The Impact of Medicare on Life Expectancy*

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Abstract

This paper estimates the causal effects of the Medicare on mortality rates and life expectancy on the program's early recipients by assessing how the introduction Medicare changed the evolution of mortality rates for affected cohorts. 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. To identify Medicare's average treatment effects, we follow a cohort approach to track Medicare's effects 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 65 for men born between 1885 and 1915 by one year on average. Medicare's effects on life expectancy at age 65 are larger for cohorts with more potential years of exposure but similar for groups of high and low socio-economic status. The effects for women are not robust across methods and specifications.

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I. Introduction

In the United States, Medicare, a federal health insurance program, is the primary source of coverage for people over the age of 65. In 2025, Medicare enrolled 68.2 million people, cost over one trillion dollars, and accounted for 21 percent of US health expenditures and 3.7 percent of Gross Domestic Product (CMS 2025). Almost everyone older than 65 is enrolled (Moon 1996), and 92 percent of them rate Medicare's performance highly (Lopes et al., 2023). As a result, many have proposed that Medicare serve as a blueprint for extending health insurance to the entire US population. But because Medicare's costs have also ballooned, many have also proposed reforms to reduce its size.

Alongside providing financial protection, which Medicare has successfully done since its inception (Finkelstein and McKnight 2008, Barcellos and Jacobson 2015, Caswell and Goddeeris 2020, Goldsmith-Pinkham, Pinkovsky, and Wallace 2021), public health insurance aims to maintain population health by providing access to basic health care. An important justification for spending on these programs thus hinges on how well it supports medical care consumption among older people and the efficacy of that care in maintaining and improving their health.

Yet, estimating causal effects of Medicare on the health and mortality of elderly recipients has proved difficult. One reason is that Medicare provides universal coverage at age 65 and has changed only a handful of times in its history. Studies focused on the age-65 discontinuity find that one-year mortality falls sharply at age 65 among people with urgent hospital admissions (Card, Dobkin et al. 2009), but not in the overall population at age 65 (Card et al. 2004; Polsky et al. 2009). Studies of Medicare's introduction in 1966 (Finkelstein and McKnight 2008, Chay, Kim, and Swaminathan 2010), its extension to people with end-stage renal disease in 1972 (Andersen 2018), and its coverage of prescription drugs in 2006 (Huh and Reif 2017) find mixed effects on mortality.[1]

Another challenge is that none of these studies observes longer-term mortality rates, when Medicare's effects may be largest. Theoretical models (Grossman 1972, Dalgaard and Strulik 2014, Lleras-Muney and Moreau 2022) and empirical studies (Leinonen, Heikkinen et al. 2001, Levinsky and Schiff 2021) document that health accumulates and deteriorates slowly.

[1] Estimated effects of recent public health insurance expansions for non-elderly adults have this pattern, too. Graves et al (2020), Miller, Johnson, and Wherry (2021) and Meyer and Wyse (2024) all find health improvements from Medicaid expansions that would have been indetectable in aggregate data (Black et al 2019).

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 suggests large long-term impacts of health insurance provision to low-income children (Boudreaux, Golberstein et al. 2016, Wherry and Meyer 2016, Thompson 2017, Wherry, Miller et al. 2018, Miller and Wherry 2019, Brown, Kowalski et al. 2020, Goodman-Bacon 2021). Whether this evidence applies to adults who are no longer growing is unknown.

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 group had health insurance in the early 1960s, they faced large medical costs, but also and often forewent potentially life-saving medical care (CHAS and NORC 1984). Soon after Medicare's passage, however, this group began to use substantially more medical care and faced lower financial risk (Finkelstein and McKnight 2008).

To measure the mortality experience of cohorts affected 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 we also observe 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 65.

We generate hypotheses about how the existence of Medicare may have affected its recipients using the theoretical model of cohort mortality in Lleras-Muney and Moreau (2022). We first show that the dynamic effects of Medicare 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, while changes to the variance of health shocks or the death threshold reduce mortality immediately but raise the slope of the age profile.

