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ABSTRACT

This paper examines the long-term effects of early-life exposure to county health departments (CHDs), a public health initiative aimed at providing affordable healthcare access to disadvantaged and underserved areas during the early decades of the 20th century, on later-life longevity. We employ Social Security Administration death records linked with the 1940 census and implement a difference-in-differences estimation strategy to compare the longevity of individuals exposed at different ages to the county-specific year of CHD establishment. We find that exposure to a CHD opening in the county of birth during fetal development and early years of life is associated with a 1-4-month increase in longevity. We observe considerable heterogeneity in the results, such that the effects are more concentrated among non-white individuals and individuals with lower-educated parents. Contemporaneous fertility and infant mortality data suggest reductions in infant mortality rates and increases in birth rates following CHD openings. Additionally, we find significant improvements in educational attainment, socioeconomic status, and income in early adulthood. We argue that enhancements in the fetal environment, improved fetal survival, and better early life health capital resulting from CHD exposure contribute to improvements in human capital during adulthood, with potential implications for later-life longevity.

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1. Introduction

The early 20th century witnessed a transformative period in the expansion of healthcare in the United States. Significant strides were made to address inequalities in healthcare access and health disparities across different sociodemographic groups, particularly in rural and underserved areas (Byrd & Clayton, 2001; Gordon, 2009). Federal initiatives like the Sheppard-Towner Act of 1921 provided essential funding for maternal and infant care services, significantly improving health outcomes in economically disadvantaged regions (Moehling & Thomasson, 2014). Public health campaigns, such as those led by the U.S. Public Health Service and the Rockefeller Sanitary Commission, targeted endemic diseases like hookworm, typhoid, pellagra, and malaria, contributing to overall improvements in population health, especially in poor regions (Beach et al., 2016; Bleakley, 2007, 2010). Notably, the establishment of County Health Departments (CHDs) brought affordable healthcare, improved sanitation, and implemented disease prevention programs in rural and low-income areas (Hoehn-Velasco, 2018, 2021). While several strands of research document the short- and medium-term impacts of these programs on the health and human capital of affected populations, understanding their long-term effects remains policy-relevant.

Several strands of research contribute to these policy discussions and provide evidence that affordable healthcare access plays an important role in shaping short-run health outcomes, with spillover effects that impact long-term health outcomes (Barbaresco et al., 2015; Black et al., 2017; Lu & Slusky, 2016). Specifically, affordable healthcare access during critical early life stages shapes infants' and children's health capital with potential implications for their long-run later-life outcomes. For instance, reducing healthcare costs through expansions in public programs is associated with improvements in infants' and children's health outcomes as well as their later-life outcomes, including education, income, disability, hospitalization, and old-age health and mortality (Boudreaux et al., 2016; Goodman-Bacon, 2021; Wherry & Meyer, 2016). Additionally, studies suggest increasing access to affordable healthcare clinics benefits maternal and infant health outcomes (Ellison et al., 2021; Hoehn-Velasco, 2018; Slusky, 2017). However, fewer studies have examined the role of availability and accessibility of affordable healthcare clinics in local areas during critical developmental ages on later-life old-age longevity. The current study fills this gap in the literature by examining the large-scale establishment of County Health Departments (CHDs) in disadvantaged counties in the United States during the early 20th century.

CHDs in the early 20th century were set up mainly in rural and low-income areas to improve public health. They offered affordable healthcare (particularly related to maternal and infant health services), sanitation improvements, and programs to prevent the spread of diseases. We employ Social Security Administration death records linked with the full count 1940 census, use the staggered establishment of CHDs, and examine the effects on later-life longevity of individuals exposed to these organizations across different ages. We observe significant improvements in longevity for in-utero and early-life exposure to CHDs. We do not observe meaningful impacts for later childhood ages, adolescence, and early adulthood ages. Exposure during the in-utero and the first four years of life is associated with a 0.9-month of higher longevity later in life. The effects reveal considerable heterogeneity. Consistent with the purpose of the CHDs, we observe larger effects among non-White individuals and individuals with low-educated mothers.

We implement a series of balancing tests to examine the association between exposure to CHDs and changes in sociodemographic and socioeconomic characteristics. We observe correlations that are indistinguishable from zero, both statistically and economically. Furthermore, event studies using county characteristics in the full count censuses 1900 – 1940 reveal no significant pattern of changes in sociodemographic and socioeconomic features of counties in the years before and after a new CHD opening. Additionally, we implement a wide array of sensitivity analyses to show the robustness of the results to alternative specifications, sample selections, estimation models, and functional forms.

Contemporaneous natality and mortality data for the years 1915 – 1940 suggest significant reductions in infant mortality rates and increases in birth rates following the establishment of a CHD in the county. Using the full count of the 1900–1940 censuses, we observe significant increases in the number of Black children following the establishment of CHDs. We also find increases in the share of male births, consistent with improvements in the fetal health environment, as fetal deaths are more prevalent among male infants (Sanders & Stoecker, 2015). These changes in the composition of births may introduce a downward bias in our estimates due to the differential longevity between nonwhites and whites. Therefore, our estimates likely underestimate the true effects.

Using the information in the 1940 census, we observe nontrivial and significant improvements in educational outcomes and measures of occupational standing and socioeconomic

status. Improvements in infants' health capital (suggested by reductions in infant mortality rates) might change the trajectory of infants' developmental outcomes, as reflected in their education and socioeconomic status in early adulthood. Based on the literature on the education-health and income-health gradient (Fletcher, 2015; Lleras-Muney, 2005; Lleras-Muney et al., 2022), these improvements might later translate into improved health outcomes and be reflected in increases in old-age longevity.

The current study makes three contributions to the literature. First, this study joins the narrow strand of research that examines the long-run effects of County Health Departments (CHDs) in the early 20th century US (Hoehn-Velasco, 2018, 2021). The most directly comparable study is Hoehn-Velasco (2021), who links the 1930 Census to the Death Master File and documents that access to CHDs at ages 0–5 is associated with an approximately 2.3-month increase in lifespan among men. While her primary focus is on income and labor-market outcomes, our analysis centers on longevity as the key outcome of interest. Our paper differs from Hoehn-Velasco (2021) along four main dimensions: data, sample, scope, and methodology. With respect to data, we use the CenSoc-Numident file, which links Social Security Administration death records for deaths occurring between 1988 and 2005 to the complete-count 1940 Census, whereas Hoehn-Velasco (2021) uses the Death Master File linked to the 1930 Census, with death years 1965–2011. With respect to sample, our analytical sample contains approximately 2.1 million individuals and includes both males and females, whereas Hoehn-Velasco (2021) is restricted to men because linking women from the 1930 Census to the Death Master File is substantially more difficult due to surname changes after marriage. With respect to scope, Hoehn-Velasco (2021) reports average lifespan effects, while our analysis examines distributional and heterogeneous effects across race, gender, maternal education, paternal socioeconomic status, CHD per-capita budget spending, and urban-versus-rural birthplace. These heterogeneity analyses allow us to evaluate whether the longevity gains were concentrated among populations more likely to benefit from local public-health infrastructure. With respect to methodology, we provide a systematic comparison across mortality data sources, using both Numident and Death Master File records, and evaluate the implications of differential death-window coverage and truncation bias. The smaller baseline longevity coefficient in our Numident analysis, 0.9 months compared with 2.3 months in Hoehn-

Velasco (2021), is consistent with differences in sample composition and mortality-window coverage.[4]

Second, our findings contribute to the literature on the long-run consequences of local healthcare clinic establishment. Studies in this area examine the establishment of maternal and child health clinics, family planning clinics, and other facility-based delivery of healthcare services in historical and contemporary settings (Bailey et al., 2012; Bhalotra et al., 2017; Bütikofer et al., 2019; Ellison et al., 2021; Hoehn-Velasco, 2018, 2021; Lu & Slusky, 2016; Marein, 2023; Slusky, 2017).[5] Our paper extends this literature by documenting that a large-scale historical episode of local clinic establishment had measurable and persistent effects on later-life longevity.

Third, our paper contributes to the narrower set of studies that examine the long-run effects of early-life access to healthcare facilities on later-life mortality outcomes (Bhalotra et al., 2017; Bütikofer et al., 2019; Hoehn-Velasco, 2021). Relative to this work, we focus on a historical episode of facility-based clinic establishment in the early 20th-century United States and document persistent effects on old-age longevity.

The rest of the paper is organized as follows. Section 2 provides a background on CHDs and the literature. Section 3 details data sources, sample selection, and econometric method. Section 4 reviews the results. Section 5 discusses mechanism channels. Finally, section 6 concludes the paper.

2. Background

This section provides the background for our analysis. Section 2.1 consolidates the institutional details of CHDs—their establishment, funding sources, services provided, and geographic spread—that we draw on throughout the paper. Section 2.2 reviews the literature on healthcare access, early-life exposures, and later-life outcomes that motivates our empirical

[4] When we restrict the DMF-based analysis to the death-year window and birth cohorts used in Hoehn-Velasco (2021)—that is, deaths through 2011 and birth cohorts 1910–1930—we obtain an identical point estimate to hers. This exercise suggests that differences between our baseline estimate and Hoehn-Velasco's estimate are explained by differences in mortality-window coverage, cohort composition, and linked data sources rather than by differences in the underlying CHD exposure measure or empirical specification.

[5] A broad literature examines the long-run consequences of early-life exposures, documenting that shocks, policies, and environmental conditions experienced during fetal development, infancy, childhood, or adolescence can shape later-life health, human capital, socioeconomic attainment, disability, and mortality (Aizer et al., 2016; J. Fletcher & Noghanibehambari, 2024, 2025; Hayward & Gorman, 2004; Van Den Berg et al., 2006).

analysis. Mechanism-specific literature is presented within the corresponding subsections of Section 5, where each channel is examined empirically.

2.1. Background of County Health Departments

In the early 20th century, the United States experienced a surge in the establishment of County Health Departments (CHDs), driven by the pioneering efforts of the U.S. Public Health Service (USPHS) and the Rockefeller Sanitary Commission (RSC). The first CHD was founded in Jefferson County, Kentucky (in 1892), and a successful pilot project in Yakima County, Washington (in 1911), showcased the effectiveness of improved water sanitation in reducing typhoid mortality (Casner, 2001; Lumsden, 1918). Building on this success, CHDs expanded across the South, addressing typhoid, pellagra, and malaria through better sanitation, nutrition, and public health measures. The RSC also launched a major campaign to combat hookworm, partnering with state health departments and transitioning to support county-level CHDs focused on child health (Bleakley, 2007). Federal initiatives, including the Sheppard-Towner Act of 1921, provided funding for maternal and infant care, while events like the 1927 Mississippi flood brought additional aid from sources such as the Red Cross (Lemons, 1969; Spencer, 1994). By the 1930s, CHDs were present in approximately 600 counties across 38 states, supported by external funding from state governments, the USPHS, and other sources (Hoehn-Velasco, 2018). This widespread network significantly reduced preventable diseases and improved public health outcomes (Lumsden, 1918, 1927).

CHDs were initially funded through local county appropriations. However, external funding sources quickly became essential for expanding these services. Major contributors included the USPHS and the Rockefeller Foundation, which provided grants to counties across the country. This support allowed counties to sustain public health programs and extended services to rural areas where private medical care was often inaccessible (Ferrell, 1936; Hoehn-Velasco, 2018). Additional funding later came through federal initiatives, such as the Sheppard-Towner Act, which focused on maternal and child health programs, helping counties establish prenatal and infant care services in underserved regions (Hoehn-Velasco, 2018).

For residents, services at these health departments were generally accessible at low or no cost, especially as many of these departments aimed to improve public health in economically disadvantaged areas. The county health movement prioritized reaching poorer, rural counties that

lacked access to healthcare facilities, rather than wealthier urban or suburban areas that already had health infrastructure. This focus on rural and lower-income areas was evident in the concentration of health departments in regions with limited access to physicians and higher rates of preventable diseases. By 1933, CHDs were concentrated in the South and the rural Midwest—regions documented to have physician-to-population ratios well below the national average and higher mortality from preventable diseases such as typhoid, hookworm, and malaria (Ferrell, 1936; Hoehn-Velasco, 2018; Lumsden, 1918, 1927).

In addition to direct medical services, CHDs offered a form of financial protection to the household as a whole rather than to any single individual. Many residents in the rural and lower-income areas that CHDs served lacked the resources to absorb the costs of acute or chronic medical needs through private care, and by delivering services at low or no cost, CHDs reduced the financial burden of healthcare for the broader family. CHDs also coordinated preventive services, vaccinations, sanitary inspections, and health education campaigns, all of which were typically delivered free of charge or at subsidized rates and were available to all household members rather than only to a designated beneficiary (Ferrell, 1936; Hoehn-Velasco, 2018).

Beyond direct healthcare provision, CHDs also engaged in a broader set of environmental and regulatory public-health activities. The early decades of the 20th century coincided with a growing recognition of lead poisoning as a public-health concern, motivated by pioneering investigations of industrial lead exposure (Hamilton, 1925) and accumulating evidence of childhood plumbism (i.e. lead poisoning) (Rabin, 1989). Although comprehensive federal restrictions on lead in paint, gasoline, and plumbing emerged much later in the 20th century, several strands of historical research document that state and municipal health authorities, often operating through county-level health departments, conducted home and workplace inspections, reported and investigated cases of lead poisoning, and enforced sanitary codes covering water supplies, housing conditions, and occupational exposures (Fee, 1990; Markowitz & Rosner, 2014; Warren, 2000). The epidemiological evidence assembled by these local departments contributed to early state-level lead reporting requirements (e.g., in Maryland, Massachusetts, and Pennsylvania) and informed the federal lead policies that emerged after our study period (Berney, 1993; Rosner & Markowitz, 1985).

2.2. Literature Background

Several strands of research provide theoretical and empirical evidence that healthcare access, especially when affordable and readily available, improves infants' and children's health outcomes with potential implications for their developmental outcomes and long-term health outcomes, including old age mortality. This section reviews the pathways and related literature background.

Principal economic theories predict that reductions in the costs of healthcare access increase healthcare demand and utilization. Empirical evidence documents that improvements in healthcare access, either by increasing the supply or reducing the cost through public programs, increase healthcare utilization, and specifically prenatal healthcare utilization (Currie & Gruber, 1996; Ellison et al., 2021; Slusky, 2017; Yu et al., 2017). Changes in maternal healthcare utilization induced by opening new CHDs may influence the trajectory of infants' long-term outcomes by affecting health endowment at birth. Prenatal healthcare utilization results in early detection of complications, nutritional guidance, management of maternal chronic conditions during pregnancy, mental health support, and general health education, all of which have important implications for pregnancy outcomes and infants' developmental outcomes later in life (Corman et al., 2019; Fletcher, 2011). For instance, in a related paper to the current study, Hoehn-Velasco (2018) examines the effects of the expansions of CHDs in the US South during the early decades of the 20th century on infant mortality rates and documents reductions in mortality rates following the establishment of new CHDs. Therefore, the establishment of CHDs can impact infants' health outcomes through various channels attributed to the relevance of prenatal healthcare utilization and improvements in infants' health capital. Hoehn-Velasco (2021) examines the effects of expansions in CHDs in the US between 1908 – 1933 on children's later life labor market outcomes and document increases in men's earnings of about 2% – 5%. Moehling & Thomasson (2014) show that public health interventions under the Sheppard Towner Act implemented in the US between 1924 – 1929 was associated with nontrivial reductions in infant mortality rates. Anderson et al. (2020) employ historical data from the US between the years 1900 – 1940 and examine the effects of Midwifery Occupational Licensing policies on infant and maternal mortality rates. They document that improvements in prenatal and postnatal care quality as a result of licensure requirements resulted in reductions in maternal and infant mortality rates. Bleakley (2007) and Bleakley (2010) examine the effects of public health interventions under the Rockefeller Sanitary

Commission's funding and US Public Health Service support to eradicate hookworm and malaria in the early 20th century by establishing vaccination campaigns, sanitation facilities, and public health education programs. They document significant improvements in human capital for children who benefited from these campaigns.[6]

The evidence in historical settings of outside of the continental US also provides a similar story. Saaritsa et al. (2024) employ historical data from rural Finland during late 19th century and early 20th century and document that accessibility of health professionals in the municipality of residence is associated with reductions in mortality rates. Marein (2023) examines the effects of expansions in County Health Department in Puerto Rico over the years 1930 – 1960 on mortality rates. He documents significant reductions in infant mortality, maternal mortality, and mortality due to infectious diseases. Bütikofer et al. (2019) examine lifecycle consequences of early life exposure to expansions in maternal and child health care centers during the early decades of the 20th century in Norway. They document significant increases in educational outcomes, height, and labor market earnings during adulthood. Bhalotra et al. (2017) examine the effects of early life exposure to an infant health intervention program in Sweden which started in the year 1931 and infants' health and later life longevity. The program provided information and support for nutrition, sanitation, and monitoring infant care. They document that the program resulted in significant reductions in infant death and improvements in later life old age longevity.

Costly healthcare can result in excessive financial burden among low-income families who face medical difficulties or have a member with pre-existing chronic conditions (Parikh et al., 2014; Saunders & Alexander, 2009). For many mothers, losing access and the expected financial burden itself affect their mental health outcomes with potential implications for pregnancy outcomes (Carney, 2021; Olafsson, 2016). In other words, mothers may substitute other health-related inputs for prenatal healthcare access. For instance, in the face of rising costs associated with healthcare needed by other family members, they might allocate resources that otherwise would have been spent on nutrition, better residential location, and better-quality supplements. Nutrition, neighborhood quality, and the general health environment during pregnancy and early life can have lasting impacts on infants' and children's later-life outcomes (Mocan et al., 2015;

[6] Studies in contemporary U.S. settings also suggest that improved healthcare access leads to increased healthcare utilization and better health outcomes (Ellison et al., 2021; Lu & Slusky, 2016, 2019; Slusky, 2017).

Neelsen & Stratmann, 2011). The opening of CHDs might reduce those financial burdens and operate as welfare transfers with a higher concentration of benefits among infants and children.

