# The Long Run Effects of Early Life Pneumonia: Evidence from the Arrival of Sulfa Drugs in America

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**Sonia Bhalotra**
*Department of Economics*
*University of Bristol*
*8 Woodland Road*
*Bristol BS8 1TN, UK*
s.bhalotra@bristol.ac.uk

**Atheendar Venkataramani**
*Washington University School of Medicine*
*660 S. Euclid Ave*
*St. Louis, MO 63108*
venkataat@wusm.wustl.edu

**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. Importantly, 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. While men and women show similar improvements on most indicators, only the estimates for the former are robust to the inclusion of birth state specific time trends. With the exception of cognitive disabilities for men and, in some specifications, family income for men and women, estimates for African Americans tend to be smaller in magnitude and less precisely estimated than those for whites. We speculate that this may be due to barriers 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

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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 infancy and 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, may be diverted away from physical and mental development. In this way, severe or repeated infections could lead to long-term "scarring" in the form of poorer health and reduced cognitive development in adulthood (Crimmins & Finch, 2006; Gluckman & Hanson, 2005). In addition, if there are dynamic complementarities in childhood endowments, any particular damage in infancy may actually 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.

Obtaining causal effects of infectious disease on later life outcomes is challenging because of likely selection into infection. A few studies have addressed this problem by using plausibly exogenous variation in infectious diseases to identify their long-run impacts. Almond (2006) uses the 1918 influenza pandemic as a natural experiment to examine the effects of *in utero* exposure to the flu on socioeconomic status and disability in adulthood. Kelly (2009) examines the effects *in utero* exposure to the 1957 Asian influenza pandemic on health and cognitive outcomes among school children in the United Kingdom. Bleakley (2010), Cutler, Fung, Kremer, Singhal, and Vogl (2010), Lucas (2010), and 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.

Despite this growing literature, there is no evidence on the long-run impacts of other important childhood infectious diseases, in particular pneumonia, which is currently the leading cause of child death in the developing world (Bhutta, 2007; Black et al., 2010). Furthermore, the aforementioned studies have examined unique pandemic conditions or (near)-total eradication campaigns. These dramatic events may have different long-term

effects than more subtle changes in the disease environment[1]. Finally, there is little evidence on the long-run impacts of reduced exposure to infections stemming from therapeutic, as opposed to preventative, interventions. For all of these reasons, it is unclear whether the findings in the extant literature can be applied to other diseases, contexts, or policies.

This paper addresses these gaps by investigating the long-run impacts of declines in pneumonia driven by the exogenous introduction of sulfonamide antibiotics (hereafter, sulfa drugs) in the United States in 1937. These agents were the first antibiotics used clinically and were quickly utilized to treat a variety of infections. The arrival of sulfa drugs led 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 the long run for schooling, income and the probabilities of employment and disability.

Specifically, we use Unites States census data from 1980-2000 to compare outcomes for cohorts born before and after the arrival of sulfa drugs, exploiting the fact that those states with higher pre-intervention pneumonia rates benefited most from the new antibiotics in terms of absolute reductions in pneumonia mortality. We focus on the impacts of birth year exposure to pneumonia given that morbidity and mortality from this disease was far more severe for this age group vis-à-vis older cohorts and that 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 result in long-term scarring.

Our models control for a host of birth state X birth year specific controls including mortality from other communicable diseases that were not responsive to sulfa antibiotics (such as diarrhea and tuberculosis) and non-communicable diseases (such as cancer and heart disease). We also control for the maternal mortality rate, which declined sharply with the arrival of sulfa drugs and may have had long-run effects through its impacts on parental investment (Jayachandran & Lleras-Muney, 2009), so as to better isolate the pneumonia effect. In addition, we estimate models with birth state X birth year socioeconomic characteristics, birth state specific time trends, and birth census region X birth year fixed

[1] Influenza mortality increased four-fold during the flu epidemics analyzed, while the sulfa-induced decline in pneumonia mortality rates examined in this paper was 30%. Thus, these 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.

effects. Finally, we allow for heterogeneity in treatment effects by estimating our models separately by gender, and by race and gender groups.

Our core findings are that cohorts born during the sulfa drug era experienced increases in years of schooling, family income, and probability of employment, as well as decreases in the probability of work-preventing disability, relative to earlier cohorts. These increases are largest for those born in states that gained most from the introduction of sulfa; that is, those with higher pre-intervention pneumonia mortality rates. We find evidence of these increases for both men and women, though only the results for men are robust to specification. We speculate that these gender differences may be driven by the fact that male children were more vulnerable to contracting infection.

We also find that the estimates for blacks are less precisely estimated and more sensitive to specification than for whites. With the exception of cognitive disability for men and, in some specifications, family income for men and women, the coefficient estimates for blacks are smaller in magnitude, as well. 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 reduced 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.