These insights suggest a new cohort approach to identifying the effects of Medicare that relies on tracing its effects on the evolution of mortality for a given cohort after they become eligible for Medicare. We pre-specify three complementary research designs that identify these effects

under distinct assumptions.[2] First, we estimate the theoretical model using pre-Medicare data for each cohort, and use the parameter estimates to form counterfactual mortality rates during their post-Medicare years. The validity of this structural design depends on the correct specification of the model and the reliability of its parameter estimates. Our second approach builds on a qualitative prediction of the model and the well-known empirically regularity first observed by Gompertz in 1825: log mortality rates in adulthood are linear in age. This motivates an interrupted time series (ITS) design that forms counterfactual mortality at older ages using the estimated the age slope of log mortality before a cohort's Medicare eligibility. This approach relies less on the structure of the model and is internally valid as long as the log-linearity assumption holds. Finally, we employ a staggered difference-in-differences (DiD) design that compares changes in log mortality across cohorts. Here we rely on a parallel trends assumption that justifies using observed mortality changes for older cohorts who gained Medicare later in life to form counterfactual mortality trends. This does not rely on any assumption about the within-cohort structure of log mortality.[3] All three designs identify the impact of Medicare as it was experienced by affected cohorts between 1966 and 2015, on average 14-18 years later.

Preliminary results from all three empirical approaches align closely for men and show that Medicare increases life expectancy for men at age 65 by about one year 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 dynamic effects on log age-specific mortality rates that are initially small, as in more recent studies of Medicare's effect at age 65 but grow with time on the program. These closely match the predictions from our theoretical model and suggest that Medicare either raises health investments or decreases the rate of aging. We also find that Medicare had somewhat greater effects on the log of mortality for lower-educated subgroups, but these effects do not necessarily translate to large differences in life expectancy except for women with the least education. In fact, we cannot reject that the LE effects are the same across education and income groups among men. The results for

[2] 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. The training sample allows us to test for heterogeneity by 1940 Census variables, select covariates, and estimating tuning parameters without inducing pre-test bias. Results in this manuscript use training data only. We will archive our full analysis plan (and code) with the Open Science Framework before conducting our main analysis.

[3] 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.

women however are inconclusive. They are highly sensitive to the various identification assumptions we make in each of our three estimation strategies.

Ours are the first estimates of the effect of Medicare on long term mortality and life expectancy, and the first to examine heterogeneity across cohorts who gained Medicare at different ages. Previous work such as Finkelstein and McKnight (2008)'s seminal study of Medicare's introduction, necessarily relies on coarser age groups available in Vital Statistics data. Our data provide accurate mortality rates by single year of age, which allow us to implement new identification approaches that uncover meaningful long-term mortality reductions. The program's introduction is also the only opportunity to observe large groups who first became eligible for Medicare at ages older than 65, and we find very similar mortality effects across these groups. Our approach as another important advantage over designs based on more recent data: all of our estimates incorporate spillover effects, which the subsequent literature suggests may be important (Finkelstein 2007; Clemens 2013). Medicare has grown tremendously in scope, scale, and cost since 1966. Our findings suggest that these outlays likely still confer meaningful benefits to population health.

Our findings also add to a growing number of reevaluations of the health effects of President Lyndon Johnson's Great Society programs. Like us, this work documents substantial long-run benefits of Medicaid (Boudreaux, Golberstein et al. 2016, Goodman-Bacon 2018, Clayton 2019, Goodman-Bacon 2021, Clay, Lewis et al. 2024), Head Start (Ludwig and Miller 2007), food assistance (Hoynes, Schanzenbach et al. 2016) and community health centers (Bailey and Goodman-Bacon 2015). A key lesson is that carefully designed studies with long time frames frequently uncover strong evidence of benefits that short-run correlational evidence misses (see also Aizer et al. 2022 and Aizer et al. 2024).[4]

II. Health Insurance and Health Care before and after Medicare

Poor health was an increasingly burdensome problem for American seniors during the decades prior to Medicare's passage. 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

[4] We are not aware of other papers that show longer-run effects for elderly people, except for Derugyina and Reif (2023) who use the model in Lleras-Muney and Moreau (2022) to demonstrate that the long-term effects of pollution on mortality are larger than short term effects.

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, Gaynor 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 an individual basis. Individual insurance markets offered medically underwritten policies, making them prohibitively expensive to older people. Blue Cross Blue Shield community rated plans, but adverse selection pushed up their premiums as well (Thomasson 2004). 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, David 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. Most "insured" people had a policy that only covered hospital and sometimes surgical expenses, not primary care, diagnostic procedures, devices, or drugs. Even those policies were far from comprehensive. Just over half of elderly insured people with hospital stays in 1962 reported that insurance paid the majority of their bills (NCHS 1964, table 11.13). Private insurance paid less than one-fifth of medical costs in one sample of Social Security beneficiaries in 1966 (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.[5] Charity providers often provided low quality care, had long wait times, and could refuse service or pursue aggressive collection practices (Stevens 1989).[6]

Statistics on the use of basic health care services reflect these problems. Figure 1 plots the number of inpatient hospital days per person 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 amendment.[7] 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, Warren et al. 1996). Part B offered optional medical and surgical insurance with 20 percent coinsurance for $3 per month (Gornick, Warren et al. 1996). 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).

