

Medical Advancements and Later Life Mortality: Evidence from Sulfa Drugs^{*}

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Abstract

We study the long-run consequences of a major early-life exposure to a medical innovation: the rapid diffusion of sulfonamide antibiotics in the late 1930s. We leverage large cross-state differences in pre-sulfa mortality from maternal infections, scarlet fever, and influenza/pneumonia to construct plausibly exogenous variation in the intensity of early-life exposure among U.S. birth cohorts. Linking these baseline differences to more than 34 million NCHS death records and 5 million Census–Numident observations, we find that children aged 0–4 in high-mortality states at the time of sulfa’s introduction experience longer adult lifespans—roughly 1.4 months per standard-deviation increase in baseline disease burden. The estimates are robust across specifications, appear in both administrative datasets, and are concentrated among children from low-SES families and rural areas. Mechanism analyses reveal reductions in infant mortality and adult cognitive, ambulatory, and functional disabilities, alongside modest but significant increases in adult education and income. The results show that major medical innovations can generate both immediate mortality gains and persistent improvements in longevity over the life course.

Keywords: Mortality, Longevity, Sulfa Drug, Early-Life Exposures

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1. Introduction

Population aging has intensified interest in the determinants of later-life health and longevity, as the share of Americans aged 65 and older is projected to rise from 17 percent in 2023 to 23 percent by 2050 (Mather and Scommegna 2024). A large literature shows that aging trajectories are shaped not only by adult exposures but by early-life environments, including nutritional deprivation, infectious disease exposure, environmental toxins, and economic or family conditions (Almond et al. 2018; Barker 1990, 1992, 1994, 1995, 1997; Cunha and Heckman 2007; Heckman 2007; Heckman et al. 2013; Currie et al. 2014; Fletcher 2015; Hayward and Gorman 2004; Lindeboom et al. 2010; Lleras-Muney et al. 2022; Van den Berg et al. 2006). Yet the long-run consequences of early-life medical innovation — as distinct from nutritional, environmental, or economic shocks — remain poorly understood at a national scale.

On the eve of World War II, American physicians gained access to sulfonamide antibiotics, the first widely effective antibacterial therapy, and within two years mortality from pneumonia, scarlet fever, and maternal infections had fallen sharply across the United States. By 1941, an estimated 10 to 15 million Americans were receiving sulfa treatments annually (Hecht 1984). Whether the survival gains produced in early childhood translated into lasting improvements in adult health and longevity is a question with direct implications for how we evaluate breakthrough medical technologies — and one that has not previously been answered at a national scale in the United States.

This paper provides the first national-scale U.S. evidence on the long-run effects of early-life sulfa exposure on adult longevity. Using more than 34 million administrative death records from the National Center for Health Statistics (NCHS) and over 5 million linked Census-Numident observations (Breen et al. 2023; Goldstein et al. 2021, 2023), we find that children aged 0–4 in states with higher pre-sulfa infectious disease burden lived meaningfully longer lives as adults. A one-standard-deviation increase in baseline mortality from sulfa-treatable diseases raises adult lifespan by approximately 1.4 months — an effect equivalent to roughly 3.6 percent of the total gain in conditional life expectancy experienced by U.S. cohorts between 1920 and 1940 (Social Security 2024). These gains are concentrated among children exposed in the first years of life, are robust across specifications and datasets, and are largest for children from low-SES families and rural areas — those who faced the greatest infectious disease burden before sulfa's arrival.

The central empirical challenge in estimating these effects is that exposure to sulfa drugs was not random. States with higher pre-sulfa disease burden may have differed from lower-burden states in ways that independently shaped adult longevity. We address this through a difference-in-differences design that exploits two sources of plausibly exogenous variation: the sharp, nationwide timing of sulfa diffusion beginning around 1937, and predetermined cross-state differences in pre-sulfa mortality from sulfa-treatable diseases measured in 1930–1936. Because sulfonamides were not patentable, large-scale production began immediately upon introduction and was not driven by state-level health investments or contemporaneous policy reforms (Jayachandran, Lleras-Muney, and Smith 2010). Baseline disease burden, measured in the years before sulfa's arrival, captures long-standing environmental and demographic conditions unrelated to subsequent medical innovation (Bhalotra and Venkataramani 2015). Interacting these predetermined state differences with age-specific exposure at the time of sulfa's diffusion generates variation in treatment intensity that is orthogonal to the factors that typically confound historical mortality research. Pre-period event-study estimates confirm that high- and low-burden states followed parallel longevity trajectories before 1937, and placebo tests using diseases unaffected by sulfa — tuberculosis and malaria — produce null results, supporting a causal interpretation of our findings.

The long-run consequences of early-life medical innovation on adult survival are theoretically ambiguous. The fetal origins hypothesis (Barker 1990, 1992, 1994, 1995, 1997) and the broader early-life origins literature document that reductions in early-life infectious disease exposure can produce persistent gains in adult health capital through improved physiological development (Almond et al. 2018; Heckman 2007). Historical evidence reinforces this view: hookworm eradication (Bleakley 2007), anti-malarial campaigns (Bleakley 2010), tuberculosis vaccination (Bütikofer and Salvanes 2018), infant home-visiting programs (Hjort et al. 2017; Bhalotra, Karlsson, and Nilsson 2017), and maternity-care expansions (Lazuka 2024) all produced lasting improvements in health and human capital. At the same time, rapid survival gains may alter the composition of survivors by allowing frailer children to live through high-risk disease environments, potentially offsetting true health improvements in adulthood (Bhalotra and Venkataramani 2015). These competing forces make the long-run impact of early-life medical innovation on longevity an empirical question. Our setting is well-suited to resolve it: the non-patentable, rapid diffusion of sulfa drugs, combined with large and predetermined cross-state

variation in disease burden, allows us to identify the direction and magnitude of the net effect while formally examining the role of selective survival.

We document four key findings. First, early-life exposure to sulfa diffusion between ages 0–4 produced meaningful and statistically significant gains in adult longevity: a one-standard-deviation increase in pre-sulfa baseline disease burden raises age at death by 1.4–1.7 months, with estimates stable across fixed-effects structures, alternative exposure windows, and multiple constructions of the baseline mortality index. Second, the longevity gains are concentrated among cohorts exposed in infancy and early childhood — exposure at birth yields the largest effect (approximately 2.5 months), with the magnitude declining monotonically as age at exposure increases, consistent with biomedical evidence that susceptibility to pneumonia and related infections is highest in the first years of life (CDC, 2024). Third, the benefits are not evenly distributed: children born in rural areas and from low-SES families experience the largest longevity gains, with the rural effect approximately one-third larger than the urban effect, reflecting higher baseline disease burdens and weaker health infrastructure in these settings. Fourth, mechanism analyses reveal that more heavily exposed cohorts exhibit statistically significant reductions in cognitive, ambulatory, mobility, and self-care disabilities in adulthood — the primary channel linking early sulfa exposure to longer lives — with improvements in schooling and earnings emerging as statistically significant but modest in magnitude secondary pathways, consistent with evidence from Medicaid’s rollout showing that early-life medical care reduces adult disability decades later (Goodman-Bacon 2016).

The aggregate stakes of these long-run effects are easily understated. Per person, the longevity gain we estimate is modest, on the order of one to two months. But sulfa drugs differed from many modern pharmaceutical innovations along two important margins: reach and timing. They were inexpensive, rapidly diffused, and used against diseases that affected infants, mothers, and children across broad segments of the population. By contrast, many late-life pharmaceutical innovations generate gains of a similar order of magnitude among much smaller or more selected patient groups. The novel cancer drugs approved by the U.S. Food and Drug Administration between 2000 and 2016 extended overall survival by a median of only 2.4 months (Ladanie et al., 2020), and reach the roughly two million Americans diagnosed with cancer each year (Siegel et al., 2026); only about a third of cancer-drug approvals in Europe (2009–2013) were supported by evidence

of a survival benefit at the time of approval (Davis et al., 2017).⁵ Statins reach a much broader population: 27.9 percent of U.S. adults aged 40 and older used a cholesterol-lowering medication in 2003-2012, and 83 percent of those users took a statin (Gu, 2014). But the measured survival gains within randomized statin trials are small, with death postponed by a median of 3.2 to 4.1 days over trial horizons of 2.0 to 6.1 years (Kristensen et al., 2015). Moreover, for a statin-like primary-prevention therapy, 93 percent of treated 50-year-old men gain no additional lifespan while the remaining 7 percent gain a mean of about eight years (Finegold et al., 2016). Sulfa combined the broad reach of a population-level therapy with the timing advantage of acting in infancy and early childhood. By 1941, sulfa drugs were administered to an estimated 10 to 15 million Americans annually (Hecht 1984), and their introduction reduced contemporaneous maternal, pneumonia, and scarlet-fever mortality by 17 to 65 percent (Jayachandran et al., 2010). As we show, these immediate mortality gains were accompanied by persistent improvements in adult longevity among cohorts exposed in early childhood. Because sulfa reached broad segments of the population and acted early in life, when each death averted returned a full expected lifetime and each developmental improvement could compound over decades, even modest individual-level gains can cumulate into large aggregate life-years. This makes the aggregate population value of sulfa plausibly first-order relative to that of many modern pharmaceuticals.

This paper contributes to three strands of literature. First, we advance the early-life origins literature (Almond et al. 2018; Barker 1990; Heckman 2007) by providing large-scale national evidence that reductions in early-life infectious disease exposure — achieved through medical innovation rather than nutritional or environmental intervention — produced persistent gains in adult survival. Prior studies in this tradition typically examine extreme shocks or narrow public health interventions; our setting captures the diffusion of a major medical technology across the full distribution of U.S. birth cohorts, allowing us to identify effects with greater external validity. Second, we extend the literature on medical innovation and longevity (Lichtenberg 2015, 2022; Wamble, Ciarametaro, and Dubois 2019; Wen et al. 2022) by documenting that the welfare effects

⁵ Access to cancer therapy is not universal. (Robbins et al., 2010) report that 15 percent of uninsured nonelderly patients with stage IV colorectal cancer received no treatment, compared with 4 percent of privately insured patients; (Walker et al., 2014) similarly show that uninsured patients with the ten deadliest cancers were much less likely to receive cancer-directed surgery and/or radiation than non-Medicaid insured patients, 62.1 percent versus 79.6 percent. Even among insured cancer patients, treatment access can be constrained by affordability. (Zafar et al., 2013) find that 46% reduced spending on food and clothing; 20% took less medication than prescribed; and 24% avoided filling prescriptions altogether to save money.

of the first widely accessible antibacterial therapy extended across the entire life course. Jayachandran, Lleras-Muney, and Smith (2010) establish that sulfa drugs produced large contemporaneous reductions in U.S. mortality from pneumonia, scarlet fever, and maternal infections. Bhalotra, Clarke, and Venkataramani (2025) examine the same U.S. setting to study how early-life sulfa exposure shaped adult education, employment, and income, and — in an important extension — demonstrate how Jim Crow-era racial discrimination moderated these long-run economic returns for Black Americans. Lazuka (2020) uses the Swedish introduction of sulphapyridine to show that reduced infant pneumonia exposure raised later-life labor income by 2.8–5.1 percent, with health operating as the stronger mechanism. Our paper contributes to and builds on these studies in two principal respects. First, while existing work focuses on midlife socioeconomic outcomes observed in census records, our primary outcome is adult longevity itself. Using more than 34 million individual administrative death records, we provide the first national-scale U.S. evidence that early-life sulfa exposure raised age at death among observed adult decedents. These data also allow us to examine cause-specific mortality and age-at-death patterns across the observed adult mortality distribution, dimensions that census-based studies cannot support. Second, our mechanism analyses document reductions in seven distinct adult disability dimensions alongside the schooling and income effects reported in prior work, providing a more comprehensive assessment of how early-life sulfa exposure shaped adult health capital. Third, we contribute to research on how early-life health investments shape adult disability and functional health (Goodman-Bacon 2016; Schiman, Kaestner and Lo Sasso 2019) by showing that sulfa exposure produced statistically significant reductions across multiple disability dimensions, with especially pronounced benefits for children from low-SES and rural backgrounds, underscoring that early medical innovations can narrow long-run health inequalities when the largest gains accrue to the most disadvantaged children.

The remainder of the paper proceeds as follows. Section II provides background on sulfa drugs and their diffusion in the United States. Section III describes our data and empirical strategy. Section IV presents main results on longevity. Section V reports heterogeneity analyses and robustness checks. Section VI examines mechanisms. Section VII concludes.

2. Background on sulfa drugs

The introduction of sulfonamide (“sulfa”) drugs in the mid-1930s represents a pivotal moment in the history of modern medicine. As the first widely effective antibacterial treatment, sulfa drugs transformed clinical practice and had profound population-level effects on mortality. Their discovery, diffusion, and adoption in the United States were part of a broader global wave of medical innovation, but the U.S. played a unique role both as an early adopter and as a setting in which the mortality impact was quickly observable at scale. Sulfonamides emerged from European laboratory research in the early 1930s, when German scientists identified the bacteriostatic properties of synthetic dyes (Lesch 2007). The earliest commercial agent, Prontosil, became available in the United States in early 1937, marking the beginning of what contemporaries referred to as the “sulfa craze” (Lesch 2007; Jayachandran et al. 2010). These early agents were only partially effective against *Streptococcus pneumoniae*, the primary cause of bacterial pneumonia, yet they were rapidly adopted because of their low cost, ease of administration, and the absence of comparably effective treatments. By 1938, a substantially more potent compound—sulfapyridine—had been developed and quickly introduced into American clinical use. Trial evidence showed dramatic reductions in case fatality rates among pneumonia inpatients, often in the range of 50–70 percent (Evans and Gaisford 1938; Gaisford 1939; Lesch 2007).

