The Long Run Economic Effects of Medical Innovation and the Role of Opportunities

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Abstract

We leverage the introduction of the first antibiotic therapies in 1937 to examine the long-run effects of early-childhood pneumonia on adult educational attainment, employment, income, and work-related disability. Using census data, we document large average improvements across all outcomes, alongside substantial heterogeneity by gender and race. Among women, health gains led to changes in marriage and fertility that partially offset their labor market improvements. Among Black Americans, we uncover a pronounced gradient linked to systemic racial discrimination in the pre–Civil Rights era: individuals born in more discriminatory Jim Crow states realized much smaller gains than those born in less discriminatory states, despite larger reductions in pneumonia exposure. There is no similar gradient among white Americans. Together, these findings highlight the central role of institutional environments in shaping whether investments in early-life health translate into long-run socioeconomic gains.

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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 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 physician Sir William Osler to coin pneumonia as the "Captain of the Men of Death."[1] Pneumonia remains the leading cause of child death worldwide, killing 700,000 children every year—more than AIDS, malaria, and tuberculosis combined. Despite this, only one third of children who have pneumonia today are able to access antibiotic therapy (World Health Organization, 2022).

In Part I of this paper, we investigate the extent to which pneumonia in early childhood inhibited human capital accumulation and economic mobility by exploiting 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). We leverage the fact that by far the largest reductions were among infants and young children. We further leverage the fact that states most burdened by pneumonia in the pre-sulfa era experienced the largest declines upon the introduction of sulfa drugs. Both facts are documented in Section 1. Our strategy essentially investigates whether the post-sulfa convergence in birth year levels of pneumonia mortality across the states after 1937 is mirrored in longer run economic 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 childhood pneumonia may have such a long reach is motivated by insights from the biomedical literature. Infections produce inflammation and result in a diversion of nutritional resources away from organ and immune and inflammatory system development toward fighting infection and protecting survival (Gluckman and Hanson, 2004b; Crimmins and Finch, 2006). Reducing infections can thus directly impact neurocognitive and physical developmental pathways, leading to compromised brain development and greater vulnerability to chronic diseases later in life. Infection in infancy is (i) more likely to occur because infants have nascent immunity, and (ii) more likely to cause longer term damage because of the greater plasticity of development at this young age, and because infancy is a resource-intensive period of rapid physical

[1] This paper substantially revises an earlier draft, circulated under the title "Shadows of the Captain of the Men of Death: Early Life Health Interventions, Human Capital Investments, and Institutions" (Bhalotra and Venkataramani, 2011), revised as (Bhalotra and Venkataramani, 2015).

and mental growth. For instance, the brain doubles in size in the first year of life (Gilmore et al., 2018), and brain growth is estimated to consume 85% of calorie intake in infancy (Finch and Crimmins, 2004). It is therefore plausible that severe or repeated infections generate permanent physiological changes.[2] These are biological mechanisms by which early childhood infections can impact the outcomes we investigate—educational attainment, employment, income and disability.

A contribution of this paper is to identify these long run impacts for pneumonia infections using a quasi-experimental approach and large-scale microdata (that medical studies seldom use). Our second core contribution is to highlight heterogeneity in the size of the impact on future outcomes, demonstrating that it depends upon economic opportunities, including access to quality education and jobs.

In particular, we estimate heterogeneity in impacts of the availability of the new antibiotics on adult economic outcomes by gender and race. To investigate gender differences in the results we allow heterogeneity by baseline gender gaps in indices of economic opportunity, and we estimate how family formation (marriage and fertility) responded to the advent of sulfa. To investigate race differences, we estimate gradients in treatment effects using two continuous measures of birth-state variation in the intensity of systemic discrimination against Black Americans under the Jim Crow laws that made discrimination de jure in the South, while it was de facto in the North. We use two related measures. The first is the share of the 1860 population that was enslaved (Nunn, 2008), which several studies have shown is predictive of racial gaps in outcomes decades later[3] and, related, the number of discriminatory Jim Crow laws, as classified by Althoff and Reichardt (2024). In the rest of this section, we summarize our findings and discuss their robustness to potential threats to identification. We then delineate the contributions of this paper relative to the existing literature.

Using microdata on the entire population in successive census files, we find that antibiotic-driven declines in pneumonia in infancy led to increases in schooling, family income, and employment in adulthood, and reductions in work-limiting disability. A back of the envelope calculation indicates that increases in schooling accounted for half of the observed increase in income. Investigating the income result further, we find a decrease in the probability of being poor, as well as an increase in the probability of being in the

[2] On cognition, the release of inflammatory molecules during infections may also directly impact the developing brain by changing the expression of genes involved in the development of neurons and the connections between them (Deverman and Patterson, 2009).

[3] See Engerman and Sokoloff (2005); Mariscal and Sokoloff (2000); Bertocchi and Dimico (2010); Sacerdote (2005); Mitchener and McLean (2003); Acharya et al. (2016); Althoff and Reichardt (2024); McMillon (2024).

top quartile of the income distribution. This is consistent with the documented increase in schooling, and a striking depiction of economic mobility triggered by improved infant health given that pneumonia is more prevalent among the poor (Britten, 1942). On average, the increase in schooling is driven by high school rather than college completion, consistent with this being a time in history when high school completion was a relevant margin (Goldin, 1998). The average estimates are comparable in magnitude to the effects of childhood medical insurance coverage on these same adulthood outcomes (Goodman-Bacon, 2021a; Cohodes et al., 2016).

Event study estimates reveal structural breaks in trend in 1937, in line with the arrival of sulfa drugs and consistent with the largest returns emerging from exposure to antibiotics in the first year of life.[4] As we have a difference-in-differences model with a continuous treatment (exposure to pneumonia), what we estimate is the average causal response (ACR), the causal response being the change in unit's potential outcome with a marginal increase in the dose (Angrist and Imbens, 1995). For comparisons between adjacent dose groups to identify the ACR, we need a 'strong parallel trends assumption', which is that the path of outcomes for lower dose units must reflect how higher dose units outcomes would have changed had they instead experienced the lower dose. This is a stronger assumption than the 'standard' parallel trends assumption but, if the data support it and we see a dose-response relationship this strengthens causal interpretation in the continuous DiD relative to the binary model (Callaway et al., 2025). To investigate this, we provide event study estimates by decile of treatment intensity. While these estimates are, naturally, noisier, they reveal a dose-response relationship.[5]

To further strengthen a causal interpretation of our results, we exploit another source of continuous variation—variation in access to sulfa drugs after they were introduced. On the premise that pharmacists were the key agent of diffusion of the new therapy (Lesch, 2007), we show that treatment effects are larger in states where the share of pharmacists per capita was higher. In sections 2.2 and 3.2 we identify and address threats to identification. We investigate—and undermine—the concern that what we capture is an underlying process of convergence across states in health and economic indicators. We consider other competing

[4] This is in line with the epidemiology of pneumonia, with infection rates higher in infancy than at any later age (Britten, 1942) (see Figure 1a), and with the greater sensitivity and malleability of human development at this critical age (Barker and Osmond, 1986).

[5] We nevertheless also estimate a model using a binary (above/below median) definition of exposure which allows us to estimate an average treatment effect on the treated (ATT). Following (Callaway et al., 2025), we compare the aggregation weights implicit in the two-way fixed effects model with the actual frequency of treatment exposures and find that they are broadly similar, but we nevertheless re-estimate the model re-weighting observations such that states are representative of their dose frequency and our findings are robust to this.

explanations of our findings, including selection on the basis of mortality, fertility, and migration, mean reversion, and confounding from historical events including World War II, the Depression, the New Deal and the Dust Bowl. We also discuss and tackle measurement error in pneumonia mortality.

Estimating the baseline model by gender and race, we see some meaningful differences. For instance, white women exhibit smaller increases in schooling and income than white men, consistent with barriers to women's participation that lowered their returns to schooling in this era (Goldin, 1991). Black men show larger increases in income and employment than white men, in line with Black children being two to three times as likely as white children to suffer pneumonia. However, on average, they show no increase in schooling, so the increases in income and employment are consistent with health being human capital. On average, black women show no education or labor market gains from sulfa exposure.[6]

In Part II of the paper, we investigate how systemic racial discrimination modified impacts of infant sulfa exposure for Black Americans. We find a sharp gradient for all outcomes, with treatment effects diminishing in the index of discrimination. In the Northern states, where institutionalized discrimination was weaker, Black Americans reaped substantial future economic gains from reduced pneumonia in infancy, indeed, they reaped higher gains than white Americans, consistent with their higher baseline pneumonia. However, these effects are eroded as one moves along a continuous measure of systemic discrimination. Schooling, employment, earnings and disability gains are diminished, and the estimated contribution of schooling to income gains is diminished. These gradients are evident for Black men and women, and they confirm that women did benefit from sulfa in the least discriminatory states.[7] The said gradients emerge for each of two measures of discrimination. Our estimates are consistent with either or both of two mechanisms potentially at play. First, systemic discrimination can directly reduce the income returns to improved early life physical and neurocognitive development by limiting available opportunities in labor markets (employment). Second,

[6]In fact they are less likely to pursue school or employment. To investigate this further, we modeled marriage and fertility as outcomes for women. We find that sulfa exposure results in black women having more children, while it leads to white women having fewer children. Black and white women are more likely to be married, particularly Black women. To study the relevance of family formation to the labor market outcomes, we stratify the sample by marriage and fertility. We find that the null and negative results for Black women are driven entirely by those who are married or have high fertility (more than 1 child). A broadly similar pattern for white women confirms that family formation in this era limited women's economic opportunities, muting impacts of improved infant health. As discussed below, the negative returns for Black women emerge entirely from discriminatory states—and this is mirrored in larger increases in fertility in these states. In an environment where access to quality schools was restricted, it seems that the returns to improved fecundity capital from the sulfa-driven increase in the health endowment (Lucas, 2010) relative to the returns from human capital were higher (Low, 2024), leading Black women to pull away from the labor market and have more children.

[7]For instance, in states in the bottom (top) decile of the number of Jim Crow Laws, income among Black men was 9.3% (3.1%) higher on account of sulfa exposure and the corresponding figures for Black women were 1.7% (-2.1%).

it can discourage investments in human capital investments (schooling), which reinforce any direct impacts on employment and earnings.

We show estimates of a similar specification for White men and women. The coefficient on the term interacting the sulfa treatment variable with the proxy for discrimination is, in general, numerically small and statistically insignificant. This "placebo" eliminates some of the potential concerns with our interpretation of the gradients. We investigate and reject a number of other concerns. Importantly, we demonstrate that Blacks systemic discrimination was not associated with reduced access to sulfa drugs. We also investigate and set aside potential concerns over measurement error, selection and growth in the supply of schools in the South.

Pulling together the estimates across the paper, the evidence suggests the following. First, the introduction of sulfa drugs that treat pneumonia, an infection that was highly prevalent among infants, led to a significant reduction in the duration and intensity of pneumonia infections and, as a result, an improvement in the health and cognitive endowment of infant children. We show that these improvements were fairly universal, and in fact larger in absolute terms among Black Americans. Second, exposure to sulfa drugs in infancy had a positive impact on employment and earnings even in states and in demographic groups that experienced no increase in schooling, confirming the role of health as human capital. Third, the improved endowments triggered responsive reinforcing investments in education. Fourth, crude simulations suggest that, where systemic discrimination was absent or small, increases in schooling explained most of the increase in income; indeed this was an era when the returns to schooling were large (Goldin, 1998). These results are consistent with dynamic complementarity, and our results on income across the distribution reinforce this. They reveal how the antibiotic-driven improvement in infant endowments was able to move individuals up the skill ladder. Fifth, we find that this was conditional on (equal) opportunities—we find that systemic discrimination prevented Southern Black Americans 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).

Contributions in relation to related research

The literature on antibiotic innovation and pneumonia Medical scientists have documented the revolutionary impacts of antibiotic innovation on the intensity and duration of bacterial infections, referring to it as a miracle life saving drug (Lesch, 2007). Using state-time series data, (Jayachandran et al., 2010) estimate that sulfa drugs reduced all-age mortality by 2 to 3 percent, confirming the contribution of modern medicine to mortality decline in the early twentieth century.

We provide the first estimates of the long run impact of antibiotic innovation on economic outcomes. Equally, we provide the first estimates of the impact of contracting pneumonia in early childhood on educational attainment, labor market outcomes and work-related disability.[8] Our findings are important today for two reasons, the first relating to the continuing scourge of pneumonia and the second, to the challenges of antibiotic resistance (globally) and antibiotic availability (in poorer countries).

Globally, about 1 in 71 children gets pneumonia each year.[9] While policymakers are actively tracking *mortality* rates, they completely miss the scars that *survivors* of infections carry. Demonstrating, as we do, the dynamic economic gains from antibiotic availability stands to influence global health priorities by dramatically increasing the benefit-cost ratio through accounting for dynamic long run impacts on future outcomes beyond the domain of health.[10] Our results are also pertinent to investment in research and development necessitated by antibiotic resistance (Cilloniz et al., 2024), and to contemporary debates concerning the pricing and distribution of available antibiotics (Hollis and Pogge, 2008; Bhalotra and Pogge, 2014).[11]

[8] In a study conducted after circulation of our original working paper on this topic (Bhalotra and Venkataramani, 2011), which she cites in her work, Lazuka (2020) showed improvements in labor income after early life exposure to sulfa drugs in Sweden. Our study, set in the US, remains distinct in that we explore a broader range of outcomes and heterogeneity in the effects of sulfa drugs on these outcomes driven by inequality in opportunity.

[9] According to the 2023 Global Burden of Disease report, 2.5 million people died of pneumonia in that year, of whom about a fourth were children under age 5. This is disproportionate given that about 10% of the world's population is under age 5. Although it is more common and deadly in low income countries, pneumonia is, overall, the eighth leading cause of death in the US (Kung et al., 2008), imposing a substantial financial burden on US healthcare, with lifetime costs estimated at 44.8 billion USD. Health experts noted a 'tripledemic' of respiratory viruses (flu, COVID-19, and RSV) contributing to surges in pneumonia in the United States in 2024, with a particular surge among children.

[10] An example of a global health initiative that could benefit from our estimates is the Advanced Market Commitment (AMC), an innovative financing mechanism that accelerates global roll-out of the pneumococcal vaccine (Kremer et al., 2020) (http://www.gavi.org/support/nvs/pneumococcal/), or SECURE, an initiative to expand access to essential antibiotics to support countries in addressing the silent pandemic of drug-resistant bacterial infections, see https://www.who.int/initiatives/secure-expanding-sustainable-access-to-antibiotics.

[11] Most children exposed to pneumonia live in poorer countries, where it is estimated that only one in three children who need antibiotics get them, see WHO 2022. at https://www.who.int/news-room/fact-sheets/detail/pneumonia The reasons include weak supply chains to remote rural areas, and prices that are higher than the poor can afford, often because of WTO regulation allowing medical patents (Otaigbe, 2025; Pogge et al., 2010; Bennett and Yin, 2019).

The literature on future economic benefits to investments in early life health There is a growing literature investigating long run economics gains to interventions that improve early life health.[12] We make two contributions to the literature. First, we provide results for pneumonia and equally, for the first antibiotics. Second, in a sharp departure from most existing work, we demonstrate the importance of economic opportunities later in the life-course—in particular equal opportunities for schooling and jobs—in realization of the full potential of a healthy start. The existing literature documenting future earnings gains from interventions that improve childhood health has tended to bundle the impacts of initial improvements in the biological endowment with the impacts of subsequent (endogenous) investments (Almond and Mazumder, 2013). We are able to disentangle these by leveraging two independent sources of exogenous variation, one that creates variation in the early life endowment (antibiotic innovation that produced a treatment for the most prevalent childhood infection), and another that generates variation in subsequent investments (indicators of systemic discrimination).

The literature on race discrimination in the pre-Civil rights era A considerable literature has demonstrated that slavery has had lasting impacts on Black Americans,[13] and that restricted access to quality schools and quality jobs in the pre-Civil Rights era in the South translated to lower returns and lower investment in human capital.[14] We show that unequal opportunities encapsulated in historical slavery and the force of the Jim Crow Laws can explain a large part of the inequality in economic benefits from reduced pneumonia infections (antibiotic access) in infancy. In doing this, we add evidence of a new channel by which contemporary Black Americans carry the legacies of systemic racial discrimination.

The rest of the paper evolves as follows. Part I provides estimates of the impact of sulfa innovation on future economic outcomes of individual exposed in early childhood. It first profiles pneumonia mortality and morbidity in the 1930 United States, marking changes in infant pneumonia created by the sulfa drug revolution. It then describes the data and research strategy. This is followed by estimates for the full population

[12] This includes, among others, studies that have investigated prevention of hookworm, typhoid, malaria or diarrhea (Bleakley, 2007; Beach et al., 2016; Cutler et al., 2010; Lucas, 2010; Venkataramani, 2012; Bhalotra and Venkataramani, 2013), studies of anti-parasitic medications (Baird et al., 2016; Croke and Atun, 2019), postnatal support programs including advice on nutrition and sanitation (Bhalotra et al., 2017, 2022), salt iodization (Adhvaryu et al., 2020), childhood vaccination (Atwood, 2022), and childhood medical insurance (Goodman-Bacon, 2021b). A broader review of studies examining the long run consequences of childhood interventions is in (Almond et al., 2018).

[13] See Engerman and Sokoloff (2005); Mariscal and Sokoloff (2000); Bertocchi and Dimico (2010); Sacerdote (2005); Mitchener and McLean (2003); Acharya et al. (2016); Althoff and Reichardt (2024); McMillon (2024).

[14] See Donohue and Heckman (1991); Card and Krueger (1993); Johnson (2011); Aaronson and Mazumder (2011); Thompson (2014); Bohren et al. (2022); McMillon (2024).

sample, and a suite of robustness checks. Part I concludes with a discussion of estimates by race and gender, which are subject to additional robustness checks. Part II examines gradients in the long-run economic impacts of the sulfa revolution by indicators for institutionalized racial discrimination. It then investigates alternative interpretations of the striking heterogeneity in impacts among Black men and women. The final section concludes.

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

1 Pneumonia and the Impacts of sulfa Drug Revolution

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 those 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 30 per 1,000 infants, nearly twice as high as for 1-4 year olds and 10 times larger than for 10-14 year olds (see Figure 1a; Britten (1942)).

Pneumonia was also 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). 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 14 and 38 pneumonia cases per 100 children under the age of 4 each year (McAllister et al., 2019). 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).[15]

[15] 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

Prior to the discovery of antibiotics, 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 drug era, it is 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.[16] 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 that the largest decline, nearly 30%, accrued to infants, consistent with their higher infection rates (Figure 2b). The trend break in the infant pneumonia rate is statistically significant (Appendix B, Table B1-B2). We also demonstrate that larger absolute reductions in pneumonia mortality were seen in states with

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 those associated with health campaigns, clean water programs or the distribution of medicines. Also, pregnant women were particularly vulnerable to influenza and Almond (2006); Schwandt (2018) establish 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.

[16] 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 (Podolsky, 2006). 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).

higher pre-sulfa drug era disease burdens (Figure 3). This pattern is evident for all age groups (Figure 3a) and, again, is stronger among infants (Figure 3b). We leverage 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 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 without prescription from pharmacists and were also extensively used by community physicians (Lesch, 2007; Lerner, 1991).

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 (1976) model (Cunha and Heckman, 2007; Heckman, 2007; Cunha et al., 2010). Here, "endowment" refers to the stock of developmental capital in infancy, and it is modifiable.

While there is no wide-spread data on individual-level access to sulfa drugs, the rich historical record points to a shock that was both abrupt and geographically diffuse. Contemporary trade journals and popular magazines advertised sulfa drugs aggressively; pharmacists could dispense them without prescription; and, at roughly one per cent of mean Black American annual income, the cost was low for a life-saving therapy (Lesch, 2007; Smith and Welch, 1989). Mirroring these accounts, structural break tests applied to state-level mortality series replicate the finding of Jayachandran et al. (2009): every state—Northern and Southern, rural and urban—exhibits a discrete drop in deaths from sulfa-treatable diseases in 1937 or 1938. This is visually striking even in descriptive trends of raw pneumonia and influenza mortality by state and year (Figure B1) with parallel timing for Black and White populations (Figure B2). A broader discussion of evidence demonstrating the reach of drugs to the Black population is available in Appendix B.1. These facts justify our empirical strategy, which treats 1937 as a nation-wide "switch-on" date and leverages cross-state

differences in baseline pneumonia risk, rather than differences in the precise year of adoption, to identify the first-stage impact of the innovation.

To gauge how thoroughly sulfa drugs penetrated different parts of society, we explore two complementary pieces of evidence. Full technical details are discussed in Appendix B.2 and B.3 respectively. First, we compare the timing of the 1937 trend break across quartiles of six state characteristics that, based on studies of the diffusion of medical innovation (Jayachandran et al., 2010; Glied and Lleras-Muney, 2008), plausibly mediate access to pharmaceutical therapies. These are income, urbanization, literacy, non-white population share, and the per-capita numbers of physicians and pharmacists. Every quartile of every characteristic shows the same 1937 shift (Figure B3), indicating that initial availability was not meaningfully staggered over time along these dimensions. While we do observe differential magnitude in trend-breaks in mortality (Tables B3-B4)[17], this is likely driven by divergence in baseline mortality rates by these characteristics. For this reason, we examine convergence in pneumonia mortality after 1937, asking whether states with initially high death rates caught up faster when they possessed more of the same diffusion-related attributes. Convergence proves broadly uniform across four of the six variables; only pharmacist and physician density accelerates the post-sulfa decline (Table B5, Figure B4). That pattern squares with historical accounts of pharmacies as the primary retail outlet for early sulfa sales (Lesch, 2007), while the absence of systematic gradients on income, education, or urbanization underscores the technology's unusually broad and rapid diffusion through the population.

2 Data and Research Strategy

2.1 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 leverage 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 a continuous differences-in-difference design similar to other work in health

[17] Over time these differences appear to be relatively minor. By year 3 post-sulfa marginal effects presented at the foot of Table B3 suggest a decline of between 42% and 61% in mortality when comparing across percentile 10 to percentile 90 of each mediator, while often declines were far more homogeneous.

and labor economics.[18] 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. We account for concerns related to continuous variation and identification in this type of approach (Callaway et al., 2025), and extend to provide event study models by state and birth cohort, as well as documenting dose responses to exposure.

The data for adult outcomes come from the 1980, 1990, and 2000 5% public use microdata samples of the United States Census (Ruggles et al., 2024). Cohorts born in 1937, the year sulfa drugs became available, were in their early 40s, 50s, and 60s at the time of these enumerations, respectively. We focus on four measures: years of education, log family income, employment, and work-limiting disability. We also consider additional or derived outcomes—high school and college completion, poverty status, cognitive disability, and (for women) marriage and completed fertility—which we present as additional results. Given multiple outcome variables, we correct for multiple hypothesis testing by consistently reporting p-values following Romano and Wolf (2005, 2016), providing strong control of the family-wise error rate. Further discussion of data and variables and all descriptive statistics are in Appendix A.

The baseline specification we estimate is:

$$Y_{istc} = \alpha + \tau \left(\text{Post sulfa}_t \times \text{Base Exposure}_s\right) + \theta_{s,rg} + (\eta_t \times \mu_d)_{rg} + \lambda_{c,rg} + X'_{st}\Gamma + \varepsilon_{istc}. \quad (1)$$

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 sulfa$_t = 1$ for cohorts in their infancy in 1937 and thereafter, and 0 for individuals not exposed to sulfa in infancy. The pre-sulfa pneumonia mortality rate in the birth state is denoted Base Exposure$_s$ and is defined as the average state-specific, all-age combined pneumonia and influenza mortality rate during 1930-1936. Base Exposure$_s$ captures treatment intensity since pneumonia reduction after 1937 was increasing in the base rate (Figure 3).[19] We expect $\tau > 0$ (for desirable outcomes) if adult outcomes mirror this pattern.[20] The Greek letters θ_s, η_t, μ_d and λ_c represent

[18] See, for example, early work such as Card (1992), Acemoglu and Johnson (2007), and Bleakley (2007).

[19] 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 and Lleras-Muney (2009), Acemoglu and Johnson (2007), Bleakley (2007, 2010), and Cutler et al. (2010), among others (see Chuard et al. (2022) of a general review of this approach). 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 1937-1938. See Appendix B, Figure B2.

[20] State-level morbidity data are not available so we follow a tradition of using mortality rates to proxy for disease exposure

fixed effects for birth state, birth year, census division, and census year respectively. In models estimated with pooled samples of both black and white men and women, fixed effects are fully interacted with race and sex dummies, indicated rg above. In later sections, we report group-specific estimates where a single set of fixed effects is included, in which case rg subscripts become redundant.

Birth year fixed-effects, η_t, are estimated separately for each census division, μ_d, implying estimates will not be confounded by any macro-level temporal shocks at the level of census regions. In this way the relevant thought experiment underlying the empirical design is that an individual within a specific birth cohort is compared to an individual within the same birth cohort and census division, but with a differential exposure to sulfa drugs. In baseline models we also control for a vector of relevant birth state and birth year varying observables (X_{st}), which we detail below. Variations in control sequences (including specifications without controls), fixed effects, as well as the incorporation of state-specific linear trends extrapolated from pre-reform periods (Bhuller et al., 2013; Goodman-Bacon, 2021b) are presented as robustness checks.

We cluster standard errors 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-'29 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.

Identifying Assumptions for Baseline Estimates The baseline model laid out in (1) relies on continuous variation in baseline pneumonia mortality rates in a fixed effect setting to measure exposure to sulfa drugs. As documented in Figure 1b, this baseline pneumonia mortality rate varies from figures as low as 80 deaths per 100,000 individuals (Oregon, Washington, California) to values as high as 120-130 deaths per 100,000 (Colorado, Nevada, Arizona). Given that sulfa drugs were first introduced in the US in 1937 and was unavailable for pre-1937 birth cohorts, we can view our setting as a continuous difference-in-differences design, with a single adoption date, or a block treatment design (using the nomenclature of Athey and Imbens (2022)). This single adoption date implies that we need not be concerned with about issues with heterogeneity in treatment effects over time contaminating 2×2 comparisons leading to undesired weights in two-way

(Bozzoli et al., 2009).

fixed effect models, as discussed by de Chaisemartin and D'Haultfœuille (2020), *inter alia*.

However, the use of continuous variation in exposure to sulfa drugs requires care, as identification in a fixed effect setting with continuous treatment exposure relies on a strong parallel trends assumption (Callaway et al., 2025). Specifically, noting that states are exposed to varying levels of baseline pneumonia mortality, our identifying assumption is that had any specific state been exposed to a different baseline pneumonia mortality level, it would have followed parallel trends with groups which had effectively received this dosage. Provided that such an assumption holds, $\widehat{\tau}$ estimated from (1) captures a weighted average of Average Causal Responses (ACR) across the distribution of baseline pneumonia mortality exposure, where the ACR captures the marginal change in outcomes given a marginal change in pneumonia exposure. We provide a full discussion of the required assumptions in Appendix C, and evidence consistent with the identifying assumptions when documenting specification and robustness checks in Section 3.2. We also discuss and document models based on binary definitions of exposure, which avoid this assumption in favor of a traditional parallel trends assumption, allowing us to estimate an ATT. In order to interpret the estimated quantity $\widehat{\tau}$ in terms of exposure to sulfa drugs, we consistently report a scaled effect size considering a representative state moving from the 75$^{\text{th}}$ percentile of baseline pneumonia mortality to the 25$^{\text{th}}$ percentile.[21]

Event-Study Specification Equation (1) defines a single-coefficient difference-in-difference style model, however the cohort-level variation in exposure can be more fully observed by estimating an event-study version of (1). Specifically, we estimate:

$$Y_{istc} = \alpha + \sum_{j=1930}^{1943} \gamma_j \left(1\{\text{Year}_t = j\} \times \text{Base Exposure}_s\right) + \theta_{s,rg} + (\eta_t \times \mu_d)_{rg} + \lambda_{c,rg} + X_{st}'\Gamma + \varepsilon_{istc} \quad (2)$$

subject to $\gamma_{1930} + \cdots + \gamma_{1935} = 0$. This implementation of the event study has been proposed by Miller (2023), where rather than using a single (arbitrarily chosen) omitted baseline period, the entire average of the pre-treatment period is used as baseline.[22] In robustness checks we document a model where a single baseline

[21] This is an inter-quartile range movement, and would imply moving from 118.9 deaths per 100,000 to 89.5 deaths per 100,000 (a decline of 29.4 deaths per 100,000). This scaling is arbitrary, but chosen given that the decline in pneumonia generated by the arrival of sulfa drugs was large. For example, between 1937 and 1938, death rates declined from 116 to 80 per 100,000, a fall of 36 deaths per 100,000 (Figure 2).

[22] As is the case with models omitting a single pre-treatment year, in this specification a single exclusion restriction is imposed implying that all coefficients are identified up to an arbitrary baseline reference category. However this procedure has the benefit of not anchoring all estimates off a single—potentially noisy—baseline period, and additionally providing point estimates and confidence intervals for all pre- and post-treatment coefficients.

coefficient is omitted for comparison, and models where estimated effects are based only on comparisons with individuals who were older at the time sulfa drugs arrived (born in 1933 or before). All other elements of (2) follow those previously described in (1).

The event study specification (2) interacts a full set of pre- and post-treatment year indicators with the baseline pneumonia (Base Exposure$_s$) mortality measure. The logic of standard event study implementations is that inspection of pre-treatment coefficients allows for a consideration of whether treated and control units were at least following similar trends prior to the reform, which may be indicative of them having followed similar trends in the post-reform period (the identifying assumption). Examining the pre-treatment coefficients in equation (2) provides a continuous interpretation of such a test: if pre-reform coefficients are close to zero, this suggests that higher versus lower pneumonia mortality cohorts experienced similar trends in human capital outcomes prior to the arrival of sulfa. However, given the strong parallel trends assumption required here, we can consider this in a more explicit way. Specifically, rather than interacting yearly pre- and post-treatment with a continuous base exposure measure, these yearly indicators can be interacted with a series of saturated indicators for base exposure intensity. In particular, we estimate such models where a series of deciles are used, and states with the lowest 10% pneumonia mortality are omitted as the reference category. In this case, we can inspect for both the parallel trends between all levels in pre-reform periods (a partial test of strong parallel trends), and inspect for any dose response in the post-treatment period.

2.2 Threats to Identification

In this section, we discuss our strategy to account for potential omitted birth state and birth year varying variables. In addition to addressing these concerns, we will also 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 birth state and sample cohorts (including robustness to excluding the war cohorts, excluding states exposed to the Dust Bowl, and removing specific birth cohorts).

Diseases not treatable with sulfa drugs: A potential concern is that Post$_t \times$Base Exposure$_s$ 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 X_{st}). We interact Post_t with pre-sulfa birth state specific mortality rates from diarrhea (under the age of 2), malaria, heart disease, tuberculosis, and cancer and include these as controls.[23]

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 $\text{Post}_t \times \text{Base Exposure}_s$ 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.[24] In contrast, puerperal sepsis accounted for 40% of maternal deaths in 1930, maternal mortality was high, at almost 7 deaths per 1,000 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 $\text{Post}_t \times \text{Base Maternal Mortality}_s$.[25]

State economy and infrastructure: Since our estimates use the cross-state convergence in pneumonia created by the introduction of sulfa drugs (Figure 2), convergent pre-trends across high-base and low-base states prior to 1937 are a concern that we investigate with the event study design (2). 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 X_{st}).[26] Controlling for income per capita in the birth-state is pertinent given early life exposures to shifting economic fortunes during the

[23] 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 et al., 2013), 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.

[24] 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 include controls for post-1937 changes in other sulfa-treatable diseases, and this did not alter the coefficient of interest; see Appendix E, Table E1.

[25] 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-á-vis* men (Jayachandran and Lleras-Muney, 2009). Our estimates test for this by separately conditioning on exposure to maternal mortality declines 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 maternal mortality decline on children because the maternal post-partum infections (puerperal sepsis) that were controlled with sulfa drugs were rarely transmitted to infants.

[26] The estimates are not sensitive to whether we directly include the characteristic (X_{st}) or we include Post-1937 multiplied by the pre-intervention level of the characteristic ($\text{Post}_t \times X_s$) to allow for discontinuous effects of X_{st} that may otherwise load on to the variable of interest.

Depression Era, which has been linked to long-run health and economic outcomes (Duque and Schmitz, 2023). We additionally consider specifications removing the depression years entirely and specifications which control for state-level New Deal spending from Fishback et al. (2003). In addition to testing for robustness to birth state specific time trends ($\theta_s \times \eta_t$) following Bhuller et al. (2013); Goodman-Bacon (2021b), in all baseline models we include census division$\times$birth year fixed effects, motivated in part by convergence in economic development between the US South (which was particularly plagued by pneumonia) and other parts of the country during the 20$^{\text{th}}$ century (Mitchener and McLean, 2003).

We note that including rich sets of control variables may amount to "over-controlling," in addition to presenting potential challenges in estimate average treatment effects due to (unintended) differential weighting of observations (Baker et al., 2025).[27] We therefore assess robustness of the coefficients to sequential addition of controls and display a suite of coefficients, including models without any time-varying controls at all.

3 Results

3.1 Pooled full sample results

Table 1 presents single-coefficient estimates of the long-run effects of early exposure to pneumonia. Each cell reports estimates of the coefficient on $\text{Post}_t \times \text{Base Exposure}_s$ from separate regressions. We find that exposure to sulfa drugs—and thereby reduced exposure to pneumonia in the birth year—led to statistically significant improvements in all outcomes investigated. On average, these results suggest that an interquartile reduction in pneumonia mortality (0.29 deaths per 1,000) was associated with a 0.06 increase in years of schooling; a 1.4% increase in family income; a 0.49% point increase in the likelihood of being employed; and a 0.23% point decrease in reporting a work-limiting disability. We adjust for multiple hypotheses testing following (Romano and Wolf, 2005)). The FWER adjusted p-values in Table 1 confirm that the estimates for schooling and income are significant at the 5% level and for employment and disability at the 10% level.

Estimates for additional outcomes are in Table F1. To assess the margin at which the increase in school-

[27] An example of over-controlling might be as follows: 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.

ing occurs, we modeled high school and college completion (Table F1). We find a robust increase in high school completion (a 2.0 % point from an IQR in pneumonia mortality), with no significant change in college.[28] Using a measure of cognitive disability available in one census year, we find a small and imprecisely estimated decline. We then look more carefully at distributional features of the decline in income. First, we model poverty using the census measure of this, and we find a 0.68% point decline. The results for schooling margin and poverty are consistent with the larger burden of pneumonia falling upon children from poorer families. We further investigated impacts of childhood sulfa exposure on position in the adult income distribution. We do this by assigning reported adult family incomes of post-sulfa cohorts to income percentiles defined on the pre-sulfa income distribution.[29] See Figure F1. Post-sulfa cohorts were significantly less likely to appear in the bottom half of the income distribution, particularly in percentiles 10-25, and they were instead significantly more likely to appear at a percentile above the 70th, especially the 75^{th}-90^{th}, but also the 90^{th}-95^{th} percentiles . Thus availability of treatment for a disease that was more likely to strike the poor not only helped individuals escape poverty later in life (Table F1), but also helped them to ascend to towards the top of the socioeconomic ladder.

Contribution of human capital accumulation to increases in adult income We estimated Mincerian returns for the sample cohorts in the 1980 census, finding 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 around 50% of the sulfa-led increase in family income can be accounted for by increased schooling. This is relevant insofar as the increase in schooling for sulfa-exposed cohorts that had a stronger infant health endowment is a reinforcing investment that contributes to their long run income gain. This becomes moot when we look at differences by race and gender (below), given diminished returns to schooling for black Americans in the South that resulted in systematically smaller income gains from a similar (or larger) improvement in their infant health endowment. A similar argument holds for women, who faced smaller returns to schooling

[28] High school rather than college has been argued to have been the primary driver of America's human capital stock growth during the early 20th century. In discussing rapid growth in high school enrollment, particularly during 1920 to 1935, Goldin (1998) observes that the economy had begun producing large numbers of white-collar jobs that demanded formal education in excess of that provided by primary school but less than that furnished by college. Even some blue-collar occupations demanded the cognitive skills furnished by a high school education, such as the ability to read manuals, interpret blue-prints, use complex formulas, and understand the fundamentals of geometry, chemistry, and electricity.

[29] Specifically, for each census wave, we computed percentiles of the family income distribution for birth cohorts born in 1930-1936 (pre-sulfa). We then assigned the 1937-1943 (post-sulfa) birth cohorts to one of these income percentiles based on reported family income in that census wave. We created a range of binary indicators denoting (mutually exclusive) membership in specific income percentiles. See Appendix A.2 for details.

than men in this era on account of restrictions (such as marriage bars) on their labor market engagement.

Flexible timing of exposure Event studies corresponding to the single-coefficient estimates are presented in Figure 4. The baseline specification models pneumonia exposure during infancy. The event study plots allow the reader to observe the impacts of exposure at other ages.[30] In general, the figures point to the importance of exposure to antibiotics during the first year of life, *i.e.* among cohorts born in 1937 or after. For instance, for education and employment, the coefficient for 1937 is the first coefficient for which the confidence interval lies above the line at zero. For income, the first significant uptick is in 1936, consistent with children's long run outcomes being sensitive to exposure from birth through to age 2. There is no medical reason that a child age thirteen months is less likely than a child age twelve months to experience long run effects from pneumonia exposure. However, it is clear that children who were aged five when sulfa drugs arrived (*i.e.* children born 1932) were robust to pneumonia exposure—these results are in line with the age distribution of pneumonia on the eve of sulfa (shown in Figure 1a), and in line with medical pathways (discussed in Section 1), the key idea being that developmental plasticity early in life can result in nutritional stressors such as infections having irreversible effects.). The patterns in our data cohere with a large literature identifying the early years of childhood as a "critical period" that shapes long-run impacts of exposure to infectious diseases (Almond and Currie, 2011; Almond and Mazumder, 2013; Barker and Osmond, 1986; Duque et al., 2018).[31] The patterns also provide a falsification test, showing no pre-trends in the outcomes.

For income, employment and disability, we use outcome data from the 1980, 1990 and 2000 censuses and not all have quarter of birth recorded. This implies partial contamination of the 1936 birth cohort with members of the (fully-exposed) 1937 birth cohort (see Appendix B). However, as discussed, it is plausible that children who were already a year old when sulfa drugs arrived were still young enough to experience the physiological changes that, in the appropriate environment, improve long run outcomes. Second, when considering education, we have a more precise dating of birth years given that both quarter and year of birth are available in the 1980 census. Third, both because there is no sharp definition of the age at which exposure to pneumonia ceases to matter for future outcomes, and because we do not have the exact date of birth, the design is "fuzzy" around the threshold. To account for this we re-estimate the single coefficient

[30] We do not expect impacts from fetal exposure because mothers of childbearing age experienced very low pneumonia infection rates (Britten, 1942). This contrasts with flu exposure during the 1918 flu pandemic, when expectant mothers experienced particularly high infection rates (Almond, 2006).

[31] The estimates for work-related disability are noisy, making it hard to identify exposure timing. The only clear pattern is that there appears a level-drop in the outcome for post-sulfa birth cohorts.

models dropping birth cohorts 1935–1937 (Table E1). The estimates are robust to this, consistent with the clear shift in education, employment and income between 1934 and 1938 in the event plots. Event studies corresponding to alternative outcomes discussed above are presented in Figure F2. We observe clear improvements in rates of high school completion and corresponding declines in the portion of households below the poverty line. We see no effect on changes in rates of college completion, and a small though imprecise decline in a measure of cognitive disability.

Average causal responses to continuous treatment exposure What we estimate is a continuous difference-in-difference model. If units with different levels of treatability (states with different baseline rates of pneumonia) have systematically different trends, then the standard event study estimates are potentially biased Callaway et al. (2025). The continuous DiD model needs to satisfy the stricter condition of strong parallel trends, see Section 2.1; Appendix C. To investigate this, we estimate the event study by decile of the baseline pneumonia rate distribution, see Figure 5. The plots are noisy as the data are now split into ten sub-groups. Nevertheless there is a dose-response pattern whereby treatment effects are larger in states with higher baseline pneumonia burdens.[32] Any time-varying confounders would have to be correlated with the pre-sulfa pneumonia burden, decile by decile, to generate these patterns.

An additional point of concern, relevant for interpretation of the single-coefficient model is that even if two-way fixed effects estimates a weighted average of dose-specific average causal responses (ACR), doses which are relatively infrequently observed in the data may attract a higher weight than doses that are more frequently observed. Following Callaway et al. (2025) we document in Figure C1 the frequency of observations for each dose observed in the true data (solid line), alongside dose-specific weights generated by the two-way FE estimates (dashed line). Overall, the two weighting schemes are relatively similar. Nevertheless, in Appendix Table C1 we document that our results are robust to two specifications designed to adjust for the concerns raised in Callaway et al. (2025). First, we re-weight our observations to be representative of the true analytical distribution of observed doses. Second, we estimate a specification in which the continuous treatment is rendered binary (above/below median).

[32] This is not as clean for employment as it is for income, education and disability but it still holds for an above/below median split of baseline intensity.

Benefit-cost ratio estimates and ACRT The 1.4% increase in mean income flowing from an IQR 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.[33] To approximate average impacts for those who contracted pneumonia and accessed antibiotics (*i.e.*, an Average Causal Response on the Treated, or ACRT), 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 ACRT estimates imply gains of 0.37 years of schooling, 3.2 percentage points in the likelihood of employment, and 9% in family income. While these impacts are large, they are 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.[34]

3.2 Robustness checks

In the preceding section we investigated sensitivity of the outcomes to the timing of exposure, and the strong parallel trends assumption. In this section we discuss additional specification checks.