Medicare 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

[5] 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.

[6] 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).

[7] Despite the long history of public health insurance bills, "the outcome of the debate over Medicare was by no means predictable" (Stevens pg. 47). A Medicare bill pushed by the Kennedy administration was narrowly defeated in 1962, but after the Democratic landslide in 1964, the newly elected President Johnson revisited the issue. The administration proposed a universal hospital insurance program for the elderly with no premiums and minimal cost-sharing. Congressional Republicans preferred a means-tested system run by the states, and the American Medical Association proposed subsidizing the purchase of private fee-for-service insurance plans. Ultimately, the legislation that created Medicare combined all three proposals. 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).

Statistics 1963, National Center for Health Statistics 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). Finkelstein and McKnight (2008) also show that extreme out-of-pocket spending fell. Figure 1a follows the literature (Chay et al. 2010, Card et al. 2008) and documents using the National Interview Survey that before Medicare's passage in 1963, the rates of hospital insurance among seniors were low and fell sharply with age.[8] Right after the passage of Medicare in 1968, the fraction of individuals with hospital insurance among the over 65 population reached more than 90% on average and no longer declined with age.

This increased insurance coverage and ability to pay was associated with substantial increases in health care use (Figure 1b). 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, consistent with previous research (Finkelstein and McKnight 2008). Today this remains true: Individuals on Medicare use health care at much higher rates than those without (Card, Dobkin et al. 2008) and they have lower medical expenditure risk (Barcellos and Jacobson 2015).[9]

III. Potential Effects of Medicare on Mortality

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. Parental health and genes, as well as *in utero* conditions endow individuals an initial level of health $H \sim N(\mu_H, 1)$ that is normally distributed in the population with a standard deviation of 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 normally distributed shocks $\varepsilon_a \sim N(0, \sigma)$. This additive law of motion for health means that ε_a captures both direct

[8] The National Health Interview Survey is a nationally representative survey of the non-institutionalized population. We use the 1963 wave (the first wave prior to Medicare's passage) and the 1968 wave (the first wave after Medicare's passage, to document trends in the fraction of individuals with hospital insurance.

[9] Since its inception there have been a few notable changes in the program. Medicare was further expanded to younger people on Social Security Disability Insurance and those with end-stage renal disease on July 1, 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.

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 dies 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 only 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 insurance structure, or equivalently, it may shift the health shock distribution.[10] Medicare may also lower the variance of health or health investment shocks (σ) for several reasons. Its insurance function transfers resources from good states (when individuals pay for premiums) to bad states when they suffer costly health shocks that are now covered by insurance, resulting in lower health in good times but higher health after bad health shocks. 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 Medicare 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 ($\underline{H}$) keeping live individuals would die in the absence of health care. Medicare cannot affect initial health, μ_H, and we assume that it does not affect mortality from accidental causes, κ.

Figure 2a plots the simulated effect on 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;[11] log mortality rates do not shift immediately, but they do rotate down so that mortality rises more slowly as people age. The reason mortality effects do not show up immediately but become visible over time is that a lot of individuals in the population will receive a boost in their health (or slow down the rate of aging) but because they are far from the death threshold these effects are not detectable with mortality rates. 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), lead to immediate

[10] Medicare is financed from payroll taxes paid by mostly ineligible workers, so the initial generations gained coverage without having paid in.
[11] There are two aging parameters. The effects of changing either are similar so we only display one.

downward shifts in log mortality rates and then steeper slopes thereafter because the less healthy surviving cohort is very sensitive to subsequent shocks. 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 resulting from of a change in each parameter. 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.

Research on Medicare and mortality, however, generally does not estimate dynamic within-cohort effects or measure life expectancy. Contemporaneous studies using the age-discontinuity in Medicare eligibility necessarily identify causal effects *at* age 65, and then only find immediate mortality reductions for severely ill patients (Polsky et al. 2009, Card et al. 2004; Card et al. 2009). 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 (Huh and Reif 2017, Andersen 2018, Dunn and Shapiro 2019).

Historical studies, on the other hand, estimate dynamic effects on aggregate age-specific mortality. 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 its first ten years. In contrast, Chay et al. (2010) use five- instead of ten-year age groups and find sharp convergence between elderly and near-elderly mortality after 1966. None of these studies estimate longer-run mortality effects within cohorts needed to quantify Medicare's effect on life expectancy, nor are they able to measure mortality for subgroups other than by race, sex, or geography.