The timing of sulfa adoption in the U.S. coincided with a period of high mortality from streptococcal infections, pneumonia, scarlet fever, puerperal sepsis, and meningitis. Pneumonia alone accounted for roughly 8 percent of all U.S. deaths in 1936 (Jayachandran et al. 2010). Against this backdrop, the availability of an affordable and effective antibacterial agent represented a major therapeutic breakthrough. Consequently, the production and sale of sulfa drugs expanded rapidly. By 1941, an estimated 10 to 15 million Americans were receiving sulfa treatments annually (Hecht 1984). This widespread diffusion occurred before the next major antibiotic, penicillin, became broadly available, making the period from 1937 to the mid-1940s a uniquely clean window for studying the effects of medical innovation on later-life mortality.

The population-level effects of sulfa drugs in the United States were substantial. Using national time-series data, Jayachandran et al. (2010) document a clear structural break in mortality from sulfa-treatable infectious diseases beginning in 1937, consistent with rapid adoption following their introduction. All-age mortality from bacterial pneumonia fell by approximately 17 percent, while infant pneumonia mortality fell by nearly 30 percent—the largest decline across any age

group. The steeper decline for infants mirrors their higher infection rates and greater baseline vulnerability to respiratory pathogens. Clinical data reinforce these patterns, noting that sulfa treatment led to rapid reductions in fever and improvements in clinical status among infants and young children (Connolly et al. 2012). The diffusion of sulfa drugs in the United States was also characterized by the absence of competing innovations that could confound mortality trends. As Jayachandran et al. (2010) emphasize, no other major changes in medical technology occurred during this period, and penicillin did not become widely available until the mid-1940s. This allowed sulfa drugs to play an outsized role in shaping the sharp decline in infectious disease mortality during the late 1930s. Indeed, maternal mortality declines in U.S. hospitals accelerated only after sulfa therapies became widespread, suggesting that earlier hospital-based care had limited efficacy in the absence of effective pharmaceuticals (Thomasson and Treber 2008).

Although much of the global scientific development occurred in Europe, sulfa drugs were adopted in the U.S. at an especially rapid pace. Their popularity stemmed in part from heavy promotion and in part from their perceived “miracle” effects relative to previous treatments. Prior to the sulfa era, therapeutic options included serum therapies, which were costly, difficult to administer, and often ineffective, especially among infants. Sulfa drugs’ oral administration and low production costs made them accessible to a broad swath of the American population, facilitating widespread diffusion across regions and socioeconomic groups (Lesch 2007). International adoption patterns provide additional context for understanding the U.S. experience. In Sweden, for example, sulfonamides were introduced into pneumonia treatment in 1939, and archival evidence shows rapid nationwide access regardless of regional socioeconomic characteristics (Lazuka 2018). Similar to the U.S., Sweden experienced steep declines in pneumonia mortality—on the order of 30 percent—within a few years of sulfa introduction. These global patterns underscore the drug’s broad therapeutic value, but the U.S. differs in scale and in the heterogeneity of pre-sulfa disease environments across states, making it an especially important context for examining distributional consequences.

3. Data

Our primary data source is the Multiple Cause of Death (MCOB) files from the National Center for Health Statistics (NCHS), covering the universe of deaths in the United States between 1979 and 2020 (NCHS, 2020). We focus on deaths occurring after 1979, as this is the first year in which

the NCHS data report state of birth, allowing us to link deaths to early-life conditions. The analysis period ends in 2020 to avoid confounding from COVID-19–related mortality, which could bias the estimates of long-term effects of early-life medical innovation. These restricted-use records include demographic details such as age, sex, race, state of birth, and state of death, which allow us to construct mortality measures at the individual level. We restrict the analysis to birth cohorts of 1921–1942 to ensure that our sample includes multiple cohorts born after the introduction of sulfa drugs, as well as several cohorts who were exposed during infancy, early childhood, and later childhood years. Using these data, we compute age at death in months and link each individual’s birth state and cohort to state-level baseline disease mortality rates from the pre-sulfa period (1930–1936). The richness and coverage of the NCHS files enable us to examine population-wide mortality responses to early-life exposure to medical innovation across race and gender groups.

To complement the analysis of the NCHS data, we employ the Social Security Administration Numerical Identification (Numident) data, extracted from the Censoc Project (Breen et al., 2023; Breen & Osborne, 2022; Goldstein et al., 2021, 2023; Osborne, 2023). The Numident data covers deaths to individuals who died between the years 1988 – 2005. The key advantage of the CenSoc-Extract Numident data is its linkage to the complete-count 1940 U.S. Census at the individual level. This linkage enables us to observe a rich set of granular geographic characteristics and detailed family attributes, which can be incorporated as additional covariates and exploited to conduct heterogeneity analyses, features that are unavailable in the NCHS data.

To measure the intensity of sulfa drug impacts, we use baseline prevalence rates of diseases that were most directly affected by sulfa drug introduction. These include maternal mortality, scarlet fever, and influenza and pneumonia death rates. Given our focus on early-life and early-childhood exposures—and consistent with medical evidence indicating that influenza and pneumonia were disproportionately prevalent among children up to age four—we use the pre-introduction influenza and pneumonia mortality rate as our primary baseline exposure measure. The state-level data on pre-introduction disease rates are extracted from Jayachandran et al. (2010), who construct disease-specific mortality rates for 1930–1936 directly from the Vital Statistics of the United States. Additional state-level covariates are drawn from the 1920–1950 decennial censuses and linearly interpolated for inter-decennial years. These include demographic and socioeconomic indicators such as literacy rate, share married, female labor force participation, socioeconomic

index, occupational income scores, and per capita numbers of midwives and nurses, all obtained from IPUMS (Ruggles et al., 2024).

To explore potential mechanisms, we combine these historical data with information from the 1970 and 1980 Decennial Censuses and the American Community Survey (ACS) 2001–2020, also obtained from IPUMS. These sources provide detailed information on adult educational attainment, labor market outcomes, and health-related disabilities for the same birth cohorts, allowing us to trace the long-run socioeconomic and health effects of early-life exposure to sulfa drugs. In addition, we examine the impacts on infant mortality and birth rates using vital statistics data compiled by (Bailey et al., 2016), which provide complementary evidence on short-run demographic responses to the diffusion of medical innovations.

We additionally use data from the National Health Interview Survey (NHIS), a nationally representative repeated cross-section of the U.S. civilian non-institutionalized population conducted by the National Center for Health Statistics (NCHS). The data are obtained from IPUMS Health Surveys (Blewett et al. 2025). The NHIS provides detailed information on adult health and socioeconomic characteristics, which we use to examine potential mechanisms linking early-life exposure to later-life outcomes. Because the NHIS does not contain information on state of birth, we proxy early-life location using the respondent’s current city of residence at the time of the survey and assign the corresponding state as the individual’s birth state. This approach introduces potential measurement error due to migration; however, such misclassification is likely to attenuate estimated effects toward zero and therefore provides a conservative test of our main results. Using the NHIS, we examine adult outcomes including height, insurance status (having no insurance and private insurance coverage), and activity limitations, which capture key dimensions of long-run health and access to care.

4. Empirical Strategy

Our empirical strategy follows prior work that exploits sharp cohort-specific reductions in infectious disease following the sudden arrival of sulfa drugs in 1936–37 (Bhalotra and Venkataramani 2015; Jayachandran et al. 2010). Sulfa compounds diffused rapidly because they were not patentable and large-scale production began immediately (Jayachandran et al. 2010). At the same time, U.S. states differed markedly in their pre-sulfa mortality burden from influenza and pneumonia—the diseases most responsive to sulfa drugs—reflecting long-standing environmental

and demographic conditions (Bhalotra and Venkataramani 2015). These features generate plausibly exogenous variation in early-life exposure intensity across birth cohorts and states. We leverage this variation using difference-in-differences (DiD) event-study models that compare birth cohorts differentially exposed to the introduction of sulfa drugs according to (i) their age at the time of diffusion and (ii) their state’s baseline disease burden. Baseline burden is measured using state-level influenza and pneumonia mortality rates in the pre-sulfa period (1930–1936). Because these conditions predate the medical innovation, interacting them with birth cohorts provides predetermined variation that is not driven by selective state or household behavior. Formally, we estimate

$$y_{isrb} = \alpha_0 + \sum_{j \neq 8} \eta_j I(AAE_b = j) \times BR_s^* + \beta X_i + \delta Z_{sb} + \xi_s + \zeta_{rb} + \varepsilon_{isrb}, \quad (1)$$

where y_{isrb} denotes the age at death (in months) of individual i , born in state s , census region r , and birth cohort b . The variable BR_s^* is the standardized pre-sulfa mortality burden from influenza and pneumonia. AAE_b is the age at which the individual encountered sulfa’s introduction, and $I(AAE_b = j)$ are event-time indicators, with age eight omitted. The coefficients η_j describe how longevity changes for cohorts first exposed at age j , relative to those turning eight in 1936, for a one-standard-deviation increase in baseline mortality. X_i includes individual controls (race, gender). Z_{sb} captures birth-state-by-birth-year characteristics—socioeconomic conditions, literacy, marriage rates, female labor force participation, occupational income scores, and per-capita midwives and nurses. State fixed effects ξ_s absorb time-invariant cross-state differences, while region-by-birth-year fixed effects ζ_{rb} allow regions to follow distinct time paths in mortality decline and economic change (Fenelon 2013). Standard errors are clustered by birth state and birth year to address serial and spatial correlation.

As a complementary specification, we summarize exposure with an indicator for whether an individual was age 0–4 in 1936–37—the window emphasized by medical and epidemiological evidence as most sensitive to infectious-disease conditions. We estimate

$$y_{isrb} = \alpha_0 + \eta EXP_b \times BR_s^* + \beta X_i + \delta Z_{sb} + \xi_s + \zeta_{rb} + \varepsilon_{isrb}, \quad (2)$$

where $EXP_b = 1$ for cohorts exposed up to age four and 0 otherwise. All other parameters and controls are defined as above. This model recovers the average treatment effect of early-life exposure to sulfa diffusion in higher-burden states.

Identification

Identification rests on two key features. First, the timing shock—the sudden diffusion of sulfa drugs beginning in 1937—is plausibly exogenous.⁶ Jayachandran et al. (2010) document that large-scale production began immediately and was not driven by contemporaneous health reforms or state-specific investments, and no other major infectious-disease medical breakthroughs occurred in the late 1930s; penicillin did not diffuse until the mid-1940s. Second, treatment intensity is based on predetermined disease burden: baseline mortality is measured in 1930–1936, well before sulfa’s arrival, and captures stable environmental and demographic conditions unrelated to subsequent medical innovation (Bhalotra and Venkataramani 2015). The inclusion of rich controls and fixed effects—birth-state, birth-year, and region-by-birth-year—mitigates concerns about omitted variables and allows for flexible regional trends in mortality and economic change. Following Bhalotra and Venkataramani (2015), we also conduct robustness checks and placebo exercises to assess potential selective survival and other threats to identification. Their work uses counterfactual survival simulations to show that differential survival does not generate spurious long-run gradients. We adopt analogous approaches and find that our estimates are stable across specifications that account for selection, alternative control structures, and placebo exposures, supporting a causal interpretation of the estimated effects.

Because treatment intensity varies continuously with pre-sulfa disease burden, identification relies on a generalized parallel-trends assumption that is stronger than in a binary DiD setting: absent sulfa diffusion, cohorts exposed at different ages would have experienced similar changes in adult longevity across the distribution of baseline disease burden, conditional on the included fixed effects and controls (Callaway, Goodman-Bacon, and Sant’Anna 2024). Appendix A5 provides supporting evidence by replacing the continuous baseline mortality measure with nonparametric quartile-based exposure groups; the estimates increase across higher-burden quartiles, consistent

⁶ Mortality trends are reported in Figure 2. Mortality declines steadily over the sample period for both all ages and children ages 0–4, with higher initial levels and a sharper reduction among young children. The decline becomes more pronounced after 1937, particularly for influenza and pneumonia mortality.

with a dose-response relationship rather than the results being driven solely by the linear functional-form assumption.

A further identification concern is selective survival: sulfa drugs saved marginal infants who previously would have died from pneumonia and related infections, but these additional survivors are, by construction, drawn from the lower tail of the health distribution. In states with higher pre-sulfa disease burden, the post-sulfa surviving cohort is therefore more negatively selected on health than in lower-burden states. This selection operates against finding longevity gains — it biases our estimates downward rather than upward, implying that our estimates are conservative lower bounds on the true physiological benefits among children whose development was genuinely improved by reduced infectious disease exposure. The same logic applies to the disability results reported in Section VI. The balancing tests in Appendix A4 confirm that early-life sulfa exposure is not associated with meaningful changes in cohort demographic composition, and the infant mortality results in Table 8 show that the relevant cohort-level reduction is modest in scale (approximately 2.5 deaths per 1,000 births). Taken together, these findings suggest that compositional change from marginal survivors is unlikely to drive the longevity effects we document; if anything, the direction of this bias reinforces the interpretation of our estimates as conservative.

