Shadows of the Captain of the Men of Death: Health Innovation, Human Capital Investment, and Institutions

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Abstract: We leverage introduction of the first antibiotic therapies in 1937 to examine impacts of pneumonia in infancy on adult education, employment, disability, income and income mobility, and identify large impacts on each. We then examine how racial segregation in the pre-Civil Rights Era moderated the long-run benefits of antibiotics among blacks. We find that blacks born in more segregated states reaped smaller and less pervasive long run benefits despite sharp drops in pneumonia exposure. Our findings demonstrate causal effects of early life health on economic mobility and the importance of an investment-rewarding institutional environment in realization of the full potential of a healthy start.

Keywords: early childhood; race; human capital production; social mobility; dynamic complementarity; institutions; segregation; infectious disease; pneumonia; medical innovation; antibiotics; education; income, disability
JEL codes: I18, H41

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Introduction

The setting for our study is America in the 1930s and early 1940s, when pneumonia, an acute, highly morbid lower respiratory tract infection, accounted for one out of every ten deaths and, barring mortality from premature birth, was the leading cause of infant mortality (Linder and Grove, 1947; Wegman, 2001). The ubiquity and ruthlessness of the disease led the iconic physician Sir William Osler to coin pneumonia as the "Captain of the Men of Death." Pneumonia remains the leading cause of child death worldwide, killing 1.6 million children every year - more than AIDS, malaria, and tuberculosis combined. Despite this, only 20% of children who have pneumonia today are able to access antibiotics (World Health Organization, 2011).

We investigate the extent to which pneumonia in early childhood inhibits human capital accumulation and economic mobility by leveraging the introduction of the first antibiotics (sulfa drugs) in 1937, which led to sharp, widespread reductions in pneumonia morbidity and mortality (Greengard, et al, 1943; Lesch, 2007; Jayachandran, et al, 2010), especially among infants and young children. We further exploit the fact that states most burdened by pneumonia in the pre-sulfa era experienced the largest declines upon the introduction of sulfa drugs. In particular, we investigate whether the post-sulfa convergence in birth year levels of pneumonia mortality across the states after 1937 is mirrored in longer run socioeconomic outcomes for cohorts born in the sulfa era for whom adult outcomes are recorded in Census micro data for 1980-2000, effectively testing whether contemporary Americans carry the scars of exposure to pneumonia in their early years.

That early pneumonia may have such a long reach is motivated by insights from the biomedical literature: inflammatory responses provoked by infections both divert nutritional resources from child development towards achieving survival and directly impact neurocognitive and physical developmental pathways. Since infancy is a period of rapid physical and mental growth, it is plausible that severe or repeated infections generate permanent physiological changes that lead to poor health and cognition later in life, and ultimately to reduced lifetime income and wellbeing (Conti and Heckman, 2013; Crimmins and Finch, 2006; Eppig et al, 2010). This paper provides the first quasi-experimental analysis of the long run impacts of the availability of antibiotics. This is pertinent to contemporary debates concerning the pricing and distribution of available drugs, and to new investment in research and development necessitated by antibiotic resistance (Hollis and Pogge,

2008; Bhalotra and Pogge, 2014). Demonstrating the socioeconomic benefits of antibiotics stands to influence the setting of global health priorities.[1]

We also seek to make broader contributions to a growing literature documenting the long-arm of early health shocks or interventions.[2] We examine impacts not only on educational attainment, disability, employment, and income, but also on position in the income distribution, which previous research has tended not to elucidate. Given that pneumonia was concentrated among the poor (Britten 1942), identifying movements towards the top of the income distribution would demonstrate that improved early health not only helps individuals escape poverty but that it can also have impacts on intergenerational mobility. This distinction is of growing interest in understanding disparities in economic opportunity across the United States (Chetty et al, 2014), and our paper provides some of the scarce evidence of the causal mechanisms by which individuals might rise up from the poverty into which they were born. Our evidence is of particular importance given that race and state differences in infant health remain large but have not featured prominently in the recent literature on intergenerational mobility in the US (Chetty et al 2014; Chetty and Hendren, 2015; Putnam, 2015), with the exception of Chay et al. (2009).

Our third contribution is to assess institutionally-driven heterogeneity in the long run returns to infant pneumonia exposure. Specifically, we estimate variation in returns among blacks by indices of the intensity of racial segregation in their birth state. Previous research suggests that segregation restricted the net returns to human capital investment for blacks, by limiting access to quality schools and skilled jobs (Card and Krueger 1993; Donohue and Heckman, 1991). This may have depressed the socioeconomic gains from antibiotic availability for blacks in one or both of two ways. First, it may have directly reduced the income returns to improved early life physical and neurocognitive development. Second, segregation may have discouraged ensuing (reinforcing) human capital investments. We investigate the second channel by modeling changes in high school

[1] For instance the Advanced Market Commitment (AMC) is "an innovative financing mechanism that accelerates global roll out of the pneumococcal vaccine against the world's leading cause of child deaths". See http://www.gavi.org/support/nvs/pneumococcal/. Related previous research has tended to focus upon preventive measures rather than therapeutics (Bleakley 2007, 2010; Cutler, et al 2010; Lucas 2010).

[2] For instance, see Case, et al (2005), Almond (2006), Currie and Moretti (2007), Case and Paxson (2009), Bleakley (2007 and 2010), Barreca (2010), Cutler, et al (2010), Lucas (2010), Venkataramani (2012), Bhalotra and Venkataramani (2013), Bhalotra, Karlsson, and Nilsson (2015). Almond and Currie (2011a, b) survey this literature.

and college completion rates as a function of sulfa exposure,[3] and testing whether these educational investments were decreasing in the degree of segregation.[4]

Existing studies tend to bundle the effects of initial changes in the biological endowment with the effects of subsequent (endogenous) investments.[5] There is little evidence regarding the importance of any subsequent investments in driving the link between early life endowments and socioeconomic position in adulthood, possibly because credible identification requires two independent sources of exogenous variation, one that creates variation in the early life endowment and another that generates variation in subsequent investments (see Almond and Mazumder, 2013). We have attempted to tackle this issue by interacting quasi-experimental variation in infant endowments created by the introduction of antibiotics with historically determined variation across race and state in institutionalized segregation.[6]

The rest of this section elaborates our results. In Part I of the paper, we show that men exposed to sulfa-driven declines in pneumonia during infancy achieved significant improvements in the probabilities of high school and college completion, cognitive disability, work-related disability, employment, income and income rank. For instance, a one standard deviation decline in pneumonia exposure is estimated to have resulted in a (cohort averaged) 1.5% [1.2%] point increase in the probability of completing high school [college], a 1.5% increase in family income, a 0.47% point decrease in the probability of having an income in the lowest quintile and a 0.41% point increase in the probability of appearing in the top quintile of the adult income distribution. These findings demonstrate the relevance of reduced pneumonia (and access to antibiotics) for human capital accumulation and economic mobility. Back of the envelope calculations suggest that more than 80% of the increase in mean income in the sample of all men can be attributed to increases in high school and college completion.

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[3] We regard increased investments in education among post-sulfa cohorts as a marker of responsive investments; our specifications account for slowly moving secular changes in the supply of school and college facilities.

[4] In line with this, Thompson (2014) shows that desegregation raised maternal investments (cognitive stimulation) in children among Southern black mothers.

[5] If the technology of human capital formation is such that returns to investments later in life are increasing in the infant endowment, then reduced infectious disease will (by improving the infant endowment) tend to stimulate reinforcing investments, which contribute to realizing the full potential of early life interventions. This notion of dynamic complementarity has been formalized and incorporated into extensions of the Becker and Tomes (1986) model (Cunha and Heckman 2007; Heckman, 2007; Cunha, Heckman, and Schennach (2010)). Here, "endowment" refers to the stock of developmental capital in infancy, and it is modifiable.

[6] As discussed, we test whether sharp changes in high school and college attainment amongst post-sulfa cohorts, conditional upon controls for gradually evolving changes in supply, are decreasing in segregation.

In Part II, we estimate equations by race and show that there are significant segregation-gradients among blacks, alongside no similar gradients among whites. Black men born in the least segregated (Northern) states reaped substantial gains from infant exposure to sulfa drugs, while blacks born in the more segregated (Deep South) states saw muted gains. Even within the South, we find a gradient in intensity of segregation. The gradients are statistically significant for high school and college completion, cognitive disability, work-related disability, employment and poverty. The gradient is large but imprecisely determined for income but, alongside the somewhat indeterminate results for mean income, we identify significant gradients in position along the income distribution. This lines up with the segregation gradient in educational capital, suggesting that endogenous skill acquisition may have acted to complement the sulfa-led improvement in infant health. In particular, both income gains, and the contribution of education to income gains are decreasing in segregation.

Thus, across several outcomes and using different indices of segregation, we find that the institutions of segregation were associated with a capping of the long run socioeconomic benefits of the antibiotic revolution. This is evident in measures of investment and mobility, consistent with segregation restricting economic opportunities among blacks. We investigate and reject a number of alternative explanations of the gradients, including that they indicate that blacks in segregated states had limited access to sulfa drugs, that they arise spuriously from mortality rates being measured with more error in more segregated states, and that blacks in segregated states were negatively selected on account of migration or higher baseline mortality rates. Our findings contribute to a literature on the legacies of racial segregation (Card and Krueger 1993; Donohue and Heckman, 1991; Johnson, 2011; Neal, 2006; Smith, 1984, Aaronson and Mazumder, 2011), showing how segregation prevented Southern blacks from fully consolidating the dynamic benefits of reduced infectious disease in infancy. As such, the potential of a generation of black children born in the post-sulfa era went underutilized at a time when America was experiencing rapid, inclusive growth as a result of the expansion of state-financed education alongside skill-biased technological change (Goldin and Katz, 2008).

The paper evolves as follows. Part I profiles pneumonia mortality and morbidity in the 1930s United States, marking changes in infant pneumonia created by the sulfa drug revolution. After describing the data and research strategy, it presents estimates for the full sample, followed by a suite of robustness checks. Part II examines racial differences and gradients in long-run impacts by indicators for institutionalized segregation. It then investigates alternative interpretations of the striking heterogeneity in impacts among blacks. The final section concludes.

Part I. The Long-Run Impacts of Early Childhood Pneumonia

1. Pneumonia and the Sulfa Drug Revolution: Profile and First Stage

Pneumonia is an acute inflammatory disease of the lung characterized by fevers, shortness of breath, and cough, typically caused by bacteria and viruses (Mandell and Wunderlink, 2011). Bacterial pneumonia (about 50% of causes) is more severe and more likely to be fatal than its viral counterpart. In 1930, pneumonia accounted for 13.8% of all infant deaths (8.9 deaths per 1000 births) in the U.S., and 22% of infant deaths other than attributed to congenital defects, premature birth, and injury (Linder and Grove, 1947). Morbidity estimates from the U.S. National Health Survey of 1934-1936 underscore the fact that pneumonia was a disease of the very young, with a case rate of 3 per 100 infants, nearly twice as high as for 1-4 year olds and 10 times larger than for 10-15 year olds (see *Figure 1a;* Britten, 1942).

Pneumonia was a disease of the poor, with estimates from the same survey showing case rates that were twice as high among infants in poor households (Britten, 1942). However, these figures are thought to represent at least a two-fold underestimate of the true pneumonia burden during this era (Klugman and Feldman, 2009), and actual morbidity rates among infants from poorer families were probably similar to rates in today's developing countries, where it is estimated that there are between 15 and 28 pneumonia cases per 100 children under the age of 4 each year (Lopez et al., 2006; Rudan et al., 2004). Pneumonia was more prevalent in the American South and some parts of the West (*Figure 1b*), consistent with its known risk factors (poverty, overcrowding, and poor nutrition) being more prevalent in these regions in the 1930s (Klugman and Feldman, 2009; van der Poll and Opal, 2009).[7]

In the pre-antibiotic era, pneumonia was a long and trying illness, resulting in an average of 39 days of disability per patient for children under 15 (Britten, 1942). Some children were afflicted with multiple episodes. With reduced oral intake, high fevers, and inflammation, pneumonia was a challenging disease to overcome. Given the degree of morbidity it caused in the pre-sulfa era, it is

[7] While there is no previous quasi-experimental analysis of long run effects of pneumonia, a pioneering study in the literature analyzed long run impacts of the influenza pandemic (Almond 2006). Epidemic infection rates are some orders of magnitude larger than endemic rates. Influenza mortality increased four-fold during the flu epidemics, which is a much larger change than the change in pneumonia mortality that we analyze here (17% all-age and 30% infant). This is relevant insofar as there is some threshold below which population level impacts are not discernible, making it difficult to generalize the results of pandemic infection studies to the case of more subtle interventions in the disease environment such as associated with health campaigns, clean water programs or the distribution of medicines. Also, pregnant women were particularly vulnerable to influenza and Almond (2005) establishes long run impacts of fetal exposure. In our setting, as discussed below, pneumonia was most prevalent among infants and we trace impacts of infant exposure.

plausible that it could have discernible long-run effects, particularly since it hit hardest during infancy, a period marked by rapid physical and mental development.

Prior to the arrival of sulfa drugs, pneumonia was primarily treated with supportive care.[8] The seeds for antibiotic therapy were sown in 1932, when German chemists conducting experiments on textile dyes discovered the antibiotic properties of sulfonamides. The first scientific evidence of their potential was published in 1935, confirmed in clinical trials conducted in the following two years (Gibberd, 1937; Kiefer, 2001; Lesch, 2007; Long and Bliss, 1937). "Sulfa" drugs first became available in the United States in early 1937. They were relatively inexpensive and heavily promoted and, as a result, quickly adopted to treat a range of conditions. The ensuing "sulfa craze" lasted until the mass-availability of the first penicillins in the mid-1940s (Lesch, 2007; Jayachandran et al, 2010). The first sulfa agents, such as Prontosil, were partially effective against *Streptococcus pneumonaie*, the microbe responsible for the majority of bacterial pneumonias; in 1938 a more effective agent, sulfapyridine became available for clinical use. Clinical trials of sulfapyridine showed reductions of 50-70% in pneumonia case fatality rates among inpatients (Evans and Gaisford, 1938; Gaisford, 1939; Lesch, 2007).

Consistent with the findings of small clinical trials, sulfa drugs had large impacts on mortality from pneumonia at the population level. Jayachandran et al, (2010) demonstrate a structural break in the time series data for all-age mortality from sulfa treatable diseases in 1937, which is evident in *Figure 2A*. They estimate that sulfa drugs led to a 17% decline in all-age pneumonia mortality. Importantly, for our purposes, we show in *Figure 2B* that the largest decline, nearly 30%, accrued to infants, consistent with their higher infection rates. The trend break in the infant pneumonia rate is statistically significant (*Appendix 2, Table 1).* In *Figure 3*, we show that larger *absolute* reductions in pneumonia mortality were seen in states with higher pre-sulfa drug era disease burdens. This pattern is evident for all age groups (*3A*) and, again, stronger among infants (*3B*). We exploit the implied convergence across states after 1937 in our identification strategy, discussed below.

In addition to reducing mortality, there is strong evidence that sulfa drugs led to reductions in the severity of pneumonia episodes (Connolly et al., 2012). Clinical trials on infants and children from the era noted rapid improvements in fever, mental status, and other physical examination

[8] Intravenous serum therapy, where antibodies to the bacteria infecting a patient were harvested in animals and introduced into the patient intravenously, was introduced among hospitalized patients in the early 1930s (Lesch, 2007). While this was successful in certain contexts (Finland, 1960), it was not widely utilized and appears to have had no impact on pneumonia mortality rates at the population level (*Figure 2*). Serum therapy was *less likely to be used in infants and young children* given greater difficulty in administration and more pronounced side effects (Connolly, et al, 2012).

findings, demonstrating that the average inpatient case of pneumonia was shorter and followed a much less severe course as a result of sulfa drug therapy (Greengard et al, 1943; Hodes et al, 1939; Moody and Knouf, 1940; Smith and Nemir, 1939). In addition to these profound impacts on hospitalized patients, sulfa drugs also led to reductions in pneumonia morbidity in the community, where roughly 70% of cases were treated in the mid-1930s (Britten 1942) because they were readily available and utilized by laypersons and community physicians (Lesch, 2007; Lerner, 1991).

2. Data and Research Strategy

2a. Data and Baseline Framework

Identification of causal effects of early life pneumonia on adult socioeconomic outcomes is challenged by selectivity in infection. We address this challenge by using the sharp birth cohort variation in pneumonia exposure created by the arrival of sulfa drugs in 1937 (*Figure 2*). We further exploit the fact that states most burdened by pneumonia in the pre-sulfa era experienced the largest declines upon the introduction of sulfa drugs (*Figure 3*), using an approach similar to that in Acemoglu and Johnson (2007) and Bleakley (2007). In particular, we investigate whether the post-sulfa convergence in birth year levels of pneumonia mortality across the states after 1937 is mirrored in longer run socioeconomic outcomes for cohorts born in the sulfa era.

The data for adult outcomes comes from the 1980, 1990, and 2000 5% public use microdata samples of the United States Census (Ruggles et al, 2010). Cohorts born in 1937, the year sulfa drugs became available, were 43, 53, and 63 years old during the census enumerations, respectively. The outcome variables are years of schooling, high school and college completion, employment status, work limiting or preventing disability, cognitive disability, physical disability, poverty status, log family income and income mobility referenced to the income distribution for pre-sulfa cohorts. Further discussion of data and variables and all descriptive statistics are in *Appendix 1*. The baseline specification is:

$$Y_{istc} = \alpha + \beta * post_t * base\_pneumonia_s + \theta_{rs} + \eta_{rt} + \lambda_{rc} + \gamma \mathbf{X}_{st} + \theta_s * \eta_t + e_{istc} \qquad (1)$$

Y_{istc} denotes an outcome recorded in adulthood for individual (*i*) of birth state (*s*) and birth year (*t*) observed in census year (*c*). The outcomes are indicators of human capital and income. $Post_t = 1$ for cohorts in their infancy in 1937 and thereafter. The pre-sulfa pneumonia mortality rate in the birth state is denoted $base\_pneumonia_s$ and defined as the average state-specific, all-age combined pneumonia and influenza mortality rate during 1930-1936. $Base\_pneumonia_s$ captures treatment

intensity since pneumonia reduction after 1937 was increasing in the base rate (*Figure 3*).[9] We expect $\beta>0$ (for desirable outcomes) if adult outcomes mirror this pattern.[10]

The Greek letters represent race-specific fixed effects for birth state, birth year, and census year. We also control for a vector of relevant birth state and birth year varying observables ($\boldsymbol{X}_{st}$), which we detail below, and birth-state specific trends ($\theta_s*\eta_t$) to account for potential unobservables. Standard errors are clustered at the birth state level to account for serial correlation in the outcomes (Bertrand et al, 2004). We restrict the sample to birth cohorts 1930-1943 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. The estimated equation is the reduced form of a system in which adult outcomes depend upon pneumonia exposure at birth and the latter is instrumented with the sharp arrival of sulfa drugs, the impact of which varies across states as a function of their pre-sulfa pneumonia burden.

2b. Threats to Inference

In this section, we discuss our strategy for controlling for potential omitted birth state and birth year varying variables. After we present the main results, we will present specification checks on measurement error in baseline mortality rates (all-age *vs* infant rates, pneumonia *vs* pneumonia combined with influenza), mean reversion, pre-trends and age at exposure (testing for impacts at ages other than infancy), selectivity in migration and fertility, survival selection, and sensitivity to the range of census years and sample cohorts (including robustness to excluding the war cohorts).

Diseases not treatable with sulfa drugs*:* A potential concern is that *post*base_pneumonia* may pick up sudden improvements in health arising independently of, but coincident with, sulfa drug availability, such as state-specific public health interventions, improvements in sanitation, and general living conditions. As a check against this we control for trends in diseases not treatable with sulfa drugs ("placebo diseases") on the premise that omitted factors will not have discriminated between sulfa-treatable and sulfa-untreatable diseases (contained in $\boldsymbol{X}_{st}$). We interact *post* with pre-

[9] Our identification strategy uses the timing of the arrival of the sulfa drug technology at the national level instead of their availability at the state level as any state differences in adoption rates are likely to be endogenous. In this regard, our approach is similar to that of Jayachandran, et al (2010) and Acemoglu and Johnson (2007), Bleakley (2007, 2010), Cutler et al. (2010), and Lucas (2010). Jayachandran, et al., show that structural breaks by state are in a tight interval around 1937 (1936-1938), and the 2008 working paper version of their paper shows that the break is in 1937 for the pneumonia mortality rate, although in 1938 for the logarithm of the rate. Using our narrower sample period, we confirm a break in 1937. See *Appendix 2 Table 1*.

[10] State-level morbidity data are not available so we follow a tradition of using mortality rates to proxy for disease exposure (Bozzoli, et al, 2009).

sulfa birth state specific mortality rates from diarrhea (under the age of 2), malaria, heart disease, tuberculosis, and cancer and include these as controls.[11]

Other diseases treatable with sulfa drugs: Sulfa drugs led to marked declines in conditions other than pneumonia, most notably scarlet fever, erysipelas, meningitis and puerperal sepsis (Jayachandran et al, 2010). Thus, the coefficient on $post_t$*$base\_pneumonia_s$ may have loaded on to it the effects of reductions in these omitted sulfa-treatable diseases. However, scarlet fever, erysipelas and meningitis accounted for a negligible fraction of infant and all-age mortality.[12] In contrast, puerperal sepsis accounted for 40% of maternal deaths in 1930, maternal mortality was high, at almost 7 deaths per 1000 births and large absolute reductions in maternal mortality occurred with the arrival of sulfa drugs (Jayachandran et al, 2010; Thomasson and Treber, 2008). We therefore control for $post_t$*$base\_maternal\ mortality$.[13]

State economy and infrastructure: Since our estimates use the cross-state convergence in pneumonia created by the introduction of sulfa drugs *(Figure 3)*, convergent pre-trends across high-base and low-base states prior to 1937 are a concern that we investigate with an event study design *(Figure 4)*. In any case, we control for birth state and birth year varying socioeconomic variables including per capita income, public health spending, and the numbers of schools, hospitals and physicians (contained in $\boldsymbol{X_{st}}$).[14] Controlling for income per capita in the birth-state is pertinent given changes in economic fortunes during the Depression Era, even if these have been shown to have had little, if any, impact on long-run health and economic outcomes (Cutler et al, 2007). In addition to including birth state specific time trends (θ_s*η_t above), we test robustness to the census division*birth year fixed effects, motivated in part by convergence in economic development

[11] The control for diarrhea is powerful as it was the second leading cause of post-neonatal death during the study era, sharing risk factors to pneumonia (Bhutta, 2014), so it will account for unobservable trends in health specific to children. Malaria was declining significantly during the study period (Barreca et al, 2012) and, like pneumonia, more prevalent in the South. Non-communicable disease trends control for health care quality and access.

[12] In 1930, the number of infant deaths in 1000 live births from these diseases was 0.1, 0.3 and 0.2, compared with 8.9 from pneumonia (Linder and Grove, 1947). We nevertheless included controls for post-1937 changes in other sulfa-treatable diseases, and this did not alter the coefficient of interest; see *Appendix 2, Table 3*.

