The Impact of Medicare's Introduction on Life Expectancy[*]

Andrew Goodman-Bacon
Federal Reserve Bank of Minneapolis and NBER

Adriana Lleras-Muney
UCLA and NBER

Joseph Price
Brigham Young University and NBER

Dahai Yue
University of Maryland, College Park

PRELIMINARY AND INCOMPLETE – DO NOT CITE OR QUOTE

(Draft, 03/17/2025)

Abstract

This paper estimates causal effects of the introduction of Medicare on mortality rates and life expectancy of the program's early recipients. We construct a new dataset of more than 18 million individuals observed in the 1940 census linked to a death record in the FamilyTree database at FamilySearch. We identify Medicare's average treatment effects on log period mortality rates using three pre-specified approaches: a design based on a simple theoretical model of cohort mortality, an interrupted time-series design, and a staggered difference-in-differences design. All three sets of results show that Medicare increases life expectancy at age 55 for men born between 1885 and 1915 by 0.74 years on average, but has negligible effects for women. Medicare's effects are larger for cohorts with more potential years of exposure and for subgroups with less education or lower income in 1940.

I. Introduction

Today, almost all countries in the world provide some form of public health insurance. Alongside financial protection, these programs aim to maintain population health by providing access to basic health care. Older people account for the largest portion of public health insurance costs because the incidence and severity of medical problems increase with age. An

[*] Research reported in this publication was supported by the National Institute on Aging of the National Institutes of Health under Award Number R21AG080416. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

important justification for this spending thus hinges on how well it supports medical care consumption among older people and the efficacy of that care.

Yet, estimating causal effects of health insurance on the health of elderly recipients has proved difficult. One reason is the lack of variation that can identify these effects in countries with universal coverage, but even in the United States, where universal public health insurance under Medicare begins at age 65, studies of health insurance typically only find effects for high-need groups. For instance, one-year mortality falls sharply at age 65 among people with urgent hospital admissions (Card et al., 2009), but not in the overall population Card et al. (2004) nor Polsky et al. (2009). Huh and Reif (2017) find that Medicare's prescription drug coverage lowers cardiovascular mortality rates at age 66 by increasing drug spending among near-poor people. [2]

Another challenge is that none of these studies observes longer-term mortality rates, when Medicare's effects may be largest. Theoretical models (Dalgaard and Strulik, 2014; Grossman, 1972; Lleras-Muney and Moreau, 2022) and empirical studies (Leinonen et al., 2001; Levinsky and Schiff, 2021) document that health is a stock that accumulates and deteriorates slowly. Therefore, the benefits of insurance, which provides access to preventive care, chronic care management and diagnostic tools, may not materialize for many years, especially among relatively healthy people. Indeed, recent evidence tracking the impact of health insurance on children suggests that there are large long-term impacts of health insurance provision. [3] Whether this evidence applies to adults who are no longer growing is unknown. Moreover, these models show that while insurance can theoretically affect health across the population, it is only likely to change mortality for sub-populations near the death threshold.

This paper estimates causal effects of the introduction of Medicare on the mortality rates and life expectancy of the program's early recipients. Medicare began providing health insurance for hospital stays and physician visits to (almost) all Americans over the age of 65 in July 1966. Only about half of this population held any kind of health insurance in the early 1960s and out-

[2] These findings echo patterns from a literature on public health insurance for non-elderly people. For instance, Medicaid, the public insurance program for the poor, reduces mortality rates for children and adults, but only when it covers those with low incomes or high health needs (Borgschulte and Vogler, 2020; Currie and Gruber, 1996a, b; Goodman-Bacon, 2018, 2021; Graves et al., 2020; Miller et al., 2021; Wherry and Meyer, 2016).

[3] Goodman-Bacon (2018, 2021) finds that the introduction of Medicaid resulted in immediate reductions in US infant and child mortality followed by reductions in mortality and disability up to 50 years later. Other work also finds evidence that Medicaid expansions during childhood led to better self-reported health at later ages, fewer chronic diseases and related hospitalizations, and lower mortality (Brown et al., 2020; Currie et al., 2008; Miller and Wherry, 2019; Thompson, 2017; Wherry and Meyer, 2016; Wherry et al., 2018).

of-pocket spending was high even among the insured. As a consequence, elderly Americans often forewent potentially life-saving medical care: almost half of respondents over age 65 in one 1963 survey said they would avoid doctors *and* hospitals "whenever possible" (CHAS and NORC 1984). Soon after Medicare's passage, however, the use of basic medical care among older Americans increased dramatically, while extreme health spending fell (Finkelstein and McKnight 2008).

To measure the mortality experience of cohorts affect by this historic change, we construct a new dataset of more than 18 million white people observed in the 1940 census who can be linked to a death record. The age profiles of mortality in our data closely match the Social Security Administration's cohort life tables, but they also include covariates that allow us to investigate *heterogeneity* in ways that are not possible using existing data sources. These data allow us to map causal effects on period/age mortality rates into a *long-run* outcome: cohort life-expectancy conditional on surviving to age 55.

We generate hypotheses about how Medicare may have affected its recipients and by how much using the theoretical model of cohort mortality in Lleras-Muney and Moreau (2022). We first show how the dynamics of Medicare's effect on log mortality rates provide evidence on how it operated: changes to the rate of health investments or the depreciation of health reduce the slope of the log-mortality-age profile, which changes to the variance of health shocks or the death threshold reduce mortality immediately but lead to steeper slopes thereafter. Moreover, the model implies that Medicare should have had larger effects on the morality of less healthy subgroups who are on average closer to the death threshold.

Next, we pre-specify three research designs that identify Medicare's average treatment effects on mortality under distinct identifying assumptions.[4] First, we estimate the theoretical model using data from before each cohort gained Medicare eligibility, and use the parameter estimates to form counterfactual mortality rates during their post-Medicare years. This validity of this

[4] To empirically ground our design choices and still conduct valid statistical inference, we use a random 20 percent of our sample to test identifying assumptions and make key specification choices. For example, our detailed microdata allows us to assess how Medicare's effects vary by SES, but we do not know a priori how best to measure SES in order to capture relevant heterogeneity and be able to detect it. Our training sample allows us to test different ways to measure SES in the 1940 Census without inducing pre-test bias. Similarly, we can condition our analysis on a range of 1940 covariates (as well as look for other types of heterogeneous effects), and we use our training sample to select covariates and choose the best conditional estimator (Sant'Anna and Zhao, 2020). We will archive our full analysis plan (and code) and the evidence from our training sample that justifies it with the Open Science Framework before conducting our main analysis. Results in this manuscript use training data only.

strategy depends on correctly specifying a model of mortality and reliably estimating its parameters only data until at least age 65. Our second approach builds on a qualitative prediction of the model: the linearity of the age profile of log mortality rates after a certain age. Our interrupted time series (ITS) design assumes that the linear trend in log mortality at pre-Medicare ages would have continued in Medicare's absence, and uses estimates of this slope to form counterfactual mortality at older ages. This relies less on the specific structure of the model, and is internally valid as long as linearity holds. Finally, we employ a staggered difference-in-differences (DiD) design that compares changes in log mortality before and after each cohort enters Medicare to the change in mortality at the same *ages* for older comparison cohorts who gained Medicare later in life. This approach uses observed mortality changes for older cohorts to form counterfactual mortality trends (ie. a parallel trends assumption), and does not rely on any specific model assumption except for comparability across cohorts.[5]

Preliminary results from all three empirical approaches align closely and show that Medicare increases life expectancy for men at age 55 by 0.74 years on average: the effect gradually increases from zero to roughly 2 years for the birth cohorts born 1885-1910 and then decreases for the 1915 birth cohort. These effects arise from gradual changes in log age-specific mortality rates that closely match the predictions from our theoretical model. The results for women, however, are close to null as predicted by our simple theory. We also find that Medicare had greater effects on life expectancy for lower-educated subgroups, and that it did improve longevity for women with less than 8 years of education.

In addition to providing the first long-term estimates of the effects of health insurance among the elderly (which is our primary contribution), we also contribute to a literature reevaluating the health effects of President Lyndon Johnson's Great Society programs. This work includes examinations of the impacts of Medicaid (Boudreaux et al., 2016; Clay et al., 2024; Goodman-Bacon, 2018, 2021; Miller et al., 2021; Wyse and Meyer, 2023), Head Start (Ludwig and Miller, 2007), and community health centers (Bailey and Goodman-Bacon, 2015). Our findings align with these previous results: like Finkelstein and McKnight (2008), we do not find large impacts in the short term for the population as a whole, but we do find effects over time. These results also add to the growing literature that documents that the short- and long-term impacts of policies can differ quite substantially.

[5] Our DiD methods extend recent identification results and estimation techniques for staggered DiD designs to a setting with a policy that is adopted by all cohorts with staggering across ages and periods.

We also contribute to the literature estimating the impacts of Medicare. In addition to reporting the first estimates of its long-term impacts, we also document substantial heterogeneity in Medicare's causal effects over time and across sub-populations, which the more recent literature has documented (Card et al., 2008). Medicare has grown tremendously in scope, scale, and cost since 1966. Today, the program serves more than 65 million Americans at a cost of $800 billion, which is one of the largest expenditures in the federal budget. Our findings suggest that these outlays likely still confer meaningful benefits to population health.

II. Health Insurance and Health Care before and after Medicare

For decades prior to Medicare's passage, poor health had been an increasingly burdensome problem for American seniors. Private health insurance, initially developed in the 1920s, exploded after World War II. The war gave millions of soldiers new experience with regular medical care and labor negotiations during the war led to a clarification in 1953 that insurance benefits provided by employers were not taxable (Thomasson, 2003). Shifts in the demand for medical care were met with rising supply as well: innovations developed during WWII such as antibiotics, blood transfusions, and ultrasounds (Van Tiggelen and Pouders, 2003) found civilian applications and the federal hospital subsidies under the 1946 Hill-Burton act greatly expanded capacity (Chung et al., 2017). These developments were good for population health, but created an intractable affordability problem for seniors.

People over age 65 generally did not get insurance through employers or on the individual market. Individual insurance markets offered medically underwritten policies, making them prohibitively expensive to older people with high expected costs. Blue Cross Blue Shield tried to offer affordable plans through "community rating," but adverse selection pushed up premiums (Thomasson, 2004). Scattered survey data from the early 1960s suggest that about half of people over age 65 held any kind of health insurance policy. The share was 40 percent in one 1960 survey (Morgan et al., 1962), 57 percent in the 1963 Survey of Health Services Utilization and Expenditure (CHAS and NORC 1984), 52 percent in the 1963 Social Security Survey of the Aged (Epstein and Murray 1967), and 54 percent (for hospital insurance) in the 1963 National Health Survey (NCHS 1964; table 6).

Underinsurance added to affordability problems. At the time, "insurance" primarily meant hospital and sometimes surgical policies (many surveys, for instance, only asked about these two forms of coverage). Even for hospital costs, insurance plans provided only partial

coverage. Just over half of elderly insured people with hospital stays in 1962 reported that insurance covered over half their bill (NCHS 1964, table 11.13). Underinsurance in terms of dollars was even more severe: among Social Security beneficiaries in 1966, private insurance covered less than one-fifth of their medical costs (Loewenstein 1971; table 10).

Without the financial protection of insurance, "[c]hoices for uninsured or underinsured elderly patients needing hospital service were to spend their savings, rely on funding from their children, seek welfare…or hope for charity from hospitals" (Stevens, 1996). Those who did seek care often faced large out of pocket spending (Epstein and Murray 1967, Table 10.11). A small number of low-income elderly people had their care financed by welfare departments or lived in state institutions.[6] Charity providers often provided low quality care, had long wait times, and could refuse service or pursue aggressive collection practices (Stevens, 1989). Therefore, in the decades leading up to Medicare's passage medical care was improving and growing in cost, but the mechanisms by which prime-age Americans paid for that care were out of reach for older people.[7]

Statistics on the use of basic health care services reflects these problems. Figure 1 plots the number of inpatient hospital days per 1,000 people in two ages groups (50-64 and 65 and older) drawn from a range of surveys and administrative data sources covering the years 1936 through 1975. Prior to 1966 these data often show small, if any, gap in the intensity of hospital use for older versus younger people, despite the measurably poorer health of the elderly.

In response to these conditions, the Johnson administration made Medicare the signature component of the 1965 Social Security Act amendments. At that point, legislative debates over public health insurance in the US were 50 years old, and a Medicare bill pushed by the Kennedy administration was narrowly defeated in 1962. After the Democratic landslide in 1964, however, the newly elected President Johnson took up the issue of health insurance for the elderly once more. The administration proposed a universal hospital insurance program for the elderly with no premiums and minimal cost-sharing. Congressional Republicans preferred a

[6] These programs included state Old Age Assistance programs, which paid for some medical costs since 1950, or a slightly more generous but short-lived program called Medical Assistance for the Aged, which mainly operated in a handful of large states. These programs were not widespread because of their means test and because they had a legal claim on the resources of a recipient's children and so many people were reluctant to accept welfare medical care.

[7] In 1959, a Senate subcommittee on aging held hearings during deliberations on a health insurance bill introduced by Senator Aime Forand. One staffer reported that "The old folks lined up by the dozen ever place we went…and they didn't talk much about housing or recreational centers or part time work. They talked about medical care" (quoted in Starr 1982, pg. 368).

means-tested system run by the states, and the American Medical Association proposed subsidizing the purchase of private fee-for-service insurance plans. Despite the long history of public health insurance bills, "the outcome of the debate over Medicare was by no means predictable" (Stevens pg. 47).

Ultimately, the legislation that created Medicare combined all three proposals.[8] Medicare had two insurance components modelled after the Blue Cross Blue Shield plans that provided most insurance coverage to seniors. Part A provided compulsory hospital insurance for people over 65 with a $40 annual deductible and no cost sharing for stays under 60 days (Gornick et al., 1996). Part B offered optional medical and surgical insurance with 20 percent coinsurance for a $3 per month (Gornick et al., 1996). (The Republican's means-tested program became Medicaid.) Upon signing it into law, President Johnson declared "no longer will older Americans be denied the healing miracle of modern medicine" (Peters and Woolley, 1999).

The program officially began on July 1, 1966. About 93% of individuals eligible for hospital insurance also enrolled in Part B (West, 1971). Medicare's introduction dramatically increased insurance coverage for the elderly; the percent of seniors with hospital insurance and surgical insurance rose from 50 percent in 1963 to 95 percent in 1968 (National Center for Health Statistics, 1963, 1968), though Finkelstein (2007) estimates an even larger 75 percentage point increase in "meaningful health insurance". It also significantly lowered the cost of health care for seniors. One survey of Social Security recipients found that in Medicare's first year the patients paid about 47 percent of medical costs compared to 77 percent in the year prior (West, 1971) and Finkelstein and McKnight (2008) show that extreme out-of-pocket spending fell. Figure 1 shows that sharp changes in the ability to pay for care corresponded with sharp changes in the use of care. After remaining fairly close for at least 30 years, the annual hospital utilization of people over 64 jumped up after 1966 to about 3.5 days per person, while it stayed flat for people between age 50 and 64.

Today this remains true: Individuals on Medicare use health care at much higher rates than those without (Card et al., 2008) and they have lower medical expenditure risk (Barcellos and

[8] Even this was a surprise: "On March 12, 1965 [Senate Finance Committee Chair, Wilbur] Mills stunned [Undersecretary of the Department of Health, Education and Welfare, Wilbur] Cohen by asking if it would be possible to amalgamate a compulsory hospitalization program, a voluntary medical insurance program, and an expansion of coverage for the indigent" (Cunningham and Cunningham pg. 143).

Jacobson, 2015).[9] As of 2025, Medicare enrolled 68.2 million people (89.4% of which are over the age of 65) and cost $1028.9 billion (about 3.7% of US Gross Domestic Products), accounting for 21% of all US health expenditures (CMS 2025).[10] Very few people over the age of 65 are not enrolled (Moon 1996), and 92% of the over 65 population rates Medicare's performance highly (Lopes et al., 2023). As a result, many have proposed that Medicare serve as a blueprint for a larger health insurance program for the entire US population.

III. Potential Effects of Medicare

We use the model developed by Lleras-Muney and Moreau (2022) to structure our hypotheses about how Medicare's introduction may have affected mortality and longevity among its initial cohorts. Individuals are endowed with an initial level of health H, which reflects parental health and genes, as well as *in utero* conditions, and is normally distributed in the population, $H \sim N(\mu_H, 1)$, where the standard deviation has been normalized to one. Thereafter, health evolves at each age a as follows: $H_a = H_{a-1} - \delta a^{\alpha} + I + \varepsilon_a$. It increases dues to the constant per-period average health investments I; it deteriorates with age at an increasing rate (given by the aging function δa^{α}); and it receives stochastic shocks ε_a, which are also assumed to be normal $N(0, \sigma)$. Note that this additive law of motion for health means that ε_a captures both direct health shocks and random variation in investments. Individuals die when their health falls below a certain threshold, $\underline{H}$, which is normalized to zero. In addition, a share κ randomly die of accidental deaths each period after age 16. This model, which ignores optimization, has been shown to provide an excellent description of the mortality of populations from birth to death in stationary environments. It also delivers log mortality curves that are linear in age after a certain age, consistent with observations by Gompertz (1825).

The simplicity of the model, which has just seven parameters $\{\mu_H, \delta, \alpha, I, \sigma, \kappa, \underline{H}\}$, defines just a handful of channels through which Medicare may change mortality. Medicare may affect health investments (I) through both a price effect and an income effect arising from its health

[9] Since its inception there have been a few notable changes in the program. Medicare was further expanded to younger people with disability status and those with end-stage renal disease in 1973. Since 1999, the elderly may opt out of the traditional fee for service (FFS) plan and enroll in a Medicare managed care plan – this is known as Part C. Finally in 2006 Medicare implemented a new voluntary insurance product (Part D) to cover prescription drugs expenditures.

[10] https://data.cms.gov/summary-statistics-on-beneficiary-enrollment/medicare-and-medicaid-reports/medicare-monthly-enrollment. And https://www.cms.gov/data-research/statistics-trends-and-reports/national-health-expenditure-data/nhe-fact-sheet

insurance structure.[11] It may also lower the variance of health or health investment shocks (σ): Medicare can be viewed as insurance, ensuring that individuals experience less severe health shocks by transferring resources from good states (when individuals pay for premiums) to bad states when they suffer costly health shocks that are now covered by insurance. This would result in lower health in good times but higher health after health shocks. If primary care or better diagnoses lower the likelihood of the onset of fatal conditions, then the incidence of negative health shocks may fall. But Medicare also reduces the financial burden of health shocks, which may help to maintain health investments which are also reflected in ε_a. An alternative view of improved access to preventive and diagnostic care is that it may slow the rate of aging (δ or α). Finally, by leading to more and better acute medical care, Medicare may lower the "threshold for dying" ($\underline{H}$).[12]

Figure 2a plots a simulated effect on the position and slope of a cohort's Gompertz curve from changes to each of these parameters. A ten percent increase in health investments (panel a) or a 5 percent decrease in the depreciation rate (panel d) at age 65 simply make the Gompertz curves flatter; log mortality rates do not shift immediately, but they do rotate down so that mortality rises more slowly as people age. By contrast, a decline in the variance of health shocks from 1 to 0.6 (panel b) or the threshold for dying from 0 to -1 (panel c) both lead to immediate downward shifts in log mortality rates and then steeper slopes thereafter. In the long-run, however, log mortality rates are higher under a smaller shock variance because while negative shocks are less negative, positive shocks as also less positive.

The difference between the two Gompertz curves from **Figure 2a**, which we plot in **Figure 2b**, are the treatment effects that we would expect to observe for a given cohort in an idealized experiment that compares the mortality of an affected and unaffected population. These predictions motivate our empirical approach because they show that dynamic treatment effects *within a cohort*, specifically the intercept and the slope of log mortality after cohorts gain Medicare eligibility, are crucial for making inferences about the program's effect on life expectancy as well as the (model-based) mechanisms by which these effects arise.

[11] The cohorts we study also enjoyed a net transfer from Medicare thanks to its pay-as-you-go structure. Medicare is financed from payroll taxes paid by mostly ineligible workers, so the initial generations gained coverage without having paid in. That transfer may increase health investments and thus health.

[12] Medicare cannot affect initial health, μ_H, since it begins at age 65, and we assume that it does not affect mortality from accidental causes, κ.

The literature on Medicare and mortality, however, has generally not estimated these kinds of dynamic effects. Contemporaneous studies using the age-discontinuity in Medicare eligibility necessarily identify causal effects *at* age 65, and then only find mortality reductions for severely ill patients (Polsky et al. 2009, Card et al. 2004 Card et al. 2009).[13] Research on the extension of Medicare to people with end-stage renal disease (in 1972) and the addition of prescription drug coverage (in 2006) find significant reductions in mortality, but only measure between 4 and 6 years of post-reform (Andersen, 2018; Dunn and Shapiro, 2019; Huh and Reif, 2017).

Historical studies, on the other hand, typically estimate dynamic effects on age-specific mortality, which does not reflect the accumulation of health within a cohort. Finkelstein and McKnight (2008) compare mortality between people ages 65-74 and 55-64, and concluded that Medicare had no discernible impact on the elderly aggregate mortality rates in first ten years.[14] In contrast, a working paper Chay et al. (2010) uses five- instead of ten-year age groups and finds sharp convergence between elderly and near-elderly mortality after 1966. A key limitation of these studies is their inability to disaggregate Vital Statistics data into subgroups for which Medicare may have had larger effects.

Randomized trials of the kinds of care that Medicare covered, however, do find long-run effects within cohorts. For example, the MRFIT randomized intervention, which aimed at lowering deaths from cardiovascular disease by lowering cholesterol, lowering blood pressure, and quitting smoking had immediate effects on health and on earnings (Stephens Jr and Toohey, 2022), but mortality declines were only detected after 10 years (Multiple Risk Factor Intervention Trial Research Group, 1982, 1990, 1996). Medicare also improves access to diagnostic and preventive services which could also benefit individuals with a delay (for evidence on colonoscopies see Niikura et al., 2017). Health insurance has also been shown to improve health behaviors and mental health in the short run (Baicker et al., 2013), which could lower mortality eventually.[15]

[13] Lichtenberg (2002) finds a significant break in mortality at age 65, but Card et al. (2009) argue that it is an artifact of the interval-smoothing procedures used to construct the data.
[14] They also find no evidence of a negative relationship between elderly mortality changes and regional pre-Medicare uninsurance rates, a point we discuss below.
[15] Later cohorts may have shifted their medical care use in anticipation of their Medicare eligibility. We discuss anticipation explicitly when we introduce our empirical strategy.

