such, it is possible that the inclusion of such controls may produce estimates that in some sense obscure the true returns to reductions in pneumonia rates resulting from therapeutic innovation. Similarly,

controlling for state trends is typically quite demanding of the data. We therefore present results with and without each set of controls.

Other robustness checks detailed in the results section involve controlling for mean reversion in the outcome variables, testing sensitivity to the end points of the sample window and checking for whether lifecycle effects may be loading onto the estimated cohort effects.

Age of exposure

In an extension of the baseline (double difference) specification, we allow for returns to sulfa exposure at other ages in childhood. In contrast to Almond (2006) and Kelly (2009) we do not expect impacts from foetal exposure because mothers of childbearing age experienced very low infection rates (Britten 1942). However, even if vulnerability to contracting pneumonia declined after age one, it was considerable at age 1-5. To investigate this, we replace *post* with a vector of birth year dummies for every year in the sample. We graph the resulting coefficients and test for a sulfa-related break. This specification also provides a useful falsification test: the presence of breaks in the coefficients in years other than around 1937 may suggest that the patterns seen in the data may be due to some process other than the introduction of sulfa drugs. Similarly, because the first sulfa drugs were less effective for treating pneumonia than sulfapyridine, which was introduced in 1938 and became widely used by 1939, the sulfa effects, if there are any, should increase in magnitude over the period 1937-1939. We assess this with the coefficient plots.

The plots confirm the gradient in long run impacts by age. This is consistent with the age profile of pneumonia morbidity and mortality. Infection rates at birth in the pre-sulfa era (1934-36) were eight to ten times as large as at age 15. The average number of days of disability suffered by patients with pneumonia peaked in childhood and reached a minimum in the age group 15-24 (Collins 1931, Britten 1942: Table 2). Consequently we expect limited if any long run benefits of sulfa to be evident for individuals who were exposed at age 15 compared with individuals exposed at birth. We use this to estimate a specification with a triple difference.

Gender and race heterogeneity

We consistently allow treatment heterogeneity by race and by race-gender. Males may have benefitted more from early life exposure to sulfa for biological reasons. Surveillance data from the mid-1930s show that they were 25% more likely to contract pneumonia than

females as infants (Britten, 1942). Males also tend to be more vulnerable to disease and early childhood shocks (Gluckman & Hanson, 2005; Low, 2000; Waldron, 1983) and so may incur more scarring from a given level of disease. The estimated impacts may however have been moderated or exaggerated by gender-specificity in the sensitivity of parental investments to health endowments. There is some evidence of a pro-male bias in contemporary times in the United States (Dahl & Moretti, 2008; Stanley & Jarell, 1998), which leads us to expect a stronger association between endowments and investments for females.

The pre-sulfa black pneumonia mortality was about twice as high as that for whites (US Vital Statistics). This alone suggests black people will have benefited more from the arrival of sulfa. However this may be turned around if they either had more limited access to sulfa drugs (Seema Jayachandran et al. 2010, Doug Almond, Ken Chay and Michael Greenstone 2006) or if they had more limited opportunities to translate their improved endowments into improved socioeconomic status.

4. Data

Data for the outcome variables were taken from the 1980, 1990, and 2000 5% samples of the United States Census (see Data Appendix for further details). The marginal sulfa cohort (i.e., those born in 1937) were 43, 53, and 63 years old at the time of each of these enumerations, respectively. We calculated means for each outcome by birth state, birth year, census year, race and gender and used the cell means in the regression analysis[7]. We estimated the equations by census year and found little evidence of lifecycle effects: estimates of pre/post sulfa cohort differences in outcomes were robust to observing the (marginal) cohorts at different ages. In addition, if sulfa exposed individuals who achieved fewer years of education were also likely to die later, then including data from later censuses may create a downward bias in the estimated impact of sulfa drugs. We find no significant evidence of this. For schooling, we similarly confirm that the estimates are very similar if we include the three census years but the estimates we report are based upon data from the 1980 census because the education variable is coded **********. We investigate indicators of physical and cognitive disability which are only available in the 2000 census. To avoid measurement

[7] The use of micro-level data produced very similar point estimates and standard errors. These results are available in the online appendix.

error, we dropped cells in the bottom 1% of the cohort size distribution (i.e., those with less than 50 persons).[8]

Data on all-age disease-specific mortality rates (expressed per 1,000 people) are from the US Vital Statistics and were gathered by Grant Miller, Adriana Lleras-Muney and Anne Case and supplemented by us. Information on the socioeconomic characteristics of states come from sources detailed in the Data Appendix. For the period of interest, state-level time series data on pneumonia mortality are aggregated with influenza mortality, so we (like Jayachandran et al 2010) work with this compound variable. Combining mortality rates from these causes may lower measurement error, given that surveillance systems may have conflated influenza and pneumonia deaths and a large portion of influenza deaths came from secondary bacterial pneumonia. However influenza, being viral, was not responsive to sulfa drugs, while many types of pneumonia were. The availability of separate influenza and pneumonia mortality rates for certain years allows us to discern the contribution of pneumonia to the compound variable. Pneumonia dominated, accounting for 75% of all-age deaths (Jayachandran et al. 2010) and 89% of neonatal deaths (our estimates from the Vital Statistics) in the influenza plus pneumonia category. More importantly, there was little change in the influenza death rate between 1930 and 1940, suggesting that the reduction in mortality rates from both causes during in the period of interest was driven primarily by reductions in pneumonia mortality.[9] *Table 1* provides descriptive statistics for each of the study variables.

5. Results

We first discuss estimates for schooling, family income, employment and work-related disability. We then examine additional indicators of disability (and, forthcoming, income). The third section presents race-specific results and the fourth discusses findings from additional falsification tests. All estimates are presented by gender.

5.1. Main Results

[8] The results are unchanged even if these cells are included in the analysis.

[9] Between 1930 and 1940 neonatal mortality from pneumonia fell from 1.6. to 1.1 per 1000 while neonatal mortality from influenza remained constant at 0.2 per 1000 (US Vital Statistics).