Measurement error in the exposure variable The estimates in Table 1 use the all-age pneumonia and influenza mortality rate averaged over the pre-sulfa period, 1930-36. In Appendix D, we explain that this choice was made to mitigate measurement error. Importantly, we demonstrate that the "portmanteau" variable transmits the relevant signals: using data from the 1940 census where these variables are independently measured, we show that the post-1937 drop in the compound rate was entirely driven by pneumonia (not influenza). We also show that the drop was sharper among infants who, at baseline, were more likely to contract pneumonia. We nevertheless investigated sensitivity of our estimates to re-specifying the baseline measure. First, we replace the compound rate with the pneumonia mortality rate in 1935 (Table E1, Panel A) and, second, we use the infant rate rather than the all-age rate (Panel B). Third, we control for a proxy for

[33] 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.

[34] 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., 2016).

measurement quality (completion of birth system registration, which we explain in the Appendix) in Panel C. Fourth, we replace the reduced form baseline estimates with their 2SLS equivalent, instrumenting the birth state and cohort varying mortality rate with Post sulfa$_t \times$ Base Exposure$_s$ (Table E1, final Panel). Our findings are robust to all of these variations.

Confounding events See Table E1. We account for state-level New Deal spending during 1933-1939 using data collected by Fishback et al. (2003) interacted with a post-sulfa indicator (Panel D). To account for the possibility that the dust storms of 1930-1936 in the Dust Bowl states drive our results, we drop these states (Panel E). We additionally document that our results are robust to removing World War II Cohorts (panel F), removing both World War II and Depression year cohorts (Panel G).

Selection *Selective Migration* – We consider whether birth state, which we use to assign exposure to pneumonia at birth, may be endogenous. If potential parents move to low pneumonia states in order to improve the life chances of their births, and the movers are positively selected, this would create a compositional effect at baseline that reinforces the tendency for low pneumonia states to have better outcomes. However, this is absorbed by state fixed effects. Once sulfa drugs were introduced, we showed that state differentials in pneumonia were narrowed. To the extent that this attenuated disease-led migration flows, we will see smaller *relative* improvements in long run outcomes in low pneumonia states after 1937 or, equally, larger treatment effects of sulfa. To investigate this we use information on migration of reproductive age individuals, aged 20-40 in the 1940 census files and find that, conditional upon state income, there is no evidence that migration induced by the disease environment may be driving our results (Appendix E, Table E2).[35,36]

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. If it went the other way, it would render our estimates conservative. We investigate this using data from the 1940 census and a specification similar to Table 1 and find no evidence of endogenous changes in sample

[35] Previous studies of the long run effects of early life interventions tend to regard the birth region as exogenously given, but this is questionable. 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.

[36] Here we have discussed selective migration of *parents* of our sample cohorts as this could 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, but we find no significant tendency for sulfa-exposure in infancy to encourage cross-state migration (Appendix E Table E3).

composition post-1937 (Appendix E, Table E4).

Selective survival – Following the advent of sulfa drugs, a greater fraction of frail children who would previously have succumbed to pneumonia survived. As the marginal survivor will be 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 more meaningful role in determining the racial differences that we document in the next section, we take a more formal approach to assessing it in Part II of the paper.

Sensitivity to controls The baseline model includes fixed effects for birth state and for census region by birth year, alongside time-varying covariates that include placebo diseases (not treatable with sulfa drugs) and that capture the socioeconomic and health environment in the state; these are consistently created as the baseline value of the variable interacted with the post-sulfa indicator). Our design thus isolates sulfa exposure from broader geographic or macro-level time-varying shocks. In Figure 6 we scrutinize the role of the controls. We start with nothing but the baseline fixed effects. Adding mortality from untreatable diseases results in a statistically insignificant increase in the coefficient. Adding socioeconomic and health variables makes no difference. That our findings are not sensitive to the controls that we use suggests that if observables and unobservables behave similarly, any relevant unobservables play a small role, if any, in driving our findings (Altonji et al., 2005; Oster, 2019). We additionally investigated sensitivity of our results to adding further controls. First, we add census wave×birth state and census wave×birth year fixed effects, to account any place or cohort-specific differences in life cycle human capital accumulation and socioeconomic outcomes. The coefficient estimates remain unchanged. We then add birth state-specific time trends. Following Bhuller et al. (2013) and Goodman-Bacon (2021b), the state-trends are estimated in the pre-treatment period, and projected forwards into the post-treatment period to avoid that they capture endogenous variation. This enlarges the treatment effects for income, education and disability. However, it drives the coefficient on employment to zero. The estimates for employment are thus less robust as for the other outcomes.[37]

[37] Employment rates for women were increasing across these cohorts, whereas they were already high for men. This may explain why employment estimates for women are more robust than for men (see below). This could explain the weakness in these pooled models.

3.3 Heterogeneity in impacts by gradients in access to sulfa drugs

We documented in Section 1 that the impact of sulfa drugs on mortality, *i.e.* the 'first stage', is increasing in the density of pharmacists per capita in the individual's birth state. After considering a number of factors including family income and urban location, this is the strongest predictor of access to sulfa drugs. In this section, we examine whether this gradient in access maps into the impact of sulfa on long term outcomes of exposed children. See Table 2, where (1) is augmented to include an interaction between sulfa exposure and baseline pharmacist coverage at the birth-state level. The gradients are all in the expected direction and statistically significant for schooling, family income and employment (similar patterns are observed for alternative outcomes in Table F2). In the case of income, the marginal effects suggest that individuals living in states in the bottom decile of pharmacist coverage gained a 1.5% uplift in income, compared to 4.5% for individuals with the greatest exposure to pharmacists. For schooling, the ratio of effect sizes in the highest vs the lowest coverages states is 4, and for employment it is three. The event study version of these interacted models is in Appendix Figure B5, where we observe clear evidence of these differences in outcomes emerging between high and low-pharmacist coverage states following the arrival of sulfa drugs for all outcomes other than disability (for which the overall estimate is an artifact of pooling the data across race and gender, which we will look at next). Overall, these results add weight to our causal design by confirming a dose-response in access (earlier we confirmed a dose-response in exposure conditional upon access).

3.4 Heterogeneity by gender and race

Table 3 reports estimates for white men, white women, Black men, and Black women; the corresponding event-study graphs are presented in Appendix Figure F3, and robustness checks discussed previously in Tables F4-F7. For white men (Panel A) we see large and precisely determined gains in schooling and income, and declines in work-limiting disability. There is no increase in employment, the baseline being high. Distributional estimates for income in Figure F4 point to shifts towards the 75th and even 90th percentile, away from the 10th to 25th.[38] For white women the point estimates are statistically significant for each of the four outcomes, larger in magnitude for employment than for men, but about half the size of the male coefficients for schooling and income. These gender differences in treatment effects are consistent with women having had lower baseline employment rates, but fewer opportunities in that era for high-skilled employment

[38] Results for other outcomes are provided in Table F3, and line up with patterns for principal outcomes.

(Goldin, 1998).

Black men show the largest increase in family income (twice as large as white men) and employment (twice as large as for white women), a substantial but imprecisely determined increase in schooling, and only a small and imprecisely determined improvement in work-related disability. The larger increase in income for Black compared with white men is consistent with Black infants having had much higher pneumonia exposure than White infants, which is reflected in the cohort averaged-estimates. Indeed, the inflation factor to scale effects on the treated into average effects would be a third to half of that for white men given higher infection rates (*i.e.* a larger portion of the population being exposed). Distributional effects for Black men point to effects closer to the median (Figure F4), with a greater likelihood of incomes concentrated in the 50^{th}-75^{th} percentiles, and a lower likelihood of appearing in the 25^{th}-50^{th} percentiles. The larger standard errors for Black men may stem from higher sampling variance in the (smaller) Black population, or from heterogeneity in treatment effect estimates, both of which we discuss in more detail below.

In contrast to the other three groups, Black women experience no economic gains from infant exposure to sulfa drugs. In fact the coefficient for schooling and employment is *negative* and the remaining estimates are imprecise. One possible explanation for the more muted impacts among women is differential exposure—male infants were about 30 percent more likely than female infants to contract pneumonia in the first year of life (Britten, 1942; Gluckman and Hanson, 2004a; Low, 2000; Waldron, 1983), so boys stood to gain more from the arrival of sulfa. Another is that there were more limited labor market returns to women's human capital (*e.g.*, marriage bars; Goldin 1991). While these are explanations of muted coefficients for white women, they do not explain the negative coefficients for Black women. We show below that differential impacts on marriage and fertility contribute to understanding the differences between Black and white women.

Measurement error in mortality rates for Black men and women The state-level pneumonia mortality rate will less accurately reflect exposure for Black individuals, a minority population in all U.S. states. In addition, vital statistics registration was known to be incomplete in rural Southern states, where the majority of Black people resided during the study period (Puffer, 1937; Shapiro and Schachter, 1952; Ewbank, 1987); see Appendix D.2, and Figure D2. While we cannot fully address what is effectively an omitted variables problem (see Bound et al., 1994), we attempt to account for measurement error in exposure for Black men

and women by controlling for the quality of vital statistics.

Fortuitously, a nationwide audit of births was conducted in 1940 (Shapiro and Schachter, 1952). Using this we derive the percentage of births registered by the state system as a proxy for quality. We find that this is systematically positively associated with earlier entry into the registration system.[39] As the concern is with bias in the coefficient on Post sulfa$_t$ $\times$ Base exposure$_s$ given non-random measurement error in Base Exposure$_s$, we introduce in the regressions, the term Post$_t$ $\times$ Birth Registration$_s$, the main effect being absorbed by state fixed effects.[40] A detailed discussion of the proxies and our approach is provided in Appendix D.2. Inclusion of these terms also helps account for the possibility that establishment of birth and death registration systems have long-run impacts on health and human capital themselves (Noghanibehambari and Fletcher, 2023). We find that controlling in this way for state heterogeneity in measurement error results in slightly larger estimated coefficients among black men and women (Table F6, F7), with the broad direction of results similar to the baseline results.

Access to sulfa drugs among Black individuals We investigate the possibility that the coefficients for Black men and women are attenuated because they had more limited access to sulfa drugs when they were introduced. The evidence suggests this was not the case. We find that Black Americans experienced sharp absolute declines in pneumonia mortality after 1937, in fact much larger declines than the white population (see Part II Section 3 below and Appendix B, where we also situate the plausibility of this result in its historical context).[41]

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,

[39] There was no corresponding national audit of the death registration system but 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 (Shapiro and Schachter, 1952). Appendix D Figure D2f reveals a positive correlation of years of entry to the national birth and death registration systems, also illustrating the range across states in years of registration.

[40] See discussion in Appendix D.2. Briefly, if outcome y depends upon (Post$_t$ $\times$ [Base Exposure $+ \nu$]$_s$) where ν_s is the error in measurement of base, then y will depend upon (Post$_t$ $\times$ Base Exposure$_s$) and (Post$_t$ $\times$ ν_s). Omission of (Post $\times$ ν_s) will bias the coefficient on (Post$_t$ $\times$ Base Exposure$_s$) if Base Exposure$_s$ is correlated with ν_s. In Appendix D Figure D3 we plot this correlation and show that it is positive.

[41] In a paper that estimates the contribution of sulfa drug innovation to aggregate declines in mortality using time series data, Jayachandran et al. (2010) conclude with a look at Black-white differences. They find no race differences in pneumonia mortality decline. For other sulfa-treatable conditions they report a smaller decline in mortality in the Black population. However, what they measure is the proportional decline in sulfa-treatable diseases; *i.e.*, differential changes in *logged* mortality. Here, we focus on absolute declines in pneumonia mortality – *i.e.*, differential changes in mortality *levels* – which best captures early life disease risk and, consequently, long run outcomes (Chuard et al., 2022).

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 frictions among the Black population in accessing sulfa drugs (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 failing to control for differences in access may bias the coefficient on $\text{Post}_t \times \text{Base Exposure}_s$. We therefore control for $\text{Post}_t \times \text{Pharmacists}_s$, allowing the main effect of pharmacists to be absorbed by state fixed effects. We include this control for access in our baseline model.[42] We additionally investigate heterogeneity in long run effects by measures of access, replicating the analysis in Section 3.3 by race and gender. Consistent with Black persons being able to access sulfa drugs, we find evidence of larger long run effects among Black men in areas where there were more pharmacists per capita (Appendix Table F8).

Having documented long run gains from sulfa drugs for Black men on average, we investigate heterogeneity in the socioeconomic benefits of antibiotic exposure for Black Americans individuals as a function of birth state systemic discrimination in Part II.

What explains limited returns for women? Our analysis takes place in an era when labor market opportunities for women were directly limited by family formation (Goldin, 1991). We investigate the role of these factors in determining the observed responses of female labor market outcomes to the introduction of sulfa drugs modified this. Details of the estimated specifications are in Appendix F.2.2. First, we interact sulfa exposure with two cross-state measures of female economic opportunity in 1940: the female-to-male employment ratio and the college-completion ratio.[43] Across outcomes, the triple-interaction coefficients are uniformly small and statistically insignificant (Table F9), indicating that restricted labor-market returns for women do not play a significant role in explaining gender difference in treatment effects.

Second, we examine family-formation. We find that Black women exposed to sulfa drugs in infancy are markedly more likely to marry and to have children at both the extensive and intensive margins. White

[42] If we do not include condition on pharmacist coverage, we still see increases in family income and declines in disability among Black men, see Figure F6.

[43] The 1930 census does not have a measure of educational attainment and for this reason we generate state-level figures at 1940. Aggregate figures from Goldin et al. (2006) suggest that college completion ratios were relatively stable among cohorts at college completion age between 1930 and 1940.

women also exhibit a (smaller) increase in marriage, but their intensive margin fertility falls (Appendix Table F10 and Figure F8). These outcomes are of substantive interest in themselves, consistent with the improved health endowment of infants born after sulfa drugs resulting in higher fecundity, with this being dominant for Black women on account of their higher baseline exposure to infant pneumonia. So as to (descriptively) study the relevance of these responses to the labor market outcomes, we stratify the sample by these endogenous variables. This exercise is very informative. We are better able to understand the null and adverse results for Black women documented in Table 3 as these are driven entirely by those who marry or have higher fertility after sulfa is introduced. The sample split reveals compelling evidence of positive impacts of childhood sulfa exposure on Black women's income income and/or employment for those who have low fertility (0 or 1 child) or are unmarried at baseline. In fact we see the same pattern for white women, which confirms that family formation in this era was a significant barrier to women's participation in the economy. The race difference within women is that, after sulfa, white women had fewer children and were more likely to be employed, while black women had more children and were less likely to be employed and less likely to earn, see Appendix Table F11. Thus for Black women but not white women, the expected returns to child-bearing appear to have risen relative to returns from schooling and market work. Put differently, sulfa exposure improved fecundity but it did not, on average, offset the school and labor market barriers imposed by systemic discrimination. We investigate this more carefully in the next section.

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

1 The Role of Institutions: Context, Data, and Research Strategy

An established literature had documented how Jim Crow era policies in the American South limited the upward mobility of Black Americans (Margo, 1990; Donohue and Heckman, 1991; Smith, 1984; Welch, 1974; Ward, 2023). Under the Jim Crow Laws passed in the post-Civil War Reconstruction era in the late nineteenth century, racial segregation was institutionalized in 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 Black Americans relative to whites. The extent of systemic discrimination was weaker (thought not absent) in Northern states and, within the South, it varied across the states. We utilize this

historically determined variation across race and state in the intensity of systemic discrimination 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 (and gender):

$$\begin{aligned} Y_{istc} &= \beta_0^{rg} + \beta_1^{rg}(\text{Post sulfa}_t \times \text{Base Exposure} \times \text{Discrimination Proxy}_s) + \beta_2^{rg}(\text{Post sulfa}_t \times \text{Base Exposure}_s) \\ &\quad + \beta_3^{rg}(\text{Post sulfa}_t \times \text{Discrimination Proxy}_s) + \theta_s^{rg} + (\eta_t \times \mu)_d^{rg} + \lambda_c + X_{st}'\Gamma^{rg} + \varepsilon_{istc}^{rg}, \end{aligned} \tag{3}$$

where notation is as in equation (1), and superscript rg highlights group-specific estimation. The coefficient β_1^{rg} on the new term, Post sulfa$_t$ $\times$ Base Exposure $\times$ Discrimination Proxy$_s$ is the discrimination-gradient in long run returns to reduced infant exposure to pneumonia associated with introduction of sulfa-drugs in 1937. For desirable outcomes (*e.g.*, income rather than work-limiting disability) for which $\beta_2^{rg} > 0$, finding that $\beta_1^{rg} < 0$ for the Black sample will confirm our hypothesis that being born in areas with greater systemic discrimination dampened the long-run impacts of early life exposure for this group.

While our proxy for systemic discrimination (described in the next subsection) is historically pre-determined, identification using the outlined approach may be compromised if our measure of systemic discrimination is not only a proxy for access to quality schools and labor markets (and hence net returns to investment) for Black Americans, but also correlated with relevant omitted variables. We now spell out how the triple difference style specification addresses relevant concerns. The fixed effects of systemic discrimination in the birth state are captured in race$\times$state specific fixed effects θ_s^{rg}, so β_1^{rg} will not simply reflect that outcomes like education and income were worse among blacks in states with higher levels if systemic discrimination. The term Post sulfa$_t$ $\times$ Discrimination Proxy$_s$ captures any convergence or divergence in outcomes after 1937 between states with different degrees of systemic discrimination that may have occurred independently of changes in pneumonia prevalence, for instance, North-South economic convergence. We allow for broader changes in the relationship between systemic discrimination and long-run outcomes over time by including race$\times$birth cohort fixed effects *in each specific* census-district. Importantly, the gradient estimates are conditional upon race$\times$state$\times$birth cohort variation in per capita income, schools, hospitals, physicians, and mortality from six "control diseases", outlined in Section 2 of Part I. So if, for example, Black men and women in more discriminatory states experienced different trends in access to public health, or higher mortality rates, this would be captured by these controls. Finally, although Base Exposure$_s$ was higher in states with higher levels of discrimination, there was considerable independent variation in Base Exposure$_s$ and

Discrimination$_s$ (Appendix D.3, Figure D4, panels (d) and (h)).

What further strengthens identification is that white men and women provide a "control group" for our analysis of systemic discrimination. If estimates of equation (3) for white men and women showed gradients of the same sign as for Black men and women, then the gradients may reflect a pan-racial process (*i.e.* not systemic discrimination) that differentially drives the returns to early life endowments in more *versus* less discriminatory 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 discrimination, with the latter impacting Black and not white outcomes.[44] Unobservables of potential concern are race×state heterogeneity in measurement of Base Pneumonia$_s$ and in access to sulfa drugs, as both may have been worse for Black Americans born in more discriminatory states, although they would also have to have evolved differently after 1937 to matter. Below, we discuss how we account for this and we also analyze selective migration and mortality.

Measures of systemic discrimination: In order to isolate the role of race-specific institutions, we use the birth state share of enslaved persons in the population in 1860 (Nunn, 2008; Acharya et al., 2016). This has been adopted broadly in the literature, and 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 McLean, 2003; Acharya et al., 2016; Althoff and Reichardt, 2024). The share of enslaved persons is effectively zero in the North and ranges from 0.01 to 0.57 in Southern states. We additionally show results with the number of Jim Crow laws passed in each state. This measure is recently validated in Althoff and Reichardt (2024), who show how the economic progress of freed Black Americans as of 1940 was decreasing in this measure.

Both measures are (i) pre-determined,[45] (ii) measured for every state, and (iii) shown in a number of studies to be predictive of modern-day racial gaps in opportunities and attitudes (*e.g.*, Nunn, 2008; Acharya

[44] 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 white over Black Americans in states with higher levels of discrimination than those with lower levels of discrimination.

[45] In the case of Jim Crow laws, these were passed from 1865–1950, however the vast majority were passed prior to 1937 (see Althoff and Reichardt, 2024, Figure IV).

et al., 2016; Althoff and Reichardt, 2024). We further document the empirical content of these measures in Appendix D.3, showing that states with higher share of enslaved persons and a greater number of Jim Crow laws display markedly larger Black-white gaps in schooling and wages in 1940. The strong, negative slope in Appendix Figures D5-D6 confirms the bivariate relation for schooling and wage ratios outside the zero-slave-share and zero-Jim Crow-law states.[46] Appendix Figure D4 shows that historical slave share varies independently of baseline pneumonia mortality and our measurement-error proxies, allowing us to isolate how discrimination mediates the long-run impact of sulfa exposure.

2 The Role of Institutions: Results

Estimates of equation (3) reveal a starkly defined and systematic tendency for the long run returns to Black men and women from infant exposure to antibiotics to be decreasing in the birth state share of enslaved persons for each of our main outcomes (Table 4). There are no similar discrimination gradients for white men and women (Table E5).[47] Thus, for white men, the introduction of sulfa drugs stimulated convergence in pneumonia across the states (Figure 3), which was mirrored in their long run economic outcomes. However, for Black men, despite convergence in pneumonia (which occurred even within the South—see Figure 7), we see a divergence in the longer run outcomes of men who were born in more vs. less discriminatory states.

Black men born in states with historical shares of enslaved persons close to zero (Northern states) experience large improvements in education, income, employment and work-limiting disability (coefficient on $\text{Post}_t \times \text{Base Pneumonia}_s$), gains that are typically larger than for white men, consistent with their initial pneumonia burden being larger.[48] All of these gains are significantly smaller for Black men born in (Southern) states with slave shares greater than zero.[49] Similar gradients are observed when we use the number of Jim Crow laws in place of the historical fraction of enslaved persons (Table 4).

At the bottom of Tables 4 (and F12 for additional outcomes) we compute outcome gains for Black men

[46] An earlier, slimmer version of this work (Bhalotra and Venkataramani, 2015) demonstrates that gradients for Black Americans emerge also with other proxies for discrimination, including a dummy for Southern states and state-specific Black–white returns to schooling, and this underscores that our results are not sensitive to the proxy we use to measure birth-state variation in systemic discrimination.

[47] As discussed in Appendix E.1.2, most coefficients are not statistically significant and any gradients in marginal effects are an order of magnitude smaller than effects among Black women and men.

[48] We observe similar large effects in other measures such as high school and college completion, and poverty—see Table F12.

[49] The *difference* in gains between states with lower and higher levels of institutionalized discrimination is in the coefficient on $\text{Post sulfa}_t \times \text{Base Exposure}_s \times \text{Discrimination Proxy}_s$. The total effects for Black men in states at different percentiles of each discrimination proxy are presented at the base of each panel as *Effect size* in Table 4.

and women at specific percentiles of the distribution of systemic discrimination (the distributions of measures considered are plotted in Figure A1). The gradients are steep. For instance, a decrease in pneumonia exposure created by the introduction of antibiotics and set at the inter-quartile range of the baseline pneumonia distribution is estimated to raise income by 11.4% for Black men born in states with no slave history, and a much smaller increase of 3.9% for Black men born in states in the highest decile of the distribution of historical shares of enslaved people. For educational attainment the corresponding figures are 0.55 years of education vs. essentially no increase. We see similarly large gaps for employment and work-related disability and also for the derived outcomes—high school, college, and poverty. It is notable that while the average impact of the sulfa shock on college attainment was not distinguishable from zero, there is a large increase of 6.55 pp in the low-discrimination states vs an increase of just under 1 pp in the high discrimination states. The gradients are steep irrespective of the measure used.[50]

Contribution of education We use Mincerian estimates of the returns to education in areas above and below the median of historical enslaved population applied to the estimates in Table 4 to approximate the contribution of education to wage gains. Black men in states with higher levels of systemic discrimination not only experienced smaller increases in income, but the contribution of education to income gains was smaller. At the lowest levels of historical slave share, income rises 11% and education explains about 60% of this while, at the 90th percentile, income rises by 4% and education explains only about 17% of this. This is consistent with sulfa exposure having led to higher skill acquisition in these states. That the more muted benefits in more discriminatory states are, in general, positive among black men is consistent with direct effects of infant health on adult incomes given evidence that Black Americans in discriminatory states experienced sharp declines in pneumonia in 1937 (Figure 7). For example, the sulfa shock may have led to improved productivity in brawn-intensive work.[51]

[50] The IQR range that we plug in is for the baseline all-race all-state pneumonia rate (0.29 fewer deaths per 100,000). As the rate among Black men and women was much higher (see Figure 7), the reported estimates are conservative. We use them so that these results are in the same "metric" as the earlier results for all individuals.

[51] In Appendix E.1.2, 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.

3 Threats to Inference and Alternative Interpretations

The estimates in Part I 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. Specification checks were reported on the pooled data and again on the data samples specific to race and gender. Here we focus on threats to identification of *discrimination-gradients* in long run impacts of sulfa, and whether the gradients may have interpretations other than the one we suggest. We discussed the main threats to identification in Section 1 of Part II, where we set out our empirical strategy. In particular, we have conditioned out the main effects of race and of systemic discrimination and allowed for trends and for post-sulfa convergence in outcomes that vary by race and state for reasons unrelated to our project. The parallel (placebo style) results showing largely absent gradients for white men and women confirm that the gradients we identify for Black men and women reflect state-specific factors that are correlated with systemic discrimination. We now investigate the main residual concerns.

Possibly the most natural alternative explanation of the discrimination gradients for Black Americans is that they reflect gradients in **access to sulfa drugs** rather than in long run outcomes conditional upon access. First, we directly investigate access by estimating race and region specific "first stage" equations which show *larger* post-1937 drops in absolute pneumonia mortality rates for Black Americans than for whites, even in the South where systemic discrimination was high (Appendix B, Table B2, also see Figure 7).

Second, though we have noted smaller returns to sulfa exposure in states with higher measures of systemic discrimination, there is evidence of positive gains for many outcomes even at high levels of systemic discrimination (see *Effect size* in Table 4). This is another result that is consistent with access. Third, we show that our proxies for systemic discrimination do not simply proxy greater access to sulfa drugs. In Appendix Table B7 we rule out that initial mortality declines are smaller in areas with higher measures of systemic discrimination, *even* if focusing on non-white mortality only, and *even* only within Southern states. Fourth, 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 unlikely that pecuniary costs of sulfa would hamper access to this life-saving drug. Finally, in Appendix B, we discuss the coherence of our findings with previous work situated in this era.

We considered whether the **Great Migration** of Black men and women from the South to the North

might have generated compositional effects that explain our finding that Blacks in the South saw smaller post-sulfa increases in economic outcomes. In fact this migration slowed after 1930, before our analysis period. Nevertheless, we consider this. Since the relatively educated were more likely to migrate northward (Vigdor, 2002; Aaronson and Mazumder, 2011), we explain in Appendix E.1.2 how accounting for this would strengthen our conclusions. We still model this and find there was in fact no significant endogenous migration (Appendix E, Table E2).

Weaker socioeconomic gains from sulfa for Black men and women born in more discriminatory states may alternatively flow from greater negative **survival selection** in this group because they experienced larger absolute declines in pneumonia mortality post-sulfa (Appendix B, Table B1). 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 E Table E6).

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 gradients in educational outcomes we find 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 systemic discrimination are absorbed by state trends and our controls for state$\times$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 levels of Base Pneumonia.

Overall, the evidence indicates that responsive reinforcing investments in education may have contributed substantially to the large sulfa-led increases in income and mobility achieved by white men and women and by Black men and women in states with lower levels of systemic discrimination. First, crude simulations suggest that, where systemic discrimination was absent or small, increases in schooling 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 born in states with less systemic discrimination] were significantly more likely, upon sulfa-drug exposure, to find a place in the top quartile [second highest quartile] may suggest dynamic complementarities. This is particularly so because the improvements in income and employment 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 employment effects 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. A number of previous studies find evidence that parental investments reinforce early life endowments, see, for example, Duque et al. (2018); Adhvaryu et al. (2024); Noghanibehambari and Fletcher (2024); Johnson and Jackson (2019). 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.

Conclusions

We demonstrated that the availability of antibiotics to treat pneumonia, an infection that particularly affected infants in the 1930s, led to increased educational attainment, income and employment, and reductions in adult disability. 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 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 systemic discrimination 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 Robinson, 2012). Our finding that Black men and women born in states with substantial systemic discrimination 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, 2019). 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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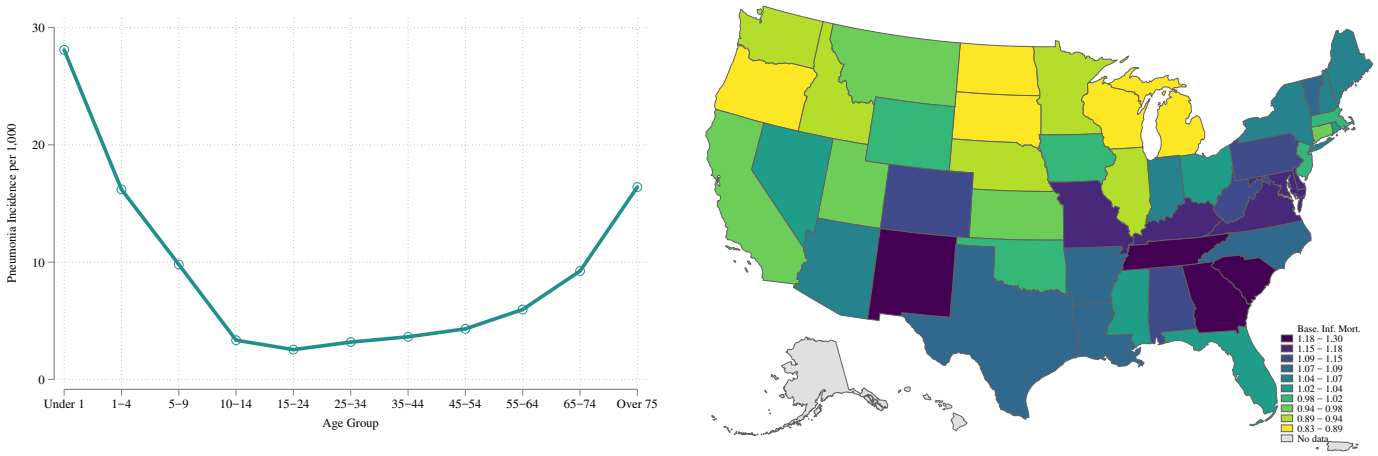
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Tables and Figures

Figure 1: Age and state variation in pneumonia

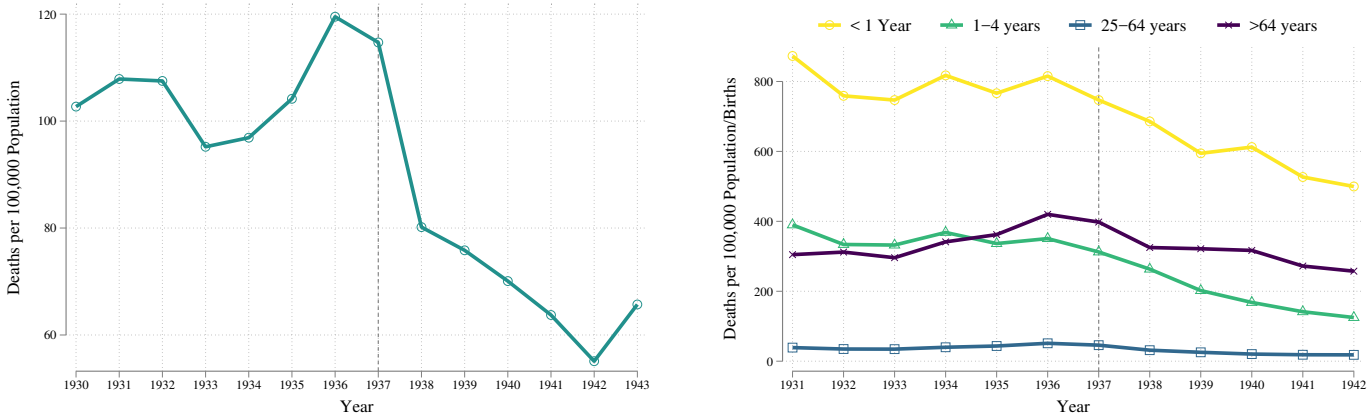


(a) Age Distribution of Pneumonia Incidence

(b) Geographic Distribution of Pneumonia Mortality

Notes: Panel (a) presents figures of incidence of pneumonia per 1,000 population in 1935-1937 by age group from a historical survey. Source: Britten 1942. Panel (b) presents deaths per 1,000 population from pneumonia and influenza averaged over 1930-1936, prior to the introduction of sulfa drugs. Source: U.S. Vital Statistics. Full details of data are provided in Section 2.1 and Appendix A.

Figure 2: Trends in pneumonia and influenza mortality

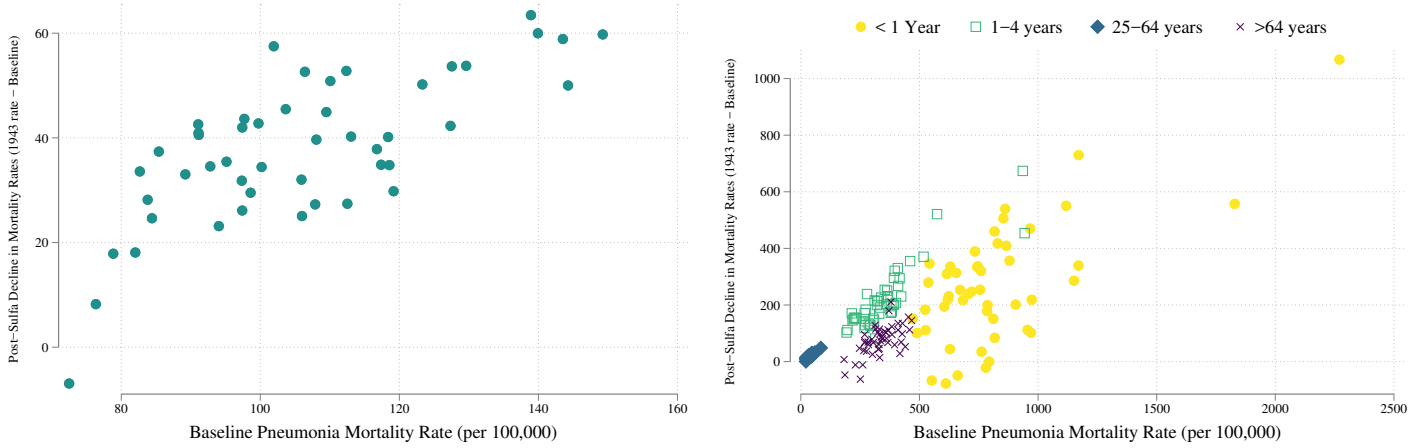


(a) All population mortality

(b) Age-specific mortality

Notes: Data from the U.S. Vital Statistics. Figure 2(a) presents all age mortality. The blip in 1936 reflects an uptick in influenza mortality from the flu pandemic in this year. The annual vital statistics data combine pneumonia and influenza mortality to avoid mis-classification error. Census data for 1930 and 1940 show that the influenza mortality rate was unchanged across the decade, clarifying that the decline shown in Figure 2(a) is driven by a sharp drop in pneumonia mortality. Figure 2(b) presents age-specific mortality rates. For individuals $<$ 1 year, rates are presented as deaths per 100,000 births. Trend breaks in the infant pneumonia mortality series are statistically significant (see Appendix Tables B1 and B2).

Figure 3: Post-sulfa convergence in pneumonia and influenza mortality rates



(a) All age

(b) By age group

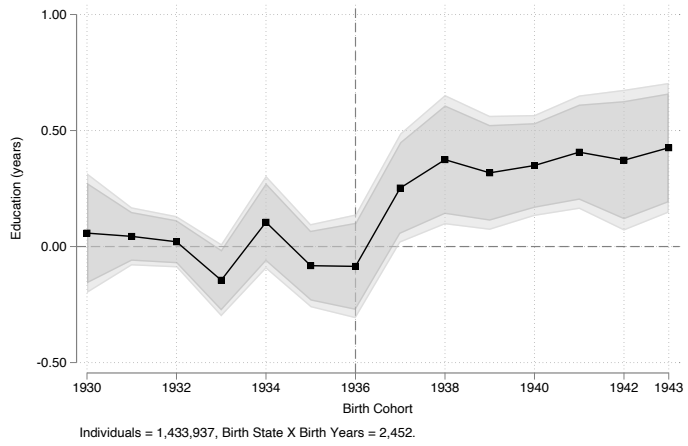
Notes: The base rate of pneumonia and influenza mortality is defined as its average over 1930-1936. Every dot is a state. Figure 3(a) presents convergence in all age mortality, while Figure 3(b) presents age-specific patterns of convergence. Rates are all expressed per 100,000 population, with the exception of $<$ 1 year olds, for whom rates are expressed per 100,000 births. Source: US Vital Statistics.

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

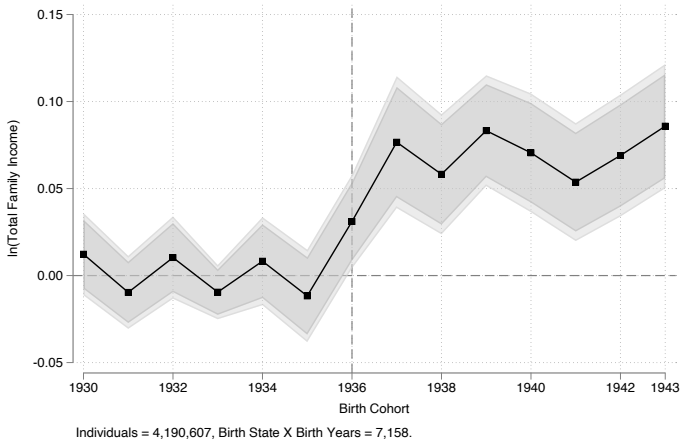
	Schooling	log(Family Income)	Employment	Work Limiting Disability
	(1)	(2)	(3)	(4)
Post Sulfa × Base Exposure	0.199**	0.0497***	0.0172**	-0.00816**
	(0.0872)	(0.0120)	(0.00736)	(0.00354)
FWER p-value	[0.049]	[0.004]	[0.091]	[0.079]
Effect size for an interquartile shift in base exposure	0.0560 years	1.399 %	0.485 pp	-0.230 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633

Notes: Each column represents a separate regression of the outcome indicated in column headers following specification (1) . Full details of the specification and baseline covariates are discussed in Section 2; robustness to alternative covariates is presented in Figure 6. The estimation sample consists of all individuals born between 1930 and 1943 (inclusive) observed in census microdata. Coefficients are presented along with standard errors clustered by birth state in parentheses. Multiple comparison adjusted p-values, which control the family-wise error rate following (Romano and Wolf, 2005) are provided in square brackets based on 10,000 bootstrap replicates. The estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) on the outcome in each column are provided as the reported effect size at the foot of the table. *** $p<0.01$; ** $p<0.05$; * $p<0.10$.