In sum, the model predicts that Medicare could have large long-term effects, some of which may not be detectable in the short run and which empirical approaches used thus far may have missed. Additional empirical evidence suggests that the benefits of medical care could increase overtime at least when measured by mortality rate declines. We estimate these dynamic benefits using various approaches.

IV. Data Sources and Outcome Measures

IV.A. Data Sources

Census-Tree data. Our primary individual-level data come from the 1940 full-count census linked to information available from FamilyTree at FamilySearch, referred to hereafter as the Census-Tree. The 1940 full-count records, released on April 2, 2012 are publicly available on the Integrated Public Use Microdata Series (IPUMS) website. FamilySearch resources "help millions of people around the world discover their heritage and connect with family members" (FamilySearch, 2025). It has a website that provides access to historical record collections and a wiki-style platform, called Family Tree, through which individuals can gather information about their ancestors. In 2020, there were over 1.2 billion individual profiles on Family Tree and over 12 million registered users, making it one of the largest genealogy websites in the world (Price et al., 2021). FamilySearch profiles contain significant information about deceased individuals, including sex, race/ethnicity, date of birth, place of birth, date of death, and state of death along with sources attached to support the validity of this information. We link the census records with profiles on the Family Tree using a matching file that FamilySearch shares with us.

Social Security Administration cohort tables. These tables track the mortality and life expectancy of men and women in the US for cohorts born in 1900 and thereafter. These data are only available at the aggregate (national) level by sex. These national-level cohort tables are constructed using death counts by five-year age groups from published vital statistics data pertaining to death registration deaths only, matched to population estimates constructed by the Census Bureau based on Census data (for 1900-1967). Because the vital registration system of the US was not complete until 1933 when all states had joined, the SSA data is not necessarily representative of the nation for these years.[16] We use these data to assess the quality

[16] These are available here: https://www.ssa.gov/oact/HistEst/CohLifeTables/2020/CohLifeTables2020.html

of the individual data and to estimate the structural model which requires data from birth to death.

IV.B Sample Construction and Sample Selection

We linked the 1940 full-count census which includes 132 million people to the Family Tree. We obtain a sample of the 49 million individuals that are linked – this process we followed to construct the sample is described in **Figure A1**. We then restrict attention to individuals born between 1885-1915 – these cohorts are estimated to be extinct and so we should observe the ages at death for all individuals. We further restrict our main analysis to whites. Their coverage in the Family Tree database is thought to be more complete than for other race groups (Price et al., 2021) and because Medicare affected access to medical facilities for Black Americans in a much different way than it did for white Americans (Anderson, Charles, Rees *forthcoming*, David Barton Smith 2016). Finally, we restrict attention to individuals without missing data regarding their ages at death (they are not missing information on birth dates or death dates on the family tree).

The final data set includes 18,485,813 individuals, accounting for 32.2% of our target population: white 1940 Census respondents born between 1885 and 1915.[17] The data includes information on individuals' year of birth and year of death from the FamilyTree and various demographics (e.g., sex, race, ethnicity) and socioeconomic variables (e.g., education) observed in the 1940 Census.

IV.C Outcome measures

Our sample includes people, i, who belong to a birth cohort $C_i = c \in \{1885,1915\}$ and die at discrete age $Y_i = a \in \{40,110\}$. Ages, periods, and cohorts are related by the identity $a = t - c$, so these deaths necessarily occur in time periods $t \in \{1925,2025\}$. We observe 1940 covariates X_i, and stratify our analysis by sex so all quantities in this section implicitly apply to men and women as measured in 1940.

[17] We rely on the year of birth recorded in the Family Tree in our analysis because the 1940 Census does not ask birth year and only reports age. Specifically, the question is, "Enter the age of the person at his last birthday before 12:01 a.m., April 1, 1940." As a result, the imputed birth year might be different from the actual birth year depending on whether the respondent's birthday is before April or not. However, the birth year variable from Family Tree was scrapped because people would have had the chance to find birth records and make corrections.

The age-specific mortality rate for cohort c equals the probability of dying at age a conditional on surviving to age a:

$$\tilde{q}_{c,a} \equiv \frac{P(Y_i = a|C_i = c)}{P(Y_i \geq a|C_i = c)} = \frac{F_c(a) - F_c(a-1)}{1 - F_c(a)} \qquad (1)$$

Where $F_c(\cdot)$ is the c.d.f. of Y_i among members of cohort c, meaning that the dynamics of $\tilde{q}_{ca}$ come from the shape of $F_c(\cdot)$.[18] We work with log mortality rates $q_{c,a} \equiv \ln(\tilde{q}_{c,a})$, and we restrict our analysis to mortality rates up to age 90 – measurement error in old ages makes mortality rates for these ages very noisy.

IV.D Representativeness and Quality of Census Tree Data

A crucial concern with the data we create is whether it is representative of the population we aim to study. As **Table 1** shows our sample (first column) appears to be representative of the target population (Whites) in the 1940 census (last column) in terms of year of birth, ethnicity, veteran status, occupation score and educational levels.[19] However, it includes poorer households, fewer females (46.3% in Census Tree versus 49.8% in the 1940 Census), more married individuals (84.2% versus 72.3%), fewer people born in the Northeast region (16.9% versus 24.3%), and fewer people who resided in Northeast in 1940 (17.8% versus, 30.5%). In fact, no existing linking methods produce samples that are consistently representative of the linkable population (Bailey et al., 2020).

However, we can re-weight the sample to make it representative and address potential selection bias (Bailey et al., 2020; Lleras-Muney et al., 2022; Yue et al., 2023a). Specifically, we estimate the probability, $p(X_i)$, of a successful match among the same population (e.g., non-Hispanic Whites) between our sample and the 1940 full-count Census using a robit model, that estimates the likelihood of a match as a function of the covariates available in the 1940 census (Newson and Falcaro, 2023; Seaman and White, 2013).[20] We then reweight matched

[18] This one-year mortality rate, $\tilde{q}_{c,a}$, equals the integral of the continuous-time hazard between age a and $a+1$. Because exact dates of birth and death are not always accurately recorded, we measure mortality rates in discrete units and use corresponding life-table methods to aggregate mortality rates into life expectancy quantities.

[19] In assessing the representativeness of our Census-Tree data and constructing the inverse probability weights of matching, we have to use the birth year variable from the census because we need birth year for census respondents both on and off the family tree. To minimize measurement bias in birth year, we further restricted the Census-Tree sample to those with birth year discrepancies between (-2, 2); it drops 5.3% of the Census-Tree sample (see **Figure A1**). Thus, the restricted Census-Tree sample of 1885-1915 cohorts can be fully matched to birth cohorts 1883-1917 in IPUMS 1940 census. We then use Census reported birth year as one of the predictors in the construction of inverse probability weights of matching.

[20] We use a robit model to estimate the inverse probability weights (IPW) because it is more suitable to handle missing-at-random values without producing extremely large weights (Newson and Falcaro, 2023; Seaman and

observations by $p(X_i)^{-1}$ (rescaled to sum to one within the matched sample) so their covariate distributions match the 1940 Census. As shown in the second column of **Table 1**, the weighted statistics are fairly comparable to those from the target population in the 1940 full-count census.

To assess the quality of death information in our data we compare the unweighted age-specific mortality rates in our data with those in the SSA cohort life tables. Since SSA life tables are for all races and only available for birth cohorts 1900 and onwards, we conduct the comparison for birth cohorts 1900-1915 from all races (although we do not use all races in our analysis) broken by sex. As shown in **Figure 3**, our mortality rates are generally comparable to the SSA life tables for all the birth cohorts and for both males and females: they follow a very similar pattern by age, increasing at an exponential rate with age, though our death rates are slightly lower, particularly in old age. We hypothesize this is likely occurring because our data include fewer Blacks and fewer immigrants, groups who tend to have a higher mortality rate than whites. As a result, the expected age at death conditional on survival to age 40 computed using our data is larger than that in SSA life tables (**Figure A2**): For males, the gap is about 1.8 years for the 1900-1904 cohorts and 1.3 years for the 1905-1915 cohorts. For females, the gap is about 2 years for the 1900-1904 cohorts and 1.5 for the 1905-1915 cohorts.

V. Empirical Strategy

V.A Potential outcomes and target parameters

The objects of interest in this paper are causal parameters that describe Medicare's effect on mortality rates or functions of those parameters that describe its effect on life expectancy. To define them, we write potential outcomes as a function of two dimensions of Medicare policy: its starting year, 1966 and its minimum age of eligibility, 65. These policy variables translate to cross-cohort variation in the age and year of Medicare eligibility. Units from cohort c become treated by Medicare at time $G_t(c) = max\{c + 65,1966\} = g_t$ which occurs when they are age $G_a(c) = G_t(c) - c = max\{65,1966 - c\} = g_a$. This structure reflects the fact that the only people who first gained Medicare after age 65 necessarily got it in 1966, and the only people who first gained Medicare after 1966 necessarily got it at age 65.

The values g_t and g_a define potential outcomes for age-at-death (Rubin 1974, Robins 1986): $Y_i(g_t, g_a)$. We use $Y_i(\infty)$ to denote potential outcomes in the absence of Medicare. Potential

White, 2013). In addition, we further truncated IPW at the 99th percentile to stabilize the weights (Seaman and White, 2013).

mortality rates $\tilde{q}_{c,a}(g_t, g_a)$ have the same form as (1) but use potential instead of realized outcomes.

Our building block target parameters are average treatment effects on the treated on the log mortality rates of each cohort at each age:[21]

$$ATT(g_a, g_t, a) \equiv q_{c,a}(g_t, g_a) - q_{c,a}(\infty) \qquad (1)$$

Since Medicare "treats" all members of a cohort we take the phrase "on the treated" to refer to the specific cohorts exposed to Medicare in year g_t at age g_a. Each $ATT(g_a, g_t, a)$ approximately equals a proportional treatment effect of Medicare relative to $\tilde{q}_{ca}(\infty)$. The collection of $ATT(g_a, g_t, a)$s are *within-cohort* dynamic event-study-type effects that fully characterize Medicare's effect on longevity, but there are too many to report them individually. We summarize these parameters in three ways.

First, we report average treatment effects by event-age, $e = a - g_a$, which answer a basic program evaluation question: how did Medicare affect average mortality rates of its beneficiaries in the years after they gained eligibility?

$$ATT(e) \equiv \sum_{g_a=65}^{A} \sum_{g_t=1966}^{T} w(g_t, g_a, g_a + e) \times ATT(g_t, g_a, g_a + e) \qquad (2)$$

These are average within-cohort dynamic effects on log mortality rates as in **Figure 2b**. The weights $w(g_t, g_a, g_a + e)$ equal the share of the population observed at event-age e who are in the (g_t, g_a) cohort. For example, event-age $e = 0$ for the 1885 cohort occurs in 1966 when they are 81 years old. Therefore, their $ATT(1966,81,81)$ parameter enters into $ATT(0)$. The 1905 cohort has $e = 0$ in 1970 when they turn 65, and so their $ATT(1970,65,65)$ parameter also enters into $ATT(0)$. Moreover, because the 1885 cohort is so much older at $e = 0$ than the 1905 cohort, they would get less weight simply because more of them will have died: $w(1966,81,81) < w(1970,65,65)$. We can compute these estimates for every year after the event therefore providing an estimate of the "impulse response" over time.

The circumstances of Medicare's introduction allow us to estimate separate effects for cohorts that first gained Medicare eligibility at different ages.[22] We report this cross-cohort

[21] We use log mortality rates for several reasons. First, studying hazards is important in duration analysis to avoid conflating changes in risk with changes in the at-risk population (Wu and Wen 2022, Deaner and Ku 2024). Second, for reasons discussed below, we believe that identification of log parameters is credible, while other transformations are not (see Roth and Sant'Anna 2024).

[22] All cohorts alive today gained Medicare at age 65, so calendar age and event-age are collinear and contemporary data cannot identify effects of *gaining* Medicare at older ages.

heterogeneity by summarizing each cohort's dynamic effects with a linear function and reporting the "shift" and a "trend-break" terms:

$$ATT(g_t, g_a, a) = \delta(g_t, g_a) + \theta(g_t, g_a)(a - g_a) \quad (3)$$

Heterogeneity in $\delta(g_t, g_a)$ and $\theta(g_t, g_a)$ across g_a is relevant to policy questions about changes to Medicare's eligibility structure. The model in section III also suggests that their magnitudes provide information about how Medicare affects health production.

Finally, we use life-table methods to combine observed mortality rates and estimated counterfactuals ($q_{c,a}(\infty) \equiv q_{c,a} - ATT(g_a, g_t, a)$) to construct counterfactual mean age at death and report average effect of Medicare on life expectancy at age 55:

$$ATT_{LE}(g_a, g_t) = E[Y_i(g_t, g_a) - Y_i(\infty) | G_t(C_i) = g_t, G_a(C_i) = g_a, Y_i \geq 55] \quad (4)$$

We describe the details in our pre-analysis plan and in appendix A.X.

Both the evidence and the model of health capital accumulation discussed in section III point to substantial heterogeneity in the effects of health insurance on mortality. Our linked data uniquely allow us to target causal effects for subgroups defined by 1940 characteristics such as education, income, and geography. These are comparable to the parameters just defined, but involve mortality rates for subgroups in 1940, $\tilde{q}_{c,a}(x) \equiv \frac{P(Y_i = a | C_i = c, X_i = x)}{P(Y_i \geq a | C_i = c, X_i = x)} = \frac{F_{c|x}(a) - F_{c|x}(a-1)}{1 - F_{c|x}(a)}$.[23]

The patterns of heterogeneity across these groups are of interest per se and can also be correlated with measures of the increase in insurance coverage created by Medicare.

V.B Identification and estimation

We rely on three distinct assumptions on untreated log mortality rates to identify the $ATT(g_a, g_t, a)$ parameters: the full structure of the model from section III, a linearity assumption that the model qualitatively implies and that is often used in empirical work, and a parallel trends assumption that requires equality of mortality trends across cohorts. All three strategies require an assumption that Medicare does not affect mortality before a cohort gains eligibility. This kind of anticipatory behavior could arise because people anticipate their own

[23] We will write these as $ATT(e; x)$, $ATT_b(e; x)$, $\delta(g_t, g_a; x)$, $\theta(g_t, g_a; x)$, and $ATT_{LE}(g_a, g_t; x)$. Note that these parameters aggregate up to the unconditional ones based on each group's sample share in 1940 as well as the way that they affect the composition of the surviving population.

eligibility, because Medicare's general equilibrium effects alter the outcomes of not-yet-eligible people, or because some subpopulations gain access prior to age 65.[24]

Assumption NA. No anticipation

For unit i from cohort $C_i = c$ eligible for Medicare at age g_a in year g_t:

$$Y_i(g_t, g_a) = Y_i(\infty) \quad if \quad Y_i(g_t, g_a) < g_a$$

Which is equivalent to:

$$\tilde{q}_{c,a}(g_t, g_a) = \tilde{q}_{c,a}(\infty) \;\; if \; a < g_a \;\; .$$

Assumption NA implies that $ATT(g_t, g_a, a) = 0$ for $a < g_a$, which allows pre-Medicare mortality to provide evidence about the validity of our research designs. We further discuss how to test for anticipation and its impact on our estimates later in the paper.

IV.B.1 Structural Design

Our first approach exploits the full structure of the model in section III to estimate counterfactual mortality rates. Using data on each cohort's mortality rates from birth until the age before they qualify for Medicare, we estimate all of the model's parameters (normalizing $\underline{H} = 0$) for each cohort. We make only one modification to the baseline model: we allow for I to be different in 1918 to account for the excess mortality associated with WWI and the flu pandemic; $\hat{I}_{flu}$. We then simulate mortality through age 95 using these values and construct counterfactual log mortality rates, $\hat{q}_{c,a}^{SIM}(\infty)$ as a function of the estimates $\{\hat{\mu}_H, \hat{\delta}, \hat{\alpha}, \hat{I}, \hat{I}_{flu}, \hat{\sigma}, \hat{\kappa}, 0\}$.

The dashed lines in **Figure 4** plot log mortality rates over the life-course predicted by the model with parameters estimated through age 65 for the 1900 cohort, and the solid line plots observed log mortality rates. The gap between the lines after age 65 generates the treatment effect estimates for this cohort. These are given by

$$\widehat{ATT}^{SIM}(g_t, g_a, a) \equiv \bar{q}_{c,a} - \hat{q}_{c,a}^{SIM} \; . \quad (5)$$

The validity of this approach depends on the correct specification of the model of cohort mortality and the ability to estimate its parameters using pre-Medicare data. While the model clearly abstracts from many features of health investment behavior, it successfully fits patterns

[24] Today, people on the Social Security Disability Insurance program, or who have end-stage renal disease, or who are widows or widowers of a Medicare-eligible spouse can get Medicare before they turn 65. None of these provisions were in place until 1972, when the 1907 cohort aged into Medicare.

of mortality for a wide range of populations (Lleras-Muney and Moreau 2022). The model fit can be assessed in a couple of ways. First, we can assess how well the model does in fitting the observed data up to age 65. Second, we can check to see that there are no "pre-trends": if the model estimates are on average unbiased then the predicted and observed mortality will be very similar up to age 65.

With this model we can estimate the impacts of Medicare on mortality starting at age 65 and until the entire cohort is extinct, providing the entire set of dynamic effects and we can use these to make predictions for life expectancy. Because we explicitly model the size and composition of the surviving population as they age, the model captures commonly observed non-linearities in later life mortality; these are particularly important for accurately estimating effects on life expectancy. In particular, there is a debate about whether log mortality rates continue to be linear in very old age or instead decelerate (e.g. Feehan, 2018). In this model mortality rates decelerate in old age due to selection (individuals who have had many positive shocks survive to the oldest ages).[25] This feature contrasts with the log linearity that is often assumed to hold in empirical work that estimates life expectancy (e.g. Chetty et al. 2016).

The structural estimation has a few limitations. The parameter estimates themselves could be influenced by early life shocks in a way that biases the model-based counterfactuals later in life. An important example of this is the 1918 flu pandemic which affected all of the cohorts we study. We account for this in the estimation, but as Lleras-Muney and Moreau (2022) discuss, these shocks are difficult to model. In addition, the model makes several critical assumptions that could be violated. First it assumes that the parameters are otherwise constant throughout the lifetime. This assumption seems reasonable for populations in stationary environments, in which we would expect that the period and cohort mortality profiles are identical. This is not true in the US for the cohorts we study. Second the model characterizes a production function, but abstracts from optimization, which is likely occurring at both individual and social levels. In the absence of data on health, incomes and health investments however it difficult to produce better estimates. Third, the objective function is poorly behaved, so the estimates are sensitive to initial guesses. To address this, we proceed in two steps. First, we estimate the model for the 1900 birth cohort repeatedly until we can no longer improve the fit. Then we estimate the model again for subsequent cohorts using the 1900 birth cohort values as initial guesses. For our main results we limit attention to cohorts for which the fit is as good

[25] Some argue that this deceleration is due to poor and sparse data quality among the oldest old (e.g. Gavrilova and Gavrilova 2019) but there is much debate about whether this is the case (e.g. see Alvarez et al. 2021).

or better than for the 1900 birth cohort – as **Table A.1** shows this results in our being able to include only the 1901 to 1908 birth cohorts for men and 1901 to 1904 cohorts for women.

Finally note that the model can only be estimated with data from birth onwards, which is only possible using complete life tables from birth to age 65 which are only available from 1900 onwards by sex. Thus, it is important to estimate the effect of Medicare under different and less parametric assumptions.

V.B.2 Interrupted Time-Series Design

Our second approach is an interrupted time-series design based on a functional form assumption on untreated log mortality rates, $q_{c,a}(\infty)$.

Assumption GM (Gompertz-Makeham). Linearity of log untreated mortality rates

For all cohorts, c, and ages between a_{c0} and a_{max}, the log of untreated period mortality rates is linear in age:

$$q_{c,a}(\infty) = \alpha_c + \beta_c(a - a_{c0}) \qquad (6)$$

This standard Gompertz-Makeham model is also implied by the model in section III (Lleras-Muney and Moreau 2022) and supported by extensive empirical evidence (e.g. Chetty et al. 2016) since Gompertz first observed it as an empirical regularity in 1825. This regularity however holds only among adults of a certain age: The log of mortality is high at young ages, falls steeply in childhood and rises in adolescence, before starting to rise in log linear fashion, as can be seen for the 1900 birth cohort in **Figure 4a**.

Under assumptions NA and GM, α_c and β_c can be obtained by regressing $q_{c,a}$ on a constant and age for each cohort using pre-Medicare ages $a < g_a$. Denoting the coefficients from this regression by $\tilde{\alpha}_c$ and $\tilde{\beta}_c$ leads to the following estimand for counterfactual mortality rates at post-Medicare ages: $\tilde{\alpha}_c + \tilde{\beta}_c(a - a_{c0})$. The dashed lines in **Figure 5** plot these linear estimates of counterfactual mortality rates and the solid lines plot observed log mortality rates. Unlike the model-based estimates, which use the full history of mortality to identify the structural parameters, this approach relies only on the linearity assumption during ages close to Medicare eligibility. Key to this approach then is to restrict attention to the range of ages during which the log-linear assumption is appropriate.

This motivates an interrupted time-series (ITS) plug-in estimator that first uses log mortality rates in our sample to estimate $\tilde{\alpha}_c$ and $\tilde{\beta}_c$ (denoted $\hat{\alpha}_c$ and $\hat{\beta}_c$) and then constructs $ATT(g_t, g_a, a)$ as follows:

$$\widehat{ATT}^{ITS}(g_t, g_a, a) \equiv \bar{q}_{c,a} - \hat{\alpha}_c - \hat{\beta}_c(a - a_{c0}) \qquad (7)$$

These are labeled as the difference between the solid and dashed lines in Figure 5.