Refer *Table 2* where each cell reports estimates of the coefficient on $post_t$*$base\_pneumonia_s$ from a separate regression. The rows denote the outcome variables and the columns different sets of control variables. We report estimates with a growing set of controls. We find positive and statistically significant impacts of exposure to sulfa on years of schooling for men. This result is remarkably robust to successive inclusion of *post*base* for mortality rates from the control diseases (which include maternal mortality), state socioeconomic and infrastructure characteristics, state specific linear time trends, and census region times birth year fixed effects. The specification with all of these controls implies a post-sulfa gain of 0.19 in years of schooling associated with shifting the pre-sulfa pneumonia death rate from the 75th to the 25th percentile of its distribution (i.e., from 1.18 to 0.92 deaths per 1,000).[10]

Men also exhibit post-sulfa improvements in the other indicators. The post-sulfa improvement flowing from a change in the pre-sulfa pneumonia death rate from the 75th to the 25th percentile of its distribution is 2.8% for family income, 0.7 percentage points (up) in the probability of being employed, and 0.62 percentage point (down) in the probability of reporting a disability limiting or preventing work. These estimates are all from column (5), the specification with the most comprehensive set of controls. For schooling, income and disability, we find effects of a similar magnitude for women in the specifications with birth state and birth year fixed effects and control diseases (columns 1 and 2). These estimates are diminished and driven to insignificance by the inclusion of state socioeconomic variables and state specific trends. However, as we shall see below, once we invoke a third difference in the specification, women exhibit improvements in schooling, employment and income that are robust to the richest set of controls.

Figures 3 and *4* present plots of the coefficients on *birth year*base*. The coefficients for men show trend breaks around 1937, consistent with the introduction of sulfa drugs. Interestingly, for most of the variables, the effect sizes climb in magnitude over the period

[10] We do not find compelling evidence of a relationship between exposure to lower maternal mortality rates and long-run outcomes. For men, the estimates on *post*base_MMR* are negative for schooling, income and employment, and positive for disability. These estimates are not robust to specification. For women, the estimates are small, positive, and insignificant for all four of these outcomes. The results suggest that either reductions in maternal mortality did not change parental investments in a way that led to long-run changes in health and socioeconomic status, or that the investment effects of maternal mortality declines through the pathways discussed earlier in the text offset each other.

1937-1939, which is consistent with the fact that the earliest sulfa drugs were less effective in treating pneumonia than sulfapyridine, which was available in 1938 and became widely used by 1939. In line with the results from *Table 2*, the sharp sulfa trend breaks are either more muted or non-evident for the estimates for women[11]. There is no evidence of trend breaks in years other than those associated with the introduction of sulfa. In particular, the coefficients for men prior to 1937 hover around zero indicating that the returns to sulfa exposure were likely limited to the birth year.[12]

5.2. Additional Disability Variables

We have so far analyzed self-reported work-related disability. This measure will carry "justification bias" if the welfare regime encourages individuals to invent or exaggerate disability (Autor & Duggan, 2003). While the importance of justification bias is contentious, we investigate additional measures of disability that are unrelated to work participation and so may serve as cleaner measures of health. These are indicators for whether individuals reported experiencing difficulties with basic physical and cognitive tasks because of physical or mental conditions, available only in the 2000 census.

The results are in *Table 3*. For men, columns (1) and (2) show negative estimates for cognitive and physical disability, with coefficients for the former being statistically significant. For cognitive difficulty, the estimated magnitudes are largest in magnitude for the specifications with birth state specific time trends: a move from the 75th to 25th percentile of the pre-sulfa baseline pneumonia mortality distribution is associated with a 0.71 percentage point post-sulfa decrease in the probability of reporting a cognitive disability. There are no significant sulfa impacts on the risk of physical disabilities. We are unable to detect any lowering of cognitive or physical disability amongst women on account of sulfa. Rather, for

[11] Indeed, the generally uptrending coefficients and the lack of a sharp trend break in 1937 is consistent with our finding large, significant coefficients in the specifications with fewer controls and with these effects being obliterated with the inclusion of birth state-specific time trends. As discussed in section 3, it is unclear whether the estimates with or without trends are more reliable. However comparing Tables 2 and 7 suggests sustained and robust improvements for women.

[12] This is consistent with pre-sulfa pneumonia mortality and morbidity rates being higher by a large margin amongst infants (Britten 1942). The age distribution of influenza and pneumonia during the 1918 influenza epidemic appears to have been unusual (e.g. Collins 1931) and is in any case different from that in endemic cases. While endemic infection is concentrated at the two tails of the age distribution, the 1918 epidemic infection rates peaked amongst women of child-bearing age. As a result, the long run effects identified in Almond (2006) arise from foetal exposure to maternal infection; there is no evidence of long run impacts associated with birth year exposure.

physically disability, the specifications with birth state specific linear time trends actually suggest positive effects[13].

5.3. Race Specific Effects

Refer *Tables 4-6*. The results for white men and women mirror those for all men and all women. It is noteworthy that the coefficients for black and white men and women in Tables 4 and 5 are similar (and insignificantly different from one another) in column 1.

However the coefficients for the black population are imprecisely estimated and tend to become smaller upon inclusion of controls from column 2 onwards. Some specifications suggest stronger impacts on income for black people. It may be that the black population accessed sulfa later than the white population, in which case "post" may need to be redefined for the former. To investigate this possibility we estimated the more general model in which we do not impose the definition of post but allow "base" to take year-specific coefficients (coefficient plots for blacks corresponding to Figures 3 and 4 are available in the online appendix). We find no evidence of a trend break in outcomes for black men and women in either 1937 or a subsequent date.

Table 6 presents results for the additional disability variables. Black men experienced larger reductions in cognitive disability from sulfa drug exposure than white men and this finding is fairly robust to specification. Moving from the 75th to the 25th percentile of the baseline pneumonia mortality distribution is associated with a 1.03-1.45 percentage point decrease in the probability that black men report a cognitive disability. Exposure to sulfa also had larger impacts on physical disability for blacks, though this is attenuated with the inclusion of additional controls and trends. The results for black women are similar to results for white women.

A reason that the estimates for blacks are so imprecisely determined may be that the state-level pneumonia mortality rate is a poor measure of pneumonia mortality for blacks since they are, on average, 10% of the population. If the ratio of white to black mortality rates were constant over time then this would not be so much of an issue. However it is plausible that the ratio varies. We therefore re-estimated the model utilizing race-specific

[13] It is possible that the positive coefficients that emerge with stronger controls in these specifications, and perhaps those reported in earlier tables, are a spurious artifact of over-controlling for unobservables. However, we are unable to establish this.

state mortality rates (these data combine all non-white races). Since some states have very small populations and this increases the potential for measurement error, we followed Jayachandran, et al (2010) and only examined data for those states in which blacks were more than 10% of the population and we further limited the sample to states with non-white populations greater than 100,000 individuals. Using these data, however, does little to alter our substantive conclusions[14].

5.4. Additional Robustness Checks

We conducted several additional falsification tests to address the robustness of our results. The first set of checks involved controlling for mean reverting shocks. Our concern here is that some negative shock in the years preceding the arrival of sulfa reduced the human capital attainment of the affected cohorts. With the resolution of the shock, these outcomes reverted back to the mean thus producing what we may mistakenly be interpreting as a sulfa effect. In order to control for mean reversion, we use the strategy employed in Bleakley (2007) and include controls for *post* interacted with the average value of the outcome of interest in each birth state for the pre-sulfa cohorts. The results, shown in *Table 7*, suggest that the inclusion of controls for mean reversion does little to alter our substantive conclusions[15].