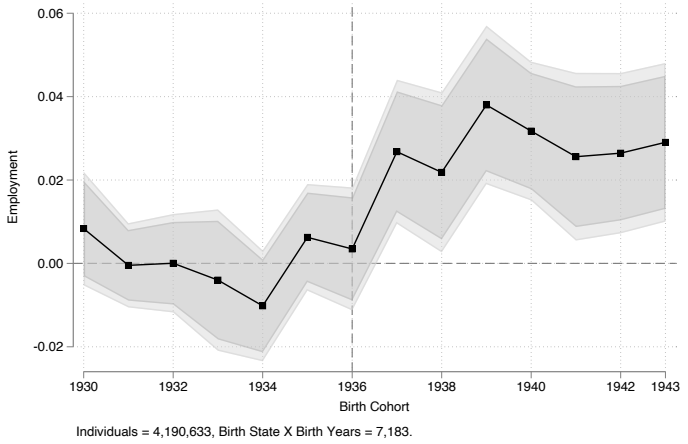
Figure 4: Event Study Estimates of Pneumonia Exposure in Infancy on Adult Outcomes



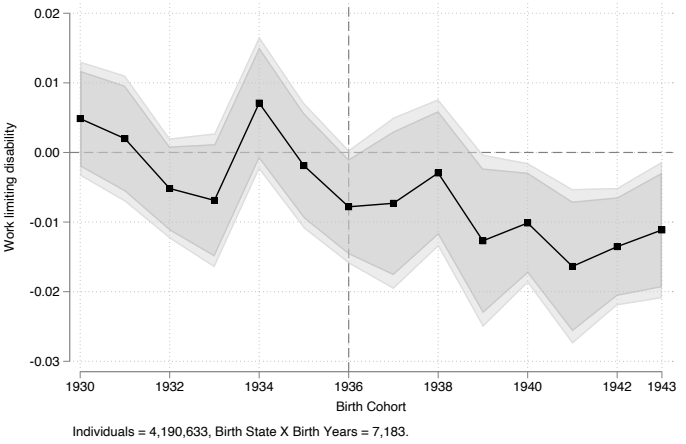
(a) Education



(b) log(Family Income)



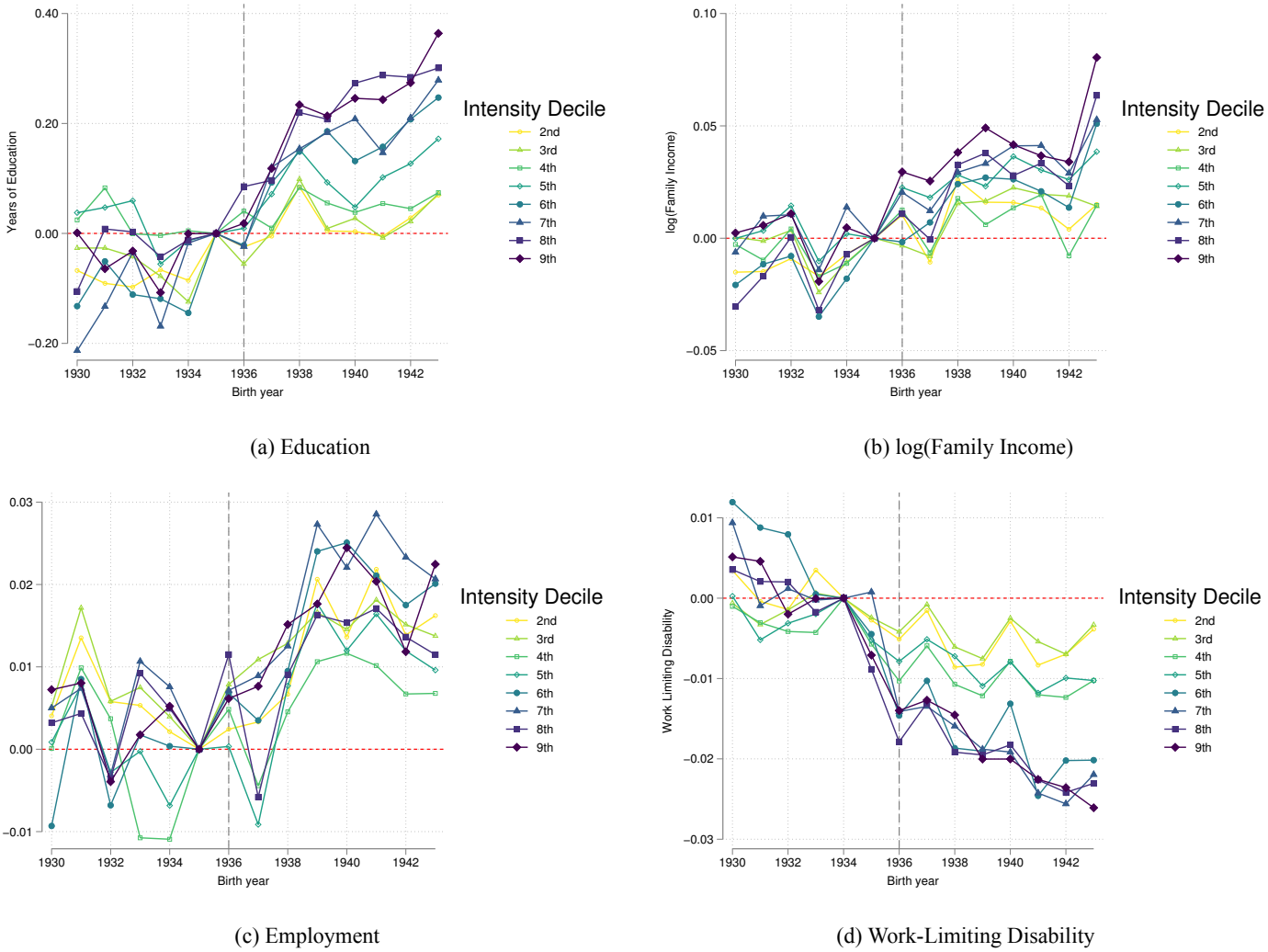
(c) Employment



(d) Work Limiting Disability

Notes: Event study estimates present impacts of exposure to sulfa on outcomes indicated in each plot caption. Each point reflects the coefficient estimated on an interaction term between the marked birth year 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 for each race× gender group and the full set of controls for mortality from other diseases and state macroeconomic and infrastructure variables (refer to notes to Table 1), as well as census division by year FEs for each race× gender group to capture geographically clustered un-observed time-varying shocks. State specific trends are omitted so as to allow us to discern the presence of pre-trends. 90 and 95% confidence intervals are presented as darker and lighter shaded areas respectively. The entire set of pre-treatment periods are conditioned to be centred on zero as the omitted baseline reference, following Miller (2023). Observation numbers for individuals and birth state by birth year by census wave groups (a maximum of 46 birth states, by 14 birth years, by 3 census waves for each of the 4 demographic group) are provided as figure notes.

Figure 5: "Strong Parallel Trends Assumption" and the Average Causal Response Function



(a) Education

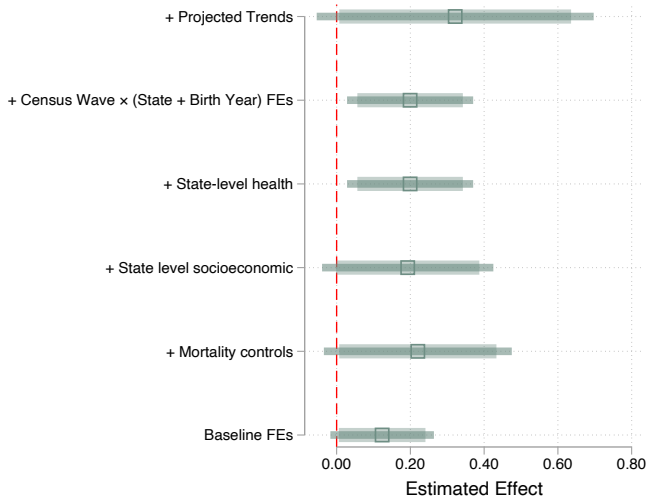
(b) log(Family Income)

(c) Employment

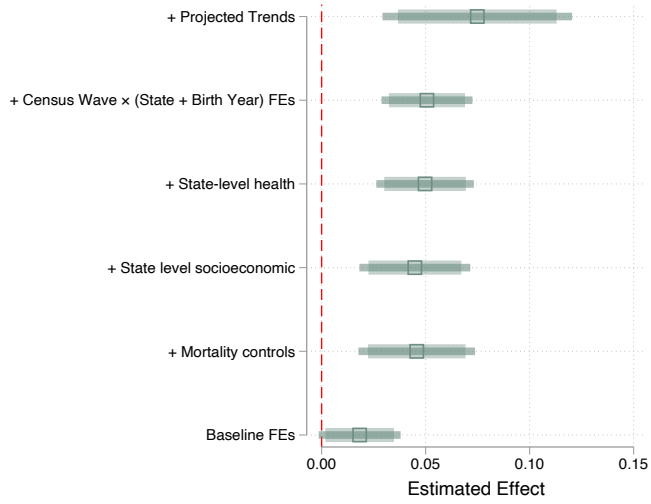
(d) Work-Limiting Disability

Notes: Each panel estimates an event study specification, where outcomes in each intensity level of treatment are compared to outcomes in the lowest treatment intensity group. Treatment intensity groups are calculated based on deciles of baseline pneumonia mortality. Year 1934 is the omitted base category, and as such each line can be interpreted as changes in outcomes when comparing higher intensity groups with the lowest intensity treatment group, standardizing these comparisons based on any baseline differences in 1934. The sample consists of all individuals from birth cohorts 1930-1943. Plots for other demographic groups are provided in Figure F5.

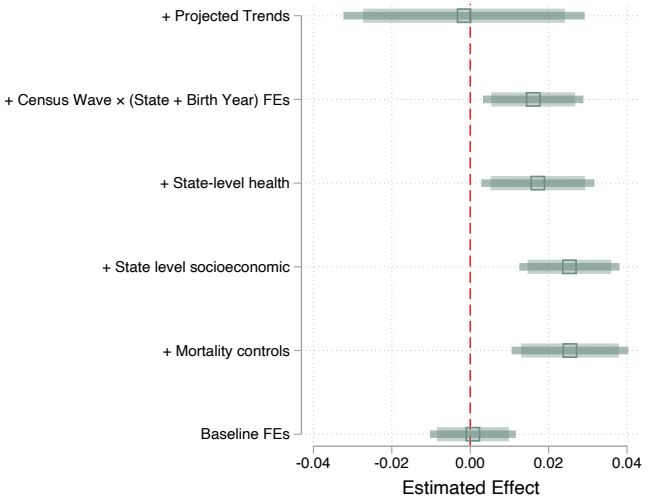
Figure 6: Robustness of estimates to alternative controls



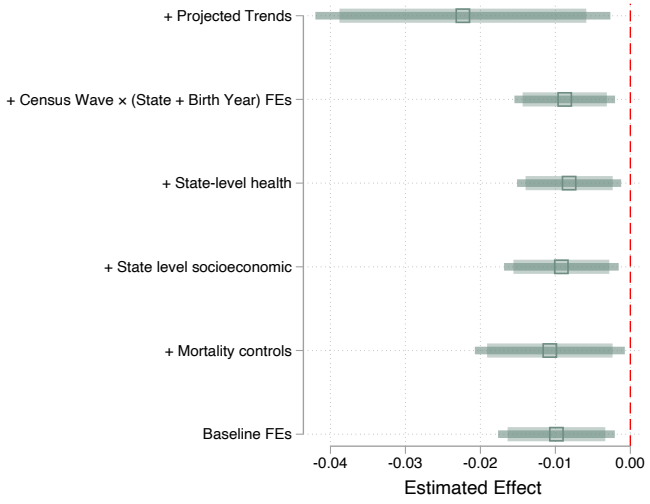
(a) Education



(b) log(Family Income)



(c) Employment



(d) Work-Limiting Disability

Notes: Robustness of single-coefficient Post×Base Exposure is documented to alternative sequences of controls, fixed effects, and trends. Each point and confidence interval refer to a single regression following (1). Baseline FEs refers to standard models with birth state and census division by birth year fixed effects. Alternative sequences of controls and trends are then presented with particular control sets indicated on the plot axes. Moving from top to bottom controls are added in a cumulative fashion, with precise controls discussed in Section 2.2. Projected trends refers to the estimation of trends following Bhuller et al. (2013); Goodman-Bacon (2021b) where state-trends are estimated in the pre-treatment period, and are projected forwards into the post-treatment period. The specification presented in Table 1 corresponds to the third specification from the top where birth state and census division-year FEs are included along with mortality, health and socioeconomic controls. The sample consists of all demographic groups (Black and white women and men), and 90% and 95% CIs are presented based on standard errors clustered by birth state.

Table 2: Gradients in Long-Run Impacts of Infant Pneumonia Exposure by sulfa Diffusion

	Schooling (1)	log(Family Income) (2)	Employment (3)	Work Limiting Disability (4)
Post Sulfa × Base Exposure	0.295***	0.0659***	0.0226***	-0.00949**
	(0.0878)	(0.0126)	(0.00775)	(0.00390)
FWER p-value	[0.013]	[0.001]	[0.019]	[0.036]
Post Sulfa × Base Exposure × Pharmacists p.c.	0.813***	0.136***	0.0454**	-0.0112
	(0.214)	(0.0366)	(0.0197)	(0.0106)
FWER p-value	[0.009]	[0.011]	[0.097]	[0.314]
Effect size for an interquartile shift at bottom decile of pharmacists p.c.	0.0188 years	0.780 %	0.279 pp	-0.179 pp
Effect size for an interquartile shift at top decile of pharmacists p.c.	0.140 years	2.807 %	0.954 pp	-0.345 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633

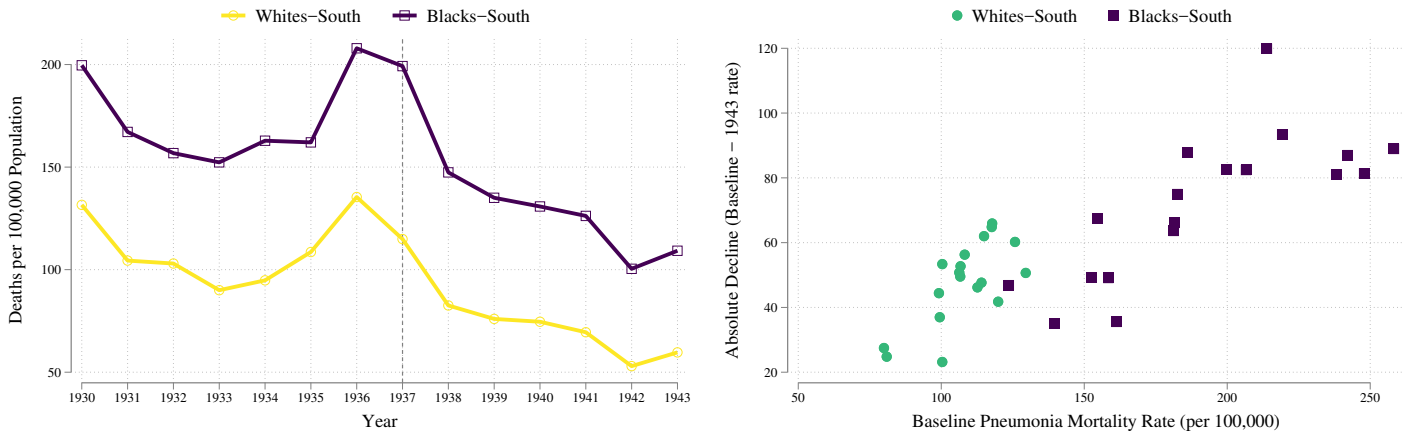
Notes: Each column represents a separate regression of the outcome indicated in column headers following specification (1), where, additionally, interactions with pharmacist coverage cerca 1930 are included. Full details of the specification and baseline covariates are discussed in Section 3.3. The estimation sample consists of all individuals born between 1930 and 1943 (inclusive) observed in census microdata. Coefficients are presented on both the main effect and interactions with pharmacist coverage, along with standard errors clustered by birth state in parentheses. Multiple comparison adjusted p-values, which control the family-wise error rate for the four tests presented across columns following (Romano and Wolf, 2005) are provided in square brackets based on 10,000 bootstrap replicates. The estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) on the outcome in each column are provided as the reported effect size at the foot of the table both in areas with low pharmacist coverage, and high pharmacist coverage. *** $p<0.01$; ** $p<0.05$; * $p<0.10$.

Table 3: Estimated Impacts of Pneumonia Exposure in Infancy on Adult Outcomes by Demographic Group

	Schooling (1)	log(Family Income) (2)	Employment (3)	Work Limiting Disability (4)
Panel A: White Men				
Post Sulfa × Base Exposure	0.402***	0.0707***	0.0100	-0.0122***
	(0.0966)	(0.0165)	(0.00758)	(0.00385)
FWER p-value	[0.003]	[0.003]	[0.217]	[0.019]
Effect size for an interquartile shift in base exposure	0.113 years	1.988 %	0.282 pp	-0.342 pp
Observations	635,279	1,829,870	1,863,597	1,863,597
Panel B: White Women				
Post Sulfa × Base Exposure	0.190*	0.0352***	0.0269***	-0.00910*
	(0.0971)	(0.0125)	(0.00749)	(0.00460)
FWER p-value	[0.080]	[0.050]	[0.010]	[0.138]
Effect size for an interquartile shift in base exposure	0.0535 years	0.989 %	0.757 pp	-0.256 pp
Observations	649,412	1,903,470	1,933,966	1,933,966
Panel C: Black Men				
Post Sulfa × Base Exposure	0.210	0.132***	0.0599*	-0.00314
	(0.217)	(0.0374)	(0.0321)	(0.0139)
FWER p-value	[0.587]	[0.019]	[0.209]	[0.847]
Effect size for an interquartile shift in base exposure	0.0590 years	3.710 %	1.685 pp	-0.0884 pp
Observations	66,597	164,497	173,715	173,715
Panel D: Black Women				
Post Sulfa × Base Exposure	-0.810***	-0.0860	-0.0756***	-0.0102
	(0.221)	(0.0520)	(0.0230)	(0.0144)
FWER p-value	[0.012]	[0.208]	[0.015]	[0.488]
Effect size for an interquartile shift in base exposure	-0.228 years	-2.417 %	-2.125 pp	-0.287 pp
Observations	82,649	212,391	219,355	219,355

Notes: Refer to notes to Table 1. Identical models are estimated, however here for demographic-specific (race× gender) groups. Each panel consists of a separate set of regressions for the group indicated in Panel titles. All other details follow those described in Table 1. *** p<0.01; ** p<0.05; * p<0.10.

Figure 7: Trends and convergence in pneumonia and influenza mortality rates: By race in the US South



(a) Trends over time

(b) Convergence in mortality

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 Table B2 demonstrates significant post-1937 trend breaks in all-age and infant pneumonia mortality for both Black and white populations in the South. The black trend breaks are larger in absolute magnitude.

Table 4: Gradients in Long-Run Impacts of Infant Pneumonia Exposure by Measures of Systemic Discrimination – Black Men and Women

	Gradient: Historical Fraction of Enslaved People				Gradient: Number of Jim Crow Laws			
	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Black Men								
Post Sulfa × Base Exposure	1.459***	0.327***	0.159***	-0.0750***	0.831**	0.252***	0.185***	-0.0606***
	(0.261)	(0.0756)	(0.0342)	(0.0247)	(0.323)	(0.0503)	(0.0383)	(0.0164)
FWER p-value	[0.019]	[0.073]	[0.072]	[0.129]	[0.062]	[0.018]	[0.019]	[0.049]
Post Sulfa × Base Exposure × Discrimination Proxy	-3.957***	-0.602***	-0.305***	0.238***	-0.0266***	-0.00497***	-0.00387***	0.00229***
	(0.753)	(0.206)	(0.0968)	(0.0643)	(0.00948)	(0.00175)	(0.00107)	(0.000463)
FWER p-value	[0.022]	[0.024]	[0.034]	[0.058]	[0.060]	[0.087]	[0.036]	[0.025]
Effect size at bottom decile of discrimination proxy	0.554 years	11.37 %	5.570 pp	-2.974 pp	0.353 years	9.306 %	6.920 pp	-2.726 pp
Effect size at top decile of discrimination proxy	0.0645 years	3.934 %	1.793 pp	-0.0292 pp	0.0231 years	3.160 %	2.135 pp	0.106 pp
Observations	65,266	161,583	170,601	170,601	66,597	164,497	173,715	173,715
Panel B: Black Women								
Post Sulfa × Base Exposure	0.421	0.0391	-0.00887	-0.109***	-0.229	0.0115	-0.0396	-0.0644**
	(0.294)	(0.0984)	(0.0312)	(0.0374)	(0.320)	(0.0755)	(0.0353)	(0.0275)
FWER p-value	[0.343]	[0.870]	[0.785]	[0.014]	[0.662]	[0.888]	[0.538]	[0.083]
Post Sulfa × Base Exposure × Discrimination Proxy	-3.154***	-0.473*	-0.323***	0.339**	-0.0199**	-0.00301	-0.00107	0.00234**
	(0.819)	(0.257)	(0.0922)	(0.136)	(0.00859)	(0.00240)	(0.000967)	(0.000975)
FWER p-value	[0.001]	[0.0657]	[0.002]	[0.025]	[0.066]	[0.537]	[0.537]	[0.066]
Effect size at bottom decile of discrimination proxy	0.233 years	2.814 %	0.923 pp	-4.304 pp	0.0244 years	1.667 %	-0.636 pp	-2.855 pp
Effect size at top decile of discrimination proxy	-0.157 years	-3.034 %	-3.075 pp	-0.109 pp	-0.222 years	-2.053 %	-1.958 pp	0.0374 pp
Observations	81,074	208,673	215,520	215,520	82,649	212,391	219,355	219,355

Notes: Each column represents a separate regression of the outcome indicated in column headers following specification (3). KIn columns (1)-(4) the historical fraction of the Black population which was enslaved is used as a proxy for systemic discrimination, while columns (5)-(8) are identical, however use the number of discriminatory Jim Crow laws as a proxy of systemic discrimination. Both measures are centered on zero such that Post sulfa × Base Exposure is cast as the effect at the mean of these variables. Full details of the specification and baseline covariates are discussed in Section 1. The estimation sample consists of all Black men (panel A) and women (panel B) born between 1930 and 1943 (inclusive) observed in census microdata. Coefficients are presented along with standard errors clustered by birth state in parentheses. Multiple comparison adjusted p-values, which control the family-wise error rate following (Romano and Wolf, 2005) are provided in square brackets based on 10,000 bootstrap replicates. The estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) on the outcome in each column are provided as the reported effect size at the foot of the table both in areas with low values for proxies of systemic discrimination, and high values for these proxies. *** p<0.01; ** p<0.05; * p<0.10.

Online Appendices for

The Long Run Economic Effects of Medical Innovation and the Role of Opportunities

Sonia Bhalotra, Damian Clarke, Atheendar Venkataramani

Not for print

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A Data Appendix

A.1 Data Sources and Variables

A.1.1 Outcome variables

Source Outcome data were drawn 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 (Ruggles et al., 2024). We collect data on eight indicators of human capital. Four of these measures are included as principal outcomes documented in the main body of the paper measuring educational attainment (years of schooling), labor market outcomes (family income and employment), and health (an indicator or work limiting disability). Four further factors which also capture educational and health measures are included as alternative outcomes in Appendix results. These are indicators of high school and college completion, an an indicator of poverty and an indicator of cognitive disability. In additional analyses we consider non-market outcomes such as marital status and fertility measures.

Census coverage A description of coverage by variable is provided in Table A1. This Table indicates both the coverage of particular variables by census waves, as well as the samples in demographic groups (Black and White males and females) for which non-missing information is recorded. With the exception of educational measures, analysis samples for principal models consist of all available data. For measures of income, poverty and employment, we pool data from the three census samples. Work limiting disability is available in all three censuses, while cognitive disability is 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 data given that years of schooling seldom change after an individual reaches their late 30s, which is 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 (refer to robustness checks discussion in Appendix E). 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 (this is documented in robustness checks, Appendix E).

Data descriptions – Principal Outcomes Specifics of the definition and/or construction of the outcome variables are as follows. We first define the four principal variables considered in the body of the paper, and then list the related variables which are presented in Appendix analyses (Appendix F.1):

1. *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). This is used to record completed years of schooling for all individuals.
2. *Log total family income* (*FTOTINC* in IPUMS): Nominal total pre-tax monetary 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 analyze family income given that this indicator is also available for all women, regardless of whether they are working or not.

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

4. *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.

5. *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 and 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.

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

7. *Cognitive disability* (*DIFFREM* in IPUMS): Denotes whether an individual has difficulty with "learning, remembering, or concentrating" due to a physical, mental, or emotional condition.

Data descriptions – Income Distribution In Appendix results, we additionally examine a series of indicators which take 1 if log(family income) is contained within specific percentiles of the pre-1937 income distribution. For percentiles $p \in \{0-1, 1-5, 5-10, 10-25, 25-50, 50-75, 75-90, 90-95, 95-99, 99-100\}$ these measures are constructed as $\mathbb{1}\{\text{logged family income} \in \text{Baseline Income Percentile } p\}$, where $\mathbb{1}$ refers to the indicator function. Baseline income percentiles refer to the pre-1937 earnings distribution observed from each census microdata file.

Data descriptions – Non-market outcomes We consider a number of non-market outcomes consisting of an individual's relationship and fertility measures. These variables are defined as follows:

1. *Number of children ever born* (*CHBORN* in IPUMS): Recorded for women only as the total number of children ever born. This is reported in a consistent way in 1980 and 1990 censuses (not available thereafter). We work with measures from 1990 census only, given that this will capture completed fertility, as it is measured between an individual's late 40s and their early 60s (1930-1943 birth cohort ages at 1990).

2. *Any child and Number of children conditional on having children*: These were computed from CHBORN as *Any child*=1 if 1 or more children were reported born, and 0 if no children were reported born. *Number of children conditional on having children* is set equal to the number of children ever born for all individuals with at least 1 child.

3. *Ever married* (*MARST* in IPUMS): Indicator variable = 1 if individual ever married, including cases where the person is now a widow, divorced, or separated. Measures are available in each of the three Census waves, however for comparability with fertility measures discussed above, analysis is conducted with measures at 1990 when individuals are aged 47-60.

4. *Age at marriage* (*AGEMARR* in IPUMS): Age at first marriage. This is the age at first marriage for all individuals who have ever been married. This is recorded only for the 1980 census.

Summary Statistics Summary statistics for all outcome variables are provided in Table A2 for the entire period under study (years 1930-1943) across all individuals. The top panel of Table A3 documents means and standard deviations of each of the 8 principal outcomes by gender and race-specific cells. Table A4 documents identical values for non-market outcomes for women, and women by race.

A.1.2 Mortality data

Source and description – mortality rates 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 and Hetzel, 1968; Linder and Grove, 1947). These data were used to extend the data series collected by Grant Miller (https://www.nber.org/research/data/vital-statistics-deaths-historical-1900-1936) who provides transcribed mortality rates for all diseases from 1900 to 1936, and Seema Jayachandran, Adriana Lleras-Muney, and Kimberly Smith (Jayachandran et al., 2010), who provide transcribed mortality rates for a sub-set of diseases up to 1950. We used these data to create birth state-specific pre-sulfa-drug era mortality 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 E we discuss issues with measurement of exposure to pneumonia and present tests of robustness to alternative measures.

Source and description – race-specific mortality rates Race-specific state mortality data for the 18 states where Black Americans comprised of over 10% of the population were generously provided by Adriana Lleras-Muney. The states in question consist of: Alabama, Arkansas, Delaware, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, Missouri, New Jersey, Oklahoma, South Carolina, North Carolina, Tennessee, Texas, Virginia and West Virginia. They were originally obtained from from yearly US Vital Statistics volumes (http://www.cdc.gov/nchs/products/vsus.htm). These data are analyzed in Appendix B and D.

Summary Statistics Summary statistics for baseline mortality rates as matched to census microdata are provided in Table A2 for the entire period under study (years 1930-1943) across all individuals. The bottom panel of Table A3 documents means and standard deviations of baseline state mortality rates by cause.

A.1.3 State level socioeconomic, infrastructure, and reporting variables

We examine sensitivity to the inclusion of a number state-level control variables. State time series data on logged state per capita income were downloaded from the Bureau of Economic Analysis website, publicly available at: 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 (https://adriana-llerasmuney.squarespace.com/data). 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 (Sylla et al., 2006). 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 (and document robustness to estimates with and without these controls). All values are based on pre-1937 measures.

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 at more length in Appendix D where we discuss concerns relating to measurement as potential drivers of documented effects.

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. The distributional and geographic variation of this measure is plotted in Figure A2. In theory, pharmacist coverage is a relevant measure for access to sulfa drugs given that drugs were initially available directly from pharmacists without prescription (Lesch, 2007). However, we also generate similar measures of availability of physicians per capita which will also mediate access to sulfa drugs. The distribution of these measures are similar (Figure A2).

A.1.4 Systemic discrimination

As a measure of systemic discrimination we use a measure from Nunn (2008), which is the state-specific share of slaves in the population in 1860. This measure is available for 38 states, and is zero for nearly all non-southern states. Densities and geographic variation of this measure is plotted in Figure A1. As an alternative measure we use the number of Jim Crow laws. These laws are classified by Althoff and Reichardt (2024) as discriminatory race-related state laws from a classification of around 800 laws, digitized from a number of sources including Murray's 1950 "States' Laws on Race and Color". In Appendix D.3 when discussing measurement, we discuss these variables' ability to capture systemic discrimination, and document that they are highly correlated with other (contemporaneous) measures of institutionalized discrimination at the time when sulfa drugs became available, such as black/white school ratios, and black/white wage return ratios. See also discussion in Althoff and Reichardt (2024) for a further validation of these laws as a measure of systemic discrimination.

In certain tests we consider results only within the north or the south of the United States. We define Southern states as 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.

A.2 Descriptive Statistics and Trends

Table A1: Outcome Variable Coverage by Demographic Group and Census Waves

Variables	Census Wave 1980		1990		2000	
Panel A: Market Outcomes						
Education Measures						
Schooling	W/M = 647,099	B/M = 67,906				
	W/W = 659,851	B/W = 83,809				
High School	W/M = 647,099	B/M = 67,906				
	W/W = 659,851	B/W = 83,809				
College	W/M = 647,099	B/M = 67,906				
	W/W = 659,851	B/W = 83,809				
Labour Market Measures						
Income	W/M = 636,193	B/M = 64,594	W/M = 627,870	B/M = 52,139	W/M = 577,391	B/M = 48,913
	W/W = 651,429	B/W = 81,799	W/W = 656,890	B/W = 68,035	W/W = 605,522	B/W = 63,692
Employment	W/M = 647,099	B/M = 67,906	W/M = 637,580	B/M = 54,724	W/M = 590,738	B/M = 52,394
	W/W = 659,851	B/W = 83,809	W/W = 665,908	B/W = 70,088	W/W = 618,646	B/W = 66,618
Poverty	W/M = 647,099	B/M = 67,906	W/M = 637,580	B/M = 54,724	W/M = 590,738	B/M = 52,394
	W/W = 659,851	B/W = 83,809	W/W = 665,908	B/W = 70,088	W/W = 618,646	B/W = 66,618
Health Measures						
Work-Limiting Disability	W/M = 647,099	B/M = 67,906	W/M = 637,580	B/M = 54,724	W/M = 590,738	B/M = 52,394
	W/W = 659,851	B/W = 83,809	W/W = 665,908	B/W = 70,088	W/W = 618,646	B/W = 66,618
Cognitive Disability					W/M = 590,738	B/M = 52,394
					W/W = 618,646	B/W = 66,618
Panel B: Non-Market Outcomes						
Family Formation Measures						
Ever Married			W/W = 665,908	B/W = 70,088		
Age at Marriage	W/W = 629,203	B/W = 74,606				
Fertility Measures						
# Children Ever Born			W/W = 595,340	B/W = 62,285		
Any Children			W/W = 595,340	B/W = 62,285		
# Childen|Any Children			W/W = 531,715	B/W = 53,149		

Notes: Estimation samples for each outcome variable by census wave are indicated in each cell. Cells coloured in blue indicate the variable is available in a given census wave. Cells coloured white indicate the variable is not available. For each variable, the number of observations is displayed for white men (W/M), Black men (B/M), white women (W/W) and Black women (B/W). In all principal models, pooled samples are used covering all census waves (by demographic group). Summary statistics by group are provided in Appendix Tables A3 (market outcomes), and A4 (non-market outcomes).

Table A2: Descriptive Statistics from Census Microdata

	Obs.	Mean	Std. Dev.	Min.	Max.
Education (years)	1,490,793	12.3	2.90	6.40	17.5
High school	1,490,793	0.74	0.44	0	1
College	1,490,793	0.33	0.47	0	1
Employed	4,354,513	0.65	0.48	0	1
log(family income)	4,270,463	10.4	0.92	0	14.2
Below poverty line	4,354,513	0.20	0.40	0	1
Work-limiting disability	4,354,513	0.13	0.33	0	1
Cognitive disability	1,380,006	0.057	0.23	0	1
Ever married	1,483,714	0.95	0.22	0	1
Age at marriage	1,403,320	22.0	4.78	12	50
# Children ever born	684,087	2.20	1.71	0	11
Any child	684,087	0.89	0.31	0	1
# Children | any child	608,111	2.47	1.61	1	11
Base pneumonia & influenza mortality	4,329,176	1.04	0.16	0.75	1.57
Female	4,354,513	0.51	0.50	0	1
Black	4,354,513	0.100	0.30	0	1
Birth year	4,354,513	1936.9	4.11	1930	1943
Pharmacists per 1,000 population	4,349,819	0.74	0.24	0	1.52
Historical slave fraction	3,994,288	0.13	0.18	0	0.57
Jim Crow laws	4,354,513	16.0	24.3	0	97

Notes: Descriptive statistics are provided for individual-level outcomes as measured in 1980, 1990 or 2000 censuses, matched with exposures to pneumonia in infancy, as well as state-level characteristics considered as long-run gradients. Full details regarding samples and data definitions are provided in Section A.1.

Table A3: Descriptive Statistics

Census Microdata

	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)	12.54	(3.13)	12.70	(3.10)	11.11	(3.03)	12.14	(2.61)	12.25	(2.56)	11.31	(2.78)
High School (= 1)	0.76	(0.43)	0.78	(0.41)	0.56	(0.50)	0.75	(0.43)	0.78	(0.42)	0.57	(0.49)
College (= 1)	0.39	(0.49)	0.41	(0.49)	0.23	(0.42)	0.29	(0.46)	0.30	(0.46)	0.22	(0.42)
ln(Family Income)	10.43	(0.89)	10.47	(0.86)	10.01	(1.03)	10.28	(0.93)	10.34	(0.90)	9.80	(1.09)
Below Poverty	0.18	(0.39)	0.16	(0.37)	0.37	(0.48)	0.23	(0.42)	0.20	(0.40)	0.47	(0.50)
Employed	0.75	(0.43)	0.76	(0.43)	0.64	(0.48)	0.55	(0.50)	0.55	(0.50)	0.54	(0.50)
Work Limiting Disability	0.13	(0.34)	0.12	(0.33)	0.19	(0.39)	0.12	(0.32)	0.11	(0.31)	0.19	(0.39)
Cognitive Disability	0.06	(0.24)	0.06	(0.23)	0.10	(0.30)	0.05	(0.23)	0.05	(0.22)	0.10	(0.30)

Birth State Baseline Mortality Rates (per 1000, N = 48 States)			**Birth State × Birth Year Socioeconomic Variables (N = 672 States×Year)**		
Pneumonia	1.06	(0.19)	log(Income per capita)	6.24	(0.53)
Tuberculosis	0.68	(0.39)	log(Hospitals per 1,000)	-2.83	(0.45)
Diarrhea	8.77	(6.02)	log(Physicians per 1,000)	0.05	(0.52)
Heart Disease	2.01	(0.61)	log(Hospitals per 1,000)	0.70	(0.66)
Maternal Mortality	0.66	(0.14)	log(Educational spending per capita)	4.14	(0.87)
Malaria	0.07	(0.14)			

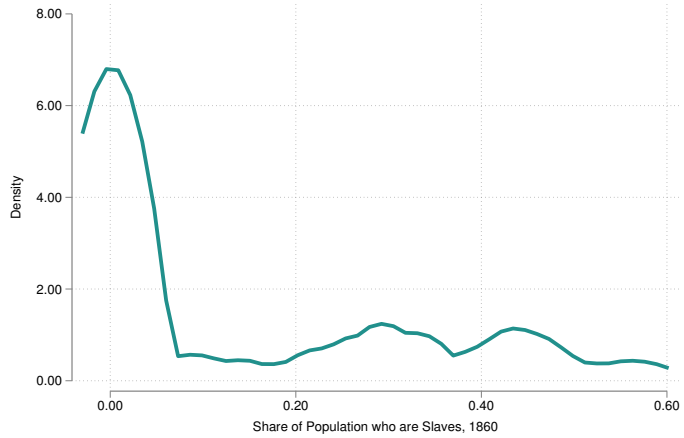
Notes: Summary statistics are presented for all principal outcome variables from census microdata by race and by sex in top panel. Bottom panel documents state-level control measures and baseline (pre-sulfa) mortality rates for the 48 mainland states covering the period 1930–1937. Census microdata summary statistics are based on all available measures in 1980-2000 census waves. Refer to Table A1 for a full description of estimation samples and variable availability by census wave.

Table A4: Descriptive Statistics – Non-market outcomes

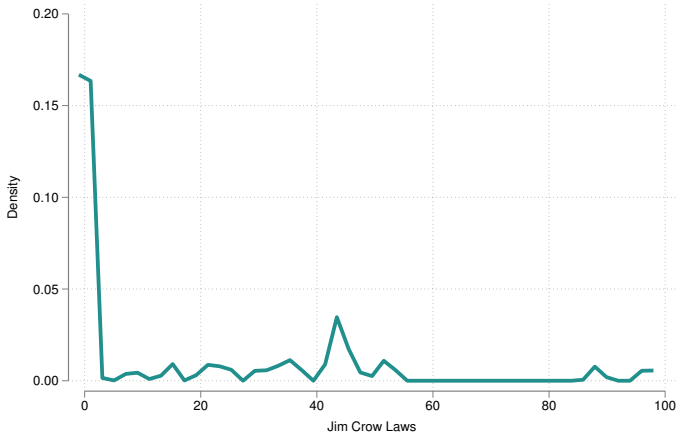
Census Microdata						
	Women		White Women		Black Women	
	Mean	SD	Mean	SD	Mean	SD
Ever married (= 1)	0.96	(0.21)	0.96	(0.19)	0.90	(0.30)
Age at Marriage	20.64	(4.33)	20.56	(4.15)	21.25	(5.55)
# Children ever born	2.20	(1.71)	2.12	(1.58)	2.93	(2.45)
Any child (= 1)	0.89	(0.31)	0.89	(0.31)	0.85	(0.35)
# Children | any child	2.47	(1.61)	2.37	(1.49)	3.43	(2.31)

Notes: Summary statistics are presented for all non-market outcome variables from census microdata. These are presented for women only, as fertility and family formation outcomes are considered for these individuals. Summary statistics are presented by race for black and white women based on all available measures of these variables in the 1980-2000 census waves. Refer to Table A1 for a full description of estimation samples and variable availability by census wave.

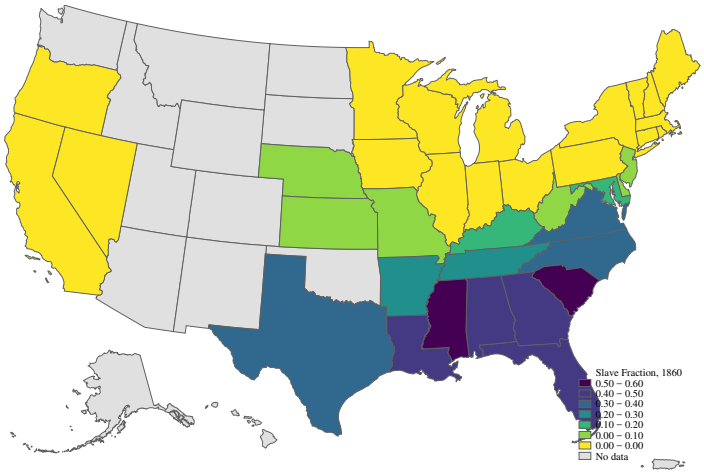
Figure A1: Historical Enslaved Population (1860) and Jim Crow Laws by State



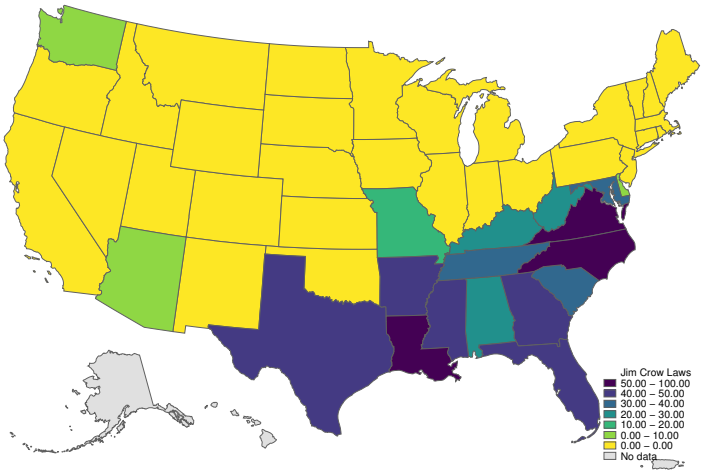
(a) Distribution of enslaved population



(b) Distribution of Jim Crow laws



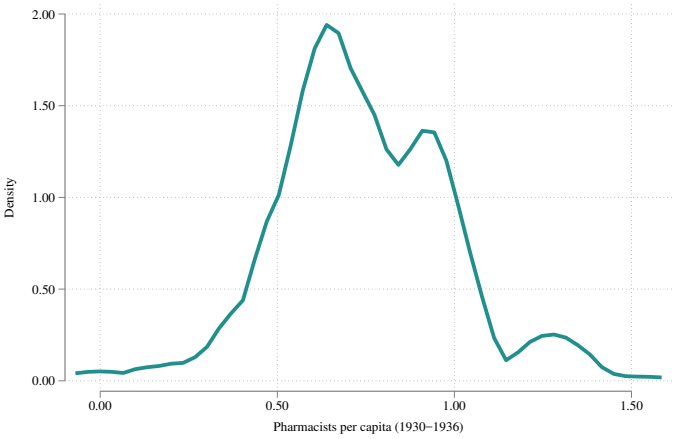
(c) Geographical Dispersion of enslaved population



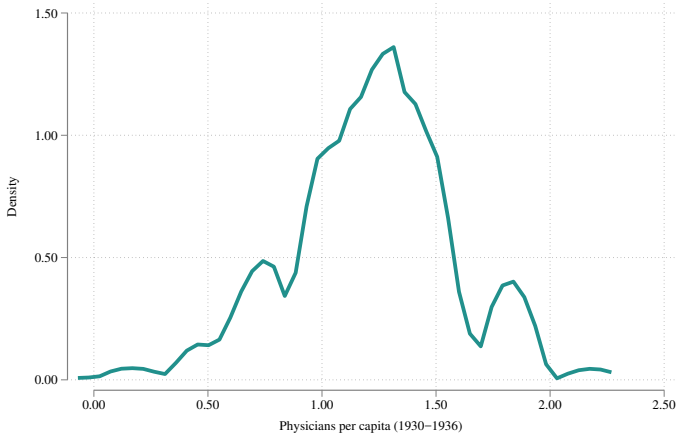
(d) Geographical Dispersion of Jim Crow laws

Notes: Plots display the proportion of each state populations which was formed of slaves in 1860, and the number of Jim Crow laws passed by each state. Source: Nunn (2008) (enslaved population) and Althoff and Reichardt (2024) (Jim Crow laws).