The ITS approach is simple to implement, but requires use to choose a range of ages, a_{c0} to a_{max}, over which we believe log mortality satisfies Assumption GM. We include data up to age $a_{max} = 90$, similar to Chetty et al. (2016) and Fletcher et al. (2023). By stopping at age 90 we avoid the issue of whether mortality is log linear among the oldest old, and we also avoid the use of potentially badly measured ages at death.[26]

We choice of a_{c0} is less clear and likely differs across cohorts and sex (as can be seen in **Figure A3a and A3b**). We select a_{c0} in two ways.[27] The simplest approach is to use ten years of pre-Medicare eligibility data ($a_{c0} = g_a - 10$). Alternatively, we use our 20% training sample to fit a series of two-slope models to log mortality rates before Medicare eligibility, selecting a_{c0} as the best fitting trend-break as in Card et al. (2008). We pool data for three cohort groups (1901-1915, 1891-1900, and 1891-1900) and select the best-fitting pre-Medicare trend break using five-fold cross-validation methods. For additional details see our pre-analysis plan (Goodman-Bacon et al. 2025).

V.B.3 Difference-in-Differences Design

Our second approach is a difference-in-differences (DiD) design based on an assumption about trends in the log of untreated mortality rates across cohorts.

Assumption PT. Parallel Trends across cohorts and ages

[26] There are many reasons why ages at death might be incorrectly measured among the oldest old. One reason is that the oldest cohorts were born before vital registration systems were complete and thus do not have birth certificates and correct dates of birth.

[27] To gauge the fit of a linear model for estimated Gompertz curves, we regress the log of mortality on age for each birth cohort by sex and report the corresponding R^2 for pre-trends, which we define as trends in log mortality from the age in 1940 to the age before Medicare eligibility age. Specifically, for birth cohort $c \in (1885,1915)$, the age window for pre-trend is: $max(40, 1940 - c)$, $max(65,1966 - c) - 1$. **Figure A4** shows that a linear model fits the Gompertz curves almost perfectly for males with an R^2 over 0.98 for all included birth cohorts. The model fit for females is also excellent for old cohorts 1885-1887 (R^2=0.99) but gradually decreases across cohorts to 0.945 for the 1904 cohort, then increases to over 0.98 for later cohorts 1910-1915.

The change in untreated log mortality rates for cohort c between ages $G_a(c) = g_a$ and a equals the average change in log untreated mortality rates between the same ages for older cohorts treated after age a and between 1 and k years after age $G_a(c)$, where $a < G_a(c) + k$

$$q_{c,a}(\infty) - q_{c,g_a-1}(\infty) = E\left[q_{\ell,a}(\infty) - q_{\ell,g_a-1}(\infty) | max\{a, g_a\} < G_a(\ell) \leq g_a + k\right] \quad (8)$$

This assumption states that we can allow different cohorts to have different levels of log mortality so long as log mortality rates have the age profiles (age slope) in the absence of treatment. As in Callaway and Sant'Anna (2021), assumption PT involves a cohort treated at age g_a and older comparison cohorts treated at later ages.[28] The difference is that k restricts the set of comparison cohorts to those who gain eligibility close to the age g_a and in doing so also restricts the ages at which $ATT(g_t, g_a, a)$ parameters can be identified. For example, with $k = 5$, the comparison group used to identify counterfactual log mortality rates for the 1900 cohort (first treated at age 66) includes cohorts born between 1899 and 1895 (treated between ages 67 and 71). This permits identification of $ATT(g_t, g_a, a)$ parameters for $a < 71$, since the oldest comparison cohort is treated by age 71.[29] A larger value of k means a larger yet older comparison that identifies parameters at older ages.[30]

Figure 6 illustrates how the PT assumption identifies counterfactual mortality changes in a simple case where we compare two cohorts at a time. To identify $ATT(1966,71,71)$, which is the effect of Medicare on mortality for the 1895 cohort in the first year they were on Medicare (i.e. at age 71 in 1966), we compare their change in mortality between ages 70 and 71 to the change in mortality for older cohorts at the same ages. In **Figure 6** we obtain nearly identical estimates when the comparison cohort is born in 1894 (ages 70 and 71 in 1964 and 1965) or in 1885 (ages 70 and 71 in 1955 and 1956). Identification of $ATT(1966,71,80)$, the effect for the same 1895 cohort at age 80, works the same way but requires a comparison cohort born at least ten years earlier (in this case, 1885).

Under assumptions NA and PT, a simple DiD estimand identifies $ATT(g_t, g_a, a)$. Plugging in sample analogues yields the following DiD estimator:

[28] Assumption PT is also a proportional hazards assumption because it is equivalent to a constant the ratio of age-specific mortality rates between cohort c and the average comparison cohort (Deaner and Ku 2024).

[29] The $\max\{a, G_a(c)\}$ notation ensures that all k comparison cohorts are used to construct pre-treatment falsification tests when $a < G_a(c)$.

[30] Assumption PT defines comparison cohorts as those whose Medicare age comes after $G_a(c)$, which means that for cohorts treated later and by definition at age 65, the only available comparison cohorts are those born in up to k years before 1901. Therefore, the cohort gap between treatment and comparison units grows as we look at younger cohorts in this design.

$$\widehat{ATT}^{DiD(k)}(g_t, g_a, a) = \left(\bar{q}_{c,a} - \bar{q}_{c,g_a-1}\right) - \sum_{\substack{\ell: \\ \max\{a,g_a\}<G_a(\ell) \\ G_a(\ell)\leq g_a+k}} w_{\ell,a}\left(\bar{q}_{\ell,a} - \bar{q}_{\ell,g_a-1}\right) \qquad (9)$$

This is a version of the Callaway and Sant'Anna (2021) estimator which we implement by adding the choice of k to the Stata command `csdid` (Callaway, Sant'Anna, Rios-Avila 2021), defining cohorts as the cross-sectional unit, age as the time variable, and $G_a(C_i)$ as the treatment variable. The weights $w_{\ell,a}$ reflect the relative size of the comparison cohorts used at age $g_a - 1$.

Intuitively the PT assumption is more likely to be satisfied for cohorts that are closer together in age than for cohorts that are not, but importantly, this is partially testable. To choose k, we again use our 20% training sample. Using all cohorts, we estimate ten pre-treatment $ATT(g_t, g_a, a)$ parameters for $k \in \{1,10\}$, and calculate the maximum period-to-period violation, M_k, which is the same quantity used by Rambachan and Roth (2023) in their bounding estimator based on potential parallel trends violations. We choose the value of k that minimizes $|M_k|$, repeating the exercise for each cohort group when we report $ATT_b(e)$ parameters.[31] In other words we include cohorts so long as they do not include large estimated pre-trends in their confidence intervals.

At a common pair of ages, the log mortality rates of different cohorts necessarily occur in different calendar years, so the PT assumption requires that the trend in "time effects" is the same across periods (see Dinas and Stoker, 2014 and Goodman-Bacon 2025). Given the turbulence of the 1960s and of the 20th century more generally, this implication of the PT assumption may not hold. For example, if one cohort was more affected than another by WWI, then its health may have deteriorated at higher rates and resulting in different age slopes.[32]

We check this in two ways. The first is a falsification test based on the same kind of comparison described in equation (9), but shifted back by 10 years. For example, we can compare changes in mortality around 1966 for cohorts who were in their mid-50s to changes in earlier years when slightly older cohorts were in their mid-50s; none of whom got Medicare at this time. In other words, we can check that the PT assumption is satisfied in the "pre-period". The second

[31] We also explore two alternative procedures, one using the F-statistic on a test of the joint significance of the pre-period estimates, and another sequential test that increases k if we fail to reject joint significance for $k-1$, and stops as soon as we reject.

[32] Several studies have documented such "scarring effects" of war. See Lleras-Muney and Moreau (2022)'s results for the 1898 French cohort and the references therein.

approach is an alternative DiD design based on comparing changes in mortality for a cohort treated at time $G_t(c)$, to the change in mortality over the same time periods for a comparison group of *younger* cohorts treated in the future. This fixes comparisons to occur in the same years, but across different ages (Goodman-Bacon 2025). We discuss this approach and report results in Appendix X.

V.B.4 Estimating shift and trend-break parameters

We estimate the shift and trend-break parameters for each cohort using post-Medicare $\widehat{ATT}(g_t, g_a, a)$ estimates the OLS formulas that that relate them to event-age:

$$\hat{\delta}_c \equiv \sum_{j=g_a}^{A} \frac{\widehat{ATT}(g_t,g_a,j)}{A-(g_a-1)} - \hat{\theta}_c^{DiD}\left(\frac{g_a+A}{2}\right) \quad (10)$$

$$\hat{\theta}_c \equiv \frac{\sum_{j=g_a}^{A} \widehat{ATT}(g_t,g_a,j)\left(j-\frac{g_a+A}{2}\right)}{\sum_{j=g_a}^{A}\left(j-\frac{g_a+A}{2}\right)^2} \quad (11)$$

These linear combinations are simple to calculate using post-estimation commands.[33]

VI. Results

VI.A Main cohort-level results

The top row of **Figure 7** plots estimates of the aggregated event-study parameters from the reduced form approaches, $ATT(e)$, for men (panel A) and women (panel B).

For men we find clear and credible evidence that Medicare reduced log mortality rates. The pre-period falsification tests are only significant in one case, never larger than 0.05, and display no obvious time-series pattern. Log mortality rates were not systematically falling relative to the model's prediction, the pre-Medicare Gompertz slope, or nearby cohorts. After gaining Medicare eligibility, however, the ATT estimates from both approaches are negative, clearly distinguishable from zero after 2 years, and growing in magnitude over time in a linear fashion. After ten years we find that Medicare reduced age-specific mortality rates for white men by 0.2 log points.

The bottom row of **Figure 7** reports the results from the structural estimation for men using the 1901-1908 birth cohorts for whom we have lifetime data and a good fit prior to age 65. The

[33] For the ITS design we can also use the following regression to estimate the shift and trend-break parameters for each cohort directly: $\bar{q}_{c,a} = \hat{\alpha}_c + \hat{\beta}_c(a - a_{c0}) + \hat{\delta}^{ITS}(g_t, g_a)1\{a \geq g_a\} + \hat{\theta}^{ITS}(g_t, g_a)(a - g_a) + \varepsilon_{ca}$.

results show that the model does an excellent job at predicting log mortality for the 25 years prior to Medicare for men. After age 65 we observe a pattern that is very similar to the pattern we obtain in the reduced form approaches: there is a small decline in the intercept at age 65 and a linear decline thereafter. Interestingly the model shows that the declines level off after ten years but remain large (-0.2 log points) up to 25 years after cohorts gain Medicare.

The top row of **Table 2** summarizes the average event-study using estimates of the average shift ($\frac{1}{31}\sum_{c=1885}^{1915}\hat{\theta}_c$) and slope changes ($\frac{1}{31}\sum_{c=1885}^{1915}\hat{\delta}_c$) in log mortality rates associated with gaining Medicare. Both the ITS and the DiD estimate small shifts (the estimate is not significant in the DiD design), but similar and statistically significant decreases in the slope. Although the structural model can only be estimated for a subset of cohorts, the results are similar. If we compute the average change in the slope is -0.01 and the change in the shift is about -0.007 — the slope estimate in particular is quite similar to the overall estimates across cohorts. Through the lens of the model, this suggests that, for men, Medicare is equivalent to increasing annual investments in health or lowering depreciation/aging rates.

These ITS and DiD estimates translate into a total gain in life expectancy of 0.8-0.9 years across the 1885-1915 birth cohorts, assuming that the effects on log mortality continue to grow linearly, as shown in **Table 3**. The structural model for the 1901-1908 cohorts only suggests gains of about 2.4 years, which results from focusing on a subset of cohorts that have relatively large benefits of Medicare (as we will show shortly).

The aggregate event-study estimates for women are substantially noisier and not robust across empirical strategies, as **Figure 7** shows. Like the results for men, the pre-period falsification tests do not show a systematic differential trend in mortality prior to Medicare eligibility using either the ITS or the DiD approaches, but the estimates are less precise and often of larger magnitudes. The post-period coefficients however do not consistently show significant effects. The ITS estimates are positive and never statistically distinguishable from zero. The DiD estimates are significant and negative only for the last three years (years 8, 9 and 10 after Medicare), but less than half as large as the results for men. The estimates based on the structural model on the other hand show substantial gains, but they also show some substantial pre-trends – that is, even though we restrict attention to cohorts with a good fit prior to age 65, the model does not match the data well. Altogether the results for women are unclear.

This is also apparent in the estimated intercept and slope changes for women in **Table 2.** Again, the ITS and the DiD approaches give opposite results. The ITS estimates statistically significant declines in the shift and increases in the slope, whereas the DiD estimates no statistically significant change in the shift but a statistically significant decline in the slope. Thus, each method gives a wildly different estimate of the life expectancy effects for women: ranging from an increase of 0.68 years if we use the ITS method to a decline of 0.5 years if we use the DiD method (**Table 3**). The structural model gives much larger estimate of a 3.1 increase, perhaps implausibly large, given the existence of important pre-trends.

VI.B Results by birth cohort

The aggregate estimates average over cohorts as described in equation 3, but we are also interested in heterogeneity in the cohort-specific effects of Medicare. **Figure 8** shows shift and slope estimates ($\hat{\theta}_c$ and $\hat{\delta}_c$) for each cohort by sex.

For men (panel A and B in the top row) the cohort specific results are very consistent with the aggregate results for most cohorts. For cohorts born 1885 to 1905 the estimates are similar: the slopes show statistically significant declines, and the intercepts are statistically insignificant. This is true in both the ITS and in the DiD approaches – while they each yield different point estimates, their confidence intervals overlap so the results are not statistically different across both methods. The estimates that are available from the structural model are typically not distinguishable from those we obtain from the ITS approach.

Starting around 1905 however we observe a change in the pattern. First, the intercept becomes negative and statistically significant, particularly in the ITS design. The change in the slope on the other hand goes from negative to positive using ITS but it remains negative using DiD. For the 1908-1915 cohorts the two approaches give diverging results.

Table 2 summarizes these observations by grouping cohorts into groups. We group the first 15 cohorts together based on our observations that they appear to be identical. We estimate a small (0.022 with the ITS design) or statistically insignificant change (less than -0.001 for the DiD design) in the shift. Both the ITS and the DiD estimates show statistically significant declines in the slope of about -0.018 to -0.023, which is similar or a bit larger than the pooled estimate for all cohorts. But these results change progressively across cohorts. In the ITS design the shift becomes more negative moving from 0.004 for the 1901-1905 cohorts to -0.064 for the 1911-1915 birth cohorts. In the DiD design the shift is positive for the 1901 cohort, negative for the

1906-19010 cohort and insignificant for the 1911-1915 cohorts. Similarly, the slope becomes less negative across cohorts in both designs, but it goes from large and negative to positive and insignificant in the ITS design, whereas the decline in the slope is smaller in the DiD design.

Figure 9 shows the implied changes in LE at age 55 across cohorts. The results are very consistent across designs for men, except the very last cohorts. Medicare leads to gains in LE that are insignificant for the 1885 cohort, but become positive by 1890, they continue to rise and max out around 2 years of life for the 1908 birth cohort. The structural estimates are generally larger, but they exhibit a similar trend upwards, increasing from roughly 2 to 2.7 years. Much of this phase-in pattern follows from differences in the age at which cohorts aged into Medicare. While the effects on period mortality are similar across cohorts, the time frame over which these benefits accumulate is mechanically different. For the most recent cohorts however, all of whom get Medicare at age 65, the effects decline. For the two last birth cohort the effects are inconsistent across methods (they are positive for the DiD but negative for the ITS).

It is not clear why the results are not robust for the most recent cohorts. One possibility is that the effects for the most recent cohorts are underestimated due to changes in Medicare. Beginning in 1973, Medicare was expanded to individuals under age 65 with disabilities or end-stage renal disease. Individuals from these two high-risk groups born after 1907 would become eligible for Medicare before age 65, which could flatten our estimation of pre-trends in log mortality and then lead to underestimated effect on e55 from our interrupted time series analyses. Another possibility is that over time Medicare generated general equilibrium effects affecting younger cohorts. For example, Hackmann et al. (2025) find evidence that large health insurance expansions result in significant expansions in the supply of health care, raising employment. Other work also suggest that Medicare may have significant spillovers onto the private health insurance market affecting the under 65 population, for example by affecting pricing for various health services (e.g. Clemens and Gottlieb 2017). Finally, it is possible that other events after 1965 and later affected the younger cohorts, even before they became eligible for Medicare. We explore this issue below.

For women the results are inconsistent across cohorts and methods as **Figure 8** shows. The slopes are generally positive in the ITS design but negative in the DiD design and they vary a lot across cohorts. Similarly, the shifts vary a lot across cohorts and do not always align across methods. This is easily seen in **Table 2** again. **Figure 9** shows that for women both the ITS

and the DiD approaches yield statistically insignificant effects on life expectancy at age 55 for almost all birth cohorts (the only significant ones are declines for the 1914 and 1915 cohorts using the ITS but not the DiD method), while the structural estimates yield very large effects.

In sum, across methods and cohorts we find that for men Medicare leads to a significant decline in log mortality rates, the size of which increases over the ten-year window for which we can estimate effects. These effects can be characterized as declines in the slope of the age profile of the log of mortality, which theoretically could result from increases in health investments or decreases in the rate of aging. The implied effects of Medicare rise across cohorts suggesting larger effects for cohorts receiving benefits at earlier ages. However, there is a decline in the impact of Medicare for the most recent cohorts though the results for these cohorts are less robust. For women we do not find consistent evidence that Medicare had a significant impact on mortality rates or life expectancy, with results varying wildly across methods and cohorts.

VI.C Assessing the validity of the identification assumptions

We now discuss a series of exercises we conduct to assess various identification issues.

Choice of window for ITS. Our main ITS estimates use the fixed 10-year window. We also estimated the model using an optimally chosen window as described in Section IV.B.2. **Figure A6** shows how the estimates compare. For men the results are insensitive to this choice except for the last five cohorts: the estimates remain positive and significant and substantial in magnitude (about 1.5 years for the 1910-1915 cohorts) if we use an optimally chosen window instead of the 10-year window, but they drop and become negative if we only use 10 years in the pre-period. For women the results are not sensitive to this choice either: most estimates remain insignificant or negative and this is true regardless of how we specify the window.

Choice of comparison cohort for DiD. Our main DiD estimates are constructed by comparing affected cohorts with unaffected cohorts born up to ten years earlier. In **Figure A7** we vary the number of cohorts that we include. In panel A we include cohorts born up to 15 years earlier. In Panel B we restrict attention to cohorts born up to 4 years earlier, and in panel C we include cohorts born up to 9 years earlier. For men (**Figure A7a**) the results are quite robust. Although the point estimates vary somewhat, the results are similar across comparison groups and in all cases show the same pattern: There is no evidence of pre-trends, there is very little change in the first two years post Medicare but there is a steady and linear decline thereafter. For women (**Figure A7b**) we continue to find insignificant effects regardless of the choice of cohorts we

use as comparisons. All in cases there are no pre-trends, and there are very few statistically significant effects.

The no-anticipation assumption. All estimation approaches rely on the assumption that individuals do not respond prior to becoming eligible. The assumption of no anticipation seems very reasonable for the cohorts that were first treated in 1966 – the passage of Medicare was difficult and unanticipated as discussed in Section II. This assumption however seems more questionable for the cohorts affected after 1966: As the population became aware of the program there might have responded to its availability, even before turning 65.

How would anticipation affect our estimates? In Appendix A.6 we consider a simple two-period model in which health and mortality evolve according to the process described in Lleras-Muney and Moreau (2022) but in which individuals can purchase medical care to improve their health. We show that regardless of how we model the impact of medical care, if Medicare lowers the cost of medical care in the second period, then in the first period individuals will increase general consumption and lower the consumption of medical care. As a result, health in the pre-Medicare period will fall and mortality will increase. Existing empirical evidence suggests that patients might indeed delay non-urgent care to age 65.[34] To test for anticipation then, we focus on the 2 years prior to reaching age 65 and test to see if mortality rates rise right before turning 65. The results are shown in Appendix X (TBA).

Other shocks correlated with Medicare. A different concern is that the passage of Medicare coincides with other policy changes that could affect both the over 65 population and the under 65 population. For example, Medicaid was created at the same time as Medicare. Poor elderly individuals are eligible for both, so in principle we are identifying the effects of the combined policy. To address this, we can take advantage of our data and look to see if the effects we estimate are larger in states that did not implement Medicaid until 1970 (Medicaid was a state program). The results are in Appendix XX (TBA).

A second concern is that events coinciding with the passage of Medicare might have affected younger individuals, potentially contaminating the control groups. For example, younger poorer individuals would be eligible for Medicaid as well starting in 1966. To test this, we

[34] Card et al. (2008) find that elective procedures rise at age 65 particularly for previously uninsured individuals consistent with patients delaying care. Similarly, Decker (2005) finds that mammography use increases sharply at age 65. Clemens and Gottlieb (2014) document that physicians' financial incentives within the Medicare program also strongly influence the provision of elective services.

focus on younger cohorts (born after 1902) – these cohorts would have benefited from these changes before becoming eligible for Medicare. For these cohorts, we estimate models in which we allow for a change in slope or intercept in 1966 in addition to changes in the intercept and slope after age 65. Results in Table A.2 show that indeed, there is some evidence that there are changes occurring in 1966 lowered mortality for men born in 1911 and later, adding suspicion to the validity of our estimates for the most recent cohorts of men. For women we also find evidence of other shocks in 1966 also mattering for the 1902 to 1909 – potentially explaining why our estimates for women are so unstable across cohorts.

In sum, our estimates for men are quite robust except for the very last cohorts, but our estimates for women are not. We discuss the reasons for these sex differentials later in the paper.