Our study adds to a growing literature on the long-run effects of early childhood health (Almond & Currie, 2010). More generally, this paper contributes to an emerging literature concerned with the origins of socioeconomic inequality in the early childhood years (Cunha & Heckman, 2009; Heckman, 2007). In terms of policy, our results underscore the importance of addressing the burden of disease from pneumonia, a disease that currently accounts for nearly 30% of under-5 deaths in the developing world but draws relatively little attention from researchers and policy makers (Bhutta, 2007). In addition, our findings also motivate increased research and focus on the returns to distribution of pharmaceuticals, such as antibiotics, in developing countries.

The rest of paper is as follows. Section 2 discusses the sulfa drug revolution and its effects on pneumonia mortality in the United States. Section 3 describes 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. Prior to the arrival of sulfa drugs, the disease was treated primarily with supportive care. In the early 1930s, a small but growing number of clinicians began to use intravenous serum therapy[2] to combat the bacterial pneumonia, which was often more severe and conferred a higher risk of death than its' viral counterpart (Lesch, 2007). 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 1942-43.

The arrival of sulfa drugs represented a 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. Early 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

[2] This involved obtaining antibodies from animals that were infected with a specific microbe. The "serum" refers to the component of animal blood comprised of fluid and antibodies, which had to be separated from the cellular component. The serum was then 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).

breaks in the time series data for sulfa treatable 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 birth state pre-sulfa pneumonia mortality rates. The key point in this figure is that states experienced a convergence in pneumonia mortality rates after 1937. This suggests that areas with a greater 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 to examine the long-run effects of pneumonia in infancy.

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. In contrast, if sulfa therapy (additionally) reduced the severity and or 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.

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). A similar result was seen for outpatients, who accounted for around 70% of all pneumonia cases (Britten, 1942), as well.

First, 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. Moreover, data on industrial workers illustrates 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).

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 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, it is difficult to draw any conclusions from these data given that measures of pneumonia incidence are sensitive to any improvements in surveillance and diagnosis. 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 cannot make any claims on whether they led to reductions in the probability of contracting the infection altogether.

Because 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% 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.

In contrast, 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 subsection). 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 (Seema Jayachandran and Adriana 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. Basic 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. Specifically, we estimate versions of the following basic specification:

$$Y_{rstc} = \beta_0 + \beta_1 * Post_t * BaseRatePNA_s + \delta_s + \zeta_t + \gamma_r + \mu_c + \theta_{rs} + \eta_{rt} + \lambda_{rc} + e_{rstc}$$

where $Y_{rst}$ is the outcome of interest for each race (*r)* X birth state (*s*) X birth year (*t*) X census year (*c*) cell, *Post* = 1 if the birth cohort was born after the initial introduction of sulfa drugs (i.e., in 1937 or thereafter), $BaseRatePNA_j$ is the pre-sulfa birth state-level pneumonia mortality rate, which we use to proxy for the pre-period pneumonia exposure, and the Greek letter terms represent fixed effects for birth state, birth year, race, census year, race X birth state, race X birth year, and race X census year, respectively. This strategy is similar in spirit to those utilized by Acemoglu and Johnson (2007), Bleakley (2007), and Lucas (2010). We cluster our standard errors at the birth state-level to account for serial correlation in the

outcomes (Bertrand, Duflo, & Mullainathan, 2004) and we weight each cell by the number of individuals within it[3].

The main outcomes we examine include years of schooling, logged household income, employment status and disability limiting or preventing work: we chose these variables in particular given their consistent use in other studies on the long-run effects of early life events. 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 starting in 1943). Our use of multiple census years allows us to observe the same birth cohorts at different ages, so as to distinguish between life cycle and age effects as well as increase the precision of our estimates.

We estimate all of our models separately by gender. This is done to account for sex-specific differences in biological responses to early childhood shocks (Low, 2000; Waldron, 1983), as well as in any parental investment behaviors that respond to health endowments. In addition, we also allow for heterogeneity in the treatment effect by estimating our models separately by gender X race groups.

The main threat to inference with the differences-in-differences type model we estimate here is the presence of other birth state X birth year characteristics that are jointly correlated with declines in pneumonia and the outcomes. To address this, we test the sensitivity of the results to the inclusion of a variety of controls reflecting birth state X birth year health and socioeconomic conditions. We interact *Post* with birth state-specific pre-sulfa mortality rates from tuberculosis, under-2 diarrhea, heart disease and cancer. As in Jayachandran, et al (2010), the former help control for secular, state specific changes (such as improvements in sanitation, public health programs, housing, etc) that influenced exposure to infectious diseases. The inclusion of non-communicable diseases helps control for factors such as health care quality and access.