5. Results

Descriptive Statistics

Figures 1 and 2 summarize the two sources of variation used in our research design. Figure 1 documents large pre-existing cross-state differences in baseline sulfa-treatable mortality, while Figure 2 shows a pronounced post-1937 decline in influenza and pneumonia mortality, particularly among children ages 0–4, consistent with the timing and age profile of sulfa’s short-run mortality effects.

Table 1 reports summary statistics for cohorts born in states with above- and below-median early-life exposure, defined by the interaction of pre-1937 sulfa-treatable mortality and being ages 0–4 at the time of sulfa’s introduction. High-exposure states were systematically more disadvantaged across all data sources, with lower mean longevity in the NCHS death records (886 vs. 903 months), a substantially larger share of Black births (22 percent vs. 4 percent), and higher pre-sulfa maternal and influenza/pneumonia mortality. County-level covariates confirm that high-exposure

states were poorer and more rural, with lower literacy, lower socioeconomic scores, fewer nurses per capita, and more midwives per capita. Similar patterns appear in the CenSoc–Numident linked sample and in Census–ACS data, where cohorts from high-exposure states complete fewer years of schooling and face higher disability prevalence. Vital statistics likewise show higher infant mortality from 1920–1950. These patterns underscore substantial baseline disadvantage and motivate the inclusion of extensive fixed effects and pre-sulfa controls.

Main Effects on Longevity

Table 2 reports our main estimates of the effect of early-life exposure to sulfa drugs on adult longevity, using age at death in months as the outcome. The analysis pools more than 34 million NCHS death records for birth cohorts 1921–1942 observed dying between 1979 and 2020. The key regressor is the standardized pre-1937 baseline mortality rate from sulfa-treatable diseases interacted with an indicator for being aged 0–4 at the time of sulfa introduction. In the specification with birth-state and birth-year fixed effects plus the full set of individual and state-level controls (column 1), a one-standard-deviation increase in baseline mortality is associated with a statistically significant increase in longevity of about 1.7 months. When we further allow for birth-region-by-birth-year fixed effects (column 2), the point estimate is slightly attenuated but remains large and precisely estimated at about 1.4 months.

Supplementary Analyses

In this section, we present additional analyses that provide further insight into the timing and structure of the effects of early-life sulfa exposure. These analyses help assess whether the estimated effects on longevity reflect genuine early-life mechanisms or are driven by spurious patterns in the data. Figure 3 through Figure 5 summarize these supplementary results and demonstrate patterns consistent with our main findings.

We begin by examining dynamic treatment effects by age at exposure. Figure 3 and Figure 4 plot the interaction between baseline mortality and indicators for specific exposure ages, using the NCHS and CenSoc-Numident samples, respectively. Both figures illustrate point estimates and 95 percent confidence intervals, with regressions including birth-state fixed effects, birth-region-by-birth-year fixed effects, and the full set of individual, family, and state controls described earlier. Across both datasets, a consistent pattern emerges: the positive effects of sulfa exposure are concentrated among cohorts exposed in early childhood, particularly in the first few years of life. In Figure 3 (NCHS), the estimated effects are close to zero or slightly negative for

exposure occurring in later childhood and become increasingly positive as the exposure age approaches birth, peaking for those exposed between ages 0 and 2. A similar though noisier pattern appears in Figure 4 (CenSoc-Numident), where the coefficients again increase in magnitude as exposure moves earlier in childhood. The alignment of these dynamic profiles across two distinct datasets reinforces the interpretation that the estimated gains in longevity reflect biologically meaningful early-life vulnerability, rather than secular trends or compositional shifts across cohorts.

A natural question raised by Figure 3 is whether the treatment definition should be restricted to infants or birth cohorts born after the introduction of sulfa drugs. While the event-study coefficients are largest for those exposed at birth or shortly thereafter, the full age profile indicates that meaningful effects extend beyond infancy. Appendix Table A-6 reports age-specific estimates across narrow exposure windows and shows a clear gradient: exposure at birth yields the largest gains (≈ 2.5 months), but the coefficients for ages 1–4 remain positive, sizable, and statistically significant, and even ages 5–7 exhibit modest positive effects. These patterns are consistent with medical evidence that susceptibility to pneumonia and related infections remains high through roughly age five and with prior work emphasizing early-childhood vulnerability (e.g., Hoehn-Velasco 2021). Redefining treatment solely on the basis of birth timing—for example, classifying only children born in 1936–37 as exposed—would therefore misclassify a large share of children who were between ages 1–4 when sulfa diffused and who, according to both the event-study and Appendix Table A-6, experienced substantial benefits. Such a specification would place these cohorts into the control group despite clear evidence of treatment effects, mechanically attenuating the estimates and complicating interpretation. The 0–4 exposure definition used in our main specification thus reflects a data-driven and medically grounded choice rather than an arbitrary cutoff. It appropriately captures the full set of early-life cohorts who were biologically vulnerable and who experienced the largest contemporaneous reductions in sulfa-treatable mortality. The combination of event-study evidence and age-specific regressions therefore supports maintaining ages 0–4 as the primary exposure window.

Figure 4 reports estimates from regressions where the outcome is an indicator for surviving beyond age Z . The estimated effects increase monotonically with the survival threshold, indicating that the impact on longevity is concentrated in the upper tail of the age-at-death distribution. In other words, the results imply increases in the probability of surviving to more advanced ages. This

pattern complements the baseline results by showing that the estimated effects are not driven by a particular cutoff, but instead become more pronounced at higher ages. Taken together, the findings suggest that the main effects on mortality operate through changes in late-life survival rather than shifts at younger or mid-range ages.

Heterogeneity by Race and Gender

Results in Table 3 examine whether the longevity effects of early-life sulfa exposure differ by race or gender. The estimates are remarkably similar across subsamples. Among whites, a one-standard-deviation increase in baseline disease burden raises age at death by roughly 1.4 months, and the corresponding estimate for Blacks is about 1.5 months. Both effects are precise and operate from different baseline mortality levels (mean age at death of 901 months for whites versus 855 months for Blacks). Gender patterns are similarly uniform: the estimated gains are approximately 1.5 months for men and 1.4 months for women. Although women live longer on average (917 months versus 877 months for men), the proportional longevity improvements are comparable. Taken together, these results show that the long-run benefits of early-life exposure to sulfa diffusion are broad-based and not concentrated in any single demographic group.

Family Background and Demographic Heterogeneity

To examine whether family socioeconomic background and place of birth moderate the long-run effects, we use the CenSoc–Numident linked sample in Table 4. These regressions, estimated on roughly 5.2 million individuals born 1920–1940, relate early-life exposure to rich parental characteristics observed in the 1940 Census. In the full sample, higher exposure is associated with an increase in age at death of about one-third of a month—a smaller estimate than in the NCHS data but still meaningful relative to the mean lifespan of 853 months.⁷ Splitting by place of birth, we find slightly larger gains among rural-born individuals (≈ 0.4 months) than among urban-born

⁷ A potential concern is that estimates based on the CenSoc–Numident sample (approximately 0.36 months) are smaller than those obtained using NCHS data (approximately 1.4 months). To assess whether this difference reflects sample composition rather than underlying differences in effects, Appendix Table A-7 re-estimates the NCHS specification across alternative death-year windows. Restricting the NCHS sample to earlier death cohorts yields substantially smaller estimates. For example, when limiting the sample to deaths occurring between 1988 and 2005, the estimated effect is 0.36 months—closely matching the magnitude observed in the Numident data. Expanding the sample to include later death years progressively increases the estimated effect, reaching approximately 1.4 months in the full 1979–2020 sample. These patterns indicate that the difference between NCHS and Numident estimates is largely driven by differences in the distribution of death years across datasets. In particular, the Numident-linked sample disproportionately captures earlier deaths, which correspond to lower mean ages at death and smaller estimated effects. By contrast, the full NCHS sample includes later deaths with higher average longevity and larger estimated impacts.

individuals (≈ 0.3 months), consistent with higher pre-sulfa disease burdens and weaker health infrastructure in rural settings. Family background reveals a clear socioeconomic gradient. Individuals with fathers above the median socioeconomic index experience longevity gains of roughly 0.5 months, compared with about 0.3 months for those with fathers below the median. A similar pattern emerges for maternal education: children whose mothers completed high school exhibit larger gains than those whose mothers did not, suggesting that better-resourced families may have been better positioned to benefit from early-life reductions in infectious-disease exposure.

Two interpretive observations follow from these heterogeneity results. First, the finding that rural-born children benefit more aligns with a convergence interpretation: before sulfa's introduction, high-burden states were systematically poorer, more rural, and less well-served by health infrastructure (Table 1), so an affordable, widely distributed antibacterial therapy disproportionately reached children whose infectious disease exposure had been greatest. Second, the SES gradient — where children of higher-SES fathers experience larger gains — is consistent with resource complementarity: better-resourced families may have been better positioned to convert early-life health improvements into sustained investments in nutrition, follow-up care, and schooling that compound over time (Currie and Stabile 2003). The similar estimated effects by race (Table 3), despite substantially higher pre-sulfa disease burdens among Black Americans, are consistent with evidence in Bhalotra, Clarke, and Venkataramani (2025) that institutional barriers — differential access to pharmacists in majority-Black communities and, in more discriminatory states, limited returns to health improvements — attenuated the conversion of early-life health gains into long-run longevity for Black cohorts.

Sensitivity to Exposure Windows and Baseline Measures

Table 5 evaluates the sensitivity of our findings to alternative definitions of the exposure window and to different constructions of the baseline mortality measure. We first estimate separate effects for exposure at birth (age 0), ages 1–4, and ages 5–7. The estimated impact is largest for exposure at birth: a one-standard-deviation increase in baseline influenza-and-pneumonia mortality is associated with a reduction in lifespan of roughly 2.5 months. The effect for exposure at ages 1–4 is smaller, about 1.5 months, and the estimate for ages 5–7 is positive but more modest at approximately 0.5 months. This pattern is consistent with evidence that early infancy is especially sensitive to infectious-disease conditions, with the influence of exposure diminishing as children

age. We next consider discrete measures of baseline disease burden. Interacting exposure with an indicator for being above the median baseline mortality yields an estimated effect of nearly 4 months, while defining exposure using the top two terciles produces an estimate of approximately 4.7 months. These patterns indicate that the long-run effects are larger in states with initially higher infectious-disease prevalence. Finally, we re-construct the baseline mortality index using alternative sets of sulfa-treatable diseases. A composite index of maternal mortality, scarlet fever, and influenza/pneumonia produces an estimate of roughly 1.5 months per standard deviation. Using maternal mortality or scarlet fever mortality individually generates coefficients of similar magnitude and statistical significance. Overall, the results are robust to alternative exposure windows and to multiple ways of measuring baseline disease burden.

Robustness to Alternative Specifications and Estimators

Table 6 reports a series of robustness checks designed to assess whether the estimated effects on longevity are sensitive to alternative specifications and identifying assumptions. Each specification targets a distinct concern relevant to historical mortality research and provides a test of the stability of the baseline findings. We begin by adding death-state fixed effects. Although treatment is assigned by state of birth and birth cohort, individuals may later migrate, and the geographic distribution of deaths may differ systematically across exposed and less-exposed cohorts. If high-exposure cohorts are more likely to die in states with different mortality regimes, medical infrastructure, or end-of-life conditions, estimates based only on birth-state exposure could partly reflect these later-life location differences. At the same time, state of death is measured after treatment and may itself be affected by early-life sulfa exposure through later-life health, income, migration, or residential choices. We therefore interpret this specification as a sensitivity check rather than as a preferred control set. The estimated coefficient remains virtually unchanged after adding death-state fixed effects, suggesting that the main results are not driven by persistent differences across states where individuals ultimately die, while also indicating that this post-treatment adjustment does not materially alter the estimated longevity effect.

Next, we introduce an extensive set of time-varying state covariates. These controls—covering demographic composition, schooling, occupational structure, and a range of socioeconomic indicators—address concerns that long-run state-level trends correlated with pre-sulfa disease environments may bias the estimates. Although the coefficient declines modestly, it remains large and statistically significant, suggesting that omitted observable state characteristics do not explain

the main findings. Note, however, that several of these covariates—including years of schooling, occupational scores, and marriage rates—are themselves potential mechanisms through which early-life sulfa exposure could improve adult outcomes (see Table 8). Including them as controls therefore risks absorbing part of the treatment effect, and this specification is best interpreted as a conservative lower bound on the true longevity gain. We also interact baseline mortality rates from other diseases with birth-year fixed effects to account for differential trends in disease environments; the results remain stable, further supporting the robustness of the estimates.

We further allow for birth-state-specific linear trends to account for the possibility that unobserved factors evolve gradually within states over time. The estimated coefficient remains positive and statistically significant, although somewhat attenuated. At the same time, it is important to note that including unit-specific trends can introduce “over-controlling” by absorbing variation that is plausibly part of the treatment effect itself, particularly when treatment intensity is correlated with pre-existing trends. As emphasized in the difference-in-differences literature (e.g., Angrist and Pischke (2009); Autor (2003); Wolfers (2006)), unit-specific trends may mechanically soak up identifying variation and bias estimates toward zero if the true effects evolve gradually over time. Therefore, while this specification provides a conservative check, it may not represent the preferred empirical specification.

We also examine robustness to functional form by replacing age at death in months with its logarithm. This proportional specification yields estimates that are consistent with the baseline results and remain highly statistically significant, indicating that the findings are not sensitive to the scaling of the outcome variable.

We then vary the cohort window. Expanding the sample to include birth cohorts 1920–1950, as well as restricting the sample to narrower cohort ranges, yields estimates that are similar in magnitude and statistically significant. These results suggest that the findings are not driven by particular cohort selections or spurious temporal patterns.