VI.D Heterogeneity by education and income

We now present results from both identification strategies for subgroups (within-sex) defined by years of completed schooling (0-7 years, 8-11 years, and 12 or more years) and whether people lived in households with above- or below-median income in 1940. We focus overall event-study parameters for log mortality, $ATT(e;x)$, and the life-expectancy parameters, $ATT_{LE}(x)$, but include subgroup estimates of the other objects of interest in appendix X.[35]

These estimates are interesting for at least two reasons. First because the previous literature has documented important heterogeneity across these dimensions. SES measures are strong predictors of having private health insurance (Card et al., 2008) and better health prior to reaching age 65 (Braveman et al., 2010). Therefore, those with lower SES may see larger health improvements when gaining Medicare (Card et al., (2004) Table 10). Even with equal access to health care, however, low-SES people still visit specialists at lower rates (Braveman et al., 2010; Dunlop et al., 2000) and benefit less from medical advances because they appear to adopt these at lower rates (Braveman et al., 2010; Dunlop et al., 2000; Glied and Lleras-Muney, 2008). Thus higher SES individuals might benefit more. We can examine this empirically.

The second reason these sub-group estimates are interesting is that they help relax identification assumptions. Because they only use a subset of the data, subgroup parameters are identified by conditional versions of the GM or PT assumptions; that is, they nonparametrically control for education or income. These conditional assumptions may be more plausible than the

[35] Recall that SSA life tables are not available for any sub-national group so we cannot estimate the effects of Medicare using the structural model for these groups.

unconditional versions discussed above. For instance, if differential mortality rates by education generate time-varying compositional changes as cohorts age, then assumptions GM or PT may fail even if they hold within education groups. Indeed, prior studies have found that education reduces mortality rates and that educational levels increased substantially for the later birth cohorts in our analysis (Goldin, 1998; Lleras-Muney, 2005; Lleras-Muney et al., 2022; Yue et al., 2023b). But the estimated effects on log mortality rates for each subgroup—identified by more refined within-group comparisons—can be aggregated to construct conditional estimates of the unconditional parameters such as $ATT(e)$. We make this comparison as a check on the validity of the aggregate identifying assumptions for both our designs.

We start by considering effects by education levels.

Figure 10 plots the event-study estimates by the three education groups separately: 0-7 years of schooling (less than middle school), 8-11 years of schooling (graduated from middle school but not high school), and 12+years of schooling (high school or above). For all groups there is very little evidence of pre-trends.

For men we find evidence that Medicare lowers log mortality rates at an increasing rate over time for all three groups. But the effects appear larger for lower education groups. Table 3 shows that indeed the implied effects on life expectancy decrease with education levels from .89 years for those with less than primary education to 0.71 for high school graduates. But this is true only for the ITS approach. The DiD approach finds the opposite pattern which larger gains for the most educated group. Furthermore these differences are small, so on net the effects do not appear to be meaningfully different by education.

For women the event studies in **Figure 10** do not show impacts except perhaps for those without primary schooling. **Table 3** shows indeed these gains are larger for the least educated women, but this is only true in the DiD estimates. The ITS estimates are insignificant in magnitude or negative. So again, the results for women are inconclusive.

VII.Discussion

VII.A Magnitudes

Our estimates for men show that Medicare obtained at age 65 increases life expectancy at age55 by as much as two years. These are very large effects. Figure A2 shows that life expectancy at

age 55 rose from about 20.5 years for the 1885 birth cohort to about 22 years for the 1915 cohort. So, the effects of Medicare that we estimate are larger than the gains observed across these 30 birth cohorts.

Are these magnitudes reasonable? Evidence from randomized trials suggests these are plausible. For example, reductions in hypertension and cholesterol and more aggressive heart disease interventions have been estimated to increase life expectancy for men by several years (Wright and Weinstein 1998). Heart disease was the main cause of death among men ages 65 and above in 1965.

The gains we estimate are large enough to justify expenditures per enrollee at the time. The estimated annual cost per enrollee in 1970 was around $368. If we assume that Medicare enrollees lived for 24 years (22 years + 2 gained), the total cost per enrollee's lifetime is less than $10,000, substantially lower than the estimated statistical value of a year of life.

VII.B Comparison with other studies of Medicare's introduction

Our approach and results contrast in several different ways with two closely related DiD studies: Finkelstein and McKnight (2008; FM) and Chay, Kim, and Swaminathan (2010; CKS). Both papers begin by comparing changes in age-specific mortality across calendar time (and cohort) for people over 65 versus people under 65. FM (Figure 3) find little evidence that the log of the number of deaths for people aged 65-74 fell after 1966 relative to log deaths for people aged 55-64. CKS (Figure 4), on the other hand, find clear evidence that mortality rates (and log mortality rates) for people aged 65-69 fell relative to those for people aged 60-64.[36] This kind of comparison estimates effects on mortality at a fixed set of ages, while our design targets dynamic effects within each cohort, consistent with our goal of understanding Medicare's effect on life expectancy.[37] Moreover, the assumption behind this "older versus younger" design are not nested with Assumption PT. It is not clear if one is more plausible, but our findings support the validity of our design based comparing within-cohort age profiles and our findings align more closely with CKS.

[36] The disagreement between these studies must stem from a combination of using log counts versus rates or log rates, or the choice of difference age windows.

[37] CKS calculate cohort life expectancy for the 1896, 1899, and 1904 cohorts, but do not estimate causal effects on these measures. Rather they assume that "all of the gains in life expectancy [across cohorts] can be attributed to the Medicare program" (pg. 23). Because our design and data yield causal effects on mortality across ages for each cohort, we are able to construct estimates of $ATT_{LE}(g_t, g_a)$.

FM also use an additional DiD design that compares changes in age-specific mortality rates before and after 1966 in sub-regions that had different health insurance coverage rates in 1963. This design effectively uses the time trends in elderly mortality in the Northeast, where Medicare led to little change in insurance thanks to widespread Blue Cross coverage, as a control group for the South, where Medicare led to very large insurance gains because Blue Cross coverage was quite rare. These comparisons show that, if anything, elderly mortality *rose* in places that saw large coverage gains relative to places with small gains. DiD designs with a continuous treatment variable, however, require additional assumptions that go beyond traditional parallel trends by imposing a kind of treatment effect homogeneity across regions. Because regional health insurance coverage was strongly correlated with factors that potentially mediate the effect of Medicare on mortality, such as income, provider capacity, or industrial mix, this assumption need not hold. CKS also show that a different "first-stage" outcome—the change in elderly hospital utilization—was actually quite similar in the North and the South after 1966, suggesting that mortality effects should be comparable as well.[38]

VII.C Discussing sex differences (TBA)

VII.D Using estimates to predict LE gains among current cohorts. (TBA)

VII.E Predicting the increases in the size of the over 65 population (TBA)

VIII. Conclusions

This paper uses a large and novel dataset that measures lifetime mortality for a wide range of cohorts and includes individual economic and demographic characteristics measured in 1940. We use these data to implement three research designs that estimate dynamic within-cohort causal effects of the introduction of Medicare on log-mortality rates. One design uses a structural model of cohort mortality based on lifetime mortality profiles (Lleras-Muney and Moreau 2022). Another is an interrupted time-series design based on a qualitative prediction of that model (linearity of log mortality later in life). Our final design is an extension of difference-in-differences with staggered treatment (Callaway and Sant'Anna 2021). For white men, all three approaches show clear evidence that Medicare reduced mortality rates by about 0.2 log points after ten years, that these effects are relatively similar across cohorts born

[38] FM also use a special tabulation of the NHIS by 11 sub-regions, but the 1963 NHIS sample design was only intended to be representative at the region level (and separately by urban residence). Measurement error in sub-regional uninsurance rates may also attenuate estimates from a dose-response type DiD design.

between 1885 and 1905, and that they generated cohort-level changes in life expectancy of up to 2 years. Our results for white women, on the other hand, are not consistent across these designs. Overall, and in contrast to some earlier work as well as contemporaneous research focused on 65-year-olds, we find clear evidence that Medicare resulted in large reductions in mortality rate and substantial life expectancy gains.

References

Alvarez, J. A., Villavicencio, F., Strozza, C., & Camarda, C. G. (2021). Regularities in human mortality after age 105. *PloS one*, *16*(7), e0253940.

Andersen MS. Effects of Medicare coverage for the chronically ill on health insurance, utilization, and mortality: Evidence from coverage expansions affecting people with end-stage renal disease. Journal of Health Economics 2018(60), 75-89.

Baicker K, Taubman SL, Allen HL, Bernstein M, Gruber JH, Newhouse JP, Schneider EC, Wright BJ, Zaslavsky AM, Finkelstein AN, Oregon Health Study G. The Oregon experiment--effects of Medicaid on clinical outcomes. N Engl J Med 2013(368), 1713-1722.

Bailey M, Cole C, Henderson M, Massey C. How Well Do Automated Linking Methods Perform? Lessons from U.S. Historical Data. Journal of Economic Literature 2020(58), 997-1044.

Bailey MJ, Goodman-Bacon A. The War on Poverty's experiment in public medicine: Community health centers and the mortality of older Americans. Am Econ Rev 2015(105), 1067-1104.

Barcellos SH, Jacobson M. The effects of Medicare on medical expenditure risk and financial strain. American Economic Journal: Economic Policy 2015(7), 41-70.

Borgschulte M, Vogler J. Did the ACA Medicaid expansion save lives? Journal of Health Economics 2020(72), 102333.

Boudreaux MH, Golberstein E, McAlpine DD. The long-term impacts of Medicaid exposure in early childhood: Evidence from the program's origin. Journal of Health Economics 2016(45), 161-175.

Braveman PA, Cubbin C, Egerter S, Williams DR, Pamuk E. Socioeconomic disparities in health in the United States: what the patterns tell us. American Journal of Public Health 2010(100 Suppl 1), S186-196.

Brown DW, Kowalski AE, Lurie IZ. Long-term impacts of childhood Medicaid expansions on outcomes in adulthood. The Review of Economic Studies 2020(87), 792-821.

Callaway B, Goodman-Bacon A, Sant'Anna PH. 2024. Difference-in-differences with a continuous treatment. (Ed)^(Eds). National Bureau of Economic Research; 2024.

Callaway B, Sant'Anna PH. Difference-in-differences with multiple time periods. Journal of Econometrics 2021(225), 200-230.

Card D, Dobkin C, Maestas N. The Impact of Nearly Universal Insurance Coverage on Health Care Utilization and Health: Evidence from Medicares. RAND Corporation: Santa Monica, CA; 2004.

Card D, Dobkin C, Maestas N. The Impact of Nearly Universal Insurance Coverage on Health Care Utilization: Evidence from Medicare. Am Econ Rev 2008(98), 2242-2258.

Card D, Dobkin C, Maestas N. Does Medicare save lives? Quarterly Journal of Economics 2009(124), 597-636.

Center for Health Administration Studies, National Opinion Research Center. 1984. Survey of Health Services Utilization and Expenditures, 1963. (Ed)^(Eds). Inter-university Consortium for Political and Social Research (ICPSR) [distributor]; 1984.

Chay KY, Kim D, Swaminathan S. 2010. Medicare, hospital utilization and mortality: Evidence from the program's origins. (Ed)^(Eds). Citeseer; 2010.

Chung AP, Gaynor M, Richards-Shubik S. Subsidies and structure: the lasting impact of the Hill-Burton program on the hospital industry. Review of Economics and Statistics 2017(99), 926-943.

Clay K, Lewis J, Severnini E, Wang X. The value of health insurance during a crisis: Effects of medicaid implementation on pandemic influenza mortality. Review of Economics and Statistics 2024(106), 1393-1402.

Clemens J, Gottlieb JD. Do physicians' financial incentives affect medical treatment and patient health? Am Econ Rev 2014(104), 1320-1349.

Clemens, J., & Gottlieb, J. D. (2017). In the shadow of a giant: Medicare's influence on private physician payments. *Journal of Political Economy*, *125*(1), 1-39.

Currie J, Decker S, Lin W. Has public health insurance for older children reduced disparities in access to care and health outcomes? Journal of Health Economics 2008(27), 1567-1581.

Currie J, Gruber J. Health insurance eligibility, utilization of medical care, and child health. Quarterly Journal of Economics 1996a(111), 431-466.

Currie J, Gruber J. Saving babies: The efficacy and cost of recent changes in the Medicaid eligibility of pregnant women. Journal of Political Economy 1996b(104), 1263-1296.

Dalgaard C-J, Strulik H. Optimal aging and death: understanding the Preston curve. Journal of the European Economic Association 2014(12), 672-701.

De Lew N. Medicare: 35 years of service. Health Care Financing Review 2000(22), 75.

Decker SL. Medicare and the health of women with breast cancer. Journal of Human Resources 2005(40), 948-968.

Dinas E, Stoker L. Age-Period-Cohort analysis: A design-based approach. Electoral Studies 2014(33), 28-40.

Dobkin C, Finkelstein A, Kluender R, Notowidigdo MJ. The Economic Consequences of Hospital Admissions. Am Econ Rev 2018(102), 308-352.

Duggan MG, Evans WN. 2008. Estimating the impact of medical innovation: A case study of HIV antiretroviral treatments. (Ed)^(Eds), Forum for Health Economics & Policy, vol. 11. De Gruyter; 2008.

Dunlop S, Coyte PC, McIsaac W. Socio-economic status and the utilisation of physicians' services: results from the Canadian National Population Health Survey. Social Science & Medicine 2000(51), 123-133.

Dunn A, Shapiro AH. Does medicare part D save lives? American Journal of Health Economics 2019(5), 126-164.

FamilySearch. 2025. FamilySearch. (Ed)^(Eds), vol. 2025. 2025.

Feehan, D. M. (2018). Separating the signal from the noise: Evidence for deceleration in old-age death rates. *Demography*, *55*, 2025-2044.

Figinski TF, Troland E. 2020. Health Insurance and Hospital Supply: Evidence from 1950s Coal Country. (Ed)^(Eds), Finance and Economics Discussion Series 2020-033. Board of Governors of the Federal Reserve System: Washington; 2020.

Finkelstein A. The aggregate effects of health insurance: Evidence from the introduction of medicare. Quarterly Journal of Economics 2007(122), 1-37.

Finkelstein A, Gentzkow M, Williams H. Sources of geographic variation in health care: Evidence from patient migration. The Quarterly Journal of Economics 2016(131), 1681-1726.

Finkelstein A, McKnight R. What did Medicare do? The initial impact of Medicare on mortality and out of pocket medical spending. Journal of Public Economics 2008(92), 1644-1668.

Finkelstein A, Taubman S, Wright B, Bernstein M, Gruber J, Newhouse JP, Allen H, Baicker K, Oregon Health Study G. The Oregon Health Insurance Experiment: Evidence from the First Year. Quarterly Journal of Economics 2012(127), 1057-1106.

Gavrilov, L. A., & Gavrilova, N. S. (2019). New trend in old-age mortality: Gompertzialization of mortality trajectory. *Gerontology*, *65*(5), 451-457.

Glied S, Lleras-Muney A. Technological innovation and inequality in health. Demography 2008(45), 741-761.

Goldin C. America's graduation from high school: The evolution and spread of secondary schooling in the twentieth century. The Journal of Economic History 1998(58), 345-374.

Goldin J, Lurie IZ, McCubbin J. Health Insurance and Mortality: Experimental Evidence from Taxpayer Outreach. Quarterly Journal of Economics 2021(136), 1-49.

Gompertz B. XXIV. On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. In a letter to Francis Baily, Esq. FRS &c. Philosophical transactions of the Royal Society of London 1825), 513-583.

Goodman-Bacon A. Public Insurance and Mortality: Evidence from Medicaid Implementation. Journal of Political Economy 2018(126), 216-262.

Goodman-Bacon A. The Long-Run Effects of Childhood Insurance Coverage: Medicaid Implementation, Adult Health, and Labor Market Outcomes. Am Econ Rev 2021(111), 2550-2593.

Gornick ME, Warren JL, Eggers PW, Lubitz JD, De Lew N, Davis MH, Cooper BS. Thirty years of Medicare: impact on the covered population. Health Care Financing Review 1996(18), 179.

Graves JA, Hatfield LA, Blot W, Keating NL, McWilliams JM. Medicaid Expansion Slowed Rates Of Health Decline For Low-Income Adults In Southern States. Health Aff (Millwood) 2020(39), 67-76.

Grossman M. On the Concept of Health Capital and the Demand for Health. Journal of Political Economy 1972(80), 223-255.

Hackmann, M. B., Heining, J., Klimke, R., Polyakova, M., & Seibert, H. (2025). *Health Insurance as Economic Stimulus? Evidence from Long-Term Care Jobs* (No. w33429). National Bureau of Economic Research.

Hausman JK. Fiscal Policy and Economic Recovery: The Case of the 1936 Veterans' Bonus. Am Econ Rev 2016(106), 1100-1143.

Huh J, Reif J. Did medicare part d reduce mortality? Journal of Health Economics 2017(53), 17-37.

Leinonen R, Heikkinen E, Jylhä M. Predictors of decline in self-assessments of health among older people—A 5-year longitudinal study. Social Science & Medicine 2001(52), 1329-1341.

Levinsky M, Schiff M. Lifetime cumulative adversity and physical health deterioration in old age: Evidence from a fourteen-year longitudinal study. Social Science & Medicine 2021(289), 114407.

Lichtenberg FR. The effects of Medicare on health care utilization and outcomes. Forum for Health Economics & Policy 2002(5).

Lleras-Muney A. The relationship between education and adult mortality in the United States. Review of Economic Studies 2005(72), 189-221.

Lleras-Muney A, Moreau F. A Unified Model of Cohort Mortality. Demography 2022(59), 2109-2134.

Lleras-Muney A, Price J, Yue D. The association between educational attainment and longevity using individual-level data from the 1940 census. Journal of Health Economics 2022(84), 102649.

Pollitz, K., Pestaina, K., Montero, A., Lopes, L., Valdes, I., Kirzinger, A., & Brodie, M. (2023). KFF Survey of Consumer Experiences with Health Insurance. *KFF (June 2023)*.

Ludwig J, Miller DL. Does Head Start improve children's life chances? Evidence from a regression discontinuity design. The Quarterly Journal of Economics 2007(122), 159-208.

Miller S, Johnson N, Wherry LR. Medicaid and Mortality: New Evidence from Linked Survey and Administrative Data. Quarterly Journal of Economics 2021(136), 1783-1829.

Miller S, Wherry LR. The long-term effects of early life Medicaid coverage. Journal of Human Resources 2019(54), 785-824.

Moon, M. (1996). What Medicare has meant to older Americans. *Health care financing review*, *18*(2), 49.

Morgan JN, David MH, Cohen WJ, Brazer HE. Income and Welfare in the United States, A Study by the Survey Research Center, Institute for Social Research, University of Michigans. McGraw-Hill: New York,; 1962.

Multiple Risk Factor Intervention Trial Research Group. Multiple Risk Factor Intervention Trial: Risk Factor Changes and Mortality Results. JAMA 1982(248), 1465-1477.

Multiple Risk Factor Intervention Trial Research Group. Mortality rates after 10.5 years for participants in the Multiple Risk Factor Intervention Trial. Findings related to a priori hypotheses of the trial. JAMA-Journal of the American Medical Association 1990(263), 1795-1801.

Multiple Risk Factor Intervention Trial Research Group. Mortality after 16 years for participants randomized to the Multiple Risk Factor Intervention Trial. Circulation 1996(94), 946-951.

National Center for Health Statistics. 1963. Health Insurance Coverage United States July 1962 - June 1963. (Ed)^(Eds), vol. 2021. 1963.

National Center for Health Statistics. 1968. Hospital and Surgical Insurance Coverage United States - 1968. (Ed)^(Eds), vol. 2021. 1968.

Newhouse JP, Group RCIE, Corporation R, Staff IEG. Free for all?: lessons from the RAND health insurance experiments. Harvard University Press; 1993.

Newson RB, Falcaro M. Robit regression in Stata. The Stata Journal 2023(23), 658-682.

Niikura R, Hirata Y, Suzuki N, Yamada A, Hayakawa Y, Suzuki H, Yamamoto S, Nakata R, Komatsu J, Okamoto M. Colonoscopy reduces colorectal cancer mortality: A multicenter, long-term, colonoscopy-based cohort study. PloS one 2017(12), e0185294.

Preston S, Heuveline P, Guillot M. Demography: Measuring and Modeling Population Processess. Wiley; 2000.

Price J, Buckles K, Van Leeuwen J, Riley I. Combining family history and machine learning to link historical records: The Census Tree data set. Explorations in Economic History 2021(80), 101391.

Rohrer K, Dundes L. Sharing the Load: Amish Healthcare Financing. Healthcare (Basel, Switzerland) 2016(4), 92.

Sant'Anna PH, Zhao J. Doubly robust difference-in-differences estimators. Journal of Econometrics 2020(219), 101-122.

Seaman SR, White IR. Review of inverse probability weighting for dealing with missing data. Statistical Methods in Medical Research 2013(22), 278-295.

Stephens Jr M, Toohey D. The impact of health on labor market outcomes: evidence from a large-scale health experiment. American Economic Journal: Applied Economics 2022(14), 367-399.

Stevens RA. In Sickness and In Wealth: American Hospitals in the 20th Centurys. Johns Hopkins University Press: London; 1989.

Stevens RA. Health care in the early 1960s. Health care financing review 1996(18), 11.

Thomasson MA. The importance of group coverage: How tax policy shaped US health insurance. Am Econ Rev 2003(93), 1373-1384.

Thomasson MA. Early evidence of an adverse selection death spiral? The case of Blue Cross and Blue Shield. Explorations in Economic History 2004(41), 313-328.

Thompson O. The long-term health impacts of Medicaid and CHIP. Journal of Health Economics 2017(51), 26-40.

Van Tiggelen R, Pouders E. Ultrasound and computed tomography: Spin-offs of the world wars. JBR-BTR 2003(86), 235-241.

West H. Five years of Medicare-A statistical review. Soc. Sec. Bull. 1971(34), 17.

Wherry LR, Meyer BD. Saving Teens Using a Policy Discontinuity to Estimate the Effects of Medicaid Eligibility. Journal of Human Resources 2016(51), 556-588.

Wherry LR, Miller S, Kaestner R, Meyer BD. Childhood Medicaid Coverage and Later-Life Health Care Utilization. Rev Econ Stat 2018(100), 287-302.