We estimate "triple-difference" models as an additional test. As explained in section 3, the idea here is to exploit the epidemiology of pneumonia. Differencing across age of exposure with respect to an intervention (sulfa) at a fixed time will remove any unobservables that influenced human capital acquisition for the post-sulfa cohort. We used cohorts born between 1915-1927 as controls for the treated ***1937***-1943 cohorts examined above. We divided the control cohorts into those who turned 15 during the sulfa drug era (that is, *post* = 1 for cohorts born between 1922-1927) and those who were this age before (*post* = 0 for the 1915-1921 birth cohorts).[16]

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[14] These estimates are in the online appendix. The race-specific mortality data are likely to be flawed to the extent that mortality reporting for non-whites was more prone to underreporting, misclassification or other measurement bias. However any such reporting bias will transmit to the race-averaged data used in the main analysis.

[15] While the coefficient on (all) men's education drops appreciably with the inclusion of the mean reversion controls, the estimates that distinguish white and black men are robust.

[16] We thank Tania Barham for suggesting this.

The results of the "triple difference" analysis are presented in *Table 8*. These are estimates with the richest set of controls, corresponding to column 5 in Table 2. For men, the estimates are remarkably similar in magnitude and significance to those in *Table 2*. Interestingly, the results for women now show large positive estimates for schooling, family income and probability of employment, all of which are significant. We also now find large impacts for all four outcomes of interest for black men, though only the work disability estimate is statistically significant. Overall, results for white men are robust to this extension and results for white women and blacks improve.

Finally we summarise a number of other checks we conducted; results are available on request. If we define *post* at 1936, we find almost no significant impacts. If we define it at 1938 then, consistent with diffusion from 1937, we estimate similar and slightly larger coefficients. The coefficients are in general robust to truncating the sample window at 1941 (war and penicillin) and also to using a narrower window, starting 1934. There is some concern in the literature that the 2000 census microdata sample used in this paper may be subject to inaccuracies in age reporting (Alexander, Davern, & Stevenson, 2010). While this problem primarily pertains to those over the age of 65, all of whom were born at least two years prior to the start of the sulfa era, we still assessed whether our results remained the same if the 2000 census was excluded. We indeed find that the substantive results are unchanged (results not shown here).

5.5. Discussion

Heterogeneity in impacts by gender and race

Section 3 set out reasons that we may expect gender and race heterogeneity. Here we consider our findings in view of the priors set out earlier. The baseline estimates (Table 2) suggest stronger impacts for men for all outcomes. The triple difference estimates in Table 8 we find significant but smaller gains in schooling and income for women. We identify larger absolute gains in employment for women but these are probably related to more contemporary changes in gender-specific returns to education and marriage. Thus the findings suggest that men benefited more from early life exposure to sulfa than women, consistent with the biological hypotheses set out in section 3 above, in particular with the fact that they face a (25%) higher probability of contracting pneumonia in infancy.

By the same token, since blacks faced a (100%) higher risk of contracting pneumonia than whites, we may have expected to see larger gains amongst them. But we don't. Jayachandran, et al (2010) provide evidence that blacks benefited less from sulfa drugs in terms of percentage declines in mortality from pneumonia and other sulfa-treatable diseases. However, (a) they still did benefit and (b) the *absolute* decline in pneumonia mortality rates was in fact larger for blacks. Both of these patterns are shown in *Figure 5*. The evidence that blacks drew a *short run* benefit from sulfa suggests that they did access sulfa drugs – despite being poorer and facing weaker access to hospitals and prescriptions. This ties in with the evidence presented in section 2 that sulfa drugs were widely used for outpatient care. Overall, the idea of barriers to sulfa uptake cannot on its own explain our failure to identify a *long run* benefit for blacks.

An explanation that suggests itself is that blacks encountered barriers to translating improved health endowments in infancy into human capital and socioeconomic returns in later life. For example, it is well known that differentials in wages, school quality and returns to education between blacks and whites were more marked prior to the Civil Rights Act of 1965 (Donohue & Heckman, 1991). Limited access to quality schools and training institutions alongside a discriminatory labor market may have depressed expected returns to human capital investment to the point that they were not been deemed worthwhile. This hypothesis is supported by the fact that the estimates for reductions in cognitive disability post-sulfa are larger in magnitude for black vis-à-vis white men (as are reductions in work disability in the specification in Table 8) while gains in schooling and employment are smaller.[17].

Selection, participation and size of effects

In this section we discuss a number of reasons why the estimated magnitudes of the long run impacts discussed earlier are likely to under-state the positive impacts of sulfa drugs. First, the estimates describe averages across the entire population. To assess the long-run returns of sulfa drugs accruing to those who actually contracted the disease, we would

[17] Another piece of supporting evidence, though more tenuous, is that the estimated income gains for black men and women are larger than those for whites in several of our specifications. This suggests that blacks may have been able to use sulfa-driven health or cognitive endowments in the marketplace to secure higher earnings as they arrived on the labour market in the post-Civil Rights Act era, although on margins that did not involve returns to schooling, their schooling having been determined prior to the Civil Rights Act.

have to divide our estimates by the fraction of infants with pneumonia. As discussed in section 2, infection or prevalence rates are difficult to pin down due to the paucity of and limitations in surveillance data from the pre-sulfa drug era. However, if we take a case rate of 25 per 100 child-years to approximate the situation in today's developing countries, our results for males suggest a 0.41-0.77 increase in years of schooling, a 4.1-11.1% increase in family income, a 1.63-2.79% increase in the probability of employment, and a 2.49-3.03% decrease in the probability of reporting a work preventing disability for individuals who actually had pneumonia as a result of the arrival of sulfa. Clearly, this is a rough calculation: it assumes that sulfa drugs only led to reductions in the severity of pneumonia without affecting the incidence rate or the distribution of who actually contracted the disease; it also assumes no within-cohort spillover effects from those afflicted by pneumonia to those that were not.