Life course theories, specifically the Fetal Origin Hypothesis and the Developmental Origins of Health and Disease suggest that in utero, early life, and early childhood conditions exert detectable impacts on later-life health, human capital, and other developmental outcomes (Almond et al., 2018; Almond & Currie, 2011; Barker, 1990). There is a recently growing body of literature that empirically investigates the influence of exposures experienced during the in-utero period and the first year of life on later life socioemotional development, cognitive development, test scores, anthropometric outcomes, disability outcomes, family formation, educational outcomes, income, hospitalization, and old age health and mortality outcomes (Case & Paxson, 2009; Cunha & Heckman, 2007; Currie & Rossin-Slater, 2015; Fletcher & Noghanibehambari, 2024, 2025). For instance, Case et al. (2005) document an association between health status during childhood and educational attainment and health outcomes during adulthood. Gu et al. (2009) show that access to healthcare during childhood increases longevity and survival outcomes in old age. Noghanibehambari & Fletcher (2023) investigate the long-term impacts of midwifery licensing laws, which enhanced midwives' knowledge, education, and quality of care, with potential improvements in prenatal and postnatal health care during the early decades of the 20th century in the US. Their findings indicate that early exposure to these policies is linked to lower cumulative mortality rates and increased longevity in later life.

Reductions in early-life exposure to infectious diseases have been shown to improve long-run outcomes including education, disability, and mortality (Almond, 2006; Beach et al., 2016; Cook et al., 2019; J. M. Fletcher, 2018b, 2018a). For instance, Almond (2006) suggests that in-utero exposure to the 1918 influenza pandemic is associated with reductions in education and increases in disability later in life. Beach et al. (2016) and Meyers & Thomasson (2021) provide evidence that exposure to typhoid and polio diseases during childhood years is associated with reductions in educational attainments later in life, respectively.

3. Data and Method

3.1. Data

The primary data source comes from the Numerical Identification (Numident) death records of the Social Security Administration extracted from the CenSoc project (Breen et al.,

2023; Breen & Osborne, 2022; Goldstein et al., 2021). The data reports deaths that occurred between the years 1988 and 2005 as reported by the Social Security Administration.[7] The CenSoc-Numident data can be linked to the full-count 1940 census at the individual level. This linked data provides four advantages that make them superior to alternative data sources in mortality studies. First, the data allows us to observe a wide array of individual and family-level characteristics observed in 1940. We use this information to control for other family-level determinants of longevity in our regressions as well as to implement balancing tests. Second, the initial data set contains roughly 8 million observations. After data merging and sample selection (explained below), we are still left with a large sample size (~2.1 M) that boosts our statistical power. Third, although the data overrepresents whites and people of higher socioeconomic status (Breen & Osborne, 2022), it still contains large subsamples of various sociodemographic and socioeconomic groups. Therefore, we are able to examine the heterogeneity of the effects across different subpopulations. Fourth, recent advancements in data linking provide us with high-match-rate cross-census linking rules from Price et al. (2021), which use a combination of name strings (first and last), reported age, birthplace, race, and household composition to probabilistically match individual records across historical census waves. The algorithm scores match candidates on these identifiers and retains the highest-scoring unique candidate as a link, achieving substantially higher match rates than earlier name-and-age-only methods. We employ these linking rules to search for individuals' county of birth/childhood in historical censuses 1880–1930. Thus, the Numident–census linked data provides the inferred county of birth or childhood, a crucial detail in our study and an exceptionally rare identifier absent in most other data sources. The combination of these four characteristics gives the Numident–census data unprecedented advantages for exploring the long-term mortality effects of early-life exposures, with geographically granular exposures and potential heterogeneity across sociodemographic subpopulations.

[7] The Berkeley Unified Numident Mortality Database (BUNMD) data covers relatively wider death window and offers a significantly larger sample size. Although the data is not linked to the 1940 census, it provides county of birth information. We replicate the main results of the paper (without parental controls) using BUNMD data for death years 1988 – 2007 in column 1 of Appendix Table A-1 and find comparable estimate. As we expand the sample the estimated coefficient becomes smaller, suggesting that the effects are more concentrated at older ages. This point is further confirmed and discussed in section 4.3. Additionally, it is important to note that the BUNMD data provides more reliable coverage for deaths occurring after 1988, so cross-death-window comparisons should be approached with caution (Breen & Goldstein, 2022; Goldstein et al., 2021).

3.2. Data Construction and Sample

The procedure for inferring early-life county of residence proceeds as follows. First, a subset of Numident records directly reports county of birth per individuals' Social Security records. For these individuals, we assign the county of birth/childhood directly from Numident records, capturing approximately 49% of observations. Second, using cross-census linking rules from Price et al. (2021), we link 1940 records to full-count historical censuses from 1900 to 1930. This process assigns 4.6% of observations to their 1900 county of residence, 8.6% to 1910, 11.7% to 1920, and 13.4% to 1930 as proxies for early-life county. Additionally, the 1940 census reports the county of residence five years prior (1935); if individuals migrated, we use the 1935 county as the early-life county, covering 1.5% of observations. For unlinked cases, we assign the 1940 county of residence, accounting for 10.5% of observations. We apply similar procedures to historical censuses to extract information on maternal literacy and paternal socioeconomic status.

We digitize the data on CHDs' year of opening from Ferrell (1936). The book details the evolution and significance of county health departments in implementing public health services, particularly in rural areas, through disease control, funding allocations, and organized community health efforts. We merge this data with Numident – census data based on county of birth/childhood. For the main analysis, we restrict the sample to counties with at least one CHD opening although we show the robustness of our results when including all counties in states with at least one CHD county. We further restrict the sample to individuals born between 1900 and 1940 to have several cohorts across various ages before and after a CHD opening. In about 9% of counties, we observe CHD closures in a few years following their opening. We remove these counties from the final sample but provide supplementary evidence that these closings are associated with lower longevity.

We further use historical censuses 1880 – 1940 to construct a county-by-year panel containing county characteristics. We use this data to add control variables in our regressions as well as to test the evolution of county features in years preceding and following CHD openings. This data is extracted from Ruggles et al. (2024). For the mechanism section, we also employ fertility and infant mortality data extracted from Bailey et al. (2016).

We define the additional outcome and control variables used in the analysis as follows. The socioeconomic index (SEI) is the Duncan Socioeconomic Index provided by IPUMS, a constructed

occupational-status measure that assigns each occupation a score based on the 1950 occupational classification scheme (Ruggles et al., 2024). The index reflects the occupational education and income associated with each occupation, using Duncan's weighted measure of occupational standing. We use paternal SEI as a control and the individual's own SEI as an outcome in the mechanism analysis. The occupational income score, used as an additional outcome and control, is an IPUMS-constructed measure that assigns each occupation the median total income of persons in that occupation in 1950, measured in hundreds of 1950 dollars. The measure is based on the 1950 occupational classification scheme and provides a time-consistent proxy for occupational economic standing. Schooling per eligible age is constructed for individuals aged six or older in 1940 as the ratio of completed years of schooling to the maximum years of schooling that the individual could have completed by their reported age (calculated as age minus six, capped at twelve); this variable captures grade progress relative to the age-appropriate schooling trajectory. Education < 9 years is a binary indicator equal to one if the individual completed fewer than nine years of schooling, i.e., never attended high school. Education < 12 years is a binary indicator equal to one if the individual completed fewer than twelve years of schooling, i.e., did not complete high school; the two measures are not equivalent, since an individual with nine to eleven years of schooling has education — 9 years but still education < 12 years. Falling behind is a binary indicator equal to one if the individual is in the bottom tercile of years of schooling within their state—by-birth-year-by-race cell.

To summarize, the analytical sample and key variables are constructed in five steps:

(1) **Mortality data.** We use the CenSoc-Numident file (Breen et al., 2023; Breen & Osborne, 2022; Goldstein et al., 2021), which links Social Security Administration death records (deaths 1988–2005) to the complete-count 1940 census at the individual level.

(2) **County of birth/childhood**. For each individual, we assign an inferred county of birth/childhood using the following hierarchy: (i) Numident-reported county of birth (49% of observations); (ii) cross-census linkage from 1940 to the 1900 census (4.6%), 1910 census (8.6%), 1920 census (11.7%), and 1930 census (13.4%), using the linking rules of Price et al. (2021); (iii) 1935 county of residence reported in the 1940 census for individuals who migrated between 1935 and 1940 (1.5%); and (iv) the 1940 county of residence for unlinked observations (10.5%).

(3) **CHD data**. We digitize county-level CHD opening years from Ferrell (1936) and merge this information into the Numident–census panel on county of birth/childhood.

(4) **Sample restrictions**. We restrict the analytical sample to (a) counties with at least one CHD opening[8], (b) individuals born between 1900 and 1940, and (c) counties that did not experience a CHD closure during our period (about 9% of CHD counties).

(5) **Exposure variable**. For the difference-in-differences specification (Equation 2), we construct a binary indicator equal to one if the individual was in utero or aged 0–4 at the time of the CHD opening in their assigned county of birth/childhood, and zero otherwise. For the event-study specification (Equation 1), we construct a vector of age-at-CHD-opening indicators relative to the omitted reference category of age 11 and above.

3.3. Summary Statistics

Summary statistics of the final sample are reported in Table 1. The average age at death is 909.3 (75.8) months (years). Roughly 69%, 49%, and 29% of the observation live beyond ages 70, 75, and 80, respectively. The bottom panel of Figure 1 depicts the geographic distribution of age at death. Figure 2 shows the density distribution of age at death in the final sample.

About 86% and 13% of individuals are whites and blacks, respectively. We define exposure as a dummy variable indicating whether an individual was in utero or up to the age of 4 at the time of the CHD opening in their county of birth/childhood. The average exposure measure in the final sample is 0.6 with a standard deviation of 0.5. The top panel of Figure 1 illustrates the geographic distribution of CHD openings across counties. These openings are more concentrated in the South and several regions in the Midwest as well as the West. Further, Figure 3 depicts the density distribution of years of CHD openings. A large portion of these openings is concentrated between the years 1920 – 1930.

3.4. Econometric Method

Our identification strategy is to examine the changes in the longevity of individuals born in different years relative to the county-specific year of CHD opening. Specifically, the method relies on the differential effects of CHDs on the longevity of individuals who were exposed to the CHD opening at different ages. The identification assumption is that the mortality outcomes of

[8] We relax this restriction in column 13 of Table 6 by including counties without CHD openings within the same state, excluding cities with separate health department expansions. The estimated coefficient remains nearly identical to the baseline result.

individuals born at older ages at the time of CHD openings follow the same path and are influenced by the same determinants as those of individuals born at younger ages (or in utero) in the absence of CHD openings. We operationalize this comparison using the difference-in-differences regressions of the following form using ordinary least squares and the Sun & Abraham (2021)'s method:

$$y_{icb} = \alpha_0 + \sum_{j \neq [<-10]} \lambda_j I(b - T_c^* = j) + \alpha_1 X_i + \alpha_2 Z_{cb} + \zeta_c + \xi_b + \varepsilon_{icb} \quad (1)$$

Where y is age at death (in months) of individual i born in county c and belongs to birth cohort b. The parameter T^* is a county-specific year of the CHD opening. The function $I(.)$ Indicates the distance (in years) between birth cohorts and year of CHD opening in the county. In other words, it produces a series of dummy variables indicating various age at exposure. We remove the coefficient of individuals with age at exposure of above 10 (i.e., $j < -10$) so that all other coefficients can be interpreted as the change in outcome with respect to these cohorts. For example, consider a county that established its CHD in 1926. An individual born in 1915 was eleven years old at the time of the CHD opening and is therefore in the omitted reference category of individuals aged ten or older at the time of opening. An individual born in 1920 was six years old at the time of opening and is assigned an age relative to CHD opening of minus six. An individual born in 1925 was in utero or in the first year of life at the time of opening and is assigned an age relative to CHD opening of zero. An individual born in 1929 was born four years after the CHD opened and is assigned a value of (positive) four, indicating that the CHD had already been operating in the county for four years before the individual's birth. Thus, each event-study coefficient compares the longevity of individuals at a given age relative to CHD opening with the omitted reference group of individuals aged eleven or older when the CHD opened.

In X, we include as individual covariates dummies for race and gender. We also include dummies for paternal socioeconomic status and maternal literacy as parental covariates. The matrix Z contains county- by-year controls. County controls include share of different occupation groups, share of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. The parameter ζ includes county fixed effects to account for time-invariant influence of county of birth in determining mortality outcomes. The parameter ξ represents birth cohort fixed effects to account

for unobserved heterogeneity in longevity across cohorts. Finally, ε is a disturbance term. Since CHDs in larger, more populated counties typically serve more people and receive higher levels of funding, we weight the regressions by county population to account for these disparities. We cluster standard errors at the county level to absorb serial correlations in error terms.

We further summarize the coefficients λ to capture the overall effects on the longevity in-utero and early-life exposure up to age 4. We use age 4 based on the results of Equation 1, discussed in section 4.1. In so doing, we define an exposure measure of the capture age at CHD opening as being less than or equal to 4. We then implement regressions of the following form:

$$y_{icb} = \alpha_0 + \gamma Exposure_{cb} + \alpha_1 X_i + \alpha_2 Z_{cb} + \zeta_c + \xi_b + \varepsilon_{icb} \quad (2)$$

All parameters in this Equation are similar to those of Equation 1. We should note that because exposure is measured at the county level by the timing of CHD openings rather than by individual-level utilization of CHD services, our coefficients capture the effect of access to (not realized use of) CHD services and should be interpreted as intent-to-treat (ITT) effects. To the extent that not all individuals in CHD counties made use of departmental services, the corresponding average treatment effect on the treated is likely larger in magnitude than the ITT estimates we report. Throughout the manuscript, we retain "exposure" as a shorthand for early-life proximity to a CHD opening, consistent with standard usage in the early-life-exposures literature (Almond et al., 2018; Bleakley, 2007, 2010; Cook et al., 2019; Hoehn-Velasco, 2018, 2021).

For the analysis regarding county-level balancing tests, we use event studies to estimate changes in county characteristics in several years before and after a CHD opening. In so doing, we implement regressions of the following form:

$$y_{ct} = \alpha_0 + \sum_{j \neq -1} \delta_j I(t - T_c^* = j) + \omega_c + \theta_t + \epsilon_{ct} \quad (3)$$

Notation follows Equation 1, with year fixed effects in place of cohort fixed effects. Weighting and standard-error clustering follow the same convention as in Equation 1.

4. Results

4.1. Main Results

The main results of Equation 1 are reported in the top and bottom panels of Figure 4 using OLS and Sun & Abraham (2021)'s method. The similarity in the magnitude and the overall pattern suggests little concern about the endogeneity of OLS-produced coefficients. For age at exposure of 5-9, we observe coefficients that are virtually zero in magnitude, suggesting that there is no difference between the longevity of these cohorts and cohorts with ages at exposure of above 10 (i.e., j>-10). The only anomaly is for age at exposure of 10, for which we observe a sudden drop in longevity.[9] The coefficients are slightly larger in magnitude for age at exposure of 0-4. We observe a gradual but nontrivial rise in the estimated coefficients for those who experienced CHD openings during in utero (i.e. j>0). Despite small variations across all in-utero exposed cohorts, the estimated coefficients become relatively stable after four years and suggest increases in the longevity of roughly 2 months.

The results of Equation 2 are reported in Table 2. The estimated coefficients are quite stable across specifications. The fully parameterized regression of column 3 suggests that exposure at ages 0-4 to CHD openings is associated with 0.9-month increases in longevity. The increase in coefficients for in utero exposure in the event study analysis is partly driven by the expansion of CHDs in size and funding over the years (Hoehn-Velasco, 2018, 2021). Additionally, the uptake and utilization of these clinics could take time due to limited dissemination of information about their availability, affordability, and effectiveness. The exposure measure in equation 2 calculates the average between ages 0-4, combining relatively smaller effects of postnatal ages with larger effects of in utero exposures as well as even larger effects of several years after the establishment

[9] Several factors may contribute to this anomalous coefficient. First, individuals exposed at age 10 in our sample were predominantly born between 1910 and 1922, with modal birth years of 1915, 1917, 1918, and 1920. These birth cohorts experienced a sequence of well-documented adverse early-life shocks during the critical developmental window—including U.S. participation in World War I (1917–1918), the 1918 influenza pandemic, and the post-war recession of 1920–1921—all of which have documented long-run effects on later-life longevity and human capital (Almond, 2006; Beach et al., 2022; J. M. Fletcher, 2018b, 2018a). Overlap with these confounding shocks may exert downward pressure on the age-10 coefficient. Second, age-10 cohorts at exposure are among the earliest-born individuals in our analytical sample and are therefore more susceptible to truncation in the observed death window (1988–2005), which captures only individuals who survived to advanced ages and may induce age-specific selection. Third, the age-10 estimate may also reflect ordinary statistical variation, given the relatively imprecise estimation of coefficients far from the treatment year. We therefore caution against interpreting the age-10 coefficient.

of these CHDs.[10] Therefore, it is not surprising to observe a smaller point estimate of 0.9-month compared with the in-utero coefficients of the event study graph.

Our estimated longevity effects are broadly consistent with those reported by Hoehn-Velasco (2021), though smaller in magnitude in the baseline specification. Using the 1930 Census linked to the DMF (covering male deaths only and death years 1965–2011), she finds that exposure to a CHD before age five increased male lifespan by approximately 0.19 years (about 2.3 months). In our analysis of Numident which covers both genders and death years of 1988 – 2005, we find an effect size of about 0.9 months. Differences in sample coverage and composition likely explain part of this gap. Our dataset includes both males and females, whereas Hoehn-Velasco's analysis is restricted to men, and our primary mortality source covers a shorter death window that may truncate late deaths, resulting in smaller effect sizes (Breen et al., 2023; Goldstein et al., 2023; Lleras-Muney et al., 2022). Moreover, our heterogeneity analysis indicates that the effects are larger among males (section 4.2). Consistent with these two interpretations, when we replicate the analysis using the male sample and expand the death coverage through 2011 (Appendix Table A-2), we obtain similar results as Hoehn-Velasco (2021)'s study. Additionally, as we expand the sample to cover death years up to 2023, the coefficient increases substantially, exceeding Hoehn-Velasco's estimate, suggesting an increase of 4.4 months. Overall, both studies provide converging evidence that early-life exposure to county health departments modestly but significantly improved longevity.