[13] Maternal mortality decline at the population level may incentivize parents to raise investments in girls' education, which would exhibit as improvements in long-run outcomes for women vis-a-vis men (Jayachandran and Lleras-Muney, 2009). Our estimates test for this and find no evidence of it. At the individual level, there are consequences for child development from a mother dying and additional impacts from changes in family size that arise because of declining infertility, a potential complication of post-partum sepsis. However, we do not expect *direct* effects of MMR decline on children because the maternal post-partum infections (puerperal sepsis) that were controlled with sulfa drugs were rarely transmitted to infants.

[14] The estimates are not sensitive to whether we directly include the characteristic (X_{st}) or we include *post-37* multiplied by the pre-intervention level of the characteristic ($post_t$*X_s) to allow for discontinuous effects of X_{st} that may otherwise load on to the variable of interest.

between the US South (which was particularly plagued by pneumonia) and other parts of the country during the 20th century (Mitchener and MacLean, 1999).[15]

3. Results

3a. Main Results

See *Table 1*. Each cell reports estimates of the coefficient on $post_t$*$base\_pneumonia_s$ from separate regressions, varying by outcome and controls. The equations were estimated by gender. We find that exposure to sulfa drugs, and thereby reduced exposure to pneumonia in the birth year, led to statistically significant improvements for men in all outcomes investigated, other than physical disability. For instance, column 4, suggests that a one standard deviation decrease in pneumonia mortality (0.19) was associated with the following impacts for men: a 0.1 increase in years of schooling, a 1.5% point and a 1.2% point increase in the probabilities of completing high school and college respectively, a 1.5% increase in family income, a 0.47% point decrease in the probability of being below 200% of the federal poverty line, a 0.43% point increase in the probability of being employed, and a 0.62% point decrease in the probability of each, reporting a work-related disability and cognitive disability. The statistical significance of our findings is robust to adjusting for multiple hypotheses testing following Aker et al (2014). See *p*-values in *Table 1*, procedure in *Appendix 2*.

We investigated impacts on position in the adult income distribution by assigning reported family incomes to pre-sulfa income quintiles[16]. The results in *Figure 5* show a significantly lower probability of appearing in the lowest quintile (0.47% points for a 1 s.d. decline in *base_pneumonia*) and an increase in the probability that sulfa exposure led to a position in the top income quintile (0.41% point increase), with statistically insignificant effects for membership of the intervening quintiles (see *Appendix 2 Table 1*). Our findings suggest that availability of treatment for a disease of poverty not only helped individuals escape poverty later in life, but also helped them ascend to the top of the socioeconomic ladder.

To assess the extent to which increases in schooling can explain the observed increases in family income, we estimated Mincerian returns for the sample cohorts in the 1980 census, finding

[15] We note that including this rich set of controls may amount to "over-controlling." For example, an immune system weakened by one infection is more likely to contract other infections, creating population level correlations in disease trends. 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. We therefore assess robustness of the coefficients to sequential addition of controls and display a suite of coefficients.

[16] Specifically, for each census wave, we computed the quintiles of the family income distribution for birth cohorts born in 1930-1936. We then assigned the 1937-1943 birth cohorts to one of these income quintiles based on reported family income in that census wave. We created 5 binary indicators denoting each income quintile. A 1 was assigned to the binary variable corresponding to the individual's income quintile and 0's assigned to the other dummies. See *Appendix 1* for details.

returns of 12% for an additional year of schooling. Applying this to the observed increases in education and income among post-sulfa cohorts suggests that more than 80% of the income increase can be accounted for by increased schooling.[17] In Part-II, we delineate contributions of education to income mobility by exploiting the finding of differential responsiveness of education across different race*state groups.

The 1.5% increase in mean income flowing from a 1 s.d. decrease in pneumonia mortality implies a benefit-cost ratio of over 10, assuming that a course of sulfa drugs cost $50 in 2008 US dollars per episode.[18] To approximate average impacts for those who contracted pneumonia and accessed antibiotics (ATT), we factor in estimates of the pneumonia morbidity burden. Using a rate of 15 cases annually per 100 children, which is similar to pneumonia attack rates in today's developing countries, this would imply an inflation of the effect estimates by a factor of 6.7 (assuming that returns to pneumonia therapy were the same across the distribution of attributes that influenced the risk of contracting the disease). The ATT estimates imply, for instance, gains of 0.7 years of schooling, 10 and 8 percentage points respectively in the likelihood of completing high school or college, and 10% in family income. While these impacts are large, they are also plausible, given that the median spell of pneumonia created more than a month of disability per patient in the year before sulfa drugs arrived (Britten, 1942) and that children often had recurrent spells.[19]

The results for men are in general robust to successive inclusion of the controls allaying concerns that they are driven by an unobserved convergence process. The estimated coefficients for women are not robust to inclusion of state socioeconomic variables and state specific trends (*Appendix 3, Table 1*). One explanation for the difference is that men were more susceptible to early life pneumonia (Gluckman and Hanson, 2005; Low, 2000; Waldron, 1983), and hence benefitted more from sulfa drugs; Britten (1942) shows that pneumonia case rates were about 30% higher for

[17] Post-sulfa cohorts exposed to a 1 s.d. reduction in pneumonia in infancy attained an additional 0.1 years of schooling (*Table 1*), so a 12% rate of return predicts a 1.2% increase in income which is 80% of the 1.5% increase in income. Mincerian estimates indicate that completion of high school, possibly with some college, was associated with a 35% increase in wages relative to not completing high school, and that completing college and above was associated with a 76% increase in wages relative to not completing high school. We estimated that a 1 s.d. reduction in pneumonia led to a 1.2% point increase in college completion (*Table 1*), which projects to a 0.9% increase in family income, which is about 75% of the estimated income effect in *Table 1*. The corresponding estimate for high school completion is a 0.5% increase in family income which is 25% so that together college and high school increases explain all of the increase in income.

[18] The estimated range is $35-$100 (Jayachandran et al., 2010). This is a crude estimate that ignores private and social costs of drug acquisition and development that were not reflected in prices.

[19] Our estimates are not large relative to related estimates in the literature. Malaria eradication is estimated to have led to a 15-27% increase in wage income (Bleakley, 2010) and about a 3-year increase in schooling (Lucas, 2010). Deworming in primary schools is estimated to have generated a 21-29% increase in income (Baird et al., 2011).

male infants. We investigate the role of limited returns to human capital investment for women (such as marriage bars and reduced labor market opportunities; see Goldin, 1991), but find no clear evidence of this. We also investigate marriage and fertility outcomes, but again find no impacts for women (*Appendix 3, Tables 2 and 3)*.

3b. Additional Robustness Checks

Measurement error: The estimates in *Table 1* use the all-age pneumonia and influenza mortality rate averaged over the pre-sulfa period, 1930-36. In *Appendix 2,* we explain that this choice was made to mitigate measurement error. We also show that the "portmanteau" term transmits the relevant signals: the post-1937 drop in the compound rate was driven by pneumonia (not influenza), and it was sharper among infants. We nevertheless investigated sensitivity of our estimates to re-specifying *base*. In Panel A of *Table 2*, we replace the compound rate with the pneumonia mortality rate in 1935. In Panel B, we use the infant rate, instrumenting with the all-age rate. The results are robust to these variations. In addition, our results are robust to estimating the 2SLS equivalent of the baseline model, where the birth state and cohort varying mortality rate is instrumented with *post-1937*base* (*Appendix 2, Table 3*).

Mean reversion: Since our strategy uses the cross-state convergence in pneumonia created by the antibiotic revolution, we investigated whether our findings are driven by mean reverting shocks. The results are robust to including the pre-1937 (1930-1936) average of the outcome variable (at the birth state X birth year X race X gender X census year level) in the equation (*Table 2, Panel C*).

Age of exposure and placebo interventions: The baseline specification models pneumonia exposure during infancy, but exposure at other ages may also have long run impacts.[20] So we replaced *post-1937* with a vector of dummies denoting the individual's birth cohort (grouped into two-year cohorts to enhance precision), and graphed coefficients on the interactions between each cohort dummy and $base\_pneumonia_s$ (*Figure 4*), controlling for state disease environment and macroeconomic variables (as in column 3 of *Table 1*).[21] The coefficient series for men show a trend break starting in 1937, the year of introduction of sulfa. We find no evidence of earlier trend breaks, which provides a falsification test (also see *Appendix 2, Table 4*). Moreover, the confidence intervals around the pre-1937 coefficients include zero and do not show a trend. This has two implications.

[20] In contrast to the case of the 1918 flu pandemic (Almond 2006), we do not expect impacts from fetal exposure because mothers of childbearing age experienced very low infection rates (Britten, 1942). However we cannot *a priori* rule out effects of being exposed at ages beyond infancy.

[21] The coefficient plots look similar if these controls are not included.

First, it provides further evidence that infancy is a "critical period" for interventions designed to control the pernicious long run effects of infectious diseases and in particular pneumonia (Almond and Currie, 2011a, b; Barker and Osmond, 1986; Eppig, et al, 2010).[22] Second, conditional on controls for other diseases and state macroeconomic conditions, it implies there were no pre-existing trends in the outcomes. We use these findings alongside the age profile of pneumonia (*Figure 1a*) to extend equation (1) to a "**triple difference**", which allows us to control more effectively for unobserved shocks that are common across the age distribution. We exploit the fact that the risk of contracting pneumonia peaks in infancy and is close to its minimum at age 10-15, to test whether we see smaller, if any, long run benefits of sulfa drugs for individuals who were first exposed when aged 10-15 (*Appendix 2, Figure 2*). This richer specification delivers similar estimates for infant exposure to those in *Table 1 (Table 2, Panel D).*

Selection: *Selective Migration* - Since the US census records state of birth, we are confident that we correctly match adult outcomes to pneumonia exposure in the birth year. However, birth state, and therefore exposure to pneumonia at birth may be endogenous if parents select the birth state of their children. Previous studies of the long run effects of early life interventions tend to regard the birth region as exogenously given, but this is questionable.[23] If potential parents who move to low pneumonia states in order to improve the life chances of their births are positively selected, this creates a compositional effect at baseline that reinforces the tendency for low pneumonia states to have better outcomes, but this is absorbed by state fixed effects. The introduction of sulfa drugs, by virtue of narrowing state differentials in pneumonia, will have attenuated disease-led migration flows and this will exhibit as smaller *relative* improvements in long run outcomes in low pneumonia states after 1937. Since our test is of post-1937 convergence in outcomes across states, a possible concern is that some of this reflects migration-led changes in population composition. We investigate this using information on 20-40 year olds in the 1930 and 1940 census files and find that, conditional upon state income, there is no evidence that migration

[22] The fact of developmental plasticity at this age creates the possibility that the damage created by nutritional stressors, such as infections, is irreversible. Consistent with our finding that pneumonia exposure matters after birth, Bozzoli et al, (2008) identify a significant impact of post-neonatal mortality rate on adult height which they argue is largely driven by pneumonia. They use longitudinal data for the 1950-1980 birth cohorts in twelve OECD-member countries, which illustrate the continuing relevance of pneumonia worldwide.

[23] Montalvo and Reynal-Querol (2007) analyze the *reverse* process, how levels of infectious diseases respond to migration patterns. That migration may respond to the disease environment is indicated in Mesnard and Seabright (2009) but we are not aware of tests of this.

induced by the disease environment may be driving our results (*Appendix 2, Table 5*).[24] *Selective fertility* - If heterogeneity in fertility responses to sulfa drugs altered the composition of births in favor of low-risk births, this would offer an alternative explanation of improved long run outcomes. We investigate this using data from the 1950 census and, conditional upon the controls in column 4 of *Table 1,* we find no evidence of endogenous changes in sample composition post-1937 (*Appendix 2, Table 5*). *Selective Survival* - With the advent of sulfa drugs, a greater fraction of frail children who would previously have succumbed to pneumonia survived. As the marginal survivor will tend to have been negatively selected, failing to account for this (as in most studies of long run outcomes) will tend to bias our estimates *downwards* (Almond, 2006; Bozzoli et al, 2009). Since selection may play a larger role in determining the racial differences that we document in the next section, we take a more formal approach to assessing it the second part of the paper.

Part II. Race-Specific Impacts and the Role of Institutions

1. Race-Specific Impacts

Estimates for white and black men are in *Table 3* (race-specific estimates for women are in *Appendix 3, Table 4*). The estimates for white men are similar to those for all men, given their numerical preponderance in the sample. For black men, we find similar increases in high school completion and larger reductions in poverty and cognitive disability, while estimates for other outcomes, including college and income, are imprecise. This imprecision may reflect measurement error in mortality rates or heterogeneity in access to sulfa drugs in the black sample. We investigated these concerns as follows.

Measurement error in mortality rates for blacks: The state-level pneumonia mortality rate is a noisier measure for blacks because they are a minority population in all states. Moreover, birth and death registration was known to be incomplete in rural Southern states, where the majority of blacks resided (Puffer, 1937; Shapiro and Schachter, 1952; Ewbank, 1987); see *Appendix 4, Figure 2*. While we cannot fully address what is effectively an omitted variables problem (see Bound, et al, 1994), we attempt to account for measurement quality as follows. Since mortality rates are a ratio of deaths to births, we used the year in which each state became part of the National Vital Statistics registration system for a) births and b) deaths. In *Appendix 4* (*Figure 2*), we validate these measures,

[24] Here we discuss selective migration of parents of our sample cohorts as this stands to bias the coefficient of interest. Selective migration of the sample cohorts *themselves* is an outcome and a potential mechanism explaining long run effects on other outcomes. We investigated this too, and we find no significant tendency for sulfa-exposure in infancy to encourage cross-state migration (*Appendix 2 Table 6).*

showing that plausible indicators of mismeasurement are a function of the year of entry variables. For births, we use data from a nationwide audit conducted in 1940 (Shapiro and Schachter, 1952) to derive the percentage of births registered by the state system as a proxy for the quality of birth registration.[25] For deaths, we use the national share of death certificates with no listed cause of death (Linder and Grove, 1947). As our concern is with bias in the coefficient on *post*base_pneumonia* given non-random measurement error in *base_pneumonia*, we introduce in the regressions, the terms *post*birth-registration* and *post*death registration* to proxy for the quality of mortality statistics. The main effects of birth and death registration years are absorbed by state fixed effects.[26] A detailed discussion of the proxies and our approach is provided in *Appendix 4*.

Access to sulfa drugs: We also investigated the possibility that the coefficients in the black sample are attenuated because blacks had more limited access to sulfa drugs when they were introduced. We establish that blacks had access to sulfa drugs, showing that they experienced sharp absolute declines in pneumonia mortality after 1937; see *section 4* below (and *Appendix 4*, where we also situate the plausibility of this result in its historical context). We nevertheless control for a proxy for state variation in access, namely, the number of pharmacists per 1,000 black population residing in majority black counties in 1940 (averaged, with population weights, over majority black counties in each state, noting that counties at the time were either predominantly black or white). Pharmacists met the bulk of demand for sulfa drugs during our study period (particularly through 1939, as sulfa drugs did not require a prescription through this time), so this is a measure of the cost of obtaining sulfa drugs among the black population (Lerner, 1991; Lesch, 2007). If access was correlated with variation in pre-intervention pneumonia mortality rates (plausible if, for instance, both were associated with being rural) then omitting to control for differences in access may bias the coefficient on *post*base_pneumonia*. We therefore control for *post*pharmacists,* allowing the main effect of pharmacists to be absorbed by the state fixed effects in the model.

Including the controls for state heterogeneity in measurement error in mortality rates and in access results in larger and more precisely determined coefficients for black outcomes, and the coefficient for income becomes statistically significant (*Table 3*). *Table 3a* presents estimates for

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[25] A dominant driving force behind incomplete *death* registration was poor enumeration of *births* that occurred outside of the hospital. Birth registration systems improved markedly between 1940 and 1950, both because of increasing shares of births in hospitals *and* improved enumeration of residual births not occurring in hospitals. See Shapiro and Schachter (1952). *Appendix 4 Figure 2F* plots the correlation of year of entry to the national birth and death registration systems, also illustrating the range across states in years of registration.

[26] See discussion in *Appendix 4*. Briefly, if *y* depends upon (*post*[base+e]*) where *e* is the error in measurement of *base*, then *y* will depend upon (*post*base*) and (*post*e*). Omission of *(post*e*)* will bias the coefficient on (*post*base*) if *base* is correlated with *e*. In *Appendix 4 Figure 5* we plot this correlation and show that it is positive.

income mobility. Sulfa exposure is associated with a significant reduction in the chances that black men have incomes in the bottom three quintiles (the quintiles are constructed for the all-race income distribution in the pre-sulfa years). There is also a significant increase in the chances of an income in the second highest quintile.[27] So, overall, the introduction of antibiotics led to significant income mobility among black men but not as much as for white men, who were more likely to achieve incomes in the top quintile (see *Figure 5* and *Appendix 2 Table 1* for all-men; the figures for white men are very similar as they numerically dominate the sample).[28] Having documented substantial (immediate and) long run gains from sulfa for black men on average, in the next section we investigate heterogeneity in the socioeconomic benefits of antibiotic exposure for blacks as a function of birth state segregation.

2. The Role of Institutions – Context, Data, and Research Strategy

It is well known that segregation-era policies in the American South limited the upward mobility of blacks (Margo, 1990; Aaronson and Mazumder, 2011; Card and Krueger, 1992; Chay, et al, 2009; Donohue and Heckman, 1991; Smith, 1984; Welch, 1974). Following the passage of the Jim Crow Laws in the post-Civil War Reconstruction era in the late nineteenth century, racial segregation was institutionalized in all public facilities in the U.S. South. While the mandate proposed "separate but equal" status for black Americans, in practice it systemized their economic and social disadvantage, effectively raising the costs of acquiring human capital and lowering the return to human capital for blacks relative to whites. The extent of segregation was weaker in the North and, within the South, it varied across the states. Here, we utilize this historically determined variation across race and state in the intensity of segregation to identify the extent to which it dampened educational investments and earnings gains flowing from improved infant endowments in the post-sulfa era. Specifically, we estimate the following equation by race:

$$Y_{irstc} = \alpha_r + \beta_{1r}*post_t*base\_pneumonia_s*segregation_s + \beta_{2r}*post_t*base\_pneumonia_s + \beta_{3r}*post_t*segregation_s + \theta_{rs} + \eta_{rt} + \lambda_{rc} + \gamma_r X_{st} + \theta_{rs}*\eta_{rt} + e_{irstc} \qquad (2)$$

[27] This is a fairly remarkable degree of social mobility, possibly facilitated by the fact that our sample birth cohorts were exposed to a boom in college education and in job opportunities. They completed their school and college choices before the Civil Rights Act of 1964, but they were exposed to its benefits on the labor market. While this may contribute to explaining how large the mobility gains were (in the absence of birth-state segregation), it is not a confounder in our analysis because the thriving economic environment and the equalization of opportunities were shared by neighboring cohorts untreated by sulfa (who did not experience these mobility gains), in other words, there was no structural break in the environment for post-1937 birth cohorts. See *Appendix 4*.

[28] The percentage point changes are larger for black men than for all men (read, "white men") but, since black infants had much higher pneumonia exposure than white infants (perhaps up to threefold), this is reflected in the cohort averaged-estimates. Indeed, the inflation factor for the ATT estimates would be a third to half of that for all men, making the estimates plausible.

Y_{irstc} is a long run outcome recorded for individual *i* of race *r* in census year *t* for birth cohort *c* born in state *s* and the remainder of the notation is as in *equation 1*. The coefficient β_{1r} on the new term, $post_t$*$base\_pneumonia_s$**segregation* is the segregation-gradient in long run returns to reduced infant pneumonia associated with introduction of sulfa-drugs in 1937. For desirable outcomes (like income rather than poverty) for which $\beta_{2r}>0$, finding that $\beta_{1r}<0$ for blacks will confirm our hypothesis that segregation dampened the long-run impacts of early life exposure for blacks. The term *base*segregation* is absorbed by state specific fixed effects.

Identification: Although segregation is predetermined, inference using this approach is hampered if segregation is not only a proxy for net returns to investment for blacks, but also correlated with relevant omitted variables. We therefore analyze potential omitted variables including race*state differences in access to health care, measurement error in mortality, and selectivity in migration and survival. All controls are effectively race-specific as equation (2) is estimated by race - in this discussion we highlight the implicit interaction with race. The fixed effects of segregation in the birth state are captured in *race*state* specific fixed effects θ_{rs}, so β_1 will not simply reflect that outcomes like education and income were worse among blacks in segregated states. The term $post*segregation_s$ captures any convergence or divergence in outcomes after 1937 between states with different degrees of segregation that may have occurred independently of changes in pneumonia prevalence, for instance, North-South economic convergence. We allow for more gradual changes in the relationship between segregation and long-run outcomes over time by including race*state linear trends and race*birth cohort fixed effects. Importantly, the gradient estimates are conditional upon race*state*birth cohort variation in per capita income, schools, hospitals, physicians, and mortality from six "control diseases". So if, for example, blacks in more segregated states experienced different trends in access to public health, or higher mortality rates, this would be captured by these controls. Note that although *base_pneumonia* was higher in more segregated states, there was considerable independent variation in *base_pneumonia* and *segregation* (*Appendix 4, Figure 3A*).

White men provide a "control group" for our analysis of segregation. If estimates of equation (2) for whites showed gradients of the same sign as for blacks, then the gradients may reflect a pan-racial process (i.e. not racial segregation) that differentially drives the returns to early life endowments in more *versus* less segregated states. In summary, unobservables that threaten our identification would have to vary by birth cohort and race and line up with the same pattern as state-

specific differences in pre-sulfa pneumonia burdens *and* state-specific differences in segregation.[29] Unobservables of potential concern are race*state heterogeneity in measurement of *base_pneumonia* and in access to sulfa drugs, as both may have been worse for blacks born in more segregated states, although they would also have to have evolved differently after 1937. Below, we attempt to account for this and we also analyze selective migration and mortality.

Measures of segregation: First, we use an indicator for birth in the U.S. South where segregation was *de jure* (see *Appendix 1* for a list of states included in the South). Segregation outside the South (henceforth "North" for expositional ease) was *de facto* and weaker. Previous work has documented North-South gradients in school access, school quality, labor market opportunities, and rates of return to skill for blacks (e.g., Donohue and Heckman, 1991), and we confirmed using the 1940 census that there were large differences in the racial education and wage gaps between the North and South (*Appendix 4, Table 2*).

In order to more definitively isolate the role of race-specific institutions, we use as a second indicator the birth state share of slaves in the population in 1860 (Nunn, 2008). This has been shown to be predictive of the quality of public schools, black suffrage, and racial gaps in education and labor market productivity in contemporary America (Engerman and Sokoloff, 2005; Mariscal and Sokoloff, 2000; Bertocchi and Dimico, 2010; Sacerdote, 2005; Mitchener and MacLean, 2003). The historical slave share is effectively zero in the North and ranges from 0.01 to 0.57 in Southern states. So, it effectively "nests" the variable "South" but augments it by introducing variation in the degree of segregation within the South.

Since an interpretation we have of segregation is that it capped returns to schooling, the third indicator we use is the state-level ratio of income returns to schooling for 25-55 year old black *versus* white men which we calculate from 1940 census micro data for the 15 states in which blacks constituted at least 10% of the population (see *Appendix 1*). We present results for all three measures to show that the story we tell is not sensitive to the measure used. In further tests, we use the slave share measure as it nests the South/North indicator. While the returns measure offers a consistency check on interpretation, it is only defined for a sub-set of states, and relative returns were not strictly exogenous to choices made by white and black families, whereas the historical slave share clearly was. In *Appendix 4*, *Table 3* and *Figure 5* show correlations between slave share, relative returns,

[29] Potential confounders of the process we model would therefore have to have changed discretely after 1937 (and in a manner that favored positive outcomes Y), changed more in states with higher pre-1937 burdens of pneumonia, *and* changed in a manner that favored whites over blacks in segregated states more than in un-segregated or less segregated states.

income ratios and education ratios by race, all of which are markers of black disadvantage in human capital accumulation and income returns.