The previous discussion concerns the infection rate in the population. Conditional upon infection, access to and use of sulfa drugs to treat pneumonia was probably not universal. In other words, we recover an intent-to-treat effect which will be smaller than the average effect of the treatment on the treated. The cost of a complete course was $28-$100 (in 2008 US $) or $4.3 per patient per day. While seemingly inexpensive, recurrent or lengthy bouts of infection may have conferred non-trivial costs for the poor. Moreover, some people may have elected not to use sulfa as it was a new drug, the side effects of which were initially unknown and once known, unpalatable.[18]

Mortality selection will tend to bias our estimates towards zero since frailer children were probably more likely to succumb to pneumonia than their healthier counterparts. With the advent of sulfa, more of these children would survive past childhood and these innately less health individuals are likely to be less productive and healthy as adults (Bozzoli, Deaton, & Quintana-Domeque, 2009). That said, we expect this bias to be small since pre-intervention death rates amongst infants were around 1% (Britten, 1942; Councell, 1963). A

[18] Sulfonamides have the potential to cause a variety of untoward reactions, including urinary tract disorders, haemopoietic disorders, porphyria and hypersensitivity reactions. When used in large doses, they may cause a strong allergic reaction, e.g. Stevens Johnson syndrome and toxic epidermal necrolysis (also known as Lyell syndrome). It is estimated that about 3% of the general population have adverse reactions when treated with sulfonamide antimicrobials (Wikipedia).

less recognized mechanism running from changes in the mortality environment in early life to later life attainments operates within family. To the extent that the index individual is less likely to have lost a sibling to pneumonia mortality, parental investments in the index child are likely to be diluted and this again suggests we will under-estimate the individual gain from sulfa.

6. Conclusions

This study provides the first evidence of long-term scarring effects of early life exposure to (endemic) pneumonia, effects that we show are not only discernible at the population level but large. It also offers some of the first estimates of the long-run socioeconomic and health returns to medical technology, in particular antibiotics. We identify robust impacts for white men of sulfa-induced reductions in pneumonia risk in infancy on adulthood indicators of education, employment, income and work-related disability risk. White women exhibit some impressive improvements in socioeconomic outcomes that are sensitive to state trends in a double difference but robust in a triple difference specification. Estimates for African-Americans are poorly determined, making it hard to rest a firm story on them. While coefficients for socioeconomic outcomes are smaller, there is some evidence of a substantial reduction in the risk of cognitive disability and work disability for black men born after the arrival of sulfa drugs as well as some evidence of income gains for black men and women. In our future work we propose to investigate a richer set of outcomes for the first generation as well as second generation outcomes.

These results are important for developing countries where acute respiratory infections are the leading cause of early childhood mortality. They point to the need for greater research and policy focus on the causes and consequences of early life pneumonia, as well as strategies to prevent and treat this disease in these areas. They indicate that medical technology, especially innovations targeting infants, may generate returns throughout the life cycle. A recent community-based intervention in India that involved treatment of pneumonia in children aged 0-4 years with co-trimoxazole showed a case-fatality rate of 0.8% in the treatment area compared with 13.5% in the control area. The cost of co-trimoxazole was US $0.025 per child per year or $2.64 per child saved (Bang et al. 1990). Evidence of this nature is still scarce and useful. However the literature fails to account for

the longer-run returns to antibiotic treatment that work through lowering the duration and severity of illness in the population that survives pneumonia.

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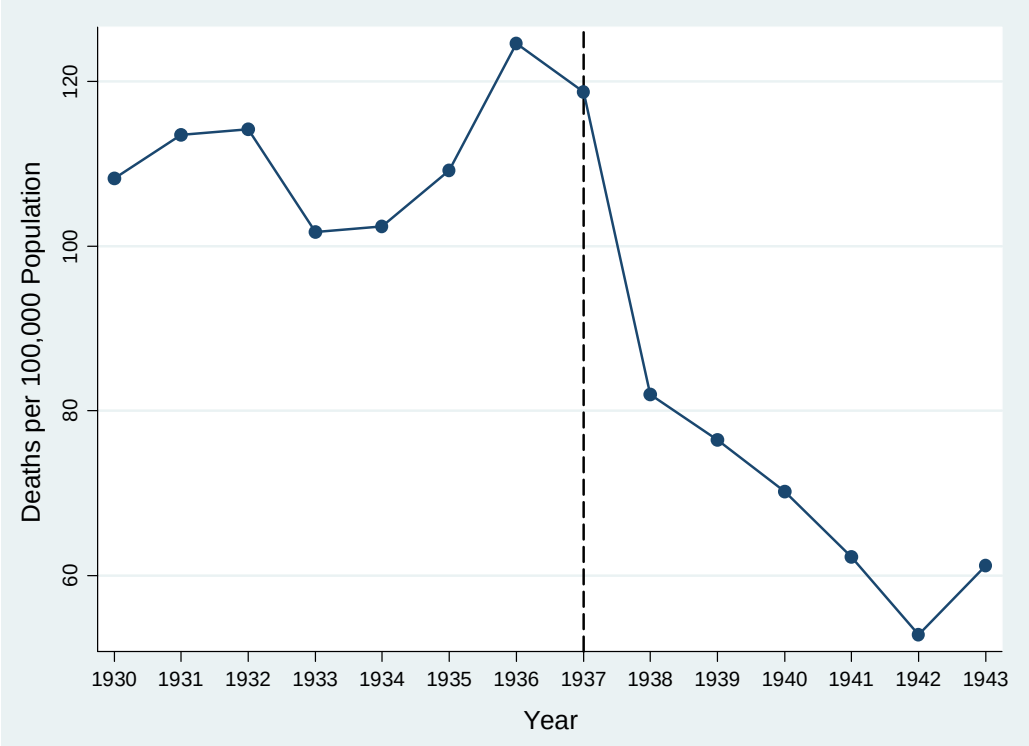
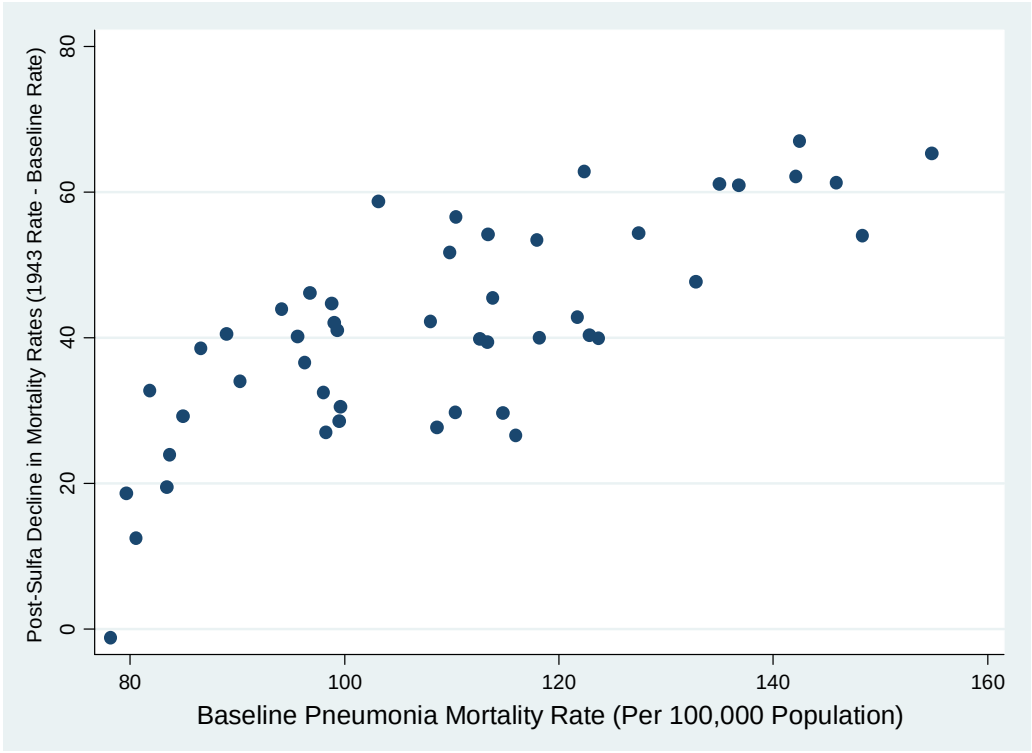
Figure 1 – Mortality from Pneumonia, United States, 1930-1943