The primary reason for the increasing coefficient size as we expand the death window is the latent effect of CHD exposure on longevity that manifests later in life. As shown in Figure 5, the effects become evident primarily at progressively older ages. In Appendix Table A-3, we present results from two subsamples divided by whether CHD openings occurred before or after the median year. This heterogeneity analysis helps identify which cohorts and death ages

[10] To examine the sensitivity of our main specification to the choice of the 0–4 exposure window, we estimate Equation 2 separately for each individual age at exposure from 1 to 10 and report the results in Appendix Table A-4. The estimates indicate that the longevity gains are statistically significant and economically meaningful for ages 1 through 8, with the largest effects at ages 4–5 (approximately 0.91 and 1.05 months, respectively) and smaller but still significant effects at younger ages (0.52, 0.64, and 0.61 months at ages 1, 2, and 3) and moderately smaller effects at older ages (0.77, 0.46, and 0.59 months at ages 6, 7, and 8). At age 9 and beyond, the estimates are statistically indistinguishable from zero. The 0–4 aggregation window adopted in our main specification is consistent with the developmental origins literature and with the cognate work of Hoehn-Velasco (2021), and it captures the period in which the coefficients are both statistically significant and economically large. The point estimates are robust to alternative groupings: aggregating ages 0–5 or 0–8 yields qualitatively similar conclusions, while restricting attention to ages 0–2 alone omits the larger effects observed at ages 3 through 5.

contribute most to the overall coefficient. The results indicate that the effects are concentrated among individuals exposed to CHD openings before 1925. This pattern holds for both the Numident data (1988–2005) and the DMF data (1975–2023). Because earlier cohorts are observed at older ages within a truncated death window, this finding provides additional evidence that the effects are concentrated at advanced ages. Consequently, as we expand the death window to include more deaths at older ages, the estimated coefficients increase in magnitude. Nevertheless, given the well-documented limitations of post-2005 death coverage in the literature, we rely on the high-mortality-coverage Numident data as our preferred specification.

One way to understand the magnitude of the effects is to compare with similar studies that examine early-life exposures on later-life mortality outcomes in similar settings. For instance, Aaronson et al. (2021) examine the effects of Rosenwald school resources in the Jim Crow South and document that a full exposure to Rosenwald schools among Blacks is associated with two-three months higher longevity. Noghanibehambari & Fletcher (2023d) find that early-life exposure to the 1930s Dust Bowl reduces the longevity of male individuals from farming families by approximately 2.5 months. Vu et al. (2023) examine the effects of in-utero exposure to lynching on the longevity of Blacks and document a reduction of roughly 3 months. Fletcher & Noghanibehambari (2024) study the effects of exposure to pesticides induced by the emergence of cicadas during fetal development on later-life longevity and document a reduction of 2.2 months. Although the estimated effects of CHDs are smaller than other exposures in these studies, we should consider that those exposures are extreme measures affecting subpopulations more vulnerable to the impact, whereas the benefits of CHDs are more marginal and observed across a broad population.

4.2. Heterogeneity analysis

These intent-to-treat effects could be considerably larger for the treated populations as they are all observed across the whole population, while the affected subpopulations for which the accessibility of affordable healthcare services is binding. For instance, based on prior research, we expect to observe larger impacts among minorities and individuals from lower socioeconomic status families (Goodman-Bacon, 2021; Slusky, 2017). Further, since health education was also among the primary missions of these health organizations, we expect to observe larger effects among lower-educated families (Bleakley, 2007; Ferrell, 1936; Hoehn-Velasco, 2018). We

examine these heterogeneities in Table 3. Column 1 restricts the sample to white individuals. We observe a slightly smaller coefficient. In column 2, we examine the results among nonwhites. The estimated coefficient becomes larger than that of whites by about 0.13 months. Given that the average age at death in this subpopulation is significantly lower, the coefficient implies an even larger impact than in the white subpopulation. In columns 3-4, we examine heterogeneity by gender. We find larger effects among males than females, 1.1 versus 0.8 months. In columns 5-6, we show the results among individuals whose mothers have less than 9 years of schooling (i.e., did not attend high school) or 9 and more years of schooling, respectively. The estimated coefficient of low-educated mothers is roughly 4% larger than high-educated mothers.

To further explore heterogeneity by CHD characteristics, we employ data from Ferrell (1936) on county-level per capita budget expenditures of CHDs, which we use as a proxy for the size, capacity, and intensity of departmental activities. We partition CHD counties into terciles of per capita budget spending and estimate the main specification separately for individuals born in counties in the top two terciles versus the bottom tercile of spending. The results are reported in columns 7–8 of Table 3. Column 7 indicates that exposure to CHDs in the top two terciles of per capita spending is associated with a 1.17-month increase in longevity, approximately 30% larger than the average effect documented in column 3 of Table 2 and statistically significant at the 1% level. By contrast, the estimated coefficient for the bottom tercile is 0.40 months (column 8), about half the average effect and not statistically distinguishable from zero. These patterns are consistent with the interpretation that better-resourced CHDs delivered more impactful public-health services.

To further investigate heterogeneity by birthplace characteristics, we use information on urban-versus-rural place of residence as reported in the historical censuses to identify whether individuals were born in urban or rural areas. We then estimate the main specification separately for the rural-born and urban-born subsamples. The results are reported in columns 9–10 of Table 3. We find a 0.31-month coefficient for the rural-born subsample (column 9), which is not statistically distinguishable from zero. By contrast, the urban-born subsample shows a 1.14-month increase in longevity (column 10), statistically significant at the 1% level. This pattern suggests that the longevity benefits of early-life CHD exposure are more concentrated among individuals born in urban areas, possibly reflecting greater concentration of healthcare infrastructure,

complementary services, and information networks in urban settings that may amplify the impact of CHD activities.

4.3. Effects of CHD Closures

To complement the main analysis of CHD openings, we examine the effects of CHD closures, which provide a natural before-and-after comparison in the subset of counties that experienced both an opening and a subsequent closure. In approximately 9% of CHD counties the department closed within a few years of opening. We apply the same difference-in-differences specification (Equation 2) to this closure subsample, with the exposure indicator equal to one if the individual was in utero or aged 0–4 at the time of the closure. The results are reported in Table 4. Across all three specifications, exposure to a CHD closure during the in-utero or early-childhood period is associated with a 1.26–1.40-month reduction in longevity, statistically significant and of similar magnitude (with the opposite sign) to the longevity gains from openings documented in Table 2. This symmetric pattern strengthens the interpretation of the main results: the same identification strategy applied to the loss of CHD access yields effects that are reversed in sign and comparable in magnitude to those from the gain of CHD access, consistent with the causal interpretation that CHDs themselves drive the longevity outcomes. As a further check on sample selection, the robustness analysis in Appendix Table A-9 retains all closure counties in the main analytical sample and yields exposure coefficients of 0.77–0.85 months, similar in magnitude to the baseline estimates in Table 2. Together, these results indicate that the exclusion of closure counties from the main sample does not materially affect the estimates while allowing closures to be examined as a separate and complementary natural experiment.

4.4. Endogeneity and Robustness Checks

One concern in the main results is the endogenous survival into adulthood and into the death window of 1988 – 2005 as a result of exposure to CHDs. For instance, the observed effects could reflect endogenous increases in the share of individuals from lower socioeconomic status families if there is a correlation between exposure and the overrepresentation of these individuals in the final sample. A similar concern is regarding endogenous migration, i.e., new CHD openings encourage migration of families with differential characteristics which correlate with health and longevity of their children. We can empirically test for these concerns by regressing observable characteristics in the final sample on the exposure measure of Equation 2, conditional on county

and cohort fixed effects. These results are reported in Table 5. We do not observe a discernible or significant correlation between observable characteristics and the exposure variable. The estimated coefficients are economically minuscule. For instance, the coefficients of white, father socioeconomic status below/above median, and mother being literate imply a change of less than 1% with respect to the mean of the outcomes (columns 1, 4, 5, and 7). The only significant coefficient is for the female outcome, suggesting a reduction of roughly 2%. This evidence aligns with the results on gender composition of births (reported in Table 7, section 5.1) and both are consistent with the *fragile male* hypothesis, that male fetuses are more susceptible to changes in prenatal health conditions (Kraemer, 2000; Sanders & Stoecker, 2015). Therefore, improvements in the prenatal environment as a result of expansions in CHDs may increase the share of male infants in our data. If these males are less healthy than the population, this compositional change in our sample would likely attenuate our effect estimates.

To further examine this concern, we employ regressions of Equation 3 to examine changes in county characteristics in several years preceding and following a new CHD opening. These results are reported in Figure 6 and Figure 7. To ease interpretations and cross-panel comparisons, we standardize outcomes. We do not observe a significant change in the share of whites, Blacks, immigrants, and married individuals (Figure 6). Although there are reductions in the share of employed individuals and the labor force participation rate several years after a CHD opening, the point estimates remain quite small in magnitude and statistically insignificant (top left and bottom left panels of Figure 7). Since the literature suggests associations between economic conditions experienced early in life and later-life mortality (Van Den Berg et al., 2006), such reductions in employment could add a downward bias in our estimates and suggest an underestimation of the true effects. However, we do not observe discernible changes in occupational income score and share of homeowners. Overall, the evidence does not support the concern regarding endogenous changes in counties' sociodemographic and socioeconomic characteristics that correlate with CHD openings.

In Table 6, we examine the robustness of the results across alternative specifications. To have a benchmark comparison, we report the results of column 3 of Table 2 in column 1 of Table 6. In column 2, we interact county fixed effects by dummies for gender, race, maternal literacy, and paternal socioeconomic status to allow for place effects of counties to have differential impacts on longevity across different subpopulations. Despite this comprehensive set of additional controls

that significantly limits the variation in the treatment, we observe a slight drop in the coefficient. In column 3, we interact birth state by birth year fixed effects so that the variation comes from early versus late adopter counties within a state-year across different ages at exposures. The estimated coefficient is quite comparable to that of column 1.

To account for early adulthood migration between birth and 1940 and the potential influence of migration on mortality (Aaronson et al., 2021), we interact birth state by the state of residence in 1940 fixed effects. The results, reported in column 4, are comparable to the main results. Additionally, studies point to the influence of the season of birth and season of death on mortality outcomes (Vaiserman, 2021). To account for the potential confounding influence of these variables, we include birth month and death month fixed effects in the regressions. The results, reported in column 5, remain comparable to those of column 1.

In columns 6-9, we examine robustness to functional form. We show the effect on log age at death in column 6. The coefficient suggests a significant increase of 0.08%, comparable to the implied percentage change of 0.09% in column 1. In columns 7-9, we investigate the effects on adverse longevity outcomes. For this analysis, we define dummy variables that indicate whether longevity falls within the lowest quantiles (Q=4, 3, 2) of the birth cohort distribution. We observe reductions in adverse longevity measures across all three columns, suggesting that the effects are more pronounced at the lower tail of longevity distribution. To complement this analysis, we examine the effects on indicators of longevity beyond various age thresholds. Specifically, we define indicators of longevity beyond ages 70 – 90 and replicate the results with these outcomes. The estimated coefficients and implied percentage change with respect to the mean of outcome are illustrated in the top and bottom panels of Figure 5. We find that the effects rise in magnitude as we look at older ages, implying the latent influence of early-life exposure to CHDs.

Studies suggest the influence of in-utero and early-life exposure to the 1918 influenza pandemic on long-term health and mortality outcomes (Almond, 2006; Beach et al., 2022; J. M. Fletcher, 2018b, 2018a). In column 10, we drop cohorts of 1918-1919. We observe slightly larger effects. Additionally, studies suggest the influence of the Dust Bowl of the 1930s, the Great Depression of the 1930s, and their associated New Deal programs in this era on long-term health and mortality outcomes (Atherwood, 2022; Cutler et al., 2007). In column 11, we remove cohorts of 1930 – 1940 from the sample. We observe a reduction in the coefficient by roughly 27%. Since

a large portion of CHD openings occurred between 1920 – 1930, eliminating cohorts of 1930 – 1940 means removing a large portion of in-utero-treated individuals from the sample. Therefore, it is not surprising to observe lower estimates. However, the fact that the estimated effect remains economically and statistically significant implies little concern about the potential confounding effects of the economic environment in the 1930s.

Although the information on birth year is extracted from the Social Security Administration, a significant portion of these cohorts were born prior to the establishment of the birth registration area. Therefore, it is not surprising that the birth year information contains measurement errors. Specifically, one documented error is the incidence of age heaping, where individuals round their birth year to the nearest multiple of five (A'Hearn et al., 2009). To examine the influence of age heaping, we exclude from the sample cohorts whose birth year is a multiple of five, i.e., born in the years 1900, 1905, 1910, 1915, 1920, 1925, 1930, 1935, and 1940. The results are reported in column 12. The estimated coefficient rises in magnitude by roughly 20%.

In column 13, we include counties without CHD openings within the same state. In this model, we avoid including cities within these states as they have experienced their own health department expansions (Hoehn-Velasco, 2018; Hoehn-Velasco & Wrigley-Field, 2022). Further, state authorities or funding agencies might reallocate funding from cities to counties or allocate funding from other resources for health departments to both counties and cities. Therefore, the trajectory of outcomes in the cities might correlate with exposure measures. Observations in these cities account for 26% of the overall Numident-census data. When we compare expansion counties with other counties without CHDs, we observe an almost identical coefficient size to that of the main results.

In column 14, we show that the effect is almost identical to the main results when we employ Sun & Abraham (2021)'s estimate. In column 15, we follow Aizer et al. (2016) and implement an Accelerated Failure Time model with an exponential function. We observe an estimated coefficient that is very similar to the logarithmic outcome from the linear regression specification of column 6.

One concern in the main results is that individuals who survive the data merging process have differential characteristics compared with un-linkable individuals and that such linking procedure correlates with their exposure to CHDs. For instance, if individuals from higher

socioeconomic status families are more likely to be in the Numident – census (hence the final sample), then the estimated effects could reflect their higher average longevity conditional on correlations between their likelihood of being in the linked data and exposure to CHDs. We can empirically test this concern by examining the association between being in the final sample from the original population and the exposure measure of Equation 2. In so doing, we use the original population of the 1940 census from cohorts of 1900 – 1940 who were born in CHD counties of the final sample. We then merge this data with the final sample records and generate a dummy variable indicating successful merging. We then regress this indicator on exposure measure, conditional on controls and fixed effects. The results are reported in Appendix Table A-5. We do not observe a discernible and significant correlation between exposure to CHD and being in the final sample from the original population.

To further address this concern, we employ Heckman (1979)'s two-stage model to control for selection bias due to such data merging. This model estimates determinants of successful merging based on observable characteristics in the regression. It then creates an Inverse Mills Ratio (IMR). In the second stage, the model adds IMR as an additional control in the longevity Equation. The results are reported in column 18. The estimated coefficient is only slightly reduced in magnitude to 0.87, suggesting that such selection bias may impose limited influence in driving the results.

A related concern is that linkage quality between Numident and the 1940 census might vary systematically across birth cohorts—for example, if earlier-born cohorts are linked at different rates than later-born cohorts—in ways that interact with CHD timing and could confound the cross-cohort comparison. To examine this directly, we regress an indicator for successful linkage into the final mortality sample on the early-life CHD access measure separately for individuals born before 1925 and those born in or after 1925. The results are reported in Appendix Table A-6. The estimated coefficients are small and statistically indistinguishable from zero in both subsamples. These results suggest that early-life access to CHDs is not systematically associated with the probability of linkage into the final mortality sample within either birth-cohort group. Therefore, the stronger estimates observed for earlier CHD openings are unlikely to be driven by differential linkage rates across cohorts. Instead, the evidence points more directly to the interaction between CHD timing and mortality-window observability: cohorts exposed through earlier CHD openings are older within the Numident death window and are therefore more likely

to be observed at ages where the longevity effects of early-life health investments may become more pronounced.

A remaining concern in the main results arises from the potential spillover influence of CHDs in neighboring counties. To address this, we calculate the total number of CHD openings in all neighboring counties and construct an exposure measure similar to the one used in the main analysis. We include this measure of neighboring counties' CHD exposure in our regressions, allowing the effects of own-county exposure to compete with those of neighboring counties. The results, reported in Appendix Table A-7, show that the effects of neighboring counties' CHD exposure are substantially smaller in magnitude and statistically insignificant.

As an additional robustness check, we restrict the analytical sample to individuals for whom the county of birth is directly reported on the Numident records, rather than inferred from cross-census linkages. This restriction reduces the sample to approximately 1.4 million observations but ensures that the identifying variation comes only from individuals whose county-of-birth assignment is not subject to potential measurement error from linkage procedures. The results are reported in Appendix Table A-8. Across all three specifications, exposure to a CHD opening at ages 0–4 is associated with a 0.81–0.90 month increase in longevity, statistically significant at the 1% level and very similar in magnitude to the corresponding estimates in Table 2. The stability of the coefficients under this stricter sample restriction suggests that the main findings are not driven by potential errors in the county-of-birth assignment from cross-census linking.

4.5. Truncation Bias

The relatively narrow coverage of death data in the Numident files may raise concerns related to truncation bias. This is particularly relevant for younger cohorts who may not have reached mortality within the observation window. In other words, if early-life CHD exposure affects not only longevity but also the likelihood of surviving into the window of observation (1988-2005), our estimates could be biased downward by selective survival. To assess whether our results are sensitive to the mortality window or subject to truncation bias, we replicate our main analysis using a different mortality dataset which covers alternative death window coverage. Specifically, we employ Death Master Files (DMF) data from the Censoc Project (Goldstein et al., 2021), which covers death years of 1975-2023. However, there are two limitations in using this

data. First, it covers deaths to male individuals only. Second, post-2005 deaths in the DMF data are highly underreported because the Social Security Administration retroactively removed state-supplied death records beginning in 2011. This change resulted in substantial declines in coverage and introduced non-random missingness across states, races, and birth cohorts, raising concerns about selection bias in analyses using recent DMF data years (Osborne, 2025).