3. The Role of Institutions - Results

Estimates of equation (2) reveal a starkly defined and systematic tendency for the long run returns to blacks from infant exposure to antibiotics to be decreasing in segregation in the birth state *(Table 4)*. This holds across a range of outcomes and for each of three indicators of segregation, and there are no similar segregation gradients for white men *(Appendix 4, Table 4)*.[30] So, for white men, the introduction of sulfa drugs stimulated convergence in pneumonia across the states (*Figure 3*), which was mirrored in outcomes. However, among black men, despite convergence in pneumonia (which occurred even within the South -see *Appendix 4, Figure 7*), we see a divergence in outcomes of men in more *vs* less segregated states. Consider the estimates for slave share. Black men born in states with a slave share close to zero (Northern states) exhibit large increases in high school and college completion, employment, income, poverty and work-limiting disability (coefficient on *post*base*), gains that are typically larger than for white men, consistent with their initial pneumonia burden being larger. All of these gains are significantly smaller for black men born in (Southern) states with slave shares greater than zero.[31] For example, a one standard deviation decrease in pneumonia exposure led to an increase of 3.7% points in the probability of completing college in states with *slave*=0, but the same change in pneumonia in states in the upper decile of the slave fraction distribution was associated with a 0.8% point increase in college completion rates.[32] The corresponding results for income are a 5.9% increase for black men born in states with no slave history, and essentially no change (a 0.5% decrease) for black men born in states with a historical slave share in the upper decile. We elaborate effect sizes more comprehensively for estimates of an extended specification of equation 2 below.

Adjusted estimates: We controlled for differences in drug access and measurement error in mortality rates for blacks using the proxies discussed in section II-1. This is now a first order concern since the segregation gradients we identify may be an artefact of greater measurement error in the Southern (and more segregated) states. To adjust for this, we include in equation (2), the

[30] As discussed in *Appendix 4*, most coefficients are not statistically significant and the odd coefficient that is suggests that white men exhibit *larger* improvements in segregated states (for example in employment), which serves to widen the racial gap in returns to sulfa as a function of segregation.

[31] The *difference* in gains between more and less segregated states is in the coefficient on *post*base*slave*. The total effects for blacks in states at different percentiles of the slave share distribution are in *Table 5* below.

[32] We have plugged in here the s.d. of the all-race all-state pneumonia rate (0.19; reported in *Appendix 1 Table 1*). As the rate among blacks was much higher (see *Figure 6*), the reported estimates are conservative but we use them so that these results are in the same "metric" as the earlier results for all-men.

additional terms *post*birth_registration*slave, post*death_registration*slave, post*birth_registration* and *post*death_registration.*[33] This is discussed in more detail in *Appendix 4*. The results are in *Table 5*.[34] The coefficients on both *post*base* and *post*base*slave* are, in general, larger. Sulfa-driven increases in high school, college, cognitive and work-related disability, employment and poverty are all decreasing in the degree of segregation states. For income, both coefficient sizes are similar to those in *Table 4* without the additional controls so there is a substantively large gradient but it is now imprecisely determined (t=1.6). Alongside these somewhat indeterminate results for mean income, we see a significant gradient in income mobility (*Table 5A)*. In particular, the tendency for black men to "escape" the bottom two quintiles shows a significant gradient in segregation: it is significant up until at least the 25th percentile of the slave share distribution but, it disappears by the 50th.

At the bottom of *Tables 5* and *5a,* we compute outcome gains for black men at specific percentiles of the distribution of slave share (the distribution is plotted in *Appendix 4, Figure 4*). The gradients are steep. For instance, a one s.d. decline in *base_pneumonia* created by introduction of antibiotics is estimated to raise high school completion rates for blacks by 5.3% points for *slave*=0 and by 3.2, 2.0 and 1.1% points respectively for slave share at the 25th, 50th and 75th percentile of the distribution. For college, the gradient is even steeper.[35] Poverty reduction ranges from 6.2% points at *slave*=0 to 0.85% points at the 75th percentile of slave share. The probability of having an income in one of the bottom two quintiles is reduced by sulfa exposure by 2.3% points when *slave*=0 and remains reduced at the 25th percentile of slave share, after which the escape from poverty is no longer evident. Similarly, the probability that sulfa exposure results in black men having an income in the top quintile is increased by 1.0% points if *slave*=0, remains positive at the 25th percentile, but is then obliterated.

Contribution of education: Using Mincerian estimates of the returns to education by race and South/non-South applied to the estimates in *Table 5* indicates that, among blacks in the Northern states, sulfa exposure led to a 6.2% increase in income, about 90% of which can be

[33] Similar to our prior argument, if outcome *y* depends upon (*post*[base+u]*segregation*) where *u* is the error in measurement of *base_pneumomia*, then *y* will depend upon (*post*u*segregation*) and (*post*u*). Omission of these terms will bias the coefficients on (*post*base*segregation*) and (*post*base*). In *Appendix 4 Figure 3*, we show that proxies for *u* are positively correlated with both *base* and *segregation* (slave share). Note that the other implied term, *slave*u* is absorbed by (race-specific) state fixed effects. Our specification now allows for trends in measurement quality correlated with segregation intensity, and it accounts for the possibility that the tendency to under-count black deaths may over-state improvements over time (Boustan and Margo, 2014).

[34] To conserve space we only show results with the added controls using the slave share, but the gradient estimates are robust to these controls using the other measures of segregation. We do not display gradients for white men with these controls as the controls create no significant changes in coefficients, but they are available on request.

[35] High school completion for blacks increased from 40% for the 1930 birth cohort to 65% for the 1943 cohort. For whites, the corresponding change was from 70 to 84%, so the overall improvement was more rapid for blacks.

explained by increases in their high school and college completion rates. Blacks in the Southern states not only experienced smaller increases in income, but the contribution of education to income gains was smaller. At the 25th percentile of slave share, income rises 3.5% and education explains about 70% of this while, at the 50th percentile, income rises by 2% and education explains about 35% of this. This and the finding of greater sulfa-related income mobility among blacks in less segregated states are consistent with sulfa exposure having led to higher skill acquisition in these states. That the more muted benefits in segregated states are, in general, positive is consistent with direct effects of infant health on adult incomes given evidence that blacks in segregated states experienced sharp declines in pneumonia in 1937 *(Appendix 4*). For example, the sulfa shock may have led to improved productivity in brawn-intensive work.[36]

4. Additional Threats to Inference and Alternative Interpretations

The estimates in Part-I using data pooled across the races were subject to a number of tests designed to challenge our contention that we are identifying long run impacts of pneumonia exposure at birth rather than something else. Here, in Part-II we consider, specifically, threats to identification of *segregation-gradients* in these long run impacts, and whether the gradients may have interpretations other than the one we suggest. First, to recapitulate, the gradient estimates are conditional upon the main effects of race and segregation and race*state specific trends which, together, account for many of the alternative hypotheses that spring to mind, for instance, that segregation had a direct impact on long run outcomes or that trends in outcomes varied by race and region for reasons unrelated to our project. Furthermore, any post-1937 convergence across the states that was unrelated to pneumonia reduction is absorbed by race-specific coefficients on the term *post*segregation*. Importantly, there is no consistent pattern of gradients in impact for white men by indicators of segregation, which undermines the potential concern that the gradients we identify for blacks reflect state-specific factors that are correlated with segregation but uncorrelated with (state and race specific) returns to human capital investment. We now investigate the main residual concerns.

As discussed, an important alternative explanation of the segregation gradients for blacks is that they reflect gradients in **access to sulfa drugs** rather than in long run outcomes conditional upon access. We have shown that the gradients persist conditional upon a proxy for state differences in access. In a companion paper, key results of which are summarized in *Appendix 4,* we directly

[36] In *Appendix 4*, we note that child labor was more prevalent in the rural South, which will have created an opportunity cost to education that may have acted to reinforce restrictions on access to quality schools and skilled jobs.

investigate access by estimating race and region specific "first stage" equations which show *larger* post-1937 drops in absolute pneumonia mortality rates for blacks than for whites, even in the South (*Appendix 4, Table 1*, also see *Figure 6*). Although we have noted smaller returns to sulfa exposure in segregated states, there are positive gains for many outcomes at the median slave share (*Table 5*), consistent with access. At $4 per course (in *1940 dollars*) sulfa drugs cost only 1% of the monthly wage for black men (as reported in Smith and Welch, 1989), making it likely they would incur this life-saving expenditure. In *Appendix 4,* we discuss the coherence of our findings with previous work situated in this era. In particular, our findings are not inconsistent with Jayachandran et al (2010) who show that *proportional* declines in mortality were larger for whites. We noted that segregation gradients could arise if **measurement error in mortality** were correlated with segregation, and we showed that the gradients are robust to attempting to control for state differences in measurement error. Again, this is elaborated in *Appendix 4*.

We considered whether the **Great Migration** of blacks from the South to the North might have generated compositional effects that explain our finding that Southern blacks saw smaller post-sulfa increases in education and other outcomes given that the relatively educated were more likely to migrate northward, even if migration slowed after 1930 (Vigdor, 2002; Aaronson and Mazumder, 2011). We explain in *Appendix 4* that accounting for this would tend to strengthen our conclusions, but we nevertheless show that there was in fact no significant endogenous migration (*Appendix 4, Table 5*). Weaker socio-economic gains from sulfa for blacks born in segregated states may also flow from greater negative **survival selection** in this group because they experienced larger absolute declines in pneumonia mortality post-sulfa (*Appendix 4, Table 1*). In general, survival selection effects tend to be too small to modify causal or "scarring" effects (Alderman et al, 2011), but we nevertheless, investigated this and, under conservative assumptions, we find only very small and insignificant changes in coefficients when estimating gradients on this sample (*Appendix 4 Table 6).*

A potential concern with our **interpretation of increases in higher education** as responsive investments may be that the observed increases were driven not by sulfa-led improvements in the infant health and cognitive endowment but instead by changes in supply. Similarly one may be concerned that the segregation gradients in high school and college that we identify are in fact just a reflection of fewer and poorer quality school and college facilities in the Southern states (Card and Krueger, 1993). However fixed cross-state differences are captured by state fixed effects in race-specific models, and any differential trends in school or college supply within-race and across states with different levels of segregation are absorbed by state trends and our

controls for state*year per capita school buildings and school expenditure (which allow for a non-linear evolution of the state-specific school infrastructure). So the only threat to our contention that changes in education reflected purposive investments in sulfa-exposed children would be if there were a sharp change in school supply that favored less segregated states over more segregated states and that affected *post-1937* and not *pre-1937* birth cohorts in states with relatively high *base_pneumonia*.

Overall, the evidence indicates that responsive reinforcing investments in higher education may have contributed substantially to the large sulfa-led increases in income and mobility achieved by white men and by black men in the Northern, less segregated states. First, crude simulations suggest that, where segregation was absent or small, increases in high school and college completion rates explained most of the increase in income (consistent with Goldin, 1998). Second, given that pneumonia had a predilection for afflicting poor families, our finding that white men [and black men in less segregated states] were significantly more likely, upon sulfa-drug exposure, to find a place in the top quintile [second highest quintile] may suggest dynamic complementarities. This is particularly so because the intergenerational mobility experienced by post-antibiotic birth cohorts lines up with increases in high school and college completion rates: where post-antibiotic improvements in education were small, income and income mobility were muted, despite similarly sharp reductions in infant pneumonia exposure after 1937. While this is not direct or conclusive evidence of complementarity between health and educational investments in production of income, it is suggestive of it.[37]

Conclusions

We demonstrated that improvements in infant endowments created by the availability of antibiotics led to increased investments in higher education, reductions in adult disability and improvements in income and income mobility. That the improvements were, on average, large is likely to have been potentiated by the unprecedented expansion of high school and college in this era (Goldin and Katz, 2008). Our results are consistent with responsive educational investments

[37] A number of previous studies find evidence that parental investments reinforce early life endowments, see, for example, Adhvaryu and Nyshadham (forthcoming), Aizer and Cunha (2012), Bharadwaj, et al (2013), Conti, et al (2011), Venkataramani (2012), and Bhalotra and Venkataramani (2013). There is nevertheless limited evidence of the *contribution* that reinforcing investments make to eventual returns (see Almond and Mazumder 2013). We contribute by showing that the initial sulfa-shock to infant endowments led to investments in education and that these investments appear to have done a lot of the heavy lifting in producing the income mobility (and income) gains where these gains are large.

playing an important role in unlocking the full potential of a healthier start. The stark manner in which the long run benefits varied for black men with markers of segregation intensity in the black sample despite a strong economic climate provides new and powerful evidence of the relevance of an institutional environment that enables and rewards investments in human capital. Our results illustrate a new and insidious legacy of segregation, bolstering evidence that the adverse consequences of extractive institutions are persistent (Acemoglu and Johnson, 2012). Our finding that blacks in segregated states failed to fully realize potentially large dynamic socioeconomic benefits from early investments coheres with recent work showing that the full realization of the education and employment benefits from diffusion of the pill since the 1960s were conditional upon the institution of equal opportunities for women (Coles and Francesconi, 2013). Our results have implications for today's developing countries where barriers to human capital accumulation (such as poor access to quality schools, imperfect information, and binding credit constraints) remain and in many of which there is institutionalized discrimination by ethnicity or gender.

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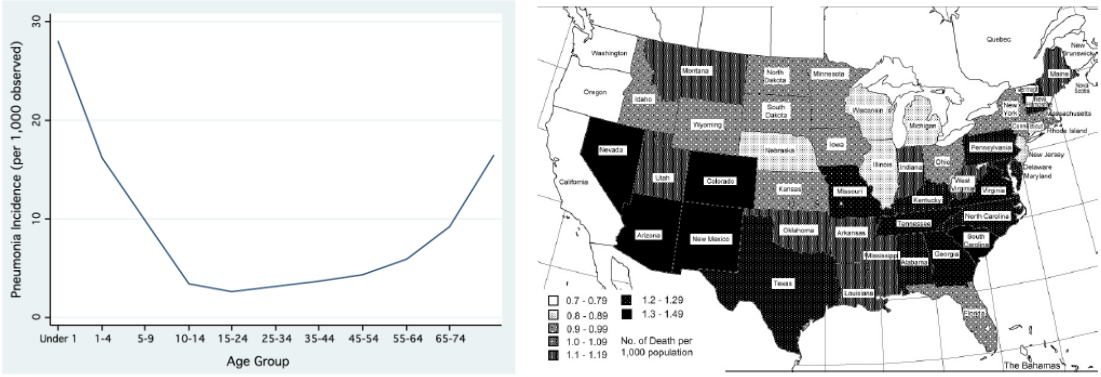
Figure 1- Age and Geographic Distribution of Pneumonia Mortality

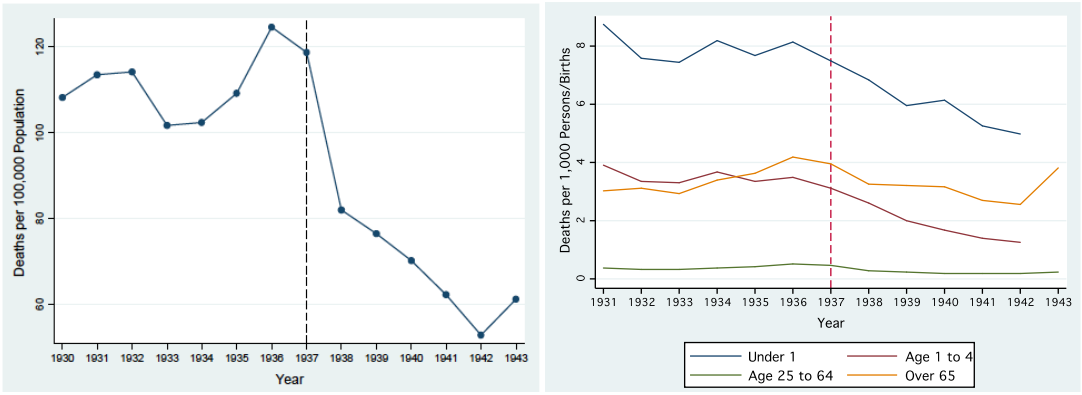
A. Age Distribution **B. Spatial Distribution**



Notes: Data for Panel A obtained from results of a nationally representative survey conducted between 1931 and 1933 (Britten, 1942). Data for Panel B from U.S. Vital Statistics.

Figure 2 – Trends in Pneumonia and Influenza Mortality

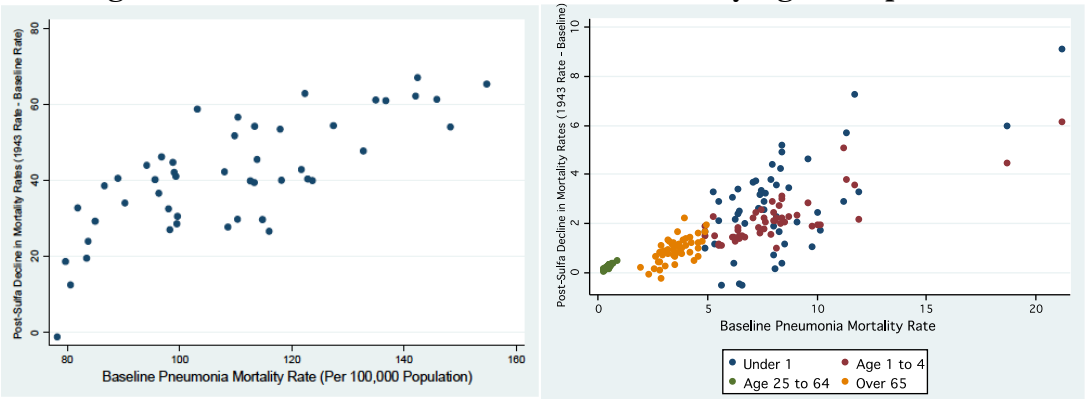
A. All-Age **B. By Age Group**



Notes: Data obtained from the U.S. Vital Statistics. Pre-1937 fluctuations in the combined series are driven by year to year variation in influenza mortality *(Appendix 2*). Trend breaks in the infant pneumonia mortality series are statistically significant (see *Appendix 2, Table 1*).

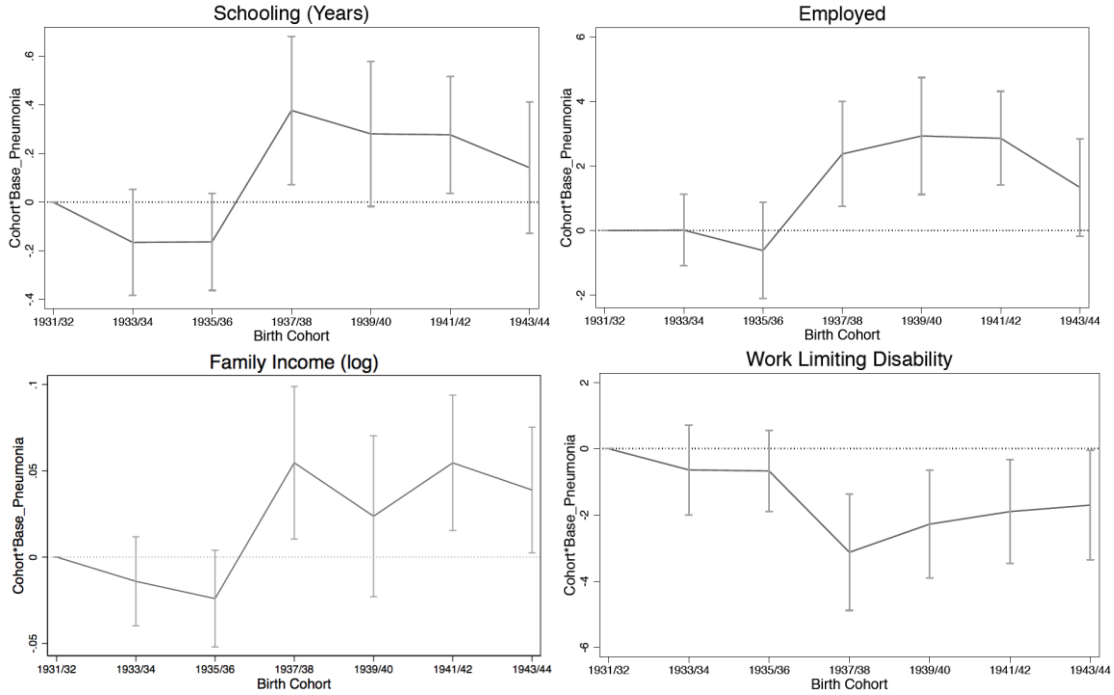
Figure 3 – Post-Sulfa Convergence in Pneumonia and Influenza Mortality

A. All-Age **B. By Age Group**



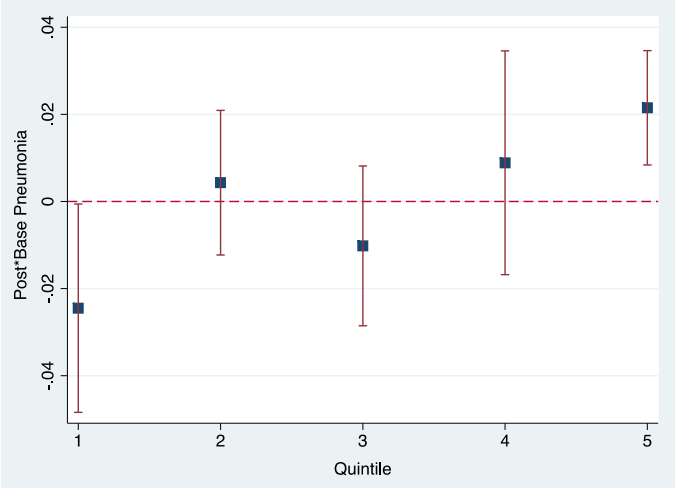
Notes: The base rate of pneumonia and influenza mortality is its average over 1930-1936. Every dot is a state. Source: US Vital Statistics.

Figure 4 – Cohort Specific Coefficient Estimates: All Men



Notes: Each point reflects the coefficient estimated on an interaction term between the marked birth year (grouped into two-year periods in order to enhance precision) and the pre-intervention (base) level of the pneumonia mortality rate in the birth-state. All models condition upon birth state and birth year fixed effects and the full set of controls for mortality from other diseases and state macroeconomic and infrastructure variables. State specific trends are omitted so as to allow us to discern the presence of pre-trends. Those in 1931/1932 form the (base) group. Joint *F*-tests (p-values) for pre-1937 and 1937 and thereafter are as follows: (1) Schooling: Pre – 1.54 (0.23), Post – 2.37 (0.066), (2) Employed: Pre – 0.47 (0.63), Post – 3.84 (0.01), (3) Family Income: Pre – 1.43 (0.25), Post – 2.59 (0.04), (4) Work Limiting Disability: Pre - 0.71 (0.50), Post – 3.54 (0.01).