Importantly, we also control for the baseline maternal mortality rate, interacted with *Post*. As discussed in the previous section, sulfa drugs led to large reductions in death from puerperal infections, which could impact long-run health and socioeconomic outcomes via increased parental investment in girls. Because we are interested in focusing on pneumonia, controlling for maternal mortality would help isolate this effect. In addition to these disease

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

rate variables, we also include controls for birth state X birth year logged state income per capita, and the numbers of schools, hospitals and physicians per capita. To control for any pre-existing trends that are not addressed these and the aforementioned controls we also estimate specifications with birth state specific linear time trends and census region X birth year fixed effects.

We acknowledge the possibility that our additional specifications may actually amount to "overcontrolling." 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 variations in disease trajectories that are in fact driven by the use of sulfa drugs rather than/in addition to 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. At another level, controlling for state trends is typically quite demanding of the data. We therefore present results with and without each set of controls.

We extend our basic specification above in two ways. First, we replace *Post* with a vector of birth year dummies and graph the resulting coefficients. This specification allows us to assess whether there were returns to sulfa exposure later in childhood. In addition, it also allows provides us 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. Along the same lines, 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 can easily assess this with the coefficient plots.

An additional source of potential bias comes from mortality selection. One might expect that frailer children were more likely to succumb from pneumonia than their healthier counterparts. With the advent of sulfa, more of these children would survive past childhood, which would bias the estimates on $Post_t$*$BaseRatePNA_s$ to zero above since such individuals are likely to be less productive and healthy as adults (Bozzoli, Deaton, & Quintana-Domeque, 2009). That said, we expect this bias to be present, but potentially small, since

pre-intervention case rates among infants were around 30 per 1,000 population, while death rates were significantly lower at around 1 per 1,000 (Britten, 1942; Councell, 1963).

Finally, it should also be noted that, other biases notwithstanding, our estimates are likely lower bounds of the true long-run impact of sulfa drugs since access to, and/or use of, these therapies was not universal (that is, we recover an intent-to-treat effect). 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.

**4. Data**

Data for our main outcome variables – years of schooling (highest year of education completed), family income (total pre-tax income owned by a family unit), employment status and work limiting/preventing disability - were taken from the 1980, 1990, and 2000 5% samples of the United States Census (see Data Appendix for further details). These data are publicly available via the Integrated Public Use Microdata Series – USA project (Ruggles, Alexander, Genadek, Goeken, Schroeder, & Sobek, 2010). 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 X birth year X census year X race X gender specific cells, and used the cell level data in our regression analysis[4]. For schooling, we only examined data from the 1980 census since the birth cohorts of interest likely finished their schooling by this time, rendering information from later censuses redundant[5]. Due to the increased potential for measurement error, we dropped cells in the bottom 1% of the cohort size distribution (i.e., those with less than 50 persons)[6]

Data on all-age disease-specific mortality rates (expressed per 1,000 people) are from the US Vital Statistics and information socioeconomic characteristics of states come from a variety of other sources, all of which are detailed in the Data Appendix. For the period of interest, state-level time series data on pneumonia mortality is often aggregated with influenza mortality, so we (like Jayachandran et al 2010) work with this compound variable.

[4] The use of micro-level data produced very similar point estimates and standard errors.
[5] In addition, if sulfa exposed cohorts who on average achieved fewer years of education were also likely to die younger, the including data from later censuses may bias downward the estimated impact of sulfa drugs.
[6] The results are unchanged even if these cells are included in the analysis.

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.

## 5. Results

We divide our discussion of the results into four subsections. The first discusses the core findings for schooling, family income, employment and disability. The second examines additional indicators of disability. The third section presents race-specific results and the fourth discusses findings from additional falsification tests.

### 5.1. Main Results

Our core results for schooling, logged family income, employment status and disability preventing work are presented in *Table 2*. Each row X column represents a separate regression, and the coefficients displayed are the estimates on $Post_t$*$BaseRatePNA_s$. The rows denote the outcome variables and the columns different sets of control variables.

We find positive and statistically significant impacts of exposure to sulfa on years of schooling for men. The estimates from column 1 imply that moving from the 75$^{th}$ to the 25$^{th}$ percentile of the pre-sulfa baseline pneumonia rate distribution (i.e., from 1.18 to 0.92 deaths per 1,000) is associated with a 0.1 (0.399*0.26) increase in the highest grade completed on average for a given cohort. The inclusion of baseline mortality rates for the control disease (which includes maternal mortality), birth state X birth year socioeconomic and infrastructure characteristics, state specific linear time trends, and census region X birth year

fixed effects, does little to alter this substantive conclusion[7]. In fact, in the specification with all of the control variables, the results imply a 0.19 increase in years of schooling if the pneumonia death rate decreased from the 75th to 25th percentile of the pre-sulfa mortality rate distribution.