We report Numident/DMF comparison results in Appendix Table A-2. We start our analysis by replicating the main results for females and males in the Numident data in columns 1 and 2, respectively. Columns 3–5 use the DMF data. In column 3, we restrict the DMF data to death years of Numident data, i.e., 1988-2005. In column 4, we include high-mortality coverage of DMF, i.e., focusing on death years of 1975-2005. Both columns 3 and 4 provide comparable point estimates to those of column 2. In column 5, we extend the DMF coverage through 2011, matching the end of the death window used in Hoehn-Velasco's (2021) sample. In column 6, we further restrict the birth cohorts to 1910–1930, corresponding to the cohorts analyzed in Hoehn-Velasco (2021). The estimated coefficient in this column is identical to hers. Finally, in column 7, we extend the DMF death window further to include deaths occurring between 1975 and 2023. We find a coefficient of 4.5, considerably larger than previous columns, suggesting that truncation might biased the estimated coefficients downward. However, the notably larger coefficient in Column 5 should be interpreted with caution as post-2005 DMF data captures less than 15% of all U.S. deaths, with substantial variation across states and demographic groups (Osborne, 2025).

Taken together, these results clarify how CHD timing, cohort composition, and the mortality observation window interact in our setting. The survival-threshold results show that the estimated effects of early-life access to CHDs become more pronounced at older survival ages (Figure 5). Consistent with this pattern, the estimates are larger for counties with CHD openings before 1925, whose exposed cohorts are mechanically older within the Numident death window (Appendix Table A-2). To rule out the alternative interpretation that earlier-adopter cohorts may be linked to the 1940 census at differential rates—producing apparent rather than genuine differences across CHD timing—we regress an indicator for successful Numident–1940 linkage on birth cohort dummies relative to the CHD opening year, with county and year fixed effects. We find no significant or economically meaningful pattern of linkage rates across cohorts (Appendix Table A-6), indicating that the larger effects we estimate for early-adopter cohorts are not driven by differential data-linking quality. The comparison with DMF-based samples further suggests

that estimated coefficients increase when the mortality window is extended (Appendix Table A-5), although estimates using post-2005 DMF deaths should be interpreted cautiously because of incomplete and nonrandom death coverage. We therefore do not interpret the larger estimates for early CHD adopters as definitive evidence that earlier departments were more effective. Rather, this heterogeneity likely reflects the joint influence of CHD timing, cohort composition, and differential observability of deaths at older ages. This consideration reinforces our interpretation of the baseline Numident estimates as reduced-form intent-to-treat effects for the observed mortality window, and suggests that effects may be understated when later-life deaths are only partially observed.

5. Mechanisms

Before presenting the empirical evidence, we outline five hypothesized channels through which early-life exposure to a CHD opening could affect later-life longevity, motivated by the literature reviewed in Section 2. First, improvements in prenatal and early-life healthcare access associated with CHDs are expected to enhance fetal survival and infant health capital, with potential downstream effects on adult health, consistent with the Fetal Origin Hypothesis and the Developmental Origins of Health and Disease (Almond & Currie, 2011; Barker, 1990). Second, improvements in early-life health capital are predicted to translate into higher educational attainment and socioeconomic status during adulthood, given the well-documented education-health and income-health gradients (Fletcher, 2015; Lleras-Muney, 2005; Lleras-Muney et al., 2022). Third, the resulting improvements in education and labor-market outcomes may shape adult residential location and place attainment, which itself influences exposure to environmental and healthcare quality in later life (Bosma et al., 2001; Mode et al., 2016). Fourth, CHDs' sanitary inspection, surveillance, and educational activities may have reduced exposure to environmental hazards such as lead, with documented long-run consequences for cognitive and cardiovascular health (Clay et al., 2014; Markowitz & Rosner, 2014; Reyes, 2015; Rosner & Markowitz, 1985). Fifth, CHDs targeted communicable diseases—particularly waterborne and vector-borne diseases such as typhoid and malaria—through sanitation, vector control, and immunization campaigns, which may have reduced early-life disease burden and improved long-run health (Beach et al., 2016; Bleakley, 2007, 2010; Cutler & Miller, 2005a). The following subsections empirically test each of these channels in turn.

5.1. Effects on Births and Infants' Health

Early-life exposures might affect later-life outcomes through various channels. As extensively discussed in section 2.2, CHDs might benefit families and specifically infants by providing affordable healthcare access, preventive and treatment services, mitigating the disease environment, and reducing the financial burden associated with healthcare. These channels suggest healing impacts of CHDs that might create new trajectories for later developmental outcomes of infants and children. However, exposure to CHD can also generate a selection mechanism by affecting the survival of infants through reductions in fetal deaths and infant mortality (Bozzoli et al., 2009). For a subset of CHD counties, we have information on births and infant deaths for the years 1915 – 1940 (from Bailey et al. (2016)). Using this data, we investigate the effects on fertility rates and infant mortality rates. In these regressions, the exposure measure is a dummy variable indicating that the year of observation is greater than or equal to the county-specific year of CHD opening. We include county fixed effects, year fixed effects, and county controls and weigh the regressions using county population. The results are reported in the top panel of Table 7. Consistent with prior research, we observe significant reductions in infant mortality rates of about 4%. Further, we observe increases in birth rates of about 3.9%. However, we do not observe a significant and discernible change in the share of births to Black mothers and White mothers. Positive effects on births might partly reflect the improvements in reproductive services that benefit the fetal development environment. Additionally, they may reflect the selective fertility of mothers who would not have given birth if there had not been access to reproductive health services through CHDs.

To complement the analysis of this section, we employ full count censuses 1900 – 1940 to construct a county by year panel of births. In so doing, we focus on children less than 10 and use age information to infer birth year. We then count the number of births in each county and year separately by race and divide by the total number of females aged 15 – 50 to calculate the birth rate. We then merge this panel with the CHD database and examine the evolution of birth rates across cohorts relative to the year of a CHD opening. These results are reported in the bottom panel of Figure 6. We do not observe a change in birth rates of whites but do find significant increases in the birth rate of nonwhites (columns 5-6). In columns 7-8, we examine the effects on the female/male ratio of births of white and nonwhite subpopulations, respectively. Across both subsamples, we observe increases in the share of male infants. Given the higher share of fetal

deaths among male infants (Sanders & Stoecker, 2015), the negative coefficients of female/male ratios may imply reductions in fetal deaths, a fact that has some reflections in increases in birth rate among nonwhites.

The signs of the coefficients in columns 5-6 align with those in columns 3-4. While these findings may indicate some degree of selection among infants—given that nonwhite individuals, particularly Black individuals, tend to have lower average longevity—an increase in the share of nonwhite infants and a decrease in the share of white infants would likely introduce a downward bias into our estimates. This suggests that our coefficients may underestimate the true effects.

5.2. Effects on Education and Socioeconomic Measures

Several strands of research document the influence of education and socioeconomic status on longevity and mortality (Chetty et al., 2016; J. M. Fletcher, 2015; Lleras-Muney, 2005; Lleras-Muney et al., 2022). Since the 1940 census provides information on these measures, we can search for potential mechanisms by examining the effects of CHDs on measures of education and socioeconomic index. The problem is that most observations in the sample, particularly treated individuals, are not old enough to have completed their education by 1940. To overcome this issue, we generate a new variable based on actual education received as a share of the maximum years of education that the individual could have attained based on their age. We call this variable schooling per eligible age. We restrict the sample to individuals above age 5 and estimate the effects of exposure on this variable using regressions of Equation 2. The results are reported in column 1 of Figure 7. We observe significant increases in schooling per eligible age. The coefficient implies a 3.1% increase with respect to the mean. In column 2, we estimate the effect on school attendance. Exposure is associated with a 1.4 percentage-points higher likelihood of school attendance, equivalent to a 2.5% change with respect to the mean of the outcome. Additionally, we define an indicator of *falling behind* which captures individuals who are at the bottom tercile of years of schooling within each state, birth year, and race. We observe reductions in *falling behind* measure, equivalent to 15.4% with respect to the mean of the outcome (column 3).

For estimations of education and socioeconomic index (columns 4-6 and 8-9), we restrict the sample to individuals above age 15. We should note that this sample selection removes a significant portion of treated individuals and limits the variation of exposure. Therefore, we

exercise caution in interpreting these results. In column 4, we observe an increase of 0.17 years of schooling. In columns 5-6, we find reductions in education less than 9 and 12 years, respectively. In column 6, we observe significant increases in college education (conditional on age more than 17). Further, exposure is associated with a 81 basis-points higher likelihood of college education (conditional on age more than 19), equivalent to a 4.9% change with respect to the mean of the outcome. However, the estimated coefficient is noisy and limit interpretations. In columns 8-9, we find significant increases in the socioeconomic index and occupational education score, respectively. With respect to the mean of the outcomes, the coefficients imply changes of 1.9% and 4%, respectively.

Lleras-Muney et al. (2022) estimate that each additional year of education is associated with 4.9 months of higher longevity. Combining their estimate and column 3 of Table 8, we can calculate an increase in longevity of about 0.83 months. This is approximately 92% of the observed effect in Table 2. Therefore, we conclude that a significant portion of the observed effects on longevity can be explained by improvements in human capital.

5.3. Effects on Place Attainments

To further explore the long-term effects of early-life exposure to CHDs, we examine "place attainment" in later life as a mediatory channel that may influence health and longevity (Meijer et al., 2012). In this analysis, "place attainment" is proxied by economic characteristics of the location in which individuals died. Specifically, we look at measures of income at the zip code and county of death. These variables reflect the economic status of the area individuals reached by the end of life, which may shape access to healthcare, environmental quality, and other determinants of mortality.

The results, presented in Table 9, suggest that early-life exposure to CHDs is associated with significant improvements in later-life place attainment. Column 1 shows that individuals exposed to a CHD opening during in-utero or early childhood lived in ZIP Codes with approximately 1.7% higher average household income at the time of death. Columns 2 and 3 suggest that they also died in counties with significantly higher per capita personal income (0.26%) and average weekly wages (0.27%), respectively.

5.4. Potential Role of Lead-Exposure Mitigation

An additional channel that may contribute to the long-run improvements in longevity relates to CHDs' role in mitigating exposure to environmental lead. A growing body of research examines the influence of lead exposure on health and human-capital outcomes in the early 20th-century US and documents nontrivial costs operating through lead-service water lines, leaded gasoline, lead-based paint, and occupational exposures (Clay et al., 2014; Feigenbaum & Muller, 2016; Fletcher & Noghanibehambari, 2025; Troesken, 2008). Although our study period predates the major federal lead restrictions of the 1970s, the historical record suggests that CHDs were active in several lead-relevant domains during these years. First, CHDs administered sanitary inspections of water systems and housing, activities that may have reduced exposure to lead in drinking water and in deteriorating lead-painted dwellings (Fee, 1990; Markowitz & Rosner, 2014). Second, CHDs collected and reported case-level surveillance data on lead poisoning, which supported the enactment and enforcement of early state-level reporting laws and child-protection statutes (Rabin, 1989; Warren, 2000). Third, CHDs disseminated public-health education on the hazards of lead paint, lead solder, contaminated water, and industrial exposures, complementing the emerging state and municipal industrial-hygiene programs that were shaped in part by Alice Hamilton's pioneering investigations of occupational lead poisoning and other toxic exposures (Hamilton, 1925; Sicherman & Hamilton, 1984). Although our data do not allow us to isolate this channel directly, since lead-related activity was bundled with the broader sanitary and inspection portfolio of CHDs, the literature suggests that any reduction in early-life lead exposure resulting from CHD openings would represent a plausible complementary mechanism for the longevity gains we document, particularly given the documented effects of lead on cognitive development, cardiovascular health, and adult mortality (Aizer & Currie, 2019; Clay et al., 2014; J. Fletcher & Noghanibehambari, 2025; Reyes, 2015).

5.5. Effects on Contemporaneous Disease Mortality

A central rationale for the establishment of CHDs in the early 20th century was the eradication of communicable diseases through improvements in water quality, sanitation, vector control, and disease surveillance (Cutler & Miller, 2005b, 2005a; Hoehn-Velasco, 2018). The Rockefeller Sanitary Commission and the U.S. Public Health Service partnered with state and county health authorities to target waterborne and vector-borne diseases such as typhoid,

hookworm, and malaria, funding sanitary inspections, water-system improvements, and antimalarial campaigns (Beach et al., 2016; Bleakley, 2007, 2010; Ferrell, 1936). Although we cannot directly observe county-level water quality or sanitation infrastructure, we examine whether CHD openings are associated with reductions in cause-specific mortality from the diseases most plausibly affected by these public-health interventions.

We employ state-level cause-specific mortality data from Miller (2006) for the years 1900–1936 and estimate regressions of cause-specific death rates per 100,000 state population on the share of states with an active CHD, including state and year fixed effects and state-level controls and weighting by state population. The results are reported in Appendix Table A-10. We document nontrivial reductions in deaths from typhoid (column 1) and malaria (column 2), consistent with CHDs' targeting of waterborne and vector-borne diseases through sanitary inspection, water-system improvements, and antimalarial campaigns (Beach et al., 2016; Bleakley, 2010; Troesken, 2008). Estimates for smallpox (column 3) and measles (column 4) are negative but smaller in magnitude and not statistically distinguishable from zero, suggesting that the contemporaneous disease-mortality effects of CHDs are concentrated among the conditions most directly addressed by sanitary and environmental interventions during this period, rather than those primarily controlled through vaccination campaigns.[11]

6. Conclusion

Despite numerous public health initiatives, expansions of social programs like Medicaid, and the spread of hospitals and clinics in recent decades, the accessibility, availability, and affordability of healthcare continue to pose significant barriers for individuals in the US, leading to adverse effects on health outcomes (Kim et al., 2017). As policy discussions around the expansion and sustainability of these public health initiatives continue, it is important to understand the effects of these programs in the long run. This study adds to these policy discussions by providing evidence of the long-term benefits of early-life exposure to County Health Departments (CHDs), a series of public health initiatives aimed at providing affordable healthcare access in disadvantaged and underserved areas, on later-life longevity.

[11] Regarding water fluoridation, we note that community water fluoridation in the United States did not begin until 1945 in Grand Rapids, Michigan, several years after the end of our study period (CDC, 1999). Fluoridation cannot therefore have contributed to the longevity effects we document, whose CHD exposures occurred between 1908 and the mid-1930s.

We employed Social Security Administration death records linked with the 1940 census to examine the effects of exposure to CHDs across various ages on later-life longevity. We implemented a difference-in-differences estimation strategy to compare the longevity of individuals exposed at different ages to the county-specific year of CHD opening. The results suggested that the benefits are concentrated primarily for in-utero and early years of life exposure. Exposure to a CHD opening in the county of birth during fetal development up to age 4 is associated with increases in longevity of roughly 0.9 months. We emphasize that our estimates capture intent-to-treat effects of access to CHD services rather than treatment-on-treated effects of individual utilization, and the corresponding effects for individuals who actually used CHD services are likely larger.

We observed considerable heterogeneity in the results, whereby the effects are more concentrated among non-White individuals and individuals with low-educated parents. We implemented balancing tests and provided evidence that the effects do not pick up on endogenous changes in sociodemographic and socioeconomic characteristics of families or endogenous evolution of county characteristics. We further tested the sensitivity of the results and showed the robustness to a wide array of alternative specifications, subsamples, functional forms, and estimation methods. Using the information from the 1940 census, we found significant increases in educational attainments, socioeconomic index, occupational educational score, and wage income. Contemporaneous fertility and infant mortality data suggested reductions in infant mortality rates and increases in birth rates following CHD openings in the county. We argued that improvements in fetal environment, fetal survival, and infants' health capital in early life as a result of CHD openings led to improvements in human capital during adulthood with potential implications for later-life longevity.

To understand the magnitude of the findings, we can estimate changes in life years gained by the programs and the associated monetary value of those gains. Applying the marginal effect of column 3 of Table 2 on the population of exposed individuals in the final sample, we estimate approximately 97,287 thousand life years gained as a result of in-utero and early-life exposure to CHDs. Further, studies suggest a Value of Statistical Life of roughly $10 million per individual (Kniesner & Viscusi, 2019). The average longevity of exposed individuals in the final sample is 71.6 years. Simplified calculations suggest that the monetary equivalent of life years gained is

approximately $13.6 billion (in 2020 dollars). The total cost of CHDs' operation during these years is estimated at roughly $1.25 billion (in 2020 dollars) (Ferrell, 1936).

Reference

A'Hearn, B., Baten, J., & Crayen, D. (2009). Quantifying Quantitative Literacy: Age Heaping and the History of Human Capital. *The Journal of Economic History*, *69*(3), 783–808. https://doi.org/10.1017/S0022050709001120

Aaronson, D., Mazumder, B., Sanders, S. G., & Taylor, E. J. (2021). Estimating the effect of school quality on mortality in the presence of migration: Evidence from the jim crow south. *Journal of Labor Economics*, *39*(2), 527–558. https://doi.org/10.1086/709783/SUPPL_FILE/17462DATA.ZIP

Aizer, A., & Currie, J. (2019). Lead and Juvenile Delinquency: New Evidence from Linked Birth, School, and Juvenile Detention Records. *The Review of Economics and Statistics*, *101*(4), 575–587. https://doi.org/10.1162/REST_A_00814

Aizer, A., Eli, S., Ferrie, J., & Muney, A. L. (2016). The Long-Run Impact of Cash Transfers to Poor Families. *American Economic Review*, *106*(4), 935–971. https://doi.org/10.1257/AER.20140529

Almond, D. (2006). Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population. *Journal of Political Economy*, *114*(4), 672–712. https://doi.org/10.1086/507154

Almond, D., & Currie, J. (2011). Killing Me Softly: The Fetal Origins Hypothesis. *Journal of Economic Perspectives*, *25*(3), 153–172. https://doi.org/10.1257/JEP.25.3.153

Almond, D., Currie, J., & Duque, V. (2018). Childhood circumstances and adult outcomes: Act II. *Journal of Economic Literature*, *56*(4), 1360–1446.