Finally, we consider alternative estimators. The Accelerated Failure Time (AFT) model provides a survival-based specification, while the Sun and Abraham (2021) estimator addresses potential biases in two-way fixed effects designs with staggered exposure timing. Both approaches produce estimates that are closely aligned with the baseline results. Overall, the consistency of the estimates across these specifications provides strong evidence that the main findings are not driven by modeling choices, sample selection, or estimator-specific concerns.

Robustness to Alternative Functional Forms of the Exposure Measure

A remaining concern is that our baseline specification assumes a linear relationship between the standardized pre-sulfa mortality index and the magnitude of long-run longevity gains. To assess whether our results are sensitive to this functional-form assumption, we re-estimate our main specification using non-parametric groupings of baseline disease burden. Specifically, we construct indicators for whether a birth state falls above the median, in the top two terciles, or in each quartile of the pre-sulfa influenza and pneumonia mortality distribution, and interact these indicators with the 0–4 exposure measure. Appendix Table A-5 reports the resulting coefficients. Across all three approaches—median splits, terciles, and quartiles—the estimated effects are qualitatively consistent with our baseline results. In each case, the coefficients increase in magnitude as we move toward higher-burden groups, with the largest effects concentrated in the top tercile and top quartile of the baseline mortality distribution. This pattern is exactly what the research design predicts: states with higher pre-sulfa infectious-disease mortality experienced larger contemporaneous declines in pneumonia and related infections following sulfa diffusion, and thus cohorts born there should exhibit greater long-run improvements. The fact that the estimates become larger in the highest-burden groups further supports this interpretation and provides reassurance that our main findings are not driven by linearity assumptions or outliers in the baseline index.

Effects on Cohort Life Expectancy

A remaining concern with the mortality analyses is that both the NCHS and CenSoc-Numident samples are based on observed deaths within specific windows. As a result, the main age-at-death estimates are observed only for individuals who survive long enough to enter the relevant death-record window and who die before the end of the observation period. If early-life sulfa exposure changed survival before entry into these data, or changed the probability of surviving beyond the end of the death-record window, the estimated effects on age at death could partly reflect selection into the observed sample.

To examine this concern, we use state-specific, sex-specific cohort life expectancy estimates, extracted from (Barbieri & Winant, 2025). These data allow us to examine the relationship between

early-life sulfa exposure and expected longevity at many different ages, including ages well before individuals enter the NCHS or CenSoc-Numident mortality windows.⁸

Figure 6 reports the estimated effect of early-life sulfa exposure on cohort life expectancy measured at various ages.⁹ The top panel reports effects in months, while the bottom panel reports effects as a percentage of mean cohort life expectancy at each age. The estimates are positive across the age distribution. In levels, the effect is already positive at young ages, rises during adolescence and early adulthood, and reaches roughly 1.5 months around ages 18 to 20. The percent effects show a similar pattern but with a different scale. The estimated effect is approximately 0.1 to 0.2 percent of mean cohort life expectancy through much of adulthood. At older ages, the percent effect rises because remaining life expectancy becomes smaller, so a given month-level change represents a larger percentage of the remaining expected lifespan. The central conclusion, however, is that the estimated effects are not confined to the ages or death windows used in the main mortality analyses.

Placebo and Falsification Tests

A natural concern in our empirical strategy is that the estimated relationship between early-life sulfa exposure and adult longevity may be driven by pre-existing state-level mortality patterns or by spurious correlations between baseline disease environments and long-run outcomes. To address this possibility, we implement two placebo tests designed to assess whether our specification mechanically generates “effects” even when exposure should be irrelevant. These exercises follow standard falsification logic in the medical-innovation literature, such as Jayachandran, Lleras-Muney, and Smith (2010), who examine diseases unaffected by sulfa drugs to verify that no discontinuities arise at the time of introduction. Our tests evaluate whether

⁸ There is an important caveat. The life expectancy data assign cohorts using state of residence rather than state of birth. This differs from our main NCHS and CenSoc-Numident analyses, which use state of birth to link individuals to early-life disease environments. Because some individuals migrate across states over the life course, state of residence is an imperfect proxy for early-life location. This measurement error is likely to attenuate the estimated effects, especially if migration weakens the relationship between current residence and childhood exposure to pre-sulfa disease environments. For this reason, the life expectancy estimates should be interpreted as complementary evidence rather than as the preferred specification. Nevertheless, they provide a useful diagnostic for evaluating whether the main findings are driven by survival into the observed death-record samples.

⁹ We begin the life-expectancy analysis at age 10 because the data start in 1941. Since sulfa treatment begins in 1936 and our main exposure window is ages 0 to 4, ages below 10 would not include untreated cohorts in the first available data year. At age 10, the 1941 data include cohorts born in 1931, who were age 5 in 1936 and therefore outside the treated exposure window.

spurious treatment effects arise when either (i) baseline mortality captures diseases not treatable by sulfa drugs, or (ii) exposure is assigned to ages at which sulfa drugs should not plausibly influence biological development.

The first placebo test replaces the baseline mortality rate from sulfa-treatable diseases with mortality from diseases for which sulfa drugs were not clinically effective—tuberculosis and malaria. This targets the concern that our estimated effects might simply reflect general cross-state mortality gradients or underlying socioeconomic disadvantage. If this were the case, interacting these placebo baseline rates with early-life exposure would also yield significant coefficients. Appendix Table A-1 shows that the estimated effects are near zero and statistically insignificant for both tuberculosis and malaria. These null results mirror the pattern documented by Jayachandran et al. (2010), who show that sulfa-related trend breaks occur only for treatable diseases. The absence of placebo effects reinforces that our results are not artifacts of unrelated mortality transitions or public-health changes but instead reflect disease-specific improvements generated by sulfa drugs.

The second placebo exercise assigns “exposure” to cohorts who would have been 16–18 or 19–21 years old in 1937—well beyond the developmental period during which infectious-disease environments are thought to have lasting effects. This test evaluates whether the interaction between baseline mortality and cohort timing might inadvertently capture omitted regional or cohort-specific trends. If such spurious trends were driving our estimates, we would observe similar effects among these older cohorts. Appendix Table A-2 shows no such evidence: across the full sample and within gender subsamples, the coefficients are small, statistically insignificant, and inconsistent in sign. These findings indicate that our specification does not mechanically generate relationships between baseline mortality and later-life outcomes when exposure is assigned outside the relevant developmental window. Instead, the effects we document are specific to cohorts plausibly influenced by early-life infectious-disease conditions.

The third placebo test further evaluates whether the empirical specification mechanically generates spurious relationships by randomly reassigning treatment across states and birth cohorts. Specifically, we shuffle the interaction between early-life exposure and baseline mortality rates across states and years, thereby breaking the true link between disease environment and timing of sulfa diffusion while preserving the overall distribution of the data. If the estimated effects were driven by chance correlations or by the structure of the regression design itself, we would expect

to recover statistically significant coefficients under these randomized assignments. Appendix Table A-3 reports the results from multiple randomization rounds. Across all specifications, the estimated coefficients are small in magnitude, statistically insignificant, and lack any consistent pattern in sign. These findings provide strong support that the baseline results are not artifacts of the empirical specification or driven by spurious correlations, but instead reflect a meaningful relationship between early-life exposure to sulfa drugs and later-life outcomes.

Balancing Tests: Demographic Composition

We next examine whether early-life exposure to sulfa drugs is associated with endogenous changes in the composition of the sample. A potential concern is that improvements in early-life survival may alter the demographic composition of cohorts observed at older ages, which could in turn influence estimated longevity outcomes. To assess this, we conduct balancing tests using demographic indicators as outcomes. Appendix Table A-4 reports the results. The estimated coefficients are extremely small in magnitude and economically negligible, with no consistent evidence of systematic compositional changes. While one coefficient is statistically significant, its magnitude is close to zero and does not suggest meaningful shifts in the underlying sample. These findings indicate that the main results are unlikely to be driven by endogenous changes in cohort composition.

Addressing Truncation of the Death Records

A natural concern in our setting is that the NCHS death files begin in 1979, which implies left truncation of the age-at-death distribution for older cohorts. Because deaths occurring prior to 1979 are not observed, individuals who died at younger ages are mechanically excluded from the sample, potentially biasing level-based measures of longevity upward. This concern is particularly relevant for cohorts born in the early 1920s, who reach older ages near the beginning of the observation window.

To assess whether truncation materially affects our conclusions, we examine how estimates vary across alternative death-year windows using the NCHS data. Appendix Table A-7 shows that restricting the sample to earlier death periods yields substantially smaller estimates. For example, limiting the sample to deaths occurring between 1988 and 2005 produces an estimated effect of 0.36 months—closely matching the magnitude observed in the CenSoc–Numident data. Expanding the death window to include later years increases the estimated effect monotonically, reaching approximately 1.4 months in the full 1979–2020 sample. This pattern indicates that

differences in estimated effects across datasets are primarily driven by the distribution of observed death years rather than truncation bias. In particular, samples restricted to earlier death windows capture individuals who die at younger ages and therefore yield smaller estimated effects, while the inclusion of later deaths raises average age at death and amplifies the estimated impact of early-life exposure.

As a complementary approach, Table 7 reports results using state-level cumulative mortality rates instead of individual-level age at death. Specifically, we define cumulative mortality as the share of individuals in a given state-cohort who die between 1980 and 2020, where the denominator is the population observed in the 1980 Census that survived to that year, and the numerator counts deaths occurring within this group over the subsequent period. By conditioning on survival to 1980, this measure avoids the mechanical truncation inherent in age-at-death outcomes while maintaining a specification analogous to our baseline design.

The estimated effects on cumulative mortality closely mirror the individual-level results, indicating that states with higher pre-sulfa disease burdens experienced larger long-run reductions in adult mortality. Taken together, these findings demonstrate that our results are not driven by left truncation of the death records and, if anything, should be interpreted as conservative estimates of the true effects. Consistent with the individual-level results, the estimates based on cumulative mortality rates indicate substantial reductions in adult mortality. In column (1), a one standard deviation increase in early-life exposure reduces cumulative mortality by approximately 3.3 percent. The effects are present across demographic groups but exhibit some heterogeneity. Among White individuals (column 2), the estimated reduction is about 1.8 percent, while for non-White individuals (column 3) the effect is larger, at approximately 4.8 percent, though both estimates are statistically significant. Splitting by gender, we find reductions of roughly 2.7 percent for females (column 4) and 3.9 percent for males (column 5). These patterns indicate that the mortality reductions associated with early-life sulfa exposure are broad-based, with somewhat larger effects among non-White individuals and males, consistent with higher baseline mortality risks in these groups.

Cause-of-Death Analyses

An additional question that frequently arises in work on historical mortality is whether estimated survival effects are concentrated among specific causes of death or reflect broad improvements in adult health. While cause-specific longevity is difficult to study directly due to left truncation in

age-at-death records, examining cause-specific mortality rates is feasible and follows common practice in the demographic and health economics literature. To address this question, Columns 6–9 of Table 7 report estimates using cumulative mortality rates disaggregated by major cause-of-death categories. The empirical design parallels our main specification: the numerator counts deaths attributed to a given cause between 1980 and 2020, and the denominator is the state-level population surviving to 1980 and observed in the 1980 Census, thereby avoiding the truncation concerns associated with individual-level age-at-death data. The results show a consistent pattern. States with higher pre-sulfa infectious disease burdens exhibit larger long-run reductions in mortality from infectious diseases (-0.025), those most plausibly affected by early-life improvements in disease environments, but also meaningful declines in chronic and non-communicable causes of death (-0.018), as well as other residual categories (-0.014). These patterns closely mirror the longevity results and provide additional evidence that early-life exposure to sulfa diffusion improved adult health broadly rather than shifting mortality across specific causes. Importantly, the coefficient on external and self-inflicted causes (-0.009) is small in magnitude and statistically insignificant, serving as a useful placebo. Because such causes are unlikely to be directly affected by early-life infectious disease environments, the absence of a meaningful effect in this category reinforces the interpretation that the main findings are not driven by compositional changes in cause-of-death assignment or by selective survival into unrelated mortality risks. Taken together, the cause-specific mortality evidence in Table 7 enhances the plausibility of our main results, as it provides an important corroborating test of mechanism, showing that the long-run gains we document reflect genuine improvements in adult health capital rather than artifacts of selective misreporting of causes or shifts across disease categories.

6. Mechanisms

Before examining specific mechanisms, it is useful to distinguish two channels through which early-life sulfa exposure could have improved adult longevity. For children who would have survived in both the pre- and post-sulfa era, reduced severity and duration of infectious disease exposure during early childhood may have improved developmental trajectories and long-run health — the sparing channel. For children who would otherwise have died, sulfa kept them alive, adding new marginal survivors to the cohort — the saving channel. Because these marginal survivors are drawn from the lower tail of the health distribution, the saving channel predicts a

modest downward drag on average adult health in high-burden states, working against the longevity and disability effects we estimate. The infant mortality result in Table 8 — a reduction of approximately 2.5 deaths per 1,000 births — provides a scale for the saving channel. Because these marginal survivors represent a small share of each cohort and their influence on our estimates runs downward, the longevity gains we document are most plausibly driven by the sparing channel: persistent physiological improvements among the large majority of children who would have survived regardless of sulfa’s availability.