Figure 5: Income Mobility Estimates: All Men

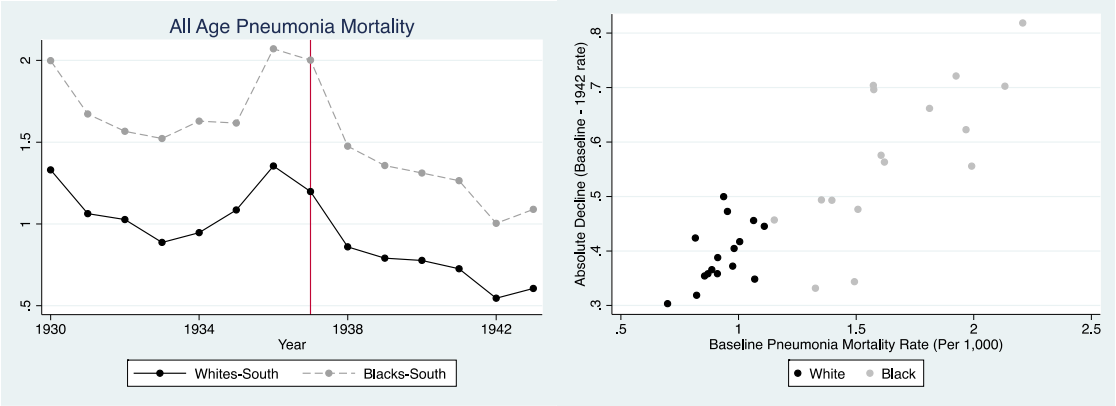


Notes: Each box-whisker plot point reflects the coefficient estimated on an interaction term between a dummy indicator for being born after 1937 (*Post)* and the pre-intervention level of the pneumonia mortality rate in the birth-state (*base*) from a separate regression. The dependent variable is a binary indicator for membership of one of the five family income quintiles marked on the x-axis. The quintiles are constructed using the pre-

intervention distribution (see *Appendix 1*). The whiskers denote 95% confidence intervals. All models include birth state and birth year fixed effect, the full set of controls for mortality from other diseases and state macroeconomic and infrastructure variables, and birth state specific time trends. The quintile regression tables underlying the plot are in Appendix 2, Table 1.

Figure 6 – Trends & Convergence in Pneumonia and Influenza Mortality Rates by Race in the US South

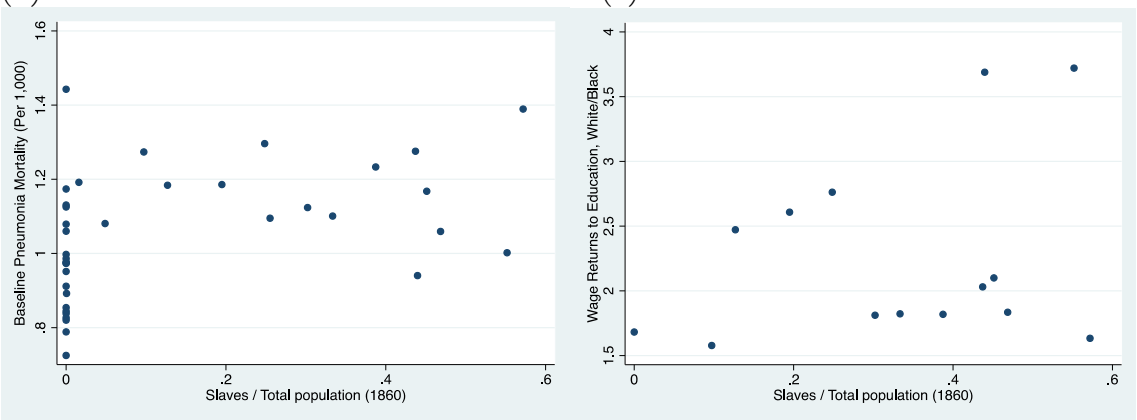
(A) (B)



Notes: Data obtained from U.S. Vital Statistics and Jayachandran, et al (2010). Panel A plots trends in all age pneumonia and influenza mortality by race. Panel B plots the difference between mortality rates post and pre-sulfa by race. Data are for U.S. South where 85% of the black population resided during the study era. *Appendix 4 Table 1* demonstrates significant post-1937 trend breaks in all-age and infant pneumonia mortality for both blacks and whites in the South. The black trend breaks are larger in absolute magnitude.

Figure 7 – Correlates of Slave Fraction

(A) (B)



Notes: Data from U.S. Vital Statistics, Nunn (2008), and 1940 US Census Micro Data. The horizontal axis has the fraction of the state population in 1860 enumerated as slaves. In Panel A the vertical is pre-sulfa pneumonia mortality (*base*), in Panel B it is the white/black wage returns to education (for the 14 Southern states with >10% black population).

Table 1: Estimated Impacts of Pneumonia Exposure in Infancy on Adult Outcomes- All Men

	(1)	(2)	(3)	(4)	(5)	N	P-Value	Effects of 1 s.d.
Schooling	0.630***	0.530***	0.360***	0.541***	0.772***	708,478	0.000	0.10
	(0.122)	(0.0934)	(0.110)	(0.0761)	(0.116)			
College	-0.0138	-0.0167	0.0093	0.0647***	0.111***	708,478	0.000	1.2% pt
	(0.0104)	(0.0101)	(0.0147)	(0.0129)	(0.0216)			
High School	0.0779***	0.0704***	0.0613***	0.0781***	0.0956***	708,478	0.000	1.5% pt
	(0.0158)	(0.0114)	(0.0152)	(0.0115)	(0.0158)			
Employment	0.0116**	0.0109**	0.0202***	0.0226**	0.0262*	2,018,898	0.084	0.4 % pt
	(0.0151)	(0.0129)	(0.0168)	(0.0220)	(0.0124)			
Family Income (log)	0.0509***	0.0492***	0.0523***	0.0645***	0.0875***	1,976,203	0.021	1.2%
	(0.00541)	(0.00493)	(0.00733)	(0.0101)	(0.00851)			
Poverty (%)	-0.0240***	-0.0237***	-0.0162**	-0.0247**	-0.013	2,018,898	0.045	-0.5 % pt
	(0.00541)	(0.00493)	(0.00733)	(0.0101)	(0.00851)			
Work Limiting Disability	-0.0157	-0.0142	-0.014	-0.0333	-0.0462**	2,018,898	0.768	-0.6% pt
	(0.0114)	(0.0104)	(0.0131)	(0.0221)	(0.0209)			
Cognitive Disability	-0.0150***	-0.0123***	-0.00869	-0.0340***	-0.0424***	627,262	0.003	-0.6% pt
	-0.00496	(0.00455)	(0.00596)	(0.0101)	(0.0114)			
Physical Disability	-0.00952	-0.0059	-0.00118	-0.0183	-0.0204	627,262	0.603	-0.3% pt
	(0.00896)	(0.00876)	(0.0110)	(0.0191)	(0.0202)			
Birth State, Birth Year Fixed Effects	Yes	Yes	Yes	Yes	Yes			
Post*BaseRate(Control Diseases)	No	Yes	Yes	Yes	Yes			
Birth State X Birth Year Controls	No	No	Yes	Yes	Yes			
Birth State Linear Trends	No	No	No	Yes	Yes			
Birth Region X Birth Year FE	No	No	No	No	Yes			

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each cell reports coefficient on *post*base_pneumonia* from a separate regresssion. Outcomes are defined in *Appendix 1*. BaseRate(Control Diseases) is a vector of pre-sulfa birth state averages for maternal mortality, and mortality from heart disease, cancer, diarrhea for children under the age of 2, malaria, tuberculosis. Birth State X Birth Year controls include per capita log state income, state education and health expenditures, schooling buildings, hospitals, physicians per capita. Region are the 9 census divisions. We obtain *p*- values consistent with multiple hypothesis testing utilizing a Bonferroni-type method modified to adjust for correlation across like outcomes. See Appendix 2 and Aker, et al (2011) and Sankoh, et al (2007) for details.The final column reports the change in the outcome variable resulting from a 1 s.d. decrease in *base_pneumonia* (0.19 deaths per 1,000).

Table 2: Robustness Checks

	Schooling	High School	College	Employed	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
as IV for Infant Pneumonia/Influenza Mortality									
*post*base_pneumonia*	0.297**	0.043**	0.037**	0.010**	0.034**	-0.005	-0.004	-0.016**	-0.008
	(0.132)	(0.022)	(0.019)	(0.005)	(0.015)	(0.004)	(0.003)	(0.007)	(0.008)
scaled impacts	0.16	2.1% pt	1.8% pt	0.5% pt	1.6 % pt	-0.25% pt	-0.2% pt	-0.7% pt	-0.35% pt
Panel B - Baseline Pneumonia Mortality in 1935									
*post*base_pneumonia*	0.255	0.0622***	0.0266	0.006	0.0652**	-0.0332***	-0.011	-0.0292**	-0.00348
	(0.181)	(0.0230)	(0.0273)	(0.014)	(0.0255)	(0.0105)	(0.009)	(0.0131)	(0.0201)
Panel C - Mean Reversion									
*post*base_pneumonia*	0.458***	0.0438**	0.0574***	0.0162*	0.0588***	-0.0213**	-0.00146	-0.0223*	-0.00865
	(0.0800)	(0.0178)	(0.0123)	(0.00938)	(0.0190)	(0.0103)	(0.0116)	(0.0122)	(0.0256)
Panel D - Triple Difference									
*post*base_pneumonia*	0.588***	0.0789***	0.0725***	0.0240***	0.0674***	-0.0264**	-0.00583	-0.0357***	-0.0205
	(0.0805)	(0.0113)	(0.0145)	(0.00877)	(0.0214)	(0.0105)	(0.00603)	(0.0101)	(0.0189)
N	721,536	721,536	721,536	2,054,718	2,011,135	2,054,718	1,952,954	637,881	637,881

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each panel describes a different robustness check and each cell reports estimates on *post*base_pneumonia* from a different regression. All models include the controls in Column (4) of *Table 1*, unless otherwise indicated. All results are for the male sample, with the sample size for each variable given in *Table 1* unless otherwise indicated. In *Panel A,* we instrument *post*base_pneumonia_infants,* which we argue is measured with error, with *post*base_pneumonia_allage*, the preferred measure that we use in Table 1. The numbers in square brackets are the scaled (by the first stage) impacts of a 1 s.d. decrease in baseline infant pneumonia on the outcomes. Specifically, we compute the estimated impacts owing to a 1 s.d. change in *post*base_pneumonia*, so as to make the impact estimates comparable to *Table 1.* First stage *F*- statistics (available upon request) are 5.3-5.5 across the different models. The models here additionally include census division X year fixed effects so as to maximize the first stage *F* statistic. In Panel B, we replace *base_pneumonia* with a single year (1935) measure of pneumonia mortality rate *that does not include influenza mortality* (Linder and Grove, 1947). In Panel C, we include a birth state X birth year X race X gender X census year pre-intervention average measure for each outcome variable interacted with *post* to address mean reversion. In Panel D, we estimate a triple difference model focusing on a birth cohort X birth state X age-at-first-exposure comparison. This is described in the text and in *Appendix 2, Figure 2*.

Table 3: Race-Specific Estimates- Impacts of Pneumonia Exposure in Infancy on Adult Outcomes

	White Men		Black Men			
	Baseline	**N**	**Baseline**	**ME + Access**	**N**	**N (ME+Acc)**
Schooling	0.593***	636,991	-0.106	-0.0532	71,487	69,181
	(0.0962)		(0.236)	(0.243)		
College	0.0632***	636,991	0.0405	0.0705	71,487	69,181
	(0.0142)		(0.0475)	(0.0590)		
High School	0.0768***	636,991	0.0738*	0.106**	71,487	69,181
	(0.0127)		(0.0384)	(0.0452)		
Employment	0.0202**	1,835,771	0.0293	0.0280	183,127	177,767
	(0.0100)		(0.0401)	(0.0457)		
Family Income (log)	0.0632***	1,802,548	0.0730	0.187**	173,655	168,704
	(0.0218)		(0.0822)	(0.0795)		
Poverty (%)	-0.0230**	1,835,771	-0.0405*	-0.0658**	183,127	177,767
	(0.0100)		(0.0242)	(0.0259)		
Work Limiting Disability	-0.0155*	1,835,771	-0.103	-0.0367	183,127	177,767
	(0.0087)		(0.0931)	(0.0305)		
Cognitive Disability	-0.0264**	573,071	-0.0925**	-0.0931*	54,191	52,673
	(0.0101)		(0.0432)	(0.0474)		
Physical Disability	-0.0135	573,071	-0.0386	-0.0244	54,191	52,673
	(0.0206)		(0.0521)	(0.0601)		

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each cell represents a separate regression. The specification is as in Column (4) of *Table 1*. In the column headed ME + Access, we add *post*year entered death registration system* and *post*year entered birth registration system* as controls for measurement error and *post*pharmacists per capita in majority black counties* (averaged to the state level, weighted by county population) to control for differences in access to sulfa drugs. Earlier entry into the death registration system is correlated with quality and completeness of death registration, and early entry to the birth registration system is correlated with the percentage of actual births registered (as revealed by a 1940 audit).

Table 3A: Income Mobility: Adjusted for Measurement Error and Access- Black Men

	Income Quintile					
	1	**2**	**1+2**	**3**	**4**	**5**
post*base	-0.0191	-0.0158	-0.0349	-0.0643**	0.0878***	0.0114
	(0.0227)	(0.0307)	(0.0358)	(0.0243)	(0.0315)	(0.0186)

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each panel X column represents a different regression. The dependent variable is a dummy variable = 1 if the individual belongs to the income quintile listed in the column header and zero otherwise. All models include the controls in column (4) of *Table 1* and the controls for access and measurement error described in *Table 3*. N = 166,568 for all models.

Table 4: Gradients in Long Run Impacts of Infant Pneumonia Exposure by Indices of Segregation- Black Men

	Schooling	High School	College	Employment	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
Panel A- South v nonSouth									
post*base	0.567	0.264**	0.231***	0.062	0.170	-0.0511	-0.150*	-0.0914	-0.0338
	(0.485)	(0.123)	(0.0767)	(0.086)	(0.197)	(0.0658)	(0.088)	(0.0848)	(0.104)
post*base *south	-0.798	-0.270**	-0.193**	-0.052	-0.123	0.0142	0.174***	-0.00727	0.0138
	(0.539)	(0.128)	(0.0783)	(0.090)	(0.217)	(0.0731)	(0.092)	(0.0896)	(0.122)
post*south	0.561	0.251*	0.190*	0.100	0.142	-0.0192	-0.192**	0.0379	-0.111
	(0.628)	(0.148)	(0.0948)	(0.098)	(0.248)	(0.0799)	(0.095)	(0.103)	(0.136)
Panel B - Slave Fraction									
post*base	0.160	0.187*	0.197***	0.127*	0.309**	-0.101**	-0.120*	-0.109	-0.0596
	(0.488)	(0.0985)	(0.0643)	(0.068)	(0.128)	(0.0398)	(0.062)	(0.0707)	(0.101)
post*base*slave	-0.469	-0.406*	-0.304**	-0.271*	-0.680**	0.179*	0.307**	0.0152	0.00729
	(1.168)	(0.231)	(0.144)	(0.145)	(0.284)	(0.0959)	(0.131)	(0.145)	(0.252)
post*slave	1.421	0.514**	0.450***	0.268	0.585	-0.120	-0.268*	0.0771	-0.0359
	(1.214)	(0.250)	(0.164)	(0.159)	(0.387)	(0.110)	(0.152)	(0.165)	(0.292)
Panel C - W-B Education Return									
post*base	0.0509	0.226***	0.139*	0.061***	0.165*	0.0119	-0.050	-0.250***	-0.284***
	(0.234)	(0.0293)	(0.0721)	(0.020)	(0.0773)	(0.0136)	(0.037)	(0.0336)	(0.0676)
post*base *returns	-0.364	-0.251***	-0.146*	-0.073***	-0.282***	-0.0342**	0.041	0.103**	0.218***
	(0.263)	(0.0344)	(0.0729)	(0.024)	(0.0877)	(0.0152)	(0.040)	(0.0351)	(0.0712)
post*returns	0.334	0.292***	0.152*	0.086***	0.318***	0.0338*	-0.065	-0.139***	-0.278***
	(0.301)	(0.0390)	(0.0833)	(0.026)	(0.0943)	(0.0161)	(0.043)	(0.0387)	(0.0783)
N	71,487	71,487	71,487	183,127	173,655	183,127	183,127	54,191	54,191
N (slave)	70,195	70,195	70,195	180,121	170,844	180,121	180,121	53,329	53,329
N (returns)	57,252	57,252	57,252	149,344	142,252	149,344	149,344	44,594	44,594

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each PanelXcolumn represents a separate regression. The corresponding estimates for white men are in Appendix 4, Table 4. The model specifications are as in Table 1, column 4 but now include *post*base_pneumonia*segregation* and the underlying main effects. The Panels show estimates for 3 measures of segregation. *South*, a dummy = 1 if birth state in the U.S. South (see Appendix 1). *Slave* denotes the 1860 slave fraction in each birth state (Nunn, 2008). *Slave* ranges from 0 to 0.57 in the microdata and is 0 for most non-Southern states. For e.g. the effect of a 1 s.d. decrease in pneumonia mortality rates for black men born in states with a slave fraction of zero is a 5.8% increase in income (0.19*0.309). The median black man was born in a state with a slave fraction of 0.38. For this individual, the predicted impact is a 1% increase in income (0.19*(0.309 - 0.38*0.680). The third segregation indicator is *Education Returns*, the ratio of income returns to schooling for white relative to black men aged 25-40 in the 1940 census, constructed for the 15 states with at least 10% blacks, all but one of which are in the U.S. South, so it identifies variation within the segregated region. *Returns* is scaled to have a minimum value of zero. The median black man was born in a state with a scaled return ratio of 0.5 (and 90% were born in states with return ratios <1). For this individual, the effective impact of income would be close to 0. *Appendix 4, Figure 4* contains density plots of *Slave* and *Returns*.

Table 5: Adjusting for Measurement Error and Access: Segregation Gradients for Black Men

	Schooling	High School	College	Employed	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
post*base	0.687	0.278***	0.361***	0.233***	0.324*	-0.144***	-0.120*	-0.229***	-0.0996
	(0.407)	(0.0699)	(0.0808)	(0.0611)	(0.160)	(0.0479)	(0.0662)	(0.0685)	(0.127)
post*base*slave	-1.488	-0.440*	-0.903***	-0.664***	-0.559	0.271**	0.302*	0.490**	0.265
	(1.071)	(0.236)	(0.206)	(0.159)	(0.350)	(0.131)	(0.153)	(0.177)	(0.356)
N	68,242	68,242	68,242	175,509	166,568	175,509	175,509	52,008	52,008
Implied Effect at Slave = 0	0.13 yrs	5.3% pt	6.9% pt	4.4% pt	6.2%	-2.7% pt	-2.3% pt	-4.4% pt	-1.9% pt
Implied Effect at 25th pctile of Slave	0.06 yrs	3.2% pt	2.6% pt	1.3% pt	3.5%	-1.4% pt	-0.85% pt	-2.0% pt	-0.63% pt
Implied Effect at 50th pctile of Slave	0.02 yrs	2.0% pt	0.17% pt	-0.49% pt	2.0%	-0.7% pt	-0.04% pt	-0.72% pt	0.07% pt
Implied Effect at 75th pctile of Slave	-0.01 yrs	1.1 % pt	-1.7% pt	-1.8% pt	0.85%	-0.16% pt	0.59% pt	0.30% pt	0.63% pr

Notes: See Notes to Table 4. This is the model specification in Table 4 (Panel B), including controls for access and the quality of mortality statistics described in the paper. Implied effect sizes refer to the estimated impact of a 0.19 decrease in *base_pneumonia*. We use the slave distribution in the microdata.

Table 5A: Income Mobility- Segregation Gradients : Adjusted for Measurement Error and Access- Black Men

	Income Quintile					
	1	2	1+2	3	4	5
post*base	-0.0758	-0.0448	-0.121**	-0.0906**	0.157**	0.0537
	(0.0601)	(0.0512)	(0.0498)	(0.0330)	(0.0581)	(0.0363)
post*base*slave	0.121	0.208	0.329**	0.115	-0.304	-0.140
	(0.124)	(0.143)	(0.157)	(0.122)	(0.178)	(0.0915)
Implied Effect at Slave = 0	-1.44	-0.85	-2.30	-1.72	2.98	1.02
Implied Effect at 25th pctile of Slave	-0.87	0.14	-0.74	-1.18	1.54	0.36
Implied Effect at 50th pctile of Slave	-0.54	0.69	0.14	-0.87	0.73	-0.02
Implied Effect at 75th pctile of Slave	-0.29	1.12	0.83	-0.63	0.09	-0.31

Notes: See Notes to Tables 3 and 4. The dependent variables are dummies= 1 if the individual belongs to the income quintile listed in the column header and zero otherwise. Controls are in column (4) of *Table 1* plus controls for access and measurement error described in the paper. global N = 166,568.

Shadows of the Captain of the Men of Death:

Health Innovation, Human Capital Investment, and Institutions

Sonia Bhalotra and Atheendar Venkataramani

Online Appendix

This is in four parts.

1. Data Sources and Variables
2. Pneumonia Mortality Measurement and Additional Robustness Checks
3. Results for Women
4. Additional Race Specific Results

Online Appendix 1 – Data Sources and Variables

Outcome variables

The outcomes data were taken from the 5% United States Census Microdata samples for 1980, 1990 and 2000. These data are publicly available via the Integrated Public Use Microdata Series – USA project at http://usa.ipums.org/usa/ (Ruggles et al., 2010).

For measures of income, poverty and employment, we pool data from the three censuses. Disability preventing work is available in all three censuses, while physical and cognitive disability are only available in the 2000 census. Models for years of schooling, high school completion, and college completion use only 1980 census data. The main reason for this is that later census files group those completing ninth grade and under into three categories and top code those who progress beyond college, whereas the 1980 census allows us to differentiate each level of schooling. In addition, using a single census allows us to avoid duplicating the data given that years of schooling seldom change after the age of 37, the age of the youngest cohort in our estimation sample in the 1980 census. Finally, using an earlier census reduces bias from potential mortality selection as the birth cohorts age. Nevertheless, the results for high school and college attainment are not substantively changed if we use later census files *(Appendix 2)*. There is some concern in the literature that the 2000 census microdata sample may be subject to inaccuracies in age reporting (Alexander et al., 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, and the substantive results were unchanged (see *Appendix 2*).

Specifics of the construction of the outcome variables are as follows:

Schooling (HIGRADE in IPUMS) – In the 1980 census, *HIGRADE* distinguishes between no schooling, nursery schooling, each grade of K-12, and college and post-graduate studies up to 8 years (top-coded thereafter).

High School and College – We computed these using the *Schooling* measure above. Specifically, we assigned *High School* = 1 for those individuals who completed grade 12 and above. *College* = 1 for those individuals who reported completing 4 years of college. These assignments were verified using the IPUMS variable *EDUC*, which categorizes years of schooling into having completed: no schooling; nursery-grade 4; grade 5-8; separate indicators for grade 9, 10, 11, and 12; and years of college, top-coding at 5.

Logged total family income (*FTOTINC* in IPUMS) – Nominal total pre-tax money income earned by the respondent's family unit in the previous calendar year. We also considered an indicator of personal income and wage income and find similar results for men as with the family income variable. We choose, however, to go with family income given that this indicator is also available for all women, regardless of whether they are working or not.