In fact, randomized trials of the kinds of care that Medicare covered do find longer-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 reducing smoking had immediate effects on health and on earnings (Stephens Jr and Toohey 2022), but mortality declines were only detected after ten years (Multiple Risk Factor

Intervention Trial Research Group 1982, Multiple Risk Factor Intervention Trial Research Group 1990, Multiple Risk Factor Intervention Trial Research Group 1996). Medicare also improves access to diagnostic and preventive services which could also benefit individuals with a delay (for evidence on colonoscopies see (Niikura, Hirata et al. 2017). Health insurance has also been shown to improve health behaviors and mental health in the short run (Baicker, Taubman et al. 2013), which could lower mortality eventually.

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 over time at least when measured by mortality rate declines. We estimate these dynamic benefits using various approaches next.

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, Buckles 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.[12] We use these data to assess the quality 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 because their coverage in the Family Tree database is more complete than for other race groups (Price, Buckles 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*, Smith 2016). Finally, we retain observations with complete data on birth and death dates.[13]

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. 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. The mean age at death in our sample, which is 72.92 for men and 79.16 for women. But there is great variance in the age at death with some dying at age 65 and other surviving to beyond age 100. The 90% percentile of the year of death in the full sample is 1994 for men and 2000 for women, and it is 1998 and 2004 for men and women born after 1901 experiencing Medicare after age 65. So the cohorts we study experienced Medicare as it operated primarily from 1966 until the mid 2000s.

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

[13] 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.

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.

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

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

$F_c(\cdot)$ is the c.d.f. of Y_i among members of cohort c, which means that the dynamics of $\tilde{q}_{ca}$ come from the shape of $F_c(\cdot)$.[14] 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.

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. **Table 1** shows that 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.[15] However, it includes poorer households, fewer women (46.3% in Census Tree versus 49.8% in the 1940 Census), more married people (84.2% versus 72.3%), fewer people either born in the Northeast (16.9% versus 24.3%) or residing in the Northeast region in 1940 (17.8% versus, 30.5%). No existing linking methods produce samples that are representative of the linkable population (Bailey, Cole et al. 2020).

[14] 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.

[15] 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.

We correct the observable imbalance by re-weighting the linked sample to make it representative of the target population (Bailey, Cole et al. 2020, Lleras-Muney, Price et al. 2022, Yue, Ettner et al. 2023). 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 (Seaman and White 2013, Newson and Falcaro 2023).[16] We then reweight matched observations by $p(X_i)^{-1}$ (inverse propensity weights, IPW, rescaled to sum to one within the matched sample) so their covariate distributions match the 1940 Census. The second column of **Table 1** shows that, as expected, reweighting brings the sample covariate means substantially closer to the target population means.

To assess the quality of death information in our data we compare the weighted 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 A2**, 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 sample include no Blacks and substantially 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 65 computed using our data is larger than that in SSA life tables (**Figure 3**). In our sample at age 65 men can expect to live 14.l 3 years and women 18.74. For every cohort we observe in both data sets, the life expectancy in the SSA data is lower than in our data as expected given the lower representation of high mortality groups. For men the gap is grows from 0.5 for the 1900 cohort to about 0.8 years for the 1915 cohort. For females, the gap shrinks from about 0.8 years for the 1900 cohort to about 0.5 years for the 1915 cohort.

Empirical Strategy

[16] In addition, we further truncated IPW at the 99th percentile to stabilize the weights (Seaman and White 2013). We include the following covariates (X_i) available in the 1940 full-count census: sex, race, ethnicity, marital status, years of schooling, household total income, family size, labor force participation, occupation income scores, year of birth, state of residence, and place of birth.

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 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 log mortality rates of each cohort at each age:[17]

$$ATT(g_t, g_a, a) \equiv q_{c,a}(g_t, g_a) - q_{c,a}(\infty) \qquad (2)$$

Since Medicare "treats" all members of a cohort we take the phrase "on the treated" to refer to all members of the specific cohorts exposed to Medicare in year g_t at age g_a. This parameter reflects many different channels through which Medicare may have affected mortality, including but not limited to the increase in insurance coverage. Each $ATT(g_a, g_t, a)$ approximately equals a proportional treatment effect of Medicare relative to $\tilde{q}_{ca}(\infty)$. The collection of $ATT(g_t, g_a, a)$s are *within-cohort* event-study effects that fully characterize Medicare's effect on longevity. Because there are too many to report individually, we summarize them in three intuitive ways.