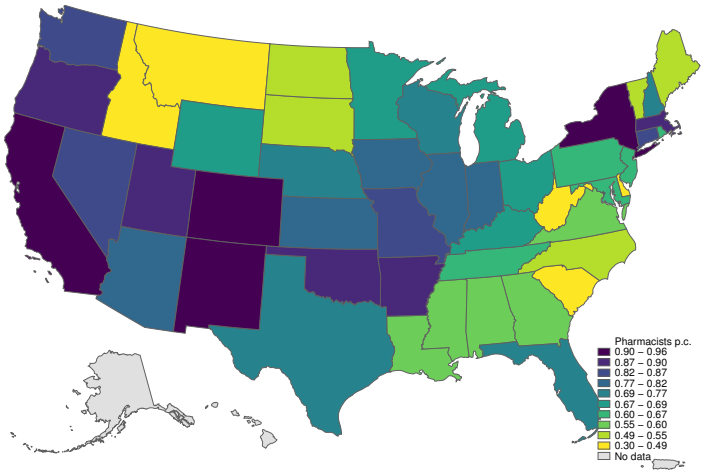
Figure A2: Historical Shares of Physicians and Pharmacists per capita



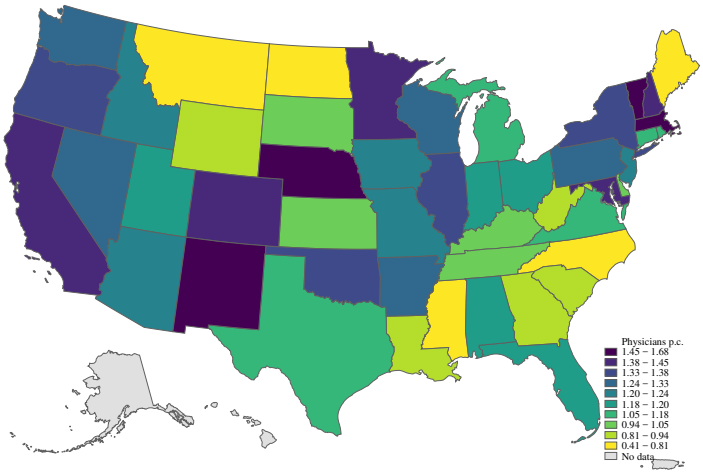
(a) Distribution of Pharmacists per capita



(b) Distribution of Physicians per capita



(c) Geographical Dispersion of Pharmacists



(d) Geographical Dispersion of Physicians

Notes: Figures plot state-level averages of pharmacists per capita (panels (a) and (c)), and physicians per capita (panels (b) and (d)), in counties where greater than 10% of the population is black, each re-expressed per 100,000 population. Top panels present densities while bottom panels present state-level averages. Measures are generated from occupational codings in 1940 IPUMS microdata.

B First-stage Results of sulfa on Mortality: Diffusion, Structural Break, and Convergence

B.1 Access to sulfa Drugs and Mortality Declines

Mortality Declines by Group We show in the paper that Black men and women who were born in an environment marked by institutionalized discrimination 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 Black Americans, even in states with higher degrees of systemic discrimination, and that the constriction of opportunities as a result of systemic discrimination played an important part in translation of first stage (pneumonia mortality) to second stage impacts (adult outcomes). We summarize evidence that supports our claim that a strong first stage—*i.e.*, Black infants had access to sulfa drugs and experienced reductions in pneumonia morbidity and mortality as a result of it—exists here.

Figure 7 of the main paper demonstrates that both Black and white individuals in the Southern states experienced declines in all-age pneumonia mortality starting in 1937, with the absolute decline being larger among Black individuals. In Table B1 of this Appendix, we reproduce "first stage" regressions using all-age as well as infant pneumonia mortality rates, and using the logarithm of the mortality rate and its level, and in Table B2 we break these results down by race and region. With the level and logarithm of both all-age and infant mortality, we find statistically significant trend breaks for Black and white individuals in the Southern as well as in the non-Southern regions. The level (absolute) breaks tend to be larger for Black people than for white, consistent with Figure 7, while the log (relative) breaks are similar, or slightly smaller. Regardless, the results demonstrate that all-age and infant pneumonia mortality declined significantly after 1937 for Black persons, including in the US South.[52] In Figure 7b of the main paper, we also demonstrate that post-1937, there was convergence in pneumonia mortality rates, even for Southern Black Americans.

This finding is consistent with other evidence from this era. For example, Boustan and Margo (2014) demonstrate large improvements in mortality for Black relative to white infants in the period 1920-1945. They attribute this to improvements in public health, specific disease eradication efforts, and secular improvements in living standards for Black families. 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 Black Americans than for whites.

While Tables B1-B2 demonstrate 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 Black Americans than for the rest of the population on account of segregation in access to health care. We investigated this further as follows.

First, the results in Table 4 of the paper serve as evidence that Black Americans in areas with greater systemic discrimination 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 systemic discrimination experienced positive gains across several outcomes, even if these gains were smaller than among Black individuals in states with less systemic discrimination. 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

[52] Table B2 shows that the absolute declines in pneumonia mortality were larger, though noisily estimated, for Northern Black individuals than for Southern Black or white individuals; but that the declines for Southern Black Americans were larger than for Southern whites.

seems unlikely that income constraints limited access for Black families.

Third, we found other evidence that Southern Black individuals accessed health innovations in this era. For example, Hollingsworth et al. (2024) find large reductions in Black infant mortality as a result of philanthropic hospital modernization and expansion in the 1920s in North Carolina. They also show that one mechanism by which these large improvements accrued was through Black infants accessing sulfa drugs in the late 1930s. In addition, Fung and Robles (2016) 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 (Alsan and Wanamaker, 2017), a clear indication that treatment was readily available in the community at the time.

Fourth, we constructed additional evidence that Black men and women were able to access antibiotics for non-life threatening infections by studying trends in rheumatic fever by race, documented in Figure B6. 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*.[53] These infections are usually not life threatening and include pharyngitis (popularly known as "Strep throat"), tonsillitis, and scarlet fever. If Southern Black men and women 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 this group, consistent with timely and efficacious treatment of streptococcal infections. Figure B6 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 Jr, 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 Black relative to white individuals 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*.

We discuss in the remaining sections of this Appendix additional evidence on the diffusion of sulfa drugs in the population, as well as convergence in mortality over time.

Sulfa Exposure and The Timing of Mortality Declines Trend breaks in mortality are clearly appreciable between years 1937 and 1938 in Figure 2. In Figure B1, this decline is visibly clear when inspecting trends in mortality at the level of each state over the period 1927–1950. We can formally test where the largest trend break occurs, which is tested by regressing year-over-year mortality changes on single year fixed effects in a regression with Newey-West standard errors, namely:

$$\Delta \ln(Mortality)_t = \alpha + \beta \mathbb{1}\{Year = j\}_t + \varepsilon_t$$

for each year from 1934,…,1943, and then conducting an F-test on the significance of β for each year and state. In Figure B2 we plot the histogram of years j found to have the largest state-level trend break for each state, overwhelmingly seeing that these fall in 1937 and 1938, both for white and Black mortality rates.

To define exposure to sulfa drugs in census microdata, we use an individual's year of birth. The year of birth provided in census microdata (*BIRTHYR* in IPUMS) is calculated based off the age that an individual reports in the census as the census year minus their age. As censuses are enumerated on April 1st of 1980,

[53] 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, 1990).

1990, and 2000, IPUMS documentation notes that this results in the calculated year of birth being 1 year later than the actual year of birth for individuals whose birth year falls after April 1st. However, in the 1980 census, IPUMS documentation states that an individual's year of birth is "further refined using Birth quarter (BIRTHQTR)."

In practice, given that the census occurs on the first day of a quarter, for the 1980 census this provides an exact mapping to birth year. However, for the 1990 and 2000 censuses, the IPUMS definition will result in a 1 year lag in birth year for around 75% of individuals. Given this, we correct the birth year for 1990 and 2000 censuses by subtracting 1 from the birth year, so that the birth year reported for each individual is correct in the majority of the cases.

This implies that for 1990 and 2000 censuses, around 25% of individuals whose birth year is recorded by us as 1936 were actually born in the first three months of 1937, and this would similarly be the case for other years. Under the assumption that sulfa became available in 1937, this decision is innocuous for individuals born from 1937 onward, as regardless of whether they were born in 1937 or 1938, they would have been exposed to sulfa from birth. For individuals recorded as being born in 1936, around one quarter of individuals would have been born in 1937, and hence exposed to sulfa from birth. For individuals recorded as being born in 1935, around one quarter would have been born in the first 3 months of 1936, and so been exposed to sulfa at the end of their first year of life. Our definitions of exposure to sulfa in the paper are coded as $\text{Post}_t = \mathbb{1}\{\text{Birth Year}_t \geq 1937\}$, and so will misclassify a very small portion of individuals (those born in the first three months of 1937 in two censuses) as un-exposed, however, the alternative is to misclassify a larger portion of individuals (those born in the last 9 months of 1936 in the two censuses) as exposed. In event study models where we examine effects by year we can inspect these coefficients directly, and we also implement robustness checks where we use only individuals exposed at an older age as the baseline reference group in event studies, or use 1935 births as our baseline reference group.

B.2 Diffusion of sulfa Drugs

We broadly observe declines in mortality with the arrival of sulfa drugs, suggestive of wide-spread diffusion and take up. Descriptively, Figure B3 documents diffusion of sulfa drugs as evidenced by mortality declines in states based on pre-sulfa state-level income per capita, the level of urbanization of the state, the literacy of residents of the state, the proportion of the state population who is not white, as well as in areas with greater pharmacist and physician coverage. These descriptive declines suggest that sulfa was widely available, which lines up with the evidence discussed in Appendix B.1 above.

Table B3 tests for differential diffusion in terms of state-level characteristics. Specifically, we estimate:

$$\begin{aligned}\ln(\text{Pneumonia Mortality})_{st} \quad = \quad & \beta_0 + \beta_1 \text{Year}_t + \beta_2 X_s + \beta_3 \text{Post 1937}_t + \beta_4 \text{Post sulfa}_t \times X_s \\ & + \beta_5 \text{Post 1937}_t \times \text{Year}_t + \beta_6 \text{Post 1937}_t \times \text{Year}_t \times X_s + \eta_{st},\end{aligned} \tag{4}$$

using state by year mortality data described in Appendix A.1.2. Here, β_3 captures immediate year-over-year declines in mortality following the arrival of sulfa, while β_5 captures any trend break which gradually emerges during the post-sulfa period. Our parameters of interest, β_3 and β_5 capture whether proportional mortality declines are larger in areas with specific state-level characteristics X_s explored in Figure B3, or whether trend breaks vary by these characteristics. In tabular output we present estimates of $\beta_3, \beta_4, \beta_5$ and β_6 in the interests of simplicity. In each case, X_s is cast as a Z-score for ease of comparability.

As observed in Figure B3, results from Table B3 (all mortality) and Table B4 (race-specific mortality) evidence large mortality declines regardless of state-level characteristics. While there are clear gradients in

terms of state level characteristics X_s in immediate declines and/or trend breaks, we present marginal effects at percentiles 10 and 90 of the characteristic X_s in table footer. In panel A (which focuses on mortality in all states), marginal effects suggest declines of anywhere between 17% and 35% at extreme percentiles of state characteristics immediately. By year 3, declines are observed to be broadly consistent across characteristics, at most ranging from around 42%-61% when considering the 90th and 10th percentile of states based on the share of urban population. Other characteristics show smaller diffusion gradients by year 3, with slightly larger proportional declines observed in states with lower income levels, lower rates of pharmacists and physicians per capita and higher rates of historically enslaved populations. In panel B we observe broadly consistent results when considering mortality rates in Southern states only, and in Table B4 present results for black and white mortality only. Regardless of the sample and state-level variable considered, we see large declines in mortality.

While evidencing broad diffusion of sulfa drugs, this is a descriptive exercise given that declines in mortality are larger in areas with higher baseline mortality. If state level characteristics also correlated with pre-sulfa incidence, we would expect this to be reflected in post-sulfa mortality declines. In the following section, we consider a more direct test of differential access to sulfa drugs by considering convergence in mortality, and whether convergence varies by state-level mortality.

B.3 Convergence in Mortality Rates Following sulfa

A richer test of how drugs diffused across the population and whether there is evidence of differential access of sulfa historically which may explain long-term patterns, considers convergence in mortality rates over time, and whether this convergence is mediated by cross-state factors. Specifically, we analyzed differences in cross-state convergence in pneumonia mortality rates by the same set of variables examined above. This test is based on the following model:

$$\begin{aligned}\text{Pneumonia Mortality}_{st} \quad = \quad & \alpha_0 + \alpha_1 \text{Post 1937}_t \times \text{Base Pneumonia}_s + \alpha_2 \text{Post 1937}_t \times X_s \\ & + \alpha_3 \text{Post 1937}_t \times \text{Base Pneumonia} \times X_s + \mu_s + \phi_t + \upsilon_{st}, \qquad (5)\end{aligned}$$

where X_s refers to the same characteristics as considered in (4). This specification is meaningful because it addresses the intensive margin of diffusion—the trend break in mortality documented in the previous sub-section simply assesses whether sulfa drugs were available in a given year in a given state, while here differences in take-up rates conditional on drug availability are detectable as faster vs. slower convergence. The estimated specification is a triple difference describing the first stage of our long-run effects analysis, which gives it conceptual clarity.

Evidence of convergence in mortality rates is presented in Table B6. Here, we consider convergence in mortality rates without interaction with characteristics X_s, and consistently see evidence of convergence in mortality rates, with rates declining more in areas with high baseline mortality. These results hold conditional on baseline disease and SES controls, as well as in specification with census division by year fixed effects. We observe evidence of convergence when considering mortality in all states between 1927-1943, as well as when considering only mortality among white and Black Americans in the 18 states for which race-specific mortality data is available. Along with standard errors clustered by state, we present 95% confidence intervals based on a wild clustered bootstrap, which consistently allows us to reject null effects.

In Table B5 we present analyses of whether cross-state convergence is mediated by factors X indicated in column headings. In panel A, we do not see evidence of convergence being greater in states with higher literacy rates, higher incomes, higher urbanization, or greater historical discrimination as proxied by histor-

ical slave shares. The only statistically significant interaction is observed when considering the coverage of pharmacists and physicians per capita. Convergence in pneumonia mortality rates after the introduction of sulfa drugs was increasing in the density of pharmacists. This is consistent with John Lesch's account of widespread uptake of sulfa drugs in the community through direct purchase at pharmacies.

We can further examine this convergence to test whether cross state convergence depends on characteristics X_s precisely following the arrival of sulfa. To do so, we effectively implement event-study analogues of (5) where rather than a single Post 1937 indicator, we test for convergence in each pre and post-reform years. As in main event studies in the paper, we follow a Miller (2023) proposal of constraining all pre-period coefficients to average 0 rather than omitting a single arbitrary based period. Namely, we estimate:

$$\begin{aligned}\text{Pneumonia Mortality}_{st} \quad = \quad & \alpha_0 + \sum_{j=1930}^{1943} \alpha_1^j \mathbb{1}\{\text{Year}_t = j\} \times \text{Base Pneumonia}_s \\ & + \sum_{j=1930}^{1943} \alpha_2^j \mathbb{1}\{\text{Year}_t = j\} \times \text{Base Pneumonia} \times X_s \\ & + \sum_{j=1930}^{1943} \alpha_3^j \mathbb{1}\{\text{Year}_t = j\} \times X_s + \mu_s + \phi_t + \upsilon_{st}, \end{aligned} \tag{6}$$

where all details follow those in (6). Results are presented in Figure B4, where we present marginal effects estimated at percentile 10 and 90 of each characteristic X_s. Here we observe quite clear evidence of convergence, with mortality declines occurring more sharply in areas with higher pre-1937 mortality specifically after the arrival of sulfa. Appreciable gradients are observed in the coverage of physicians and pharmacists, but not across the range of other variables considered. This gradient in access owing to pharmacists is shown to map into long-term outcomes in the body of the paper (Table 2), and similarly this is clear if long-term event study specifications are exampled, as documented in Figure B5.

While we do not observe evidence of gradients in mortality in terms of systemic discrimination as proxied by the historical slave share of a state's population and the number of Jim Crow laws passed in each state, arguments that long term outcomes among black populations depend on gradients in systemic discrimination may also reflect differential rates of uptake or access to sulfa historically among this group. In Table B7 we test for this, examining whether initial convergence in mortality rates among black populations varies by the historical slave share and the number of Jim Crow laws in these states. We observe some weak evidence that mortality convergence among Black Americans in the south may have been larger in areas with a lower historical share of enslaved persons (Table B6). Such patterns are observed *only* in specifications which weight by state population (Panel C, column 4), and are imprecisely estimated. However they do point to potential 'first stage' differences in access. In each case, the slave share has been re-standardized as a Z-score, and so the effect size in panel C suggests that a 1 standard deviation higher historical slave share (0.15, when weighted by state population, or 0.18 if unweighted), results in a rate of convergence which is 0.129 higher than the baseline rate of -0.467. This value of 0.15 is quite large, approximately capturing a movement from the lowest to the 80th percentile of historical slave share. We do not observe clear evidence of gradients when considering the number of Jim Crow laws, or in alternative specifications documented in Table B6.

Table B1: Trend Breaks in 1937 in Infant and All-Age Mortality Rates from Pneumonia and Influenza

	Levels		Logs	
	All-age Mortality (per 1,000 population)	Infant Mortality (per 1,000 births)	All-age Mortality (per 1,000 population)	Infant Mortality (per 1,000 births)
	(1)	(2)	(3)	(4)
Post 1937	-0.290***	-1.003***	-0.299***	-0.147***
	(0.0124)	(0.119)	(0.0144)	(0.0175)
Year	0.0133**	-0.122*	0.0105*	-0.0133*
	(0.00596)	(0.0612)	(0.00527)	(0.00755)
(Post 1937) × Year	-0.0516***	-0.105*	-0.0660***	-0.0305***
	(0.00918)	(0.0537)	(0.00865)	(0.00709)
Observations	667	621	667	621
Mean Dep. Var.	0.94	7.25	-0.12	1.91

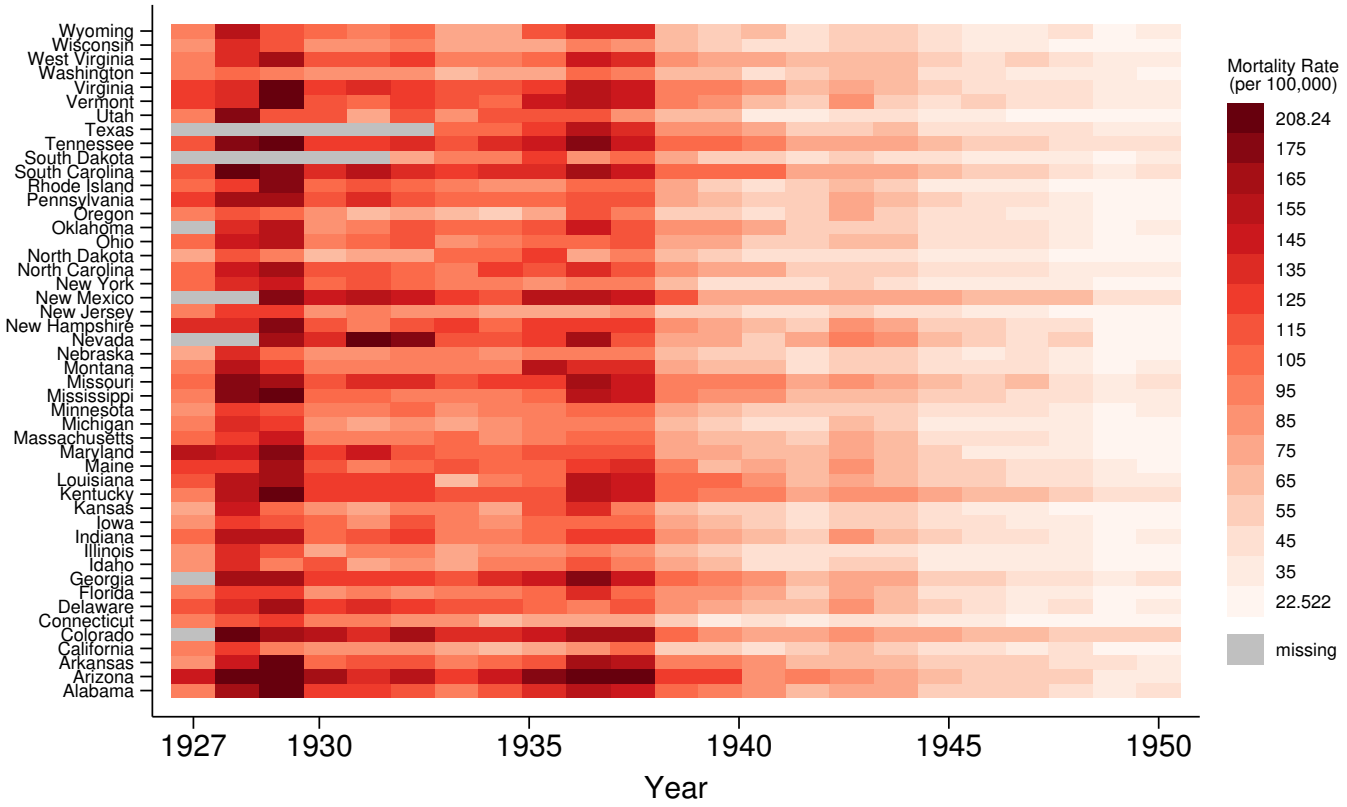
Notes: Each column presents a separate model, regressing the level or log of the dependent variable denoted in the column header on variables indicated in the table as well as state fixed effects. The level and log allow us to assess absolute and relative trend breaks, respectively. The sample includes observations for 48 states over the period 1930-1943 (max $N = 672$). In the case of infant mortality, data is available for the period 1931-1943 (max $N = 624$). See Appendix A.2 for data sources. Standard errors are clustered by state. *** $p<0.01$; ** $p<0.05$; * $p<0.10$.

Table B2: Trend Breaks in 1937 in Infant and All-Age Mortality Rates from Pneumonia and Influenza

	Levels		Logs		Levels		Logs	
	All-age Mortality (1)	**Infant Mortality** (2)	**All-age Mortality** (3)	**Infant Mortality** (4)	**All-age Mortality** (5)	**Infant Mortality** (6)	**All-age Mortality** (7)	**Infant Mortality** (8)
	Black-South				Black-North			
Post 1937	-0.952***	-1.164***	-0.411***	-0.0870***	-0.0362	-6.229	-0.506***	-0.184*
	(0.116)	(0.392)	(0.0347)	(0.0277)	(2.452)	(4.713)	(0.0465)	(0.0939)
Year	0.272***	0.166	0.139***	0.0210	-0.781	-0.120	-0.0638	-0.0512
	(0.0232)	(0.183)	(0.00849)	(0.0123)	(0.510)	(1.000)	(0.0845)	(0.0488)
Post 1937 × Year	-0.353***	-0.467	-0.188***	-0.0408*	0.283	-1.020	0.0402	-0.0443
	(0.0245)	(0.320)	(0.0111)	(0.0195)	(0.571)	(0.961)	(0.0866)	(0.0504)
Observations	188	175	188	175	236	204	226	186
Mean Dep. Var.	1.92	12.63	0.58	2.47	18.13	19.65	1.69	2.89
	White-South				White-North			
Post 1937	-0.490***	-0.0556	-0.486***	-0.000598	-0.424***	-0.562**	-0.420***	-0.0445
	(0.0229)	(0.151)	(0.0283)	(0.0285)	(0.0275)	(0.243)	(0.0252)	(0.0358)
Year	0.154***	0.172**	0.189***	0.0352*	0.146***	0.326***	0.178***	0.0554***
	(0.00623)	(0.0724)	(0.0185)	(0.0178)	(0.0115)	(0.106)	(0.0151)	(0.0150)
Post 1937 × Year	-0.197***	-0.507***	-0.246***	-0.0925***	-0.163***	-0.804***	-0.202***	-0.162***
	(0.00906)	(0.110)	(0.0225)	(0.0226)	(0.0114)	(0.126)	(0.0142)	(0.0199)
Observations	188	175	188	175	236	204	236	204
Mean Dep. Var.	0.86	6.18	-0.22	1.78	0.84	5.44	-0.23	1.62

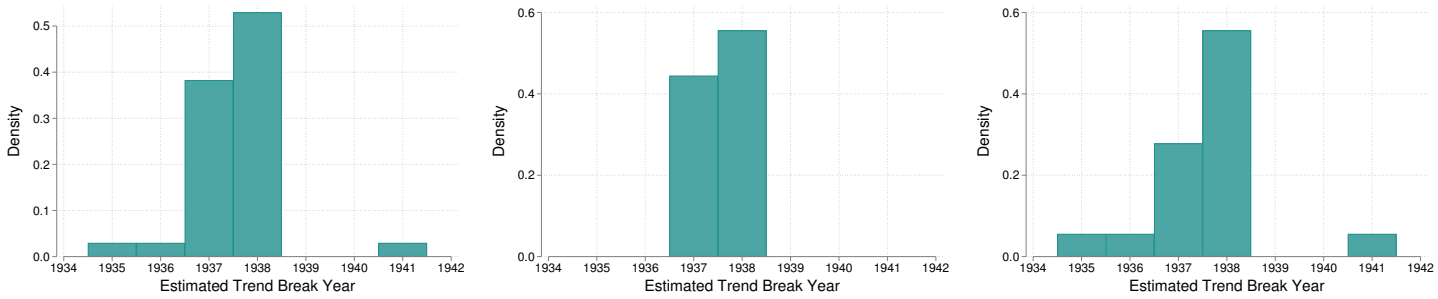
Notes: Each column presents a separate regression where mortality is measured per 1,000 population (all age) or per 1,000 births (infant). All models include state fixed effects and weight by state×race population. Sample includes yearly observations between 1930-1943, and in the case of infant mortality, 1931-1943. See Appendix A.1.2 for details on data coverage. Standard errors are clustered by state. *** p<0.01; ** p<0.05; * p<0.10.

Figure B1: State-level Mortality Trends Over Time



Notes: State by year cells present pneumonia and influenza mortality rates per 100,000 from US Vital Statistics data. Refer to Appendix A.2 for additional data details.

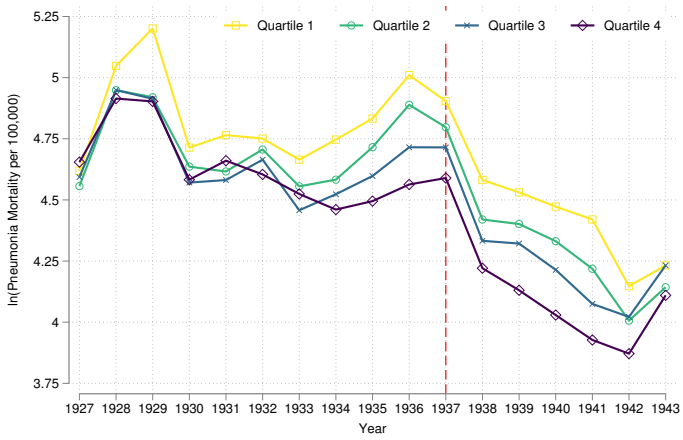
Figure B2: Estimated Timing of Trend Break by Race



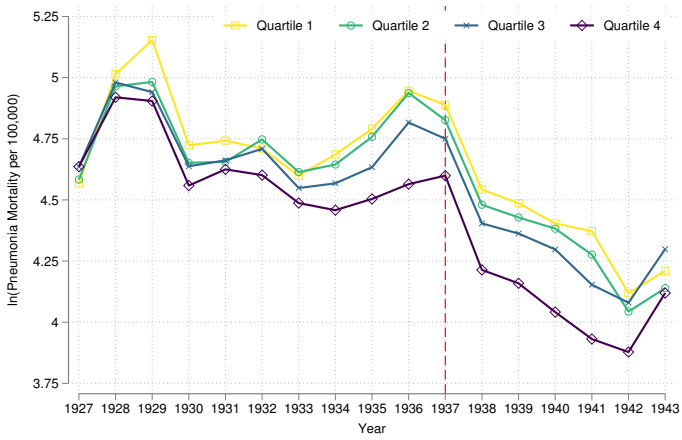
(a) All Races (b) Whites (c) Black Americans

Notes: Histograms plot years in which the principal trend break is observed in rates of pneumonia and influenza mortality rates, considering each state, and potentially all years between 1934 and 1943. The principal trend break year for each state is estimated as the year between 1940 and 1943 in which trend breaks are determined by finding the largest observed discrete change in period-to-period mortality rates and prevailing trends from a joint F-test of a Newey-West model. Years with maximum F-statistics for each state (principal trend-breaks) are plotted in each panel, for all races, and for race-specific mortality rates.

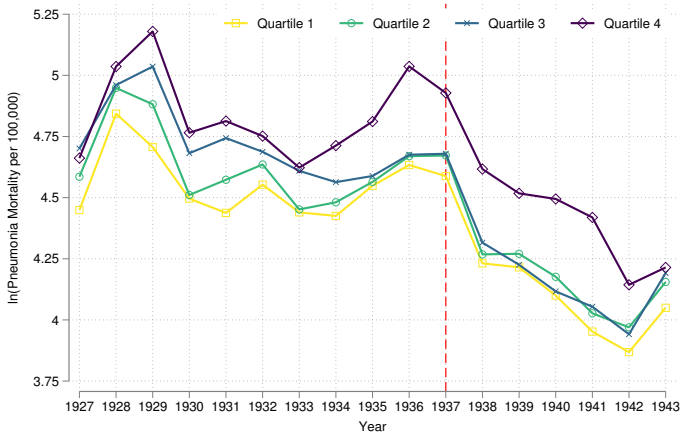
Figure B3: Trend Breaks in Pneumonia Mortality by State-Level Characteristics



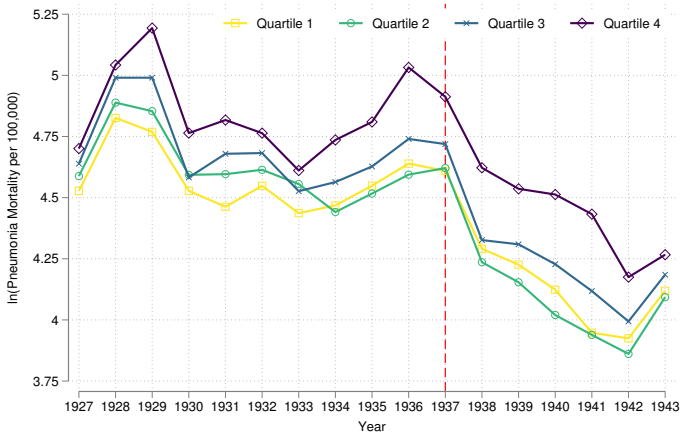
(a) Quartiles of State Income Per Capita



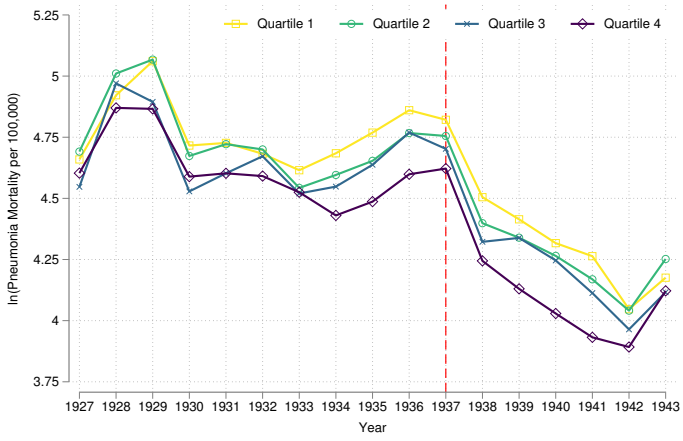
(b) Quartiles of Urbanization



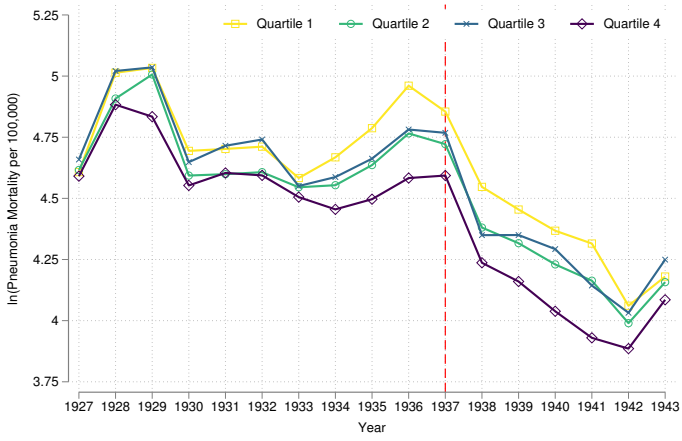
(c) Quartiles of Illiteracy



(d) Quartiles of Non-White Population Share



(e) Quartiles of Pharmacists per Capita



(f) Quartiles of Physicians per Capita

Notes: Temporal variation in rates pneumonia mortality are plotted by quartiles of state-level aggregate variables. Each state-level aggregate (indicated in plot titles) refers to averages calculated from 1930 census microdata.

Table B3: State-Level Trend Breaks by State Characteristics at Baseline

$X =$	Illiteracy (1)	Inc. p.c (2)	Urban (3)	Phys. p.c. (4)	Pharm. p.c. (5)	Slave frac. (6)	Jim Crow (7)
Panel A: All States							
Post 1937	-0.294***	-0.284***	-0.283***	-0.292***	-0.290***	-0.282***	-0.294***
	(0.0123)	(0.0125)	(0.0126)	(0.0148)	(0.0149)	(0.0114)	(0.0110)
Post 1937 $\times X$	0.0651***	-0.0356***	-0.0376***	-0.0301*	-0.0403***	0.0684***	0.0573***
	(0.0124)	(0.00756)	(0.0108)	(0.0169)	(0.0147)	(0.0126)	(0.0126)
Post 1937 $\times$ Year	-0.0679***	-0.0807***	-0.0823***	-0.0737***	-0.0706***	-0.0725***	-0.0699***
	(0.00806)	(0.00569)	(0.00566)	(0.00752)	(0.00877)	(0.00591)	(0.00657)
Post 1937 $\times$ Year $\times X$	-0.0222***	0.0311***	0.0346***	0.0305***	0.0204***	-0.0380***	-0.0280***
	(0.00522)	(0.00471)	(0.00475)	(0.00581)	(0.00752)	(0.00608)	(0.00748)
Observations	667	667	667	667	667	515	667
Effect at Percentile 10	-0.35	-0.24	-0.23	-0.25	-0.24	-0.33	-0.33
Effect at Percentile 90	-0.19	-0.34	-0.34	-0.33	-0.35	-0.17	-0.22
Effect at Percentile 10 (year 3)	-0.50	-0.60	-0.61	-0.60	-0.53	-0.47	-0.49
Effect at Percentile 90 (year 3)	-0.50	-0.43	-0.42	-0.43	-0.47	-0.58	-0.54
Panel B: Southern States Only							
Post 1937	-0.289***	-0.273***	-0.260***	-0.252***	-0.242***	-0.294***	-0.296***
	(0.0105)	(0.0206)	(0.0255)	(0.0166)	(0.0173)	(0.0145)	(0.0273)
Post 1937 $\times X$	0.0721***	-0.0652***	-0.0626**	-0.0822**	-0.0531	0.0768***	0.0576**
	(0.0150)	(0.0180)	(0.0281)	(0.0281)	(0.0335)	(0.0170)	(0.0252)
Post 1937 $\times$ Year	-0.0972***	-0.0823***	-0.0947***	-0.102***	-0.113***	-0.0895***	-0.0965***
	(0.0141)	(0.0124)	(0.0177)	(0.0132)	(0.0134)	(0.0135)	(0.0137)
Post 1937 $\times$ Year $\times X$	-0.0156*	0.0402***	0.0302	0.0294	-0.00849	-0.0247**	-0.0120
	(0.00736)	(0.00986)	(0.0179)	(0.0178)	(0.0150)	(0.00898)	(0.00835)
Observations	221	221	221	221	221	221	221
Effect at Percentile 10	-0.35	-0.19	-0.18	-0.13	-0.17	-0.35	-0.33
Effect at Percentile 90	-0.18	-0.38	-0.36	-0.36	-0.32	-0.16	-0.22
Effect at Percentile 10 (year 3)	-0.60	-0.59	-0.58	-0.57	-0.48	-0.56	-0.60
Effect at Percentile 90 (year 3)	-0.54	-0.43	-0.50	-0.55	-0.70	-0.56	-0.56

Notes: Each column presents a separate regression of the natural logarithm of rates of pneumonia and influenza mortality per 1,000 on a time trend, Post-1937 indicator, Post-1937 by time indicator, full interaction with state-level means of the measures indicated in column headers, and state fixed effects. For the sake of presentation, only Post-1937 and interaction terms are presented. State-level means are calculated from 1930 census microdata, and each of these variables are re-standardized as a Z-score for comparability. Observations are state by year cells between 1930 and 1943, for all 48 mainland states in panel A, and only for Southern state in panel B. Regressions are weighted by population. Standard errors are clustered by state. *** $p<0.01$; ** $p<0.05$; * $p<0.10$.

Table B4: State-Level Trend Breaks by State Characteristics at Baseline and by Race

$X =$		Illiteracy	Inc. p.c	Urban	Phys. p.c.	Pharm. p.c.	Slave frac.	Jim Crow
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Black Population								
Post 1937	-0.111***	-0.267***	-0.215***	-0.186***	-0.169***	-0.139***	-0.294***	-0.202***
	(0.0382)	(0.0329)	(0.0473)	(0.0549)	(0.0338)	(0.0393)	(0.0435)	(0.0493)
Post 1937 × Year	-0.0671***	-0.0484***	-0.0362***	-0.0470***	-0.0561***	-0.0659***	-0.0350**	-0.0543***
	(0.00592)	(0.0141)	(0.00872)	(0.0121)	(0.00849)	(0.00730)	(0.0153)	(0.0111)
Post $\times X$		0.114***	-0.117***	-0.112**	-0.135***	-0.0806*	0.141***	0.0674**
		(0.0277)	(0.0366)	(0.0490)	(0.0402)	(0.0433)	(0.0322)	(0.0291)
Post × Year $\times X$		-0.0134*	0.0342***	0.0291**	0.0256**	0.00463	-0.0242**	-0.00944
		(0.00674)	(0.00688)	(0.0117)	(0.00973)	(0.00897)	(0.00877)	(0.00561)
Observations	298	298	298	298	298	298	282	298
Effect at Percentile 10 (year 3)		-0.48	-0.30	-0.30	-0.25	-0.25	-0.45	-0.39
Effect at Percentile 90 (year 3)		-0.30	-0.35	-0.37	-0.41	-0.44	-0.28	-0.31
Panel B: White Population								
Post 1937	-0.173***	-0.202***	-0.191***	-0.184***	-0.185***	-0.178***	-0.207***	-0.202***
	(0.0150)	(0.0114)	(0.0128)	(0.0144)	(0.0124)	(0.0151)	(0.0111)	(0.0142)
Post 1937 × Year	-0.0692***	-0.0555***	-0.0513***	-0.0549***	-0.0629***	-0.0716***	-0.0463***	-0.0506***
	(0.0126)	(0.0158)	(0.00612)	(0.00900)	(0.0137)	(0.0121)	(0.0138)	(0.0157)
Post $\times X$		0.0420***	-0.0360***	-0.0340**	-0.0605***	-0.0207	0.0495***	0.0312**
		(0.0109)	(0.00879)	(0.0131)	(0.0204)	(0.0268)	(0.0105)	(0.0140)
Post × Year $\times X$		-0.0199**	0.0389***	0.0430***	0.0307*	-0.00966	-0.0330***	-0.0209*
		(0.00824)	(0.00430)	(0.00855)	(0.0159)	(0.0111)	(0.0106)	(0.0108)
Observations	298	298	298	298	298	298	282	298
Effect at Percentile 10 (year 3)		-0.35	-0.45	-0.47	-0.42	-0.33	-0.31	-0.33
Effect at Percentile 90 (year 3)		-0.40	-0.21	-0.20	-0.33	-0.47	-0.43	-0.39

Notes: Refer to Notes to Table B3. Identical models are estimated, however here for race-specific mortality rates. Observations are state by year cells between 1927 and 1943 for the 18 states for which race-specific mortality rates are available. Regressions are weighted by the population of each race in the state. Standard errors are clustered by state. *** $p<0.01$; ** $p<0.05$; * $p<0.10$.