Anderson, D. M., Brown, R., Charles, K. K., & Rees, D. I. (2020). Occupational Licensing and Maternal Health: Evidence from Early Midwifery Laws. *Journal of Political Economy*, *128*(11), 4337–4383. https://doi.org/10.1086/710555

Atherwood, S. (2022). Does a prolonged hardship reduce life span? Examining the longevity of young men who lived through the 1930s Great Plains drought. *Population and Environment 2022*, 1–23. https://doi.org/10.1007/S11111-022-00398-W

Bailey, M., Clay, K., Fishback, P., Haines, M., Kantor, S., Severnini, E., & Wentz, A. (2016). U.S. County-Level Natality and Mortality Data, 1915-2007. *Inter-University Consortium for Political and Social Research*. https://doi.org/https://doi.org/10.3886/E100229V4

Bailey, M. J., Hershbein, B., & Miller, A. R. (2012). The Opt-In Revolution? Contraception and the Gender Gap in Wages. *American Economic Journal: Applied Economics*, *4*(3), 225–254. https://doi.org/10.1257/APP.4.3.225

Barbaresco, S., Courtemanche, C. J., & Qi, Y. (2015). Impacts of the Affordable Care Act dependent coverage provision on health-related outcomes of young adults. *Journal of Health Economics*, *40*, 54–68. https://doi.org/10.1016/j.jhealeco.2014.12.004

Barker, D. J. P. (1990). The fetal and infant origins of adult disease. *BMJ: British Medical Journal*, *301*(6761),

1111.

Beach, B., Brown, R., Ferrie, J., Saavedra, M., & Thomas, D. (2022). Reevaluating the Long-Term Impact of In Utero Exposure to the 1918 Influenza Pandemic. *Journal of Political Economy*, *130*(7), 1963–1990. https://doi.org/10.1086/719757/SUPPL_FILE/2016445DATA.ZIP

Beach, B., Ferrie, J., Saavedra, M., & Troesken, W. (2016). Typhoid Fever, Water Quality, and Human Capital Formation. *The Journal of Economic History*, *76*(1), 41–75. https://doi.org/10.1017/S0022050716000413

Berney, B. (1993). Round and Round It Goes: The Epidemiology of Childhood Lead Poisoning, 1950-1990. *The Milbank Quarterly*, *71*(1), 3. https://doi.org/10.2307/3350273

Bhalotra, S., Karlsson, M., & Nilsson, T. (2017). Infant health and longevity: Evidence from a historical intervention in Sweden. *Journal of the European Economic Association*, *15*(5), 1101–1157.

Black, B., Espín-Sánchez, J. A., French, E., & Litvak, K. (2017). The long-term effect of health insurance on near-elderly health and mortality. *American Journal of Health Economics*, *3*(3), 281–311. https://doi.org/10.1162/AJHE_A_00076/ASSET/IMAGES/LARGE/AJHE.2017.00076-FIGURE06.JPEG

Bleakley, H. (2007). Disease and Development: Evidence from Hookworm Eradication in the American South. *The Quarterly Journal of Economics*, *122*(1), 73–117. https://doi.org/10.1162/QJEC.121.1.73

Bleakley, H. (2010). Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure. *American Economic Journal: Applied Economics*, *2*(2), 1–45. https://doi.org/10.1257/APP.2.2.1

Bosma, H., Dike Van De Mheen, H., Borsboom, G. J. J. M., & Mackenbach, J. P. (2001). Neighborhood socioeconomic status and all-cause mortality. *American Journal of Epidemiology*, *153*(4), 363–371.

Boudreaux, M. H., Golberstein, E., & McAlpine, D. D. (2016). The long-term impacts of Medicaid exposure in early childhood: Evidence from the program's origin. *Journal of Health Economics*, *45*, 161–175. https://doi.org/10.1016/j.jhealeco.2015.11.001

Bozzoli, C., Deaton, A., & Quintana-Domeque, C. (2009). Adult height and childhood disease. *Demography*, *46*(4), 647–669. https://doi.org/10.1353/DEM.0.0079

Breen, C. F., & Goldstein, J. R. (2022). Berkeley Unified Numident Mortality Database: Public administrative records for individual-level mortality research. *Demographic Research*, *47*, 111–142. https://doi.org/10.4054/DEMRES.2022.47.5

Breen, C. F., & Osborne, M. (2022). *An Assessment of CenSoc Match Quality*. https://doi.org/10.31235/OSF.IO/BJ5MD

Breen, C. F., Osborne, M., & Goldstein, J. R. (2023). CenSoc: Public Linked Administrative Mortality Records for Individual-level Research. *Scientific Data 2023 10:1*, *10*(1), 1–12. https://doi.org/10.1038/s41597-023-02713-y

Bütikofer, A., Løken, K. V., & Salvanes, K. G. (2019). Infant Health Care and Long-Term Outcomes. *The Review of*

Economics and Statistics, *101*(2), 341–354. https://doi.org/10.1162/REST_A_00790

Byrd, W. M., & Clayton, L. A. (2001). *An American health dilemma: Race, medicine, and health care in the United States 1900-2000*. Routledge.

Carney, M. H. (2021). The impact of mental health parity laws on birth outcomes. *Health Economics*, *30*(4), 748–765. https://doi.org/10.1002/HEC.4217

Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics*, *24*(2), 365–389. https://doi.org/10.1016/J.JHEALECO.2004.09.008

Case, A., & Paxson, C. (2009). Early Life Health and Cognitive Function in Old Age. *American Economic Review*, *99*(2), 104–109. https://doi.org/10.1257/AER.99.2.104

Casner, N. (2001). "Do It Now!" Yakima, Wash, and the Campaign Against Rural Typhoid. *American Journal of Public Health*, *91*(11), 1768–1775.

CDC. (1999). Achievements in Public Health, 1900–1999: Fluoridation of Drinking Water to Prevent Dental Caries. *Morbidity and Mortality Weekly Report*, *48*(41), 933–940. https://www.cdc.gov/mmwr/preview/mmwrhtml/mm4841a1.htm

Chetty, R., Stepner, M., Abraham, S., Lin, S., Scuderi, B., Turner, N., Bergeron, A., & Cutler, D. (2016). The Association Between Income and Life Expectancy in the United States, 2001-2014. *JAMA*, *315*(16), 1750–1766. https://doi.org/10.1001/JAMA.2016.4226

Clay, K., Troesken, W., & Haines, M. (2014). Lead and Mortality. *The Review of Economics and Statistics*, *96*(3), 458–470. https://doi.org/10.1162/REST_A_00396

Cook, C. J., Fletcher, J. M., & Forgues, A. (2019). Multigenerational Effects of Early-Life Health Shocks. *Demography*, *56*(5), 1855–1874. https://doi.org/10.1007/S13524-019-00804-3

Corman, H., Dave, D., & Reichman, N. E. (2019). The Effects of Prenatal Care on Birth Outcomes: Reconciling a Messy Literature. *Oxford Research Encyclopedia of Economics and Finance*. https://doi.org/10.1093/ACREFORE/9780190625979.013.375

Cunha, F., & Heckman, J. (2007). The Technology of Skill Formation. *American Economic Review*, *97*(2), 31–47. https://doi.org/10.1257/AER.97.2.31

Currie, J., & Gruber, J. (1996). Health Insurance Eligibility, Utilization of Medical Care, and Child Health. *The Quarterly Journal of Economics*, *111*(2), 431–466. https://doi.org/10.2307/2946684

Currie, J., & Rossin-Slater, M. (2015). Early-life origins of life-cycle well-being: research and policy implications. *Journal of Policy Analysis and Management : [The Journal of the Association for Public Policy Analysis and Management]*, *34*(1), 208–242. https://doi.org/10.1002/PAM.21805

Cutler, D. M., Miller, G., & Norton, D. M. (2007). Evidence on early-life income and late-life health from America's Dust Bowl era. *Proceedings of the National Academy of Sciences*, *104*(33), 13244–13249.

Cutler, D., & Miller, G. (2005a). The role of public health improvements in health advances: The twentieth-century United States. *Demography 2005 42:1*, *42*(1), 1–22. https://doi.org/10.1353/DEM.2005.0002

Cutler, D., & Miller, G. (2005b). *Water, Water, Everywhere: Municipal Finance and Water Supply in American Cities*. https://doi.org/10.3386/W11096

Ellison, J., Griffith, K., Thursby, M., Slusky, D. J. G., & Bor, J. (2021). The Impact of Driving Time to Family Planning Facilities on Preventive Service Use in Ohio. *American Journal of Preventive Medicine*, *60*(4), 542–545. https://doi.org/10.1016/J.AMEPRE.2020.11.009

Fee, E. (1990). Public Health in Practice: An Early Confrontation with the "Silent Epidemic" of Childhod Lead Paint Poisoning. *Journal of the History of Medicine and Allied Sciences*, *45*(4), 570–606. https://doi.org/10.1093/JHMAS/45.4.570

Feigenbaum, J. J., & Muller, C. (2016). Lead exposure and violent crime in the early twentieth century. *Explorations in Economic History*, *62*, 51–86. https://doi.org/10.1016/J.EEH.2016.03.002

Ferrell, J. A. (1936). *History of County Health Organizations in the United States, 1908-33* (Issue 222). US Government Printing Office.

Fletcher, J. M. (2011). The medium term schooling and health effects of low birth weight: Evidence from siblings. *Economics of Education Review*, *30*(3), 517–527. https://doi.org/10.1016/j.econedurev.2010.12.012

Fletcher, J. M. (2015). New evidence of the effects of education on health in the US: Compulsory schooling laws revisited. *Social Science & Medicine*, *127*, 101–107. https://doi.org/10.1016/J.SOCSCIMED.2014.09.052

Fletcher, J. M. (2018a). Examining the long-term mortality effects of early health shocks. *Applied Economics Letters*, *26*(11), 902–908. https://doi.org/10.1080/13504851.2018.1520960

Fletcher, J. M. (2018b). New evidence on the impacts of early exposure to the 1918 influenza pandemic on old-age mortality. *Biodemography and Social Biology*, *64*(2), 123–126. https://doi.org/10.1080/19485565.2018.1501267

Fletcher, J., & Noghanibehambari, H. (2024). The siren song of cicadas: Early-life pesticide exposure and later-life male mortality. *Journal of Environmental Economics and Management*, *123*, 102903. https://doi.org/10.1016/J.JEEM.2023.102903

Fletcher, J., & Noghanibehambari, H. (2025). Early-life lead exposure and male longevity: Evidence from historical municipal water systems. *Explorations in Economic History*, *97*, 101701. https://doi.org/10.1016/J.EEH.2025.101701

Goldstein, J. R., Alexander, M., Breen, C., Miranda González, A., Menares, F., Osborne, M., Snyder, M., & Yildirim, U. (2021). Censoc Project. In *CenSoc Mortality File: Version 2.0. Berkeley: University of California*. https://censoc.berkeley.edu/data/

Goldstein, J. R., Osborne, M., Atherwood, S., & Breen, C. F. (2023). Mortality modeling of partially observed cohorts using administrative death records. *Population Research and Policy Review*, *42*(3), 36.

Goodman-Bacon, A. (2021). The Long-Run Effects of Childhood Insurance Coverage: Medicaid Implementation, Adult Health, and Labor Market Outcomes. *American Economic Review*, *111*(8), 2550–2593. https://doi.org/10.1257/AER.20171671

Gordon, C. (2009). *Dead on arrival: The politics of health care in twentieth-century America* (Vol. 29). Princeton University Press.

Gu, D., Zhang, Z., & Zeng, Y. (2009). Access to healthcare services makes a difference in healthy longevity among older Chinese adults. *Social Science & Medicine*, *68*(2), 210–219. https://doi.org/10.1016/J.SOCSCIMED.2008.10.025

Hamilton, A. (1925). *Industrial poisons in the United States* (Issue 219). Macmillan.

Hayward, M. D., & Gorman, B. K. (2004). The long arm of childhood: The influence of early-life social conditions on men's mortality. *Demography 2004 41:1*, *41*(1), 87–107. https://doi.org/10.1353/DEM.2004.0005

Heckman, J. J. (1979). Sample selection bias as a specification error. *Econometrica*, *47*(1), 153–161. https://doi.org/10.2307/1912352

Hoehn-Velasco, L. (2018). Explaining declines in US rural mortality, 1910–1933: The role of county health departments. *Explorations in Economic History*, *70*, 42–72. https://doi.org/10.1016/J.EEH.2018.08.003

Hoehn-Velasco, L. (2021). The Long-term Impact of Preventative Public Health Programs. *The Economic Journal*, *131*(634), 797–826. https://doi.org/10.1093/EJ/UEAA071

Hoehn-Velasco, L., & Wrigley-Field, E. (2022). City health departments, public health expenditures, and urban mortality over 1910–1940. *Economic Inquiry*, *60*(2), 929–953. https://doi.org/10.1111/ECIN.13038

Kim, T. J., Vonneilich, N., Lüdecke, D., & von dem Knesebeck, O. (2017). Income, financial barriers to health care and public health expenditure: A multilevel analysis of 28 countries. *Social Science & Medicine*, *176*, 158–165. https://doi.org/10.1016/J.SOCSCIMED.2017.01.044

Kniesner, T. J., & Viscusi, W. K. (2019). The Value of a Statistical Life. *Oxford Research Encyclopedia of Economics and Finance*. https://doi.org/10.1093/ACREFORE/9780190625979.013.138

Kraemer, S. (2000). The fragile male. *BMJ : British Medical Journal*, *321*(7276), 1609. https://doi.org/10.1136/BMJ.321.7276.1609

Lemons, J. S. (1969). The sheppard-towner act: Progressivism in the 1920s. *The Journal of American History*, *55*(4), 776–786.

Lleras-Muney, A. (2005). The Relationship Between Education and Adult Mortality in the United States. *The Review of Economic Studies*, *72*(1), 189–221. https://doi.org/10.1111/0034-6527.00329

Lleras-Muney, A., Price, J., & Yue, D. (2022). The association between educational attainment and longevity using individual-level data from the 1940 census. *Journal of Health Economics*, *84*, 102649. https://doi.org/10.1016/J.JHEALECO.2022.102649

Lu, Y., & Slusky, D. J. G. (2016). The Impact of Women's Health Clinic Closures on Preventive Care. *American Economic Journal: Applied Economics*, *8*(3), 100–124. https://doi.org/10.1257/APP.20140405

Lu, Y., & Slusky, D. J. G. (2019). The Impact of Women's Health Clinic Closures on Fertility. *American Journal of Health Economics*, *5*(3), 334–359. https://doi.org/10.1162/AJHE_A_00123

Lumsden, L. L. (1918). *Rural sanitation: a report on special studies made in 15 Counties in 1914, 1915, and 1916* (Issue 94). US Government Printing Office.

Lumsden, L. L. (1927). Cooperative Rural Health Work of the Public Health Service in the Fiscal Year 1927. *Public Health Reports (1896-1970)*, 2545–2597.

Marein, B. (2023). Public health departments and the mortality transition in Latin America: Evidence from Puerto Rico. *Journal of Development Economics*, *160*, 102980. https://doi.org/10.1016/J.JDEVECO.2022.102980

Markowitz, G., & Rosner, D. (2014). *Lead wars: the politics of science and the fate of America's children* (Vol. 24). Univ of California Press.

Meijer, M., Röhl, J., Bloomfield, K., & Grittner, U. (2012). Do neighborhoods affect individual mortality? A systematic review and meta-analysis of multilevel studies. *Social Science & Medicine*, *74*(8), 1204–1212.

Meyers, K., & Thomasson, M. A. (2021). Can pandemics affect educational attainment? Evidence from the polio epidemic of 1916. *Cliometrica*, *15*(2), 231–265. https://doi.org/10.1007/S11698-020-00212-3/TABLES/14

Miller, G. (2006). *Vital Statistics Deaths: Historical 1900–1936*. National Bureau of Economic Research. https://www.nber.org/research/data/vital-statistics-deaths-historical-1900-1936

Mocan, N., Raschke, C., & Unel, B. (2015). The impact of mothers' earnings on health inputs and infant health. *Economics and Human Biology*, *19*, 204–223. https://doi.org/10.1016/j.ehb.2015.08.008

Mode, N. A., Evans, M. K., & Zonderman, A. B. (2016). Race, neighborhood economic status, income inequality and mortality. *PloS One*, *11*(5), e0154535.

Moehling, C. M., & Thomasson, M. A. (2014). Saving Babies: The Impact of Public Education Programs on Infant Mortality. *Demography*, *51*(2), 367–386. https://doi.org/10.1007/S13524-013-0274-5

Neelsen, S., & Stratmann, T. (2011). Effects of prenatal and early life malnutrition: Evidence from the Greek famine. *Journal of Health Economics*, *30*(3), 479–488.

Noghanibehambari, H., & Fletcher, J. (2023). Long-Term Health Benefits of Occupational Licensing: Evidence from Midwifery Laws. *Journal of Health Economics*, *92*, 102807. https://doi.org/10.1016/J.JHEALECO.2023.102807

Noghanibehambari, H., & Fletcher, J. M. (2024). Dust to Feed, Dust to Grey: The Effect of In-Utero Exposure to the Dust Bowl on Old-Age Longevity. *Demography*. https://doi.org/10.1215/00703370-11140760

Olafsson, A. (2016). Household Financial Distress and Initial Endowments: Evidence from the 2008 Financial Crisis. *Health Economics*, *25*, 43–56. https://doi.org/10.1002/hec.3426

Osborne, M. (2025). *CenSoc-DMF Weights: Technical Documentation for CenSoc-DMF Data Version 4.0*.