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

[17] We use log mortality rates for several reasons. First, parameters defined in logs are easier to interpret across ages with different mortality rates (Deaner and Ku 2024). Second, for reasons discussed below, we believe that identification of log parameters is credible, while other transformations are not (Roth and Sant'Anna 2023).

$$ATT(e) \equiv \sum_{g_a=65}^{A} \sum_{g_t=1966}^{T} w(g_t, g_a) \times ATT(g_t, g_a, g_a + e) \qquad (3)$$

The weights $w(g_t, g_a)$ equal the share of the population who survived to their Medicare age who are in the (g_t, g_a) cohort. The collection of $ATT(e)$ parameters around Medicare eligibility are average within-cohort dynamic effects on log mortality rates, as in **Figure 2b**.

The circumstances of Medicare's introduction mean that cohorts born before 1901 gained Medicare eligibility after age 65. These are the only cohorts in Medicare's history to gain access later in life, and thus Medicare's effects for them are of special relevance to questions about changing eligibility policy. We report cross-cohort heterogeneity by summarizing each cohort's dynamic effects with a linear function and reporting the intercept term and slope terms:

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

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 ($q_{c,a}(\infty) \equiv q_{c,a} - ATT(g_t, g_a, a)$) to construct counterfactual mean age at death and report average effect of Medicare on life expectancy at age 65:

$$ATT_{LE}(g_t, g_a) = E[Y_i(g_t, g_a) - Y_i(\infty) | G_t(C_i) = g_t, G_a(C_i) = g_a, Y_i \geq 65] \qquad (4)$$

It is important to compute life expectancy gains in addition to impacts on mortality because the former is shaped both by how Medicare changes mortality at different ages and the level of mortality thereafter (Dahl, Kreiner et al. 2024). Thus a group that experiences the largest drop in mortality (in absolute or relative terms) is not necessarily the group that will experience the largest gains in life expectancy.

Both the evidence and the model of health capital accumulation discussed in section III point to potential 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, $\tilde{q}_{c,a}(x) \equiv \frac{P(Y_i = a | C_i = c, X_i = x)}{P(Y_i \geq a | C_i = c, X_i = x)} = \frac{F_{c|x}(a) - F_{c|x}(a-1)}{1 - F_{c|x}(a)}$. 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 $ATT(g_t, g_a, a)$ parameters: the full structure of the model from section III, linearity of untreated log mortality rates, and a parallel trends assumption across cohorts. All three strategies require an assumption that Medicare does not affect mortality before a cohort gains eligibility. These effects could arise because people anticipate their own 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.[18]

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:

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

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 model is estimated using the Simulated Method of Moments (SMM), and we target the differences in the survival curve at each age as the objective function.

The dashed line in **Figure 4** plot log mortality rates predicted by the model for the 1900 cohort with parameters estimated on data through age 65. The solid line plots observed log mortality

[18] 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 1973, when the 1908 cohort aged into Medicare. [MORE RECENT EVIDENCE ON SHIFTING OF CARE, WHETHER ITS PLAUSIBLE HERE]

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. The structure of the model embeds several strong assumptions, such as constant parameters and no optimization.[20] We also face several limitations estimating the model's parameters. The most practical challenge is that the model requires data on complete life tables from birth to age 65, which means we can only use the SSA life tables for cohorts born in 1900 and after. Moreover, the importance of infant and child mortality in pinning down parameters like the initial health stock and the accident rate mean that early-life shocks could bias our 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. Third, the objective function is poorly behaved, so the estimates are sensitive to initial guesses. To address this, we first estimate the model for the 1900 birth cohort repeatedly until we can no longer improve the fit. Then we use these parameter values as initial guesses for subsequent cohorts. Our main results refer to cohorts for which the fit is as good or better than for the 1900 birth cohort: the 1901 to 1908 birth cohorts for men and 1901 to 1904 cohorts for women (see **Table A.1**).

Despite these limitations, this model successfully fits patterns of mortality for a wide range of populations and can successfully replicate the observed effects of high SES or of temporary or permanent shocks to health occurring at some point in the lifetime (Lleras-Muney and Moreau 2022). It is easily used to estimate the effects of Medicare on life expectancy and to make counterfactual predictions. It is not clear that there are better alternatives to do this in the demography or economics literature. We assess the model's fit in our context using both mean squared error in the survival curve and by plotting pre-Medicare differences between observed and predicted log mortality. If the model estimates are on average unbiased then the predicted and observed mortality will be very similar up to age 65. We view this design as a bridge between the qualitative predictions of the model discussed above, and the empirical estimates

[20] Optimizing models with these features are well understood, but without detailed data on health, incomes, and investments they are not estimable (e.g. Khwaja 2010).

of the $ATT(g_a, g_t, a)$ parameters. We also use two additional approaches that rely on less parametric assumptions and can be implemented using our linked data.

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 theoretical model 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.