Table B5: Mortality Convergence by State-level Measures

	(1) Illiteracy	(2) Income p.c.	(3) Urbanization	(4) Physicians	(5) Pharmacists	(6) Slave Share (1860)	(7) Jim Crow Laws
Panel A: All Individuals							
Post Sulfa × Base Exposure	-0.394***	-0.403***	-0.395***	-0.327***	-0.298***	-0.418***	-0.416***
	(0.0572)	(0.0441)	(0.0546)	(0.0441)	(0.0347)	(0.0707)	(0.0514)
Post Sulfa × Base Exposure × State Var	0.0688	-0.0620	-0.0261	-0.107***	-0.109***	-0.0464	-0.0343
	(0.0643)	(0.0413)	(0.0573)	(0.0382)	(0.0360)	(0.0537)	(0.0721)
Observations	798	798	798	798	798	620	798
R-Squared	0.867	0.868	0.866	0.866	0.866	0.873	0.868
Panel B: Black Individuals							
Post Sulfa × Base Exposure	-0.567***	-0.506***	-0.655***	-0.635***	-0.599***	-0.446***	-0.518***
	(0.100)	(0.151)	(0.108)	(0.0555)	(0.0591)	(0.0883)	(0.0689)
Post Sulfa × Base Exposure × State Var	0.0943	-0.0264	-0.250**	-0.112**	-0.0908	0.0452	-0.0287
	(0.0711)	(0.116)	(0.0903)	(0.0454)	(0.0553)	(0.0988)	(0.0575)
Observations	298	298	298	298	298	282	298
R-Squared	0.868	0.869	0.870	0.868	0.868	0.873	0.868
Panel C: White Individuals							
Post Sulfa × Base Exposure	-0.257***	-0.293**	-0.286**	-0.315***	-0.331***	-0.215***	-0.197**
	(0.0715)	(0.103)	(0.110)	(0.101)	(0.0955)	(0.0583)	(0.0824)
Post Sulfa × Base Exposure × State Var	-0.176**	0.0939	0.0448	0.186	-0.0648	-0.211***	-0.226**
	(0.0732)	(0.0750)	(0.0982)	(0.195)	(0.151)	(0.0504)	(0.0799)
Observations	298	298	298	298	298	282	298
R-Squared	0.901	0.900	0.900	0.900	0.900	0.900	0.901

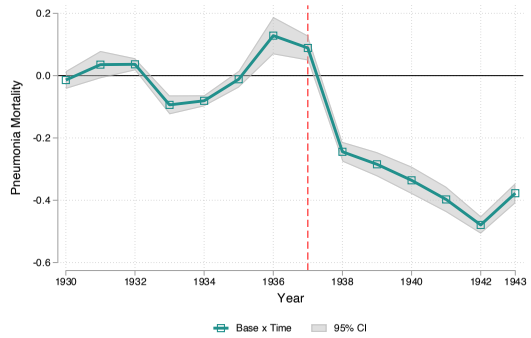
Notes: Each column regresses rates of influenza and pneumonia mortality on baseline mortality in the state (mean from 1930-1936) interacted with post sulfa, as well as interactions with state-level baseline variable, indicated in table headers, along with state and year fixed effects. Panel A is based on all mortality, while panels B and C are race-specific mortalities in states for which we have Black and white-specific mortality rates. In these models, all base pneumonia rates refer to race-specific mortality rates in the same state. *** $p<0.01$; ** $p<0.05$; * $p<0.10$.

Table B6: Convergence in Rates of Pneumonia Mortality

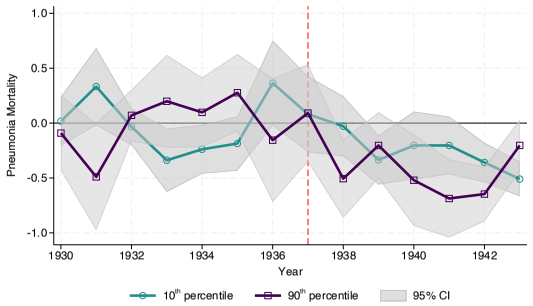
	Influenza Mortality (post-1937)			
	(1)	(2)	(3)	(4)
Panel A: All Races				
Post Sulfa × Base Exposure	-0.300***	-0.306***	-0.401***	-0.495***
	(0.0511)	(0.0638)	(0.0684)	(0.0654)
95% CI Wild Bootstrap	[-0.40, -0.20]	[-0.46, -0.19]	[-0.56, -0.26]	[-0.64, -0.35]
Observations	667	667	655	655
Mean Influenza Mortality	0.90	0.90	0.91	0.91
Panel B: White Only				
Post Sulfa × Base Exposure	-0.261***	-0.471**	-0.491***	-0.744***
	(0.0763)	(0.171)	(0.122)	(0.187)
95% CI Wild Bootstrap	[-0.49, -0.05]	[-0.71, 0.00]	[-0.70, -0.14]	[-1.26, -0.41]
Observations	298	298	298	298
Mean Influenza Mortality	0.97	0.97	0.97	0.97
Panel C: Black Only				
Post Sulfa × Base Exposure	-0.550***	-0.557***	-0.434***	-0.479***
	(0.0943)	(0.117)	(0.0719)	(0.103)
95% CI Wild Bootstrap	[-0.78, -0.38]	[-0.87, -0.36]	[-0.58, -0.29]	[-0.65, -0.18]
Observations	298	298	298	298
Mean Influenza Mortality	1.75	1.75	1.75	1.75
Controls				
State & Year FEs	Y	Y	Y	Y
Census Div-Year FEs		Y	Y	Y
Disease Controls			Y	Y
SES Controls				Y

Notes: Each cell represents a separate regression where the influenza and pneumonia mortality rate (per 1,000) is regressed on a Post-sulfa (Post-1937) indicator, multiplied with the baseline mortality rate. In Panel A, the sample consists of state-by year cells covering 1930-1943 of the 48 mainland US states. Panels B and C report race-specific mortality rates, and consist of only 18 states with race-specific mortality rates reported. In these cases, CIs based on a wild cluster bootstrap are also reported given relatively low cluster size. Controls indicated in Table footer are described Section 2.2. *** p<0.01; ** p<0.05; * p<0.10. Standard errors clustered by state are reported in parentheses.

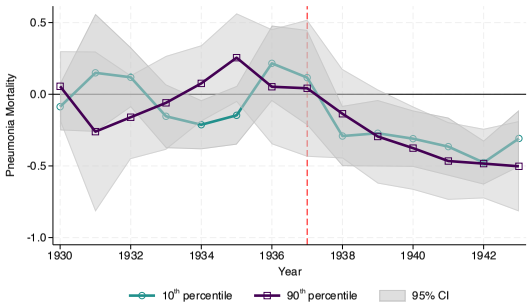
Figure B4: Convergence Event Studies



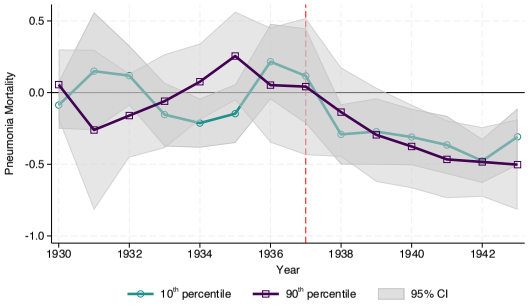
(a) Baseline



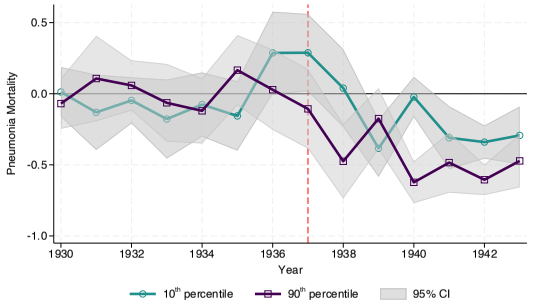
(b) Urbanization Interaction



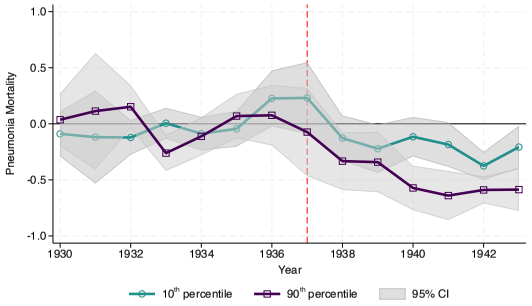
(c) Illiteracy Interaction



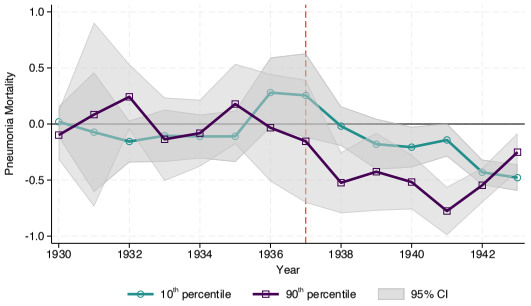
(d) Non-White Population Share Interaction



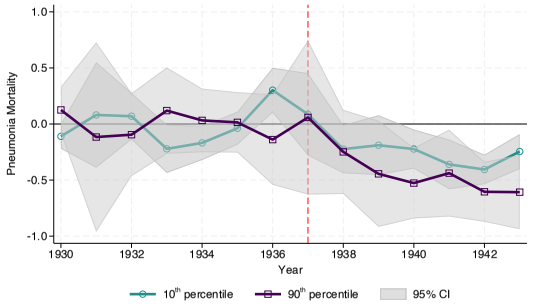
(e) Physicians per capita Interaction



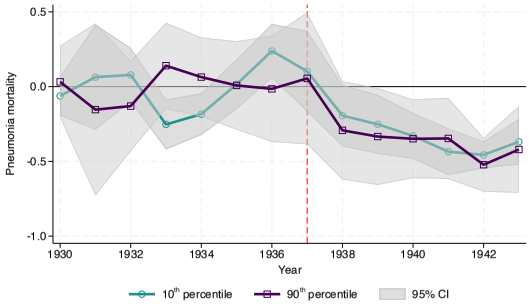
(f) Pharmacist per capita Interaction



(g) Baseline per capita income Interaction



(h) 1860 Slave share Interaction



(i) Jim Crow laws

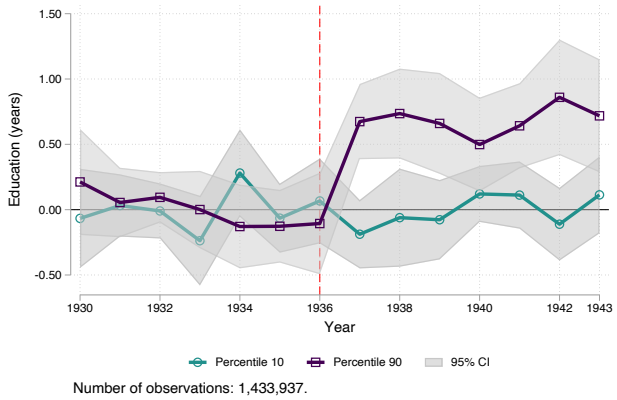
Notes: Each plot presents coefficients from a regression of state-level mortality on baseline mortality times a full set of year fixed effects as well as state fixed effects (panel (a)). Panels (b)-(i) implement a similar specification, however now also including interactions with baseline mortality time year fixed effects times the state-level characteristic indicated in plot titles. This is the "first stage" with negative coefficients indicating convergence. In panels (b)-(i), marginal effects are reported at the 10^{th} and 90^{th} percentile for each characteristic indicated in plot captions. Gray shaded areas represent 95% confidence intervals based on standard errors clustered by state.

Table B7: First Stage Access and Systemic Discrimination

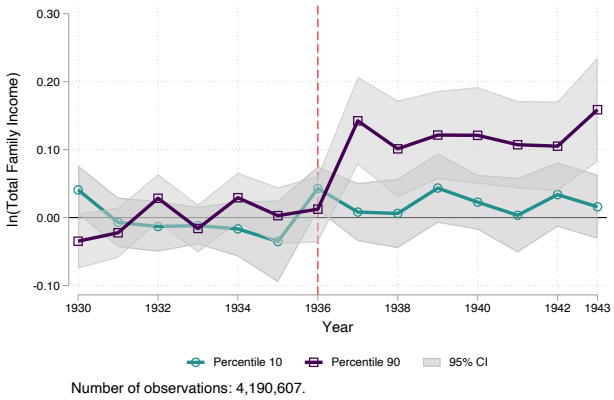
	Gradient: Historical Fraction of Enslaved People				Gradient: Number of Jim Crow Laws			
	Unweighted Models		Population weighted		Unweighted Models		Population weighted	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: All States, Total Mortality								
Post Sulfa × Base Exposure	-0.340***	-0.418***	-0.292***	-0.403***	-0.340***	-0.416***	-0.292***	-0.400***
	(0.0503)	(0.0729)	(0.0612)	(0.0961)	(0.0503)	(0.0530)	(0.0612)	(0.0733)
Post Sulfa × Base Exposure × Discrimination Proxy		-0.0464		-0.0298		-0.0343		-0.0320
		(0.0554)		(0.0704)		(0.0743)		(0.0842)
95% CI Wild Bootstrap		[-0.18, 0.12]		[-0.17, 0.16]		[-0.18, 0.15]		[-0.18, 0.18]
Observations	798	620	798	620	798	798	798	798
R-Squared	0.87	0.87	0.90	0.90	0.87	0.87	0.90	0.90
Panel B: All States, Black Mortality								
Post Sulfa × Base Exposure	-0.588***	-0.446***	-0.550***	-0.589***	-0.588***	-0.518***	-0.550***	-0.524***
	(0.0615)	(0.0911)	(0.0943)	(0.0942)	(0.0615)	(0.0711)	(0.0943)	(0.0719)
Post Sulfa × Base Exposure × Discrimination Proxy		0.0452		0.145*		-0.0287		-0.00321
		(0.102)		(0.0790)		(0.0594)		(0.0462)
95% CI Wild Bootstrap		[-0.32, 0.30]		[-0.24, 0.29]		[-0.19, 0.10]		[-0.10, 0.16]
Observations	298	282	298	282	298	298	298	298
R-Squared	0.87	0.87	0.88	0.89	0.87	0.87	0.88	0.88
Panel C: Southern States, Black Mortality								
Post Sulfa × Base Exposure	-0.575***	-0.438***	-0.537***	-0.467***	-0.575***	-0.546***	-0.537***	-0.520***
	(0.0611)	(0.0833)	(0.0946)	(0.0729)	(0.0611)	(0.0732)	(0.0946)	(0.0882)
Post Sulfa × Base Pneumonia × Discrimination Proxy		0.0529		0.129*		-0.0559		-0.0211
		(0.0832)		(0.0668)		(0.0561)		(0.0432)
95% CI Wild Bootstrap		[-0.25, 0.22]		[-0.19, 0.25]		[-0.24, 0.06]		[-0.18, 0.12]
Observations	265	265	265	265	265	265	265	265
R-Squared	0.88	0.89	0.89	0.90	0.88	0.88	0.89	0.89

Notes: Each cell presents a separate regression where the influenza and pneumonia mortality rate (per 1,000) is regressed on a Post-sulfa (Post-1937) indicator, multiplied with the baseline mortality rate, as well as interactions with the historical share of enslaved persons in each state (columns (1)-(4)) or the number of Jim Crow laws (columns (5)-(8)), and state and year fixed effects. In panel A, the sample consists of state-by year cells covering 1927-1943 of the 48 mainland US states. Panels B and C report mortality rates for Black individuals only, based on the 18 states with race-specific mortality rates reported (panel B), and only the Southern states among these states (panel C). Confidence intervals based on a wild cluster bootstrap are presented in the table footer for the Post × Base Pneumonia × Slave Share term. *** p<0.01; ** p<0.05; * p<0.10.

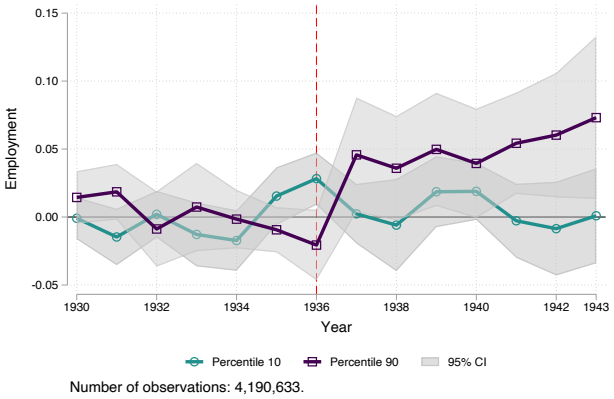
Figure B5: Pharmacist Interaction Long Term Event Studies



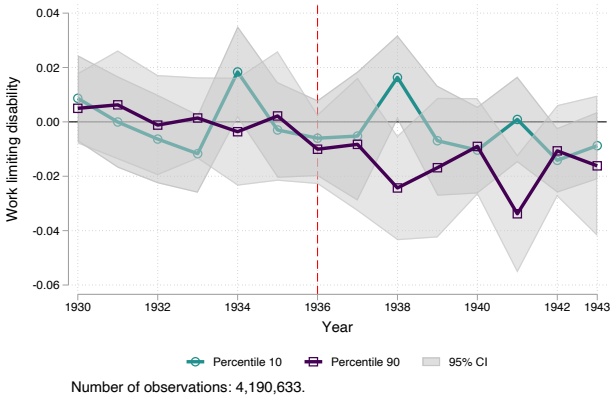
(a) Education



(b) ln(Family Income)



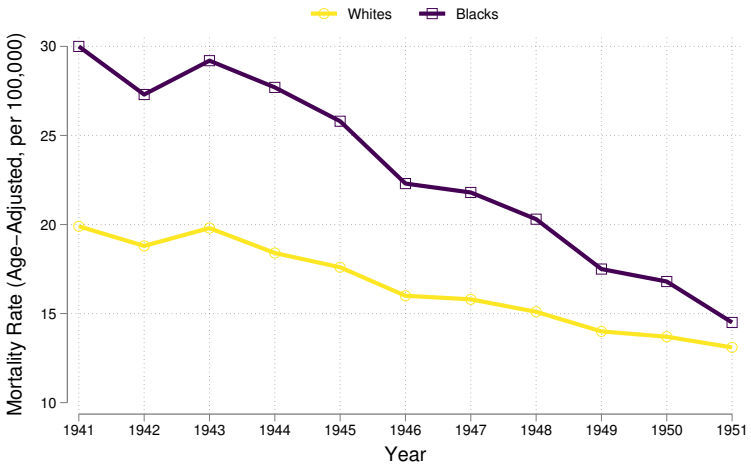
(c) Employment



(d) Work Limiting Disability

Notes: Each plot presents an event study where long term outcomes are regressed on baseline mortality times a full set of lags and leads, full interactions between lags and leads and pharmacist coverage, and state and year fixed effects. Marginal effects are reported for an interquartile range shift in exposure to sulfa (0.29 fewer deaths per 1,000) at percentiles 10 and 90 of pharmacist coverage.

Figure B6: Rheumatic fever mortality rates by race in the US South



Notes: Trends document mortality rates for rheumatic fever in Southern US states. Source is US Vital Statistics.

C Modeling and Identification Details

C.1 Further details related to estimation and identification

In what follows, we will refer to treatment intensity for a particular state as p, for pneumonia mortality, and the set of all mortality rates as $\mathcal{P}$. Following the notation of Callaway et al. (2025), the Average Causal Response (ACR) is defined as:

$$ACR(p) = \frac{\partial E[Y_{istc}(p)]}{\partial p},$$

capturing the average effect of a marginal change in exposure to pneumonia on outcomes of interest Y. Callaway et al. (2025) show that under a "strong parallel trend" assumption, the parameter τ from (1) captures the following quantity:

$$\tau = \sum_{p_j \in \mathcal{P}} w(p_j) \frac{ACR(p_j)}{p_j - p_{j-1}},$$

that is, a weighted sum of ACRs. The weights, w sum to one, and are defined as:

$$w(l) = \frac{(E[P|P \geq l] - E[P]) \Pr(P \geq l)}{\sigma_P^2},$$

where σ_P^2 refers to the variance of baseline pneumonia mortality. In practice, if we consider a pre-treatment outcome Y_{t-1} and post-treatment outcome Y_t (for ease of notation, subscript isc is left implicit), the strong parallel trend assumption required states that for all $p \in \mathcal{P}$:

$$E[Y_t(p) - Y_{t-1}(0)] = E[Y_t(p) - Y_{t-1}(0)|P = p].$$

In words, this assumption states that observed trends in outcomes for units with a particular baseline dosage p (the quantity on the right-hand side above), are a good counterfactual for the trends which all other units would have followed, had they instead been assigned that particular baseline mortality rate. This can be seen as a multi-valued generalization of the parallel trends assumption, given that the standard parallel trends assumption simply requires that the trends in outcomes for untreated units are a good counterfactual for trends in outcomes to treated units, had they not been treated.

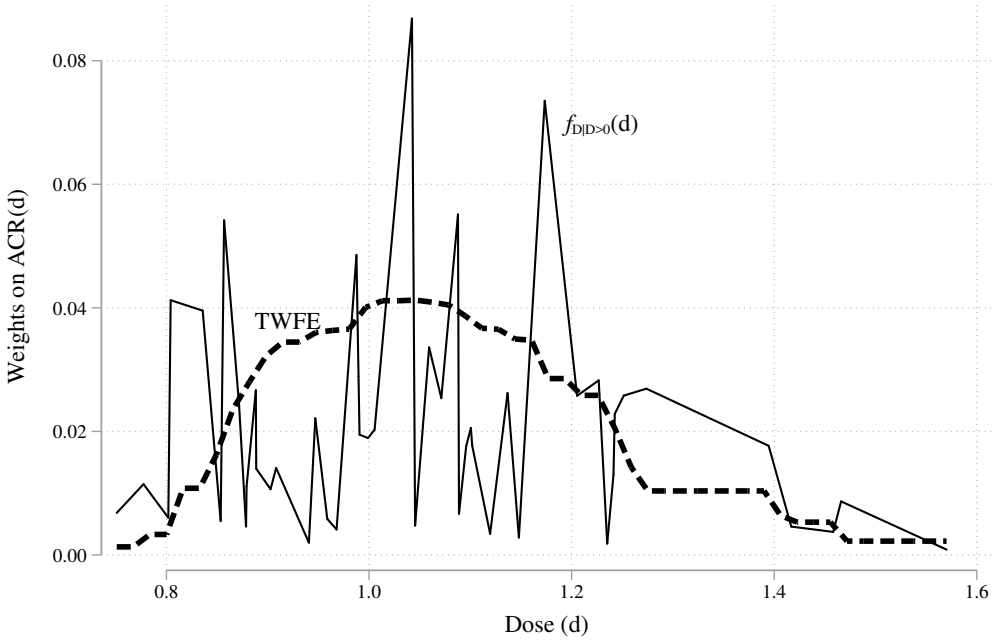
This strong parallel trends assumption cannot be explicitly tested, as it refers to counterfactual treatment assignments which are never observed. Nevertheless, we can seek to shed some light on this in a number of ways. In particular, we can examine variable dose responses and seek to determine whether there are parallel pre-trends by dose response groups, and we can consider the weighting functions w to ensure that this does not lead to aggregation in an undesired comparisons. We examine these tests in Figure 5 and C1. Figure 5 examines how outcomes of interest evolve in states with distinct baseline pneumonia levels, p, compared to the evolution of outcomes in states with the lowest baseline pneumonia level. This is analogous to event study models in (2), but rather than considering a continuous treatment dose measure, we consider indicators for belonging to specific treatment intensity deciles. Here we observe, for education, employment and income, a dose response function, where outcomes in states with the highest treatment intensity improved most, with treatment effects tending to decline with treatment intensity. While the pre-trends are noisy (and estimates for work-related disability are less clean), on average the pre-trends do not deviate from the zero line until 1937, when the positive outcomes tend to rise. While this does not necessarily imply that counterfactual trends would have been flat in post-treatment periods, the lack of a noteworthy trend *between each treatment*

intensity group in the pre-reform period is reassuring for this research design. In Figure C1 we document the aggregation weights which are implicit in two-way FE models (dashed line), compared to the actual frequency of treatment exposures (solid line). That these quantities are broadly similar suggests that results are unlikely to be driven by undesired weighting, and indeed, in Panel B of Appendix Tables C1 we observe that if we re-weight observations such that states are representative of their dose frequency, results are broadly similar.

If, however, we seek to avoid a strong parallel trends assumption entirely, we can also consider a discretization of the treatment variable, where rather than considering the full support of $\mathcal{P}$, we simply generate a measure of Highly Treated $= 1\{\text{Baseline Pneumonia} > \text{Median(Baseline Pneumonia)}\}$, in which case we return to standard identifying assumptions of parallel trends, where we assume that had highly treated units not been highly treated by sulfa, they would have followed the trends which were observed in less treated areas. We consider this specification as an alternative model, and in Panel C of Appendix Tables C1 observe that the takeaway in terms of this discretized model is similar with clear increases in education and incomes among individuals more highly exposed to the benefits of sulfa drugs.

C.2 Tables and Figures

Figure C1: Estimand Weights: Two-Way Fixed Effects Weights versus Treatment Distribution Weights



Notes: Figure documents weights of units based on their "dose" of sulfa exposure (baseline mortality rates). Implicit weights assigned to individual units within the two-way fixed effect model estimated in this paper are presented as a dashed black line, while empirical weights based on the frequency of doses observed in our sample of individuals born 1930–1943 are documented as a solid black line. TWFE weights are calculated following Callaway et al. (2024, proposition 4).

Table C1: Two-way Fixed Effects, Average Causal Responses and Binary Treatments Individuals

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A - Baseline Model				
Post Sulfa × Base Exposure	0.199**	0.0497***	0.0172**	-0.00816**
	(0.0872)	(0.0120)	(0.00736)	(0.00354)
Effect size for an interquartile shift in base exposure	0.0560 years	1.399 %	0.485 pp	-0.230 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel B - Reweighting based on ACR function				
Post Sulfa × Base Exposure	0.213*	0.0414***	0.0159**	-0.0113***
	(0.107)	(0.0149)	(0.00748)	(0.00393)
Effect size for an interquartile shift in base exposure	0.0599 years	1.165 %	0.448 pp	-0.319 pp
Observations	1,433,935	4,110,227	4,190,632	4,190,632
Panel C - Binary Treatment Measure				
Post Sulfa × High Base Exposure	0.0624**	0.0145***	0.00558**	-0.00208**
	(0.0271)	(0.00412)	(0.00240)	(0.00102)
Observations	1,433,937	4,110,228	4,190,633	4,190,633

Notes: Each column represents a separate regression. Estimates are for all individuals (black and white women and men). Coefficient reported is on Post×Baseline Pneumonia (panels A and B) or Post×Highly Exposed, where Post identifies cohorts born after 1936 and Baseline Pneumonia Influenza is the average pneumonia+influenza mortality rate between 1930–1936. Highly exposed refers to states with a baseline mortality rate above the median. Sample definitions and controls follow those defined in Table 1. Panel A presents identical models as in Table 1 for comparison. Panel B re-weights observations so that estimates can be considered to be representative of doses in the sample. In this procedure, units are re-weighted by the ratio of analytic weights to TWFE weights documented in C1. In panels A and B where a continuous treatment measure is used, estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) is provided in panel footers as *Effect Size*. *** p<0.01; ** p<0.05; * p<0.10.

D Measurement Concerns

D.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 exposure 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, 1987) which, together, introduces noise in infant mortality rates (which are number of deaths divided by number of births in a year). In Appendix Table E1, 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 1a and dependent variable means in Table B1 of Appendix B where we discuss sulfa's impact on mortality). 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 2b).

It is nevertheless important to demonstrate that the "first stage" holds with infant mortality rates as it does with all-age rates. See Table B1, 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_t (=1 for 1937 an onwards, 0 otherwise), Year_t (a trend in birth year), Post $\times$ Year_t, and state fixed effects, using Vital Statistics data for 1930-1943. We find negative and statistically significant trend breaks (the coefficient on Post $\times$ Year_t) for both the level and log models and for both infant and all-age mortality rates.

Pneumonia versus 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 E1, panel A, 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 D1 below by gender and race. For both genders and both races, it is clear that pneumonia dominated influenza in prevalence, and that it was pneumonia that showed a significant decline after 1937. If one compares the 1930-36 pre-intervention period with the post-intervention

period, there is little decline in influenza mortality rates, the drop from 1937 to 1938 being in part an artifact of an influenza pandemic in 1936-37 (which also led to an uptick in pneumonia cases since pneumonia is often caused by influenza).

Figure D1 also shows that mortality rates from both diseases were higher for men than for women, and for Black persons 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 greater for Black Americans than for whites but the declines are evident 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 1,166 per 100,000 infants overall, 1,375 and 951 for boys and girls respectively. We discuss measurement error in pneumonia mortality as it applies to race in Section D.2.

D.2 Measurement Error in Mortality Rates by Race

In the previous sub-section 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 (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 sub-section, 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 Black Americans resided in the mid-1930s (Ewbank, 1987). State and race-specific time-invariant differences in measurement are captured in our specifications by race$\times$state fixed effects, and general trends in the quality of vital statistics data are absorbed by race$\times$year fixed effects. Linearly evolving state-specific secular improvements in measurement are accounted for by race$\times$birth state specific pre-1937 time trends which we document in Figure 6. We are nevertheless concerned that our estimates may carry a bias if the divergence we identify in long run outcomes of sulfa-exposure for Black Americans in more versus less discriminatory states arises spuriously on account of differential trends in the quality of data on pneumonia mortality rates in states with higher or lower rates of institutionalized discrimination. 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 mortality rates of Black Americans 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 Schachter, 1952). 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 D2(a) 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 can 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 D2(b) 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 Black Americans than for whites because more than 85% of Black Americans lived in the South during the sulfa drug era. As discussed in the paper, this is relevant to our identification of discrimination gradients. Figures D2(c) and D2(d) 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. Figure D2(e) uses data from Linder and Grove (1947), which show prominent clumping in last digit of recorded age of death for Black individuals but not whites, another form of measurement error. In Figure D2(f) 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 under-reporting 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.[54] 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 D3 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 variance σ_u^2. The probability limit of the OLS estimator, b, is:

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

The bias is thus equivalent to $p\lim[(X^{*\prime}X^*)^{-1}X^{*\prime}(-uB+e)]$. 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 under-reporting 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^*.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 Exposure. Replacing Post sulfa$_t$ $\times$ Base Exposure$_s$ with Post sulfa$_t$ $\times$ (Base Exposure $+ u$)$_s$ in equation (1) in the paper by race produces the additional term Post sulfa$_t \times u_s$ which we proxy with Post$_t \times$ registry completeness$_s$. These estimates are displayed in Table E1, panel C.

[54] 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 and because, at any adult age, the population at risk is given by some earlier birth cohort.

D.3 Historical Share of Enslaved Persons and Jim Crow laws as Measures of Discrimination

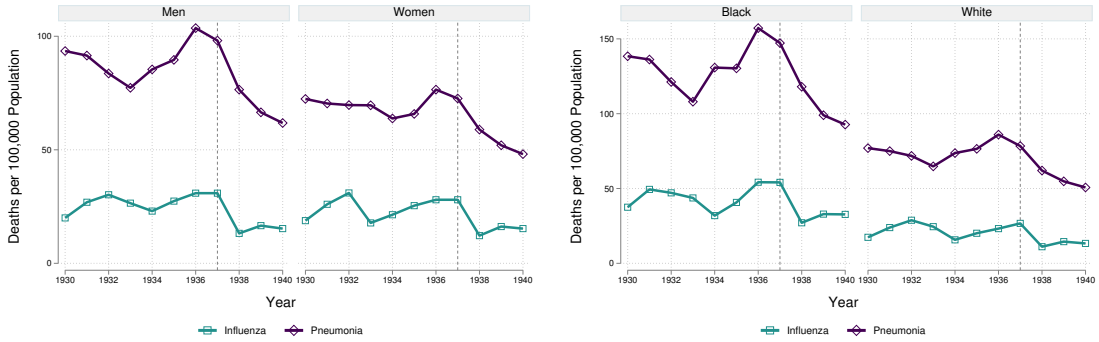
In the main text, we define and discuss the use of historical slave share and the number of Jim Crow laws as measures of systemic discrimination. Details on the source and descriptive statistics are discussed in Appendix A.1.4. Here, we further define the empirical content of this measure.

First, to demonstrate that this pre-determined measure has contemporaneous relevance for other measures of institutionalized discrimination, we collect a number of relevant proxies of contemporaneous indicators of discrimination in 1940. This consists of black/white differentials in average schooling and in average wages. These measures are generated from data for men aged 25-55 in the IPUMS 1940 Census 1% sample. These define relative levels in the population, capturing differences in levels of these measures between black and white males. Figures D5 and D6 documents a very strong pattern between measures of wage and schooling ratios (observed within all states) and historical slave share and Jim Crow laws outside of states with 0s in each measure.

In the paper we wish to consider how returns to sulfa are mediated by systemic discrimination. As such we demonstrate independent variation in measures of discrimination, baseline pneumonia mortality rates, and markers of measurement error discussed in Section D.2. These are presented in Figure D4. In a previous working paper version of this work, we documented that gradient estimates are robust to considering these alternative proxies of discrimination instead of historical slave fraction or Jim Crow laws (Bhalotra and Venkataramani, 2015).

D.4 Tables and Figures

Figure D1: Pneumonia vs. Influenza Mortality Rates by Sex and Race

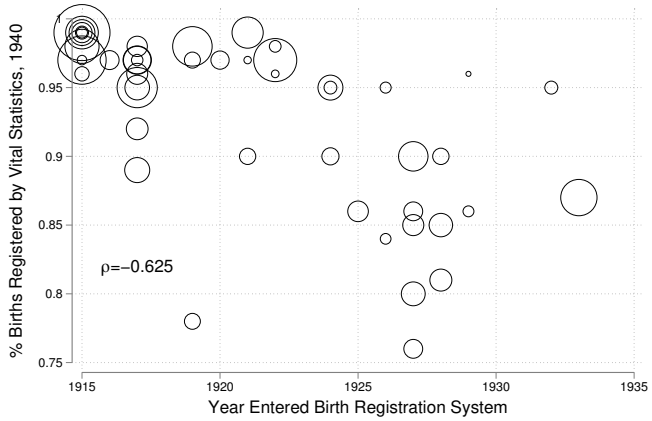


(a) Rates by Sex

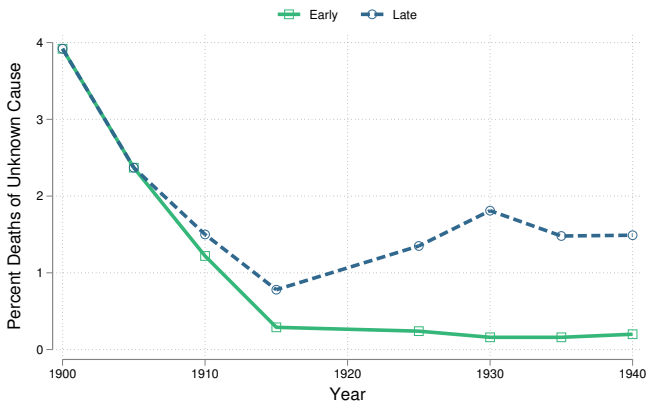
(b) Rates by Race

Notes: Data are recorded from United States Vital Statistics compendium (Linder and Grove, 1947, pp. 258-289). Rates by sex are reported in Vital Statistics "Specific death rates for selected causes, by sex", and rates by race are reported in Vital Statistics "Specific death rates for selected causes, by race".

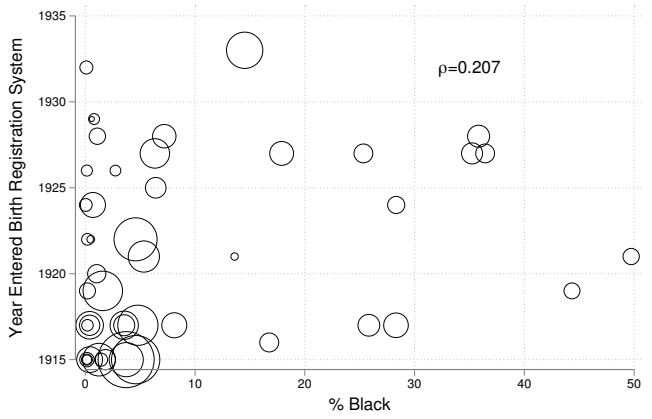
Figure D2: Proxies for measurement quality of mortality rates



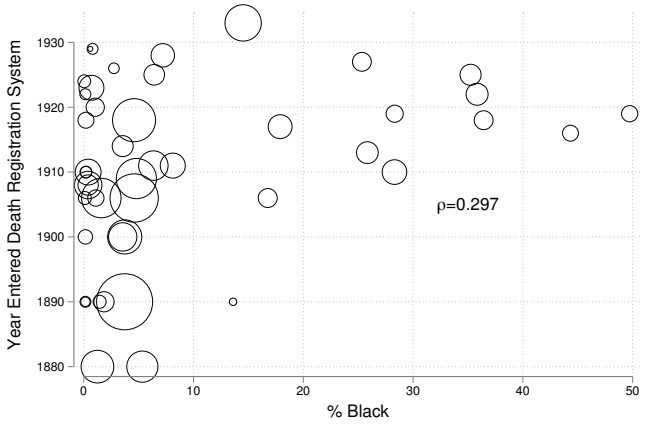
(a) Birth Registration System Entry and 1940 Registration



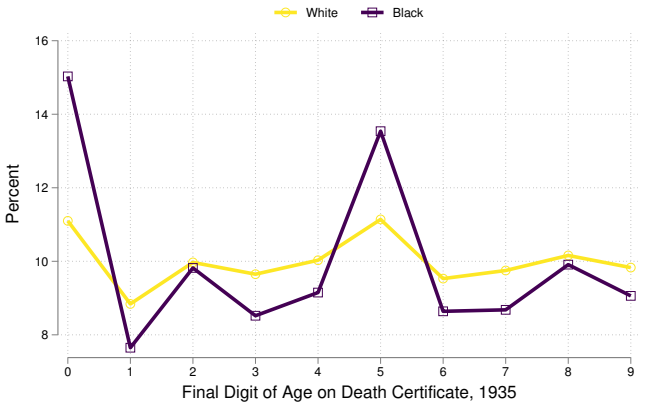
(b) Death Registration System Entry and Cause of Death



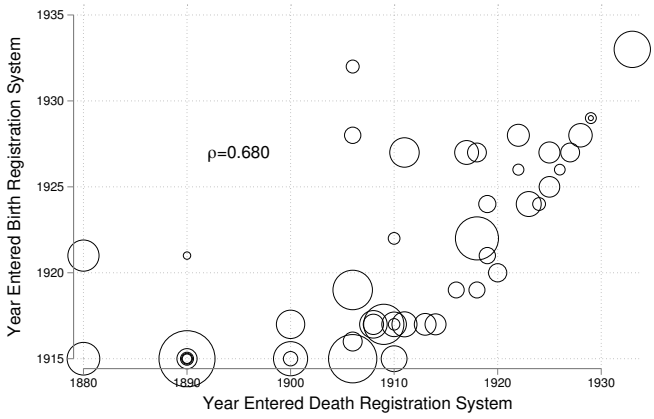
(c) Birth Registration System Entry and Black Population Share



(d) Death Registration System Entry and Black Population Share



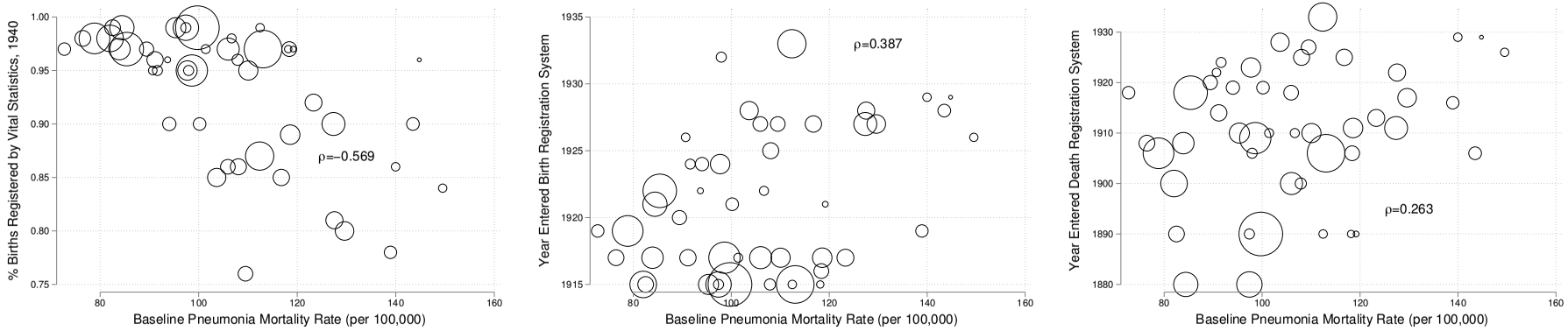
(e) Proxy for Errors in Age at Death by Race



(f) Birth and Death Registration System Entry Date

Notes: Panel (a) plots a measure of birth registration completeness in 1940 (from Shapiro and Schachter (1952)) 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 Black Americans 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. Panel (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.

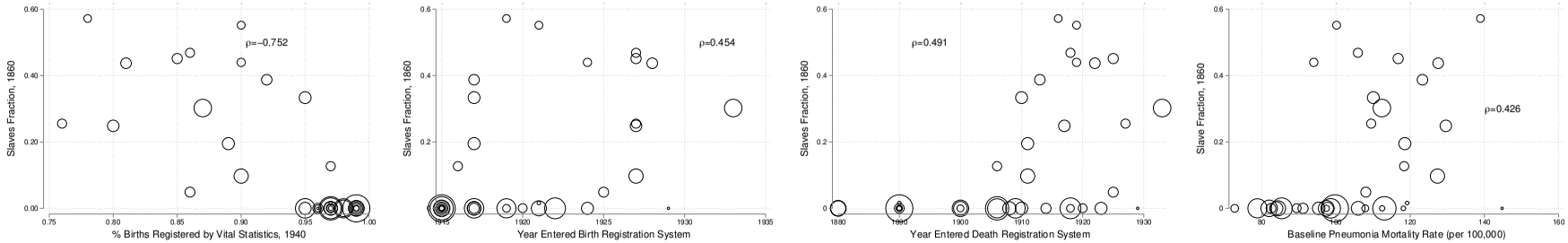
Figure D3: Proxies for measurement error and baseline pneumonia mortality



(a) Birth Registration Completeness and Pneumonia Mortality (b) Birth Registration Year and Pneumomia Mortality (c) Death Registration Year and Pneumomia Mortality

Notes: Bivariate plots show raw correlations between state-level baseline pneumonia mortality (1930-1936) and proxies of measurement error discussed in Section D.