Poverty - Indicator for whether family income is 200% below the federal household poverty line or less. We constructed this using the *POVERTY* variable in IPUMS (which specifies the percentage above the poverty line for a given reported level of income).

Employed (EMPSTAT in IPUMS) - Individual employment = 1 if the individual reports current employment and 0 otherwise.

Work limiting disability (DISABWRK in IPUMS) – 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 (DIFFREM in IPUMS) – Denotes whether an individual has difficulty with "learning, remembering, or concentrating" due to a physical, mental, or emotional condition.

Physical disability (DIFFPHYS in IPUMS)- Denotes whether the respondent has a condition that "substantially limits one or more basic daily tasks, such as walking, climbing stairs, reaching, lifting, or carrying."

For the *quantile dummy models*, we created five binary indicators that indicate assignment of each individual to quintiles of the pre-intervention income distribution. The quintiles were constructed using the income distribution, as observed in each census wave, for the pre-intervention cohorts (1930-1936) birth cohorts. Individuals with incomes, for example, in the second quintile of the pre-sulfa income distribution in a given census year were assigned a 1 for the second quintile dummy variable, and a zero for each of the other dummy variables. The estimates are similar if we assign individuals to quintiles of the income distribution for all birth cohorts in the sample (1930-1943).

Mortality data

State time series data on mortality rates from influenza and pneumonia, under-2 diarrhea, malaria, heart disease, cancer and tuberculosis, and the maternal mortality ratio were obtained from various volumes of the US Vital Statistics (Grove, 1968; Linder, 1947; United States Bureau of the Census, 1930-1943). These data were used to extend the data series collected by Grant Miller (http://www.nber.org/data/vital-statistics-deaths-historical/), and Seema Jayachandran, Adriana Lleras-Muney, and Kimberly Smith (Jayachandra, et al, 2010, http://www.aeaweb.org/articles.php?doi=10.1257/app.2.2.118). We used these data to create birth state-specific pre-sulfa drug era rates for each disease by averaging the cause-specific mortality rates between 1930 and 1936 (varying the time period over which we compute baseline rates does not change our substantive results). In the text we refer to these pre-sulfa mortality rates as *base* rates. In *Appendix 2* we discuss issues with measurement of exposure to pneumonia and present tests of robustness to alternative measures.

Race specific state mortality data for the 18 states where blacks comprised of >10% of the population were generously provided by Adriana Lleras-Muney. The states in question include: Alabama, Arkansas, Delaware, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, Missouri, New Jersey, Oklahoma, South Carolina, North Carolina, Tennessee, Texas, Virginia, West Virginia. They were originally obtained from from yearly US Vital Statistics volumes (http://www.cdc.gov/nchs/products/vsus.htm). They are analysed in *Appendix 4*.

State level socioeconomic and infrastructure variables

State time series data on logged state per capita income were downloaded from the Bureau of Economic Analysis website (http://www.bea.gov/regional/spi/). Data on the number of

schools, doctors, hospitals, and educational expenditures per capita were drawn from Adriana Lleras-Muney's website (http://www.econ.ucla.edu/alleras/research/data.html). These data were originally collected from various volumes of the *Biennial Survey of Education* (schools and expenditures) and the American Medical Association's *American Medical Directory* (doctors and hospitals). For state per capita health expenditures, we used data from ICPSR 6304, "State and Local Government [United States]: Sources and Uses of Funds, Census Statistics, Twentieth Century [Through 1982]" (http://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/6304). These data were originally collected from various reports from the US Census bureau. The only year in our estimation sample for which there were data on health expenditures was 1932. We use these pre-intervention data interacted with a linear trend.

We acquired data on the completeness of birth registration and the year of entry into the death registration system as proxies for measurement error in the vital statistics data from Linder and Grove (1947). These variables are discussed in *Appendix 4*. As a measure of access to sulfa drugs, we used data on the number of pharmacists for counties with black share of the total population of > 10% in the IPUMS 1940 Census Microdata. This is transformed to a state level measure using population weighted averages for the counties.

Indicators of institutionalized racial segregation

In the second part of the paper, we examine whether the long-run impacts of reduced early life pneumonia for black men were muted by institutional constraints on acquiring and utilizing human capital using a number of different measures of institutionalized segregation. These are defined below, and described in Part-II of the main paper and in *Appendix 4*.

North-South - We define *South* to include Alabama, Delaware, District of Columbia, Florida Georgia, Kentucky, Maryland, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, West Virginia, Arkansas, Louisiana, Texas and Missouri. For expositional ease, our use of the term *North* refers to all other regions of the US.

Slave Fraction - The state-specific share of slaves in the population in 1860 (Nunn, 2008). Available for 38 states. This is zero for nearly all non-southern states.

Returns to Education Ratio – This variable denotes the ratio of white to black returns to years of schooling. Using data for men aged 25-55 in the IPUMS 1940 Census 1% sample, we estimated the following (population weighted) Mincerian wage equations for each state and race cell:

$$Ln(Wage\_income_{ijs}) = \beta_{0,js} + \beta_{1,js}*Schooling_{ijs} + \beta_{2,js}*Experience_{ijs} + {}_{\beta 3,js}*Experience_{ijs} + e_{ijs}$$

where *i* index the individual, *j* indexes race (white versus black), and *s* indexes the state. *Wage Income* refers to yearly wage and salary incomes (the 1940 census did not separately enumerate farm and business income), schooling refers to years of completed schooling (*HIGRADE*, as above), and experience is equivalent to reported age minus expected age at final year of schooling or, for those reporting primary schooling or less, it is reported age minus legal working age (derived from child labor and compulsory schooling laws relevant in the state of birth when the individual was aged six – see data set for Lleras-Muney (2002): http://www.econ.ucla.edu/alleras/research/data.html). We estimated this model for those states with > 10% black population and for whom we had at least 1,000 observations for

black men in the census sample. The states include: Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, Missouri, New Jersey, North Carolina, South Carolina, Tennessee, Texas, and Virginia. We use the white-black ratio of β_1 in estimating our models.

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Census Micro Data												
	Men		White Men		Black Men		Women		White Women		Black Women	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Schooling (Years Completed)	12.30	(3.28)	12.48	(3.21)	10.74	(3.50)	11.99	(2.73)	12.11	(2.66)	11.08	(3.06)
High School (=1)	0.74	(0.44)	0.77	(0.42)	0.54	(0.50)	0.74	(0.44)	0.77	(0.42)	0.56	(0.50)
College (=1)	0.38	(0.49)	0.40	(0.49)	0.22	(0.42)	0.29	(0.45)	0.29	(0.46)	0.22	(0.41)
Ln(Family Income)	10.42	(0.89)	10.46	(0.86)	9.99	(1.03)	10.26	(0.93)	10.32	(0.89)	9.79	(1.09)
Below Poverty	0.18	(0.39)	0.16	(0.37)	0.38	(0.49)	0.23	(0.42)	0.20	(0.40)	0.47	(0.50)
Employed	0.73	(0.44)	0.74	(0.44)	0.63	(0.48)	0.53	(0.50)	0.53	(0.50)	0.52	(0.50)
Work Disability	0.09	(0.29)	0.09	(0.28)	0.14	(0.34)	0.08	(0.27)	0.07	(0.26)	0.14	(0.35)
Cognitive Difficulty	0.06	(0.24)	0.06	(0.23)	0.10	(0.30)	0.05	(0.23)	0.05	(0.22)	0.10	(0.30)
Physical Difficulty	0.19	(0.39)	0.18	(0.38)	0.26	(0.44)	0.19	(0.39)	0.18	(0.38)	0.29	(0.45)

Birth State Baseline Mortality Rates (Per 1000, N = 48 States)			**Birth State X Birth Year Socioeconomic Variables (N = 669)**		
Pneumonia	1.06	(0.19)	Log Income Per Capita	6.18	(0.49)
Tuberculosis	0.64	(0.37)	Log Hospitals Per 1,000	-2.83	(0.46)
Diarrhea	8.22	(5.65)	Log Physicians Per 1,000	0.14	(0.36)
Cancer	0.96	(0.31)	Log Schools Per 1,000	0.72	(0.65)
Heart Disease	2.09	(0.63)	Log Educational Spending Per Capi	4.27	(0.79)
Maternal Mortality	0.64	(0.12)			
Malaria	0.03	(0.07)			

Notes: Sample sizes for education variables (Schooling, High School, College - 1980 census only): Men - 708,478 (White - 636,991, Black - 71,487), Women -739,845 (White - 651,165, Black - 88,680). Sample sizes for Family Income, Poverty, Employed, Work Disability: Men - 2,018,898 (White - 1,835,771, Black - 173,665), Women - 2,151,234 (White - 1,887,723, Black - 225,407). Sample sizes for Cognitive and Physical Difficulty (2000 census only): Men - 627,262 (White - 573,071, Black - 54,191), Women - 679,720 (White - 609,744, Black - 70,076).

Online Appendix 2 - Pneumonia Mortality Measurement and Additional Robustness Checks

1. Measurement of Pneumonia Mortality Rates

All-age *vs* infant rates: As discussed in the main text, we pursue our analysis using all-age pneumonia and influenza mortality rates averaged over 1930-1936 as the measure of pre-intervention or baseline rates (*base_pneumonia* in equations 1 and 2). We use the all-age rate in lieu of the infant rate because of the known underreporting of infant births (and to a lesser, extent, deaths) during the study era, particularly in the rural South (Linder and Grove, 1947; Ewbank, 1940) which, together, introduces noise in infant mortality rates (which are number of deaths divided by number of births in a year). In *Table 2* of the main paper, we show that if instead of directly using the all-age rate, we use it to instrument the infant rate then we can recover similar estimates. This is consistent with two stylized facts that point to the all-age series being an appropriate proxy for the infant series. First, pneumonia has dramatically higher morbidity and mortality rates among infants than for any other age group (main text, *Figure 1* and dependent variable means in *Table 2* of this Appendix). Second, and related to the first point, overall trends of the infant and all-age series are similar i.e. the all-age rate tracks the infant rate, the sharp break in trend in 1937 is evident in both series and sharper in the infant series (main text, *Figure 2*).

It is nevertheless important to demonstrate that the "first stage" holds with infant mortality rates as it does with all-age rates. See *Table 2* of this Appendix, where we use the level and the logarithm of the all-age and the infant pneumonia and influenza mortality rates as dependent variables. These are regressed on *Post* (=1 for 1937 an onwards, 0 otherwise), *Year* (a trend in birth year), *Post*Year,* and state fixed effects, using Vital Statistics data for 1930-1943. We find negative and statistically significant trend breaks (the coefficient on *Post*Year*) for both the level and log models and for both infant and all-age mortality rates.

Pneumonia *vs* the combined rate for pneumonia and influenza: A second potential concern with the pneumonia exposure measure we use is that it combines pneumonia with influenza mortality. However, this may serve to reduce measurement error for the following reasons. First, the two diseases share symptoms, for example, fevers, cough, malaise, and shortness of breath, and therefore may have been difficult to distinguish, particularly in the 1930s where radiographs were not widely used. Second, superimposed secondary pneumonia was often the proximal cause of death for those afflicted initially with influenza, complicating any genuine separation of the two.

We expect no bias from including influenza with pneumonia mortality counts because pneumonia, having a large bacterial component, was treatable with sulfa drugs while influenza, being viral, was not. So upon the introduction of antibiotics, the entire change in the combined rate is driven by the drop in pneumonia. In fact decadal data separating the two causes of death show that the infant influenza mortality rate held constant between 1930 and 1940, even as the infant pneumonia mortality rate fell substantially. Note that pneumonia dominated the combined series, with 8.9 deaths per 1,000 in 1930 compared with 1.3 deaths per 1,000 live births from influenza.

Although annual time series by state are only available for the compound measure, state level quinquennial data that separate deaths from pneumonia *vs* influenza are available for 1930, 1935, and 1940 in Linder and Grove (1947), and we uses these to investigate more formally whether using the compound variable might drive our results. In *Table 2* of the

main paper, we replaced the compound measure with pneumonia for the year 1935, and showed that our findings are robust to this change.

The separate series are plotted in *Figure 1* below by gender and race. For both genders and both races, it is clear that pneumonia dominated influenza in prevalence, and that it was only pneumonia that showed a significant decline after 1937. If one compares the 1930-36 pre-intervention period with the post-intervention period, there is no significant decline in influenza mortality rates, the drop from 1937 to 1938 is an artefact of an influenza pandemic in 1936-37 (which also led to an uptick in pneumonia cases since pneumonia is often caused by influenza).

Figure 1 also shows that mortality rates from both diseases were higher for men than for women, and for blacks than for whites. Both the gender gap and the race gap were larger for pneumonia than for influenza. Absolute declines in pneumonia mortality rates were greater for men than women and for greater for blacks than for whites but the declines are evidence in each of the groups. The graphs show the all-age rates but we confirmed that the infant rates exhibit similar patterns, for instance, in 1940 infant pneumonia mortality was 1166 per 100000 infants overall, 1375 and 950.9 for boys and girls respectively.

We discuss measurement error in pneumonia mortality as it applies to race in *Appendix 4*.

2. Additional Results and Robustness Checks

Multiple Hypothesis Correction: We obtain p-values consistent with multiple hypothesis testing utilizing a Bonferroni-type method modified to adjust for correlation across like outcomes, see Aker, et al (2011) and Sankoh, et al (2007) cited in the main paper for details. We compute intra-group correlations for the education variables, employment, family income, and poverty, and the three disability variables, respectively. We then compute the adjusted p-values as follows: $p\_adjusted = 1 - (1 - p(k))g(k)$ where $p(k)$ is the p-value for the k-th outcome, and $g(k) = M1-r(.k)$, where M is the number of outcomes in the group and r(.k) is the mean correlation among other (non-k) elements in the group. Our findings are robust to using the method of Kling, et al (2007).

Income mobility estimates: The regression estimates corresponding to *Figure 4* in the main paper are in *Table 1* of this Appendix. As is also clear from the confidence intervals in *Figure 4*, the estimated probabilities for membership of quintiles 1 and 5 are statistically significant, while the probabilities of membership of the middle quintiles are not. The construction of quintiles is detailed in *Appendix 1*. If instead of estimating independent linear probability models for each of the quintiles, we use an ordered logit specification, we find a similar pattern of results but now the coefficients are statistically significant for each of the five quintiles. They show a significantly lower tendency for sulfa-exposed cohorts to be in the bottom three quintiles and a significantly higher tendency for them to have incomes that place them in the top two quintiles. See *Table 1A* of this Appendix.

Triple difference specification in Section I-3b of paper: We extended the sample back to birth cohorts 1915 and onwards and defined *treated* as 1 for individuals exposed to sulfa during infancy (i.e. born in or after 1937) and as 0 for individuals who had their first exposure at age 10-15. *Post* is now equal to 1 for treated individuals born on or after 1937 and untreated individuals who were 10-15 on or after 1937 (see *Figure 2 in this Appendix*). The regressor of interest is now *post*treated*base_pneumonia* and the underlying two-way interactions and main effects are included. The estimated equation is

$$Y_{stc} = a + \lambda_1 * post_c * base\_pneumonia_s * treated_c + \lambda_2 * post_c * base\_pneumonia_s + \lambda_3 * base\_pneumonia_s * treated_c + \theta_{rs} + \eta_{rt} + \lambda_{rc} + \gamma X_{st} + \theta_s * \eta_t + e_{stc}$$

The terms $post_c * treated_c$, $post_c$ and $treated_c$ are absorbed by birth year fixed effects and $base_s$ by birth state fixed effects. The controls are as in column 4 of *Table 1* of the paper. Subscripts are s for state of birth, c for year of birth and t for year of enumeration (census). Any omitted variables would now have to have affected infants and not 10-15 year olds, in high *base_pneumonia* states more than in low *base* states, *post-1937* more than *pre-1937*.

Placebo break years: In *Table 4* of this Appendix, we complement our analysis of cohort trends breaks in the long-run impacts of early pneumonia exposure by defining a series of fake breaks. We replace *Post* with each year in the interval [1935,1939]. These results show that the first positive jumps (for positive outcomes like income; the jumps being negative for negative outcomes like poverty) and the largest significant jumps are found when 1937 is set as the break year, consistent with the arrival of antibiotics in that specific year. As expected, there is no systematic significant tendency for outcomes to improve post-1935 or post-1936 and the significant positive coefficients for post-38 and post-39 are consistent with sulfa drugs having already arrived in the preceding year or two years.

Selective migration of parents of sample cohorts: See Section I-3b of the paper where the potential bias is elaborated. We select 20-40 year olds as the population group most likely to give birth during the sample period. Births and deaths in this age range are limited, which allows us to focus on changes in population created by migration. We regress the logarithm of population in each state-year cell on *post*base_pneumonia*, and the controls in equation (1), including income. The results are in *Table 5* of this Appendix. There is no evidence of selective migration.[1]

Selective migration of the sample cohorts*:* The more conventional concern is that the birth cohorts of interest may have migrated between birth and the census date at which their adult outcomes are recorded, and that this may have influenced returns for some. So, we modelled migration as an outcome. The dependent variable *migration* is defined as residing in a state different to the birth state at the time of enumeration. We find no significant impact of sulfa-exposure on the propensity to migrate (*Table 6* of this Appendix). An implication of this is that the gains in economic mobility achieved by post-sulfa cohorts were not achieved by moving to opportunity but, more likely, by being skilled to exploit opportunity; the caveat being that we have here only analyzed inter-state migration and, since we do not full birth histories, we cannot identify age of migration or return migration.

Selective fertility*:* See Section I-3b of the paper where the issue is elaborated. The 1950 census is used because it records characteristics of the parents of the birth cohorts in our sample. We estimate the specification in *equation (1),* but using parental characteristics (age,

[1] Controlling only for year and state fixed effects, we see the expected weakening of migration along a state-pneumonia gradient: after 1937, states with higher pre-1937 pneumonia had larger populations, consistent with smaller outflows following sulfa-led convergence in disease levels. However, this effect is small and rendered insignificant once we control for state income per capita at baseline (which the main models in the paper control for).

race, education, work status of mother, and household income) as dependent variables. The results are in *Table 5* of this Appendix).

Additional robustness checks: *Table 3* of this Appendix presents checks that complement those reported in the main text in Section I-3b. These are listed and briefly discussed here.

-Using *post*base_pneumonia* as an instrument for birth cohort X birth state (yearly) pneumonia and influenza mortality (Panel A). This check formalizes the intuition behind the reduced form estimates in the main paper by explicitly modeling the implied first stage relationship. This check also addresses potential measurement error in both the yearly pneumonia mortality series and the baseline measure, though the similar effect size magnitudes suggest that this is not a major issue.

-Adding *post*scarlet fever, post*meningitis, post*typhoid* and *post*diphtheria* as additional controls. These diseases were impacted by the antiobiotic revolution in 1937 but they were far less prevalent, among infants and overall, than pneumonia (Panel B). Consistent with this epidemiology, controlling for these conditions does not create significant changes in our estimates.

-*Removing Arizona and New Mexico* (Panel C). These states had the two highest baseline pneumonia mortality rates in the sample (1.44 and 1.49 deaths per 100,000 individuals, respectively, against a mean (sd) of 1.06 (0.19), and may be deemed outliers. Our results were substantively unchanged removing these states, which is not surprising given the (relatively) small number of sample individuals born in either area.

-*Restricting the sample to 1935-1941* (Panel D). This restriction allows us to exclude early Depression Era cohorts and World War II cohorts, with the results remaining similar to baseline results.

-*Excluding (Panel E) or using exclusively (Panel F) data from the 2000 Census*. With regards to the former, there is some concern in the literature that the 2000 census microdata sample may be subject to inaccuracies in age reporting (Alexander et al., 2010; see discussion in *Appendix 1*). With regards to the latter, we focus on this single census so as to assess the potential of bias from mortality selection as the birth cohorts in our sample begin to age by the 2000 census (the 1937 cohort is 63 in 2000). In both cases, the results are broadly consistent with the findings from pooling all census years. We do not estimate models for education in this census since, after 1980, single years of schooling below 10^{th} grade and above five years of college were not distinguishable (i.e., grouped).

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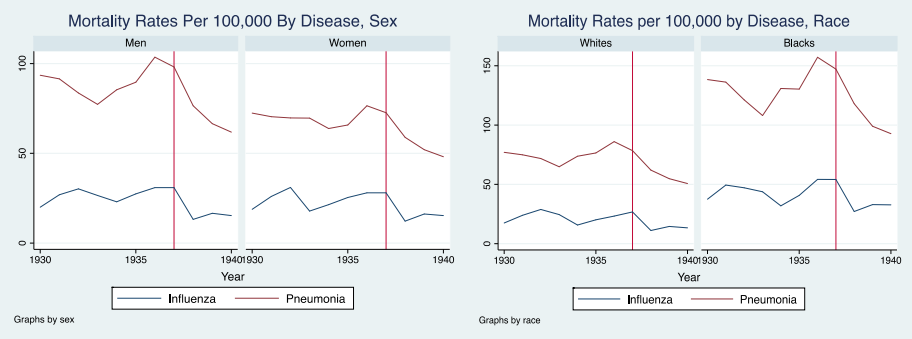
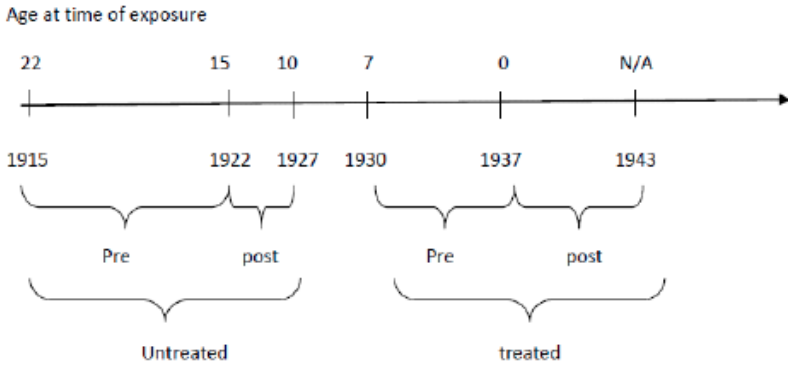
Linder, Forrest E., and Robert D. Grove. 1947. *Vital Statistics in the United States 1900-1940*. Washington, DC: United States Government Printing Office.

Figure 1: Pneumonia vs Influenza Mortality Rates by Gender and Race



Source: Quinquennial data from Vital Statistics records.

Figure 2 – Triple Difference Schema



Notes: See main text for details.

Table 1: Income Mobility Estimates

	Income Quintile				
	1	**2**	**3**	**4**	**5**
post*base	-0.0245*	0.00431	-0.0102	0.00887	0.0215***
	(0.0122)	(0.00847)	(0.00936)	(0.0131)	(0.00677)

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each cell is the coefficient from a different regression. The dependent variable is a dummy variable = 1 if the individual belongs to the income quintile listed in the column header and zero otherwise. All models include the controls in column (4) of *Table 1*. *N = 1.976.203* for all models. Construction of quintiles uses the income distribution for pre-sulfa cohorts.