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

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

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

Unlike the model-based estimates that use the full history of mortality to identify the structural parameters, the ITS approach relies only on assumption GM during ages close to Medicare eligibility. Key to this approach then is to determine the range of ages, a_{c0} to a_{max}, during which log-linearity is appropriate. We set $a_{max} = 90$, similar to Chetty et al. (2016) and

Fletcher et al. (2023), which avoids bias from non-linearities in log mortality and mismeasurement of age at death for the oldest old.[21]

The 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.[22] 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.

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.[23]

Assumption PT. Parallel Trends across cohorts and ages

The change in untreated log mortality rates for cohort c between ages $G_a(c) = g_a$ and a equals the change in log untreated mortality rates between the same ages for older cohorts, ℓ, treated after age a and between 1 and k years after age $G_a(c)$; that is, for all ℓ such that $max\{a, g_a\} < G_a(\ell) \leq g_a + k$:

$$q_{c,a}(\infty) - q_{c,g_a-1}(\infty) = q_{\ell,a}(\infty) - q_{\ell,g_a-1}(\infty)\,. \tag{8}$$

As in Callaway and Sant'Anna (2021), assumption PT involves a cohort treated at age g_a and comparison cohorts treated "later." In our case, later means "at a later age", which means the comparison cohorts are older. We assume that these cohorts' mortality-age profiles would have had the same slope at the same ages in the absence of Medicare.

[21] 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.

[22] 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.

[23] 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).

The detail unique to our analysis is that k restricts the set of comparison cohorts so that we only assume parallel trends for those who gain eligibility close to the age g_a. This restricts the ages at which $ATT(g_t, g_a, a)$ parameters can be identified. For example, with $k = 5$, Assumption PT means that cohorts born between 1899 and 1895 identify counterfactual log mortality rates for the 1900 cohort. This permits identification of $ATT(g_t, g_a, a)$ parameters from age 66, when the 1900 cohort gained Medicare, to age 70 since the oldest comparison cohort is treated by age 71.[24] A larger value of k means a larger yet older comparison group that identifies parameters at older ages.[25]

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:

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

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 measured at age $g_a - 1$.

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

[25] 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.

Intuitively assumption PT 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. For example, WWI and the 1918 flu had different mortality effects across cohorts may lead to non-parallel slopes in log mortality thereafter.[26] We control this by choosing k using our 20% training sample. For a group of 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.[27] In other words we include cohorts so long as they do not include large estimated pre-trends.

At a common pair of ages, however, the log mortality rates of different cohorts necessarily occur in different calendar years, so assumption PT requires that the trend in "time effects" is the same across periods (see Dinas and Stoker 2014). We check this in two ways. First, we compare changes in mortality around 1966 for cohorts who were in their 50s or early 60s, and thus not on Medicare, to changes in earlier years when slightly older cohorts were the same ages. As long as the existence of Medicare does not affect mortality of older adults too young to get it, then this approach identifies calendar time shocks arising in 1966. Second, we implement an alternative DiD design that compares log mortality changes in calendar time for cohorts treated at time $G_t(c)$ to *younger* cohorts treated in the future. This fixes comparisons to occur in the same years, but across different ages and, in the absence of spillovers, identifies the same parameters defined above. We report results from these approaches and discuss what they imply about Medicare's spillover effects onto younger people in Appendix X.

V.B.4 Estimating aggregated parameters

We report aggregates of the estimated $\widehat{ATT}(g_t, g_a, a)$ parameters as described in section V.A. $\widehat{ATT}(e)$ parameters are simple cross-cohort averages of the building block parameters. We estimate linear parameters for each cohort's mortality effects using the OLS formulas for the intercept and slope from a regression of $\widehat{ATT}(g_t, g_a, a)$ on post-Medicare event-ages 0 to A:

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

[26] 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.

[27] 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.

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

These linear combinations are simple to calculate using post-estimation commands.[28]

Finally, we use life table methods described in appendix A.X, to estimate $ATT_{LE}(g_t, g_a)$ parameters. Because the structural model identifies counterfactuals at all ages, we have estimates $\widehat{ATT}^{SIM}(g_t, g_a, a)$ for each cohort's entire post-Medicare lifespan. An advantage of the model is that it captures commonly observed non-linearities in later life mortality; these are particularly important for accurately estimating effects on life expectancy.[29] When calculating causal effects on life expectancy based on the ITS or DiD designs, which we only estimate within an age window around Medicare, we assume that the estimated linear trend across ages in each cohort's $\widehat{ATT}(g_t, g_a, a)$ parameters continues until age 90.