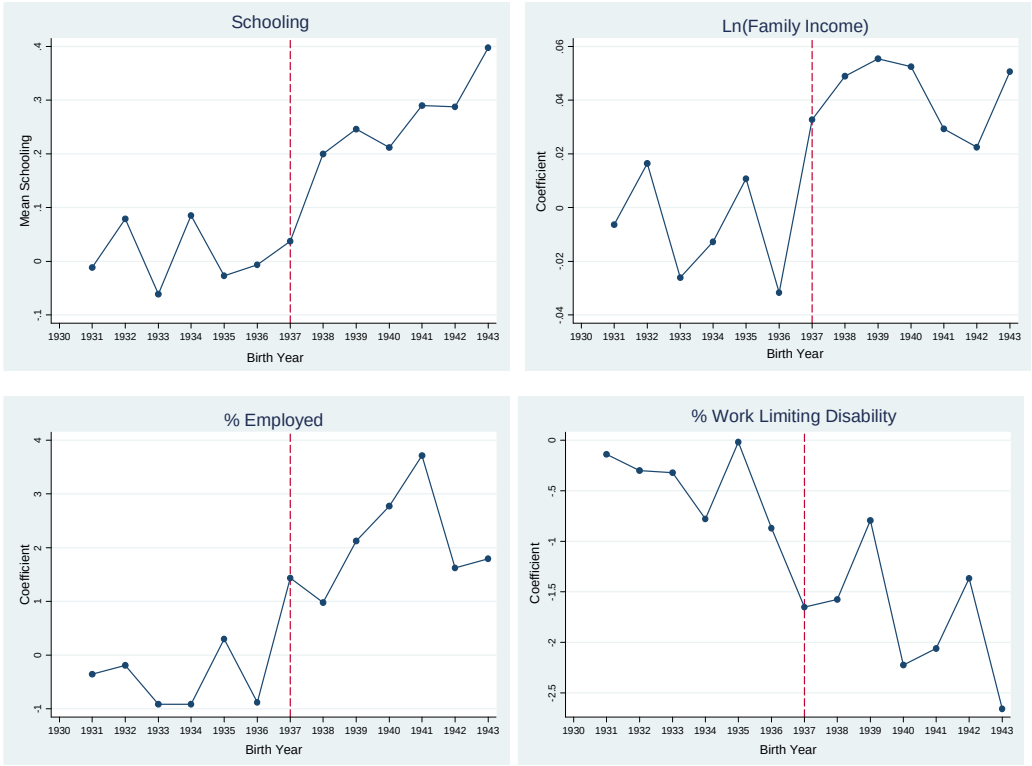
Notes: Total mortality from influenza and pneumonia for all age groups. The dashed-vertical line represents the year in which sulfa antibiotics first became available in the United States. Data source: US Vital Statistics

Figure 2 – Post-Sulfa Reduction in Pneumonia Mortality Rates by Baseline Rates



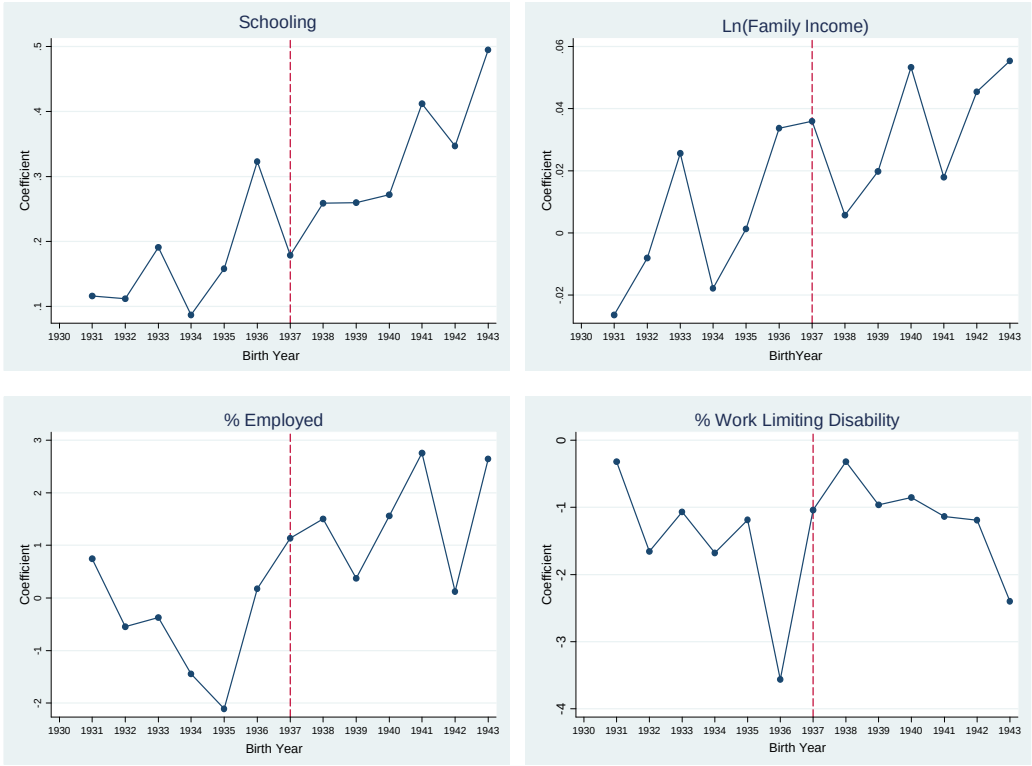
See Notes for *Figure 2*

Figure 3 – Coefficients on *Birth Year* X *BaseRatePNA* Interactions, Men



Notes: Each point reflects the estimate on the interaction between the marked birth year and *BaseRatePNA*. All models include birth state and birth year fixed effects, *Post*BaseRate(Control Diseases)*, and birth state X birth year macroeconomic and infrastructure variables (i.e., the same control vector as used in Column 3 of *Table 2*). The vertical line denotes the year sulfa drugs became available in the United States (agents more efficacious against pneumonia became available in 1938). See the notes for *Table 2* for further details.