Table 8 examines a broad set of potential mechanisms through which early-life exposure to sulfa-induced reductions in infectious-disease mortality may have translated into long-run gains in adult longevity. The table brings together evidence from Census microdata, the ACS, vital statistics, and the National Health Interview Survey, allowing us to evaluate effects on adult human capital, labor-market outcomes, disability, health insurance use, anthropometric outcomes, and early-life survival conditions.

Education. Exposure is associated with a modest reduction in the probability of having less than a high school education and a small increase in years of schooling. While both estimates are statistically significant, their magnitudes are small relative to cross-cohort variation in educational attainment. Using recent evidence that one additional year of schooling corresponds to roughly five additional months of life expectancy (Lleras-Muney et al., 2022), the implied contribution of the schooling changes in Table 8 is approximately 0.25 months of additional longevity. This represents about 18 percent of the reduced-form effects, suggesting that education may account for a meaningful but limited share of the overall impact. These results indicate that education operates as a secondary channel rather than the primary mechanism through which early-life exposure affects later-life mortality.

Adult earnings. Coefficients on log wage income and log total income are positive and statistically significant, while reliance on welfare income declines. Taken together, these patterns point to improved labor market outcomes among more heavily exposed cohorts, consistent with enhanced human capital and productivity over the life cycle. These findings suggest that early-life exposure contributed to better economic circumstances in adulthood, which may in turn support improved health and longevity.

Disability and functional health. More substantial evidence emerges from the disability measures. Across a wide set of adult functional limitations—including cognitive, ambulatory,

mobility, self-care, vision–hearing impairment, vision disability, and work disability—exposure to sulfa diffusion is consistently associated with reductions in disability risk. The uniform pattern across these indicators points to broad improvements in adult health capital among individuals more intensively exposed in early childhood. These findings align with the overall pattern of enhanced longevity and suggest that reductions in early-life infectious disease exposure had persistent effects on adult physical and cognitive functioning.

An important caveat is that disability measures are self-reported and available only for survivors observed in the ACS or Census data. If sulfa exposure increased survival among frailer individuals, the surviving pool in high-exposure states would be negatively selected on health, creating a headwind against detecting disability reductions. The estimated disability improvements should therefore be interpreted as conservative lower bounds on the true gains in adult functional health.

Marital status. Column (15) shows that a one standard deviation increase in exposure raises the probability of being married by 0.003 (0.3 percentage points) relative to a mean of 0.71, indicating a modest but precisely estimated increase in marital attachment. This finding is consistent with a large literature documenting that marriage is associated with improved health and lower mortality risk (Gardner & Oswald, 2004; van den Berg & Gupta, 2015; Waite & Gallagher, 2001), suggesting that early-life health improvements may translate into greater social stability and indirectly contribute to long-run longevity gains.

Early-life survival conditions. The vital statistics results show that cohorts with higher exposure experienced larger contemporaneous reductions in infant mortality, consistent with the well-documented short-run impact of sulfa drugs. As shown in column (16) of Table 8, a one standard deviation increase in exposure reduces infant mortality by approximately 2.5 deaths per 1,000 births. These effects are heterogeneous by race: column (17) indicates a decline of about 1.6 deaths per 1,000 births among Whites, while column (18) shows a larger reduction of approximately 4.8 deaths per 1,000 births among non-Whites, consistent with higher baseline mortality risks in these populations. In contrast, crude birth rates (column 20) and the sex ratio at birth (column 19) remain largely unchanged, suggesting that fertility behavior and the gender composition of births did not respond meaningfully to sulfa diffusion. Taken together, these patterns indicate that the observed long-run outcomes are unlikely to be driven by changes in cohort composition and instead reflect improvements in early-life survival and health conditions, particularly among more disadvantaged groups.

Midlife NHIS outcomes. Columns 21-24 supplement these long-run outcomes with midlife measures from the National Health Interview Survey, including anthropometric outcomes, insurance coverage, and additional disability indicators. The estimates are directionally consistent with the broader evidence, pointing to improved midlife health and access to resources. In particular, the results suggest gains in physical stature, increased private insurance coverage, and reductions in activity limitations. These findings are consistent with a large literature linking greater adult height to improved early-life conditions and lower mortality risk (Bozzoli et al., 2009; Wilson, 2019). Similarly, the increase in insurance coverage aligns with evidence that access to health insurance is associated with better health outcomes and reduced mortality (Goldin Ithai Lurie Janet Mccubbin et al., 2020; Goodman-Bacon, 2021). Taken together, these results reinforce the view that early-life exposure contributed to sustained improvements in health and economic well-being over the life cycle.

Because the NHIS does not report individuals' state of birth, we construct early-life exposure using the respondent's current state of residence. This introduces measurement error if individuals migrate between birth and the time of the survey, such that the assigned exposure deviates from true early-life exposure. To assess the extent of this measurement error, we use 5% 1980 census data in which both birth state and current state are observed and estimate the following relationship:

$$Exposure_i^{birth} = \alpha + \kappa Exposure_i^{current} + \beta X_i + \epsilon_i$$

where $Exposure_i^{birth}$ and $Exposure_i^{current}$ denote exposure constructed using birth state (the true measure), and Exposure using current state (proxied measure). Under the classical measurement error framework, when a mismeasured regressor is used in place of the true variable, the estimated coefficient is biased toward zero (attenuation bias). The magnitude of this attenuation is proportional to the signal-to-noise ratio κ , such that the estimated coefficient equals κ times the true effect.

The estimates of κ are reported in Appendix Table A-8. We find values of κ between 0.50 and 0.58, indicating that roughly half of the variation in current-state exposure corresponds to true early-life exposure. This implies that regression coefficients using current-state exposure are attenuated toward zero by approximately 40–50 percent. Accordingly, estimates based on NHIS data should be interpreted as conservative, representing lower bounds of the true effects of early-life exposure.

7. Conclusion

This paper provides new evidence on the long run effects of one of the earliest large scale antibacterial therapies: the introduction of sulfonamide drugs in the late 1930s. Prior research has established that sulfa drugs sharply reduced contemporaneous mortality from pneumonia, scarlet fever, and maternal infections (Jayachandran, Lleras Muney & Smith 2010) and generated improvements in later life socioeconomic outcomes in Sweden and the United States (Lazuka 2020; Bhalotra & Venkataramani 2015). Using more than 34 million U.S. death records and over 5 million linked Census–Numident observations, we provide the first national scale U.S. evidence on how this landmark medical innovation shaped adult longevity.

Our research design exploits predetermined cross state variation in baseline mortality from sulfa treatable diseases, interacted with age specific exposure at the time of sulfa diffusion. Across both administrative datasets, we find that individuals exposed between ages 0–4 in high burden states lived longer lives. A one standard deviation increase in pre sulfa disease burden raises adult lifespan by roughly 1.4 months, with highly consistent estimates across specifications, fixed effects structures, and robustness checks. Dynamic event study estimates show that these longevity gains are concentrated among children exposed at birth or shortly thereafter, consistent with biomedical evidence that infancy is a period of heightened vulnerability to infectious disease. The benefits are not evenly distributed. Children from low SES families and those born in rural counties—who faced higher infectious disease burdens prior to sulfa diffusion—experience the largest gains in longevity. These patterns align with historical evidence that access to effective medical therapies was unequal and that disadvantaged regions benefited most from reductions in early life infectious disease risk.

Our mechanism analyses reveal sustained improvements in adult health and socioeconomic outcomes. Early life exposure leads to small but statistically significant increases in adult schooling and income, but more importantly, to statistically significant reductions in cognitive, ambulatory, mobility, and self care disabilities. These improvements in functional health are consistent with the long run gains in longevity and point to persistent enhancements in adult health capital rather than survivor selection or latent frailty. Vital statistics confirm that the exposure measure captures large short-run improvements in infant survival, while birth rates remain unchanged.

To help contextualize the magnitude of these findings, it is useful to consider their place in the broader history of U.S. mortality improvements. Life expectancy conditional on survival to age 37 increased from 36.79 years for the 1921 cohort to 40.72 years for the 1941 cohort, a gain of 3.28 years (Social Security, 2024). The longevity gain we estimate—roughly 1.4 months per standard deviation increase in baseline mortality—constitutes about 3% of this total improvement.¹⁰

In our vital statistics sample, approximately 21.946 million births occurred post-sulfa. Given our estimate of 2.5 fewer infant deaths per 1,000 live births, this translates into 54,865 fewer infant deaths over the sample period. Given the average U.S. life expectancy for the 1940 cohort, 62.9 years when averaging men and women (Social Security, 2024), this number translates into 3.451 million life-years gained. Moreover, using the average share of exposed individuals in the final sample, ages 0–4 post-sulfa (roughly 0.366), the size of the final sample (34,473,143), and the average effect reported in the main results (1.4 months), we calculate approximately 1.473 million life-years gained due to later-life increases in longevity. Combining the contemporaneous effects on infant mortality and later-life longevity, we compute roughly 4.9 million life-years gained.¹¹

These population-level life-year benefits are larger than those generated by many other modern pharmaceutical innovations. For instance, modern oncology therapeutics extend survival by a median of roughly 2.4 months and reach only the small share of the population diagnosed with cancer in a given year (Ladanie et al., 2020; Siegel et al., 2026). Statins deliver larger average gains, but to a screened, high-risk adult population and late in life, with benefits concentrated in a small minority of users (Finegold et al., 2016). In randomized trials, statins postponed death by only 3.2 to 4.1 days over trial follow-up (Kristensen et al., 2015), while lifetime models imply larger gains of around 0.3 years of total life expectancy and 0.7 years of cardiovascular-disease-free life expectancy (Ferket et al., 2012). To translate these per-patient figures into comparable aggregates, even under the generous assumption that all of the roughly 2 million Americans diagnosed with cancer each year (Ladanie et al., 2020; Siegel et al., 2026) received a therapy delivering the median survival gain, a single annual cohort would accrue only about 0.4 million life-years. Scaling the realized trial-horizon estimates across the roughly 35 million U.S. adults

¹⁰ We use the 1921-1942 birth cohorts because they define the analytic sample. We condition on survival to age 37 because individuals born in 1942 must survive to approximately age 37 to enter the NCHS death-record observation window, which begins in 1979.

¹¹ We should note that the two channels are complementary. The contemporaneous channel reflects deaths averted, while the long-run channel reflects the lasting physiological benefit conferred on the far larger group of children who would have survived in any case. A complete welfare accounting of medical innovation requires both.

taking statins implies a similar total of 0.3–0.4 million life-years; lifetime simulation models project substantially larger gains, but these remain unrealized in trial data, accrue late in life, and are concentrated among a small minority of users.

A welfare interpretation further emphasizes the economic significance of these long-run effects. Using a Value of Statistical Life (VSL) of \$10 million for U.S.-born individuals (Kniesner & Viscusi, 2019) and the average longevity in our sample, we estimate the value of a life-year at approximately \$133,973. Using our calculated 4.9 million life-years gained, we calculate monetary benefits of approximately \$656.5 billion.

Our findings contribute to several strands of literature. First, they deepen the early life origins literature by showing that reductions in early life infectious disease exposure can generate persistent improvements in adult longevity (Almond, Currie & Duque 2018; Barker 1990). Second, they extend the medical innovation literature by documenting long run benefits of the first widely accessible antibacterial therapy. Third, the heterogeneity results contribute to ongoing work on the distributional consequences of medical technologies by showing that early life health innovations can narrow long run health inequalities when the largest gains accrue to disadvantaged children. Finally, our results underscore the importance of a life course perspective in evaluating medical innovations: the benefits of early life improvements in infectious disease environments accumulate across decades, shaping health and economic trajectories well into adulthood.

These findings speak directly to contemporary global health policy. Low- and middle-income countries continue to experience rapid declines in early-life infectious disease mortality due to antibiotics, vaccines, and maternal health interventions. Our results highlight that such technologies generate substantial long-run benefits, reinforcing — not weakening — the case for continued investment. To ensure that early-life survival gains translate into long-run improvements in adult health and longevity, they may need to be complemented by ongoing investments in nutrition, preventive care, and early childhood health environments. Notably, our heterogeneity results suggest that the long-run longevity gains from such interventions may be largest for children in the most disease-burdened environments — precisely the settings where these technologies are being most actively deployed today. Evaluations of medical innovations should therefore consider both immediate mortality reductions and their persistent life-course impacts.