Figure D4: Cross-state correlations of indicators of the quality of mortality rates, base pneumonia and slave fraction

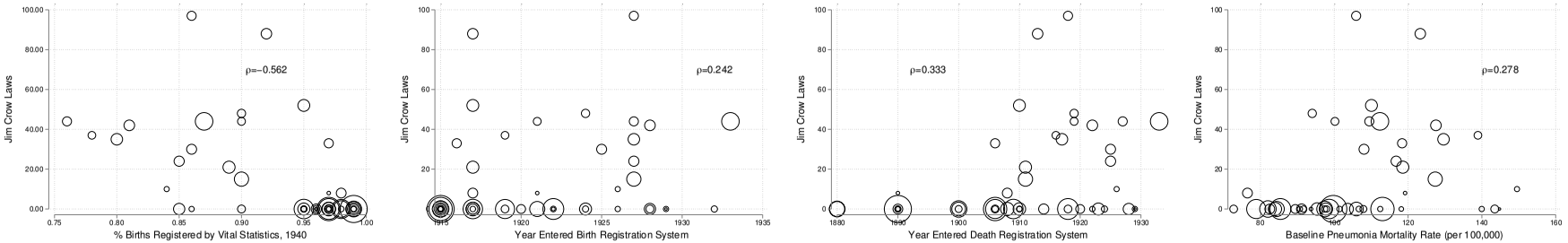


(a) Slave fraction and birth registration completeness

(b) Slave fraction and birth registration entry year

(c) Slave fraction and death registration entry year

(d) Slave fraction and base pneumonia mortality



(e) Jim Crow laws and birth registration completeness

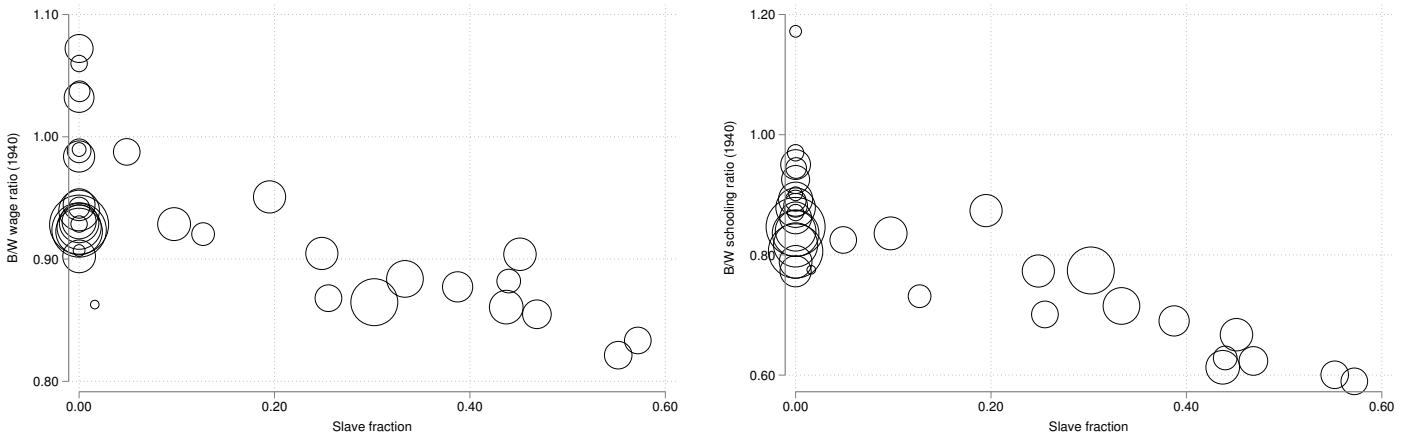
(f) Jim Crow laws and birth registration entry year

(g) Jim Crow laws and death registration entry year

(h) Jim Crow laws and base pneumonia mortality

Notes: Correlations are presented between proxies of discrimination and indicators of the quality of Vital Statistics registration—birth registry completeness, and the years of entry to the birth and death registration systems respectively (panels (a)-(c) and (e)-(g)), and with baseline pneumonia mortality (panels (d) and (h)). Proxies of discrimination are the historical share of enslaved persons (top row) and number of Jim Crow laws (bottom row) Each point represents a state, with sizes relative to state populations.

Figure D5: Historical share of enslaved persons and wage and schooling differentials

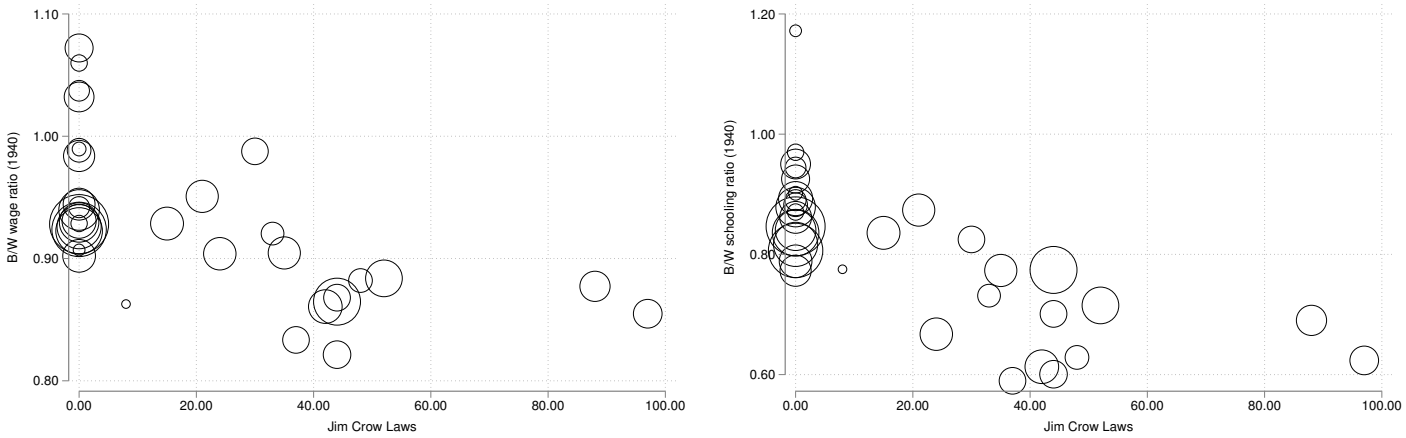


(a) Slave share and B/W wage ratio

(b) Slave share and B/W schooling ratio

Notes: Plots display contemporary (1930) state-level correlates with the historical share of enslaved persons. Right-hand panel presents the white to black wage ratio estimated from 1930 census microdata and left-hand panel presents the white to black schooling ratio estimated from 1930 census microdata.

Figure D6: Jim Crow laws and wage and schooling differentials



(a) Jim Crow laws and B/W wage ratio

(b) Jim Crow laws and B/W schooling ratio

Notes: Plots display contemporary (1930) state-level correlates with the number of Jim Crow laws. Right-hand panel presents the white to black wage ratio estimated from 1930 census microdata and left-hand panel presents the white to black schooling ratio estimated from 1930 census microdata.

E Specification and Robustness Checks

E.1 Additional Details on Robustness Checks

In this Appendix we collect additional robustness checks. We lay out key details of each of these tests below. Results are presented in Section E.2 in the order they are discussed in the text.

E.1.1 Long-run impacts of sulfa – Additional Results and Further Checks

Placebo break years In Table E7 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 alternative breaks. We replace Post sulfa with each year in the interval [1933,1938]. These results show that the first consistently positive jumps (for positive outcomes like income; the jumps being negative for negative outcomes like disability) 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-1933 or post-1934 and the significant positive coefficients for post-1935 and 1936 are consistent with much of the post- group actually being exposed, while post-1938 are consistent with sulfa drugs having already arrived in the preceding year.

Selective migration of parents of sample cohorts See Section 3.2 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. 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 sulfa$_t$ $\times$ Base Exposure$_s$, and the controls in equation (1). The results are presented in Table E2 of this Appendix. There is no evidence of selective migration.[55]

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. To test this, 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 E3 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 3.2 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, race, education, work status of mother, and household income) as dependent variables. The results are in Table E4 of this Appendix.

[55] 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).

Additional robustness checks Table E1 of this Appendix presents checks that complement those reported in the main text in Section 3.2. These are listed and briefly discussed here, where letters refer to the Panel of Table E1 which corresponds to each set of results.

A Instead of using combined pneumonia and influenza mortality as a measure of baseline exposure, we use a measure of pneumonia mortality available in 1935. For discussion, refer to Appendix D.1.

B Instead of using all age mortality as a measure of baseline exposure, we use infant mortality rates owing to pneumonia and influenza. For discussion of this, refer to Appendix D.1.

C Controls for measurement error in mortality rates are incorporated. Specifically, Post sulfa$_t$ is also interacted with birth system registration completeness in 1930, which we document is a proxy for vital statistics data quality. For discussion of this, refer to Appendix D.2

D Incorporates state-level controls for New Deal spending from Fishback et al. (2003) interacted with Post$_t$.

E Removes states exposed to the Dust Bowl (New Mexico, Oklahoma, Texas, Nebraska, Kansas and Colorado) from the estimation sample.

F Removes cohorts born 1941 and beyond as cohorts born during US involvement in World War II.

G Restricts the sample to 1935-1941. This restriction allows us to exclude early Depression Era cohorts and World War II cohorts, with the results remaining similar to baseline results.

H Removes the sample of birth cohorts born immediately around sulfa's arrival (1935-1937). This 'donut' design compares individuals exposed in infancy (1938 birth cohort onwards), with individuals exposed from only later infancy (1934 and prior birth cohorts).

I-J Excluding (Panel K) or using exclusively (Panel L) 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 A.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.

K Using Post$_t$ $\times$ Base Exposure$_s$ as an instrument for birth cohort $\times$ birth state (yearly) pneumonia and influenza mortality. 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.

Event Studies and Baseline Years Our baseline event studies follow a suggestion of Miller (2023) of setting all pre-sulfa year dummies equal to zero as a baseline comparison instead of arbitrarily selecting a single pre-treatment baseline reference period. In Figure E1 we present versions which set birth cohort 1935 as the omitted baseline group (race- and gender-specific estimates are presented in Figure F3). Alternatively, if we are concerned that individuals which were exposed not during their very early life, but rather the first few years of life do not act as an ideal control, we can consider baseline comparisons based on older children. Figure E2 presents versions in which the baseline reference group of older children who were completely unexposed during the first 5 years of their life as baseline (birth cohorts 1930-1931).

E.1.2 Gradients in Long-Run Impacts: Additional Results and Further Checks

As discussed in the paper, the main threats to identification and interpretation of the gradients in long-run impacts for black men by institutional discrimination are differences in access to sulfa drugs, migration, and survival selection. These issues were addressed in the paper. In this section we further elaborate, and describe additional robustness checks.

Discrimination gradients for white men and women Table E5 of this Appendix reports the analogue of Table 4 in the paper for white men and women. In sharp contrast to the case for black men and women, there is no evidence that the long run returns to sulfa drugs were smaller in more discriminatory states. For most outcomes, the coefficients on Post sulfa$_t$ $\times$ Base Exposure$_s$ $\times$ Discrimination Proxy$_s$ are not significantly different from zero, and gradients documented at the foot of the table are small in magnitude—typically an order of magnitude smaller than gradients documented in Table 4. This suggests that the gradients for black men and women are driven by a process specific to this population group rather than by institutional features of discriminatory states that worsened outcomes for everyone.

The Great Migration Refer to section 3 of the paper which motivates discussion of whether selective black migration from the South to the North might explain the discrimination 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 Black Americans 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 sulfa$_t$ $\times$ Discrimination Proxies$_s$, which is included in models presented in Table 4. 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 3 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 sulfa$_t$ $\times$ Base Exposure $\times$ Discrimination Proxy$_s$ 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 Black Americans in the North and South. Hence, accounting for South to North migration would only strengthen the discrimination 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 E2 of this Appendix). In Appendix Table E3, 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 Black individuals because they had higher mortality rates then this could bias both Post sulfa$_t$ $\times$ Base Exposure$_s$ and Post sulfa$_t$ $\times$ Base Exposure$_s$ $\times$ Discrimination Proxy$_s$. 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 more discriminatory states that we find. So as to estimate the empirical significance of differential selective survival rates of Black Americans in more vs. less discriminatory 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 E6 of this Appendix).

Age heaping If numeracy was correlated with discrimination 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 discrimination 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 (Appendix Figure E3).

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, 2010; Venkataramani, 2012). This is pertinent for Black Americans in the South, 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 discrimination gradients may be that, in response to the positive health shock, Black men and women 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 Black persons 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 raise 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 Black Americans in the South than for any other group, but we see the opposite. So, accounting for the Civil Right Acts of the 1960s would only strengthen our finding that Southern Black men and women 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 sulfa$_t$ $\times$ Discrimination Proxy$_s$ helps account for any birth-cohort differences in participation in the Civil Rights movement across states with different levels of discrimination.

An Event-study Model of Long-Run Gradients Specifications examining long-term gradients based on discrimination proxies in (3) are based on single coefficient models. We thus may be concerned that any gradients observed in outcomes are picking up differential trends, or that Post sulfa$_t$ $\times$ Discrimination Proxy$_s$ is otherwise not adequately capturing level differences between more and less discriminatory areas. For this reason, we implement full event study versions of (3), allowing us to determine whether outcome gradients

only appear in cohorts born after the arrival of sulfa. We estimate:

$$\begin{aligned} Y_{istc} \quad = \quad & \alpha + \sum_{j=1930}^{1943} \gamma_j \left(1\{\text{Year}_t = j\} \times \text{Base Pneumonia}_s\right) \\ & + \sum_{j=1930}^{1943} \delta_j \left(1\{\text{Year}_t = j\} \times \text{Base Pneumonia}_s \times \text{Discrimination}_s\right) \\ & + \theta_s + (\eta_t \times \mu_r) + \lambda_c + X'_{st}\Gamma + \varepsilon_{istc}, \end{aligned} \tag{7}$$

where all details follow (2), however now with the added inclusion of the series of interaction terms and corresponding coefficients δ_j.

In Figures E4 (historical fraction of enslaved persons) and E5 (Jim Crow laws) we present results for these models respectively for Black men and women, and White men and women. These present marginal effects at the 10$^{\text{th}}$ and 90$^{\text{th}}$ percentile of Discrimination Proxy$_s$, which represent the quantities $\gamma_j + \delta_j \times$ Discrimination Proxy Percentile for each year j. We observe in each case that there is little evidence of deviation of pre-sulfa trends from 0 prior to fully affected birth cohorts, while clear changes are observed following sulfa. For Black men and women, such impacts are appreciable in areas with low proxies for discrimination but not in areas with high discrimination, while for White men and women, no such gradient is evident.

E.2 Tables and Figures

Table E1: Full Robustness Checks

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A - Alternative Base Measures				
Post Sulfa × Base 1935 Exposure	0.311***	0.0572***	0.0286***	-0.0132***
	(0.0694)	(0.0149)	(0.00761)	(0.00280)
Effect size for an interquartile shift in base exposure	0.0640 years	1.179 %	0.589 pp	-0.273 pp
Observations	1,350,829	3,875,492	3,950,318	3,950,318
Panel B - Infant Pneumonia Measure				
Post Sulfa × Base Exposure (Infant)	0.0323***	0.00397***	0.00151***	-0.000269
	(0.00862)	(0.00144)	(0.000538)	(0.000289)
Effect size for an interquartile shift in base exposure	0.0420 years	0.515 %	0.197 pp	-0.0349 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel C - Measurement Error Controls				
Post Sulfa × Base Exposure	0.172**	0.0583***	0.0187**	-0.00858**
	(0.0801)	(0.0113)	(0.00759)	(0.00367)
Effect size for an interquartile shift in base exposure	0.0484 years	1.640 %	0.525 pp	-0.241 pp
Observations	1,433,935	4,110,227	4,190,632	4,190,632
Panel D - Controlling for Depression & New Deal Spending				
Post Sulfa × Base Exposure	0.245**	0.0554***	0.0180**	-0.00707*
	(0.0946)	(0.0144)	(0.00736)	(0.00394)
Effect size for an interquartile shift in base exposure	0.0688 years	1.556 %	0.506 pp	-0.199 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel E - No Dust Bowl States				
Post Sulfa × Base Exposure	0.362***	0.0472***	0.0140	-0.0108***
	(0.0736)	(0.0118)	(0.0100)	(0.00322)
Effect size for an interquartile shift in base exposure	0.102 years	1.328 %	0.392 pp	-0.302 pp
Observations	1,263,857	3,628,879	3,699,045	3,699,045
Panel F - Excluding WW II Cohorts				
Post Sulfa × Base Exposure	0.213**	0.0486***	0.0206***	-0.00857*
	(0.0996)	(0.0139)	(0.00600)	(0.00455)
Effect size for an interquartile shift in base exposure	0.0599 years	1.366 %	0.578 pp	-0.241 pp
Observations	1,081,022	3,062,341	3,122,572	3,122,572
Panel G - 1935–1941 Cohorts Only				
Post Sulfa × Base Exposure	0.292***	0.0507***	0.0109*	-0.0118*
	(0.0922)	(0.0132)	(0.00598)	(0.00590)
Effect size for an interquartile shift in base exposure	0.0821 years	1.426 %	0.307 pp	-0.333 pp
Observations	687,376	1,993,810	2,032,795	2,032,795
Panel H - 'Donut' specification (no 1935–1937 Cohorts)				
Post Sulfa × Base Exposure	0.216*	0.0470***	0.0271***	-0.00622

Continued on next page

Table E1 – continued from previous page

	Schooling	log(Family Income)	Employment	Work Limiting Disability
	(0.127)	(0.0162)	(0.00748)	(0.00615)
Effect size for an interquartile shift in base exposure	0.0608 years	1.321 %	0.762 pp	-0.175 pp
Observations	792,813	2,229,988	2,274,368	2,274,368
Panel I - Excluding 2000 Census				
Post Sulfa × Base Exposure	0.199**	0.0402**	0.0203**	-0.0148***
	(0.0872)	(0.0170)	(0.00861)	(0.00533)
Effect size for an interquartile shift in base exposure	0.0560 years	1.131 %	0.569 pp	-0.416 pp
Observations	1,433,937	2,814,710	2,862,237	2,862,237
Panel J - 2000 Census Only				
Post Sulfa × Base Exposure	0.309***	0.0694**	0.00620	0.00428
	(0.0649)	(0.0275)	(0.0117)	(0.00862)
Effect size for an interquartile shift in base exposure	0.0869 years	1.952 %	0.174 pp	0.120 pp
Observations	1,328,396	1,295,517	1,328,396	1,328,396
Panel K - Measurement Error, 2SLS				
Pneumonia-Influenza Mortality Rate	-0.398**	-0.0920***	-0.0331**	0.0176**
	(0.179)	(0.0256)	(0.0155)	(0.00673)
Effect size for an interquartile shift in base exposure	0.0560 years	1.294 %	0.466 pp	-0.247 pp
Observations	1,422,298	4,079,298	4,158,902	4,158,902

Notes: Each column-panel represents a separate regression. Models include the same controls as those presented in Table 1, and represent robustness checks of these estimates. The sample of interest here is individuals of all demographic groups (Black and white women and men). The models are otherwise identical to those generating the core estimates, with the following additions or changes: Panel A: Baseline rate measure (combining influenza and pneumonia) is replaced with an alternative measure which reports pneumonia mortality rates for 1935. Panel B: Baseline rate measure is replaced with the average pre-intervention pneumonia-influenza mortality rates for infants. Panel C: Controls for Post_t × Birth System Registration Completeness in 1930_s, a measure of birth under-reporting that proxies for vital statistics quality. Panel D: New Deal spending (Fishback et al., 2003) is interacted with post-sulfa, and included as an additional control in all models. Panel E: States affected by the Dust Bowl (New Mexico, Oklahoma, Texas, Nebraska, Kansas, and Colorado) are removed from the estimation sample. Panel F: Cohorts born during US involvement in World War II (1941 onwards) are excluded. Panel G: We restrict our sample to the 1935-1941 birth cohorts, which excludes both the early Depression years and World War II. Panel H: Removes the cohorts born in the immediate pre- and post-sulfa arrival periods (1935–1937) as a 'donut' specification. Panel I: Removes 2000 census data. Panel J: Uses only 2000 census data. Panel K: Uses Post×Baseline pneumonia-influenza mortality as an IV for annual influenza-pneumonia mortality in the birth year. Here we expect a sign-reversal as positive treatment indicator in the reduced form is replaced with a mortality rate in 2SLS. Standard errors clustered at the birth state level are reported in parentheses. In each case, the estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) is provided in panel footers as *Effect Size*.

*** p<0.01; ** p<0.05; * p<0.10.

Table E2: Testing for Endogenous South-North Migration of Parents

	Full Sample		Some High School +	
	(1)	(2)	(3)	(4)
Base Exposure	-0.103*	-0.00937	-0.161*	-0.0188
	(0.0543)	(0.0120)	(0.0848)	(0.0221)
Mean of Dependent Variable	0.0194	0.0194	0.0284	0.0284
Observations	44,271	44,271	26,218	26,218

Notes: Marginal effects are presented from Probit regressions of the probability of moving Northward between 1935 and 1940 among those Black individuals of child bearing age living in Southern states in 1935 as a function of Base Pneumonia mortality rates. Data consist of all individuals observed in the 1940 census. Controls in column 1 include age and sex FE, and standard errors clustered by state are consistently reported. In column 2 we additionally add state per capita income, under-2 diarrheal mortality, and heart disease mortality. In columns 3 and 4, estimates are presented for individuals with "Some High School and Beyond" as this represents the median of the black schooling distribution. Standard error clustered by states are included in parentheses.
*** p<0.01; ** p<0.05; * p<0.10.

Table E3: Testing for Migratory Responses

	All	White	Black	Black
	(1)	(2)	(3)	(4)
Panel A: Men and Woman				
Post Sulfa × Base Exposure	-0.0128	-0.00486	-0.0633*	-0.0643**
	(0.0146)	(0.0132)	(0.0318)	(0.0301)
Post Sulfa × Base Exposure × Slave Fraction				-0.00957
				(0.0203)
Observations	4,329,176	3,899,448	429,728	422,737
Panel B: Men				
Post Sulfa × Base Exposure	-0.00731	0.00152	-0.0697*	-0.0623
	(0.0155)	(0.0135)	(0.0411)	(0.0411)
Post Sulfa × Base Exposure × Slave Fraction				-0.0376
				(0.0240)
Observations	2,103,072	1,913,370	189,702	186,565
Panel C: Women				
Post Sulfa × Base Exposure	-0.0181	-0.0110	-0.0580*	-0.0659**
	(0.0144)	(0.0135)	(0.0302)	(0.0254)
Post Sulfa × Base Exposure × Slave Fraction				0.0118
				(0.0229)
Observations	2,226,104	1,986,078	240,026	236,165

Notes: Model specification is as in Table 1. Now the dependent variable is migration, which is = 1 if the individual reports 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. Standard errors clustered by state are provided in parentheses.
*** p<0.01; ** p<0.05; * p<0.10.

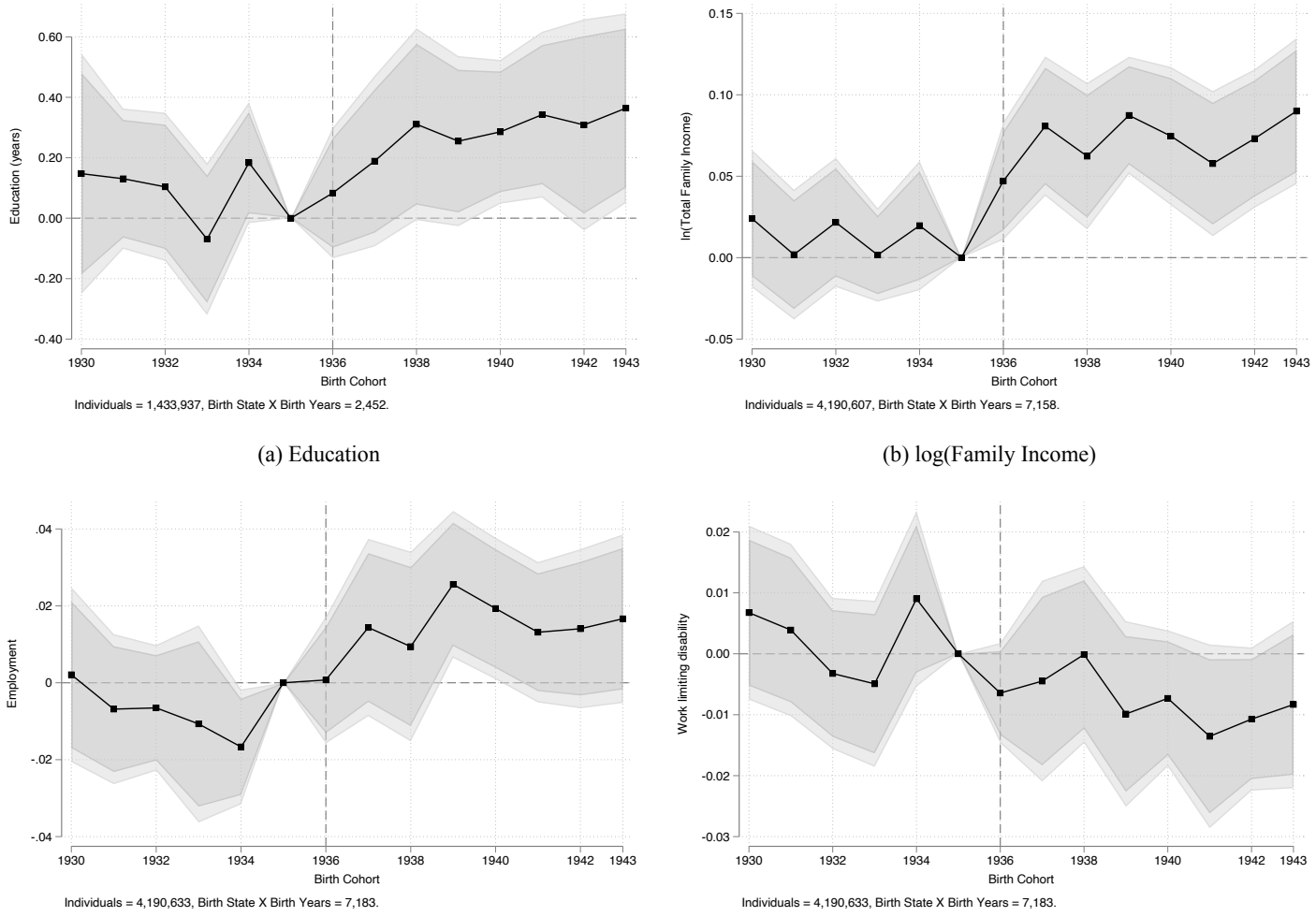
Table E4: Testing for Fertility Selection

	Mother's Education	Mother's Age	Mother's Working	Household Income	Black
Post Sulfa × Base Exposure	-0.131	0.201	-0.0137	-186.3	0.0148
	(0.122)	(0.600)	(0.0152)	(114.3)	(0.0103)
Observations	439,168	439,168	439,168	85,248	485,766

Notes: Each cell presents a separate regression of maternal or household characteristics of mothers observed in 1940 census microdata to test for selection. Model specifications are the same as in Table 1, but work with data from the 1940 census. Standard errors clustered by state are presented in parentheses.

*** p<0.01; ** p<0.05; * p<0.10.

Figure E1: Event Study Estimates With a Single Baseline Year



(a) Education

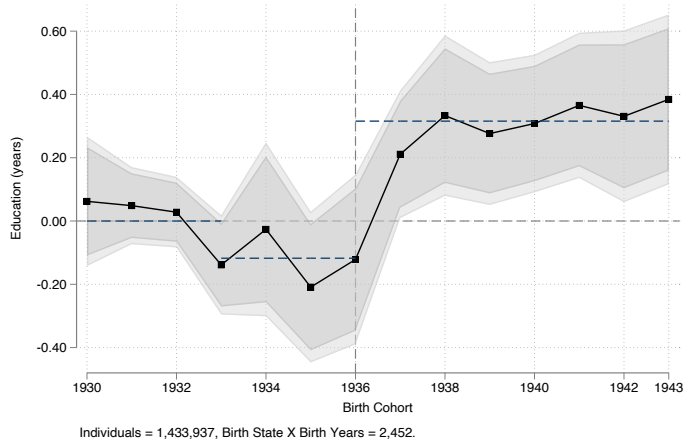
(b) log(Family Income)

(c) Employment

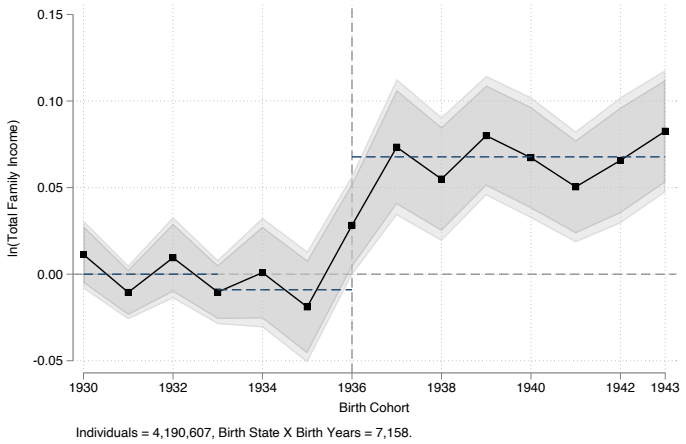
(d) Work-Limiting Disability

Notes: Refer to notes to Figure 4. Identical models are presented, however here using a single baseline year of 1935 as the omitted reference group. All other details follow Figure 4.

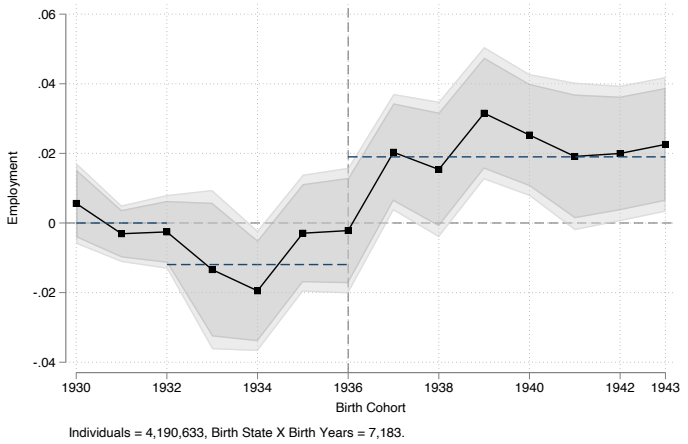
Figure E2: Event Study Estimates Using Older Exposed as Baseline



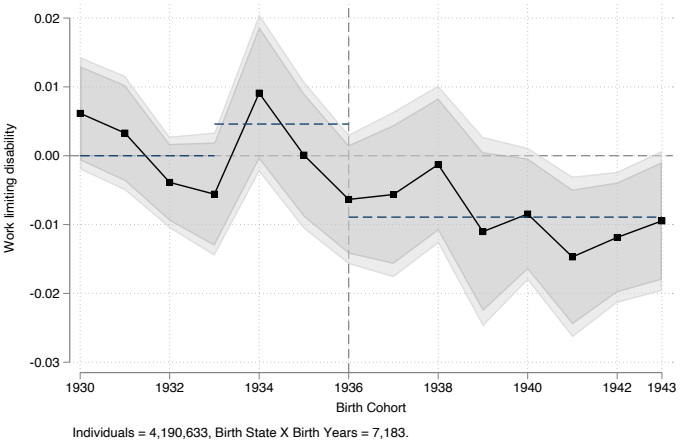
(a) Education



(b) log(Family Income)



(c) Employment



(d) Work-Limiting Disability

Notes: Refer to notes to Figure 4. Identical models are presented, however here using birth cohorts from 1930-1933 (older children at sulfa's arrival) as the baseline reference group. Dashed lines represent average coefficients among the baseline reference years (constrained at zero), those exposed after birth but at a young age (cohorts 1934-1936), and those exposed from birth onwards (cohorts 1937-1943). All other details follow Figure 4.

Table E5: Gradients in Long Run Impacts of Infant Pneumonia Exposure by Measures of Systemic Discrimination – White Men and Women

	Gradient: Historical Fraction of Enslaved People				Gradient: Number of Jim Crow Laws			
	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: White Men								
Post Sulfa × Base Exposure	0.489***	0.0577**	0.0261*	-0.0109***	0.337***	0.0714***	0.00975	-0.0131***
	(0.143)	(0.0248)	(0.0129)	(0.00382)	(0.101)	(0.0183)	(0.00886)	(0.00405)
FWER p-value	[0.225]	[0.437]	[0.311]	[0.308]	[0.021]	[0.008]	[0.305]	[0.020]
Post Sulfa × Base Exposure × Discrimination Proxy	0.174	-0.0146	-0.0599*	0.0228**	0.00878**	-0.0000206	-0.0000946	0.000173
	(0.448)	(0.0775)	(0.0348)	(0.0112)	(0.00394)	(0.000745)	(0.000321)	(0.000124)
FWER p-value	[0.930]	[0.858]	[0.418]	[0.355]	[0.157]	[0.978]	[0.950]	[0.464]
Effect size at bottom decile of discrimination proxy	0.131 years	1.676 %	0.951 pp	-0.390 pp	0.0555 years	2.016 %	0.316 pp	-0.446 pp
Effect size at top decile of discrimination proxy	0.153 years	1.496 %	0.211 pp	-0.107 pp	0.164 years	1.990 %	0.199 pp	-0.232 pp
Observations	583,489	1,683,128	1,713,946	1,713,946	635,279	1,829,870	1,863,597	1,863,597
Panel B: White Women								
Post Sulfa × Base Exposure	0.315***	0.0433***	0.0343**	-0.00849	0.228**	0.0358***	0.0283***	-0.00845*
	(0.103)	(0.0125)	(0.0128)	(0.00612)	(0.0981)	(0.0127)	(0.00813)	(0.00471)
FWER p-value	[0.142]	[0.138]	[0.200]	[0.295]	[0.083]	[0.049]	[0.016]	[0.136]
Post Sulfa × Base Exposure × Discrimination Proxy	-0.401	0.0139	-0.0591	0.00280	-0.00374	0.000491	-0.000309	0.0000328
	(0.321)	(0.0442)	(0.0404)	(0.0191)	(0.00365)	(0.000464)	(0.000342)	(0.000192)
FWER p-value	[0.506]	[0.946]	[0.542]	[0.891]	[0.660]	[0.754]	[0.641]	[0.877]
Effect size at bottom decile of discrimination proxy	0.103 years	1.168 %	1.178 pp	-0.249 pp	0.0809 years	0.786 %	0.933 pp	-0.252 pp
Effect size at top decile of discrimination proxy	0.0536 years	1.340 %	0.447 pp	-0.214 pp	0.0346 years	1.394 %	0.551 pp	-0.212 pp
Observations	596,683	1,751,194	1,779,217	1,779,217	649,412	1,903,470	1,933,966	1,933,966

Notes: Refer to Notes to Table 4. This Table is identical however now presents results for white men (panel A) and white women (panel B).

*** p<0.01; ** p<0.05; * p<0.10.

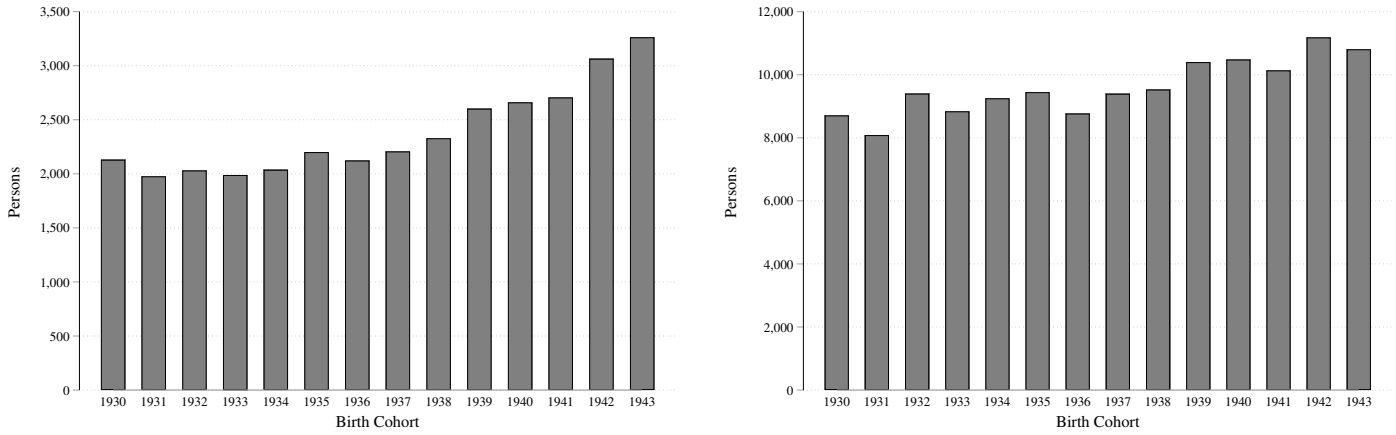
Table E6: Gradients in Long Run Impacts of Infant Pneumonia Exposure by Measures of Systemic Discrimination Accounting for Survival Section – Black Men

	Gradient: Historical Fraction of Enslaved People				Gradient: Number of Jim Crow Laws			
	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Baseline Sample								
Post Sulfa × Base Exposure	1.459***	0.327***	0.159***	-0.0750***	0.831**	0.252***	0.185***	-0.0606***
	(0.261)	(0.0756)	(0.0342)	(0.0247)	(0.323)	(0.0503)	(0.0383)	(0.0164)
FWER p-value	[0.019]	[0.073]	[0.072]	[0.129]	[0.062]	[0.018]	[0.019]	[0.049]
Post Sulfa × Base Exposure × Discrimination Proxy	-3.957***	-0.602***	-0.305***	0.238***	-0.0266***	-0.00497***	-0.00387***	0.00229***
	(0.753)	(0.206)	(0.0968)	(0.0643)	(0.00948)	(0.00175)	(0.00107)	(0.000463)
FWER p-value	[0.022]	[0.024]	[0.034]	[0.058]	[0.060]	[0.087]	[0.036]	[0.025]
Effect size at bottom decile of discrimination proxy	0.554 years	11.37 %	5.570 pp	-2.974 pp	0.353 years	9.306 %	6.920 pp	-2.726 pp
Effect size at top decile of discrimination proxy	0.0645 years	3.934 %	1.793 pp	-0.0292 pp	0.0231 years	3.160 %	2.135 pp	0.106 pp
Observations	65,266	161,583	170,601	170,601	66,597	164,497	173,715	173,715
Panel B: Selection Adjusted Sample								
Post Sulfa × Base Exposure	3.132***	0.465***	0.260***	-0.220***	2.173***	0.360***	0.264***	-0.173***
	(0.491)	(0.0883)	(0.0358)	(0.0432)	(0.471)	(0.0545)	(0.0345)	(0.0339)
FWER p-value	[0.024]	[0.050]	[0.017]	[0.044]	[0.009]	[0.008]	[0.004]	[0.020]
Post Sulfa × Base Exposure × Discrimination Proxy	-4.474***	-0.624**	-0.317***	0.268**	-0.0472***	-0.00665***	-0.00497***	0.00389***
	(1.100)	(0.229)	(0.104)	(0.100)	(0.0117)	(0.00182)	(0.00100)	(0.000849)
FWER p-value	[0.033]	[0.097]	[0.077]	[0.093]	[0.049]	[0.030]	[0.039]	[0.049]
Effect size at bottom decile of discrimination proxy	1.043 years	15.33 %	8.450 pp	-7.152 pp	0.822 years	13.10 %	9.643 pp	-6.600 pp
Effect size at top decile of discrimination proxy	0.490 years	7.613 %	4.537 pp	-3.835 pp	0.238 years	4.868 %	3.490 pp	-1.790 pp
Observations	66,442	164,447	173,465	173,465	67,783	167,382	176,600	176,600

Notes: Refer to Notes to Table 4. This Table reports results for black men as in the original analysis (panel A), and accounting for survival selection in a conservative way (panel B). To account for the fact that pneumonia mortality rates were higher for black men, and even higher for black men in the Southern, more discriminatory 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).