Parikh, P. B., Yang, J., Leigh, S., Dorjee, K., Parikh, R., Sakellarios, N., Meng, H., & Brown, D. L. (2014). The Impact of Financial Barriers on Access to Care, Quality of Care and Vascular Morbidity Among Patients with Diabetes and Coronary Heart Disease. *Journal of General Internal Medicine*, *29*(1), 76–81. https://doi.org/10.1007/S11606-013-2635-6/TABLES/5

Price, J., Buckles, K., Van Leeuwen, J., & Riley, I. (2021). Combining family history and machine learning to link historical records: The Census Tree data set. *Explorations in Economic History*, *80*, 101391. https://doi.org/10.1016/J.EEH.2021.101391

Rabin, R. (1989). Warnings unheeded: a history of child lead poisoning. *American Journal of Public Health*, *79*(12), 1668–1674. https://doi.org/10.2105/AJPH.79.12.1668

Reyes, J. W. (2015). Lead exposure and behavior: effects on antisocial and risky behavior among children and adolescents . *Economic Inquiry*, *53*(3), 1580–1605. https://doi.org/10.1111/ECIN.12202

Rosner, D., & Markowitz, G. (1985). A "gift of God"?: The public health controversy over leaded gasoline during the 1920s. *American Journal of Public Health*, *75*(4), 344–352. https://doi.org/10.2105/AJPH.75.4.344

Ruggles, S., Flood, S., Sobek, M., Backman, D., Chen, A., Cooper, G., Richards, S., Rodgers, R., & Schouweiler, M. (2024). *IPUMS USA: Version 15.0 [dataset]*. https://doi.org/10.18128/D010.V15.0

Saaritsa, S., Simanainen, E., & Ristola, M. (2024). Nurses, doctors, and mortality: the effectiveness of early health professionals in rural Finland, 1880–1938. *European Review of Economic History*, *28*(1), 91–119. https://doi.org/10.1093/EREH/HEAD011

Sanders, N. J., & Stoecker, C. (2015). Where have all the young men gone? Using sex ratios to measure fetal death rates. *Journal of Health Economics*, *41*, 30–45. https://doi.org/10.1016/J.JHEALECO.2014.12.005

Saunders, M. R., & Alexander, G. C. (2009). Turning and churning: Loss of health insurance among adults in medicaid. *Journal of General Internal Medicine*, *24*(1), 133–134. https://doi.org/10.1007/S11606-008-0861-0/METRICS

Sicherman, B., & Hamilton, A. (1984). *Alice Hamilton, a life in letters*. Harvard University Press.

Slusky, D. J. G. (2017). Defunding women's health clinics exacerbates Hispanic disparity in preventive care. *Economics Letters*, *156*, 61–64. https://doi.org/10.1016/J.ECONLET.2017.04.013

Spencer, R. (1994). Contested Terrain: The Mississippi Flood of 1927 and the Struggle to Control Black Labor. *The Journal of Negro History*, *79*(2), 170–181. https://doi.org/10.2307/2717627

Sun, L., & Abraham, S. (2021). Estimating dynamic treatment effects in event studies with heterogeneous treatment effects. *Journal of Econometrics*, *225*(2), 175–199. https://doi.org/10.1016/J.JECONOM.2020.09.006

Troesken, W. (2008). Lead Water Pipes and Infant Mortality at the Turn of the Twentieth Century. *Journal of Human Resources*, *43*(3), 553–575. https://doi.org/10.3368/JHR.43.3.553

Vaiserman, A. (2021). Season-of-birth phenomenon in health and longevity: epidemiologic evidence and mechanistic considerations. *Journal of Developmental Origins of Health and Disease*, *12*(6), 849–858. https://doi.org/10.1017/S2040174420001221

Van Den Berg, G. J., Lindeboom, M., Portrait, F., Berg, G. J. Van Den, Lindeboom, M., Portrait, F., den Berg, G. J., Lindeboom, M., & Portrait, F. (2006). Economic Conditions Early in Life and Individual Mortality. *American Economic Review*, *96*(1), 290–302. https://doi.org/10.1257/000282806776157740

Vu, H., Noghanibehambari, H., Fletcher, J., & Green, T. (2023). Prenatal Exposure to Racial Violence and Later Life Mortality among Males: Evidence from Lynching. *SSRN Electronic Journal*. https://doi.org/10.2139/SSRN.4613372

Warren, C. (2000). *Brush with death: a social history of lead poisoning*. JHU Press.

Wherry, L. R., & Meyer, B. D. (2016). Saving teens: Using a policy discontinuity to estimate the effects of medicaid eligibility. *Journal of Human Resources*, *51*(3), 556–588. https://doi.org/10.3368/jhr.51.3.0913-5918R1

Yu, S. W. Y., Hill, C., Ricks, M. L., Bennet, J., & Oriol, N. E. (2017). The scope and impact of mobile health clinics in the United States: A literature review. *International Journal for Equity in Health*, *16*(1), 1–12. https://doi.org/10.1186/S12939-017-0671-2/TABLES/1

Tables

Table 1 - Summary Statistics

	Mean	SD	Min	Max
Age at death (months)	909.25	106.43	565	1269
Log age at death	6.81	.12	6.34	7.15
Age at death > 70	.69	.46	0	1
Age at death > 75	.49	.5	0	1
Age at death > 80	.29	.45	0	1
Birth year	1921.86	8.61	1900	1940
Death year	1997.64	4.93	1988	2005
White	.86	.34	0	1
Black	.13	.34	0	1
Female	.51	.5	0	1
Exposure	.6	.49	0	1
Year of CHD opening	1923.57	5.1	1908	1933
Father socioeconomic status below median	.41	.49	0	1
Father socioeconomic status above median	.38	.49	0	1
Father socioeconomic status missing	.21	.41	0	1
Mother literate	.57	.5	0	1
Mother Literacy Missing	.39	.49	0	1
Schooling per eligible age	.4	.18	0	3.33
School attendance	.42	.49	0	1
Bottom-decile of state-cohort-level education	.18	.39	0	1
Years of schooling	9.52	3.12	0	20
Education < high school	.37	.48	0	1
Education < 12 years	.66	.47	0	1
Education college – more	.14	.35	0	1
Socioeconomic index	27.52	20.75	3	96
Occupational education score	13.27	18.99	1	93.9
Observations		2,147,634		

Table 2 - Early-life Exposure to County Health Departments and Later-life Longevity

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Exposure	.91997***	.93074***	.90867***
	(.19868)	(.2042)	(.19207)
Observations	2147634	2147634	2147634
R-squared	.70239	.70243	.70244
Mean DV	887.680	887.680	887.680
County FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Individual Controls	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.

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

Table 3 - Heterogeneity in Early-life Exposure to County Health Departments and Later-life Longevity

	Outcome: Age at Death (Months), Subsamples				
	Whites	Nonwhites	Females	Males	Mother education < high school
	(1)	(2)	(3)	(4)	(5)
Exposure	.8469***	.9846**	.79336***	1.08972***	.9266**
	(.2058)	(.4446)	(.24269)	(.27039)	(.44322)
Observations	1855927	291701	1093511	1054123	917146
R-squared	.7023	.69839	.7244	.66001	.60823
Mean DV	888.672	873.663	906.116	870.775	875.922
	Mother education ≥ high school	Top-Two Terciles of Per capita Budget Spending	Bottom Tercile of Per capita Budget Spending	Rural-Born	Urban-Born
	(6)	(7)	(8)	(9)	(10)
Exposure	.88963***	1.17407***	.401	.31156	1.14341***
	(.22993)	(.21491)	(.30712)	(.23074)	(.25078)
Observations	1230488	1227915	919719	1114466	1033168
R-squared	.74258	.6983	.69307	.71077	.69766
Mean DV	896.191	878.931	905.858	885.277	888.474

Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Each column reports estimates for the subsample listed in the column heading. Columns 7–8 split the sample by county-level CHD per-capita budget spending, with columns 7 and 8 corresponding to counties in the top two terciles and bottom tercile of spending, respectively. Columns 9–10 split the sample by rural versus urban birthplace. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender, where applicable. Family controls include maternal education and paternal socioeconomic status, where applicable. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Table 4 - Exploring the Effects of CHD Closures on Later Life Longevity

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Exposure to CHD Closure	-1.25922*	-1.29701**	-1.40134***
	(.6503)	(.64538)	(.51879)
Observations	736586	736586	736586
R-squared	.69536	.69542	.69543
Mean DV	899.451	899.451	899.451
County FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Individual Controls	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The treatment variable is an indicator equal to one if a county health department closed in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. These estimates examine whether the loss of CHD access during early life is associated with later-life longevity. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Table 5 - Balancing Tests: Early-life Exposure to County Health Departments and Changes in Sociodemographic and Socioeconomic Characteristics of the Final Sample

	Outcomes:							
	White	Black	Female	Father Socioeconomic Status Below Median	Father Socioeconomic Status Above Median	Father Socioeconomic Status Missing	Mother Literate	Mother Literacy Missing
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Exposure	-.00957	.00539	-.00967*	.00004	-.00662	.00658	.00491	.00359
	(.00824)	(.00575)	(.00532)	(.00495)	(.01265)	(.01133)	(.00624)	(.01024)
Observations	2147634	2147634	2147634	2147634	2147634	2147634	2147634	2147634
R-squared	.12595	.14296	.03374	.05089	.07262	.1059	.4312	.47165
Mean DV	0.934	0.060	0.478	0.279	0.542	0.179	0.540	0.425

Notes. Standard errors, clustered at the county level, are reported in parentheses. Each column reports a separate regression in which the dependent variable is the sociodemographic or family-background characteristic listed in the column heading. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. These regressions test whether early-life access to CHDs is systematically associated with observable predetermined characteristics in the final linked sample. All regressions include county fixed effects and birth-year fixed effects and are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Table 6 - Robustness Checks

	Column 3 of Table 2	County by Covariates FE	Birth State by Birth Year FE	1940 State by Birth State FE
	(1)	(2)	(3)	(4)
Exposure	.90867***	.75341***	.81215***	.92973***
	(.19207)	(.19063)	(.20832)	(.20921)
Observations	2147634	2147628	2147223	2147062
R-squared	.70244	.7026	.70338	.70299
Mean DV	887.680	887.680	887.683	887.664
	Birth Month and Death Month FE	Log Age at Death	Age at Death: First Quartile Within Birth Cohort Distribution	Age at Death: First Tercile Within Birth Cohort Distribution
	(5)	(6)	(7)	(8)
Exposure	.87196***	.00096***	-.00482***	-.00645***
	(.19011)	(.0002)	(.00134)	(.00151)
Observations	2147634	2147634	2147634	2147634
R-squared	.7037	.69566	.00299	.00339
Mean DV	887.680	6.782	0.248	0.331
	Age at Death: Below Median Birth Cohort Distribution	Excluding Birth Cohorts of 1918-1919	Excluding Birth Cohorts of 1930-1940	Excluding Age Heaping
	(9)	(10)	(11)	(12)
Exposure	-.00701***	.93904***	.6691***	1.12063***
	(.00132)	(.22079)	(.24011)	(.20183)
Observations	2147634	1961072	1712926	1708030
R-squared	.00402	.71221	.54825	.70247
Mean DV	0.497	883.055	927.981	887.033
	Including All Counties in CHD States	Sun & Abraham (2021)'s Estimate	Accelerated Failure Time Model	Heckman (1979) Two-Step Model
	(13)	(14)	(15)	(16)
Exposure	.93599***	.90867***	.00097***	.8784***
	(.20559)	(.19207)	(.0002)	(.2337)
Observations	5970199	2147634	2147634	22782844
R-squared	.70331	.70244	---	---
Mean DV	912.952	887.680	887.680	887.680

Notes. Standard errors, clustered at the county level, are reported in parentheses. Unless otherwise indicated, the outcome is age at death measured in months. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Column 1 reproduces the preferred specification from column 3 of Table 2. Column 2 adds county-by-covariate fixed effects. Column 3 adds birth-state-by-birth-year fixed effects. Column 4 adds 1940-state-by-birth-state fixed effects. Column 5 adds birth-month and death-month fixed effects. Column 6 uses log age at death as the outcome. Columns 7–9 use indicators for whether age at death falls in the first quartile, first tercile, or below the median of the birth-cohort-specific longevity distribution. Columns 10–12 exclude the 1918–1919 birth cohorts, the 1930–1940 birth cohorts, and birth years ending in 0 or 5, respectively. Column 13 includes all counties in states with at least one CHD county. Column 14 reports the Sun and Abraham (2021) estimate. Column 15 reports results from an accelerated failure time model. Column 16 reports results from a Heckman two-step model. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls unless the column-specific specification replaces or augments them. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Table 7 - Exploring Mechanisms: Effects on Contemporaneous Fertility and Infant Mortality

	Vital Statistics Data, Outcomes:			
	Log Infant Mortality Rate	Log Birth Rate	Share of Births to Blacks	Share of Births to Whites
	(1)	(2)	(3)	(4)
CHD Open in County-Year	-.03994**	.03926**	.00054	-.0139
	(.01594)	(.01932)	(.00644)	(.01213)
Observations	3827	3828	3585	3585
R-squared	.69729	.89273	.96773	.93276
Mean DV	4.266	4.117	0.403	0.592
	Full-Count Census 1900-1940, Outcomes:			
	Log Birth Rate, Whites	Log Birth Rate, Non-Whites	Female/Male Ratio, Whites	Female/Male Ratio, Non-Whites
	(5)	(6)	(7)	(8)
CHD Open in County-Year	-.01447	.15094***	-.00244	-.0015
	(.01065)	(.05799)	(.00162)	(.01175)
Observations	30774	26780	30774	26780
R-squared	.93107	.95568	.04247	.07486
Mean DV	3.192	0.832	0.974	1.057

Notes. Standard errors, clustered at the county level, are reported in parentheses. Columns 1–4 use Vital Statistics data; columns 5–8 use the full-count censuses from 1900–1940. The treatment variable is an indicator equal to one for county-years after a CHD had opened in the county. Columns 1–4 examine contemporaneous changes in infant mortality, birth rates, and the racial composition of births. Columns 5–8 examine birth rates and the female-to-male birth ratio by race using birth cohorts inferred from the full-count censuses. All regressions include county fixed effects, year fixed effects, and county controls. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Table 8 - Exploring Mechanisms: Effects on Education and Socioeconomic Measures

	Outcomes:								
	Schooling Per Eligible Age	School Attendance	Bottom-Decile of State-Cohort-Level Education	Years Of Schooling	Education < High School	Education < 12 Years	Education College – More	Socioeconomic Index	Occupational Education Score
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Exposure	.01231***	.0137**	-.03584***	.16965***	-.0128*	-.03159**	.00808	.58469**	.61276**
	(.00206)	(.00641)	(.00644)	(.06526)	(.00702)	(.01234)	(.00748)	(.27956)	(.24606)
Observations	1920936	1963234	1920936	1400747	1426227	1426227	961355	624632	626841
R-squared	.63456	.6799	.1546	.29151	.21285	.27552	.10589	.18772	.11761
Mean DV	0.423	0.547	0.190	10.214	0.258	0.622	0.162	31.465	14.992

Notes. Standard errors, clustered at the county level, are reported in parentheses. Each column reports a separate regression in which the dependent variable is the education or socioeconomic outcome listed in the column heading. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Schooling per eligible age is defined as completed years of schooling divided by the maximum years of schooling the individual could have completed by their age in 1940. School attendance is an indicator for whether the individual was attending school at the time of the 1940 Census. The bottom-decile education measure is an indicator for whether the individual falls in the bottom decile of schooling within their state-by-birth-cohort cell. Education < high school is an indicator for completing fewer than 9 years of schooling, while Education < 12 years is an indicator for completing fewer than 12 years of schooling. College education is an indicator for having attended at least some college. The socioeconomic index and occupational education score are occupation-based measures from IPUMS. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1

Table 9 - Exploring Mechanisms: Effects on Later-Life Place Attainments

	Outcomes:		
	Log Household Income in 2005 in Death Zip Code	Log Per Capita Personal Income in Death County in Death Year	Log Average Weekly Wages in Death County in Death Year
	(1)	(2)	(3)
Exposure	.01656***	.00259**	.00269***
	(.00556)	(.00109)	(.00096)
Observations	1374335	1360238	1360238
R-squared	.05734	.10795	.09766
Mean DV	11.206	10.672	10.794

Notes. Standard errors, clustered at the county level, are reported in parentheses. Each column reports a separate regression in which the dependent variable is a measure of later-life place attainment based on the individual's ZIP code or county of death. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Column 1 uses the log of average household income in 2005 in the individual's ZIP code of death. Column 2 uses the log of per-capita personal income in the individual's county of death in the year of death. Column 3 uses the log of average weekly wages in the individual's county of death in the year of death. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.