Wright, J. C., & Weinstein, M. C. (1998). Gains in life expectancy from medical interventions—standardizing data on outcomes. *New England Journal of Medicine*, *339*(6), 380-386.

Wyse A, Meyer B. 2023. Saved By Medicaid: New Evidence on Health Insurance and Mortality from the Universe of Low-Income Adults. (Ed)^(Eds). 2023.

Yue D, Ettner SL, Needleman J, Ponce NA. Selective mortality and nonresponse in the Health and Retirement Study: implications for health services and policy research. Health Services and Outcomes Research Methodology 2023a(23), 313-336.

Yue D, Ponce NA, Needleman J, Ettner SL, Lleras-Muney A. Increased Schooling Reduces Hospitalization Later in Life: New Evidence with Optimal Instruments from the United States. American Journal of Health Economics 2023b(0), null.

Yusuf S, Zucker D, Peduzzi P, Fisher LD, Takaro T, Kennedy JW, Davis K, Killip T, Passamani E, Norris R, et al. Effect of coronary artery bypass graft surgery on survival: overview of 10-year results from randomised trials by the Coronary Artery Bypass Graft Surgery Trialists Collaboration. Lancet 1994(344), 563-570.

Table 1. Characteristics of white individuals linked to the 1940 full-count census

	20% Census-Tree data (N=3,697,163)		1940 full-count census (N=57,416,201)
	w/o IPW	w/ IPW	Percent or Mean
Birth cohorts based on 1940 census			
1883-1884	0.12%	0.45%	3.76%
1885-1889	10.56%	11.22%	10.91%
1890-1894	15.00%	13.71%	13.09%
1895-1899	15.85%	14.18%	13.51%
1900-1904	17.29%	15.85%	14.95%
1905-1909	17.90%	17.07%	15.75%
1910-1914	20.95%	23.09%	20.91%
1915-1917	2.32%	4.44%	7.12%
Female	46.31%	48.98%	49.79%
Hispanics			
Not Hispanic	99.36%	98.70%	98.49%
Mexican	0.45%	0.96%	1.07%
Puerto Rican	0.01%	0.04%	0.09%
Cuban	0.01%	0.03%	0.04%
Other	0.17%	0.27%	0.31%
Regions of residence in 1940			
Northeast	17.80%	28.36%	30.50%
Midwest	37.25%	33.70%	32.74%
South	30.94%	25.81%	25.01%
West	14.01%	12.12%	11.74%
Regions of birth			
Northeast	16.87%	24.50%	24.27%
Midwest	39.42%	33.31%	31.86%
South	32.30%	26.73%	25.69%
West	6.72%	5.33%	5.06%
US or outlying areas	0.02%	0.06%	0.12%
Abroad	4.67%	10.07%	13.00%
Marital status			
Married, spouse present	84.19%	74.89%	72.29%
Married, spouse absent	1.81%	3.34%	3.75%
Separated	0.00%	0.00%	0.00%
Divorced	1.16%	1.86%	1.87%
Widowed	2.04%	3.26%	3.94%
Never married/single	10.80%	16.64%	18.14%
Veteran status			
N/A	97.30%	97.43%	97.49%
Not a veteran	1.14%	1.11%	1.11%
Veteran	0.32%	0.28%	0.25%
Unknown	1.24%	1.18%	1.15%
Educational levels			
0-7 years	22.25%	22.44%	23.59%
8-11 years	45.94%	44.54%	44.45%
12+ years	29.89%	30.92%	29.60%
Missing	1.91%	2.11%	2.36%
Years of schooling (SE)	9.35 (3.25)	9.32 (3.41)	9.16 (3.46)
Household total annual income in 1940 US dollars (SE)	1295.45 (1876.93)	1556.21 (2838.90)	1533.34 (2491.17)
Occupational income score in 1940 (SE)	14.96 (14.59)	15.26 (14.57)	14.99 (14.40)

Notes: The weights are estimated using a Robit model that predicts a match as a function of the following as covariates: female dummy, years of schooling, age, 6 marital status dummies, number of children, labor force participation dummy, occupation dummies, occupation income scores, wage and salary income, veteran status (yes, no, unknown), place of birth, and state of residence in 1940.

Table 2: Estimates of Medicare's average treatment effect on the shift and Slope log of mortality by cohort group

Cohort group:	ITS Design		DiD Design	
	Shift:	Slope	Shift:	Slope
		Panel A. Men		
1885-1915	-0.023***	-0.015***	0.008	-0.018***
	(0.007)	(0.001)	(0.011)	(0.002)
1885-1900	0.022***	-0.023***	-0.000	-0.018***
	(0.007)	(0.001)	(0.007)	(0.001)
1901-1905	0.004	-0.018***	0.091***	-0.021***
	(0.014)	(0.002)	(0.030)	(0.006)
1906-1910	-0.097***	-0.014***	-0.073***	-0.024***
	(0.014)	(0.002)	(0.019)	(0.004)
1911-1915	-0.064***	0.004*	0.028	-0.012***
	(0.016)	(0.002)	(0.021)	(0.004)
		Panel B. Women		
1885-1915	-0.030***	0.008***	0.017	-0.008***
	(0.009)	(0.001)	(0.014)	(0.003)
1885-1900	0.021**	-0.001	0.017*	-0.002
	(0.009)	(0.001)	(0.010)	(0.002)
1901-1905	0.02	0.011***	0.156***	-0.019***
	(0.02)	(0.003)	(0.034)	(0.006)
1906-1910	-0.099***	0.010***	-0.063*	-0.015**
	(0.019)	(0.003)	(0.032)	(0.006)
1911-1915	-0.110***	0.019***	-0.062	0.001
	(0.021)	(0.003)	(0.040)	(0.007)

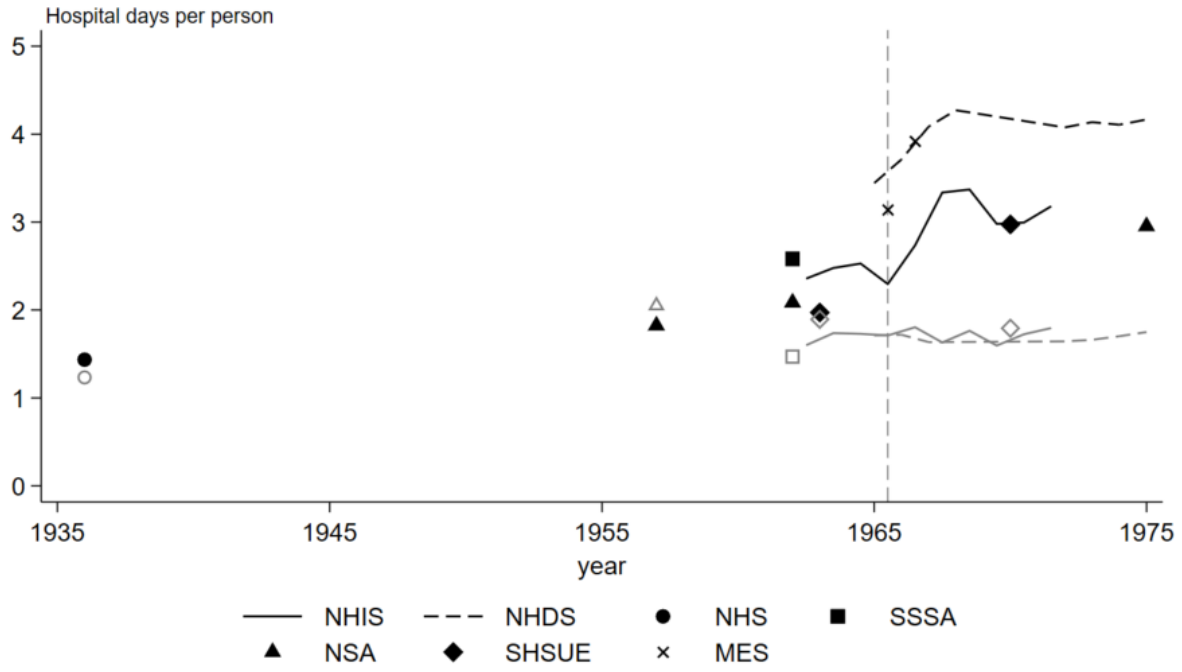
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. We then compute the logarithm of mortality as the outcome for regressions. $EventAge$ denotes the number of years relative to the Medicare eligibility age by birth cohort. All models control for birth cohort fixed effects (FE) and cohort specific linear trends over age ($Cohort\ FE * EventAge$).

Table 3. Estimates of Medicare's average treatment effect on Life Expectancy at Age 55 overall, by education, and by income in 1940

	ITS Estimates	DiD Estimates
Panel A. Males		
All men	0.82	0.93
Education		
0-7 years of schooling	0.89	0.77
8-11 years of schooling	0.78	0.85
12+ years of schooling	0.71	0.88
Household total income in 1940		
Less than or equal to Median	0.88	1.01
Above the Median	0.73	0.83
Panel B. Females		
All women	-0.50	0.68
Education		
0-7 years of schooling	0.01	1.17
8-11 years of schooling	-0.41	0.31
12+ years of schooling	-1.02	0.45
Household total income in 1940		
Less than or equal to Median	-0.69	0.51
Above the Median	-0.28	0.82

Notes: This table reports the estimated life expectancy gains associated with gaining access to Medicare. The Appendix contains the details of our computations. All estimates are produced using the CensusTree data. Standard errors will be produced by bootstrapping.

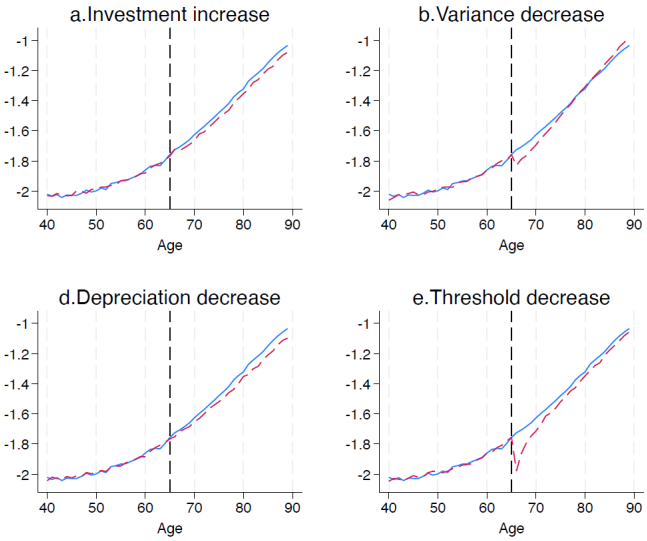
Figure 1. Health Care utilization before and after Medicare



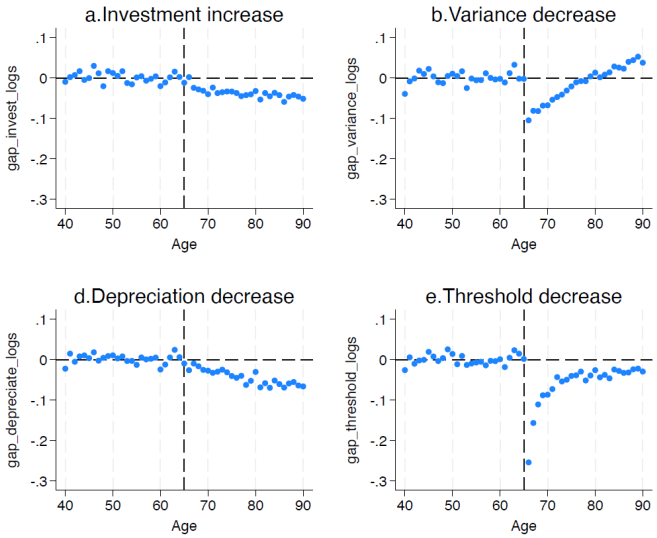
Notes: The figure shows days of hospital care per person from several data sources for individuals ages 65 and older who became eligible for Medicare in July 1966 (black markers) and individuals ages 50-64 who did not (gray markers). The data sources are: National Health Interview Survey (NHIS), the National Hospital Discharge Survey (NHDS), the National Health Survey (NHS), the Social Security Survey of the Aged (SSSA), the National Survey of Aging (NSA), the Survey of Health Services Utilization and Expenditure (SHSUE), and the Medicare Evaluation Study (MES).

Figure 2. Simulated changes of Gompertz curves after permanent unanticipated shocks at age 65

a. log of mortality with and without parameter changes at age 65

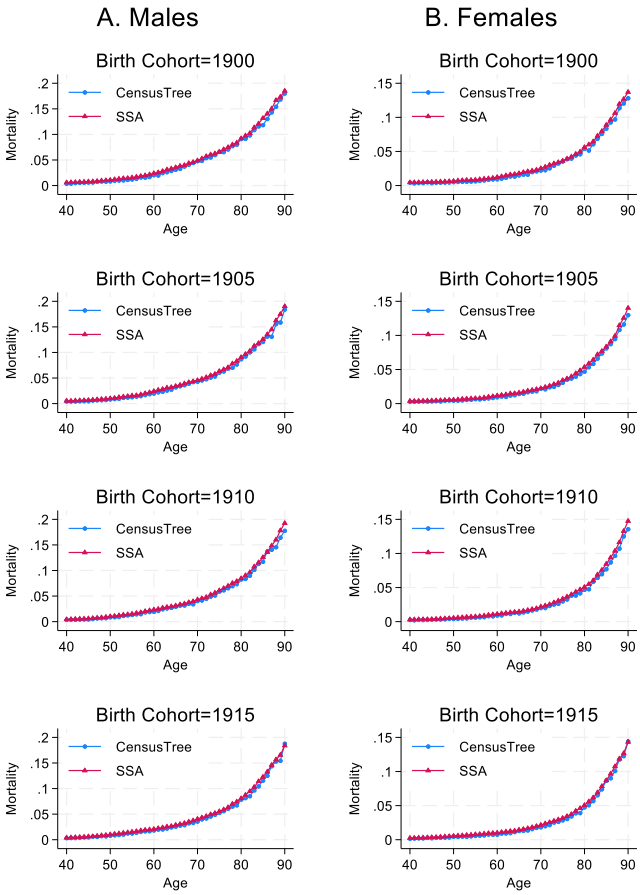


b. Effects of parameter changes at age 65 on log mortality



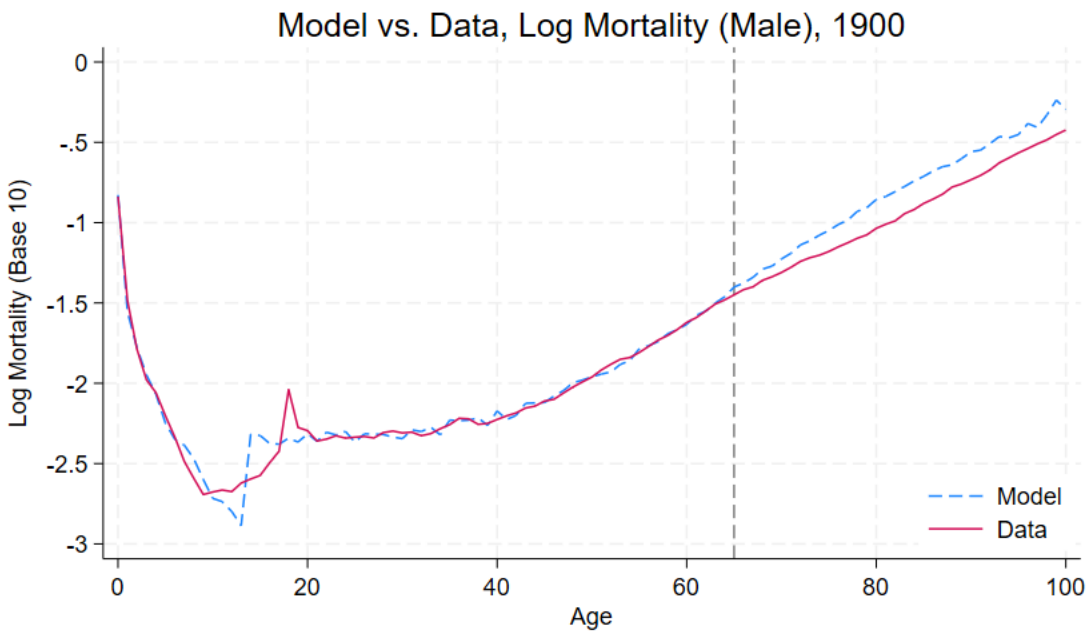
Notes: The figures show simulated results based on Lleras-Muney and Moreau (2022)'s unified model for cohort mortality, with parameter values for the baseline model given by $I = 0.4$, $\delta = 0.0006$, $\sigma = 1$, $\alpha = 1.7$, $\mu_0 = 0.9$, and an adolescent accident shock $\kappa = 0.008$. The threshold for dying is set at 0. In the top panel, the blue line shows the evolution of mortality rates by age for the baseline population and the red dashed line shows shifted Gompertz curves after permanent shocks are simulated at age 65: I is increased by 10% (investment increase), the variance σ is decreased to 0.6 (variance decrease), accidents are decreased to 0.004 (accident decrease), the depreciation rate δ is lowered by 5% (depreciation decrease), and finally the threshold is lowered from 0 to -1 (threshold decrease). In the bottom panel we show the implied evolution of the gaps in logs, which are given by the distance between the solid blue line and the dashed red line.

Figure 3. Comparison of mortality between Census Tree and SSA life tables for selected cohorts



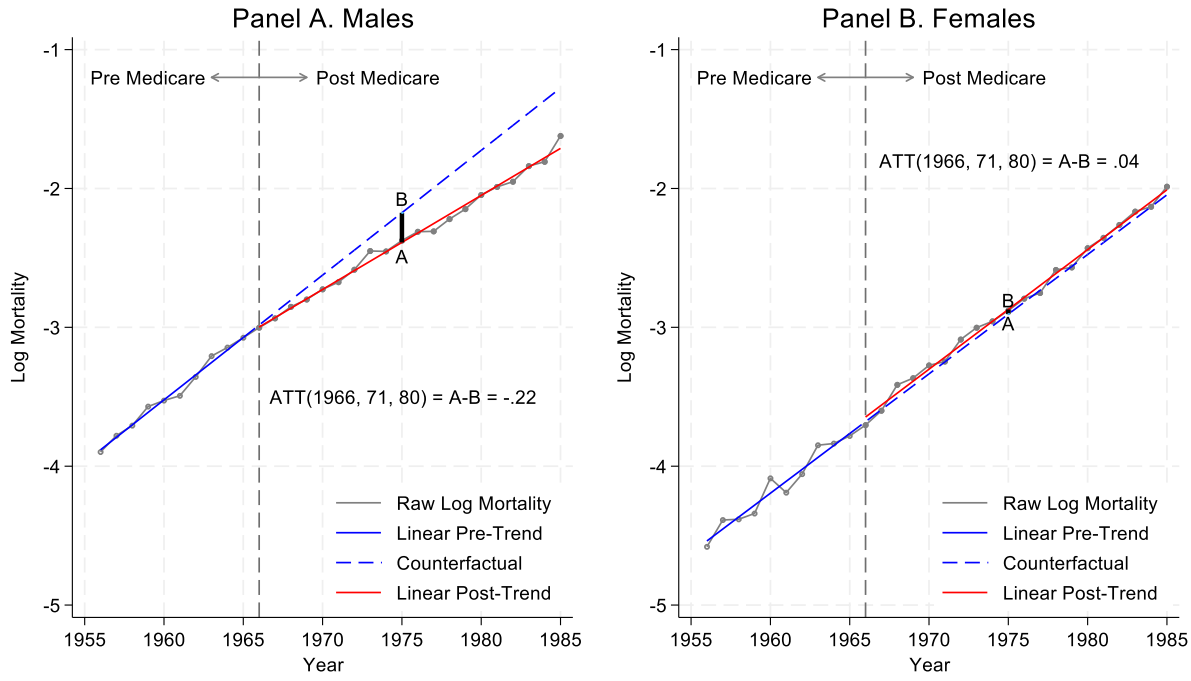
Notes: Shown are age specific mortality rate by cohort, sex, and data source. The CensusTree data represent a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. We compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. SSA represents mortality data from the Social Security Administration life tables. Similar results were found for other birth cohorts 1885-1915.

Figure 4. Identification of Medicare's effect on one cohort's log mortality rates in the structural design



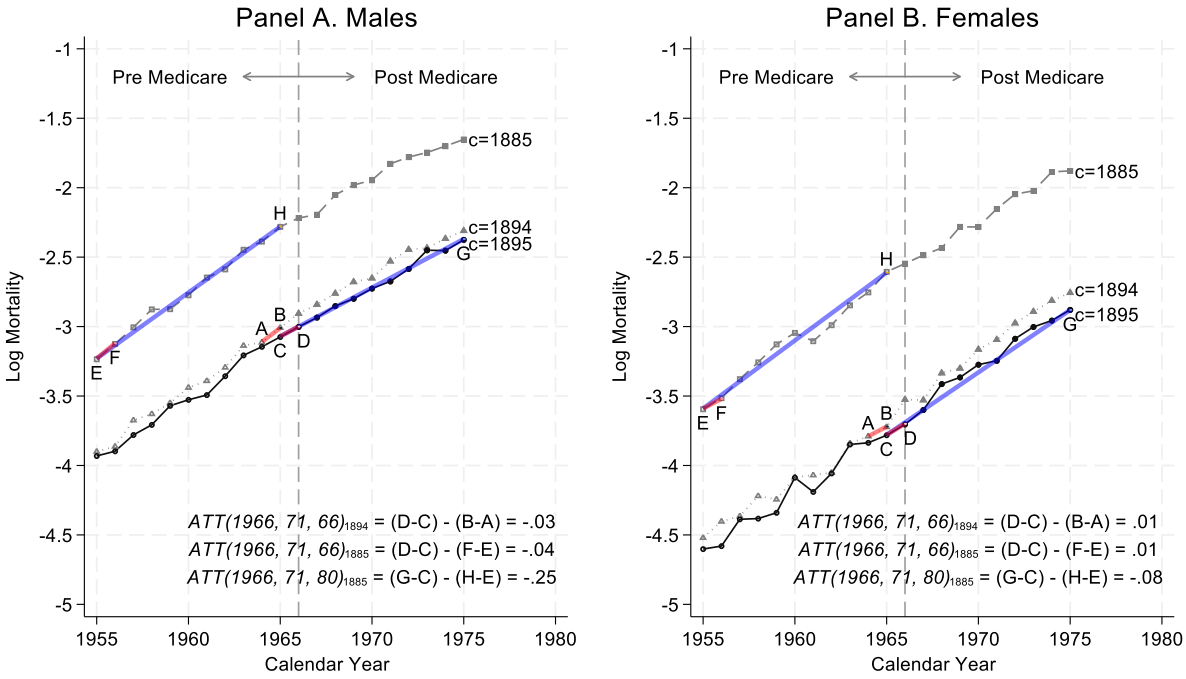
Notes: This figure demonstrate how the model can be used for identification using the Social Security Administration cohort tables (dashed blue line) starting at birth. We first estimate the model using data up to age 65. We then project mortality rates beyond age 65 using the estimated parameters (solid red line). The estimated parameters for this cohort and other cohorts we study are presented in Table A.1.