Table 2: Trend Breaks in 1937 in Infant and All-Age Mortality Rates from Pneumonia and Influenza

	Levels		Logs	
	All-age Mortality Rate (per 1000 inhabitants)	**Infant Mortality x 1000 Births**	**All-age Mortality Rate (per 1000 inhabitants)**	**Infant Mortality x 1000 Births**
Post	-0.075**	-0.732**	-0.047*	-0.077*
	(0.032)	(0.265)	(0.023)	(0.043)
Year	0.002	0.284***	-0.000	0.044**
	(0.009)	(0.095)	(0.008)	(0.017)
PostxYear	-0.095***	-0.604***	-0.103***	-0.094***
	(0.015)	(0.128)	(0.014)	(0.022)

Notes: *** - p<0.01, ** - p<0.05, * - p<0.1. Each column represents a separate regression, regressing the level or log of the dependent variable denoted in the column header on *Post* (=1 for 1937 and above), *Year, Post*Year*, and state fixed effects. The level and log allow us to assess absolute and relative trend breaks, respectively. The sample includes observations for 48 states over the period 1930-1943 (N = 672). See *Appendix 1* for data sources.

Table 3: Further Robustness Checks

	Schooling	High School	College	Employed	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
Panel A - *Post*Baseline_pneumonia* as an IV for birth state*cohort pneumonia rates									
*post*base_pneumonia*	-1.939**	-0.273**	-0.231**	-0.084*	-0.240**	0.091*	0.016	0.133**	0.072
	(0.950)	(0.135)	(0.109)	(0.046)	(0.116)	(0.048)	(0.021)	(0.064)	(0.088)
	[0.10]	[1.4 % pt]	[1.2 % pt]	[0.5 % pt]	[1.3%]	[-0.5 % pt]	[-0.08 % pt]	[-0.7 % pt]	[-0.4% pt]
Panel B - Adding Additional Control Diseases									
*post*base_pneumonia*	0.566***	0.0863***	0.0781***	0.0339***	0.0487**	-0.0206**	-0.0079	-0.0313***	-0.0177
	(0.0782)	(0.0136)	(0.0169)	(0.009)	(0.0190)	(0.00931)	(0.0084)	(0.0114)	(0.0210)
Panel C - Removing Outlier States									
*post*base_pneumonia*	0.511***	0.0757***	0.0623***	0.0213**	0.0654***	-0.0260**	-0.00291	-0.0367***	-0.0184
	(0.0738)	(0.0120)	(0.0137)	(0.00901)	(0.0223)	(0.0100)	(0.00622)	(0.00957)	(0.0193)
N	702980	702980	702980	2,004,392	1,962,052	2,004,392	1,905,091	622817	622817
Panel D - 1935-1941 Cohorts Only									
*post*base_pneumonia*	0.412***	0.0711***	0.0493***	0.0125	0.0779***	-0.0246**	-0.00572	-0.0238**	0.00917
	(0.118)	(0.0168)	(0.0171)	(0.0104)	(0.0243)	(0.0115)	(0.00904)	(0.0115)	(0.0251)
N	400,631	400,631	400,631	1,155,426	1,130,846	1,155,426	1,094,801	365,957	365,957
Panel E - Excluding 2000 Census									
*post*base_pneumonia*	0.541***	0.0781***	0.0647***	0.0140	0.0582**	-0.0196*	0.0667		
	(0.0761)	(0.0115)	(0.0129)	(0.00985)	(0.0279)	(0.0115)	(0.205)		
N	708,478	708,478	708,478	1,391,636	1,365,437	1,391,636	1,391,636		
Panel F - 2000 Census Only									
*post*base_pneumonia*				0.0442**	0.0795*	-0.0363*		-0.0340***	-0.0183
				(0.019)	(0.044)	(0.021)		(0.0101)	(0.0191)
N				627,262	610,766	627,262		627,262	627,262

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each panel describes a different robustness check and each column X cell reports estimates on post*base_pneumonia from a different regression. All models include the controls in Column (4) of Table 1 in the main paper, and all results are for the male sample, with the sample size for each variable given in Table 1 of the main paper unless otherwise indicated. In Panel A, we replace the reduced form in the main paper with 2SLS. To the extent that one of cohort-state pneumonia or base_pneumonia is measured with error, this specification can be thought of addressing measurement error. The implied effect sizes (scaled (by the first stage) impacts of a 1 s.d. decrease in baseline infant pneumonia on the outcomes), which are given in the square brackets, are almost exactly the same size as those presented in the main paper. First stage F-statistics (available upon request) are 5.3-5.5 across the different models. Of note, the coefficients are flipped in sign which is as to be expected - greater pneumonia rates in infancy are associated with worse outcomes.

In Panel B, we add to our specification post*scarlet fever, post*meningitis, post*typhoid fever, and post*diphtheria mortality. Of these, meningitis in particular declined markedly with the arrival of sulfa dtugs, though mortality rates were far lower than for pneumonia. Diphtheria and typhoid mortality rates, too, were low compared to pneumonia mortality. In Panel C, we remove New Mexico and Arizona, two outliers in the base_pneumonia distribution. In Panel D, we restrict our sample to the 1935-1941 birth cohorts, which excludes both the early Depression years and World War II. Our findings are effectively unchanged when looking at this restricted sample. In Panel E, we exclude the 2000 census from the sample due to concerns around inaccuracies in age reporting that may impact some sample members (in particular, the 1935 birth cohorts and earlier - Alexander, et al, 2010). We do not have estimates for cognitive and physical disability as these variables are only available for the 2000 census. Finally, in Panel F, we examine outcomes in the 2000 census only. The education estimates are not included here given that individual years of schooling are not available after the 1980 census.

Table 4: Adult Outcomes Associated with Fake Trend Breaks in Birth Years Surrounding 1937

	Schooling	High School	College	Employment	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
Panel A - Post = 1 for 1935 onwards									
*post*base_pneumonia*	-0.164	-0.00714	-0.0424***	-0.00687	-0.0210*	0.00186	0.00515	0.00933	0.00365
	-0.12	-0.0199	-0.0148	-0.00608	-0.0124	-0.00632	-0.00478	-0.00766	-0.0157
Panel B - Post = 1 for 1936 onwards									
*post*base_pneumonia*	0.0642	0.00925	-0.0102	-0.00454	-0.0191	0.000147	-0.00217	-0.00624	-0.0177
	(0.124)	(0.0166)	(0.0176)	(0.00911)	(0.0176)	(0.00602)	(0.00646)	(0.0106)	(0.0153)
Panel C - Post = 1 for 1937 onwards									
*post*base_pneumonia*	0.541***	0.0781***	0.0647***	0.0226**	0.0645***	-0.0247**	-0.00442	-0.0340***	-0.0183
	-0.0761	-0.0115	-0.0129	-0.00888	-0.022	-0.0101	-0.00631	-0.0101	-0.0191
Panel D - Post = 1 for 1938 onwards									
*post*base_pneumonia*	0.411***	0.0361*	0.0551***	0.0141**	0.0382***	-0.00511	0.000726	-0.0119	-0.0197
	-0.12	-0.0181	-0.0166	-0.00606	-0.0134	-0.00673	-0.00591	-0.00841	-0.0134
Panel E - Post = 1 for 1939 onwards									
*post*base_pneumonia*	0.201*	0.0109	0.0310**	0.0203**	0.0298**	-0.00812	-0.000851	-0.012	-0.0101
	-0.102	-0.0157	-0.0139	-0.00864	-0.0141	-0.00626	-0.0051	-0.0077	-0.0104

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each panel X column represents a different regression, with the reported coefficient the estimate on *post*base_pneumonia*. The models are identical to those presented in column 4 of *Table 1* in the main paper, except *Post* is refined to be =1 for a number of placebo years (1935, 1936, 1938, and 1939). The positive impact estimates are *largest* when Post is set to 1937 (Panel C, hich identical to column 4 of *Table 1*), which is consistent with the arrival of sulfa drugs that year. The sample sizes are the same as in *Table 1*.

Table 5: Further Robustness Checks- Selection Issues

Panel A - Fertility Selection		Panel B - Selective Migration	
Mother's Education	0.0502	**Full**	0.031
	(0.170)		(0.071)
Mother's Age	-0.913	**High School**	-0.127
	(0.653)		(0.201)
Mother Working	-0.0274	**White**	0.060
	(0.0192)		(0.078)
Log(Household Income)	0.0421	**Black**	0.072
	(0.0802)		(0.281)
Black	0.0039		
	(0.011)		

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors corrected for birth state level clustering in parenthesis. As before, reported coefficients are those on *post*base_pneumonia* in equation 1 in the text and each cell represents a different regression. In Panel A the outcomes of interest are characteristics of the parents of the 1930-1943 birth cohorts. We use 1950 census data to estimate these regressions as most of the sample cohorts were living with their parents at this time and therefore their information is available. The controls included in this model are the same as col. 4 of Table 1. Sample sizes: Mother's education, age, labor force, race = 445,924; family income = 82.730 (was only asked of a random subset of census households in 1950). Panel B uses data are from the US Census Microdata for 1930 and 1940, collapsed to the state year level. We effectively have data for three time points: 1930, 1935, and 1940. 1935 data come from retrospective state of residence questions in the 1940 census. We treat 1930 and 1935 as pre-intervention years and 1940 as a post-intervention year. The dependent variable in all models is log(population) of individuals age 20-40 years in 1930. We choose this age group given their higher propensity to migrate as well as to remove population changes occuring on account of births and deaths (which are concentrated at the tails of the age distribution). Population structure effects will be small given that we are focusing on the same birth cohorts over a short period of time. For these models, the controls are the same as in col. 3 of Table 1. We do not include state specific time trends given that we only have three time points. N = 147 state-years.

Table 6: Migration of Sample Birth Cohorts

	All Men	Black Men	Black Men
*post*base_pneumonia*	-0.0114	-0.0221	-0.0421
	(0.0114)	(0.0657)	(0.0867)
*post*base_pneumonia*slave fraction*			0.0921
			(0.182)
N	2,018,898	173,646	170,844

Notes: *** P<0.01, ** p<0.05, * p<0.01. Model specification is as in Table 1, col 4. Now the depedent variable is migration, which is = 1 if the individual reporting living in a different state than the birth state at the time of census enumeration. Here we use the sample of individuals born in 1930-1943. For black men, we include the same access and measurement error corrections as described in the main text and Table 3.

Online Appendix 3 – Results for Women

Estimates of the long run returns to birth year exposure to pneumonia for women are in *Table 1* and correspond to estimates in column 4 of *Table 1* in the text. In contrast to the case for men, the coefficients are small and not significantly different from zero, except for work-limiting and cognitive disability for which the coefficients are positive. In the absence of controls, the coefficients for men and women are similar, but the coefficients for women are much more sensitive to controls.[1]

In this section, we investigate potential explanations for the gender difference in long run effects of pneumonia exposure. One is that pneumonia morbidity and mortality were greater among male infants than among female infants at baseline. We obtained incidence data for children (under age 5) from Britten (1942), who reports results of a 1934-1936 US Public Health Service national survey, and these show that pneumonia incidence rates were over 30% higher for boys *(Figure 1a in this Appendix).* There is also a large gender difference in mortality from pneumonia; see *Figure 1b* below which plots pre-intervention all-age influenza and pneumonia mortality.[2] The (unadjusted) absolute decline in pneumonia mortality between the baseline period (1930-1936) and 1940 was 50% larger for men (a decline of 32 versus 21 deaths per 100,000 for men and women, respectively). As mortality is an extreme case of morbidity, mortality differences proxy severe rates of infection. So, overall, it is credible that boys gained more from exposure to antibiotic therapy in infancy than girls because of their higher risks of contracting (severe) pneumonia.

A competing explanation is that following exposure to antibiotics in infancy, subsequent complementary investments were larger among men than among women. This was an era when women faced restricted access to the labor market, for example, Goldin (1991) examines the confluence of social consensus and inefficient personnel practices in restricting opportunities for married women. "Marriage Bars" prohibited the hiring of women and allowed for termination of employment contracts after marriage, greatly limiting female labor supply during the Depression Era and through to the early 1950s (Goldin, 1991). Their disappearance was ultimately driven by changing social norms with regards to women's work, a growing clerical sector, and rising female education (Costa, 2000; Goldin, 2006). However, for the marginal sulfa cohort member born in 1937, the existence of restrictions on employment will have tended to diminish returns to human capital investments through their early and middle childhood. This, in turn, may have discouraged educational investments reinforcing sulfa-led improvements in early life health and cognitive development.

We test the hypothesis that social and institutional constraints on women dampened the long-run returns to improved early life health for women by interacting the exposure term, *post*base_pneumonia* with indicators of gender differences in educational investments and labor market outcomes. In particular, we create two measures – the female-male

[1] It is unclear why the disability rates should be worse for exposed women. In the absence of state trends, the coefficients for disability are insignificant rather than positive. The sensitivity to controls suggests to us that the estimates are not reliable, and so these results may be artefactual. It is also pertinent to observe that in Table 2 of this appendix we see that the odd positive signs on disability tend to disappear as the relative share of women who complete college increases.

[2] For a discussion of the use of influenza and pneumonia mortality rather than pneumonia mortality alone, see Section I-3b of the main paper and *Appendix 2*. We use the all-age rate because the infant mortality rate is not available by gender for this period.

employment and college-completion ratios for individuals aged 25-40 in the 1940 census. We also considered gender ratios of high school completion and Mincerian returns to schooling, but these exhibited less variance than the chosen measures. The ratio of female to male employment ranged from 0.17 to 0.67 across the US states in 1940 and the college completion ratio from 0.45 to 1.15, making it pertinent to investigate if this spatial variation influenced the incentives for younger women to invest in human capital. Rendall (2010), for instance, argues that female labor force participation rates during this period were converging with male rates ahead of any changes in wages, suggesting that this variable proxies for incentives to invest in human capital for women (given their comparative advantage in brain-intensive as opposed to brawn-intensive tasks relative to men).

The results are in *Table 2* of this Appendix. The hypothesis is that, even if the average effects for women are small, they increase systematically as the female-male gap in college completion and employment narrows. This implies a positive coefficient on the triple interaction term. However, looking across outcomes, we see insignificant or negative coefficients. So, there is no evidence of gradients in returns in these variables, or at least we do not have sufficient power to detect significant effects for women in regions with relatively high relative participation of women in college and the labor market. This contrasts with our findings for black men in less segregated states in the main paper, but it may be that the sample of women that did benefit was small and/or that we need higher resolution data (i.e., below the birth state-birth cohort level) to identify gradients for women.

As we find no evidence that women's education and labor market outcomes benefited from sulfa-drug exposure in infancy, even women in states where college completion and labor force participation of women were relatively high, we proceeded to explore impacts on marriage market outcomes. Using data from the 1980 census, we investigated marital status, age of first marriage, spouse age, spousal education, and completed fertility (children ever born). As seen in *Table 3* of this Appendix, we find little evidence of impacts of exposure on most of these outcomes, though we see a strong negative association between sulfa exposure and spousal age.

In *Table 4* we present results for women disaggregated by race, corresponding to the estimates for men in Table 4 of the main paper. For black women, we additionally control for measurement error as discussed in the paper. There are no robust impacts for women of either race.

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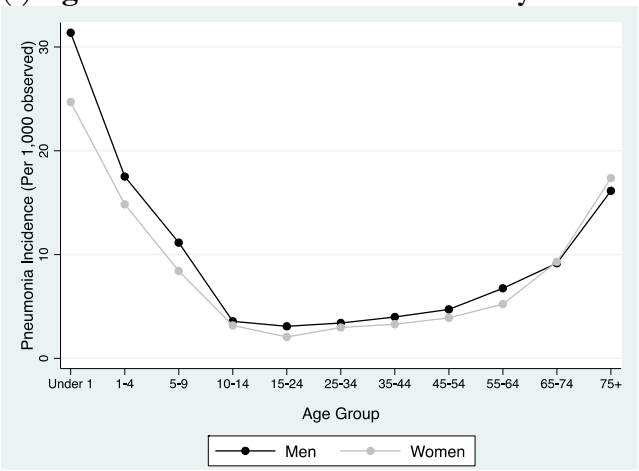
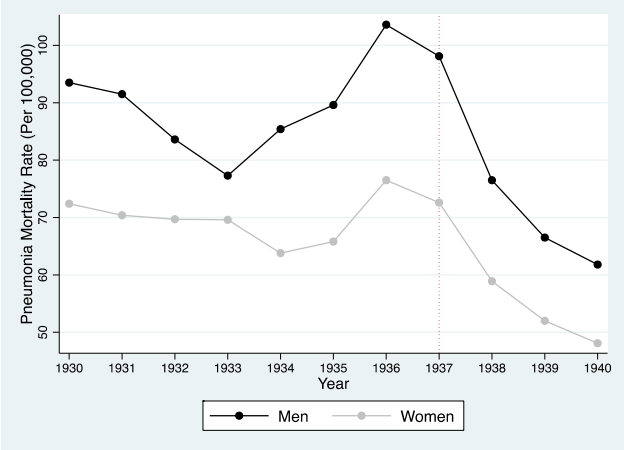
Figure 1 – Pneumonia by Gender

(a) **Age Profile of Pneumonia Morbidity**



(b) **Trends in Pneumonia Mortality**



Notes: Panel A plots nationwide, sex specific pneumonia incidence rates from 1934-1936 as reported by Britten (1942). Panel B plots national trends in all-age pneumonia mortality by sex from 1930-1940 (source: Linder and Grove, 1947). The higher-level lines are the lines for men.

Table 1: Estimates for Women of Impacts of Sulfa Exposure in Infancy on Adult Outcomes

	Coef on post*base_pneumonia	N	P-Value (Multiple hypothesis adjustment)	Effect of 1 S.D. Decrease in Baseline Pneumonia
Schooling	-0.00285	739,845	1.000	-0.001
	(0.143)			
College	0.00610	739,845	0.835	0.1% pt
	(0.0178)			
High School	-0.0224	739,845	0.420	-0.4% pt
	(0.0224)			
Employment	0.00650	2,151,234	0.992	-0.1% pt
	(0.0104)			
Family Income (log)	-0.00940	2,113,130	0.995	-0.2% pt
	(0.0297)			
Poverty	-0.00618	2,151,234	0.939	-0.1 % pt
	(0.0139)			
Work Limiting Disability	0.0370**	2,151,234	0.926	0.7% pt
	(0.0158)			
Cognitive Disability	0.0224**	679,720	0.064	0.5% pt
	(0.00995)			
Physical Disability	0.0274	679,720	0.222	0.5% pt
	(0.0167)			

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each row contains a different adult outcome. The controls are birth state and birth year fixed effects, post*baserate for the six control diseases listed in the main paper, birth state health expenditure in 1930*birth year, state and year varying controls for income, physicians, education, and birth state specific trends. Estimates reported are coefficient on *post*base_pneumonia*. The corresponding estimates for men are in Table 1 column 4 of the main paper; notes to that table define multiple hypothesis testing.

Table 2: Gradients in Long Run Returns to Sulfa Exposure of Women by Female-Male Ratios of Employment and College Completion

	Schooling	High School	College	Employed	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
Panel A - Female/Male Employment Share									
post*base_pneumonia	0.592*	0.0538	0.0362	0.022	-0.0384	0.0166	0.618	0.0461*	0.0690**
	(0.337)	(0.0647)	(0.0484)	(0.029)	(0.0614)	(0.0313)	(2.306)	(0.0239)	(0.0340)
post*base_pneumonia*employment	-3.650**	-0.458	-0.178	-0.101	0.172	-0.125	3.813	-0.149	-0.268
	(1.723)	(0.433)	(0.245)	(0.180)	(0.310)	(0.162)	(12.18)	(0.124)	(0.187)
post*employment	3.897**	0.477	0.198	0.076	-0.193	0.182	-0.613	0.135	0.211
	(1.879)	(0.438)	(0.268)	(0.183)	(0.348)	(0.172)	(12.98)	(0.134)	(0.192)
Panel B - Female/Male College Completion									
post*base_pneumonia	0.00646	0.0126	0.00646	0.012	0.0162	-0.0202	0.581	0.0456***	0.0571***
	(0.0218)	(0.0234)	(0.0218)	(0.012)	(0.0366)	(0.0181)	(1.105)	(0.0121)	(0.0194)
post*base_pneumonia *college_ratio	-0.00182	-0.117***	-0.00182	-0.021	-0.0807	0.0451	1.517	-0.0749**	-0.101**
	(0.0523)	(0.0427)	(0.0523)	(0.026)	(0.0607)	(0.0305)	(1.680)	(0.0304)	(0.0469)
post*college_ratio	-0.00207	0.141**	-0.00207	0.021	0.119	-0.0616	-3.053	0.104**	0.115*
	(0.0742)	(0.0566)	(0.0742)	(0.035)	(0.0854)	(0.0422)	(2.337)	(0.0411)	(0.0665)
N	739,845	739,845	739,845	2,151,234	2,113,130	2,151,234	2,052,480	679,720	679,720

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each cell contains the results from a separate regression. The dependent variable for each regression is provided in the column header. Model specifications are the same as in Table 1 but here we include interactions between post*base_pneumonia with female-male employment for 25-50 year olds and college completion for 25-40 year olds in the 1940 Census. These variables are chosen as markers of returns to human capital investment for women relative to men. The ratio variables are scaled such that zero reflects the lower bound of that particular variable.

Table 3: Impacts of Sulfa Exposure in Infancy on Marriage Market Outcomes of Women

	Coef	N
Ever Married (=1)	-0.00873	749,873
	(0.00839)	
Age of First Marriage	0.128	568,745
	(0.156)	
Spouse Age	-0.588**	568,745
	(0.264)	
Spouse Education	0.126	568,745
	(0.129)	
Children Ever Born	0.0428	749,873
	(0.0680)	

Notes: ** - $p<0.05$. Each cell represents the estimated coefficient on *post*base_pneumonia* from a separate regression. Robust standard errors, clustered at the birth state level, in parenthesis. Model specification and covariates as in Table 1 of this appendix. We use the 1980 census as, in this, the marginal sulfa birth cohort is aged 43 at enumeration. Changes in marital status, age of first marraige and fertility after this point are likely to be small.

Table 4: Race-Specific Estimates for Women- Adult Outcomes as a Function of Sulfa Exposure in Infancy

	White Women		Black Women		
	Coef	**# Obs**	**Coef**	**ME**	**# Obs**
Schooling	-0.0120	651,165	-0.128	0.228	83,963
	(0.158)		(0.251)	(0.459)	
College	0.00318	651,165	0.0362	0.0822	83,963
	(0.0200)		(0.0352)	(0.0614)	
High School	-0.0321	651,165	0.0347	0.147	83,963
	(0.0267)		(0.0498)	(0.086)	
Employment	0.0102	1,918,476	-0.0378	-0.0624	232,758
	(0.00996)		(0.0356)	-0.0647	
Family Income (log)	-0.0203	1,887,723	0.0616	0.0190	225,407
	(0.0291)		(0.0774)	(0.116)	
Poverty (%)	-0.00739	1,918,476	0.00552	0.0348	232,758
	(0.0148)		(0.0229)	(0.0361)	
Work Limiting Disability	0.0501***	1,918,476	0.0110	0.0429	232,758
	(0.0184)		(0.0583)	(0.052)	
Cognitive Disability	0.0176	609,644	0.0470*	-0.0251	70,076
	(0.0113)		(0.0250)	(0.0482)	
Physical Disability	0.0255*	609,644	0.107	0.276**	70,076
	(0.0137)		(0.0784)	(0.125)	

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Model specification and controls are the same as in *Table 1 of this Appendix*. The column headed ME adjusts for measurement error as described in the main paper (see Table 3 notes). Corresponding results for men are in Table 3 of the main paper.