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

Figures

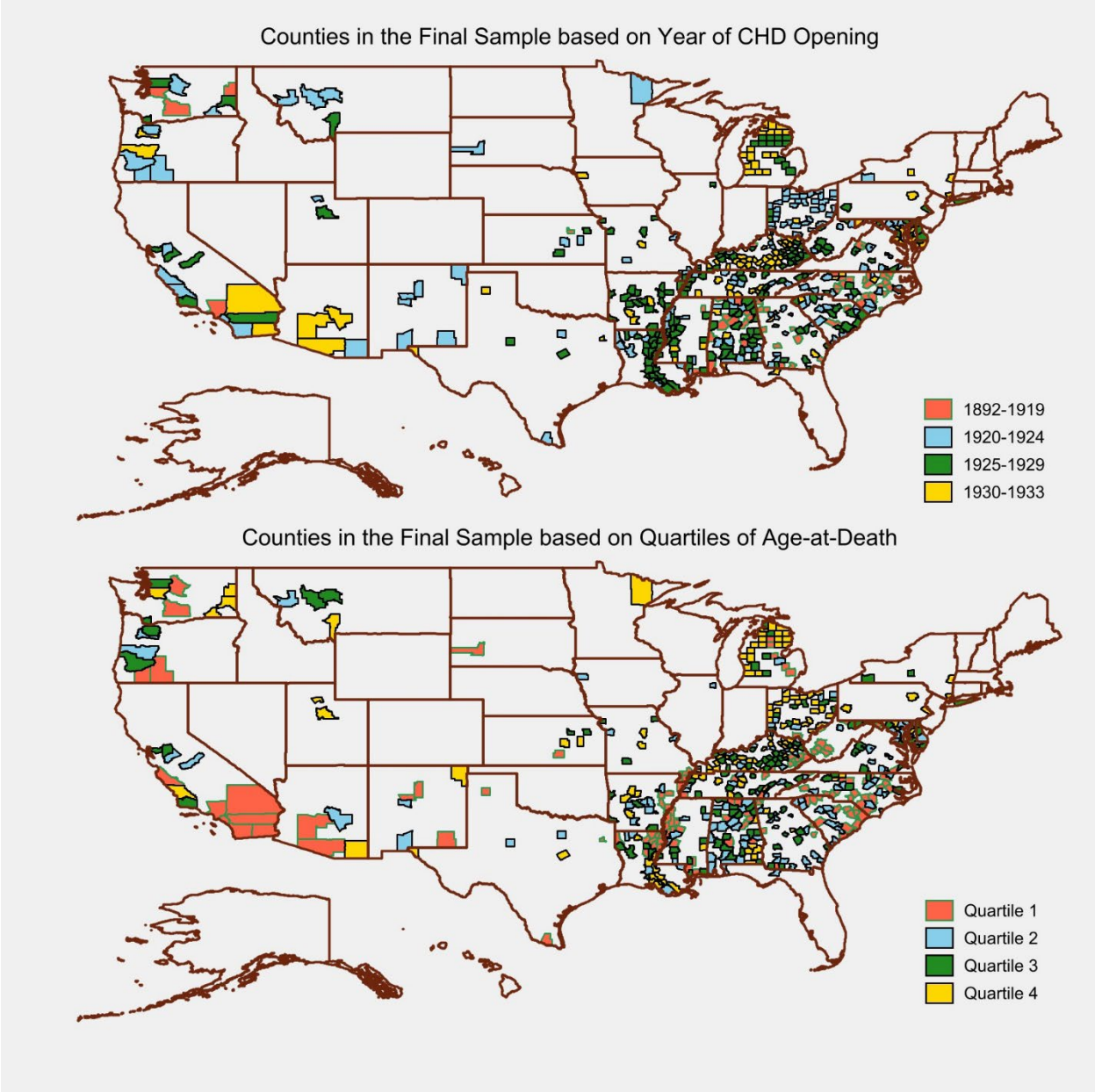

Figure 1 - Geographic Distribution of CHD Opening (Top Panel) and Age At Death (Bottom Panel) in the Final Sample

Notes. The top panel shows the geographic distribution of County Health Department (CHD) openings across U.S. counties, with shading indicating the year of the first CHD opening in each county; counties with no CHD opening through 1933 are shown unshaded. By 1933 (the end of the CHD-opening years in our data), approximately 600 counties across 38 states had established a CHD (Ferrell, 1936). Using the 1940 full-count census, these CHD counties contained approximately 32.8% of the total U.S. population in 1940. The bottom panel shows the geographic distribution of average age at death (in months) for the final analytical sample, computed from Numident death records (1988–2005) linked to the 1940 census. Sample restrictions are described in Section 3.2.

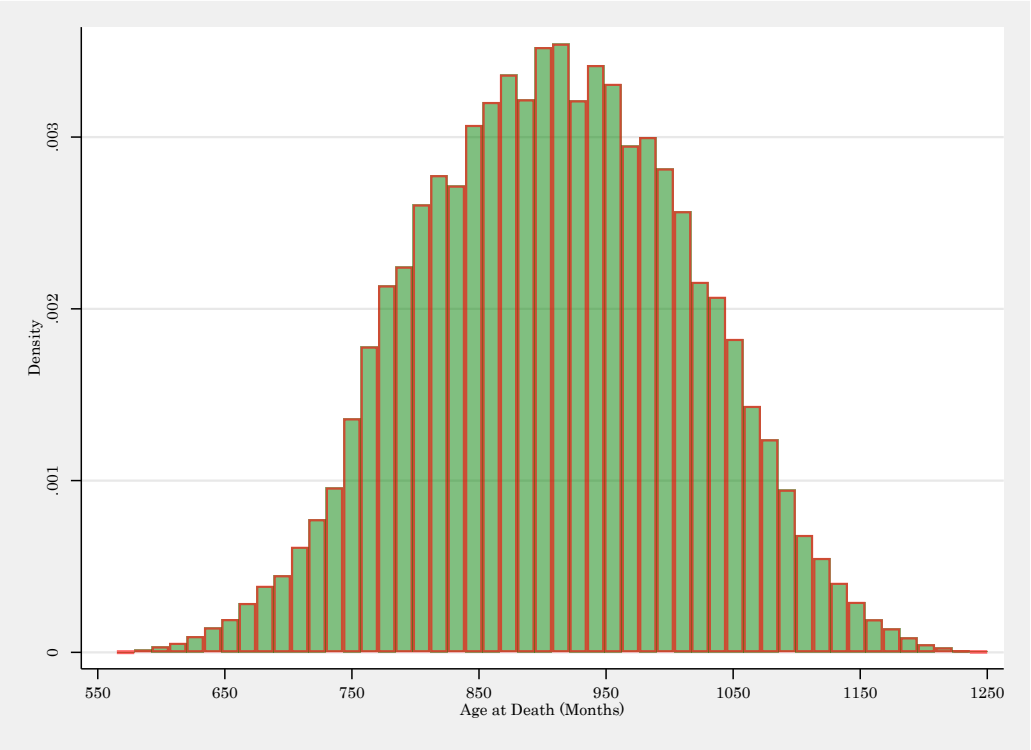

Figure 2 - Density Distribution of Age at Death

Notes. The figure shows the density distribution of age at death (in months) in the final analytical sample. The sample consists of individuals born between 1900 and 1940 in U.S. counties that experienced at least one CHD opening between 1908 and 1933, linked to Social Security Administration Numident death records covering the death window 1988–2005, as described in Section 3.2. The mean age at death is 909.3 months (75.8 years), with a standard deviation of 75.8 months. Approximately 69%, 49%, and 29% of the sample died beyond ages 70, 75, and 80, respectively. The total sample size is approximately 2.1 million individuals.

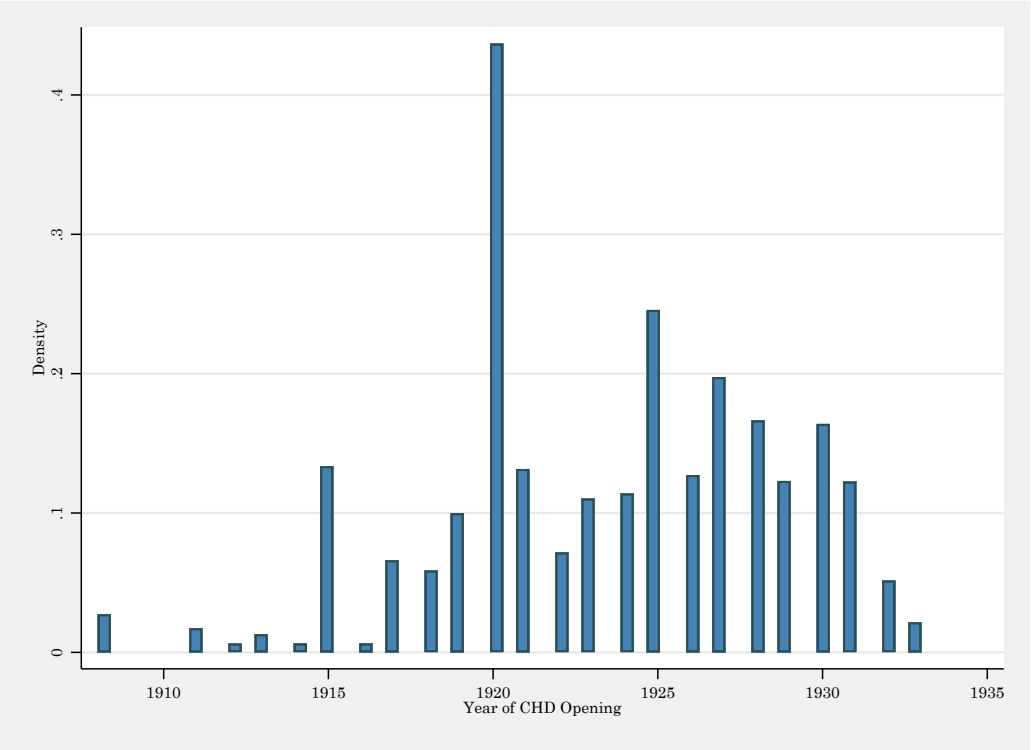

Figure 3 - Distribution of Years of CHD Openings

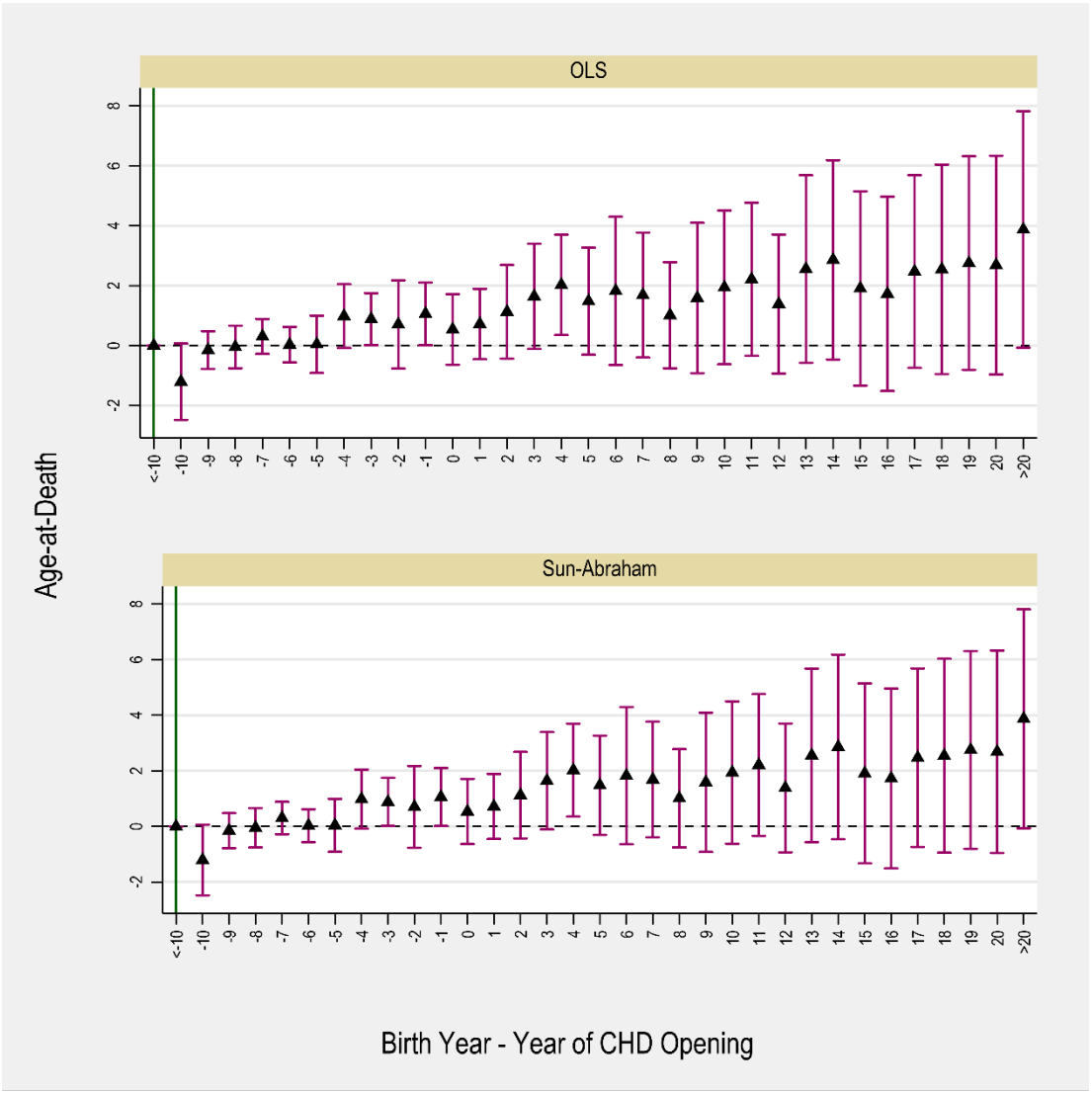

Figure 4 - Event Study Analyses to Examine the Effects of Early-life Exposure to County Health Departments and Later-life Longevity

Notes. The figure reports point estimates and 95% confidence intervals from event-study regressions of age at death, measured in months, on indicators for birth year relative to the year of CHD opening in the individual's county of birth/childhood. Positive values on the x-axis indicate cohorts born after a CHD had opened; negative values indicate cohorts who were already born when the CHD opened. The omitted reference category is individuals whose birth year is more than 10 years before the CHD opening. Standard errors are clustered at the county level. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.

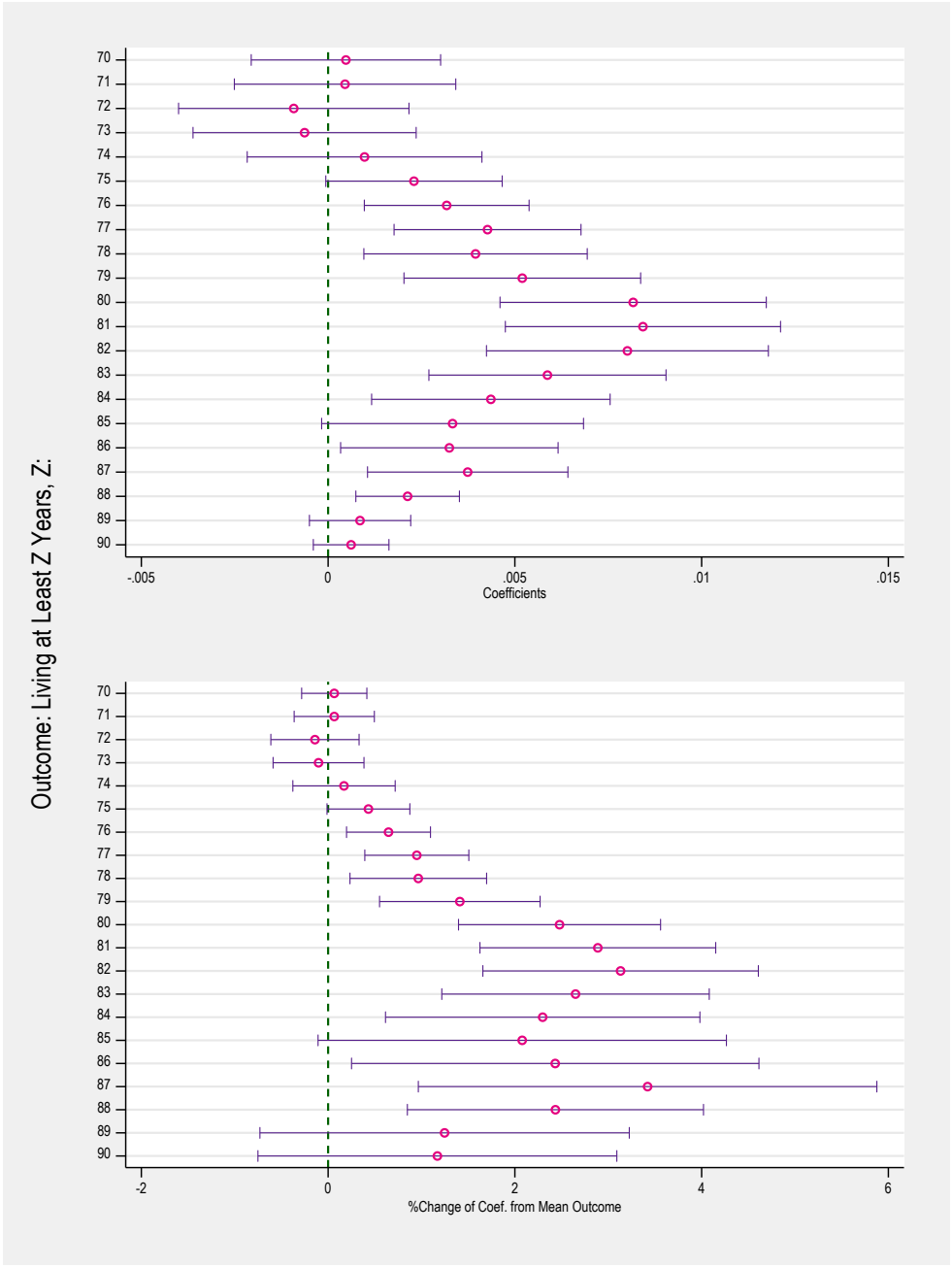

Figure 5 - The Effects of Early-life Exposure to County Health Departments and Living beyond Various Survival Age Thresholds

Notes. The figure reports point estimates and 95% confidence intervals from regressions in which the outcome is an indicator equal to one if the individual lived at least to age Z, where Z ranges from 70 to 90. The top panel reports coefficient estimates, and the bottom panel reports the percentage change implied by each coefficient relative to the mean of the corresponding outcome. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Standard errors are clustered at the county level. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.