Figure 5. Identification of Medicare's effect on one cohort's log mortality rates in the interrupted time series design



Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. The figures show the log mortality (Gompertz curve) for the 1895 cohort by sex. The blue solid line is a linear fit of log mortality against age over 10 years prior to Medicare eligibility age (pre-trend). The blue dashed line is a linear project of the pre-trend of log mortality after Medicare eligibility to age 90, representing the counterfactual without Medicare's introduction. The red solid line represents the linear fit of the post-Medicare trend in log mortality from Medicare eligibility age to age 90.

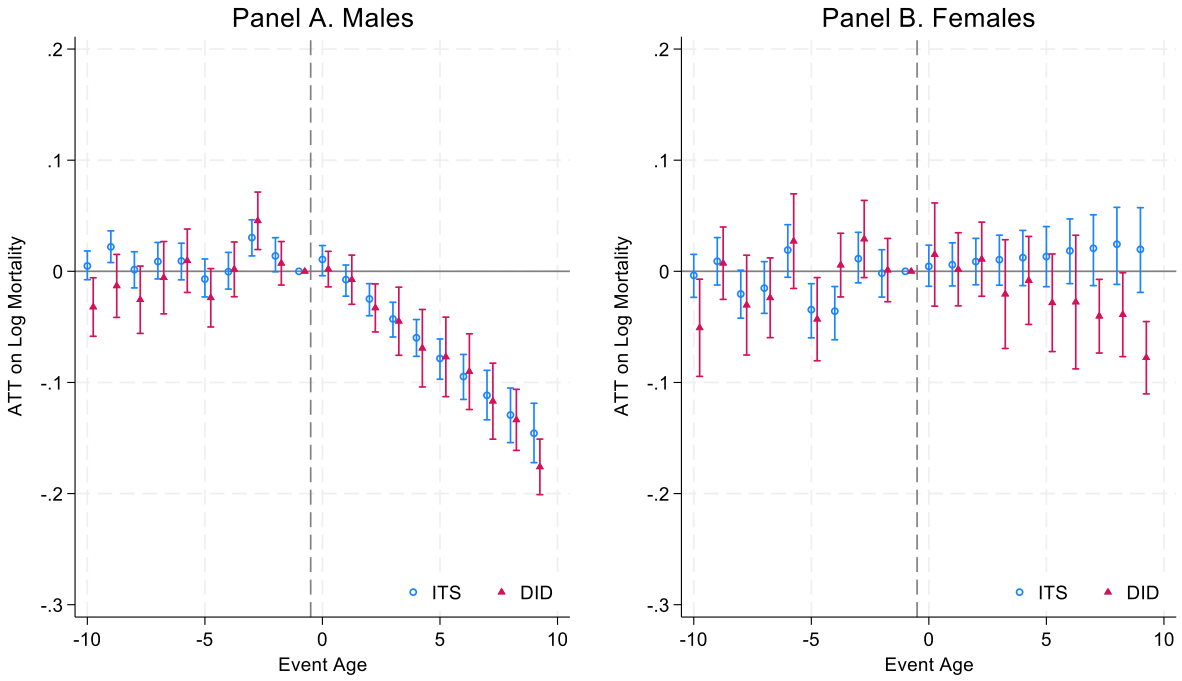
Figure 6. Identification of Medicare's effect on one cohort's log mortality rates in the difference-in-differences design



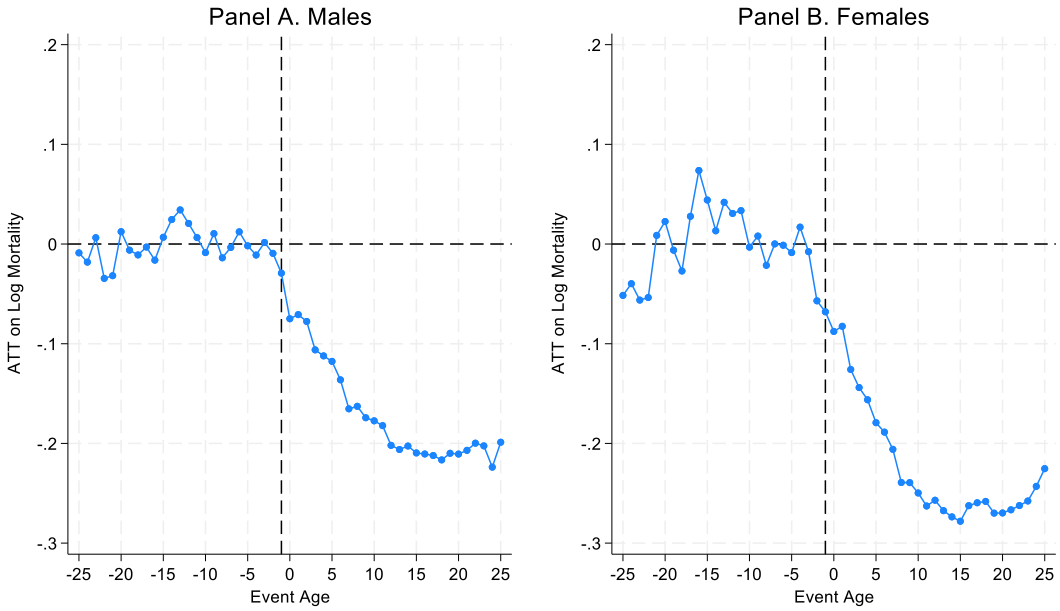
Notes: This figure highlights the estimator for $ATT(g_t, g_a, t)$ for the 1895 cohort as an example. We describe the treatment effect for the Medicare's introduction year ($t = 1966$) and 9 years after the introduction. This effect is identified by comparing the change in the (log) mortality rate before (C) and after treatment (D) (at ages 70 and 71) for the 1895 cohort, to the change for each of the 1885-1894 cohorts in the previous year prior to becoming eligible for Medicare. The identifying assumption is parallel trends between pairs of cohorts and pairs of ages ($PT(c, a)$), namely that the change in mortality for the 1885-1894 cohort between ages 70 and 71 (e.g., B-A, or F-E) serves as a counterfactual for the change in mortality for the 1895 cohort because they would have evolved in parallel if the 1895 cohort had not been treated. This assumption can be partially tested by checking whether the mortality rates of the two cohorts were in fact parallel prior to age 71. Similarly, the treatment effect for the 1895 cohort 9 years after Medicare's introduction use (and only use) the 1885 cohort as the comparison group. The implied estimate of the effect of Medicare 9 years after its introduction year for the 1895 cohort is illustrated in this figure as (G-C)-(H-E), which -0.25 for males and -0.08 for females.

Figure 7. Estimates of Medicare's average treatment effect on log mortality rates by event-age

a-1885-1915 cohorts – ITS and DiD approaches

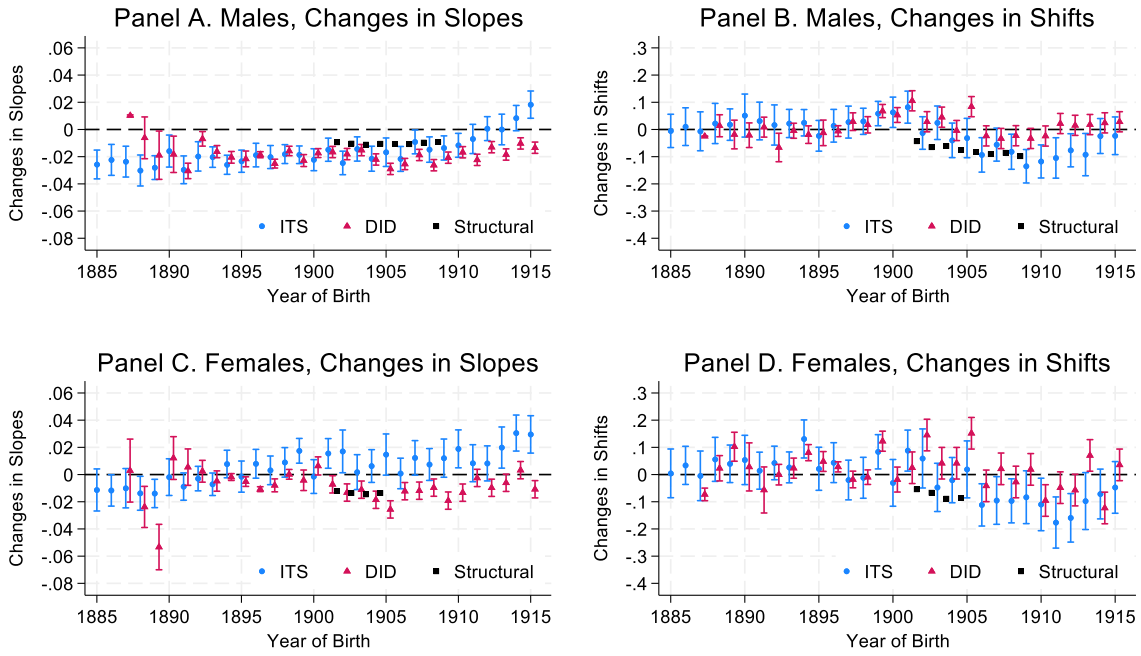


b-Structural Approach for selected cohorts



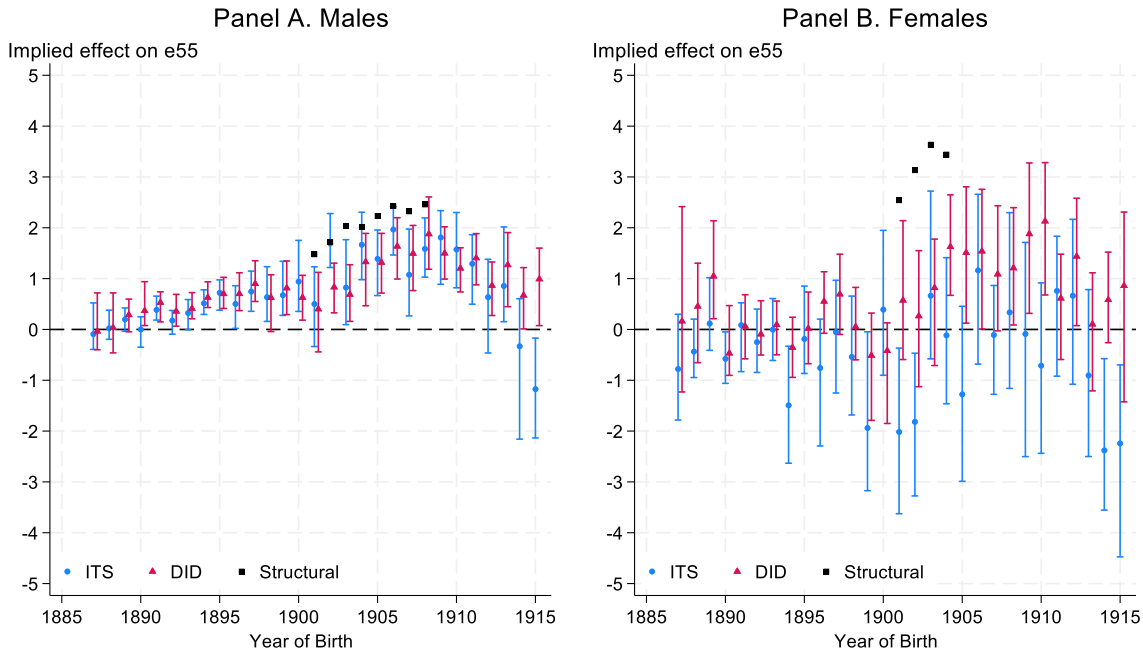
Notes: The figure 5a plots the estimates of $ATT(e)$ parameters from equation (Y) for men and women using both the ITS and DiD designs across all birth cohorts. Figure 5b reports the estimates for the subset of cohorts available in the SSA life tables for which we have complete cohort data from birth to death and for which the structural estimation has an acceptable fit (see text for details). The bottom panel therefore only includes the 1901-1908 birth cohorts for men and the 1901-1904 birth cohorts for women.

Figure 8. Estimates of Medicare's average treatment effect on the Intercept and Slope of log Mortality rates by age



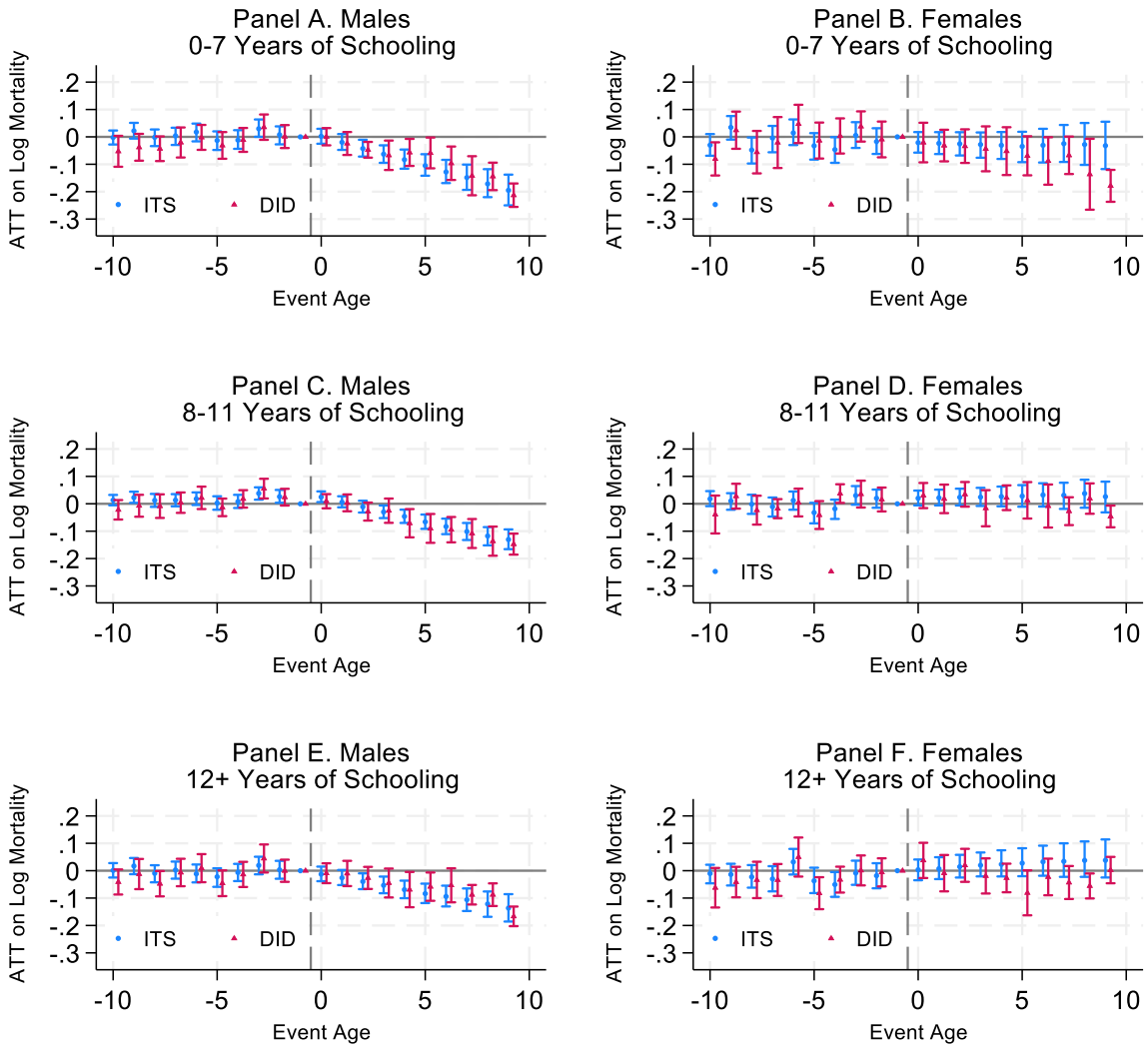
Notes: We use an interrupted time series approach to estimate changes in slopes and levels of log mortality, after Medicare's introduction in 1966, by gender and birth cohort using three different methods. The blue circle (ITS approach) and red triangle (DiD) estimates are from a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. The black squares estimates are derived from the Social Security Administration life tables, which are available for the subset of cohorts available in the SSA life tables for which we have complete cohort data from birth to death and for which the structural estimation has an acceptable fit (see text for details). Therefore, they only include the 1901-1908 birth cohorts for men and the 1901-1904 birth cohorts for women. We compute the slope and intercept estimates for these cohorts only using the first 10 years of data to match what we estimate in the other 2 approaches.

Figure 9. Estimates of Medicare's average treatment effect on life expectancy at age 55 by cohort (50 resamples)



Notes: Shown are the implied effects of Medicare's introduction on life expectancy at age 55 based on our ITS, DiD and structural estimation approaches by sex, birth cohort, and data source. The blue and red estimates are from a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rates using the inverse probability weights, separately by sex and birth cohort. We use a bootstrapping approach with 1,000 resamples to obtain the standard error and 95% confidence intervals for our analysis using Census Tree microdata. The black dots correspond to the structural model estimates which are only available for selected cohorts. Therefore, they only include the 1901-1908 birth cohorts for men and the 1901-1904 birth cohorts for women. We have not computed standard errors yet for these estimates.

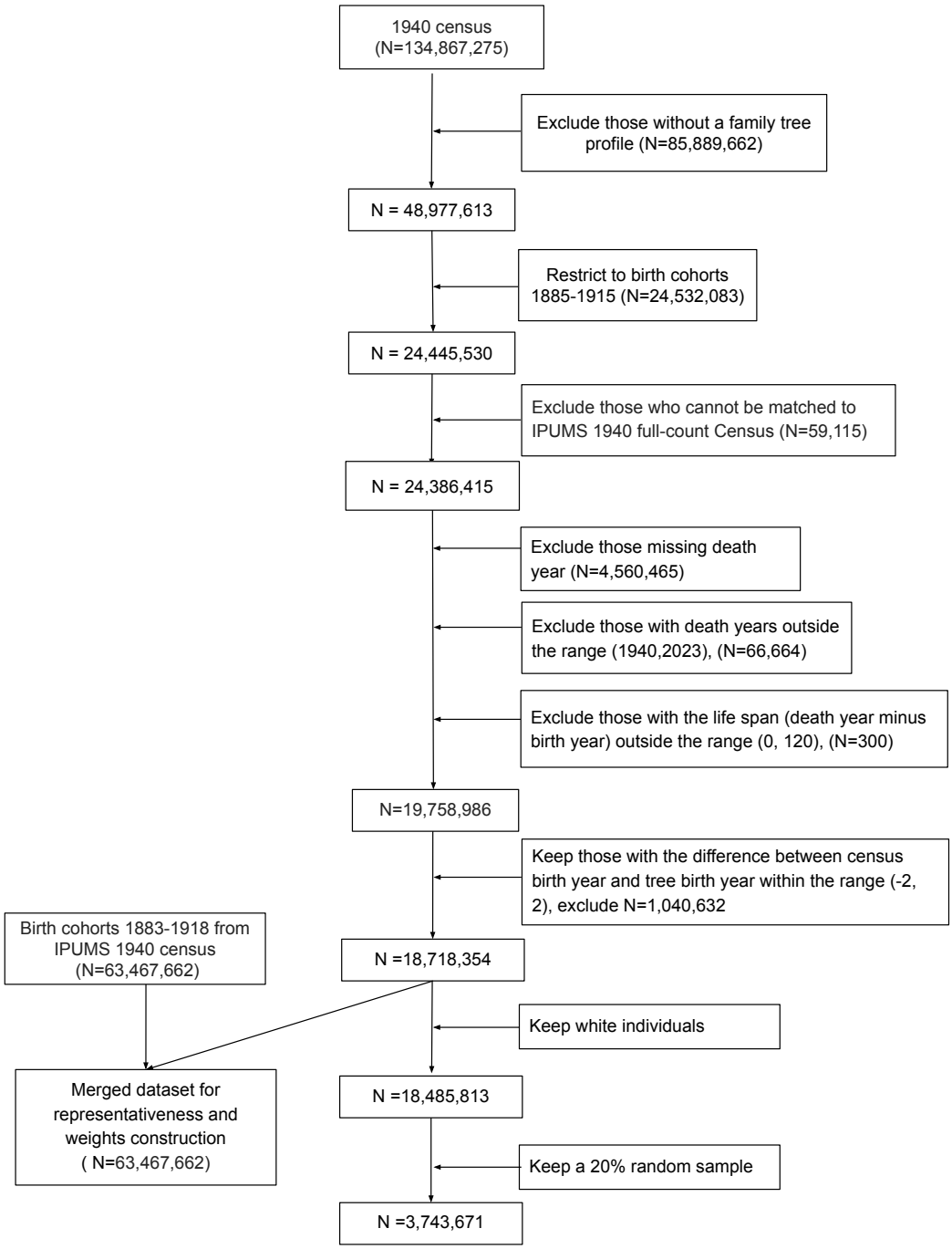
Figure 10. Estimates of Medicare's average treatment effect on log mortality rates by event-age and education group



Appendix

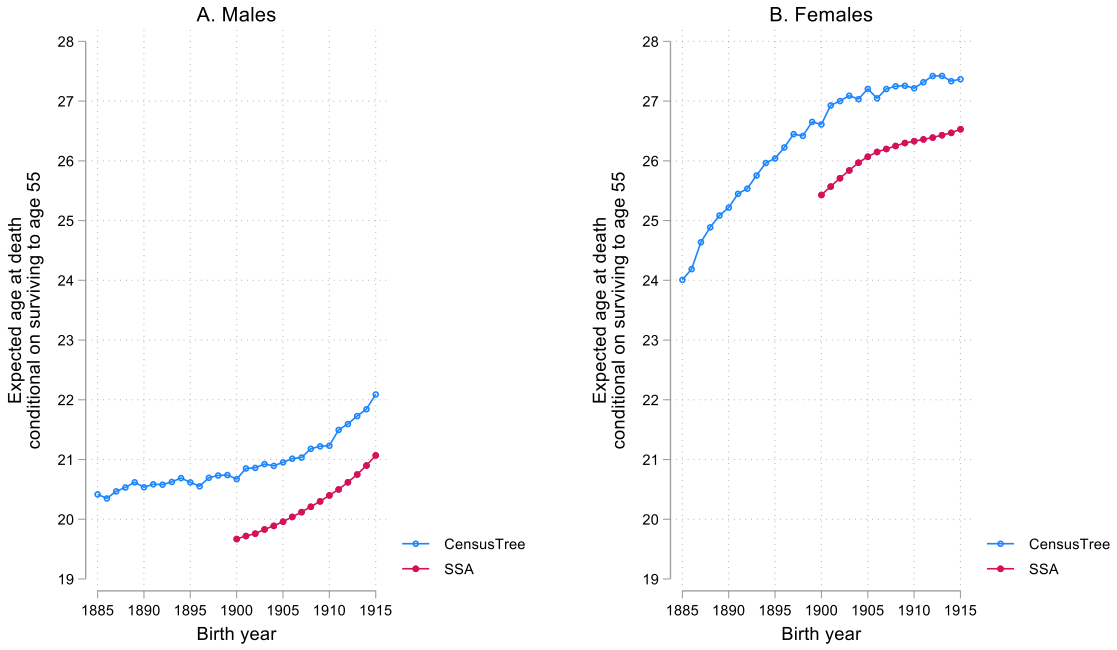
1. Figures
2. Tables
3. Computation of implied effect on ex55 based on the ITS analysis
4. Computation of implied effect on ex55 based on the DiD analysis (TBA)
5. Details on the estimation of the structural model (TBA)
6. Behavioral responses to Medicare (TBA).