We find a similar pattern of results for the other long-run indicators. For the same shift in the pneumonia mortality rate discussed above, our results suggest a 2.8% increase in family income, a 0.7 percentage point increase in the probability of being employed, and a 0.62 percentage point decrease in the probability of reporting a disability limited or preventing work for men. These estimates are all taken from column (5), the specification with all control variables included.

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 (column 1). However, the inclusion of birth state X birth year socioeconomic and disease environment controls reduces the magnitude of the estimates, and the inclusion of birth state specific linear trends obliterates them completely.

*Figures 3* and *4* present plots of the coefficients on the birth year X baseline pneumonia rate interactions for men and women, respectively. 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 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-existent for the estimates for women[8].

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[7] It is important to note that 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*BaseRateMMR* are negative for schooling, income and employment, and positive for disability. The 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.

[8] Indeed, the generally uptrending coefficients and the lack of a sharp trend break in 1937 explains why we find large, significant coefficients in the specifications with fewer controls and why these estimates are obliterated with the inclusion of birth state-specific linear time trends.

For both men and women, we do not find evidence of trend breaks that occur in years other than those associated with the introduction of sulfa. This finding further supports our interpretation of the coefficient on $Post_t$*$BaseRatePNA_s$ as representative of the long-run effects of the reduction in childhood pneumonia exposure rather than some other process. The results also suggest that the returns to sulfa exposure were likely limited to the birth year as the coefficient estimates for men prior to 1937 hover around zero[9].

Another point to address before moving forward pertains to the nature of the gender differences found in the results. Men may have benefitted more from early life exposure sulfa relative to females for several reasons. First, it is known that men are more vulnerable to disease and other shocks in early childhood (Gluckman & Hanson, 2005; Low, 2000; Waldron, 1983). Indeed, surveillance data from the mid-1930s shows that male infants were 25% more likely to contract pneumonia than female infants (Britten, 1942). In the same vein, it is possible that, conditional on contracting an infectious disease, male children are more likely to be scarred from such an experience than female children. It is also possible is that the sensitivity of parental investments to health endowments may differ by gender, though given generally pro-male gender biases noted in the United States, one would expect the association between endowments and investments to be larger for females than males (Dahl & Moretti, 2008; Stanley & Jarell, 1998).

### 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). Although this 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 presented in *Table 3*. For men, columns (1) and (2) show negative estimates for cognitive and physical disability, with coefficients for the former being

[9] This, too, supports our interpretation of the findings in *Table 2*: pneumonia was far more prevalent in infancy than in other parts of childhood (Britten, 1942), so the long-run returns to sulfa in later years should be much lower (or non-existent) when compared to birth year exposure.

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 impacst 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 physically disability, the specifications with birth state specific linear time trends actually suggest positive effects[10].

**5.3. Race Specific Effects**

*Tables 4* and *5* present estimates for schooling, income, employment and work disability for whites and African-Americans, respectively. The substantive results for white men and women mirror those found in *Table 2*. In contrast to white men, the coefficients for black men are generally imprecisely estimated and not robust to specification. Most of the coefficients for schooling and work disability are smaller than those for white men. Several of the coefficients for logged household income are larger in magnitude than for whites but this finding is sensitive to specification; the estimates on employment flip signs with the inclusion of additional controls. The findings for black women and white women are substantively similar, though the estimates for household income are larger in magnitude and more robust to specification for the former. However, these effects are generally imprecisely estimates. Coefficient plots (not shown here) for black men and women confirm the generally null pattern of results observed in *Tables 4* and *5*.

*Table 6* presents results for the additional disability variables. The results for black men suggest a larger negative effect of exposure to sulfa drugs on cognitive difficulty vis-à-vis whites. This finding is generally robust to specification and several of the coefficients are statistically significant. In terms of magnitudes, 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 of reporting a cognitive disability. Exposure to sulfa also had larger impacts on physical disability for blacks, though this finding disappears with the

[10] 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.

inclusion of additional controls and trends. The results for black women are substantively similar to their white counterparts.

Why do the results differ by race? One potential explanation for this is that pneumonia rates for blacks were relatively unaffected by the arrival of sulfa drugs due to poor access to medical technology and quality health care, more generally. While Jayachandran, et al (2010) provide evidence that blacks did indeed benefit less from the new antibiotics in terms of percentage declines in pneumonia mortality (a) they still did benefit and (b) the *absolute* decline in pneumonia mortality rates was actually larger for blacks. Both of these patterns are shown in *Figure 5*. Thus, the lack of a short run benefit cannot explain the differential black-white results.