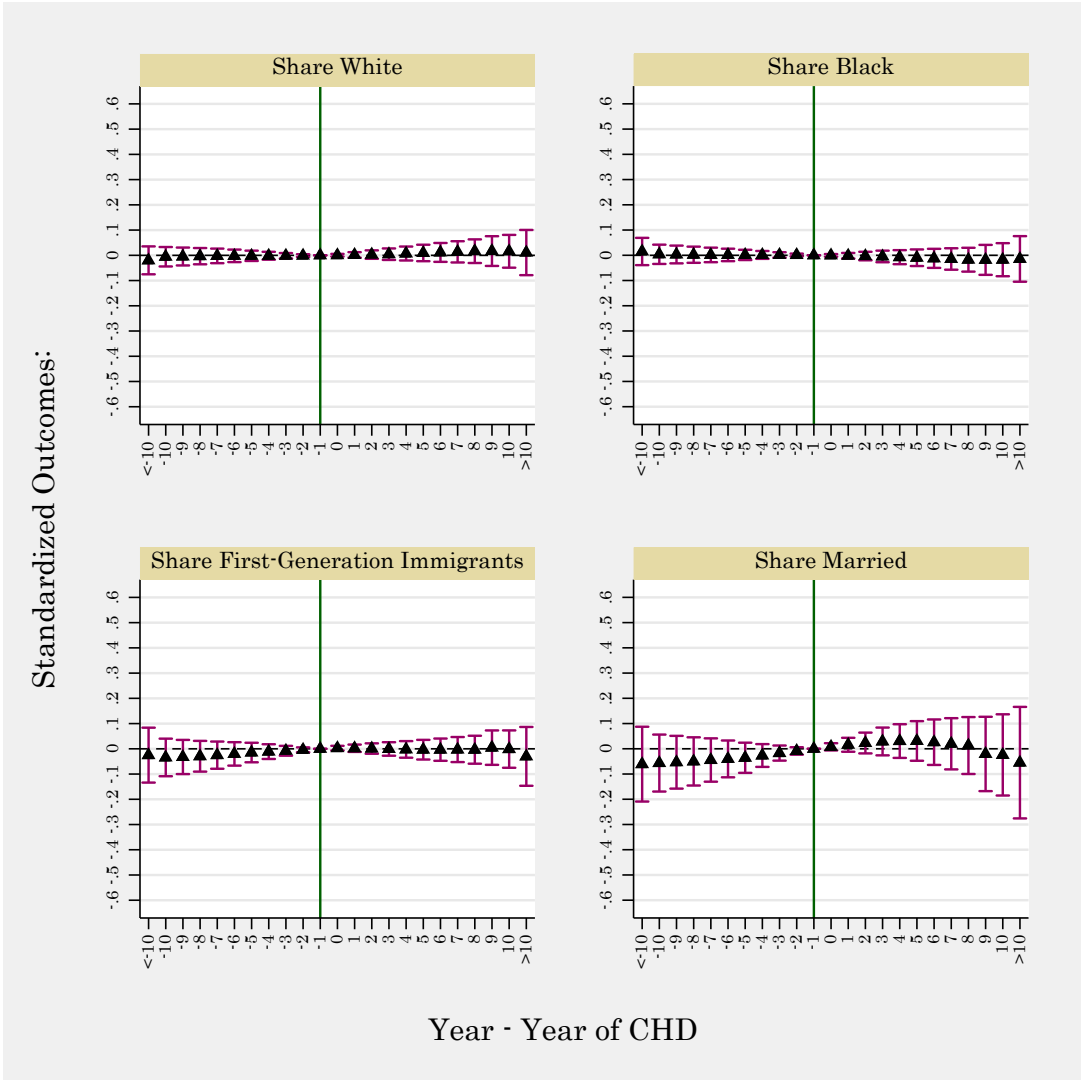

Figure 6 – Event Study Analyses to Examine the Evolution of County Outcomes in Different Years Relative to the Year of CHD Opening

Notes. The figure reports point estimates and 95% confidence intervals from county-level event-study regressions examining the evolution of county characteristics around the year of CHD opening. Outcomes are standardized to have mean zero and standard deviation one. The x-axis reports years relative to the CHD opening year, with negative values indicating years before the opening and positive values indicating years after the opening. The omitted reference period is the year immediately before the CHD opening. Standard errors are clustered at the county level. All regressions include county fixed effects and year fixed effects and are weighted by county population.

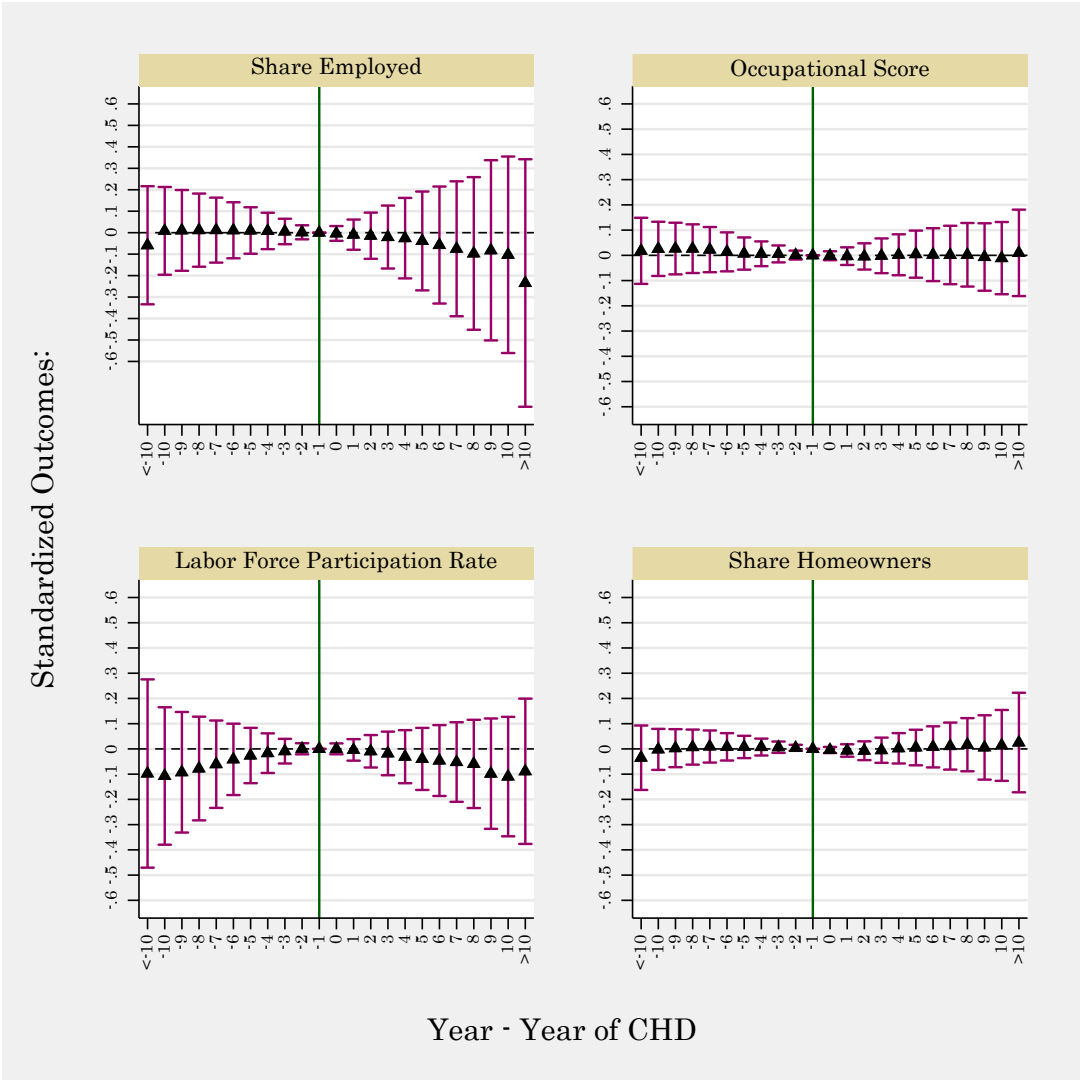

Figure 7 – Event Study Analyses to Examine the Evolution of County Outcomes in Different Years Relative to the Year of CHD Opening

Notes. The figure reports point estimates and 95% confidence intervals from county-level event-study regressions examining the evolution of county characteristics around the year of CHD opening. Outcomes are standardized to have mean zero and standard deviation one. The x-axis reports years relative to the CHD opening year, with negative values indicating years before the opening and positive values indicating years after the opening. The omitted reference period is the year immediately before the CHD opening. Standard errors are clustered at the county level. All regressions include county fixed effects and year fixed effects and are weighted by county population.

Appendix A

Appendix Table A-1 - Replicating the Main Results Using BUNMD Data

	Outcome: Age at Death (Months)	
	Death years 1988 – 2007	Death years 1970 – 2007
	(1)	(2)
Exposure	1.00345***	.87235***
	(.24127)	(.33019)
Observations	5181577	5689235
R-squared	.65742	.51895
Mean DV	883.521	861.874

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The table replicates the baseline specification using the Berkeley Unified Numident Mortality Database, which provides county-of-birth information and broader mortality coverage than the linked CenSoc-Numident–1940 Census sample. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Column 1 restricts the sample to deaths occurring between 1988 and 2007; column 2 uses deaths occurring between 1970 and 2007. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-2 - Evaluating Potential Truncation Bias

	Outcome: Age at Death (Months)						
	Females, Numident, Death Years 1988-2005	Males, Numident, Death Years 1988-2005	Males, DMF, Death Years 1988-2005	Males, DMF, Death Years 1975-2005	Males, DMF, Death Years 1975-2011	Males, DMF, Death Years 1975-2011, Birth Years 1910-1930	Males, DMF, Death Years 1975-2023
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Exposure	.79336***	1.08972***	.84179**	1.05431***	2.72863***	2.34868***	4.46877***
	(.24269)	(.27039)	(.34427)	(.39354)	(.49068)	(.3631)	(.68679)
Observations	1093511	1054123	908142	1453662	1550815	982092	1596247
R-squared	.7244	.66001	.72456	.42549	.35807	.15292	.29191
Mean DV	906.116	870.775	891.888	849.348	858.081	872.284	864.249

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Columns 1–2 use the CenSoc-Numident data for female and male deaths occurring between 1988 and 2005. Columns 3–6 use the Death Master File for male deaths under alternative death-window restrictions: 1988–2005, 1975–2005, 1975–2011, and 1975–2023. The comparison across columns evaluates whether the estimated effects vary with the mortality source, gender composition, and length of the observed death window. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender, where applicable. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-3 - Heterogeneity by CHD Opening Years

	Outcome: Age at Death (Months)			
	CHD opening year < 1925 Numident (1988-2005)	CHD opening year ≥ 1925 Numident (1988-2005)	CHD opening year < 1925 DMF (1975-2023)	CHD opening year ≥ 1925 DMF (1975-2023)
	(1)	(2)	(3)	(4)
Exposure	1.0821***	.45825	2.52858***	-.48286
	(.36972)	(.34375)	(.75069)	(.76494)
Observations	1107874	1039760	818978	777269
R-squared	.69881	.69809	.30859	.24967
Mean DV	878.417	902.980	853.905	879.969

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Columns 1–2 use the CenSoc-Numident data for deaths occurring between 1988 and 2005 and split the sample by whether the county's CHD opened before 1925 or in/after 1925. Columns 3–4 use the Death Master File for deaths occurring between 1975 and 2023 and apply the same CHD-opening-year split. This table evaluates whether estimated effects differ by the timing of CHD adoption and whether such heterogeneity is sensitive to mortality data source and death-window coverage. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-4 - Examining the Effects across Various Ages at Exposure

	Outcome: Age at Death (Months), Subsamples									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Age at Exposure ≤ 1	.52328***									
	(.19208)									
Age at Exposure ≤ 2		.63625***								
		(.14323)								
Age at Exposure ≤ 3			.60715***							
			(.18489)							
Age at Exposure ≤ 4				.90867***						
				(.19207)						
Age at Exposure ≤ 5					1.04696***					
					(.19206)					
Age at Exposure ≤ 6						.76753***				
						(.22016)				
Age at Exposure ≤ 7							.45777**			
							(.21032)			
Age at Exposure ≤ 8								.58716*		
								(.29888)		
Age at Exposure ≤ 9									.23961	
									(.27)	
Age at Exposure ≤ 10										-.02208
										(.30925)
Observations	2147634	2147634	2147634	2147634	2147634	2147634	2147634	2147634	2147634	2147634
R-squared	.70244	.70244	.70244	.70244	.70244	.70244	.70244	.70244	.70244	.70244
Mean DV	887.680	887.680	887.680	887.680	887.680	887.680	887.680	887.680	887.680	887.680

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. Each column estimates a separate specification using an alternative cumulative age-at-access cutoff. For example, column 1 defines the treatment variable as an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or by age 1, while column 4 corresponds to the baseline definition of access in utero or by age 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. All regressions include county fixed effects, birth-year fixed effects, individual controls, family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-5 - Exploring Endogenous Merging between Death Records in the Final Sample and the Original Population in the 1940 Census

	Outcome: Successful Merging between Final Sample Records and the Original Population in the 1940 Census		
	(1)	(2)	(3)
Exposure	-.01224	-.00983	-.00776
	(.00956)	(.00814)	(.00495)
Observations	22782844	22782844	22782844
R-squared	.02448	.02687	.02711
Mean DV	0.096	0.096	0.096
County FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Individual/Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is an indicator equal to one if an individual from the original 1940 Census population is successfully linked into the final mortality sample, and zero otherwise. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. These regressions test whether early-life access to CHDs is systematically associated with the probability of appearing in the final linked mortality sample. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-6 - Endogeneity of Data Linking Process across Cohorts

	Outcome: Successful Merging between Final Sample Records and the Original Population in the 1940 Census, Subsample:	
	Birth Cohort < 1925	Birth Cohort ≥ 1925
	(1)	(2)
Exposure	-.00841	-.00028
	(.00521)	(.0025)
Observations	13394013	9388831
R-squared	.03558	.0148
Mean DV	0.112	0.083
County FE	✓	✓
Birth Year FE	✓	✓
Individual/Family Controls	✓	✓
County Controls	✓	✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is an indicator equal to one if an individual from the original 1940 Census population is successfully linked into the final mortality sample, and zero otherwise. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. These regressions test whether early-life access to CHDs is systematically associated with linkage into the final mortality sample across cohort subsamples. All regressions include county fixed effects, birth-year fixed effects, individual/family controls, and county controls. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-7 - Exploring Spillover Effects of Neighboring Counties' CHD

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Own-County Exposure to	.89793***	.90832***	.89757***
CHD	(.22946)	(.23463)	(.20218)
Neighboring-County	-.112	-.11404	-.05546
Exposure to CHD	(.07053)	(.07042)	(.04948)
Observations	2146863	2146863	2146863
R-squared	.70239	.70243	.70244
Mean DV	887.679	887.679	887.679
County FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Individual Controls	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. Own-county access is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. Neighboring-county access is an analogous indicator based on CHD openings in counties adjacent to the individual's county of birth/childhood. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-8 - Robustness to Restricting to Observations with County of Birth Reported on Numident Records

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Exposure	.89922***	.89307***	.80568***
	(.28712)	(.28135)	(.27468)
Observations	1408558	1408558	1408558
R-squared	.69438	.69442	.69444
Mean DV	884.270	884.270	884.270
County FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Individual Controls	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The table replicates the baseline specification after restricting the sample to individuals whose county of birth is directly reported in the Numident records, rather than inferred from cross-census linkage or later residence. The treatment variable is an indicator equal to one if a county health department had opened in the individual's directly reported county of birth while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-9 - Robustness to Including Counties that Experienced County Health Closures

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Exposure	.78999***	.77475***	.84974***
	(.15892)	(.15986)	(.16463)
Observations	2884220	2884220	2884220
R-squared	.69902	.69907	.69908
Mean DV	894.815	894.815	894.815
County FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Individual Controls	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the county level, are reported in parentheses. The outcome is age at death measured in months. The table replicates the baseline specification after including counties that experienced CHD closures, which are excluded from the main analytical sample. The treatment variable is an indicator equal to one if a county health department had opened in the individual's county of birth/childhood while the individual was in utero or between ages 0 and 4. The estimates should be interpreted as intent-to-treat effects of access to CHD services, rather than effects of individual utilization. Individual controls include indicators for race and gender. Family controls include maternal education and paternal socioeconomic status. County controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by county population.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table A-10 - Exploring Mechanisms: Effects on Contemporaneous Disease Mortality

	Outcomes, Death Rate per 100k, Cause:			
	Typhoid	Malaria	Smallpox	Measles
	(1)	(2)	(3)	(4)
Share of Counties with Health Department	-5.387***	-.192	-.18	-1.245
	(1.991)	(1.364)	(.372)	(1.347)
Observations	1109	1109	1109	1109
R-squared	.9	.902	.341	.551
Mean DV	9.297	2.844	0.333	6.106
	Scarlet fever	Whooping cough	Diphtheria	Influenza
	(5)	(6)	(7)	(8)
Share of Counties with Health Department	.787	-.878	1.362	2.586
	(1.425)	(.908)	(2.493)	(6.108)
Observations	1109	1109	1109	1109
R-squared	.777	.727	.896	.897
Mean DV	4.002	7.557	11.270	36.596
	Meningitis	Diabetes	Circulatory	Pneumonia
	(9)	(10)	(11)	(12)
Share of Counties with Health Department	-2.325	.887	-24.313**	.852
	(2.047)	(.663)	(9.808)	(9.892)
Observations	1109	1109	1109	1109
R-squared	.918	.958	.969	.893
Mean DV	6.112	18.034	187.794	109.640

Notes. Standard errors, clustered at the state level, are reported in parentheses. The outcomes are state-level cause-specific death rates per 100,000 population. The treatment variable is the share of counties in the state with an active county health department in a given year. Columns 1–12 report separate regressions for the cause of death listed in each column heading. These regressions examine whether the expansion of CHDs is associated with contemporaneous changes in mortality from waterborne, vector-borne, infectious, and chronic causes of death. All regressions include state fixed effects, year fixed effects, and state-level controls. State controls include the shares of different occupation groups, shares of different age groups, labor force participation rate, literacy rate, average family size, average socioeconomic index, and average occupational income score. Regressions are weighted by state population.
*** p<0.01, ** p<0.05, * p<0.1.