Online Appendix 4 – Additional Race Specific Results

This Appendix is organized as follows. First, we provide additional evidence to support our contention that blacks in segregated (Southern) states were able to access sulfa drugs. Second, we discuss and analyze the problem of greater incompleteness and inaccuracy in pneumonia mortality rates in the Southern states. Third, we provide descriptive evidence on the measures of institutional segregation used in the paper. Fourth, we present additional robustness checks, extending the discussion in Part II-4 of the paper. The first and second sections of this Appendix summarize results from a companion paper, Bhalotra and Venkataramani (*in progress*).

1. Access to Sulfa Drugs

We show in the paper that blacks who were born in an environment marked by institutionalized segregation reaped smaller and less pervasive gains from infant exposure to reduced pneumonia created by the introduction of antibiotics. We contend that there was a clear "first stage" for blacks, even in segregated states, and that segregation played a part in translation of first stage (pneumonia mortality) to second stage impacts (adult outcomes). In our companion paper, results of which are summarized here, we bolster the evidence that supports this claim.

Figure 6 of the main paper demonstrates that both blacks and whites in the Southern states experienced declines in all-age pneumonia mortality starting in 1937, with the absolute decline being larger among blacks. In *Table 1* of this Appendix, we reproduce "first stage" regressions by race and region using all-age as well as infant pneumonia mortality rates, and using the logarithm of the mortality rate and its level. With the level and logarithm of both all-age and infant mortality, we find statistically significant trend breaks for blacks and whites in the Southern as well as in the non-Southern regions. The level (absolute) breaks tend to be larger for blacks than for whites, consistent with *Figure 6*, while the log (relative) breaks are smaller. Regardless, the results demonstrate that all-age and infant pneumonia mortality declined significantly after 1937 for blacks, including in the US South.[1] In *Figure 7* in this Appendix, we also demonstrate that *post-1937*, there was convergence in pneumonia mortality rates, even for Southern blacks; this is the analogue of *Figure 3* in the main paper.

This finding is consistent with other evidence from this era. For example, Boustan and Margo (2014) demonstrate large improvements in infant mortality for blacks relative to whites in the period 1920-1945. They attribute this to improvements in public health, specific disease eradication efforts, and secular improvements in living standards for blacks. We contribute here some of the scarce specific evidence of a particular mechanism (sulfa drugs) incident in this period which lowered disease prevalence for a disease that had considerably higher baseline rates for blacks than for whites.

While *Table 1* of this Appendix demonstrates sulfa-driven improvements in pneumonia mortality (extensive margin), and mortality is widely thought to scale up with morbidity (Bozzoli et al, 2009, Almond, 2006), we might worry that the manner in which mortality scaled with morbidity was different for Southern blacks than for the rest of the population on account of segregation in access to health care. We investigated this further as follows.

[1] *Table 1* shows that the absolute declines in pneumonia were larger for Northern blacks than for Southern blacks or whites; but that the declines for Southern blacks were larger than for Southern whites.

First, the results in *Tables 4 and 5* of the paper serve as evidence that blacks in segregated benefited from sulfa drugs on the "intensive margin"; i.e the drugs were available for survivors of infant pneumonia infections. This is because we find that post-sulfa black cohorts born into states with the median level of segregation experienced positive gains across several outcomes, even if these gains were smaller than among blacks in less segregated states. Second, as stated in the paper, the cost of a whole course of sulfa drugs was equivalent to only about 1% of the monthly wage of black men in the 1930s, which is a relatively small cost for a life-saving drug, so it seems unlikely that income constraints limited access for black families.

Third, we found other evidence that Southern blacks accessed health innovations in this era. For example, Fung and Robles (*Forthcoming*) demonstrate large impacts of antenatal syphilis testing laws passed during the late 1930s and 1940s on black neonatal mortality. Neonatal mortality is a manifestation of maternal syphilis, which is generally either asymptomatic or mildly symptomatic, so their results suggest that black women did have access to medical therapies for conditions that were not immediately life threatening for them. These results square with the Tuskegee Syphilis Study in which researchers had to actively use chicanery to prevent black subjects from seeking syphilis treatment, a clear indication that treatment was readily available in the community at the time.

Fourth, we constructed additional evidence that blacks were able to access antibiotics for non-life threatening infections by studying trends in rheumatic fever by race. Rheumatic fever is a disease of the heart, joints, and or/brain that can *occur only after an infection caused by Group A streptococcal bacteria.*[2] These infections are usually not life threatening and include pharyngitis (popularly known as "Strep throat"), tonsillitis, and scarlet fever. If Southern blacks were less able to access antibiotics to treat strep-A infections than others, then it follows that rheumatic fever would decline relatively slowly for them. But, in contrast to this prediction, the evidence is that there were dramatic declines in rheumatic fever for Southern blacks, consistent with timely and efficacious treatment of streptococcal infections. *Figure 1* of this Appendix displays race-specific rheumatic fever mortality for the US South, and a dramatic convergence in rheumatic fever mortality rates over the 1940s. The largest declines occurred in the penicillin era (Bisno, 1990; Denny, 1994; Massell, et al, 1988): pre-1940 data on rheumatic fever were not available, but there was no major change in health care access for blacks relative to whites between 1937 when sulfa arrived and the early 1940s when penicillin arrived, so we can attribute these declines to *increased rates of treatment of antecedent non-life threatening bacterial infection.*

As explained in the paper, we nevertheless allow for heterogeneity in access to sulfa drugs across race and state by including a measure of the density of pharmacists in counties populated by blacks (allowing for a discontinuity in its effects after 1937), and our finding of segregation gradients stands up to this.

2. Measurement Error in Mortality Rates

In *Appendix 2* we explained that measurement error in infant mortality rates was greater than in all-age rates, and that pneumonia was often the end result of influenza and had similar symptoms to influenza, so the combined influenza and pneumonia mortality rate

[2] It is thought that antibodies produced following an infection caused by Group A streptococcal bacteria cross react with tissues around blood vessels, heart valves, and joints to cause rheumatic fever (Bisno, 1991).

(available by state and year) was likely to be measured with less error than the pneumonia (only) mortality rate (available quinquennially at the state level).

In this Appendix, we discuss the fact that under-reporting of births and deaths and inaccuracies in assignment of causes of death were very likely greater in the Southern states where 85% of blacks resided in the mid-1930s (Ewbank, 1987). State and race-specific time-invariant differences in measurement are captured in our specifications by race*state fixed effects, and general trends in the quality of vital statistics data are absorbed by race*year fixed effects. Linearly evolving state-specific secular improvements in measurement are accounted for by race*birth state specific time trends. We are nevertheless concerned that our estimates may carry a bias if the divergence we identify in long run outcomes of sulfa-exposure for blacks in more *versus* less segregated states arises spuriously on account of differential trends in the quality of data on pneumonia mortality rates in the more *vs* less segregated states. As discussed in the paper, we attempt to adjust for differences in measurement quality across state and race using proxies for the quality of vital statistics data around the time of the sulfa revolution. The proxies are the years in which a state entered the national birth and death registration systems respectively, obtained from Linder and Grove (1947). These were national conglomerates of states using similar best practices in vital statistics recording, so new entrants to the national registration system will have upgraded to national surveillance standards.

We nevertheless explore them here as previous work using black mortality rates has tended not to discuss measurement issues very much. First, we attempt to validate these measures against more substantive indicators of quality. The comprehensiveness of natality registration was analyzed in a nationwide vital statistics audit conducted in 1940 and 1950 by the US Public Health Service (Shapiro and Schacter, 1950). The audits utilized decennial census data from those years as the "gold standard" for measurement of the total number of births in the US and compared these totals to the number of births recorded by each state in their vital statistics in that year to yield an index of birth registration completeness (fraction of births recorded in the census that were also recorded in the state vital statistics). *Figure 2A* plots this completeness variable against the timing of entry into the National Birth Registration System. The figure shows considerable state variation in the completeness of registration in 1940, with better performance associated with earlier entry into the registration system.

There was no corresponding national audit of the death registration system. However, we were able to assess the relationship between the year of entry to death registration and a direct indicator of data quality available at the national (but not state) level, namely, the percentage of death certificates with no listed cause of death. *Figure 2B* shows that later entry into the death registration system is associated with a greater prevalence of un-coded causes of death. Note that according to historical documents, the death registration system included deaths outside hospitals and registration laws typically required a death certificate filled out by an appropriate authority in order to obtain a permit for disposal, cremation, or burial of a corpse (Wilcox, 1933).

We next investigate Ewbank's (1987) contention that vital statistics measurements were more error prone for blacks than for whites because more than 85% of blacks lived in the South during the sulfa drug era. As discussed in the paper, this is relevant to our identification of segregation gradients. *Figures 2C* and *2D* plot the years of entry into the birth and death registration systems against the percentage of the state population that was black. Both plots show that these proxies for registration data quality are inversely related to black population share, the relationship being more prominent for completeness of birth

registration. *Figure 2E* uses data from Linder and Grove (1947), which show prominent clumping in last digit of recorded age of death for blacks but not whites, another form of measurement error. In *Figure 2F* we plot the year of birth registration against the year of death registration, illustrating the range in these years across states and their positive correlation.

As discussed, historical research identifies underreporting as the common pathology in birth and death registration. Since the pneumonia mortality rate for children is a (scaled) function of deaths/births, this could in principle lead to over-estimation or under-estimation in mortality rates, although under-estimation seems more likely.[3] However, measurement error in the variable *base_pneumonia* (the pre-intervention pneumonia mortality rate) is larger in states with higher rates of *base_pneumonia (Figure 3E-3F* in this Appendix), and the expected bias is downward. To fix ideas, considering the following general measurement model from Bound, et al. (1994):

$$Y = BX + e$$

Suppose that the econometrician does not observe X, but does observe X*:

$$X^* = X + u$$

where u is a mean zero error term with standard deviation σ^2_u. The probability limit of the OLS estimator, b, is:

$$b = (X^{*\prime}X^*)^{-1} X^{*\prime}(X^*B - uB + e)$$

$$= B + (X^{*\prime}X^*)^{-1} X^{*\prime}(-uB + e)$$

The bias is thus equivalent to $\text{plim}[(X^{*\prime}X^*)^{-1}X^{*\prime}(- uB + e)]$. When $cov(u,X) = 0$, we have the classical measurement error case, and estimates of B are biased downwards. If the net effect of the error is distributed around zero, which is reasonable given that the direction of measurement error in any given year depends on the relative underreporting of births vis-à-vis deaths, this implies that $cov(u,X) > 0$, which would also bias downward estimates of B.

As discussed in Bound, et al. (1994), the proportional bias arising from measurement error can be conceptualized as being equal to the coefficient on X^* from a regression of u on X^*.[4] While such a regression is hypothetical, this motivates our inclusion of proxies for data quality using the years of entry to the birth and death registration systems respectively. In the paper, X is *base_pneumonia*. Replacing *post*base_pneumonia* with *post*(base_pneumonia+u)* in estimates of equation (1) in the paper by race produces the additional term *post*(u)* which we proxy with *post*year of birth registration* and *post*year of death registration*. The resulting estimates are displayed in *Table 3* of the main paper.

The more general specification, equation (2), allows returns to vary by birth state segregation, which creates the additional term of interest, *post*base_pneumonia*segregation*. Replacing this with *post*[base+u]*segregation* yields the additional terms *(post*u*segregation)* and *(post*u)*, omission of which will bias the coefficients on *(post*base*segregation)* and *(post*base)* if

[3] Birth registration quality will also matter for measurement error in all-age mortality rates because changes in all-age pneumonia and influenza mortality rates were primarily driven by infant and child pneumonia mortality (see *Appendix 2*) and because, at any adult age, the population at risk is given by some earlier birth cohort.

[4] Two-stage least squares approaches using an instrumental variable Z will ameliorate measurement error bias with $cov(u, X) = 0$. It may also do so in cases where $cov(u, X) \neq 0$ as long as $cov(u, Z) \neq 0$.

base is correlated with *u* (which *Figures 3E-3F* below show it is). The other additional term, *segregation*u* is absorbed by the (race-specific) state fixed effects. As above, we include two proxies for *u*, which are the years of birth and death registration respectively. Estimates are in *Table 5* of the paper. This is a fairly demanding test since the years of registration tended to be later in states with higher segregation (proxies by 1860 slave share) (see *Figure 3C-3D* below) (and also in states with higher pre-intervention (*base*) pneumonia- see *Figure 3E-3F*below). The figures show positive associations but they also demonstrate a fair degree of independent variation in the variables, available for identification.

3. Measures of Institutional Segregation

In the main text, we define and discuss three different markers of institutional segregation, the source and construction of which are discussed in *Appendix 1*. Here, we further define their empirical content. First, we plot the densities of the fraction of the state population that were slaves in 1860, and the ratio of white to black returns to education estimated from the 1940 census (*Figure 4*); both measures exhibit considerable variation across the states. Notes to *Figure 4* report summary statistics for the two variables. *Figure 5* plots the correlation of these variables across the states. The third measure of segregation used in the paper is simply South (where segregation was *de jure*) *vs* "North". In *Table 2* of this Appendix, we show that the black/white ratio in completed schooling, earned income and returns to schooling was lower in the South than in the North and that the 1860 slave fraction was much higher. *Table 3* shows the correlation matrix between these measures of black disadvantage. We demonstrate independent variation in institutional segregation, baseline pneumonia mortality rates, and markers of measurement error and access in *Figure 3* below.[5]

4. Gradients in Long-Run Impacts – Additional Results and Further Checks

As discussed in the paper, the main threats to identification and interpretation of the segregation gradients in long-run impacts for black men by institutional segregation are differences in measurement error and access to sulfa drugs. These issues were addressed in the paper and elaborated above. In this section we describe additional robustness checks.

Segregation gradients for white men - *Table 4* of this Appendix reports the analogue of *Table 4* in the paper for white men. In sharp contrast to the case for black men, there is no evidence that the long run returns to sulfa drugs were smaller in more segregated states. For most outcomes, the coefficients on *post*base*segregation* are not significantly different from zero.[6] This suggests that the gradients for black men are driven by a process specific to this

[5] For reasons detailed in the text, although we analyze three measures of segregation to demonstrate robustness of our findings to choice of measure, further checks are done with the 1860 slave fraction as this nests "South", and is available for all states and more clearly pre-determined than the race ratio of education returns.

[6] A notable exception is that sulfa-exposed white men in segregated states exhibited *higher* employment rates than in less segregated states, which *reinforces* the evidence that the racial gap in returns was larger in segregated states. The only other significant coefficients on the triple interaction term for white men are in panel C, where we use white relative to black returns, and the sample excludes most Northern states, so the heterogeneity explored is essentially within the segregated states (see the main text). Rates of high school completion of white men are increasing in the white-black gap, and this tendency is stronger in segregated states, again consistent with the evidence in the main paper of wider racial gaps in these states.

population group rather than by institutional features of segregated states that worsened outcomes for everyone.

The Great Migration - Refer section II-4 of the paper which motivates discussion of whether selective black migration from the South to the North might explain the segregation gradients that we identify. Previous work shows that the relatively educated were more likely to move northward (Vigdor, 2002; Aaronson and Mazumder, 2011). One might worry that this drives the stronger sulfa-led gains for blacks in the North relative to the South. The impact of any changes in North-South migration driven by economic opportunities that happened to coincide with the health shock in 1937 will be absorbed by *post*segregation* or *post*south*. Migration would only be a concern if it was induced by high pneumonia levels. However, we have shown that the introduction of sulfa drugs stimulated convergence in disease levels across states (*Figure 2* of the paper), which, given higher initial disease burdens in the South, implies South-North convergence.

So if migration were disease-led then it will have exhibited as South-North convergence (or a positive coefficient on *post*base*south* for positive outcomes like education and income). In fact we estimate a negative coefficient on this variable (*Table 4 in the paper)*, which indicates divergence of outcome-gains between blacks in the North and South. Hence, accounting for South to North migration would only strengthen the segregation gradients that we estimate. Estimates of migration equations were nevertheless obtained and they show that, conditional upon state income, there was no association between *base_pneumonia* and the propensity of the parents of our sample cohorts to migrate Northward *(Table 5 of this Appendix).* In *Appendix 2, Table 6,* we show for all-men and also for black-men, estimates of an equation modelling migration of members of the sample birth cohorts (the children). Again we see no evidence of endogenous migration.

Selective mortality- If sulfa-led mortality declines were larger in states with higher pneumonia burdens, and larger among Southern blacks because they had higher mortality rates then this could bias both *post*base* and *post*base*segregation.* Since the introduction of sulfa drugs was a positive shock, the marginal survivor was negatively selected and so this could produce the more muted long run effects in segregated states that we find. So as to estimate the empirical significance of differential selective survival rates of blacks in more *vs* less segregated states, we use race and state specific pneumonia mortality rates to replace individuals in the pre-1937 sample who died but who would have survived had sulfa drugs been available. Alderman et al. (2011) conduct a similar exercise. We assigned these individuals the lowest possible values of the outcome variables (for instance, a zero for high school and college completion and poverty status, or income at the mean of the bottom quintile). We find no appreciable change in gradients estimated on the simulated sample (*Table 6 of this Appendix*).

Age heaping- If numeracy was correlated with segregation and also with a tendency to "heap" age at values ending in 0 and 5 then the (classical) measurement error created by age heaping may express as segregation gradients. We investigated this by using the 1980 census data to plot frequencies by birth cohort, and there is no evidence of age heaping or of this being greater in the South (*Figure 6*).

The one anomaly among the 27 coefficients (9 outcomes x 3 measures of segregation) is for cognitive disability.

Race differences in child labor- Post-1937 improvements in child health may have raised the returns to child labor or early entry into the labor market alongside raising the returns to schooling (Bleakley, 2010b; Venkataramani, 2012). This is pertinent for Southern blacks, who lived in predominantly rural areas and for whom child labor laws had no significant impact on educational attainment, presumably because of a paucity of black schools and states being more likely to exempt black children from child labor laws (Lleras-Muney, 2002). It follows that another explanation for the segregation gradients may be that, in response to the positive health shock, blacks chose to invest their children's time in labor rather than schooling. However, this would remain a reflection of relatively low returns to schooling for blacks in this era.

Civil Rights legislation- The marginal cohort was 27 when the Civil Rights Act of 1964 was passed. This was too late in the life course to have influenced investments in education. It will have tended to raised income conditional upon education for our sample cohorts but, for this to bias our estimates, access to civil rights would need to have discriminated between close neighbors in the pre and post 1937 cohorts, which seems implausible. In any case, if the Civil Rights movement were in any way driving our results, we would see *larger* improvements in outcomes for blacks in the South than for any other group, but we see the opposite. So, accounting for the Civil Right Acts of the 60s would only strengthen our finding that Southern blacks did not benefit as much from a positive health shock in infancy, particularly in the realm of education.

In any case, intercept changes in outcomes associated with cohort differences in exposure to the 1960s Civil Rights movement are controlled for through the inclusion of birth cohort fixed effects, and inclusion of *post*segregation* helps account for any birth-cohort differences in participation in the Civil Rights movement across states with different levels of segregation.

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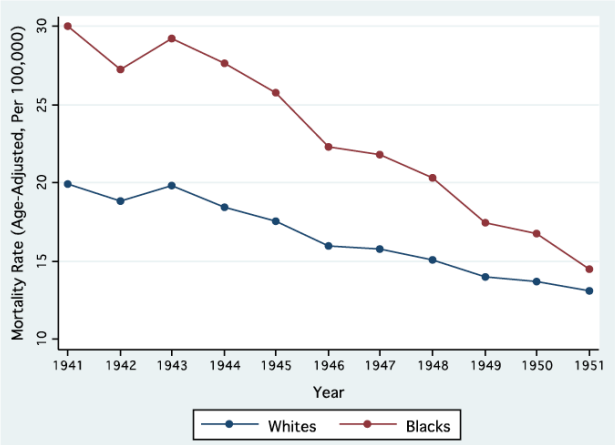
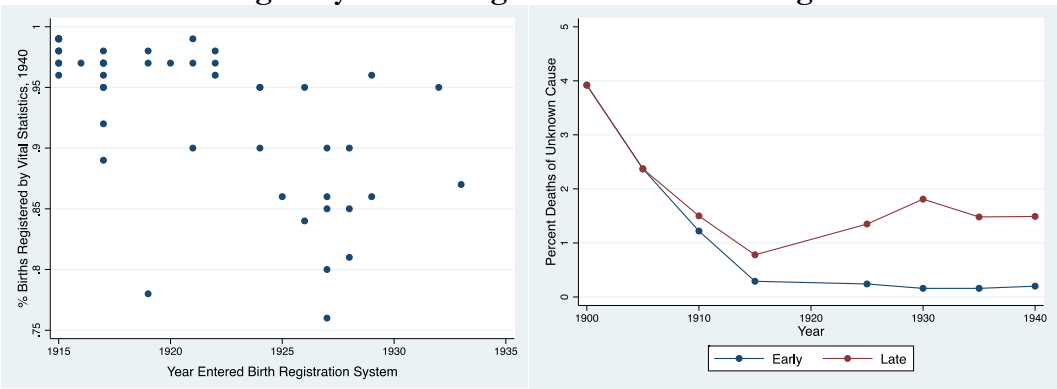
Figure 1 – Rheumatic fever mortality rates by race in the US South



Notes: Source is US Vital Statistics. Data are for the Southern states. Blacks have higher rates, which converge towards rates for whites over the course of the penicillin era. Data prior to 1941 are not available.