*** p<0.01; ** p<0.05; * p<0.10.

Figure E3: Age heaping among Black Americans in Southern vs. non-Southern states

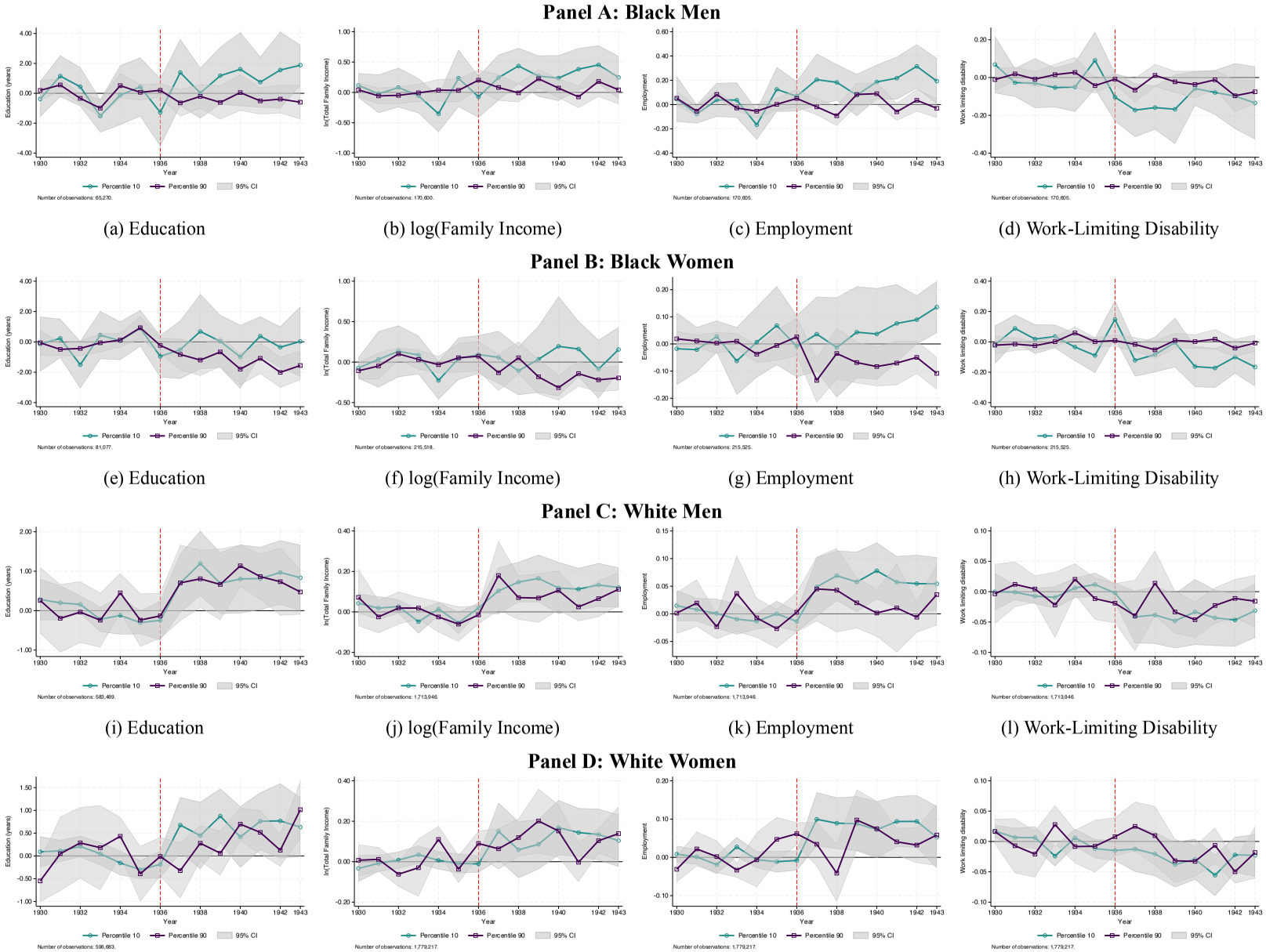


(a) Non-Southern States

(b) Southern States

Notes: These are plots of cohort size by birth cohort in the 1980 census sample for Black Americans. 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.

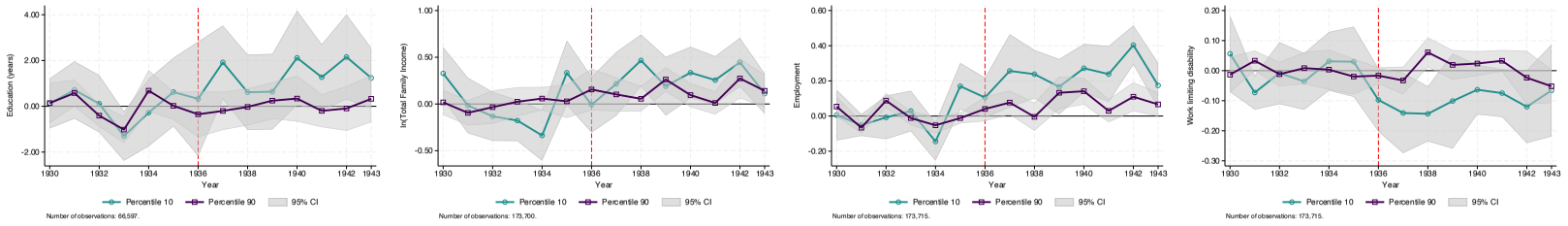
Figure E4: Long-term Returns: Historical Fraction of Enslaved Persons Mediation Event Studies



Notes: Each plot presents estimates from (7) at the 10th and 90th percentile of the historical enslaved persons discrimination proxy. These marginal effects and their 95% CIs are presented based on the coefficients γ_j and δ_j in (7), with years j indicated on the horizontal axis of plots. Coefficients for γ_j are constrained to be mean 0 in the pre-treatment period as a baseline reference, and an identical reference (mean 0) is used for δ_j. Other details follow main event studies discussed in Figure 4.

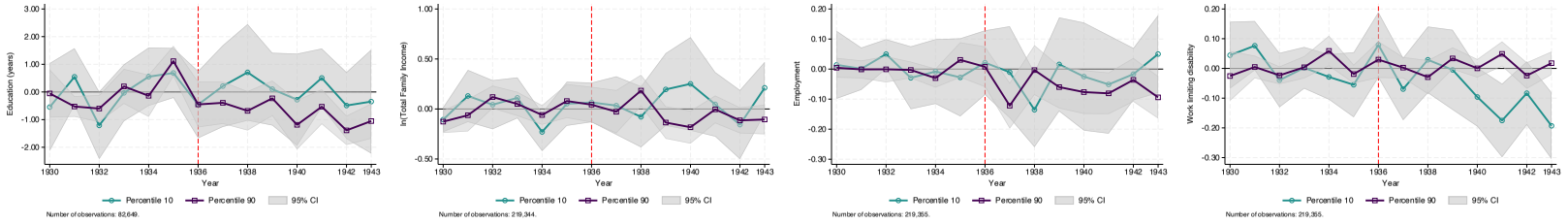
Figure E5: Long-term Returns: Jim Crow Law Mediation Event Studies

Panel A: Black Men



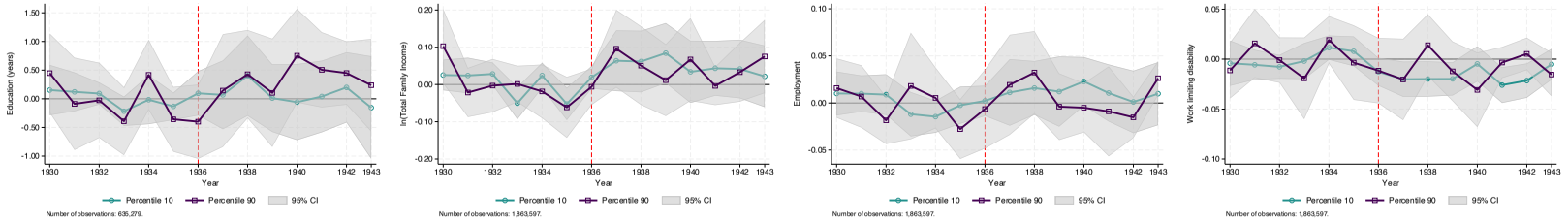
(a) Education (b) log(Family Income) (c) Employment (d) Work-Limiting Disability

Panel B: Black Women



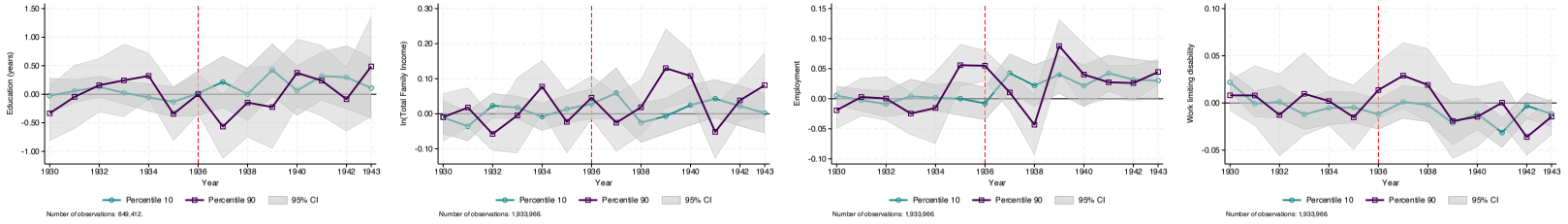
(e) Education (f) log(Family Income) (g) Employment (h) Work-Limiting Disability

Panel C: White Men



(i) Education (j) log(Family Income) (k) Employment (l) Work-Limiting Disability

Panel D: White Women



(m) Education (n) log(Family Income) (o) Employment (p) Work-Limiting Disability

Notes: Refer to Note to Figure E4. Identical event studies are shown, however now with gradients in terms of Jim Crow laws.

Table E7: Adult Outcomes Associated with False Trend Breaks in Birth Years Surrounding 1937

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A: Post$_t$ = 1 for 1933 onwards				
Post Sulfa 1934 × Base Exposure	-0.0324	0.00996	0.000794	-0.00417
	(0.102)	(0.0119)	(0.00596)	(0.00377)
Effect size for an interquartile shift in base exposure	-0.00910 years	0.280 %	0.0223 pp	-0.117 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel B: Post$_t$ = 1 for 1934 onwards				
Post Sulfa 1935 × Base Exposure	0.0488	0.0196*	0.00414	-0.00201
	(0.0997)	(0.00986)	(0.00674)	(0.00365)
Effect size for an interquartile shift in base exposure	0.0137 years	0.552 %	0.116 pp	-0.0564 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel C: Post$_t$ = 1 for 1935 onwards				
Post Sulfa 1936 × Base Exposure	0.0220	0.0234**	0.0114**	-0.00683*
	(0.0796)	(0.00999)	(0.00524)	(0.00355)
Effect size for an interquartile shift in base exposure	0.00619 years	0.658 %	0.319 pp	-0.192 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel D: Post$_t$ = 1 for 1936 onwards				
Post Sulfa 1937 × Base Exposure	0.108	0.0449***	0.0130**	-0.00874***
	(0.0759)	(0.00882)	(0.00528)	(0.00297)
Effect size for an interquartile shift in base exposure	0.0303 years	1.262 %	0.367 pp	-0.246 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel E: Post$_t$ = 1 for 1937 onwards				
Post Sulfa 1938 × Base Exposure	0.199**	0.0497***	0.0172**	-0.00816**
	(0.0872)	(0.0120)	(0.00736)	(0.00354)
Effect size for an interquartile shift in base exposure	0.0560 years	1.399 %	0.485 pp	-0.230 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633
Panel F: Post$_t$ = 1 for 1938 onwards				
Post Sulfa 1935 × Base Exposure	0.0488	0.0196*	0.00414	-0.00201
	(0.0997)	(0.00986)	(0.00674)	(0.00365)
Effect size for an interquartile shift in base exposure	0.0137 years	0.552 %	0.116 pp	-0.0564 pp
Observations	1,433,937	4,110,228	4,190,633	4,190,633

Notes: Each column represents a separate regression. The models are identical to those presented in Table 1, except Post is refined to be =1 for a number of alternative years (1933, 1934, 1935, 1936, and 1938). The positive impact estimates are largest when Post is set to 1937 (Panel E, which is identical to Table 1), which is consistent with the arrival of sulfa drugs that year. In each case, the estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) is provided in panel footers as *Effect Size*.

*** p<0.01; ** p<0.05; * p<0.10.

F Alternative Outcomes and Full Results by Demographic Group

F.1 Additional Outcomes

In the body of the paper we present results for 4 main outcomes: educational attainment, family income, employment and work-limiting disability. We additionally generate results on a number of derived measures or alternative outcomes (whether an individual completes high school and college, whether their family income is below 200% of the national poverty line and whether they report cognitive disability), which are presented in this Appendix.

Results are presented which correspond to each of the main exhibits in the paper. Specifically, results which correspond to Table 1 are presented in Appendix Table F1. Results which correspond to Table 3 are presented in Appendix Table F3. Results which correspond to Table 2 are presented in Appendix Table F2. Results which correspond to Table 4 are presented in Appendix Table F12. Results which correspond to Figure 4 are presented in Appendix Figure F2.

We additionally present results examining effects of sulfa exposure on family income by specific percentiles of the (pre-sulfa) income distribution. These are displayed in Appendix Figure F1 (all individuals), and Appendix Figure F4 (group-specific estimates).

F.2 Alternative Gender by Race Estimates

F.2.1 Principal Results

We present results for each demographic group which correspond to all main results in the paper. In the case of long-term impacts presented in Table 1, race and gender-specific estimates are already presented in main text as Table 3 For event studies in Figure 4, group-specific results are presented as Figure F3. In the case of long-run returns based on exposure to sulfa diffusion (Table 2), group-specific results are presented in Table F8. The robustness of estimates by group (corresponding to Figure 6) is presented as Figure F6.

F.2.2 Additional Results for Women

Estimates of the long run returns to birth year exposure to pneumonia for women are in Table 3. Results are presented for both Black and White women and men, and in general, results for women are more muted than those for men. In the case for black women, unlike all other groups we observe, certain results suggest *declines* in long term measures of human capital, rather than improvements. What's more, results in Figure F6 suggest that with some exceptions, these results are not highly dependent upon control sets. For example, in the case of black women, when considering both education and total family income, across all control sequences we observe evidence consistent with either significant negative effects or at most null effects in the long run. White women on the other hand generally have results broadly consistent with those of white men, though typically more muted in magnitude.

In this section, we investigate potential explanations for the gender difference in long run effects of pneumonia exposure, as well as the difference in long run effects between black women and white women. One explanation of differential responses by gender 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 F7a in this Appendix). There is also a large gender difference in mortality from pneumonia; see Figure F7b below which plots all-age influenza and pneumonia mortality rates over time.[56] 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, $\text{Post}_t \times \text{Base Pneumonia}_s$ with indicators of gender differences in educational investments and labor market outcomes. In particular, we create two measures—the female-male 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 (2017), 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 F9 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 very little support for this, with largely insignificant 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 discriminatory 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 clear evidence that black women's education and labor market outcomes benefited from sulfa-drug exposure in infancy, and observe small effects among white women, we proceeded to explore impacts on marriage market outcomes. Using data from the 1980 census, we investigated marital status, as well as a range of measures of fertility (both at the extensive and intensive margin). As seen in Table F10 and

[56] For a discussion of the use of influenza and pneumonia mortality rather than pneumonia mortality alone, see Section 3.2 of the main paper and Appendix D. We use the all-age rate because the infant mortality rate is not available by gender for this period.

Figure F8 of this Appendix, we observe clear impacts of sulfa exposure on fertility and marriage rates of black women, with more muted effects among white women. Specifically, we observe that black women exposed to the impacts of sulfa are substantially more likely to ever have been married or be currently married (in 1980), substantially more likely to any children, and have more children (both conditional and unconditional on having any children). Among white women, while we observe a slight increase in the likelihood of marriage we see, if anything, declines in fertility. These results potentially rationalize the observed effects on long-term outcomes the positive endowment shocks to women allow Black women to better meet their desired fertility, through some increase in the marginal returns to a pregnancy in the face of high risk of loss otherwise. Interactions between fertility and labor market outcomes and higher education would thus point to declines in labor supply and education among black women which would not be expected among white women.

To examine this directly we can also stratify by marriage and fertility and examine long-term returns to sulfa exposure among women. We present results of this analysis in Table F11, where we additionally augment the main 4 outcomes presented in the paper with an indicator of whether women are themselves employed as well as educational attainment by level. Here we observe evidence which suggests that changes in marriage and fertility explain the divergent patterns between black and white women. Among Black women, declines in education, family income and employment are driven by married women, with unmarried women having, if anything, improved outcomes as a result of sulfa exposure. Similarly, if stratifying by high and low fertility, among black women declines in labor market and educational outcomes are driven by women who have high fertility (2 or more births),[57] with low fertility women observed to gain from sulfa on the labor market. Among white women—where sulfa had no impacts on realized fertility, no such differential effects are observed, with improvements in labor market outcomes among both married and unmarried women, and high- and low-fertility women.

[57] We split the sample in this way to ensure a sufficiently large sample in each group. If we instead stratify at 0 versus $>$ 0 births, the 0 birth sample is too small among black women to estimate precise effects.

F.3 Tables and Figures

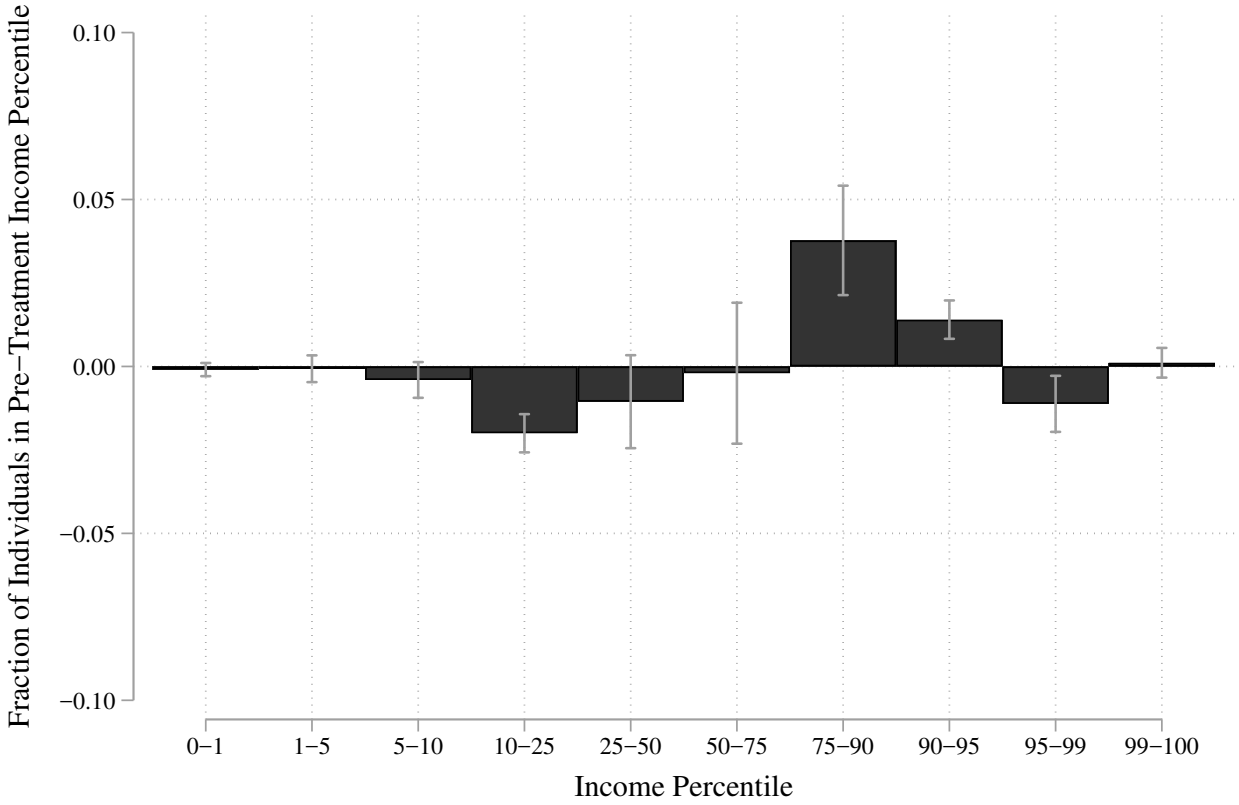
Table F1: Estimated Impacts of Pneumonia Exposure in Infancy on Alternative Outcomes

	High School	College	Poverty	Cognitive Disability
Post Sulfa × Base Exposure	0.0703***	-0.00410	-0.0240***	-0.00591
	(0.0201)	(0.0119)	(0.00752)	(0.00661)
FWER p-value	[0.030]	[0.731]	[0.033]	[0.631]
Effect size for an interquartile shift in base exposure	1.978 pp	-0.115 pp	-0.675 pp	-0.166 pp
Observations	1,433,937	1,433,937	4,190,633	1,328,396

Notes: Refer to notes to Table 1. Identical models are estimated, however here for alternative outcomes indicated in each column header.

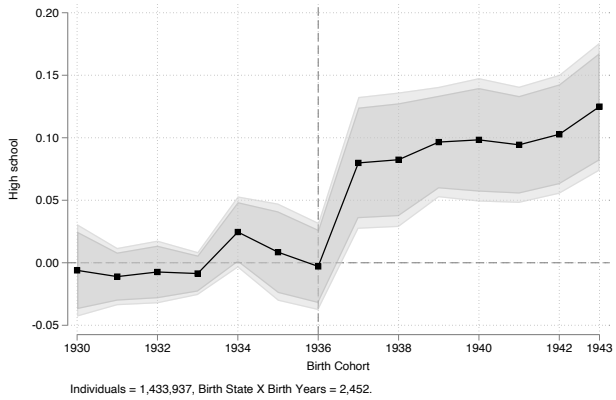
*** p<0.01; ** p<0.05; * p<0.10.

Figure F1: Heterogeneous Effects of sulfa Drugs Across the Income Distribution

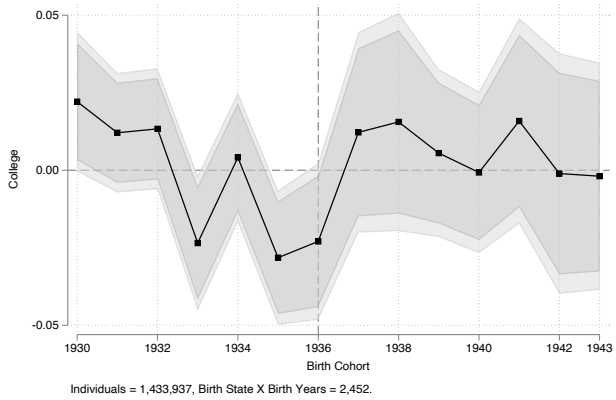


Notes: Distributional impacts of sulfa exposure on log(Family Income) are presented. Implementation follows Isen et al. (2017), reporting coefficients (solid bars) and confidence intervals of 10 separate regressions. Each regression follows result from Table 1, where the dependent variable is an indicator of an individual having a family income in the income percentile reported on the x-axis. These income percentiles refer to percentiles of income in the pre-1937 period in the full sample. Confidence intervals are based on standard errors clustered by state.

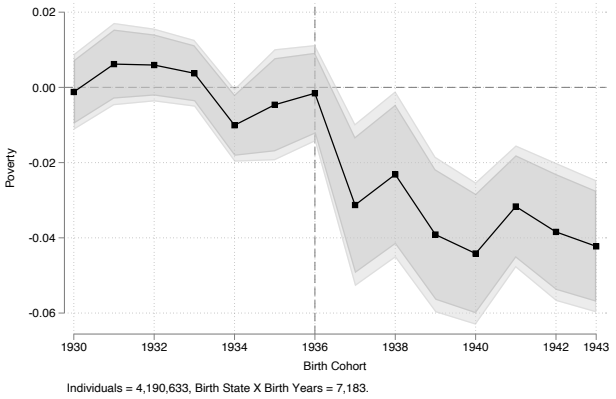
Figure F2: Event Study Estimates of Pneumonia Exposure in Infancy on Alternative Outcomes



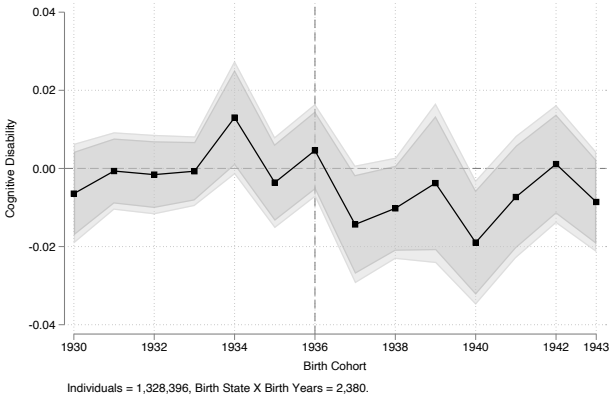
(a) High school



(b) College



(c) Poverty



(d) Cognitive Disability

Notes: Refer to Notes to Figure 4. Identical models are estimated, however here for alternative outcomes indicated in each plot title.

Table F2: Gradients in Long Run Impacts of Infant Pneumonia Exposure by sulfa Diffusion, Alternative Outcomes

	High School	College	Poverty	Cognitive Disability
Post Sulfa × Base Exposure	0.0796***	0.0122	-0.0329***	-0.00323
	(0.0198)	(0.0111)	(0.00734)	(0.00642)
FWER p-value	[0.008]	[0.515]	[0.005]	[0.640]
Post Sulfa × Base Exposure × Pharmacists p.c.	0.0790*	0.139***	-0.0745***	0.0227*
	(0.0459)	(0.0343)	(0.0177)	(0.0118)
FWER p-value	[0.111]	[0.002]	[0.003]	[0.148]
Effect size for an interquartile shift at bottom decile of pharmacists p.c.	1.616 pp	-0.750 pp	-0.337 pp	-0.270 pp
Effect size for an interquartile shift at top decile of pharmacists p.c.	2.793 pp	1.316 pp	-1.447 pp	0.0682 pp
Observations	1,433,937	1,433,937	4,190,633	1,328,396

Notes: Refer to notes to Table 2. Identical models are estimated, however here for alternative outcomes indicated in each column header.
*** p<0.01; ** p<0.05; * p<0.10.

Table F3: Estimated Impacts of Pneumonia Exposure in Infancy on Alternative Adult Outcomes by Demographic

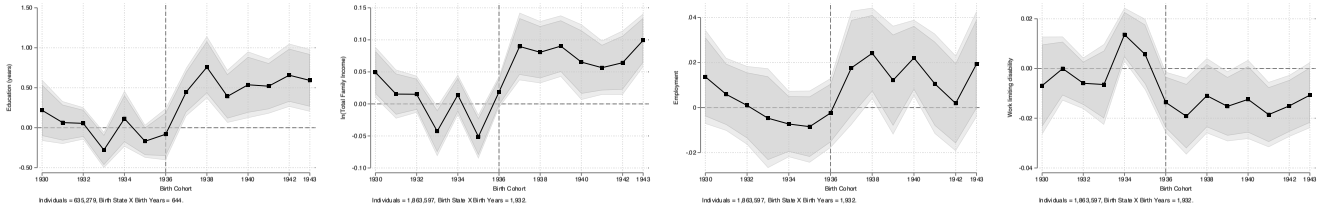
	High School	College	Poverty	Cognitive Disability
Panel A: White Men				
Post Sulfa × Base Exposure	0.0803***	0.0184	-0.0223***	0.00323
	(0.0192)	(0.0138)	(0.00766)	(0.00906)
FWER p-value	[0.003]	[0.352]	[0.026]	[0.722]
Effect size for an interquartile shift in base exposure	2.257 pp	0.518 pp	-0.628 pp	0.0908 pp
Observations	635,279	635,279	1,863,597	590,738
Panel B: White Women				
Post Sulfa × Base Exposure	0.0929***	-0.0108	-0.0258***	-0.0136**
	(0.0291)	(0.0128)	(0.00826)	(0.00590)
FWER p-value	[0.040]	[0.404]	[0.036]	[0.101]
Effect size for an interquartile shift in base exposure	2.612 pp	-0.304 pp	-0.725 pp	-0.383 pp
Observations	649,412	649,412	1,933,966	618,646
Panel C: Black Men				
Post Sulfa × Base Exposure	0.0573*	0.0627	-0.0405	-0.0313
	(0.0299)	(0.0397)	(0.0302)	(0.0213)
FWER p-value	[0.281]	[0.385]	[0.202]	[0.327]
Effect size for an interquartile shift in base exposure	1.610 pp	1.763 pp	-1.138 pp	-0.880 pp
Observations	66,597	66,597	173,715	52,394
Panel D: Black Women				
Post Sulfa × Base Exposure	-0.0198	-0.113***	0.0190	-0.0375
	(0.0576)	(0.0270)	(0.0169)	(0.0243)
FWER p-value	[0.752]	[0.007]	[0.480]	[0.368]
Effect size for an interquartile shift in base exposure	-0.557 pp	-3.188 pp	0.535 pp	-1.055 pp
Observations	82,649	82,649	219,355	66,618

Notes: Refer to notes to Table 3. Identical models are estimated, however here for alternative outcomes indicated in column headers.

*** p<0.01; ** p<0.05; * p<0.10.

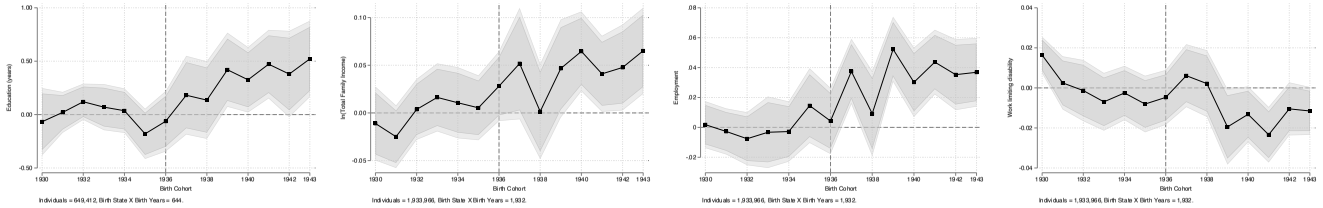
Figure F3: Event Study Estimates of Pneumonia Exposure in Infancy on Adult Outcomes – Other Groups

Panel A: White Men



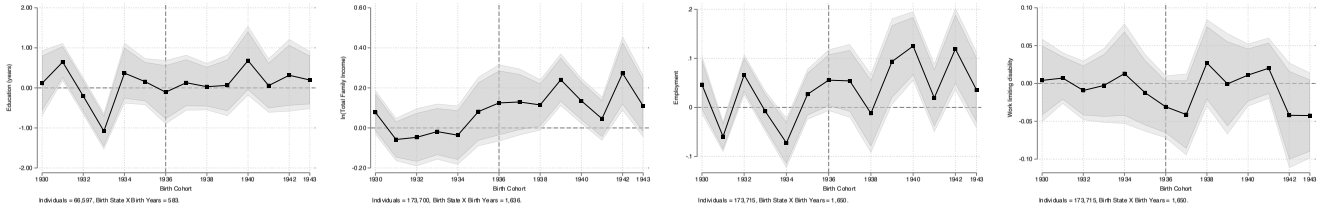
(a) Education (b) log(Family Income) (c) Employment (d) Work Limiting Disability

Panel B: White Women



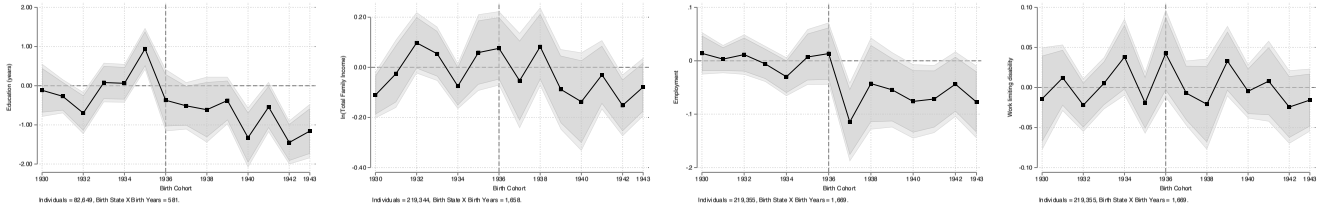
(e) Education (f) log(Family Income) (g) Employment (h) Work Limiting Disability

Panel C: Black Men



(i) Education (j) log(Family Income) (k) Employment (l) Work Limiting Disability

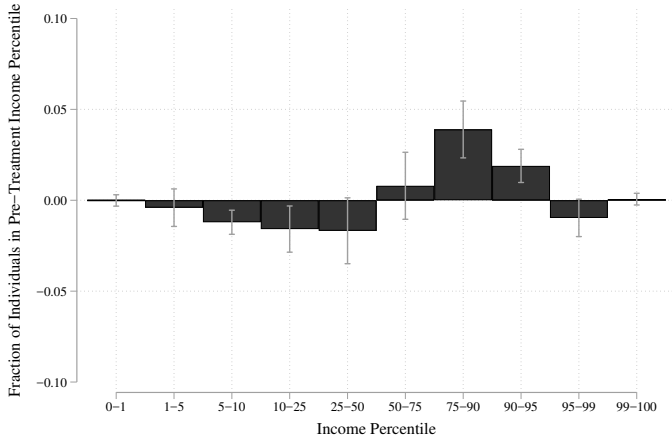
Panel D: Black Women



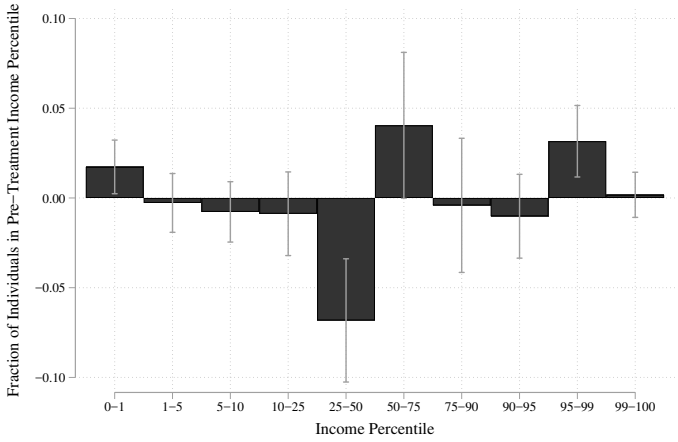
(m) Education (n) log(Family Income) (o) Employment (p) Work Limiting Disability

Notes: Refer to Notes to Figure 4. Identical event studies are displayed, however now for the sample of white men (panel A), Black men (panel B), white women (panel C) and Black women (panel D).

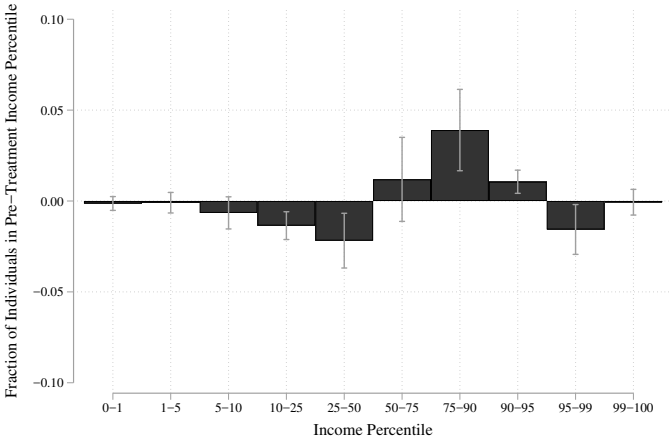
Figure F4: Heterogeneous Effects of sulfa Drugs Across the Income Distribution (group-specific baseline distributions)



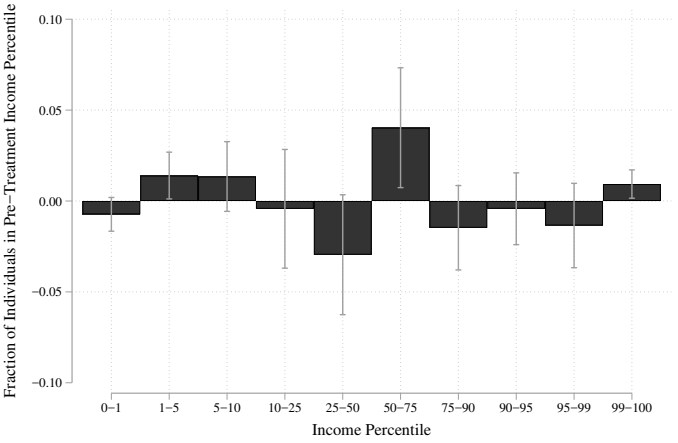
(a) White Men



(b) Black Men



(c) White Women

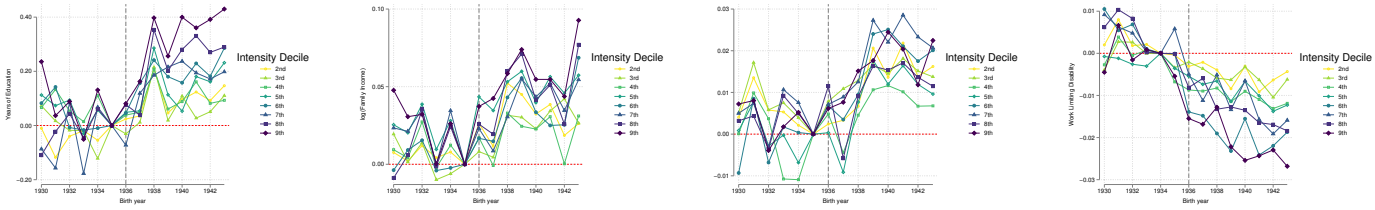


(d) Black Women

Notes: Distributional impacts of sulfa exposure on log(Family Income) are presented for each demographic group. Implementation follows Isen et al. (2017), reporting coefficients (solid bars) and confidence intervals of 10 separate regressions. Each regression follows result from Table 1, where the dependent variable is an indicator of an individual having a family income in the income percentile reported on the x-axis. These income percentiles refer to percentiles of income in the pre-1937 period in the full sample. Confidence intervals are based on standard errors clustered by state.

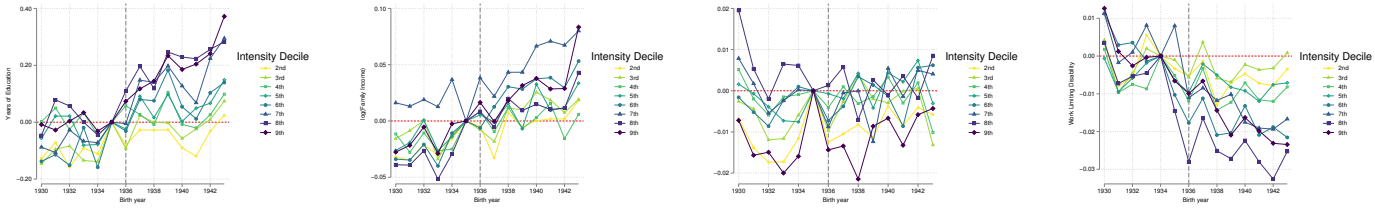
Figure F5: "Strong Parallel Trends Assumption" and the Average Causal Response Function – Other Groups

Panel A: White Men



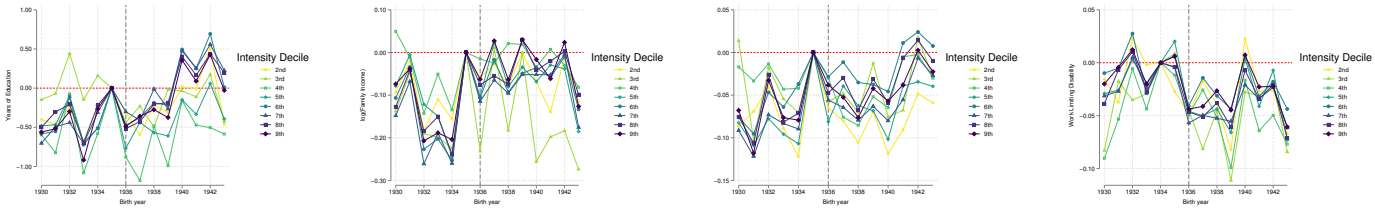
(a) Education (b) log(Family Income) (c) Employment (d) Work-Limiting Disability

Panel B: White Women



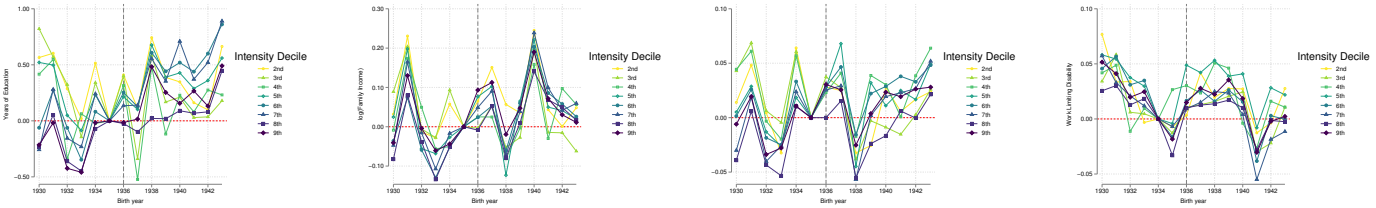
(e) Education (f) log(Family Income) (g) Employment (h) Work-Limiting Disability

Panel C: Black Men



(i) Education (j) log(Family Income) (k) Employment (l) Work-Limiting Disability

Panel D: Black Women



(m) Education (n) log(Family Income) (o) Employment (p) Work-Limiting Disability

Notes: Refer to notes to Figure 5. Identical specifications are estimated, however here for alternative demographic groups indicated in Panel headings.