Another possibility is measurement error in baseline mortality rate variable, especially as it pertains to blacks. Almost all states had significantly larger white populations vis-à-vis blacks, so the overall state mortality rate is more reflective of the former population than the latter. To address this, we utilize baseline mortality rates constructed from data on non-white populations in each state (available from the US Vital Statistics). Because these data combine all non-white races, and since some states have very small populations thus increasing the potential for measurement error, we followed Jayachandran, et al (2010) and only examined data for those states with greater than 10% black populations; we also limited ourselves to only those states with non-white populations greater than 100,000 individuals. Using these data, however, does little to alter our substantive conclusions (results not shown here)[11].

A final possibility is that blacks did not gain experience long-term gains from short run reductions in pneumonia mortality due to barriers in translating improved health endowments in infancy into human capital and socioeconomic returns in the pre-Civil Rights era. 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 in 1965 (Donohue & Heckman, 1991). Thus, human capital accumulation may not have responded to improved health endowments for blacks vis-à-vis whites due to due to reduced access to quality schools and training institutions and/or a discriminatory labor market, both of which would depress returns to human capital investment to the point that they may not have been

[11] Of course, this finding must be taken with a grain of salt: it is possible that the race-specific mortality data are flawed, too, if mortality reporting for non-whites was more prone to underreporting, misclassification, and/or other measurement biases.

deemed worthwhile. Our "barriers" hypothesis is supported by the fact that the estimates for cognitive disability are larger in magnitude for black vis-à-vis white men, despite generally smaller estimates on schooling and employment. This suggests that endowments for blacks were indeed influenced by reduced exposure to pneumonia, but that these improvements did not translate into increased schooling and employment prospects[12].

### 5.4. Additional Robustness Checks

We conducted several additional falsification tests to assess the robustness of our findings. The first set of checks addresses 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 those 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 drug effect. In order to control for mean reversion, we 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[13].

We also estimate "triple-difference" models as an additional test. The idea here is to exploit the epidemiology of pneumonia. While infants are very likely to contract and/or die from pneumonia older children (particularly those over the age of 10) are not. However, these older children may be affected by other processes that could impact human capital acquisition and long-run health and socioeconomic outcomes. These same processes may confound the estimates on $Post_t$*$BaseRatePNA_s$. Thus, to additionally control for any other birth state X birth year processes that are not captured by the control variables used in *Table 2*, we used the birth cohorts born between 1915-1927 as controls for the treated 1930-1943 cohorts examined above. We divided the control cohorts into those who turned 15 years old

[12] Another piece of supporting evidence, though more tenuous, is that the income estimates for black men and women were larger than those for whites in several of the specifications. This suggests that blacks were able to use their sulfa-driven health or cognitive endowments in the marketplace to secure more earnings in the post-Civil Rights Act era, though on margins that did not involve returns to schooling.

[13] 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.

during the sulfa era (that is, *Post* = 1 for cohorts born between 1922-1927) and those who turned 15 beforehand (*Post* = 0 for the 1915-1921 birth cohorts)[14].

The results of the "triple difference" analysis are presented in *Table 8*. For men, the estimates are similar in magnitude and significance as those presented 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 statistically significant. With respect to the race specific results, we now find large impacts for all four outcomes of interest for black men, though only the probability of work disability estimate is statistically significant. Thus, while there are differences between the double and triple-difference estimates for some population groups, they support the baseline findings that sulfa-induced declines in pneumonia exposure had important long-run impacts.

Finally, 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).

## 6. Conclusions

In this study, we examine the long-run returns to reductions in pneumonia exposure, a leading cause of death in United States in the first half of the 20th century, that were driven by the introduction and widespread diffusion of sulfonamide antibiotics. Our core findings are that birth year exposure to sulfa drugs led to increases in educational attainment, income, and the probability of employment and decreases in the probability of work-limiting disability. Importantly, these increases were largest for those born in states with higher pre-intervention pneumonia mortality rates, the same states that experienced the largest absolute reductions in pneumonia mortality from sulfa drugs. Only the results for men are robust to specification. In terms of race, with the exception of cognitive disability for men and, in some specifications, family income for men and women, the results for African-Americans are generally smaller in magnitude and imprecisely estimated when compared to estimates for whites.

[14] We thank Tania Barham for providing us this idea.

This study provides the first evidence of a long-term scarring effect of early life exposure to pneumonia, as well as the some of the first estimates of the long-run socioeconomic and health impacts to medical technology, in particular antibiotics. Our findings, which contribute to a growing literature on the impacts of early life health on health and socioeconomic inequalities later in life, have several policy implications. First, our results are important for developing countries, where acute respiratory infections are the leading cause of early childhood mortality. Our findings 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. Second, the results indicate that failure to account for the longer-run returns may lead to underinvestment in beneficial technologies. Indeed, the results further 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.