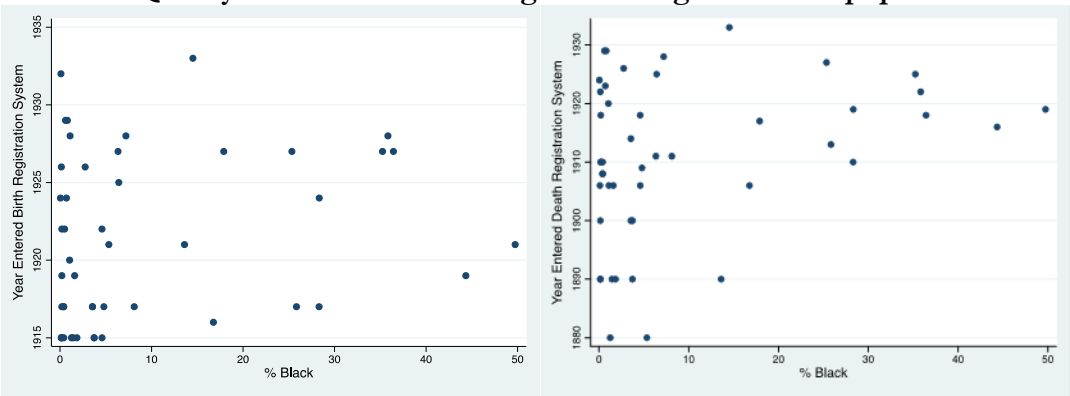
Figure 2 – Proxies for measurement quality of mortality rates

2A & 2B: Validating entry to birth registration and death registration



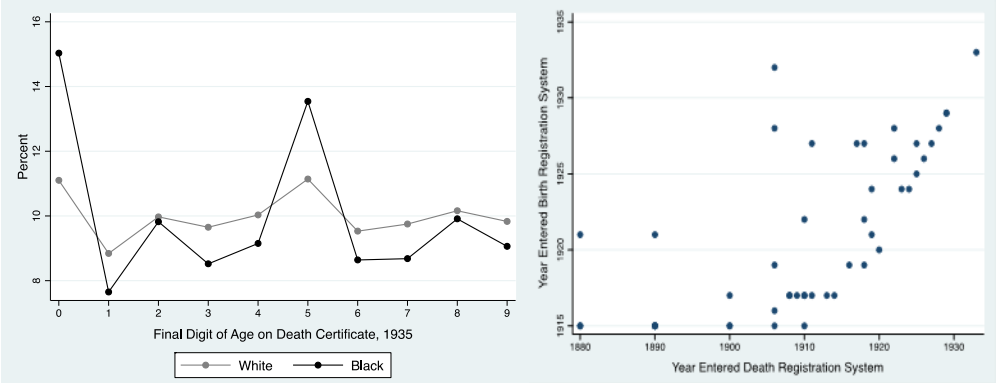
R = -0.63

2C & 2D: Quality of birth and death registration against black population share



R = 0.21 R=0.30

2E: Errors in age at death by race **2F: Birth against death registration year**



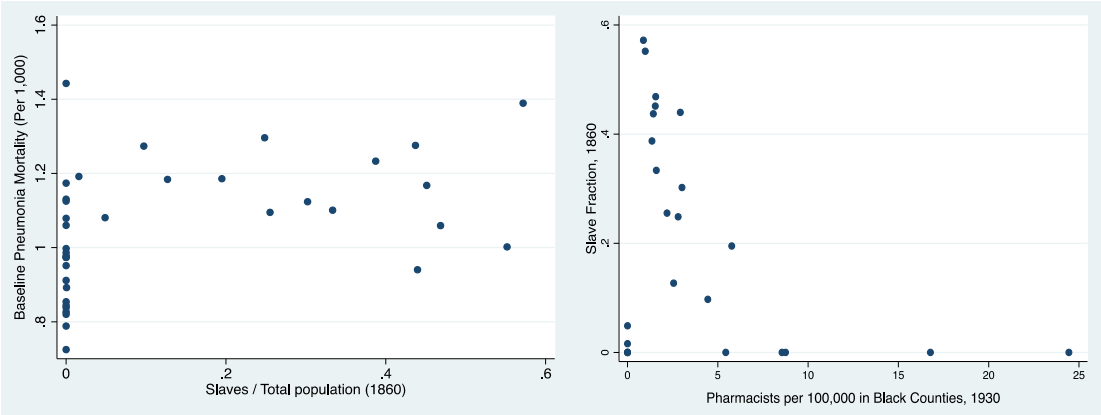
R = 0.68

Notes: Panel A plots a measure of birth registration completeness in 1940 (from Shapiro and Schacter, 1950) against year of entry into the birth registration system. Later entry is generally associated with less accurate reporting. Panel B plots a proxy for death registration system quality, the proportion of deaths

without an identified cause, against year for the group of states joining the death registration system before 1910 ("early") versus those joining after ("late"). These data are obtained at the national level from Linder and Grove (1947). Starting in 1910, unrecorded cause of death declined to close to zero for early registration states but not for late registration states. Unidentified cause of death data are not available at the *state*year* level, but the association here validates our use of timing of entry into the death registration system as a proxy. Panels (C) and (D) show that the quality of mortality rate data was worse in states with a higher fraction of blacks in the population, consistent with these being more rural states. Panel (E) uses national data from Linder and Grove (1947) on the frequency of 0-9 as the final digit in the recorded death certificate age by race, which is another proxy of death registration quality. For non-whites, there is a more prominent spike at 0 and 5, suggesting greater mismeasurement in age in this group. Panels (F) shows the relationship between the year of entry to the birth registration system and year of entry to the death registration system across states.

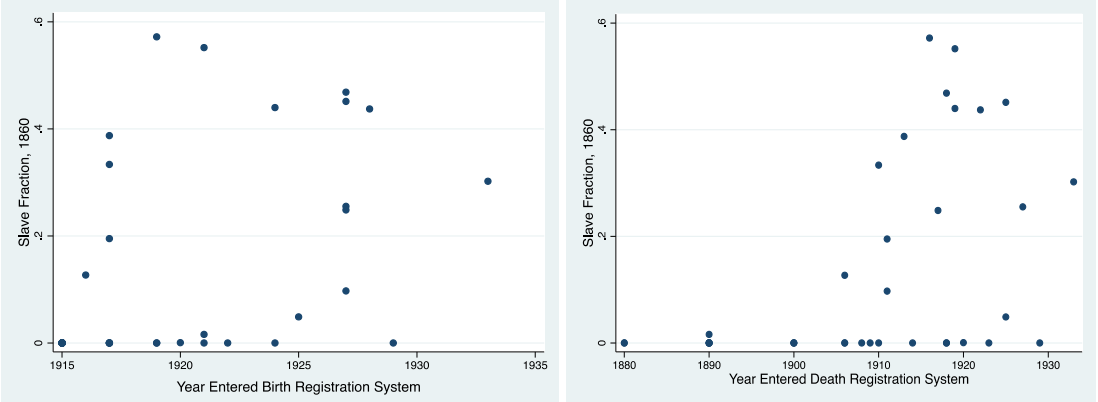
Figure 3 – Cross-state scatters of indicators of the quality of mortality rates, access to pharmacists, base pneumonia and slave fraction

3A: Slave fraction against base pneumonia **3B: Slave fraction against pharmacists**



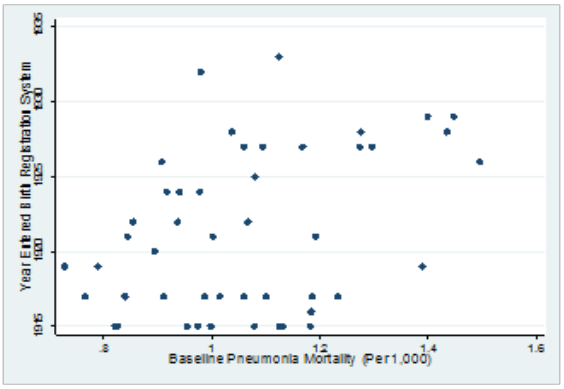
R=0.43 R=-0.38

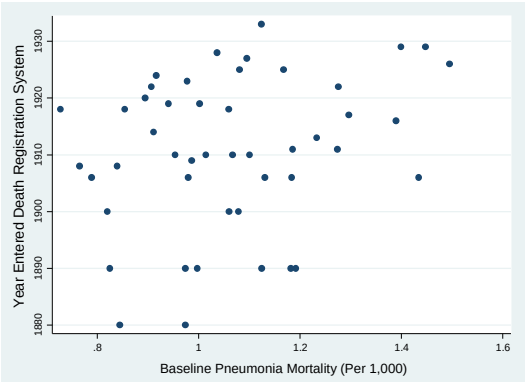
3C & 3D: Correlation of years of birth and death registration with slave fraction



R = 0.45 R=0.49

3E& 3F: Correlation of years of birth and death registration with base pneumonia

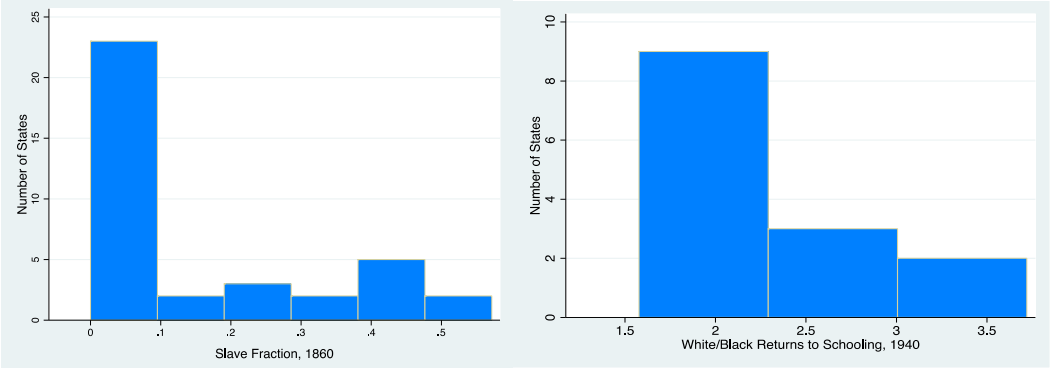




R = 0.39 R=0.26

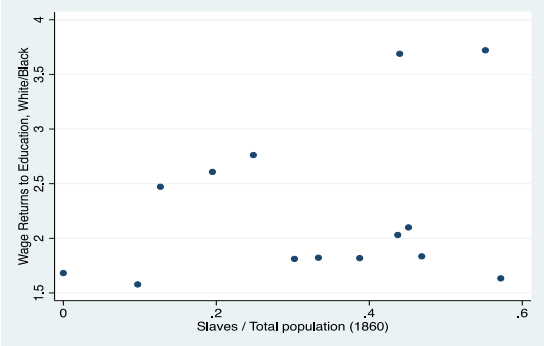
Notes: These figures explore the relationship of the 1860 slave fraction variable, which we use as a proxy for institutional segregation, with *base_pneumonia (A),* the density of pharmacists in black-residential counties *(B)*, and the two indicators of the quality of Vital Statistics registration- the years of entry to the birth and death registration systems respectively *(C and D*). Panels *E and F* show a positive but dispersed relationship between the quality of mortality data in this era and pre-intervention pneumonia mortality rates.

Figure 4 – Density plots for indices of segregation



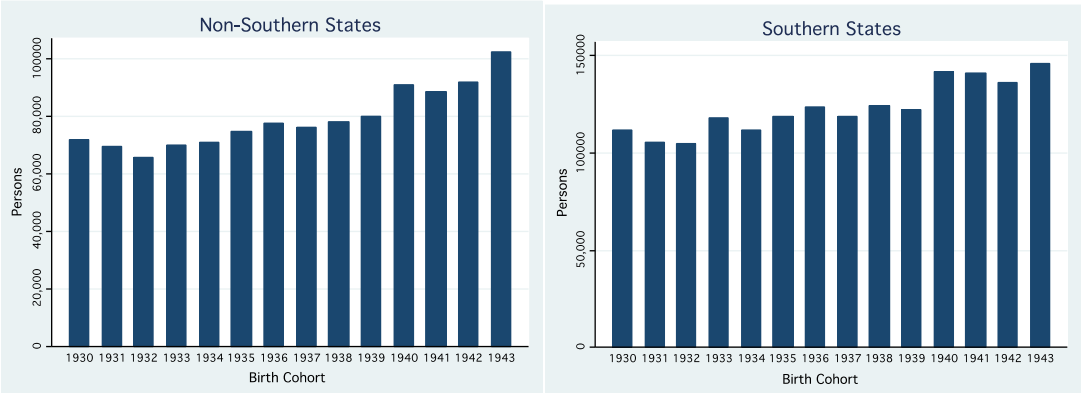
Notes: Histogram plots of the 1860 slave share and 1940 white to black Mincerian income returns to schooling ratio. Using state averages: Mean (sd, range): slave fraction: 0.133 (0.192, [0, 0.57]), white/black returns: 2.55 (1.35, [1.58, 6.73]). In the microdata sample used in the estimation, the median slave fraction was 0.38 and mean 0.33. The percentiles were as follows: 10^{th}=0, 25^{th} =0.25, 50^{th}= 0.39, 75^{th} =0.45, 90^{th}= 0.55. In the state sample, the overall median is 0.04, but conditioning out the zeros, it is 0.24.

Figure 5: Correlation of slave share and black-white ratio of returns to education in the Southern states



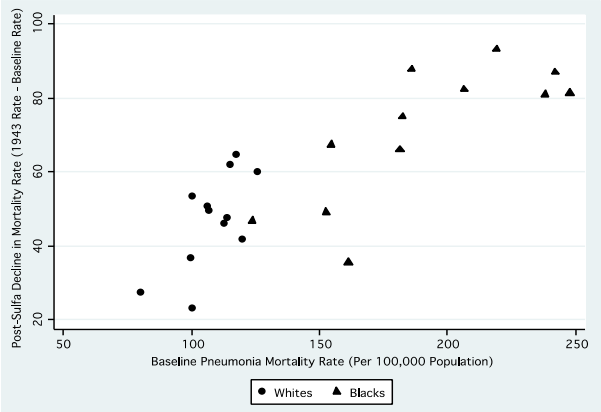
Notes: Scatter plot of slave share in 1860 against race ratio of income returns to education for adults observed in the 1940 census ; see *Appendix 1*.

Figure 6 – Age heaping among Blacks in Southern vs non-Southern states



Notes: These are plots of cohort size by birth cohort in the 1980 census sample for blacks. For a given enumeration date, heaping in birth year is mirrored in heaping in age. There is no evidence of age heaping in either region and, in particular, no evidence of greater age heaping in the Southern states.

Figure 7 – Race Specific Convergence: Southern States Only



Notes: The triangles indicate black mortality rates and the circles white rates. Each triangle or circle is a state in the South. Race-specific mortality data were provided by Adrian Lleras-Muney.

Table 1: Trend Breaks in Pneumonia and Influenza Mortality Rates- by Race, Age and Region, in Levels and Logarithms

	Levels		Logs		Levels		Logs	
	All-age Mortality Rate (per 1000 inhabitants)	**Infant Mortality x 1000 Births**	**All-age Mortality Rate (per 1000 inhabitants)**	**Infant Mortality x 1000 Births**	**All-age Mortality Rate (per 1000 inhabitants)**	**Infant Mortality x 1000 Births**	**All-age Mortality Rate (per 1000 inhabitants)**	**Infant Mortality x 1000 Births**
	Black-South				**Black-North**			
Post	-0.158**	-1.128**	-0.072*	-0.100**	-0.606	-10.285**	-0.297	-0.449**
	(0.063)	(0.413)	(0.038)	(0.043)	(0.356)	(4.036)	(0.175)	(0.151)
Year	0.065***	0.359**	0.039***	0.036**	0.143*	2.436***	0.071*	0.111***
	(0.014)	(0.145)	(0.008)	(0.013)	(0.073)	(0.808)	(0.035)	(0.030)
PostxYear	-0.200***	-0.601***	-0.136***	-0.054***	-0.239***	-3.441***	-0.140***	-0.174***
	(0.016)	(0.183)	(0.011)	(0.014)	(0.079)	(0.836)	(0.040)	(0.032)
	White-South				**White-North**			
Post	-0.216***	-0.607**	-0.176***	-0.098*	-0.422***	-0.285	-0.385**	0.033
	(0.035)	(0.274)	(0.029)	(0.050)	(0.138)	(0.700)	(0.142)	(0.127)
Year	0.055***	0.262**	0.047***	0.050*	0.068**	0.134	0.061***	0.021
	(0.013)	(0.098)	(0.011)	(0.024)	(0.023)	(0.198)	(0.020)	(0.030)
PostxYear	-0.142***	-0.549***	-0.155***	-0.097***	-0.121***	-0.652***	-0.143***	-0.134***
	(0.017)	(0.137)	(0.016)	(0.028)	(0.030)	(0.215)	(0.036)	(0.039)

Notes: *** - p<0.01, ** - p<0.05, * - p<0.1. Each column represents a separate regression. All models include state fixed effects and weight by state X race population. And all standard errors are corrected for clustering at the state level. All models weight by state X race population. Sample includes yearly observations between 1930-1943. The sample size for the South is 188 state*years and for North is 20. See *Appendix 4* text for details.

Table 2: Slavery, Education, Income in the South vs Non-South: Correlations

	South	Non-South
Ratio of Years of Schooling	0.72	0.94
	(0.09)	(0.13)
Ratio of Earned Income	0.88	0.95
	(0.04)	(0.07)
Ratio of Returns to Schooling	0.44	0.58
	(0.14)	(-)
Slave Fraction in 1860	0.00002	0.31
	(0.0001)	(0.18)

Notes: With the exception of slave fraction, we computed state level estimates of black-white ratios for each of the variables shown above using 1940 Census microdata. The slave fraction data come from Nunn (2008), who originally gleaned these from 1960 Census microdata. The means across states are provided in each cell, with the standard deviation in parentheses. There are 17 states in the South and 31 states in the North. For the returns to schooling variable, we have data for 14 Southern states and 1 Non-Southern State, since cell sizes in the 1940 census for states outside of this sample were quite small. The states for which we have data for this variable overlap almost exactly the states for which we have race-specific mortality data (see *Appendix 1*). Slave fraction data is available for 21 Non-South and 16 Southern states.

Table 3: Correlations Of Indicators of Segregation

	Slave Fraction in 1860	White Black Returns to Schooling	White Black Schooling Ratio	White Black Income Ratio
Slave Fraction in 1860	1			
White Black Returns to Schooling	0.26	1		
White Black Schooling Ratio	0.87	0.25	1	
White Black Income Ratio	0.79	0.12	0.83	1

Notes: Correlation matrix for 15 states for which we have each of the variables displayed above. See *Appendix 1* for details on sources. Ratios expressed as White/Black so as to define all variables in terms of white relative advantage.

Table 4: Segregation-Gradients in Impacts of Infant Pneumonia on Adult Outcomes: White Men

	Schooling	High School	College	Employment	Family Income	Poverty	Work Disability	Cognitive Disability	Physical Disability
Panel A - North/South									
post*base	0.412***	0.0343	0.0832***	-0.002	0.0524*	-0.0297*	-0.00691	-0.0215	-0.00852
	(0.122)	(0.022)	(0.018)	(0.009)	(0.031)	(0.015)	(0.0104)	(0.0132)	(0.0253)
post*base *south	0.338	0.0883**	-0.0166	0.060**	0.0549	0.0146	-0.01613	-0.0184	0.00703
	(0.231)	(0.036)	(0.043)	(0.028)	(0.044)	(0.025)	(0.0204)	(0.0280)	(0.0463)
post*south	-0.337	-0.101**	0.0173	-0.063*	-0.0759	-0.0131	0.01233	0.0230	-0.0212
	(0.294)	(0.043)	(0.053)	(0.033)	(0.052)	(0.030)	(0.0254)	(0.0337)	(0.0554)
Panel B - Slave Fraction									
post*base	0.532***	0.0628***	0.0885***	0.007	0.0613**	-0.0287**	-0.01501	-0.0283**	-0.0197
	(0.113)	(0.018)	(0.014)	(0.008)	(0.026)	(0.012)	(0.0126)	(0.0128)	(0.0257)
post*base*slave	0.437	0.0883	-0.0936	0.128***	0.0295	0.066	-0.01804	-0.00721	-0.0134
	(0.484)	(0.064)	(0.085)	(0.038)	(0.090)	(0.043)	(0.0454)	(0.0472)	(0.0995)
post*slave	-0.363	-0.119	0.0797	-0.100*	-0.0528	-0.0735	0.00599	-0.0340	-0.0476
	(0.556)	(0.077)	(0.089)	(0.050)	(0.112)	(0.059)	(0.053)	(0.0576)	(0.119)
Returns									
post*base	0.503***	0.0345**	0.0538***	0.033***	0.0792***	-0.0143	-0.04679	-0.0521***	-0.000121
	(0.167)	(0.015)	(0.015)	(0.010)	(0.013)	(0.011)	(0.0068)	(0.00545)	(0.0358)
post*base *returns	0.311	0.0947***	0.0366	0.009	-0.0246	0.0256	0.0247	0.0674***	-0.0354
	(0.217)	(0.022)	(0.034)	(0.012)	(0.019)	(0.033)	(0.0181)	(0.00839)	(0.0457)
post*returns	-0.331	-0.106***	-0.0323	-0.021	0.0149	-0.0234	-0.0299	-0.0615***	0.0427
	(0.240)	(0.023)	(0.036)	(0.013)	(0.020)	(0.034)	(0.0189)	(0.00922)	(0.0511)
N	636991	636991	636991	1835771	1802548	1835771	1,835,771	573,071	573,071
N (slave)	601797	601797	601797	1774954	1746544	1774954	1,774,954	564,026	564,026
N (returns)	207156	207156	207156	588546	577185	588546	588,546	181,893	181,893

Notes: *** = p<0.01, ** = p<0.05, * = p<0.1. Robust standard errors adjusted for clustering at the birth state level are in parentheses. Each panel X column contains the results from a separate regression. The dependent variable for each regression is provided at the top of the table. Models and included controls are identical to those in *Table 4* of the main text except are estimated for the white male sample.

Table 5: Testing for Endogenous South-North Migration of Parents

	(1)	(2)
Full Sample	-0.051**	-0.001
	(0.033)	(0.011)
Some High School and Beyond	-0.076**	0.004
	(0.046)	(0.007)

Notes: Probit regressions of the probability of moving Northward between 1935 and 1940 among those of black individuals of child bearing age living in Southern states in 1935 (see *Appendix 1* for definitions of North and South) as a function of *post*base_pneumonia*. Controls in column 1 include age and sex as well as state and year FE. In column 2 we additionally add state per capita income, under-2 diarrheal mortality, and heart disease mortality. Each cell represents a probit marginal effect from a separate regression. For the subgroup analysis, we chose "Some High School and Beyond" as it represents the median of the black schooling distribution. However, similar results obtain if we use high school completion and above. The results in column 1 suggest that blacks living in high pneumonia were *less* likely to migrate after the arrival of sulfa drugs in 1937. However, this association is completely purged with the addition of the other state level controls, in particular state per capita income.

Table 6: Accounting for Survival Selection- Black Men

Baseline Sample	**Income**	**Educ**	**College**	**High School**	**Employment**	**Poverty**	**Work Disability**	**Cognitive Disability**	**Physical disability**
post*base	0.309**	0.0978	0.145	0.194**	0.108*	-0.0866**	-0.358*	-0.120*	-0.110
	(0.128)	(0.469)	(0.106)	(0.0776)	(0.0597)	(0.0416)	(0.178)	(0.0646)	(0.102)
post*base*slave	-0.679**	-0.374	-0.327	-0.270*	-0.258**	0.172*	0.652*	0.0695	0.0795
	(0.284)	(1.054)	(0.242)	(0.152)	(0.119)	(0.0907)	(0.376)	(0.141)	(0.256)
N	170,844	66,969	66,969	66,969	170,844	170,844	170,844	49,911	49,911
Selection Adjusted Sample	**Income**	**Educ**	**College**	**High School**	**Employment**	**Poverty**	**Work Disability**	**Cognitive Disability**	**Physical disability**
post*base	0.305**	0.0541	0.142	0.188**	0.107*	-0.0838**	-0.358*	-0.118*	-0.110
	(0.128)	(0.487)	(0.107)	(0.0788)	(0.0599)	(0.0409)	(0.179)	(0.0648)	(0.102)
post*base*slave	-0.677**	-0.283	-0.321	-0.259	-0.256**	0.169*	0.647*	0.0634	0.0818
	(0.286)	(1.086)	(0.244)	(0.155)	(0.119)	(0.0894)	(0.376)	(0.142)	(0.256)
N	170,920	67,045	67,045	67,045	170,920	170,920	170,920	49,928	49,928

Notes: To account for the fact that pneumonia mortality rates were higher for black men, and even higher for black men in the Southern, more segregated states, we adjusted our estimates for survival selection as follows. First, we computed the number of individuals prior to 1937 who would have been "saved" if sulfa drugs were available starting in 1930 using differences in race-specific pneumonia mortality rates for each state. We then added observations to our estimation sample reflecting these "lost" men, assigning the worst possible outcomes (log income of 0, no high school or college, etc), so as to attempt the most severe check on selection possible. We then re-estimate the specifications shown in Table 4. The mean mortality rate from pneumonia and influenza for our sample cohorts was 11.2/1000 live births (about 1 percent).