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Tables

Table 1 - Summary statistics

	<i>Above-Median Exposure</i>		<i>Below-Median Exposure</i>	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
<i>NCHS Data:</i>				
Age at death (months)	885.8	136.0	903.3	135.3
Birth year	1930.2	6.2	1929.7	6.1
Death year	2004.0	10.9	2005.0	10.8
White	0.78	0.42	0.96	0.20
Black	0.22	0.41	0.04	0.19
Female	0.48	0.50	0.47	0.50
Maternal mortality rate 1930 – 1936	6.8	0.90	5.3	0.73
Scarlet fever death rate 1930 – 1936	1.6	0.90	2.4	0.91
Influenza death rate 1930 – 1936	1.3	0.11	0.96	0.10
Exposure age $\leq 4 \times$ baseline rate (STD)	0.43	0.61	-0.30	0.50
<i>County covariates:</i>				
Share literate	0.67	0.33	0.74	0.34
Socioeconomic index	23.7	3.0	30.1	2.5
Share married	0.62	0.02	0.60	0.02
Share of females in labor force	0.25	0.05	0.27	0.05
Occupational income score	19.5	2.0	23.6	1.5
Per capita midwife	2.9	3.0	1.8	2.1
Per capita nurse	2.3	0.81	3.8	1.1
Observations	14,966,073		19,507,070	
<i>Censoc-Numident Data:</i>				
Age at death (months)	851.4	81.0	854.3	80.3
Birth year	1927.4	5.5	1927.2	5.4
Death year	1998.3	4.8	1998.4	4.8
White	0.85	0.36	0.97	0.17
Black	0.15	0.36	0.03	0.16
Female	0.45	0.50	0.43	0.50
Father's SEI below median	0.47	0.50	0.38	0.48
Father's SEI above median	0.35	0.48	0.47	0.50
Father's SEI missing	0.18	0.39	0.15	0.36
Mother's education < high school	0.58	0.49	0.55	0.50
Mother's education high school	0.24	0.43	0.29	0.45
Mother's education college	0.05	0.21	0.06	0.23
Mother's education missing	0.13	0.34	0.11	0.31
Observations	2,483,390		2,703,580	
<i>Census-ACS Data:</i>				
Years of Schooling	11.3	3.5	12.4	3.1
Education < high school	0.13	0.34	0.06	0.24
Education < 12 years	0.28	0.45	0.16	0.37
Log wage income	10.3	1.1	10.4	1.2
Log total income	10.2	1.1	10.4	1.1
Log well for income	0.21	1.3	0.15	1.1
Cognitive disability	0.10	0.30	0.07	0.26
Ambulatory disability	0.25	0.43	0.20	0.40
Mobility disability	0.12	0.33	0.10	0.30
Self-care disability	0.08	0.27	0.06	0.24
Vision – hearing disability	0.14	0.35	0.12	0.33
Vision disability	0.07	0.26	0.05	0.22
Work disability	0.15	0.36	0.12	0.32
Being in mental institution	0.14	3.7	0.15	3.8
Observations	16,201,704		16,548,016	
<i>Vital Statistics Data:</i>				
Infant mortality rate (per 1,000 births)	60.7	21.2	47.8	15.8
Birth rate (per 1,000 women aged 15-45)	94.2	17.6	85.0	22.3
Observations	667		677	

Notes. The NCHS data covers the years 1979 – 2020. The Censoc-Numident data covers the years 1988 – 2005. The census – ACS data covers the years 1970 – 2020. The vital statistics data covers the years 1920 – 1950. The variable “Being in mental institution” is multiplied by 100 in this table.

Table 2 - Main Results

	<i>Outcome: Age at death (months)</i>	
	(1)	(2)
Exposure age $\leq 4 \times$ baseline rate (STD)	1.7*** (0.1)	1.4*** (0.1)
Observations	34,473,143	34,473,143
R-squared	0.15	0.15
Mean DV	895.7	895.7
Birth state FE	✓	✓
Birth year FE	✓	✓
Controls	✓	✓
Birth region by birth year FE		✓

Notes. Standard errors, clustered on state and year, are in parentheses. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Table 3 - Heterogeneity by race and gender

	<i>Outcome: Age at death (months), Subsamples:</i>			
	Whites	Blacks	Males	Females
	(1)	(2)	(3)	(4)
Exposure age $\leq 4 \times$	1.4***	1.5***	1.5***	1.4***
baseline rate (STD)	(0.1)	(0.2)	(0.1)	(0.1)
Observations	30,290,136	4,183,007	17,990,745	16,482,398
R-squared	0.14	0.14	0.11	0.16
Mean DV	901.3	855.3	876.6	916.5

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Table 4 - Family Background and Demographic Heterogeneity Using CenSoc-Numident

	<i>Outcome: Age at death (months), Subsamples:</i>						
	Full Sample	Urban born	Rural born	Father's SEI below median	Father's SEI above median	Mother's education < high school	Mother's education ≥ high school
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Exposure age ≤ 4 × baseline rate (STD)	0.36*** (0.08)	0.31** (0.13)	0.40*** (0.10)	0.26** (0.11)	0.47*** (0.13)	0.39*** (0.10)	0.35*** (0.12)
Observations	5,186,951	2,583,388	2,603,563	2,182,590	2,148,571	2,912,117	2,274,834
R-squared	0.51	0.50	0.51	0.51	0.50	0.48	0.53
Mean DV	852.9	855.1	850.7	845.2	850.6	852.9	852.8

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/family/state controls. Individual controls include dummies for race and gender. Family controls include dummies for maternal education and paternal socioeconomic index. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1920 – 1940 who died between the years 1988 – 2005.

*** p<0.01, ** p<0.05, * p<0.1

Table 5 - Sensitivity to Exposure Windows and Baseline Rates

	<i>Outcome: Age at death (months)</i>					
	(1)	(2)	(3)	(4)	(5)	(6)
Exposure age ≤ 0 $\times$ baseline rate (STD)	2.5*** (0.2)					
Exposure ages 1-4 $\times$ baseline rate (STD)	1.5*** (0.2)					
Exposure age 5-7 $\times$ baseline rate (STD)	0.57*** (0.14)					
Exposure age ≤ 4 $\times$ baseline rate above-median		3.9*** (0.3)				
Exposure age ≤ 4 $\times$ baseline rate Top-Two Tercile			4.7*** (0.3)			
Exposure age ≤ 4 $\times$ baseline maternal mortality + scarlet fever + influenza/pneumonia (STD)				1.5*** (0.2)		
Exposure age ≤ 4 $\times$ baseline maternal mortality rate (STD)					1.3*** (0.2)	
Exposure age ≤ 0 $\times$ baseline scarlet fever rate (STD)						0.49*** (0.15)
Observations	34,473,143	34,473,143	34,473,143	33,469,292	34,473,143	33,469,292
R-squared	0.15	0.15	0.15	0.15	0.15	0.15
Mean DV	895.7	895.7	895.7	896.2	895.7	896.2

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Table 6 - Robustness to Alternative Specifications and Estimators

	<i>Outcome: Age at death (months), Specification:</i>					Outcome: log age at death
	Main results	Adding death state FE	Adding more state controls	Interacting other disease rates with birth year FEs	Adding State Trend	
	(1)	(2)	(3)	(4)	(5)	(6)
Exposure age $\leq 4 \times$ baseline rate (STD)	1.4*** (0.1)	1.4*** (0.1)	1.2*** (0.1)	1.5*** (0.1)	0.36*** (0.13)	0.0015*** (0.0001)
Observations	34,473,143	34,468,120	34,473,143	33,469,292	34,473,143	34,473,143
R-squared	0.15	0.15	0.15	0.15	0.15	0.14
Mean DV	895.7	895.7	895.7	896.2	895.7	6.8
	Expanding birth cohorts to 1920 – 1950	Using birth cohorts of 1920-1929 and 1941-1950	Using birth cohorts of 1930-1940	Accelerated Failure Time (AFT) model	(Sun & Abraham, 2021)'s estimator	
	(7)	(8)	(9)	(10)	(11)	
Exposure age $\leq 4 \times$ baseline rate (STD)	1.8*** (0.1)	2.4*** (0.1)	1.0*** (0.2)	0.0015*** (0.0001)	1.5*** (0.1)	
Observations	43,662,138	29,089,927	14,572,211	34,473,143	34,473,143	
R-squared	0.28	0.35	0.07	---	0.15	
Mean DV	872.9	877.9	862.9	6.8	895.7	

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

Additional state controls of column 3 includes population, share of whites, share of Blacks, share of Hispanics, share of other races, share of females, share of homeowners, share of institutionalized population, share of population in different occupation/gender groups, share of married individuals, female labor force participation rate, school attendance rate, gender specific socioeconomic index, gender specific occupational income scores, and gender specific occupational prestige score.

The disease mortality rates used in column 4 include malaria, tuberculosis, and meningitis.

*** p<0.01, ** p<0.05, * p<0.1

Table 7 - Analysis of Cumulative Mortality Rates and Heterogeneity by Cause of Death

	<i>Outcome: Log Cumulative Mortality Rate, Subsample:</i>								
	Full sample	Whites	Nonwhites	Females	Males	Infectious Diseases	Chronic/Non-communicable Diseases	All other Diseases	External and Self-Inflicted Causes
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Exposure age ≤ 4 $\times$ baseline rate (STD)	-0.033*** (0.007)	-0.018** (0.007)	-0.048*** (0.011)	-0.027*** (0.008)	-0.039*** (0.009)	-0.025*** (0.007)	-0.018*** (0.006)	-0.014** (0.007)	-0.009 (0.007)
Observations	137,720	68,860	68,860	68,860	68,860	101,354	101,354	101,354	101,354
R-squared	0.53	0.50	0.49	0.53	0.53	0.36	0.38	0.36	0.32
Mean DV	4.4	5.6	3.3	4.3	4.6	3.3	5.0	3.7	2.5

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions are weighted using state level population in 1980. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1920 – 1950 who died between the years 1979 – 2020.

Infectious Diseases include: TB, Syphilis, Influenza, Pneumonia, other Infectious Diseases

Chronic/Non-communicable Diseases include: Neoplasm Diseases, Diabetes, Cardiovascular Diseases, Peptic Ulcer, Nephritis

*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 8 - Exploring Mechanism Channels Using Census, American Community Survey, Vital Statistics, and National Health Interview Survey Data

	<i>Outcomes:</i>					
	Years of schooling	Education < 8 years	Education < 12 years	Log wage income	Log total income	Log welfare income
	(1)	(2)	(3)	(4)	(5)	(6)
Exposure age $\leq 4 \times$ baseline rate (STD)	0.0476*** (0.0070)	-0.0155*** (0.0011)	-0.0116*** (0.0011)	0.0067*** (0.0015)	0.0039*** (0.0012)	-0.0118*** (0.0014)
Observations	11,991,790	33,577,216	33,577,216	16,872,920	29,157,256	30,669,623
R-squared	0.12	0.09	0.11	0.14	0.16	0.01
Mean DV	11.8	0.09	0.21	10.3	10.3	0.16
	Cognitive disability	Ambulatory disability	Mobility disability	Self-care disability	Vision – hearing disability	Vision disability
	(7)	(8)	(9)	(10)	(11)	(12)
Exposure age $\leq 4 \times$ baseline rate (STD)	-0.0028*** (0.0005)	-0.0016** (0.0007)	-0.0028*** (0.0006)	-0.0017*** (0.0004)	-0.0018*** (0.0006)	-0.0038*** (0.0007)
Observations	15,546,669	15,546,669	21,585,419	21,585,425	15,546,669	7,418,424
R-squared	0.02	0.05	0.04	0.02	0.04	0.03
Mean DV	0.09	0.22	0.11	0.07	0.13	0.06
	Work disability	Being in mental institution	Married	Infant mortality rate (per 1,000)	Infant mortality rate, Whites (per 1,000)	Infant mortality rate, Nonwhites (per 1,000)
	(13)	(14)	(15)	(16)	(17)	(18)
Exposure age $\leq 4 \times$ baseline rate (STD)	-0.0045*** (0.0004)	-0.0001* (0.0001)	0.0030*** (0.0005)	-2.5* (1.4)	-1.6** (0.8)	-4.8** (2.0)
Observations	19,377,144	6,626,289	33,577,216	882	812	802
R-squared	0.03	0.0003	0.05	0.73	0.74	0.71
Mean DV	0.13	0.0010	0.71	42.9	41.4	60.6
	Sex Ratio	Birth Per population (per 1,000)	Height	Having no insurance	Private insurance	Activity limitation
	(19)	(20)	(21)	(22)	(23)	(24)
Exposure age $\leq 4 \times$ baseline rate (STD)	0.0010 (0.0010)	-0.64 (0.59)	0.11** (0.04)	-0.01** (0.01)	0.02*** (0.01)	-0.01** (0.01)
Observations	1,344	1,344	27,163	14,769	14,737	27,163
R-squared	0.16	0.99	0.53	0.03	0.05	0.02
Mean DV	1.1	75.3	67.0	0.10	0.86	0.13

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions of columns 1-14 and 17-20 include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data for columns 1-14 covers birth cohorts of 1920 – 1950. The sample is restricted to individuals at least 25 years old observed in censuses 1970 – 2000 and ACS data 2001 – 2020. The data for columns 15-16 covers birth and infant death data of the years 1920 – 1950. Regressions of columns 15 – 16 is similar to columns 1 – 14 except that they do not include individual covariates. Columns of 21 – 24 use the National Health Interview Survey (NHIS) data for the years 1974 – 1984.

The years-of-schooling variable in column 1 is based on the continuous measure of educational attainment reported in the 1970–1980 Censuses. The educational dummy variables in columns 2–3 are constructed using the harmonized education variable provided by IPUMS, which is consistently defined across the 1970–2000 Censuses and the American Community Survey (ACS) for the period 2001–2020.