Figure A1. Sample flowchart of Census Tree Sample



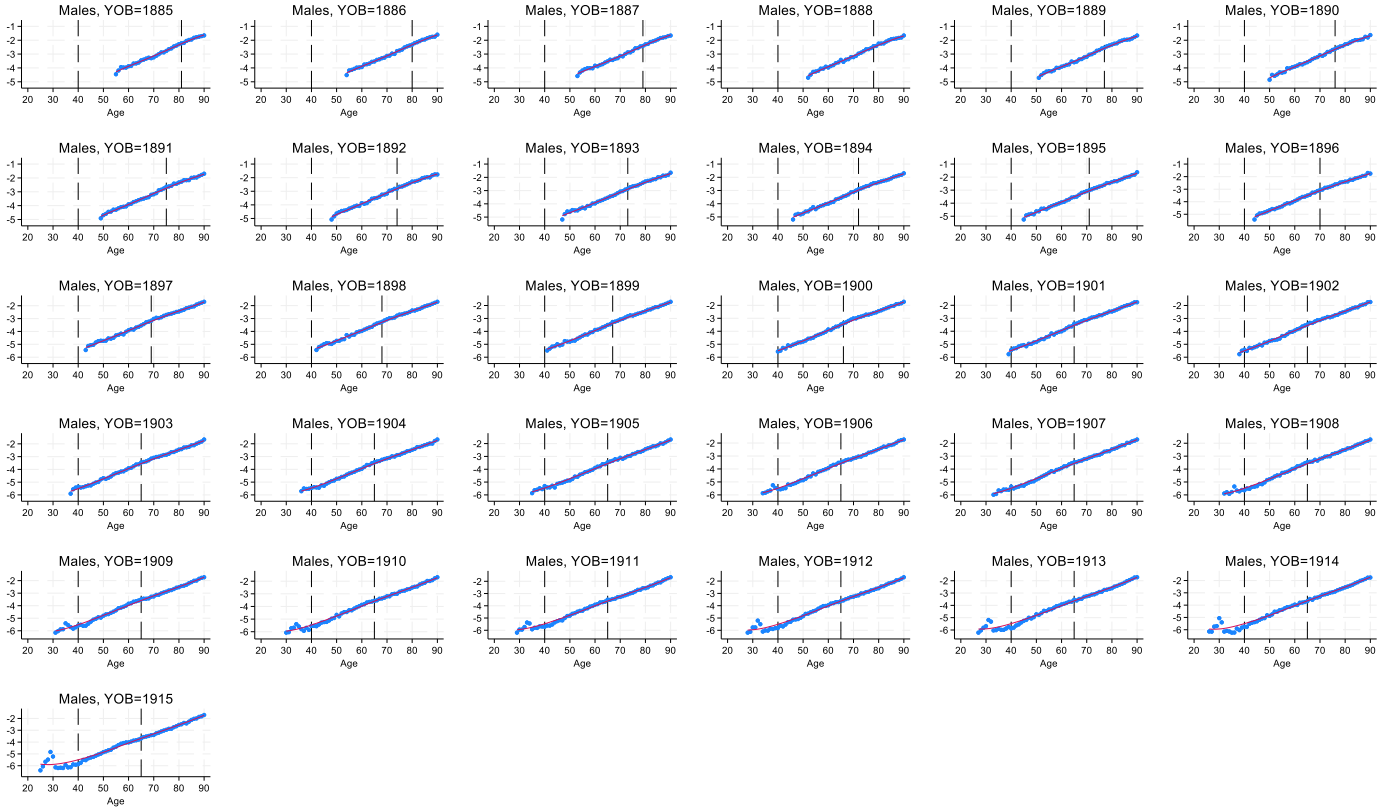
Notes: We rely on the year of birth recorded in the Family Tree in our analysis because the 1940 Census does not ask for birth year and only reports age. However, the birth year variable from Family Tree was scrapped because people would have had the chance to find birth records and make corrections. To exclude Census Tree observations with potential errors in birth year, we restricted the analytic sample to those the difference between census computed birth year and Census Tree recorded birth year within the range (-2, 2). We use Census Tree year of birth in our analysis. To construct the inverse probability weights (IPW), we the sample (N=18,718,354) to the IPUMS 1940 full-count census and further restricted to birth cohorts 1883-1917 based on 1940-census-computed birth year, leading to a sample of 63,467,662.

Figure A2. The expected age at death conditional on survival to age 55.



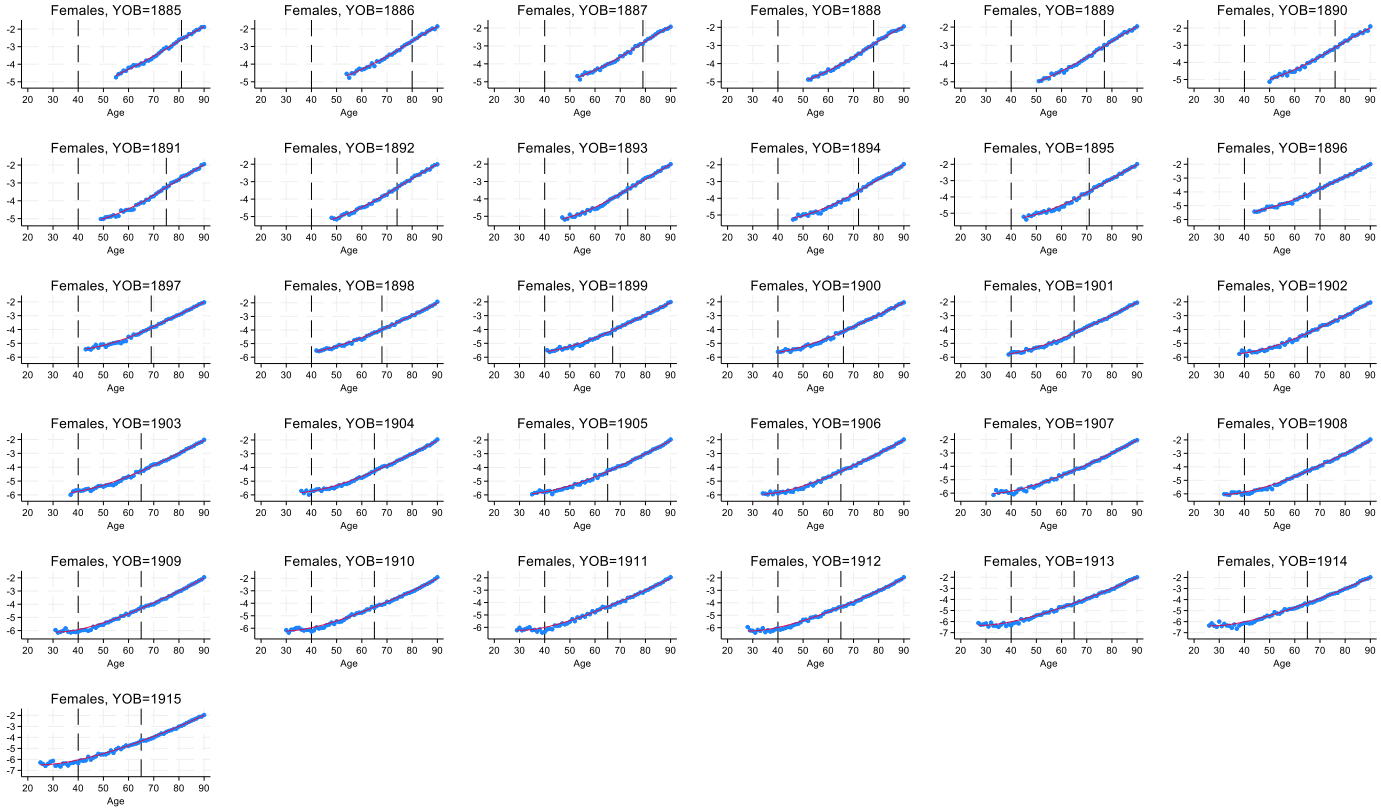
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort.

Figure A3a. Gompertz curves for studied birth cohorts of white males. (updated)



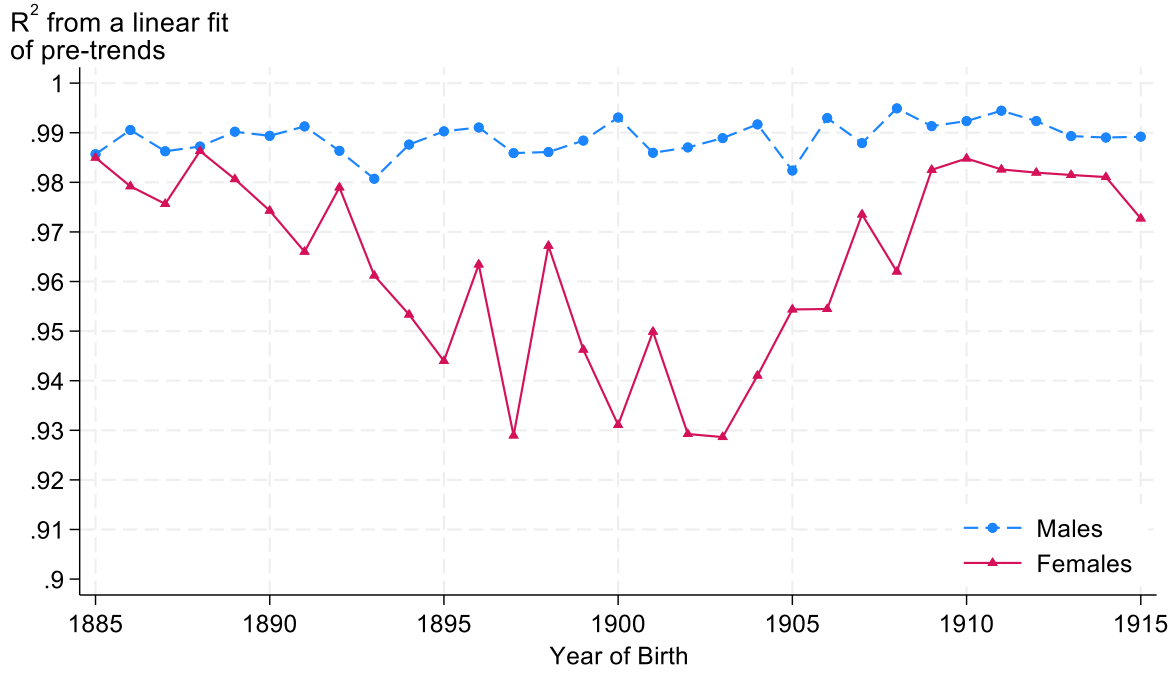
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. We truncate the age at death at age 90. The y-axis is the log of mortality rates and the x-axis is age. We observe cohorts starting in 1940 which is why the curves start at different ages for different cohorts.

Figure A3b. Gompertz curves for studied birth cohorts of white females.



Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. We truncate the age at death at age 90. The y-axis is the log of mortality rates and the x-axis is age. We observe cohorts starting in 1940 which is why the curves start at different ages for different cohorts.

Figure A4. Estimated R^2 from a linear regression model of log mortality by sex and birth cohort over the pre-trends period.



Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. We truncate the age at death at age 90. We obtain the R^2 from a linear regression model of log mortality on age in the age window from $Max(40, 1940 - yob)$ to $(1966 - yob)$, by sex for each birth cohort. We then plot the R-squared against birth cohorts. Whites only.

Figure A.5 Estimates of Medicare's average treatment effect on log mortality rates by event-age and cohort group

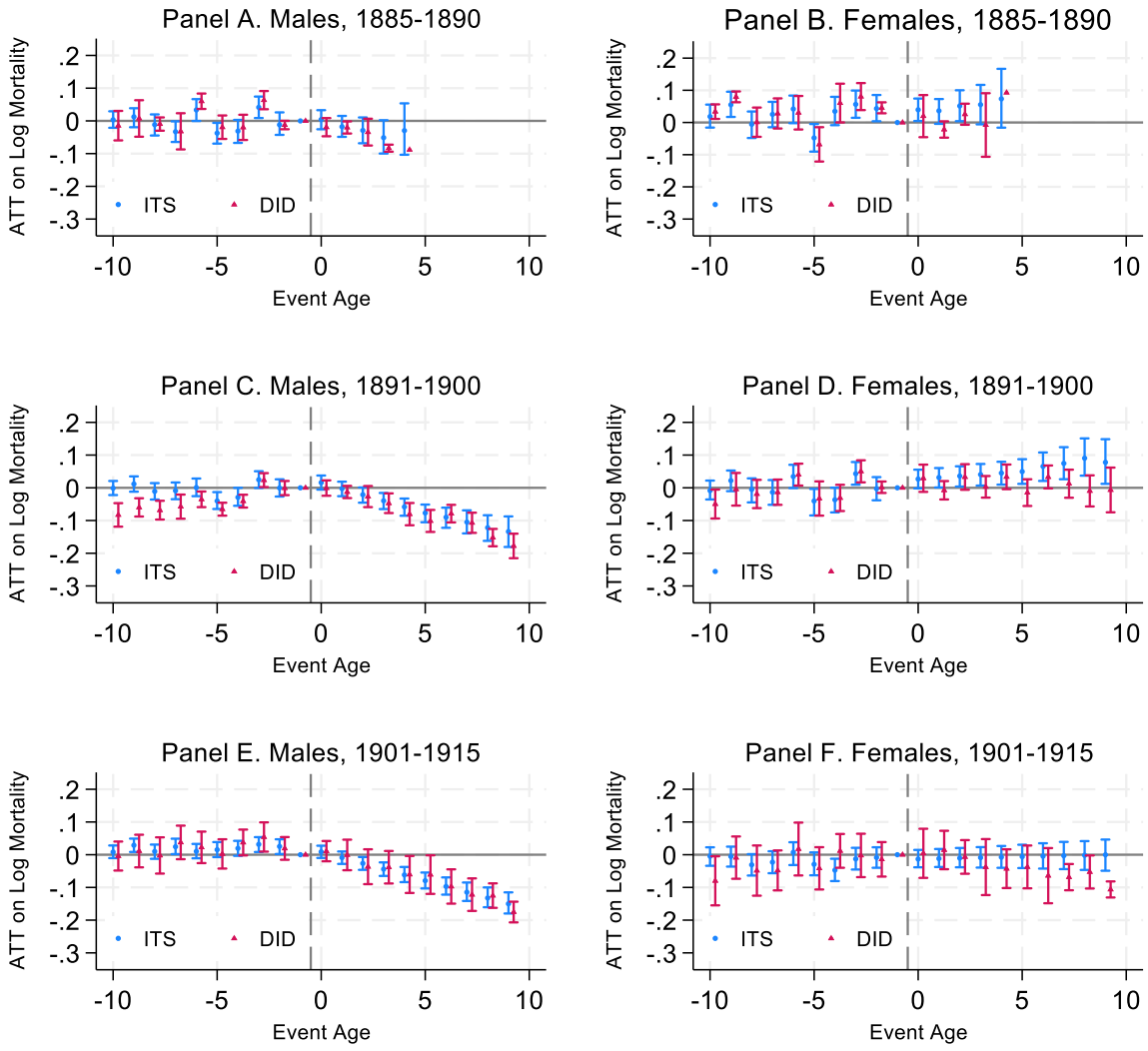
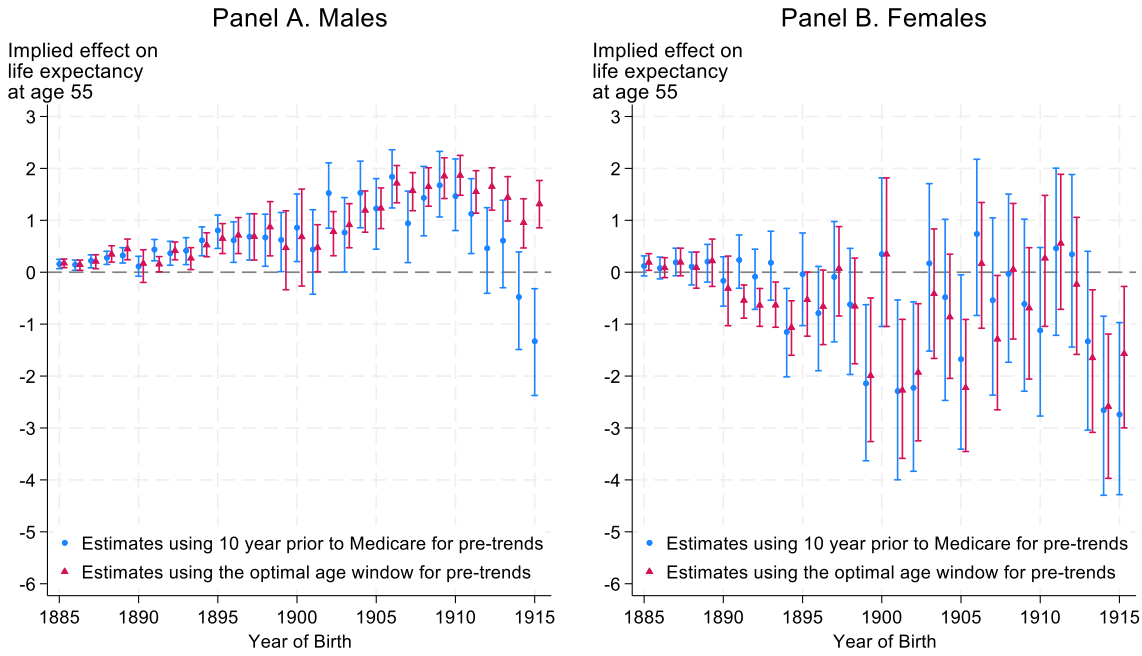


Figure A6. Implied effect of Medicare on life expectancy at age 55 using the ITS approach with the optimal age window for pre-trends.



Notes: Shown are implied effect of Medicare's introduction on life expectancy at age 55 based on our interrupted time series (ITS) approach, by sex, birth cohort, and approach. All estimates are from a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. The blue circle estimates are estimated treatment effects from ITS analysis that uses 10 year prior to Medicare eligibility age to fit a linear pre-trend of Gompertz curves. The red triangle estimates are also estimated treatment effects but use the age window from selected break age to one year prior to Medicare to fit a linear trend of Gompertz curves. The selected break age from a 5-fold cross validation is 46 for 1901-1915 male cohorts, 59 for 1891-1900 male cohorts, 70 for 1885-1890 male cohorts, 52 for 1901-1915 female cohorts, 56 for 1891-1900 female cohorts, 69 for 1885-1890 male cohorts. We use a bootstrapping approach with 1,000 resamples to obtain the standard error and 95% confidence intervals for our analysis using Census Tree microdata.

Figure A7. Robustness checks by restricting comparison cohorts.

Figure 7a. Event-study estimates for males.