Further work in this area should seek to broaden the outcome measures examined. While the measures of adult attainments we examine follow the bulk of the literature on the long-run effects of early childhood health, it would be useful to have a richer range of outcomes, include better measures of health status and cognitive test scores. In addition, while we are able to establish robust connections between early life exposure to sulfa and later life outcomes, we are not able to comment on the exact nature of the causal chain linking the two. Future work would do well to understand the extent to which biological pathways and parental investment decisions interact to link early childhood events to later life outcomes. Finally, an interesting area of future research would be to better understand the causes of race and gender differences in the long-run returns to infectious diseases in young children. While we have attempted to advance a hypothesis centering on barriers to translating better endowments into human capital accumulation to explain these findings, a thorough explanation awaits more detailed theoretical frameworks and data.

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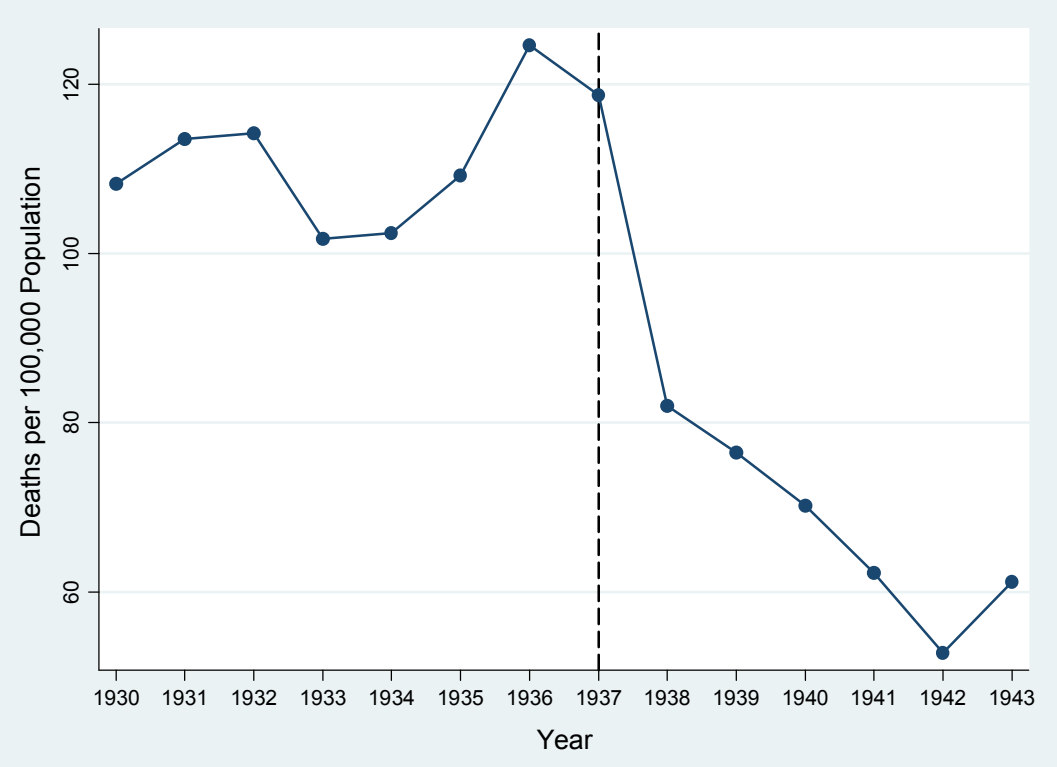
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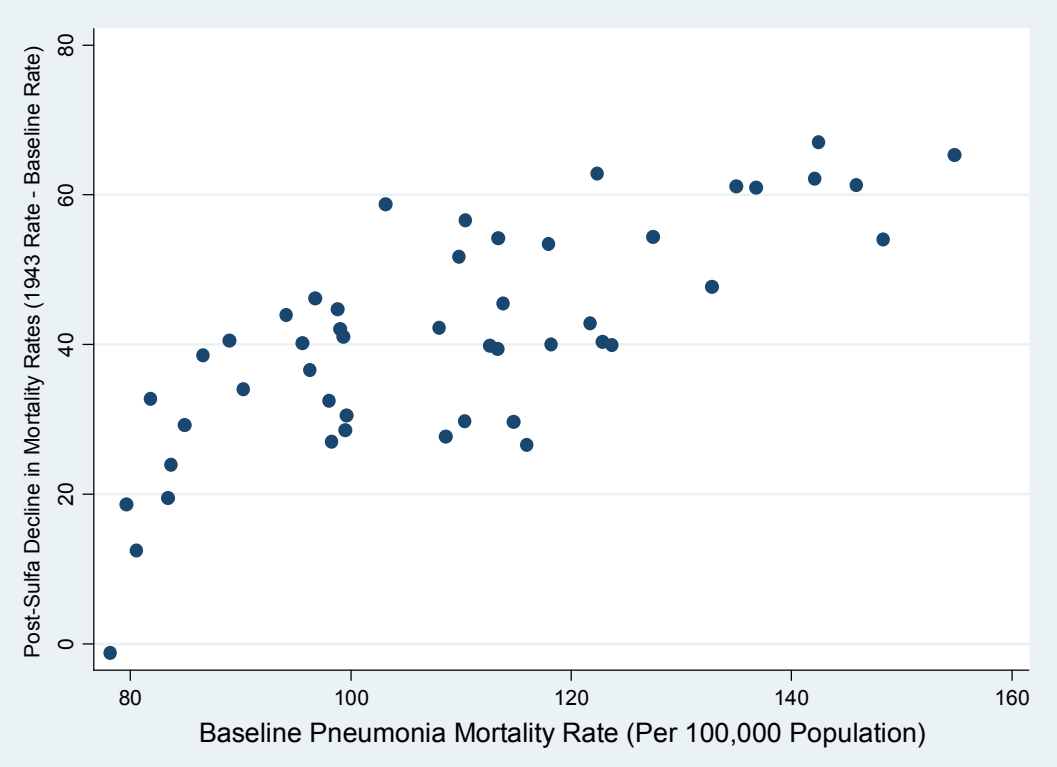
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**Figure 1 – Mortality from Pneumonia, United States, 1930-1943**