Figure 4 – Coefficients on *Birth Year* X *BaseRatePNA* Interactions, Women



Notes: See notes for *Figure 3*.

Figure 5 – Pneumonia Mortality Rates by Race, United States, 1930-1940

Source: US Vital Statistics

Table 1 – Sample Descriptives

Census Variables	Men	Women	White Men	White Women	Black Men	Black Women
Schooling	12.64 (0.87)	12.29 (0.67)	12.79 (0.72)	12.40 (0.59)	11.20 (0.84)	11.48 (0.70)
Family Income	63667.42 (12498.54)	56367.86 (11148.51)	65694.67 (11238.07)	58713.07 (9420.62)	45060.67 (6847.58)	38527.27 (5876.59)
% Employed	73.98 (21.49)	53.36 (16.27)	75.16 (21.37)	53.45 (16.17)	63.22 (19.48)	52.73 (16.99)
% Work Disability	13.36 (5.19)	12.22 (5.07)	12.69 (4.51)	11.24 (3.92)	19.54 (6.67)	19.67 (6.47)
% Cognitive Difficulty	6.02 (2.86)	5.42 (2.46)	5.63 (1.78)	4.84 (1.52)	9.83 (3.39)	10.09 (3.34)
% Physical Difficulty	18.37 (5.12)	18.79 (5.68)	17.65 (4.49)	17.52 (1.52)	25.39 (5.55)	28.95 (5.74)
Birth State Baseline Mortality Rates (Per Thousand, N = 48 States)						
Pneumonia	1.06 (0.19)					
Under-2 Diarrhea	8.22 (5.65)					
Maternal Mortality	6.34 (1.24)					
Tuberculosis	0.64 (0.37)					
Heart Disease	2.09 (0.64)					
Cancer	0.96 (0.31)					
Birth State X Birth Year Socioeconomic Variables (N = 669)						
Income Per Capita	544.72 (274.63)					
Hospitals Per 1,000	0.067 (0.042)					
Physicians Per 1,000	1.21 (0.36)					
Schools Per 1,000	2.52 (1.90)					
Educational Spending Per Capita	87.04 (52.09)					

Notes:
-Figures provided are means, with standard deviations in parentheses
-See main text and the Data Appendix for details on variable definitions and construction
-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. Note that for the regressions, cell-level means are used as observations instead of unit record data
-Census family income figures reflect 2000 dollars
-Baseline mortality rates reflect average mortality rates for each birth state over the period 1930-1936. Rates for pneumonia, tuberculosis, heart disease and cancer reflect deaths per 1,000 total population. Diarrheal rates are computed per 1,000 live births Maternal mortality rates are per 1,000 live births, as well

Table 2 – Main Results for Schooling, Income, Employment and Disability by Gender

	(1)	(2)	(3)	(4)	(5)
Men					
Schooling	0.399***	0.297***	0.332***	0.555***	0.742***
(N = 1154)	(0.105)	(0.0921)	(0.112)	(0.0908)	(0.147)
Ln(Family Income)	0.0387***	0.0362***	0.0493***	0.0753***	0.107***
(N = 3405)	(0.0138)	(0.0125)	(0.0152)	(0.0185)	(0.0200)
% Employed	1.565***	1.241**	2.441***	2.531**	2.676
(N = 3405)	(0.573)	(0.523)	(0.566)	(1.102)	(1.743)
% Work Limiting Disability	-2.918***	-2.088***	-1.448***	-1.013	-2.394***
(N = 3405)	(0.599)	(0.485)	(0.491)	(1.036)	(0.803)
Women					
Schooling	0.410***	0.363***	0.174	0.00201	-0.0219
(N = 1161)	(0.0848)	(0.0781)	(0.111)	(0.131)	(0.172)
Ln(Family Income)	0.0545***	0.0479***	0.0325**	-0.0140	0.0326
(N = 3448)	(0.0147)	(0.0139)	(0.0146)	(0.0180)	(0.0253)
% Employed	-1.018	-0.783	1.983**	0.561	-0.624
(N = 3448)	(0.651)	(0.620)	(0.782)	(1.170)	(1.564)
% Work Limiting Disability	-2.487***	-1.583***	0.147	1.263	1.901**
(N = 3448)	(0.691)	(0.539)	(0.561)	(0.769)	(0.893)
Controls					
Birth State, Birth Year FE	Yes	Yes	Yes	Yes	Yes
Post*BaseRate(Control Diseases)	No	Yes	Yes	Yes	Yes
Birth State X Birth Year Macro	No	No	Yes	Yes	Yes
Birth State Linear Trends	No	No	No	Yes	Yes
Birth Census Region X Birth Year FE	No	No	No	No	Yes

Notes

-*** - p<0.01, ** - p<0.05, * - p<0.10

-Robust standard errors, corrected for clustering at the birth state level, in parenthesis

-Each estimate is from a different regression and represents the estimate on Post*BaseRatePNA

-N refers to the number of Birth State X Birth Year X Race X Gender X Census Year cells

-Each cell is weighted by its population in the regression analysis; unweighted regressions produce substantively similar results

-The outcome variables Schooling, Ln(Family Income), % Employed and % Work Limiting Disability are discussed in the main text as well as in the **Data Appendix**

-BaseRate(Control Diseases) includes pre-sulfa birth state averages for maternal mortality, heart disease, cancer, under 2 diarrheal, and tuberculosis mortality

-"Birth State X Birth Year Macro" includes controls for logged state per capita income per capita educational expenditures, and per capita school buildings, hospitals, and physicians by birth state and birth year

Table 3 – Additional Disability Variables by Gender

	(1)	(2)	(3)	(4)	(5)
Men					
% Cognitive Disability	-1.40**	-0.996*	-0.499	-2.28*	-2.74
(N = 1124)	(0.601)	(0.552)	(0.706)	(1.26)	(1.70)
% Physical Disability	-1.23	-0.960	-0.687	-0.141	1.97
(N = 1124)	(0.980)	(1.10)	(1.29)	(2.58)	(2.44)
Women					
% Cognitive Disability	-0.903	-0.229	1.32*	2.09*	0.818
(N = 1142)	(0.605)	(0.523)	(0.707)	(1.16)	(1.17)
% Physical Disability	-1.04	-0.441	0.695	3.32**	3.45
(N = 1142)	(0.886)	(0.806)	(0.971)	(1.48)	(2.06)
Controls					
Birth State, Birth Year FE	Yes	Yes	Yes	Yes	Yes
Post*BaseRate(Control Diseases)	No	Yes	Yes	Yes	Yes
Birth State X Birth Year Macro	No	No	Yes	Yes	Yes
Birth State Linear Trends	No	No	No	Yes	Yes
Birth Census Region X Birth Year FE	No	No	No	No	Yes