*** p<0.01, ** p<0.05, * p<0.1

Figures

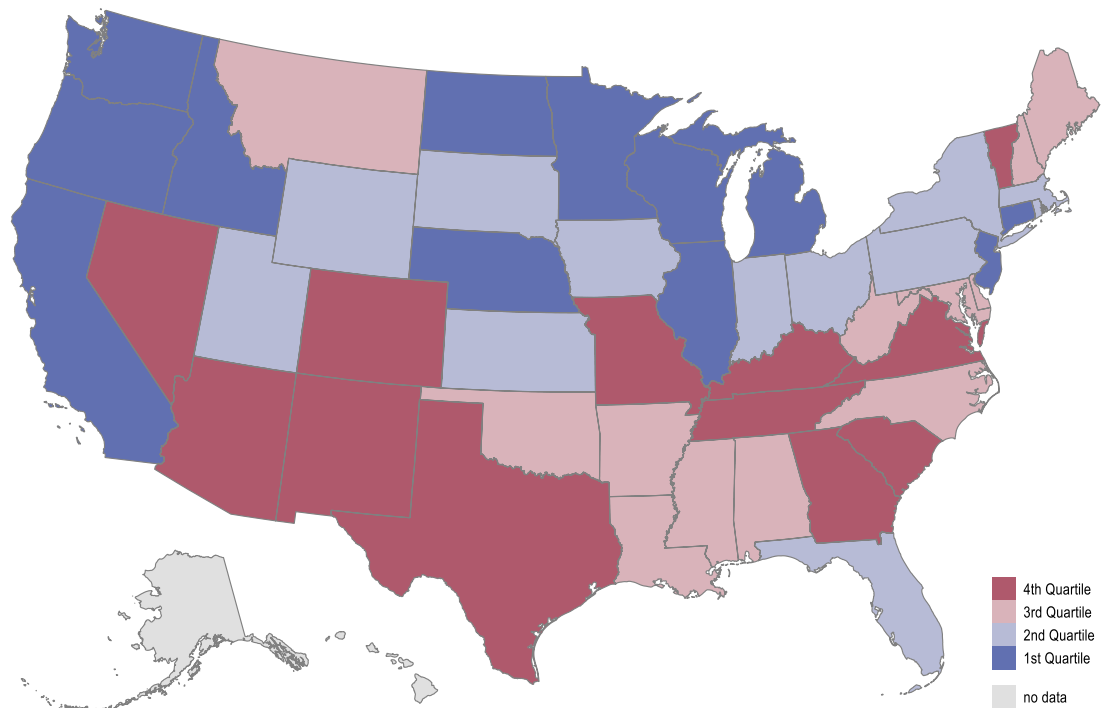


Figure 1 - Spatial Variation in Baseline Exposure Across States

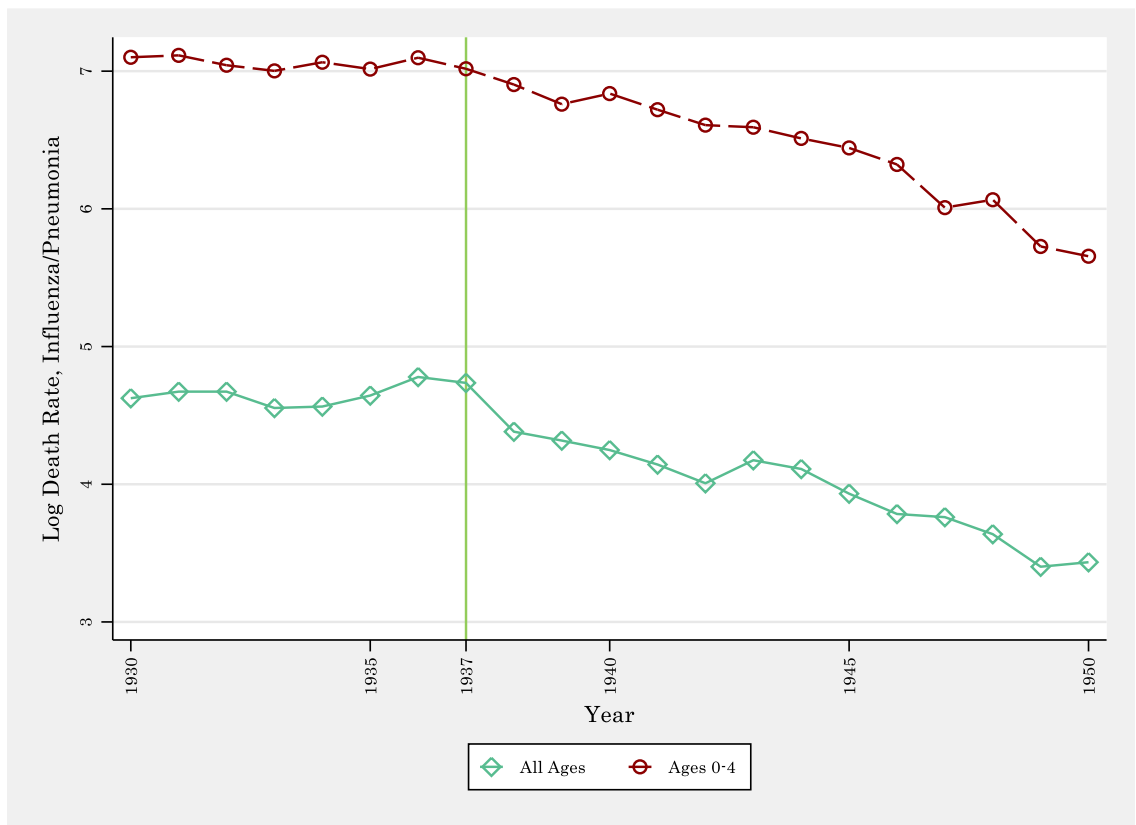


Figure 2 - Timeseries Evolution of Death Rate by Age 1930-1950

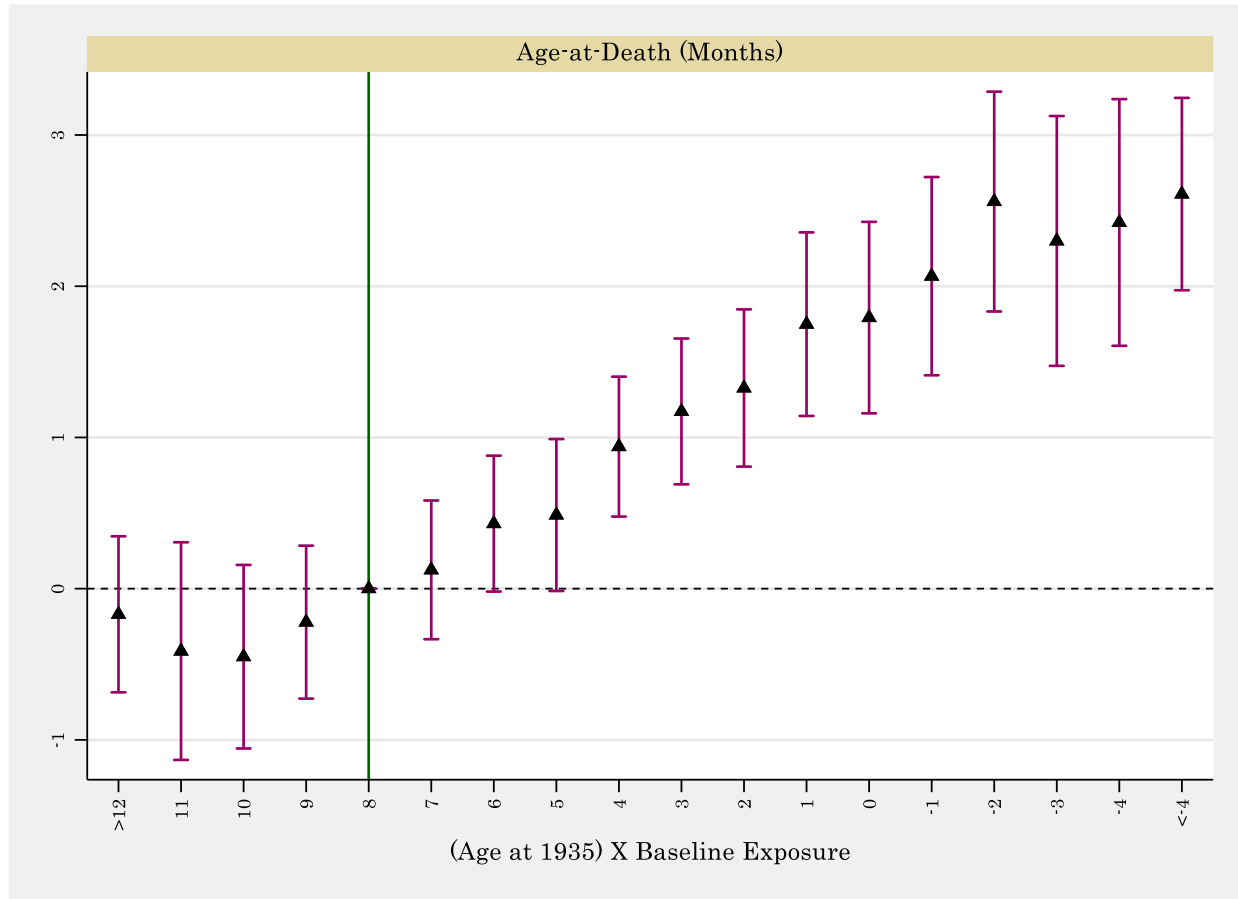


Figure 3 - Dynamic Effects of Sulfa Drug Introduction Across Exposure Ages Using NCHS Data

Notes. The point estimates and 95% confidence intervals are illustrated. Standard errors are clustered by birth state and birth year. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

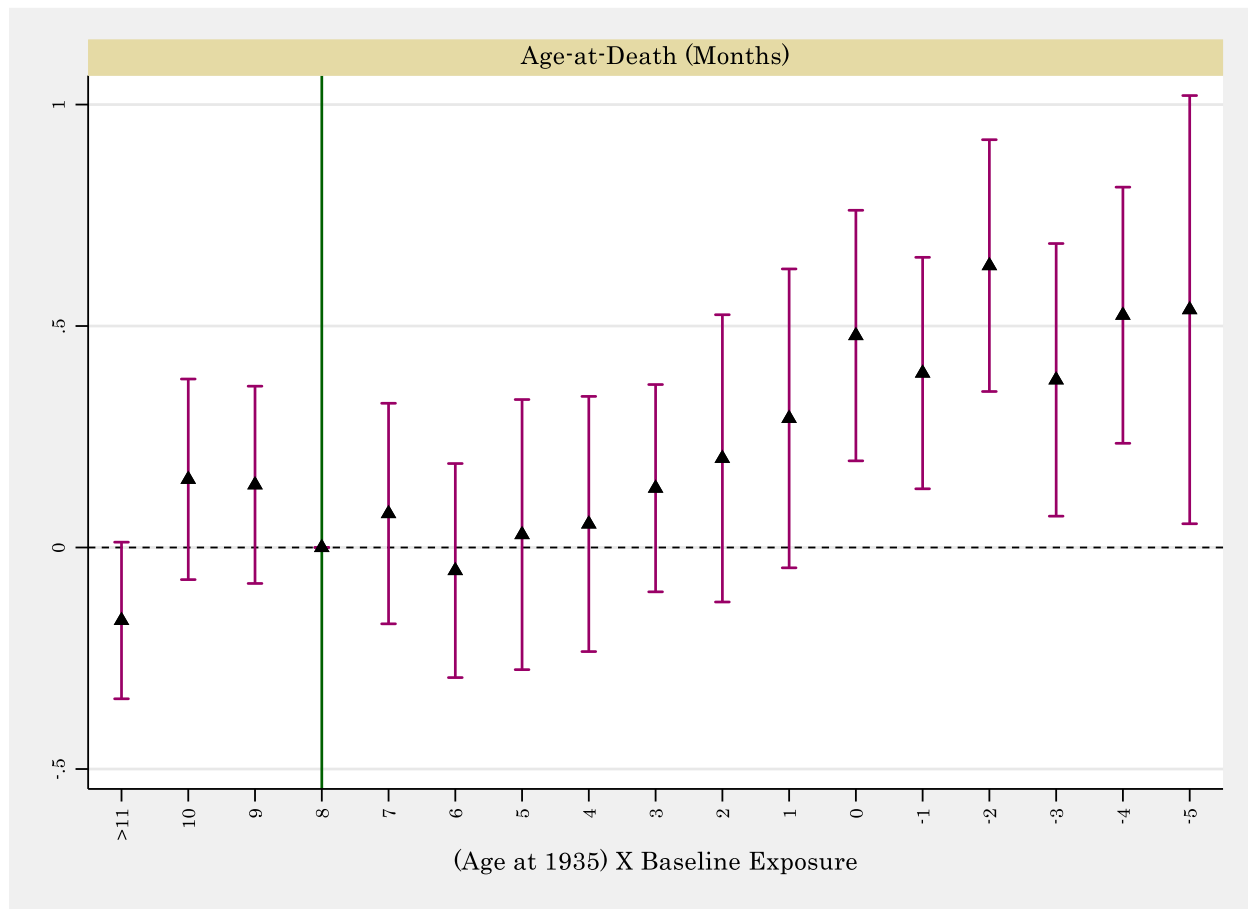


Figure 4 - Dynamic Effects of Sulfa Drug Introduction Across Exposure Ages Using Censoc-Numident Data

Notes. The point estimates and 95% confidence intervals are illustrated. Standard errors are clustered by birth state and birth year. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/family/state controls. Individual controls include dummies for race and gender. Family controls include dummies for maternal education and paternal socioeconomic index. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1920 – 1940 who died between the years 1988 – 2005.