Table F4: Full Robustness Checks – White Men

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A - Alternative Base Measures				
Post Sulfa × Base 1935 Exposure	0.384***	0.0745***	0.0271***	-0.00896**
	(0.117)	(0.0211)	(0.00597)	(0.00390)
Effect size for an interquartile shift in base exposure	0.0790 years	1.534 %	0.558 pp	-0.185 pp
Observations	600,049	1,730,239	1,761,722	1,761,722
Panel B - Infant Pneumonia Measure				
Post Sulfa × Base Exposure (Infant)	0.0315**	0.00391*	0.00164**	-0.000339
	(0.0139)	(0.00201)	(0.000671)	(0.000369)
Effect size for an interquartile shift in base exposure	0.0409 years	0.508 %	0.213 pp	-0.0440 pp
Observations	635,279	1,829,870	1,863,597	1,863,597
Panel C - Measurement Error Controls				
Post Sulfa × Base Exposure	0.345***	0.0779***	0.00785	-0.0129***
	(0.0918)	(0.0163)	(0.00792)	(0.00381)
Effect size for an interquartile shift in base exposure	0.0971 years	2.191 %	0.221 pp	-0.364 pp
Observations	635,277	1,829,869	1,863,596	1,863,596
Panel D - Controlling for Depression & New Deal Spending				
Post Sulfa × Base Exposure	0.479***	0.0780***	0.0115	-0.0105**
	(0.111)	(0.0187)	(0.00859)	(0.00468)
Effect size for an interquartile shift in base exposure	0.135 years	2.194 %	0.322 pp	-0.295 pp
Observations	635,279	1,829,870	1,863,597	1,863,597
Panel E - No Dust Bowl States				
Post Sulfa × Base Exposure	0.518***	0.0668***	0.00878	-0.0156***
	(0.107)	(0.0141)	(0.00842)	(0.00362)
Effect size for an interquartile shift in base exposure	0.146 years	1.878 %	0.247 pp	-0.439 pp
Observations	558,432	1,612,816	1,642,001	1,642,001
Panel F - Excluding WW II Cohorts				
Post Sulfa × Base Exposure	0.459***	0.0630***	0.0186**	-0.0184***
	(0.116)	(0.0188)	(0.00728)	(0.00534)
Effect size for an interquartile shift in base exposure	0.129 years	1.773 %	0.523 pp	-0.517 pp
Observations	477,891	1,360,800	1,385,775	1,385,775
Panel G - 1935–1941 Cohorts Only				
Post Sulfa × Base Exposure	0.581***	0.0936***	0.0270***	-0.0231**
	(0.142)	(0.0197)	(0.00863)	(0.00869)
Effect size for an interquartile shift in base exposure	0.163 years	2.631 %	0.758 pp	-0.651 pp
Observations	304,253	888,908	905,494	905,494
Panel H - 'Donut' specification (no 1935–1937 Cohorts)				
Post Sulfa × Base Exposure	0.513***	0.0500**	0.0112	-0.0119**
	(0.131)	(0.0242)	(0.00826)	(0.00584)

Continued on next page

Table F4 – continued from previous page

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Effect size for an interquartile shift in base exposure	0.144 years	1.406 %	0.316 pp	-0.334 pp
Observations	350,593	992,384	1,010,634	1,010,634
Panel I - Excluding 2000 Census				
Post Sulfa × Base Exposure	0.402***	0.0512**	0.0155*	-0.0193***
	(0.0966)	(0.0197)	(0.00906)	(0.00655)
Effect size for an interquartile shift in base exposure	0.113 years	1.440 %	0.436 pp	-0.544 pp
Observations	635,279	1,252,479	1,272,859	1,272,859
Panel J - 2000 Census Only				
Post Sulfa × Base Exposure	0.462***	0.107***	-0.0112	0.00215
	(0.114)	(0.0273)	(0.0154)	(0.0134)
Effect size for an interquartile shift in base exposure	0.130 years	3.020 %	-0.316 pp	0.0605 pp
Observations	590,738	577,391	590,738	590,738
Panel K - Measurement Error, 2SLS				
Pneumonia-Influenza Mortality Rate	-0.776***	-0.135***	-0.0211	0.0258***
	(0.245)	(0.0408)	(0.0150)	(0.00828)
Effect size for an interquartile shift in base exposure	0.109 years	1.901 %	0.297 pp	-0.363 pp
Observations	630,394	1,817,070	1,850,493	1,850,493

Notes: Refer to notes to Table E1. Identical robustness tests are displayed, however now for the sample of white men instead of all groups. Standard errors clustered at the birth state level are reported in parentheses.

*** p<0.01; ** p<0.05; * p<0.10.

Table F5: Full Robustness Checks – White Women

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A - Alternative Base Measures				
Post Sulfa × Base 1935 Exposure	0.218**	0.0421**	0.0342***	-0.0199***
	(0.0913)	(0.0178)	(0.00799)	(0.00417)
Effect size for an interquartile shift in base exposure	0.0449 years	0.868 %	0.705 pp	-0.410 pp
Observations	613,028	1,798,242	1,826,792	1,826,792
Panel B - Infant Pneumonia Measure				
Post Sulfa × Base Exposure (Infant)	0.0337***	0.00425***	0.000996**	-0.000850**
	(0.00877)	(0.00157)	(0.000475)	(0.000395)
Effect size for an interquartile shift in base exposure	0.0438 years	0.552 %	0.129 pp	-0.110 pp
Observations	649,412	1,903,470	1,933,966	1,933,966
Panel C - Measurement Error Controls				
Post Sulfa × Base Exposure	0.179*	0.0357***	0.0260***	-0.00857*
	(0.0919)	(0.0121)	(0.00787)	(0.00441)
Effect size for an interquartile shift in base exposure	0.0503 years	1.004 %	0.732 pp	-0.241 pp
Observations	649,412	1,903,470	1,933,966	1,933,966
Panel D - Controlling for Depression & New Deal Spending				

Continued on next page

Table F5 – continued from previous page

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Post Sulfa × Base Exposure	0.210**	0.0418***	0.0271***	-0.00844*
	(0.103)	(0.0135)	(0.00745)	(0.00477)
Effect size for an interquartile shift in base exposure	0.0590 years	1.176 %	0.762 pp	-0.237 pp
Observations	649,412	1,903,470	1,933,966	1,933,966
Panel E - No Dust Bowl States				
Post Sulfa × Base Exposure	0.328***	0.0402***	0.0283***	-0.0107**
	(0.0802)	(0.0109)	(0.00781)	(0.00504)
Effect size for an interquartile shift in base exposure	0.0923 years	1.130 %	0.796 pp	-0.301 pp
Observations	570,739	1,676,361	1,702,869	1,702,869
Panel F - Excluding WW II Cohorts				
Post Sulfa × Base Exposure	0.0997	0.0374**	0.0258***	-0.00454
	(0.105)	(0.0184)	(0.00778)	(0.00760)
Effect size for an interquartile shift in base exposure	0.0280 years	1.051 %	0.726 pp	-0.128 pp
Observations	490,479	1,418,797	1,442,174	1,442,174
Panel G - 1935–1941 Cohorts Only				
Post Sulfa × Base Exposure	0.269**	0.0291	-0.00775	-0.00607
	(0.117)	(0.0203)	(0.0128)	(0.00642)
Effect size for an interquartile shift in base exposure	0.0757 years	0.818 %	-0.218 pp	-0.171 pp
Observations	309,960	917,214	931,692	931,692
Panel H - 'Donut' specification (no 1935–1937 Cohorts)				
Post Sulfa × Base Exposure	0.0499	0.0381*	0.0455***	-0.00699
	(0.121)	(0.0197)	(0.00862)	(0.0106)
Effect size for an interquartile shift in base exposure	0.0140 years	1.070 %	1.279 pp	-0.197 pp
Observations	360,039	1,032,879	1,050,327	1,050,327
Panel I - Excluding 2000 Census				
Post Sulfa × Base Exposure	0.190*	0.0322*	0.0341***	-0.0174**
	(0.0971)	(0.0187)	(0.00945)	(0.00701)
Effect size for an interquartile shift in base exposure	0.0535 years	0.906 %	0.958 pp	-0.488 pp
Observations	649,412	1,297,948	1,315,320	1,315,320
Panel J - 2000 Census Only				
Post Sulfa × Base Exposure	0.256**	0.0472	0.0139	0.00790
	(0.102)	(0.0506)	(0.0154)	(0.00938)
Effect size for an interquartile shift in base exposure	0.0721 years	1.327 %	0.391 pp	0.222 pp
Observations	618,646	605,522	618,646	618,646
Panel K - Measurement Error, 2SLS				
Pneumonia-Influenza Mortality Rate	-0.400**	-0.0696**	-0.0514***	0.0191**
	(0.185)	(0.0263)	(0.0178)	(0.00827)
Effect size for an interquartile shift in base exposure	0.0563 years	0.979 %	0.723 pp	-0.269 pp
Observations	644,317	1,889,192	1,919,375	1,919,375

Notes: Refer to notes to Table E1. Identical robustness tests are displayed, however now for the sample of white women. Standard errors clustered at the birth state level are reported in parentheses.

*** p<0.01; ** p<0.05; * p<0.10.

Table F6: Full Robustness Checks – Black Men

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A - Alternative Base Measures				
Post Sulfa × Base 1935 Exposure	0.628**	0.248***	0.0897***	-0.0330
	(0.279)	(0.0382)	(0.0330)	(0.0219)
Effect size for an interquartile shift in base exposure	0.129 years	5.110 %	1.848 pp	-0.679 pp
Observations	61,497	151,427	159,819	159,819
Panel B - Infant Pneumonia Measure				
Post Sulfa × Base Exposure (Infant)	0.0369*	0.0164***	0.00516	-0.00140
	(0.0216)	(0.00507)	(0.00399)	(0.00185)
Effect size for an interquartile shift in base exposure	0.0479 years	2.130 %	0.670 pp	-0.182 pp
Observations	66,597	164,497	173,715	173,715
Panel C - Measurement Error Controls				
Post Sulfa × Base Exposure	0.275	0.142***	0.106***	-0.0141
	(0.250)	(0.0395)	(0.0239)	(0.0133)
Effect size for an interquartile shift in base exposure	0.0774 years	4.005 %	2.986 pp	-0.396 pp
Observations	66,597	164,497	173,715	173,715
Panel D - Controlling for Depression & New Deal Spending				
Post Sulfa × Base Exposure	-0.0477	0.165***	0.122***	-0.00717
	(0.367)	(0.0537)	(0.0390)	(0.0168)
Effect size for an interquartile shift in base exposure	-0.0134 years	4.635 %	3.424 pp	-0.202 pp
Observations	66,597	164,497	173,715	173,715
Panel E - No Dust Bowl States				
Post Sulfa × Base Exposure	0.622	0.262***	0.0836**	-0.0281
	(0.397)	(0.0752)	(0.0397)	(0.0258)
Effect size for an interquartile shift in base exposure	0.175 years	7.377 %	2.350 pp	-0.789 pp
Observations	60,107	148,194	156,385	156,385
Panel F - Excluding WW II Cohorts				
Post Sulfa × Base Exposure	0.229	0.131**	0.0293	0.0369***
	(0.212)	(0.0597)	(0.0406)	(0.0125)
Effect size for an interquartile shift in base exposure	0.0643 years	3.686 %	0.824 pp	1.037 pp
Observations	50,325	123,334	129,914	129,914
Panel G - 1935–1941 Cohorts Only				
Post Sulfa × Base Exposure	0.294	0.123***	0.0820*	0.0112
	(0.206)	(0.0406)	(0.0473)	(0.0238)
Effect size for an interquartile shift in base exposure	0.0825 years	3.446 %	2.306 pp	0.316 pp
Observations	32,955	82,642	87,177	87,177
Panel H - 'Donut' specification (no 1935–1937 Cohorts)				
Post Sulfa × Base Exposure	-0.0922	0.0655	-0.00846	0.0609***
	(0.325)	(0.0740)	(0.0458)	(0.0180)

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Table F6 – continued from previous page

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Effect size for an interquartile shift in base exposure	-0.0259 years	1.840 %	-0.238 pp	1.712 pp
Observations	36,629	89,179	93,977	93,977
Panel I - Excluding 2000 Census				
Post Sulfa × Base Exposure	0.210	0.207***	0.0687**	0.00984
	(0.217)	(0.0386)	(0.0306)	(0.0258)
Effect size for an interquartile shift in base exposure	0.0590 years	5.813 %	1.933 pp	0.277 pp
Observations	66,597	115,584	121,321	121,321
Panel J - 2000 Census Only				
Post Sulfa × Base Exposure	-0.189	-0.0484	0.0282	-0.0522
	(0.232)	(0.0667)	(0.0580)	(0.0375)
Effect size for an interquartile shift in base exposure	-0.0530 years	-1.361 %	0.793 pp	-1.468 pp
Observations	52,394	48,912	52,394	52,394
Panel K - Measurement Error, 2SLS				
Pneumonia-Influenza Mortality Rate	-0.380	-0.185***	-0.0811*	0.00816
	(0.286)	(0.0533)	(0.0464)	(0.0189)
Effect size for an interquartile shift in base exposure	0.0534 years	2.605 %	1.140 pp	-0.115 pp
Observations	65,863	162,830	171,951	171,951

Notes: Refer to notes to Table E1. Identical robustness tests are displayed, however now for the sample of Black men. Standard errors clustered at the birth state level are reported in parentheses.

*** p<0.01; ** p<0.05; * p<0.10.

Table F7: Full Robustness Checks – Black Women

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A - Alternative Base Measures				
Post Sulfa × Base 1935 Exposure	-0.0877	-0.0466	-0.0826**	-0.0501***
	(0.290)	(0.0717)	(0.0376)	(0.0181)
Effect size for an interquartile shift in base exposure	-0.0181 years	-0.961 %	-1.703 pp	-1.032 pp
Observations	76,255	195,584	201,985	201,985
Panel B - Infant Pneumonia Measure				
Post Sulfa × Base Exposure (Infant)	-0.0348	0.000925	-0.00392*	-0.00247*
	(0.0252)	(0.00515)	(0.00206)	(0.00132)
Effect size for an interquartile shift in base exposure	-0.0452 years	0.120 %	-0.510 pp	-0.321 pp
Observations	82,649	212,391	219,355	219,355
Panel C - Measurement Error Controls				
Post Sulfa × Base Exposure	-0.709***	-0.0438	-0.0505**	-0.0138
	(0.227)	(0.0488)	(0.0193)	(0.0143)
Effect size for an interquartile shift in base exposure	-0.199 years	-1.232 %	-1.419 pp	-0.389 pp
Observations	82,649	212,391	219,355	219,355
Panel D - Controlling for Depression & New Deal Spending				

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Table F7 – continued from previous page

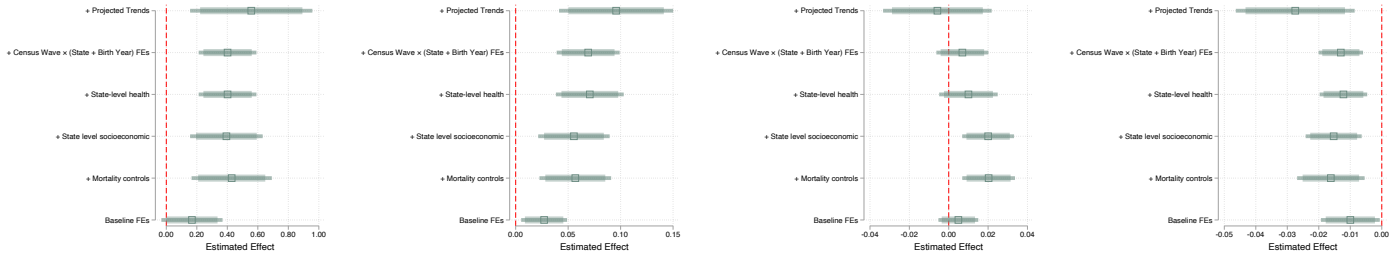
	Schooling	log(Family Income)	Employment	Work Limiting Disability
Post Sulfa × Base Exposure	-0.995***	-0.0590	-0.107***	0.00364
	(0.318)	(0.0924)	(0.0369)	(0.0240)
Effect size for an interquartile shift in base exposure	-0.280 years	-1.660 %	-3.019 pp	0.102 pp
Observations	82,649	212,391	219,355	219,355
Panel E - No Dust Bowl States				
Post Sulfa × Base Exposure	-0.252	-0.119	-0.148***	-0.0744***
	(0.344)	(0.0979)	(0.0456)	(0.0196)
Effect size for an interquartile shift in base exposure	-0.0710 years	-3.355 %	-4.160 pp	-2.091 pp
Observations	74,579	191,508	197,790	197,790
Panel F - Excluding WW II Cohorts				
Post Sulfa × Base Exposure	-0.674**	-0.0523	-0.0757***	-0.0363**
	(0.270)	(0.0698)	(0.0267)	(0.0140)
Effect size for an interquartile shift in base exposure	-0.190 years	-1.470 %	-2.129 pp	-1.020 pp
Observations	62,327	159,410	164,709	164,709
Panel G - 1935–1941 Cohorts Only				
Post Sulfa × Base Exposure	-0.724***	-0.0657	-0.0521	-0.0338
	(0.262)	(0.0780)	(0.0328)	(0.0209)
Effect size for an interquartile shift in base exposure	-0.204 years	-1.848 %	-1.464 pp	-0.951 pp
Observations	40,208	105,046	108,432	108,432
Panel H - 'Donut' specification (no 1935–1937 Cohorts)				
Post Sulfa × Base Exposure	-0.429	-0.0738	-0.0773***	-0.0532**
	(0.326)	(0.0699)	(0.0286)	(0.0211)
Effect size for an interquartile shift in base exposure	-0.121 years	-2.075 %	-2.173 pp	-1.497 pp
Observations	45,552	115,546	119,430	119,430
Panel I - Excluding 2000 Census				
Post Sulfa × Base Exposure	-0.810***	-0.00255	-0.0944***	0.0110
	(0.221)	(0.0574)	(0.0313)	(0.0172)
Effect size for an interquartile shift in base exposure	-0.228 years	-0.0718 %	-2.655 pp	0.309 pp
Observations	82,649	148,699	152,737	152,737
Panel J - 2000 Census Only				
Post Sulfa × Base Exposure	0.396	-0.291***	-0.0219	-0.0821**
	(0.278)	(0.0686)	(0.0304)	(0.0318)
Effect size for an interquartile shift in base exposure	0.111 years	-8.190 %	-0.616 pp	-2.308 pp
Observations	66,618	63,692	66,618	66,618
Panel K - Measurement Error, 2SLS				
Pneumonia-Influenza Mortality Rate	1.029***	0.141*	0.0977***	0.0219
	(0.325)	(0.0719)	(0.0339)	(0.0189)
Effect size for an interquartile shift in base exposure	-0.145 years	-1.984 %	-1.373 pp	-0.308 pp
Observations	81,724	210,206	217,083	217,083

Notes: Refer to notes to Table E1. Identical robustness tests are displayed, however now for the sample of Black women. Standard errors clustered at the birth state level are reported in parentheses.

*** p<0.01; ** p<0.05; * p<0.10.

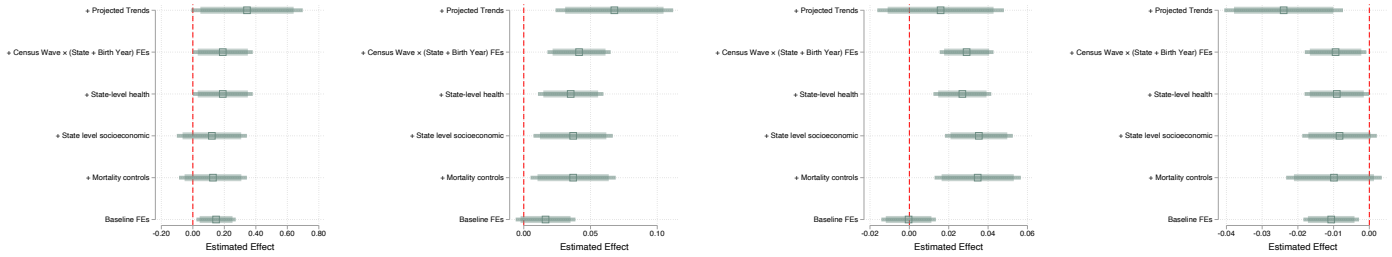
Figure F6: Robustness of estimates to alternative controls – Other Groups

Panel A: White Men



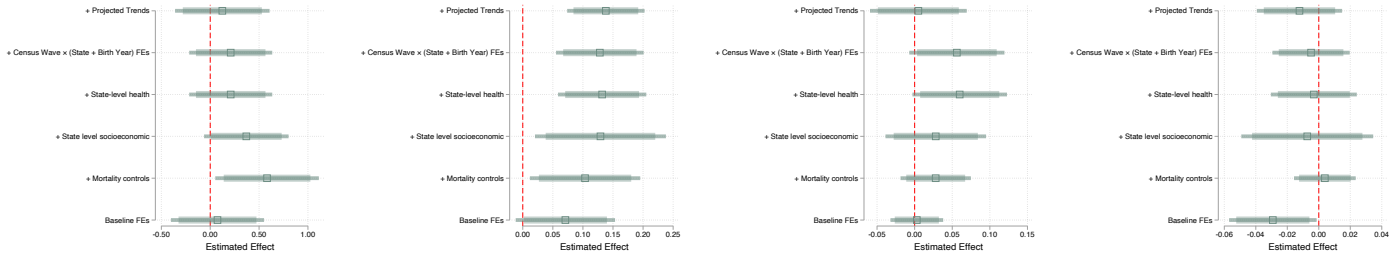
(a) Education (b) log(Family Income) (c) Employment (d) Work-limiting Disability

Panel B: White Women



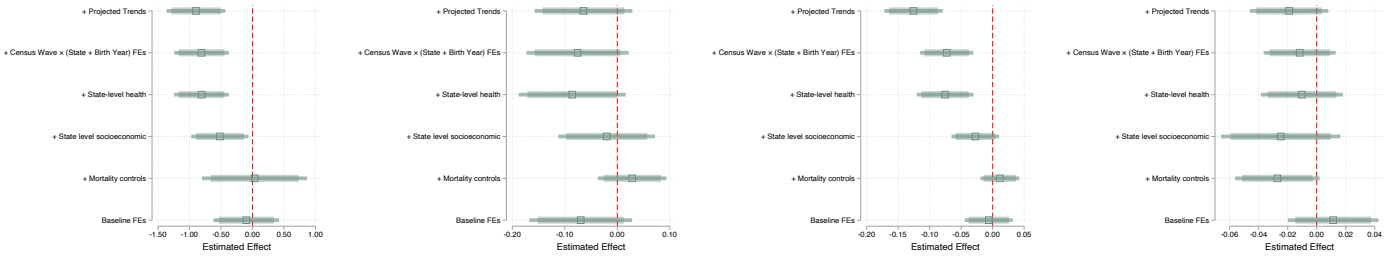
(e) Education (f) log(Family Income) (g) Employment (h) Work-limiting Disability

Panel C: Black Men



(i) Education (j) log(Family Income) (k) Employment (l) Work-Limiting Disability

Panel D: Black Women



(m) Education (n) log(Family Income) (o) Employment (p) Work-Limiting Disability

Notes: Refer to Notes to Figure 6. Identical robustness checks are displayed, however here for alternative demographic groups indicated in Panel headings.

Table F8: Gradients in Long Run Impacts of Infant Pneumonia Exposure by sulfa Diffusion – Demographic Groups

	Schooling	log(Family Income)	Employment	Work Limiting Disability
Panel A: White Men				
Post Sulfa × Base Exposure	0.515***	0.0834***	0.0154*	-0.0120**
	(0.0891)	(0.0174)	(0.00848)	(0.00457)
FWER p-value	[0.000]	[0.001]	[0.100]	[0.048]
Post Sulfa × Base Exposure × Pharmacists p.c.	1.036***	0.115***	0.0489*	0.00130
	(0.222)	(0.0410)	(0.0266)	(0.0126)
FWER p-value	[0.003]	[0.060]	[0.203]	[0.923]
Effect size for an interquartile shift at bottom decile of pharmacists p.c.	0.0632 years	1.441 %	0.0484 pp	-0.348 pp
Effect size for an interquartile shift at top decile of pharmacists p.c.	0.218 years	3.151 %	0.777 pp	-0.329 pp
Observations	635,279	1,829,870	1,863,597	1,863,597
Panel B: White Women				
Post Sulfa × Base Exposure	0.233**	0.0508***	0.0299***	-0.0115**
	(0.106)	(0.0117)	(0.00823)	(0.00475)
FWER p-value	[0.061]	[0.004]	[0.011]	[0.079]
Post Sulfa × Base Exposure × Pharmacists p.c.	0.400	0.141***	0.0269	-0.0213*
	(0.276)	(0.0358)	(0.0175)	(0.0108)
FWER p-value	[0.169]	[0.005]	[0.276]	[0.185]
Effect size for an interquartile shift at bottom decile of pharmacists p.c.	0.0341 years	0.315 %	0.629 pp	-0.154 pp
Effect size for an interquartile shift at top decile of pharmacists p.c.	0.0937 years	2.420 %	1.029 pp	-0.471 pp
Observations	649,412	1,903,470	1,933,966	1,933,966
Panel C: Black Men				
Post Sulfa × Base Exposure	0.682*	0.262***	0.191***	-0.0429*
	(0.375)	(0.0650)	(0.0368)	(0.0223)
FWER p-value	[0.094]	[0.003]	[0.000]	[0.137]
Post Sulfa × Base Exposure × Pharmacists p.c.	2.549*	0.705**	0.716***	-0.217**
	(1.414)	(0.307)	(0.150)	(0.101)
FWER p-value	[0.095]	[0.102]	[0.000]	[0.092]
Effect size for an interquartile shift at bottom decile of pharmacists p.c.	-0.00927 years	1.810 %	-0.272 pp	0.505 pp
Effect size for an interquartile shift at top decile of pharmacists p.c.	0.370 years	12.30 %	10.39 pp	-2.728 pp
Observations	66,597	164,497	173,715	173,715
Panel D: Black Women				
Post Sulfa × Base Exposure	-0.801**	-0.123	-0.0994**	-0.0558**
	(0.352)	(0.0877)	(0.0444)	(0.0275)
FWER p-value	[0.124]	[0.191]	[0.097]	[0.105]
Post Sulfa × Base Exposure × Pharmacists p.c.	0.0506	-0.199	-0.129	-0.246**
	(1.286)	(0.349)	(0.169)	(0.115)
FWER p-value	[0.971]	[0.778]	[0.782]	[0.160]
Effect size for an interquartile shift at bottom decile of pharmacists p.c.	-0.229 years	-1.891 %	-1.780 pp	0.372 pp
Effect size for an interquartile shift at top decile of pharmacists p.c.	-0.222 years	-4.853 %	-3.698 pp	-3.295 pp
Observations	82,649	212,391	219,355	219,355

Notes: Refer to note to Table 2. Identical models are estimated, however here for alternative demographic groups indicated in Panel headings.

*** p<0.01; ** p<0.05; * p<0.10.

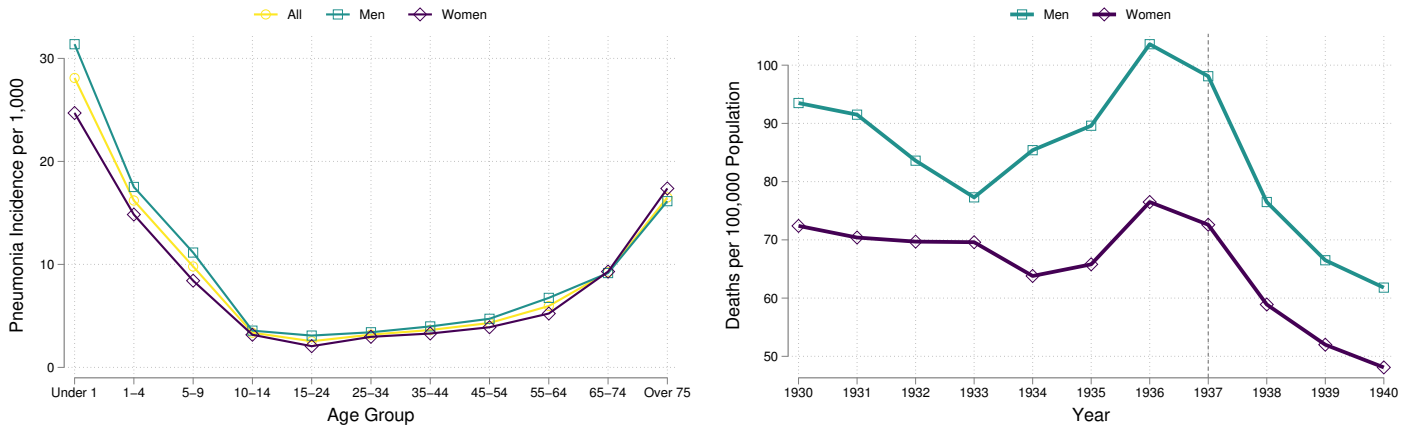
Table F9: Gradients in Long Run Impacts of Infant Pneumonia Exposure by F/M Employment and Education Ratios

	Gradient: Female/Male Employment Ratio				Gradient: Female/Male College Ratio			
	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability	Schooling	log(Family Income)	Employ-ment	Work Limiting Disability
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A – White Women								
Post Sulfa × Base Exposure	0.188*	0.0359**	0.0224**	-0.00628	0.210**	0.0377***	0.0266***	-0.00921**
	(0.0960)	(0.0149)	(0.00834)	(0.00472)	(0.0874)	(0.0112)	(0.00732)	(0.00454)
FWER p-value	[0.125]	[0.078]	[0.063]	[0.219]	[0.080]	[0.027]	[0.018]	[0.104]
Post Sulfa × Base Exposure × Gender Ratio	0.304	-0.00245	0.102	-0.0650	0.612**	0.0678	0.00382	-0.0170
	(1.100)	(0.190)	(0.0864)	(0.0514)	(0.276)	(0.0439)	(0.0162)	(0.0138)
FWER p-value	[0.954]	[0.990]	[0.594]	[0.610]	[0.186]	[0.391]	[0.819]	[0.396]
Effect size at bottom decile of female/male ratio	0.0454 years	1.016 %	0.380 pp	-0.0178 pp	0.0270 years	0.706 %	0.729 pp	-0.170 pp
Effect size at top decile of female/male ratio	0.0594 years	1.005 %	0.849 pp	-0.316 pp	0.0941 years	1.449 %	0.771 pp	-0.356 pp
Observations	649,412	1,903,470	1,933,966	1,933,966	649,412	1,903,470	1,933,966	1,933,966
Panel B – Black Women								
Post Sulfa × Base Exposure	-1.137***	-0.0544	-0.0677	0.00417	-0.789***	-0.0657	-0.0637***	-0.0169
	(0.314)	(0.0851)	(0.0451)	(0.0209)	(0.235)	(0.0559)	(0.0233)	(0.0156)
FWER p-value	[0.002]	[0.779]	[0.333]	[0.843]	[0.003]	[0.468]	[0.029]	[0.300]
Post Sulfa × Base Exposure × Gender Ratio	3.983	-0.486	-0.0484	-0.148	0.915	-0.158	-0.118	0.106
	(4.281)	(1.048)	(0.485)	(0.237)	(1.082)	(0.366)	(0.157)	(0.128)
FWER p-value	[0.791]	[0.833]	[0.927]	[0.888]	[0.883]	[0.691]	[0.680]	[0.804]
Effect size at bottom decile of female/male ratio	-0.417 years	-0.343 %	-1.784 pp	0.477 pp	-0.270 years	-1.023 %	-1.170 pp	-1.027 pp
Effect size at top decile of female/male ratio	-0.234 years	-2.573 %	-2.007 pp	-0.200 pp	-0.169 years	-2.751 %	-2.468 pp	0.130 pp
Observations	82,649	212,391	219,355	219,355	82,649	212,391	219,355	219,355

Notes: Panels A and B present estimates for White and Black women respectively. Model specifications are are as in Table 1, additionally including Post×Base Exposure×F/M Ratios and the underlying main effects. Columns (1)-(4) consider interactions with F/M employment ratios in each state, while columns (5)-(8) consider interactions with F/M college completion ratios. Coefficients are presented along with standard errors clustered by birth state in parentheses. Multiple comparison adjusted p-values, which control the family-wise error rate following (Romano and Wolf, 2005) are provided in square brackets based on 10,000 bootstrap replicates. The estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) on the outcome in each column are provided as the reported effect size at the foot of the table both in areas with low values for the F/M gender ratios, and high values for these measures.

*** p<0.01; ** p<0.05; * p<0.10.

Figure F7: 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 F10: Estimated Impacts of Pneumonia Exposure in Infancy on Nonmarket Outcomes

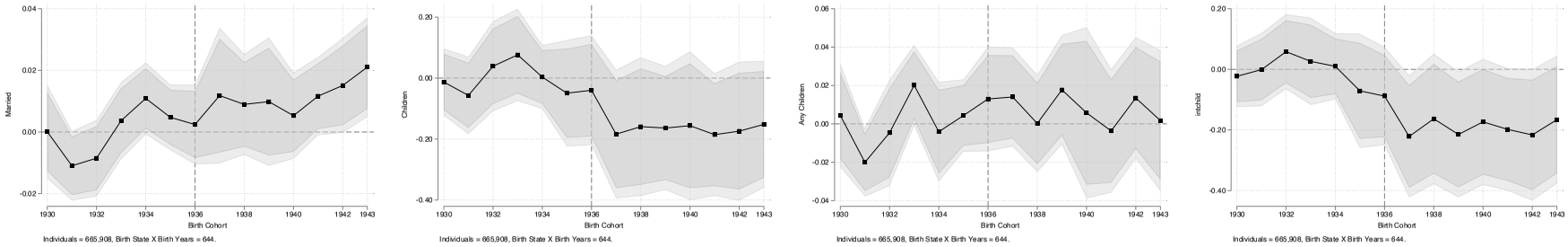
	Ever Married	Currently Married	# Children Ever Born	Any Child	# Children | Any Child
Panel A: White Women					
Post Sulfa × Base Exposure	0.0129***	0.0313**	-0.134*	0.00489	-0.148**
	(0.00428)	(0.0120)	(0.0762)	(0.00814)	(0.0695)
FWER p-value	[0.026]	[0.058]	[0.182]	[0.554]	[0.106]
Effect size for an interquartile shift in base exposure	0.364 pp	0.880 pp	-0.0378 children	0.137 pp	-0.0417 children
Observations	665,908	665,908	595,340	595,340	531,715
Panel B: Black Women					
Post Sulfa × Base Exposure	0.0825***	0.0615*	1.217***	0.167***	0.718***
	(0.0230)	(0.0362)	(0.219)	(0.0252)	(0.250)
FWER p-value	[0.028]	[0.144]	[0.002]	[0.001]	[0.054]
Effect size for an interquartile shift in base exposure	2.319 pp	1.729 pp	0.342 children	4.692 pp	0.202 children
Observations	70,087	70,087	62,284	62,284	53,146

Notes: Refer to Notes to Table 1. Identical models are estimated however here for non-market (family formation) outcomes of individuals exposed to sulfa at birth. Models are estimated for White women (Panel A) and Black women (panel B). Standard errors clustered by birth state are presented in parentheses. Multiple comparison adjusted p-values, which represent family-wise error rates (Romano and Wolf, 2005), provided in square brackets. The estimated effect of an inter-quartile range movement in pneumonia mortality (0.29 fewer deaths per 1,000) on the outcome in each column are provided as *Effect Size*.

*** p<0.01; ** p<0.05; * p<0.10.

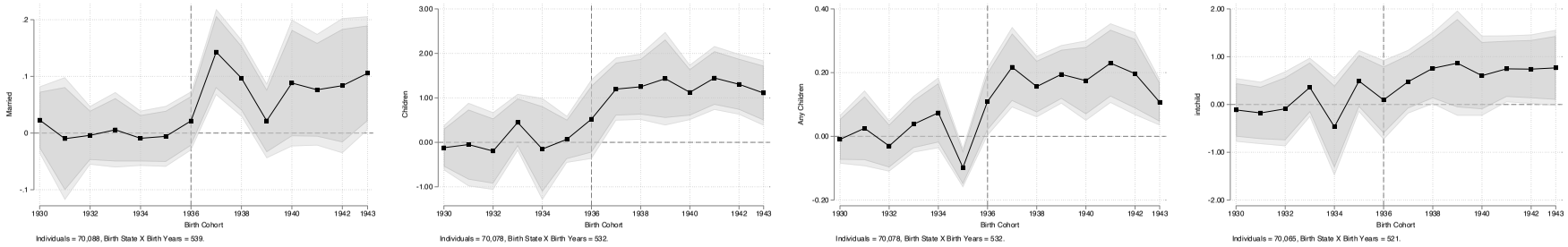
Figure F8: Event Study Estimates of Pneumonia Exposure in Infancy on non-Market Outcomes

White Women



(a) Ever married (b) Total Children (c) Any Child (d) Child|Any Child

Black Women



(e) Ever married (f) Total Children (g) Any Child (h) Child|Any Child

Notes: Refer to Note to Figure 4. Similar event studies are estimated following (2), however here for non-market (family formation) variables. Models are estimated for White women (top panel) and Black women (bottom panel). All other details follow those in Figur 4.

Table F11: Estimated Impacts of Pneumonia Exposure in Infancy on Long-term Outcomes by Marriage and Fertility

	Married					Unmarried				
	Schooling	log(Family Income	Employment	High School	College	Schooling	log(Family Income	Employment	High School	College
Panel A – Black Women										
Post Sulfa × Base Exposure	-0.997***	-0.226***	-0.159**	-0.0925	-0.198***	-0.369	0.256**	0.0272	-0.0493	-0.0107
	(0.353)	(0.0762)	(0.0670)	(0.0569)	(0.0423)	(0.309)	(0.103)	(0.0454)	(0.0441)	(0.0462)
Observations	32,236	31,906	32,236	32,236	32,236	37,844	36,121	37,844	37,844	37,844
Panel B – White Women										
Post Sulfa × Base Exposure	0.0841	0.0468	0.0419***	0.0220	-0.0175	0.111	0.0478	0.0624***	0.0395*	-0.00890
	(0.100)	(0.0323)	(0.0149)	(0.0143)	(0.0211)	(0.153)	(0.0349)	(0.0164)	(0.0221)	(0.0307)
Observations	497,937	495,294	497,937	497,937	497,937	167,971	161,596	167,971	167,971	167,971
	High fertility					Low fertility				
	Schooling	log(Family Income	Employment	High School	College	Schooling	log(Family Income	Employment	High School	College
Panel C – Black Women										
Post Sulfa × Base Exposure	-0.857***	-0.220**	-0.151**	-0.160***	-0.125**	0.00533	0.387**	0.209***	0.0863	0.0169
	(0.271)	(0.0904)	(0.0565)	(0.0378)	(0.0594)	(0.512)	(0.183)	(0.0667)	(0.0725)	(0.0768)
Observations	41,098	40,047	41,098	41,098	41,098	21,181	20,585	21,181	21,181	21,181
Panel D – White Women										
Post Sulfa × Base Exposure	0.137	0.0499	0.0668***	0.0281	-0.00140	-0.0258	0.116**	0.0460***	0.0112	-0.0276
	(0.132)	(0.0338)	(0.0130)	(0.0196)	(0.0237)	(0.115)	(0.0478)	(0.0118)	(0.0171)	(0.0272)
Observations	355,207	351,392	355,207	355,207	355,207	240,133	237,609	240,133	240,133	240,133

Notes: Refer to notes to Table 1. Identical models are estimated, however estimating separately for women who are married (left-hand side columns) and those who are unmarried (right-hand side columns). Standard errors clustered by birth state are presented in parentheses.

*** p<0.01; ** p<0.05; * p<0.10.

Table F12: Gradients in Long Run Impacts of Infant Pneumonia Exposure by Indices of Discrimination, Alternative Variables – Black Men and Women

	Gradient: Historical Fraction of Enslaved People				Gradient: Number of Jim Crow Laws			
	High School	College	Poverty	Cognitive Disability	High School	College	Poverty	Cognitive Disability
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Black Men								
Post × Base Exposure	0.219***	0.175***	-0.00516	-0.0423	0.143***	0.148**	-0.0229	-0.0482
	(0.0495)	(0.0554)	(0.0478)	(0.0582)	(0.0467)	(0.0566)	(0.0488)	(0.0455)
FWER p-value	[0.068]	[0.124]	[0.920]	[0.716]	[0.069]	[0.109]	[0.646]	[0.512]
Post Sulfa × Base Exposure × Discrimination Proxy	-0.606***	-0.453**	-0.0558	-0.0318	-0.00401***	-0.00274	-0.000592	0.00106
	(0.115)	(0.185)	(0.130)	(0.128)	(0.00137)	(0.00167)	(0.00154)	(0.00123)
FWER p-value	[0.044]	[0.183]	[0.889]	[0.807]	[0.081]	[0.376]	[0.717]	[0.639]
Effect size at bottom decile of discrimination proxy	8.353 pp	6.555 pp	0.0574 pp	-1.075 pp	5.821 pp	5.384 pp	-0.379 pp	-1.831 pp
Effect size at top decile of discrimination proxy	0.855 pp	0.952 pp	-0.633 pp	-1.469 pp	0.862 pp	1.999 pp	-1.111 pp	-0.514 pp
Observations	65,266	65,266	170,601	51,486	66,597	66,597	173,715	52,394
Panel B: Black Women								
Post × Base Exposure	0.333***	-0.125***	-0.00114	-0.121***	0.115	-0.0666*	0.0233	-0.0604**
	(0.0587)	(0.0381)	(0.0360)	(0.0283)	(0.0809)	(0.0342)	(0.0329)	(0.0286)
FWER p-value	[0.027]	[0.015]	[0.975]	[0.008]	[0.416]	[0.305]	[0.500]	[0.286]
Post Sulfa × Base Exposure × Discrimination Proxy	-0.627***	-0.184	0.0961	0.0925	-0.00440*	-0.00123	-0.000329	0.000355
	(0.178)	(0.119)	(0.121)	(0.0773)	(0.00223)	(0.00104)	(0.00102)	(0.000903)
FWER p-value	[0.098]	[0.360]	[0.466]	[0.442]	[0.246]	[0.539]	[0.766]	[0.911]
Effect size at bottom decile of discrimination proxy	11.65 pp	-2.842 pp	-0.380 pp	-3.731 pp	5.207 pp	-1.323 pp	0.801 pp	-1.855 pp
Effect size at top decile of discrimination proxy	3.898 pp	-5.120 pp	0.807 pp	-2.587 pp	-0.231 pp	-2.845 pp	0.394 pp	-1.417 pp
Observations	81,074	81,074	215,520	65,568	82,649	82,649	219,355	66,618

Notes: Refer to notes to Table 4. Identical models are estimated, however here for alternative outcomes indicated in column headers.

*** p<0.01; ** p<0.05; * p<0.10.