Notes
-See Notes for Table 2

Table 4 – Results for Schooling, Income, Employment and Disability for White Men and Women

	(1)	(2)	(3)	(4)	(5)
White Men					
Schooling	0.401***	0.326***	0.416***	0.607***	0.868***
(N = 655)	(0.107)	(0.0929)	(0.114)	(0.111)	(0.177)
Ln(Family Income)	0.0351**	0.0335**	0.0497***	0.0647***	0.106***
(N = 1965)	(0.0146)	(0.0129)	(0.0155)	(0.0226)	(0.0298)
% Employed	1.421**	1.157*	2.449***	2.110*	2.770
(N = 1965)	(0.636)	(0.576)	(0.612)	(1.251)	(2.141)
% Work Limiting Disability	-3.161***	-2.346***	-1.689***	-1.249	-3.159***
(N = 1965)	(0.650)	(0.546)	(0.567)	(1.087)	(0.821)
White Women					
Schooling	0.409***	0.393***	0.264***	0.00563	-0.0391
(N = 1161)	(0.0854)	(0.0760)	(0.0932)	(0.147)	(0.227)
Ln(Family Income)	0.0522***	0.0479***	0.0359**	-0.0241	0.0222
(N = 1965)	(0.0163)	(0.0151)	(0.0163)	(0.0208)	(0.0358)
% Employed	-1.536**	-1.193*	1.936**	1.199	-0.0708
(N = 1965)	(0.681)	(0.672)	(0.820)	(1.160)	(1.742)
% Work Limiting Disability	-2.736***	-1.863***	-0.111	1.302	2.266*
(N = 1965)	(0.761)	(0.574)	(0.576)	(0.865)	(1.229)
Controls					
Birth State, Birth Year FE	Yes	Yes	Yes	Yes	Yes
Post*BaseRate(Control Diseases)	No	Yes	Yes	Yes	Yes
Birth State X Birth Year Macro	No	No	Yes	Yes	Yes
Birth State Linear Trends	No	No	No	Yes	Yes
Birth Census Region X Birth Year FE	No	No	No	No	Yes

Notes
-See Notes for Table 2

Table 5 – Results for Schooling, Income, Employment and Disability for Black Men and Women

	(1)	(2)	(3)	(4)	(5)
Black Men					
Schooling	0.380	-0.0534	-0.411*	-0.0503	-0.0707
(N = 499)	(0.302)	(0.240)	(0.235)	(0.197)	(0.224)
Ln(Family Income)	0.0792	0.0537	0.00696	0.121	0.0337
(N = 1439)	(0.0535)	(0.0461)	(0.0558)	(0.116)	(0.101)
% Employed	3.183	1.438	-2.178	1.625	-1.764
(N = 1440)	(2.687)	(2.593)	(2.408)	(4.067)	(3.500)
% Work Limiting Disability	-0.187	1.023	-0.579	-1.035	0.251
(N = 1440)	(1.547)	(1.335)	(1.696)	(3.807)	(4.384)
Black Women					
Schooling	0.415	0.0783	-0.488	-0.194	-0.279
(N = 506)	(0.345)	(0.288)	(0.369)	(0.305)	(0.357)
Ln(Family Income)	0.0760*	0.0358	0.0115	0.0629	0.110
(N = 1483)	(0.0418)	(0.0422)	(0.0478)	(0.0737)	(0.101)
% Employed	3.786**	3.210**	0.404	-4.836	-2.977
(N = 1483)	(1.558)	(1.350)	(2.219)	(2.979)	(2.770)
% Work Limiting Disability	-0.171	0.637	1.718	1.607	-1.302
(N = 1483)	(1.530)	(1.719)	(1.734)	(3.149)	(3.151)
Controls					
Birth State, Birth Year FE	Yes	Yes	Yes	Yes	Yes
Post*BaseRate(Control Diseases)	No	Yes	Yes	Yes	Yes
Birth State X Birth Year Macro	No	No	Yes	Yes	Yes
Birth State Linear Trends	No	No	No	Yes	Yes
Birth Census Region X Birth Year FE	No	No	No	No	Yes

Notes
-See Notes for Table 2

Table 6 – Additional Disability Variables by Gender X Race Groups

	(1)	(2)	(3)	(4)	(5)
White Men					
% Cognitive Disability	-1.07	-0.649	-0.0209	-1.67	-1.44
(N = 655)	(0.658)	(0.630)	(0.755)	(1.37)	(1.92)
% Physical Disability	-0.932	-0.592	-0.205	0.169	2.31
(N = 655)	(1.03)	(1.16)	(1.38)	(2.86)	(2.72)
White Women					
% Cognitive Disability	-1.09*	-0.453	1.02	1.65	0.483
(N = 655)	(0.626)	(0.521)	(0.654)	(1.19)	(1.59)
% Physical Disability	-0.891	-0.340	1.49	3.60***	4.10*
(N = 655)	(0.893)	(0.846)	(0.978)	(1.32)	(2.18)
Black Men					
% Cognitive Disability	-5.26***	-5.59***	-4.67**	-5.23	-3.97
(N = 469)	(1.55)	(1.85)	(2.23)	(6.16)	(7.49)
% Physical Disability	-4.75*	-6.07**	-0.451	0.149	2.17
(N = 469)	(2.54)	(2.95)	(4.27)	(6.29)	(9.81)
Black Women					
% Cognitive Disability	0.935	1.93	4.24	1.58	-0.195
(N = 487)	(1.77)	(1.75)	(2.68)	(3.72)	(3.77)
% Physical Disability	-2.50	-1.63	0.367	10.6	4.17
(N = 487)	(3.13)	(3.54)	(3.33)	(9.88)	(9.41)
Controls					
Birth State, Birth Year FE	Yes	Yes	Yes	Yes	Yes
Post*BaseRate(Control Diseases)	No	Yes	Yes	Yes	Yes
Birth State X Birth Year Macro	No	No	Yes	Yes	Yes
Birth State Linear Trends	No	No	No	Yes	Yes
Birth Census Region X Birth Year FE	No	No	No	No	Yes