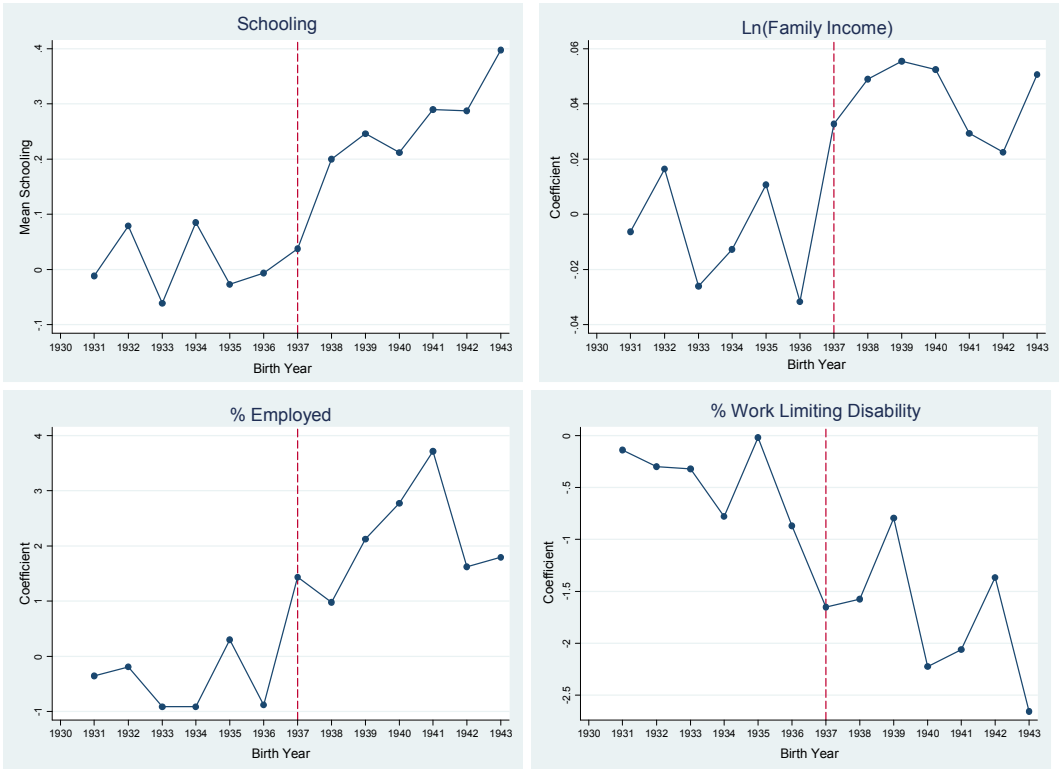

**Source:** US Vital Statistics

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



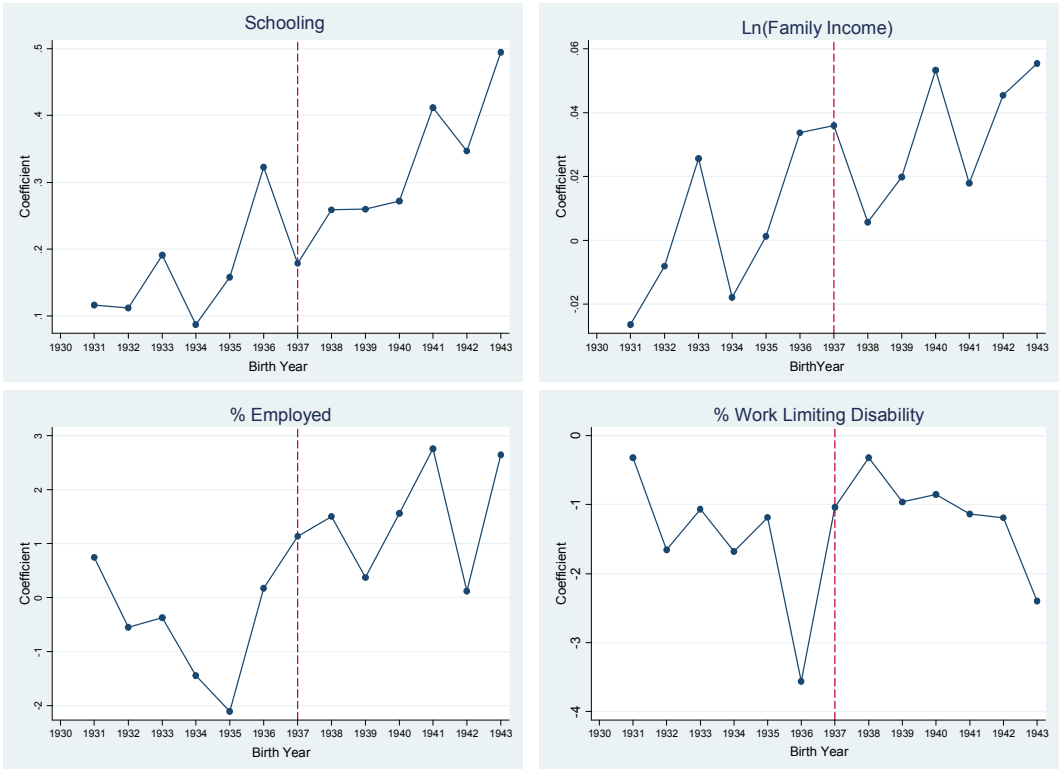

**Source:** US Vital Statistics

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



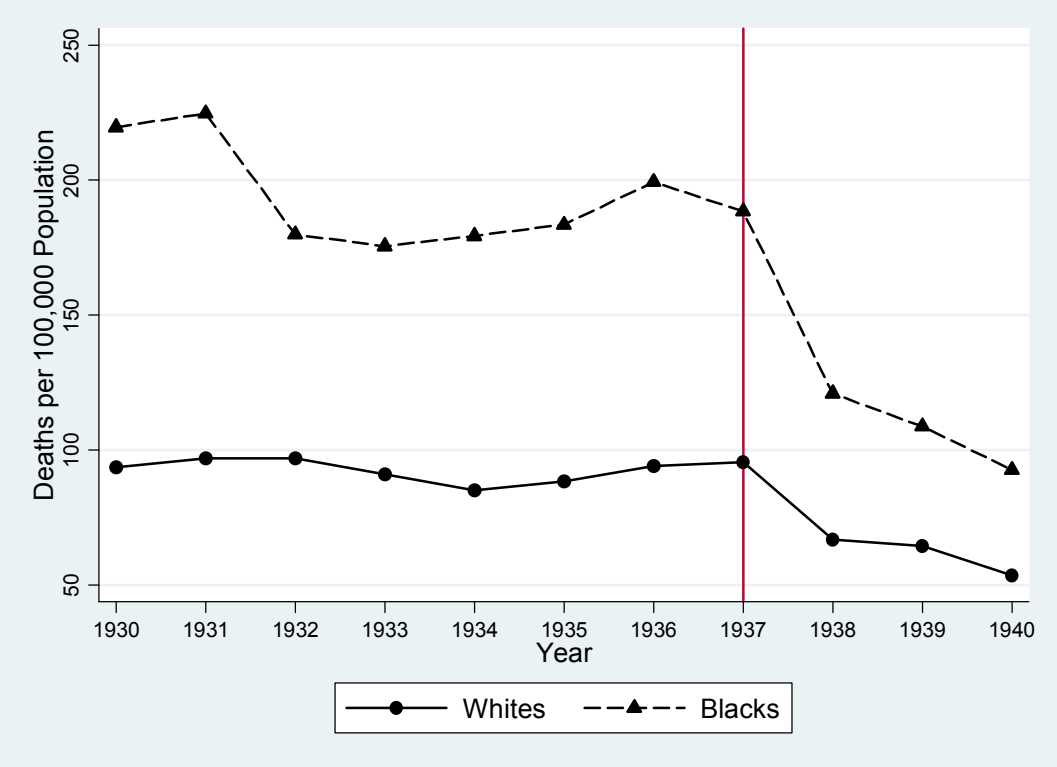

**Notes:** Each point reflects the estimate on the interaction between the marked birth year and *BaseRatePNA*. All models include birth state and birth year fixed effects, *Post*BaseRate(Control Diseases)*, and birth state X birth year macroeconomic and infrastructure variables (i.e., the same control 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/). 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 these 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.