V. The Effect of Medicare on Mortality Rates and Life Expectancy

VI.A Main cohort-level results

Figure 7a presents our main estimates of Medicare's causal effects on mortality. It plots $\widehat{ATT}(e)$ parameters from each of our two reduced form designs 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 trend. 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 all three approaches are negative, clearly distinguishable from zero after two 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

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

[29] 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). 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).

men by about 15 percent ($\widehat{ATT}(e) \approx 0.16$), an estimate that is remarkably similar across designs.

Figure 7b 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 results show that the model does an excellent job at predicting log mortality for the 20 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. Evidently, the conclusion that Medicare reduced mortality of white men does not depend on which of our three identifying assumptions hold. Interestingly the model shows that the declines level off after ten years but remain large (-0.2 log points) up to 20 years after cohorts gain Medicare. This last result highlights the value of the model since it would be hard to predict this pattern based on the reduced form results alone.

The top row of **Table 2** summarizes the average event-study using estimates of the average intercept ($\frac{1}{31}\sum_{c=1885}^{1915}\hat{\theta}_c$) and slope changes ($\frac{1}{31}\sum_{c=1885}^{1915}\hat{\delta}_c$) in log mortality rates associated with gaining Medicare across all cohorts. The intercept estimates are small in both the ITS (-0.023, s.e. = 0.007) and DiD designs (0.008, s.e. = 0.011), but the slope estimates are negative, close (-0.015 for ITS and -0.018 for DiD), and very precisely estimated (ITS s.e. = 0.001, DiD s.e. = 0.002). 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 in the first ten years it is -0.01 and the change in the intercept 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 an average gain in life expectancy for men of 1-1.13 years across the 1885-1915 birth cohorts, as shown in **Table 3**. Our confidence intervals rule out changes smaller than about 0.8 years; a meaningfully large increases in longevity. The structural model suggests gains of about 2.4 years, which come from focusing on the 1901-1908 cohorts who have relatively large benefits of Medicare (as we will show and discuss shortly).

The aggregate event-study estimates for women vary widely in sign, magnitude, and significance across empirical strategies, as **Figure 7** shows. The ITS estimates are slightly

positive, but never statistically distinguishable from zero. The DiD estimates are negative, only significant after eight years, and less than half as large as the results for men. The structural estimates on the other hand, show large mortality reductions, very similar to our findings for men. These differences do not appear to stem from failures of our identifying assumptions in the pre-period. The ITS and DiD point estimates suggest that pre-Medicare mortality rates were roughly linear and comparable across cohorts, but they are imprecise and cannot rule out potentially meaningful bias (in either direction). The structural results for women, however, show that log mortality rates fall fairly sharply relative to the model predictions at age 64. Thus, 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 inconclusive, implying that Medicare may either increase or decrease log mortality rates depending on the identifying assumption.

The inconclusive effects for women are also apparent in their estimated intercept and slope changes in **Table 2.** The ITS estimator yields statistically significant declines in the intercept and increases in the slope, whereas the DiD estimator does not indicate a change in the intercept but does show a statistically significant decline in the slope. The two methods therefore give wildly different estimates for women's life expectancy: ranging from an increase of 0.75 years using the DiD estimates to a *decline* of 0.5 years using ITS (**Table 3**). The structural model gives much larger, perhaps implausibly large, estimate of a 3.1 increase given the existence of pre-trends.

VI.B Results by birth cohort

Figure 8 shows intercept 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 closely resemble the aggregate results for most cohorts. For cohorts born between 1885 to 1905 the slopes show statistically significant declines, and the intercept changes 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 this pattern. First, the estimated intercepts become 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 cohort groups. For the 1885-1900 cohorts, we estimate a small (0.022 with the ITS design) or statistically insignificant change (less than -0.001 for the DiD design) in the intercept, and statistically significant declines in the slope (-0.018 for DiD and -0.023 for ITS) that are similar to the pooled estimate. These results change progressively across cohorts, however. In the ITS design the intercept 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 intercept 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. Overall both approaches suggest significant declines in the slopes, and while they change somewhat across cohorts we cannot reject they are the same.

Figure 9 shows the implied changes in LE at age 65 across cohorts. The results are very consistent across designs for men. 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. 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. Cohorts becoming eligible earlier are exposed to the reductions for longer, explaining their greater gains. This highlights that conclusions based on mortality rate changes cannot necessarily be applied to life expectancy.

The structural estimates are generally larger for any given cohort, but they exhibit a similar trend upwards, increasing from roughly 2 to 2.7 years. The reason these SM estimates are larger than the reduced form ITS and DiD is that mortality rates in the data slow down at very old ages. The model delivers this pattern, which results in higher life expectancy than when we use the DiD and ITS estimates because in that case we need to assume that mortality rates continue to grow linearly at all ages (see Appendix A.3 for details).