Notes

-See notes for Table 2

Table 7 – Specifications with Mean Reversion Controls

	(1)	(2)	(3)	(4)
	Schooling	Ln(Family Income)	% Employed	% Work Disability
All Men	0.351*	0.0999***	2.605	-1.218
	(0.189)	(0.0231)	(1.774)	(0.923)
All Women	-0.403*	0.0267	-0.983	2.858***
	(0.206)	(0.0281)	(1.515)	(0.948)
White Men	0.756***	0.0696*	2.855	-3.196***
	(0.214)	(0.0372)	(2.143)	(0.887)
White Women	-0.104	0.0521	-0.301	2.691**
	(0.302)	(0.0370)	(1.734)	(1.286)
Black Men	-0.0728	0.0157	-2.046	0.0472
	(0.239)	(0.101)	(3.674)	(4.274)
Black Women	-0.375	0.0647	-3.627	-1.564
	(0.340)	(0.107)	(2.942)	(3.472)

Notes

-*** - p<0.01, ** - p<0.05, * - p<0.10

-Robust standard errors, corrected for clustering at the birth state level, in parenthesis

-Each estimate is from a different regression and reflects the coefficient on Post*BaseRatePNA.

-These models control for Post*BaseRate(Outcome) in order to control for mean reverting shocks that are jointly correlated with trends in pneumonia and the outcomes

-All models include the controls in Column 5 of Table 2. Refer to the Table 2 notes for further details.

-Refer to Tables 2, 4 and 5 for information on sample sizes. Observations are weighted by cell population.

Table 8 – Triple Difference Specifications

	(1)	(2)	(3)	(4)
	Schooling	Ln(Family Income)	% Employed	% Work Disability
All Men	0.800***	0.107***	2.676	-2.394***
	(0.219)	(0.0200)	(1.743)	(0.803)
All Women	0.467**	0.0603***	4.314***	-0.410
	(0.222)	(0.0180)	(1.159)	(1.219)
White Men	0.851***	0.131***	1.578	-0.993
	(0.225)	(0.0256)	(1.188)	(1.211)
White Women	0.581***	0.0441**	-0.164	0.802
	(0.197)	(0.0193)	(1.196)	(0.689)
Black Men	0.430	0.0875	5.418	-8.392***
	(0.655)	(0.0826)	(3.431)	(2.328)
Black Women	-0.479	0.0621	0.233	-0.830
	(0.742)	(0.0711)	(2.674)	(3.553)

Notes

-*** - p<0.01, ** - p<0.05, * - p<0.10

-Robust standard errors, corrected for clustering at the birth state level, in parenthesis

-Each estimate is from a different regression and represents the estimate on the triple difference Post*BaseRatePNA*Treated, where Treated = 1 refers to those who were infants during the period 1932-1943 The control cohort includes those who turned 10 during this same period. For the control cohort, Post = 1 if the cohort turned 10 years old in 1937 or thereafter.

-All models include controls for Post*BaseRatePNA, Post*Treated, Treated*BaseRatePNA, birth state and birth year fixed effects, Post*BaseRate(Control Diseases), birth state specific linear time trends, and census region of birth X birth year fixed effects

-Refer to main text for further details; refer to Table 2 notes for details regarding the control variables.

-Also, refer to Tables 2, 4 and 5 for sample size information. Observations are weighted cell population.

Data Appendix

Outcomes – These were all taken from the 1980 5%, 1990 5% and 2000 5% United States Census Microdata samples (available via IPUMS-USA, http://usa.ipums.org/usa/). These data are publicly available via the Integrated Public Use Microdata Series – USA project (Ruggles, Alexander, Genadek, Goeken, Schroeder, & Sobek, 2010). We aggregated all data into birth state X birth cohort X race (white and other) X gender X census year cells. Data on the 2,019,930 men, 2,137,468 women, 1821,471 white men, 198,459 black men, 1,897,973 white women and 248,495 black women in the three census samples who were born between 1930 and 1943 (the period of interest in the study) were used to create cell level means.

Schooling – Represents the highest grade of schooling completed. This variable was constructed using the IPUMS variable *HIGRADE*. Since schooling was likely completed before the age of 30 for most sample individuals, we used only the 1980 census data for this variable.

Logged Total Family Income – From the IPUMS variable *FTOTINC*. Describes the (nominal) total pre-tax money income earned by the respondent's family unit in the previous calendar year.

Employed - Uses the IPUMS variable *EMPSTAT*, which distinguishes between current employment, unemployment and not being in the labor force. For each individual, we set employment = 1 if the individual reports current employment and 0 otherwise.

Work Limiting Disability – The IPUMS variable *DISABWRK* Indicates a physical or mental health condition that causes difficulty working, limits the amount or type of work, or prevents working altogether. The disability cannot be transient (e.g., pregnancy) and must have been present for at least six month prior to survey. We coded any limitation in the ability to work (either certain limitations or the inability to work altogether) as representing disability.

Cognitive Disability – From the IPUMS variable *DIFFREM*, which denotes whether an individual has difficulty with cognitive tasks due to a physical or mental illness. This variable is only available in the 2000 census.

Physical Disability – From the IPUMS variable *DIFFPHYS*. Denotes if the respondent has a condition that limits basic tasks of daily living that involve movement (walking, running, lifting, etc). This variable is only available in the 2000 census.

Baseline Pneumonia Rates and Disease Variables – State X Year data on pneumonia, under-2 diarrheal, heart disease, cancer, and tuberculosis mortality, as well as the maternal mortality ratio, were taken from various volumes of the US Vital Statistics (Grove, 1968; Linder, 1947; United States Bureau of the Census, 1930-1943). We also made use of US Vital Statistics data collected by Grant Miller (http://www.nber.org/data/vital-statistics-deaths-historical/) and Seema Jayachandran, Adriana Lleras-Muney, and Kimberly Smith (http://www.aeaweb.org/articles.php?doi=10.1257/app.2.2.118). We used the State X Year data to create state-specific baseline rates for each disease by averaging mortality rates between 1930 and 1936.

Of note, for several of the years in the period 1930-1943, pneumonia mortality counts were combined with influenza mortality counts. As such, we follow Jayachandran, et al (2010) and work with a combined pneumonia/influenza mortality rate. As noted in the main text, influenza mortality rates remained stable during the study period and, prior to sulfa, accounted for ~25% of the combined mortality rate.

In addition, we also used Race X State X Year mortality figures for pneumonia and several other diseases. We thank Adriana Lleras-Muney for providing use these data, which were originally taken from yearly US Vital Statistics volumes (http://www.cdc.gov/nchs/products/vsus.htm).

Socioeconomic Characteristics and Infrastructure Variables – State X Year data on logged state per capita income were taken 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). 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 1939.