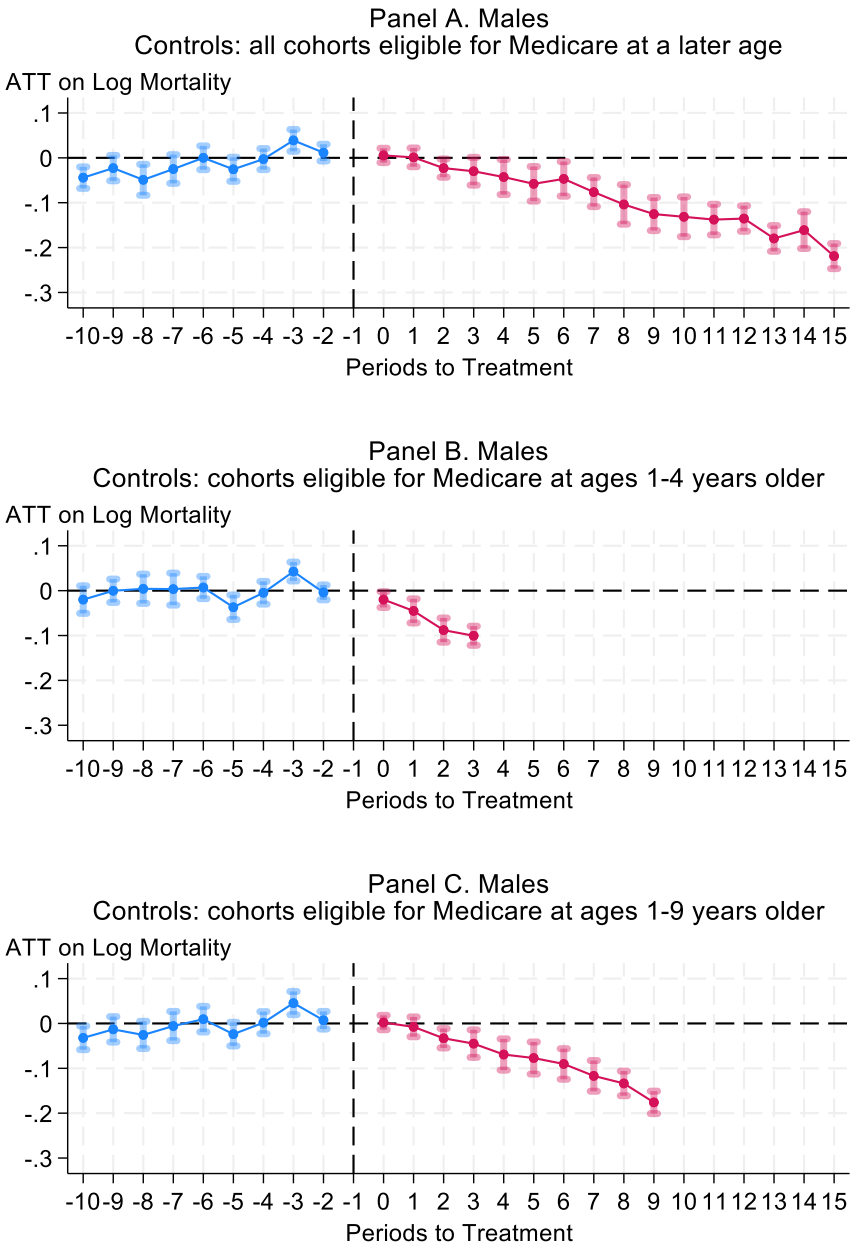
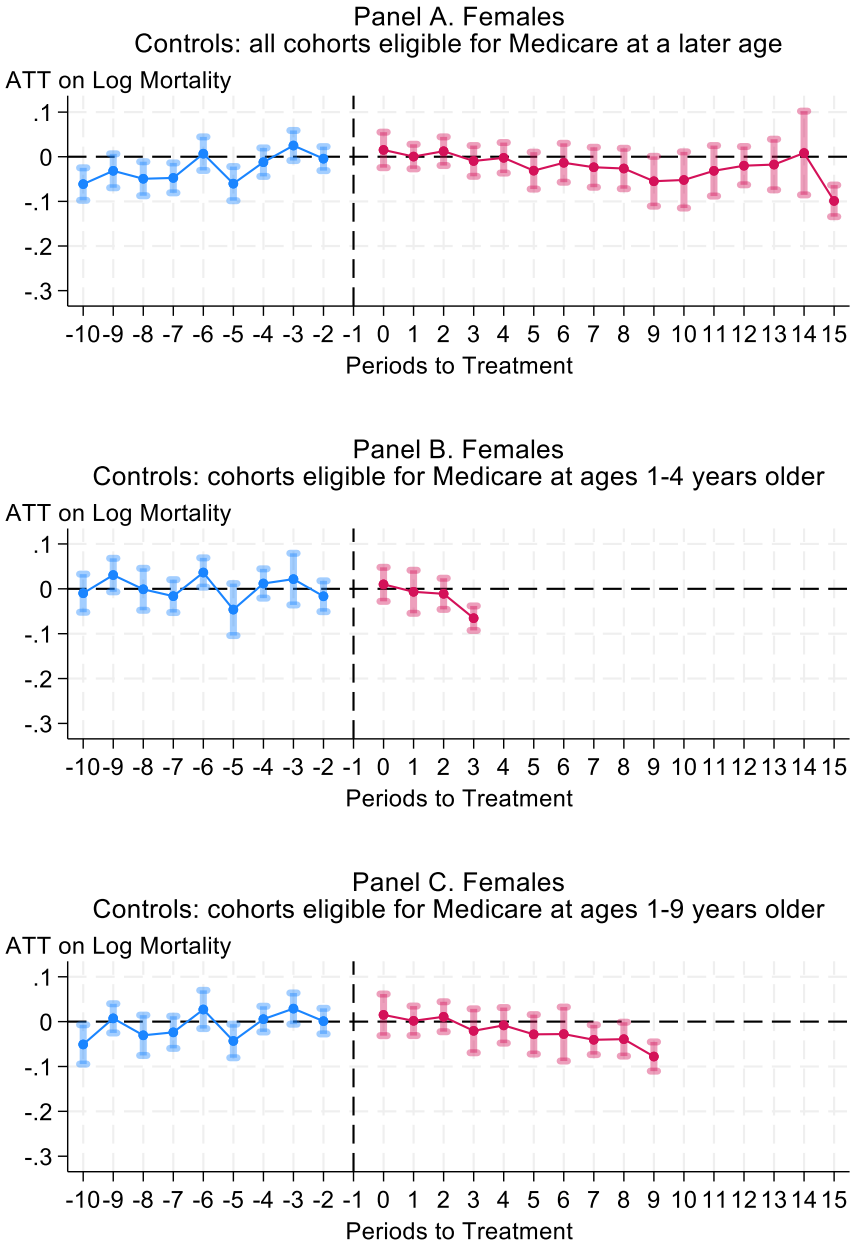


Figure 7b. Event-study estimates for females.



Appendix Table 1a: Parameter estimates by cohort. Men born 1900-1915, SSA cohort tables

Year	I	δ	σ_ε	α	μ_H	κ	I_{flu}	Fit
1900	0.1884	0.00058	0.3954	1.5374	1.0333	0.00359	0.1439	0.6398
1901	0.1871	0.00061	0.4007	1.5303	1.1133	0.00334	0.1481	0.4803
1902	0.1903	0.00061	0.4047	1.5399	1.1294	0.00324	0.1536	0.2678
1903	0.1930	0.00061	0.4077	1.5474	1.1483	0.00317	0.1612	0.3573
1904	0.1993	0.00062	0.4124	1.5463	1.1307	0.00299	0.1610	0.2612
1905	0.2057	0.00062	0.4153	1.5517	1.1198	0.00292	0.1656	0.4252
1906	0.2123	0.00063	0.4176	1.5519	1.1129	0.00283	0.1696	0.4742
1907	0.2218	0.00065	0.4298	1.5467	1.1442	0.00252	0.1690	0.4807
1908	0.2247	0.00065	0.4273	1.5452	1.1617	0.00245	0.1699	0.6200
1909	0.2304	0.00067	0.4301	1.5420	1.1845	0.00235	0.1738	0.9524
1910	0.2371	0.00067	0.4291	1.5433	1.1722	0.00219	0.1721	1.1078
1911	0.2359	0.00068	0.4337	1.5428	1.2512	0.00206	0.1772	1.4623
1912	0.2497	0.00069	0.4363	1.5423	1.2493	0.00198	0.1790	2.0922
1913	0.2662	0.00070	0.4356	1.5383	1.2355	0.00182	0.1785	2.7505
1914	0.2688	0.00070	0.4273	1.5397	1.2656	0.00179	0.1799	4.2626
1915	0.2802	0.00071	0.4157	1.5345	1.2677	0.00181	0.1802	6.3186

Notes: This table shows the parameter estimates from the model. We use the Social Security Administration cohort tables for each birth cohort to estimate the model proposed by Lleras-Muney and Moreau (2022). WE make one modification to the basic model: we allow for the investment parameter to change in 1918, to account for the extra-mortality in the year of the flu pandemic. We experimented with modeling this shock differently, but this choice does not affect the estimates of Medicare substantially. The fit measure is given by the value of the objective function used to find the parameters – we minimize the difference between the observed and predicted survival curves at every age. More precisely the fit measure is the sum of squared deviations from the survival curve up to the age we fit to - in this case, 0-65.

Appendix Table 1b: Parameter estimates by cohort. Women born 1900-1915, SSA cohort tables

Year	I	δ	σ_ε	α	μ_H	κ	I_{flu}	Fit
1900	0.2360	0.00078	0.4593	1.5191	1.1603	0.00397	0.2272	0.6116
1901	0.2351	0.00077	0.4649	1.5247	1.2353	0.00380	0.2303	0.5141
1902	0.2399	0.00078	0.4688	1.5331	1.2480	0.00370	0.2391	0.3774
1903	0.2430	0.00078	0.4733	1.5422	1.2706	0.00361	0.2493	0.4558
1904	0.2461	0.00079	0.4725	1.5471	1.2522	0.00352	0.2580	0.4929
1905	0.2475	0.00080	0.4730	1.5491	1.2459	0.00338	0.2627	0.6963
1906	0.2548	0.00079	0.4761	1.5602	1.2385	0.00331	0.2694	0.8584
1907	0.2603	0.00079	0.4816	1.5655	1.2653	0.00321	0.2747	0.9528
1908	0.2660	0.00078	0.4859	1.5759	1.2872	0.00313	0.2819	1.1748
1909	0.2708	0.00078	0.4894	1.5852	1.3090	0.00304	0.2890	1.5844
1910	0.2761	0.00079	0.4903	1.5823	1.3009	0.00291	0.2883	2.0652
1911	0.2433	0.00074	0.4696	1.5805	1.3870	0.00266	0.2683	2.3380
1912	0.2735	0.00078	0.5013	1.5750	1.3970	0.00242	0.2756	2.4573
1913	0.2918	0.00078	0.5052	1.5802	1.3826	0.00238	0.2813	3.3434
1914	0.3124	0.00078	0.5171	1.5849	1.4087	0.00223	0.2854	3.8250
1915	0.3213	0.00078	0.5175	1.5938	1.4165	0.00214	0.2908	4.8505

Notes: This table shows the parameter estimates from the model. We use the Social Security Administration cohort tables for each birth cohort to estimate the model proposed by Lleras-Muney and Moreau (2022). WE make one modification to the basic model: we allow for the investment parameter to change in 1918, to account for the extra-mortality in the year of the flu pandemic. We experimented with modeling this shock differently, but this choice does not affect the estimates of Medicare substantially. The fit measure is given by the value of the objective function used to find the parameters – we minimize the difference between the observed and predicted survival curves at every age. More precisely the fit measure is the sum of squared deviations from the survival curve up to the age we fit to - in this case, 0-65.

Table A.2: Testing whether other events in 1965 affected mortality rates

a. Results for men

Cohort:	1902	1903	1904	1905	1906	1907	1908
year	0.079**	0.080**	0.082**	0.079**	0.086**	0.082**	0.088**
	(49.04)	(48.47)	(50.55)	(32.98)	(45.50)	(35.17)	(42.16)
post1966	0.099	0.096*	0.097*	0.090	0.025	0.016	-0.009
	(1.67)	(1.68)	(1.99)	(1.43)	(0.56)	(0.34)	(-0.24)
slope1966	-0.003	-0.055	-0.014	-0.003	0.007	0.008	0.004
	(-0.06)	(-0.74)	(-0.42)	(-0.10)	(0.43)	(0.58)	(0.41)
postage65	0.000	0.085	-0.011	-0.008	-0.094*	-0.085	-0.099*
	(.)	(0.71)	(-0.15)	(-0.09)	(-1.72)	(-1.52)	(-2.34)
slopeage65	-0.007	0.044	0.002	-0.005	-0.022	-0.018	-0.019*
	(-0.12)	(0.59)	(0.05)	(-0.17)	(-1.41)	(-1.34)	(-2.22)
constant	-158.929**	-160.178**	-164.413**	-158.525**	-173.184**	-165.743**	-176.976**
	(-50.51)	(-49.92)	(-52.04)	(-34.00)	(-46.81)	(-36.23)	(-43.36)
F-test1966	4.784	1.509	2.785	1.683	0.682	0.532	0.084
p-value1966	0.013	0.232	0.072	0.197	0.511	0.591	0.919
F-testAge65	0.013	0.276	0.095	0.022	1.477	1.178	2.967
p-valueAge65	0.908	0.760	0.910	0.978	0.239	0.317	0.062

Cohort	1909	1910	1911	1912	1913	1914	1915
year	0.090**	0.089**	0.097**	0.096**	0.104**	0.094**	0.095**
	(33.72)	(32.80)	(34.80)	(33.14)	(26.74)	(25.61)	(18.92)
post1966	0.035	-0.016	-0.046	0.028	0.004	0.080*	0.042
	(0.80)	(-0.40)	(-1.24)	(0.80)	(0.10)	(2.25)	(0.97)
slope1966	-0.004	-0.001	-0.014*	-0.019**	-0.028**	-0.024**	-0.017**
	(-0.47)	(-0.15)	(-2.44)	(-3.66)	(-4.78)	(-4.85)	(-2.82)
postage65	-0.130**	-0.128**	-0.105**	-0.087*	-0.090*	-0.031	-0.099**
	(-2.79)	(-3.11)	(-2.87)	(-2.66)	(-2.37)	(-0.99)	(-2.78)
slopeage65	-0.012	-0.014*	-0.007	-0.002	0.000	0.007*	0.001
	(-1.48)	(-2.19)	(-1.32)	(-0.49)	(0.11)	(1.97)	(0.26)
constant	-180.499**	-179.416**	-194.753**	-193.691**	-208.423**	-188.776**	-191.095**
	(-34.68)	(-33.74)	(-35.73)	(-34.04)	(-27.43)	(-26.34)	(-19.46)
F-test1966	0.329	0.128	4.854	6.753	11.431	16.123	5.598
p-value1966	0.722	0.880	0.012	0.003	0.000	0.000	0.007
F-testAge65	4.219	4.836	4.409	4.627	4.465	4.977	5.689
p-valueAge65	0.021	0.013	0.018	0.015	0.017	0.011	0.006

Notes: F-test1966 denotes the joint F test of changes in both levels and slopes in 1966, and p-value1966 denotes the corresponding P value. F-testAge65 denotes the joint F test of changes in both levels and slopes at age 65, and p-value1966 denotes the corresponding P value. We restricted the age range to (40, 90).
*p<0.1, ** p<0.05.

Table A.2: Testing whether other events in 1965 affected mortality rates
b. Results for women

Cohort	1902	1903	1904	1905	1906	1907	1908
year	0.054**	0.050**	0.051**	0.056**	0.057**	0.064**	0.058**
	(20.45)	(21.43)	(20.54)	(19.92)	(21.31)	(22.82)	(18.07)
post1966	0.107	0.311**	0.144*	0.039	0.131*	0.117*	0.126*
	(1.10)	(3.82)	(1.93)	(0.52)	(2.09)	(2.02)	(2.13)
slope1966	0.089	-0.066	0.066	0.043	0.043*	0.003	0.029*
	(0.92)	(-0.62)	(1.28)	(1.22)	(1.93)	(0.16)	(2.13)
postage65	0.000	0.058	-0.094	-0.023	-0.137*	-0.061	-0.103
	(.)	(0.34)	(-0.81)	(-0.23)	(-1.78)	(-0.91)	(-1.58)
slopeage65	-0.055	0.104	-0.028	-0.009	-0.009	0.026	0.005
	(-0.56)	(0.98)	(-0.55)	(-0.27)	(-0.40)	(1.63)	(0.36)
_cons	-111.198**	-103.827**	-104.394**	-115.357**	-117.292**	-130.045**	-118.751**
	(-21.46)	(-22.57)	(-21.63)	(-20.90)	(-22.34)	(-23.82)	(-18.96)
F-test1966	6.865	10.129	8.067	2.369	11.247	3.482	9.777
p-value1966	0.002	0.000	0.001	0.105	0.000	0.039	0.000
F-testAge65	0.319	1.745	0.402	0.035	3.328	7.819	4.132
p-valueAge65	0.575	0.186	0.672	0.965	0.045	0.001	0.023

Cohort	1909	1910	1911	1912	1913	1914	1915
year	0.066**	0.072**	0.077**	0.071**	0.077**	0.076**	0.079**
	(20.51)	(20.35)	(18.99)	(16.26)	(14.85)	(13.49)	(10.44)
post1966	0.097*	0.103*	0.003	-0.016	0.015	0.055	-0.074
	(1.82)	(1.96)	(0.05)	(-0.31)	(0.27)	(1.01)	(-1.13)
slope1966	0.010	0.002	0.010	0.012	-0.004	-0.007	0.001
	(0.95)	(0.19)	(1.21)	(1.56)	(-0.51)	(-0.99)	(0.06)
postage65	-0.067	-0.105*	-0.176**	-0.144**	-0.080	-0.070	-0.094*
	(-1.18)	(-1.96)	(-3.32)	(-2.89)	(-1.58)	(-1.50)	(-1.75)
slopeage65	0.017*	0.020*	0.008	0.012*	0.024**	0.030**	0.018**
	(1.68)	(2.34)	(1.09)	(1.83)	(3.91)	(5.66)	(3.26)
_cons	-134.733**	-146.728**	-156.482**	-145.768**	-156.510**	-153.804**	-160.258**
	(-21.41)	(-21.17)	(-19.71)	(-16.93)	(-15.42)	(-14.02)	(-10.83)
F-test1966	3.669	2.402	0.809	1.216	0.168	1.162	0.713
p-value1966	0.033	0.102	0.452	0.306	0.846	0.322	0.496
F-testAge65	7.540	15.088	14.911	14.853	18.915	30.813	13.017
p-valueAge65	0.001	0.000	0.000	0.000	0.000	0.000	0.000

Notes: F-test1966 denotes the joint F test of changes in both levels and slopes in 1966, and p-value1966 denotes the corresponding P value. F-testAge65 denotes the joint F test of changes in both levels and slopes at age 65, and p-value1966 denotes the corresponding P value. We restricted the age range to (40, 90).
*p<0.1, ** p<0.05.

Section A.3. Computation of implied effect on ex55 based on the ITS analysis

Using the 1905 male cohort as an example, we shall describe the approach we use to compute the implied effect on life expectancy at age 55 based on changes in slopes and levels of Gompertz curves after Medicare eligibility age. We use a 20% random sample of whites from our census-tree data for this analysis.

Figure A. Gompertz curve of 1905 white male cohort

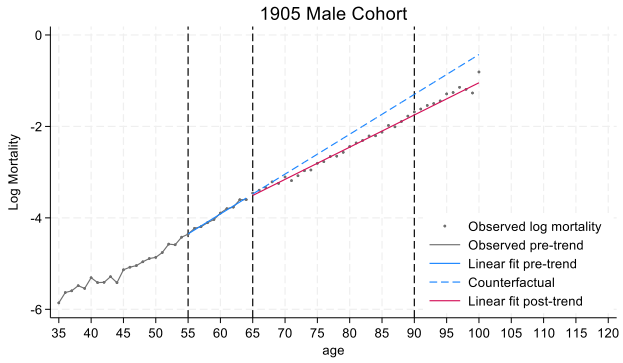
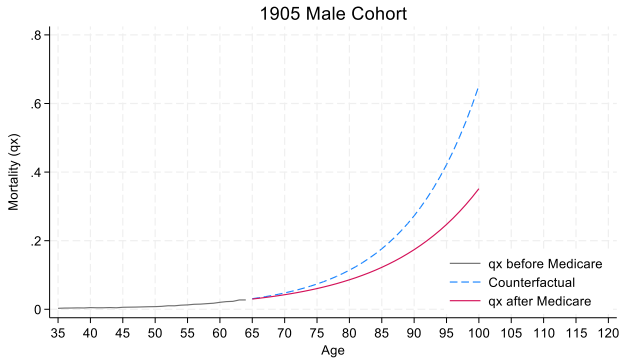


Figure B. Mortality (qx) with and without Medicare



Notes: Analytic data include a 20% random sample of whites born in 1905 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights.

The Gomperz curve for the cohort is illustrated in Figure A. It is consistent with the Gompertz Law of Mortality that log mortality is pretty linear in age. The Gompertz curve generally follows a linear function until age 97 and then fluctuates after that. We use the age window 55-64 to fit pre-trends in log mortality before Medicare, and 65-90 to fit the post-Medicare trends in log mortality.

First, we compute life expectancy if there were no Medicare at age 65 for the 1905 white male cohort (the counterfactual). We assume that without Medicare, the log mortality will follow the pre-trends. We run a linear regression of log mortality on age restricting to age range 55-64 and then predict log mortality for age 65-100—the dashed blue line Figure A. We then exponentiate the observed log mortality before age 65 (grey line) and predicted log mortality (dashed blue line) to obtain the mortality (qx_nopolicy) by age for the counterfactual scenario if Medicare did not exit, shown in Figure B. Using the life table with qx_nopolicy by age over 35-100, we compute the life expectancy at age 55 (**ex55_nopolicy**).

Second, to compute the life expectancy with Medicare (**ex55_policy**), we first fit a linear regression of log mortality on age for post-Medicare period (age 65 to age 90) and then predict log mortality for ages 65-100. The predicted log mortality for post-Medicare period is the red solid line in Figure B. Similarly, we convert the log mortality prior to Medicare at age 65 (grey line) and the predicted log

mortality after Medicare (red line) to mortality (qx_policy) shown in Figure B. With the qx_policy from age 35 to 100, we compute the life expectancy at age 55 (**ex55_policy**).

The implied effect of Medicare on life expectancy at age 55 is calculated as ex55_policy minus ex55_nopolicy. We then use a bootstrapping approach with 1,000 resamples of the Census Tree microdata to obtain the standard error and 95% confidence intervals.

Section A.4. Computation of implied effect on ex55 based on the DiD analysis

Section A.5. Details on the structural estimation

Section A.6. Behavioral responses to Medicare.