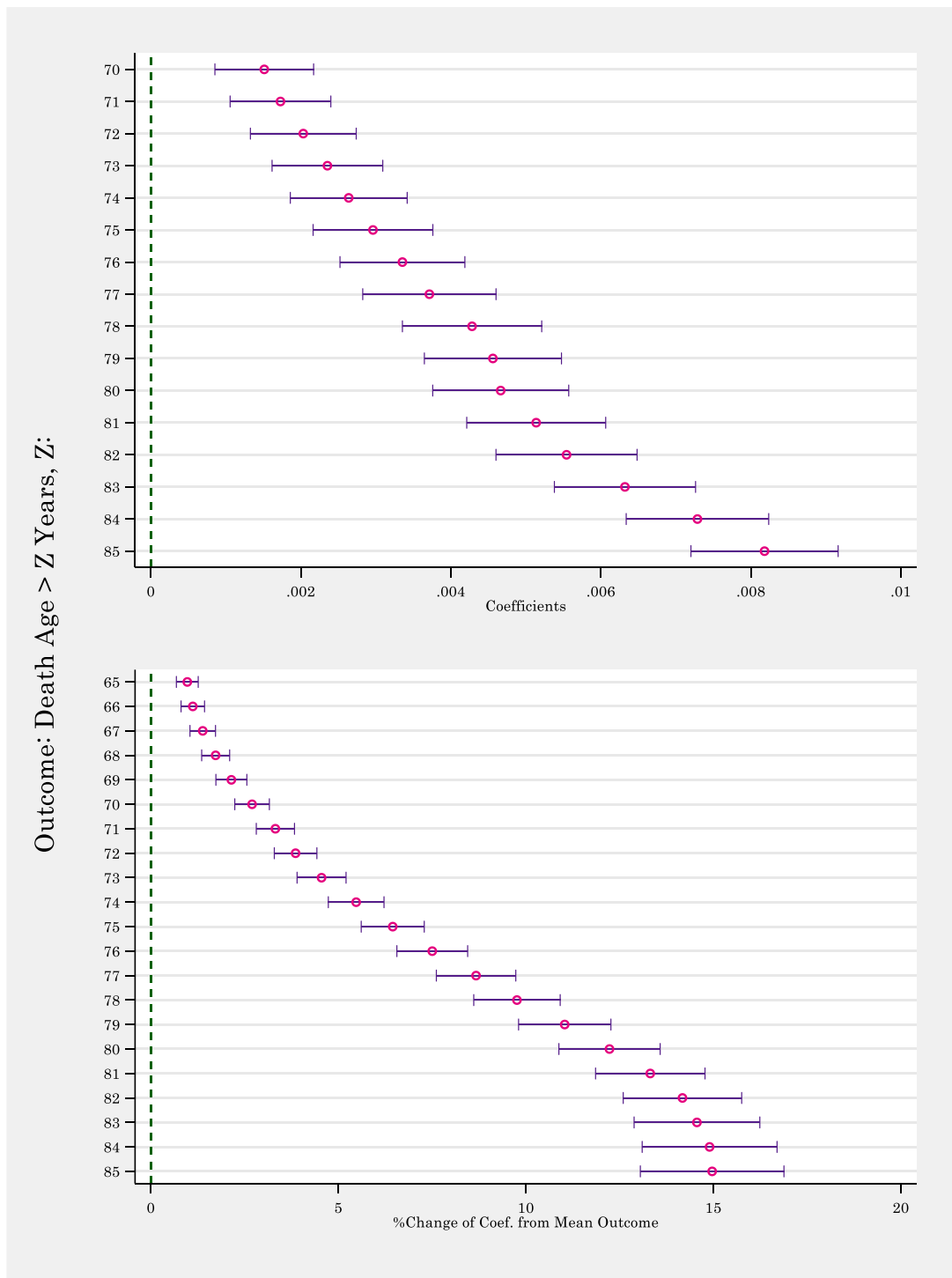


Figure 5 - Sulfa Drug Introduction and Longevity: Effects at Increasing Survival Thresholds

Notes. The point estimates and 95% confidence intervals are illustrated. Standard errors are clustered by birth state and birth year. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

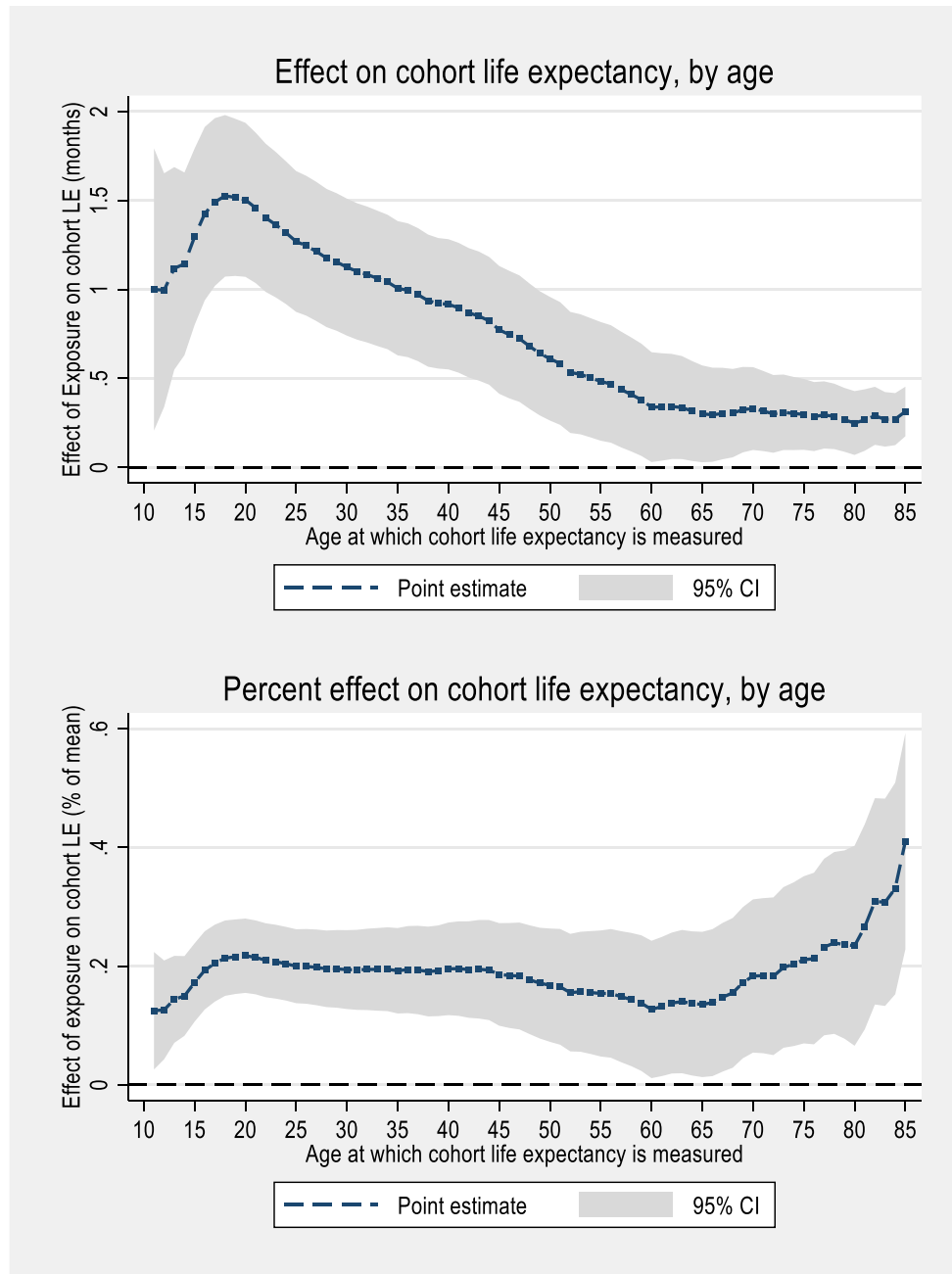


Figure 6 - Early-Life Sulfa Exposure and Cohort Life Expectancy Across the Age Distribution

Notes. The point estimates and 95% confidence intervals are illustrated. Standard errors are clustered by birth state and birth year. Regressions include birth state fixed effects, birth region by birth year fixed effects. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1920 – 1950.

Appendix A

Appendix Table A-1 - Placebo Tests

	<i>Outcome: Age at death (months)</i>	
	(1)	(2)
Exposure age $\leq 4 \times$ baseline	0.13	
Tuberculosis (STD)	(0.13)	
Exposure age $\leq 4 \times$ baseline Malaria (STD)		0.09 (0.12)
Observations	34,473,143	34,473,143
R-squared	0.15	0.15
Mean DV	895.7	895.7

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-2 - Placebo Tests: Age at Exposure of 16-18 Vs 19-21

	<i>Outcome: Age at death (months)</i>		
	Full sample	Males	Females
	(1)	(2)	(3)
Exposure age 16-18 × baseline	-0.20	-0.10	-0.30
Rate (STD)	(0.14)	(0.16)	(0.19)
Observations	10,865,658	5,803,498	5,062,160
R-squared	0.06	0.01	0.01
Mean DV	965.9	989.3	939.0

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-3 - Placebo Tests: Shuffling Treatment across States and Years

	<i>Outcome: Age at death (months), Round Number:</i>				
	(1)	(2)	(3)	(4)	(5)
Exposure age $\leq 4 \times$ baseline	-0.02	0.02	-0.07**	-0.03	-0.03
Rate (STD)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Observations	34,473,143	34,473,143	34,473,143	34,473,143	34,473,143
R-squared	0.15	0.15	0.15	0.15	0.15
Mean DV	895.7	895.7	895.7	895.7	895.7
	(6)	(7)	(8)	(9)	(10)
Exposure age $\leq 4 \times$ baseline	0.01	0.04	0.02	0.04	0.02
Rate (STD)	(0.03)	(0.04)	(0.03)	(0.03)	(0.03)
Observations	34,473,143	34,473,143	34,473,143	34,473,143	34,473,143
R-squared	0.15	0.15	0.15	0.15	0.15
Mean DV	895.7	895.7	895.7	895.7	895.7

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-4 - Balancing Tests: Endogenous Changes in the Sample's Demographic Composition

	<i>Outcomes:</i>	
	White	Female
	(1)	(2)
Exposure age $\leq 4 \times$ baseline rate (STD)	-0.0002 (0.0005)	-0.0005** (0.0002)
Observations	34,473,143	34,473,143
R-squared	0.18	0.0036
Mean DV	0.88	0.48

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects and birth region by birth year fixed effects. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-5 - Examining Robustness to Functional Form of Independent Variable

	<i>Outcome: Age at death (months)</i>		
	(1)	(2)	(3)
Exposure age $\leq 4 \times$ baseline	2.8***		
Rate Above-Median	(0.3)		
Exposure age $\leq 4 \times$ baseline		3.6***	
Rate 2 nd Tercile		(0.2)	
Exposure age $\leq 4 \times$ baseline		4.3***	
Rate 3 rd Tercile		(0.3)	
Exposure age $\leq 4 \times$ baseline			3.4***
Rate 2 nd Quartile			(0.2)
Exposure age $\leq 4 \times$ baseline			4.4***
Rate 3 rd Quartile			(0.4)
Exposure age $\leq 4 \times$ baseline			4.5***
Rate 4 th Quartile			(0.3)
Observations	34,473,143	34,473,143	34,473,143
R-squared	0.15	0.15	0.15
Mean DV	895.7	895.7	895.7

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-6 - The Effects across Various Ages at Exposure to the Introduction of Sulfa Drug

	<i>Outcome: Age at death (months)</i>	
	(1)	(2)
Exposure age $\leq 0 \times$ baseline rate (STD)	2.3*** (0.3)	1.9*** (0.3)
Exposure age 1-4 $\times$ baseline rate (STD)	1.3*** (0.3)	1.1*** (0.3)
Exposure age 5-6 $\times$ baseline rate (STD)	0.57*** (0.17)	0.41** (0.19)
Exposure age 7-10 $\times$ baseline rate (STD)	-0.06 (0.22)	0.08 (0.25)
Exposure age 11-15 $\times$ baseline rate (STD)	-0.14 (0.28)	0.03 (0.29)
Observations	34,473,143	34,473,143
R-squared	0.15	0.15
Mean DV	895.7	895.7
Birth state FE	✓	✓
Birth year FE	✓	✓
Controls	✓	✓
Birth region by birth year FE		✓

Notes. Standard errors, clustered on state and year, are in parentheses. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-7 - Comparing the Results of NCHS Data across Various Death Years

	<i>Outcome: Age at death (months), Subsamples:</i>			
	Death Years 1988-2005	Death Years 1979-2005	Death Years 1988-2020	Death Years 1979-2020
	(1)	(2)	(3)	(4)
Exposure age $\leq 4 \times$	0.36***	0.46***	1.2***	1.4***
baseline rate (STD)	(0.05)	(0.08)	(0.1)	(0.1)
Observations	13,383,022	16,477,047	31,379,118	34,473,143
R-squared	0.57	0.37	0.22	0.15
Mean DV	825.4	794.9	918.6	895.7

Notes. Standard errors, clustered on state and year, are in parentheses. Regressions include birth state fixed effects, birth region by birth year fixed effects, and individual/state controls. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1921 – 1942 who died between the years 1979 – 2020.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-8 - Examining Signal-to-Noise in the NHIS Data

	<i>Outcome: Exposure based on Birth-State (STD)</i>	
	(1)	(2)
Exposure based on Current-State (STD)	0.58*** (0.02)	0.50*** (0.02)
Observations	2,159,073	2,159,073
R-squared	0.36	0.59
Mean DV	-0.000	-0.000
Current state FE	✓	✓
Birth year FE	✓	✓
Controls		✓

Notes. Standard errors, clustered on state, are in parentheses. Individual controls include dummies for race and gender. Birth state controls include share literate, share married, average socioeconomic index, average occupational income score, female labor force participation rate, per capita midwife, and per capita nurse. The data covers birth cohorts of 1920 – 1950 observed in the 1980 census.

“STD” denotes that the exposure variables are standardized.

*** p<0.01, ** p<0.05, * p<0.1