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 intercept changes 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 65 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 decline in the slope as a result of Medicare is the same across cohorts. However, the implied effects of Medicare on life expectancy rise across cohorts because cohorts receiving benefits at earlier ages were exposed to them for longer. 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 Spillovers

Medicare was so large its introduction had widespread effects on providers (Finkelstein 2007) and medical device patenting (Clemens and Olsen 2021). With spillovers, the identification strategies we use all identify $ATT(g_t, g_a, s, a)$ under their respective assumptions—that is, our parameters include spillover effects (see Appendix A7 for a formal discussion of spillover effects and how they affect our estimates). There is no way to use cross-cohort variation alone to separately identify their direct and spillover components. Furthermore, for all the cohorts becoming eligible in 1966, our identification strategies are not biased by spillovers because they use pre-1966 data to identify its effects.

Spillovers become a concern for cohorts born after 1901: if there are large spillovers then using mortality profiles post 1966 and pre age 65 would bias our estimates. However, we can partially test if there is evidence on spillovers on younger, near elderly individuals. We conduct two exercises to assess these. First, we can test if there is a change in the intercept or the slope of the log of mortality of non-eligible individuals in 1966. Results in **Table A.3** 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 these 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.

Second, we estimate "placebo" DiD models using cohorts before they are eligible for Medicare but after 1966. We observe the mortality rates of the 1902-1915 cohorts potentially affected by spillovers in 1966 but not directly covered by Medicare. They spend progressively longer periods of time exposed to *others'* Medicare coverage. The 1902 cohort potentially experiences spillovers for just one year—when they are 64 years old in 1966—while the 1915 cohort experiences 14 years of spillovers—from 1966 to 1979 when they are 51 to 64 years old. We can compare the change in e.g. one year mortality in 1966 for the 1902-1908 birth cohorts (ages 64 and younger in 1966) to that of older cohorts at the same age prior to 1966, as we do in the main estimation. Appendix Figure AX1 reports estimates based on this kind of DiD approach. We find no evidence of spillover effects on younger people. The point estimates are small and not statistically significant for the younger and for the older subsets of these spillover cohorts, except perhaps for the youngest cohorts after 9 or 10 years.

There are two reasons why our analysis should capture the timing of Medicare's likely spillovers. First, Finkelstein (2007) finds immediate and large changes in hospital capacity, aggregate utilization, and entry. We find no health effects among the cohorts who without Medicare who may have gone to these new and larger hospitals. Moreover, even if Medicare's most important spillovers took time to materialize, our estimates for the 1908-1915 cohorts cover up to seven years after Medicare, a reasonable time frame over which to expect spillover mortality effects. It is also possible that Medicare's spillovers were small for people without Medicare coverage and that the main spillovers were within markets and benefitted the over 65 population primarily.

VI.D 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, restricting attention to cohorts born up to 4 years earlier, 11 years earlier or up to 15 years earlier. For men (**Figure A7a**) the results are quite robust. Although the point estimates vary somewhat (the declines are a bit larger if we use more recent cohorts), 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.

Weighting schemes.

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 (those born 1902 and later): 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.[30] 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. Medicare's passage took place during a decade of large transformations in the safety net and social insurance programs as a result of the War on Poverty under the Johnson administration and further changes in the 1970s under the Nixson administration. We now discuss which other programs may have affected the elderly or the near elderly, and test to see if these changes affect our findings

Medicaid was created at the same time as Medicare. Poor elderly individuals are eligible for both (they are the so called "dual eligibles"), so in principle we are identifying the effects of the combined policy, at least for this group, which accounts for about 20% of seniors. To assess if Medicaid is partly responsible for the gains we see, we can 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 **Table A.2**. We do not find this to be the case.

There are other programs that may have affected elderly and ear elderly adults. The Older Americans Act was also passed in 1965. Old Age Assistance (OAA) for poor elderly individuals became more generous. Finally, community health centers (CHC) were created to expand the supply of health care providers in areas that were lacking medical personnel and services (mostly rural areas and inner cities). We collect additional data on these programs to test these hypotheses (TBA).

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. Also to the extent we can test them, we do not find evidence of spillovers at least in the first few years of the program.

VI.E The effects of Medicare by education and income

[30] 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.

We now present ITS and DiD results 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. SSA life tables are not available for any sub-national group so we cannot estimate the structural model for education and income sub-groups.

These estimates are interesting for at least two reasons. First, it is not obvious whether Medicare should have larger or smaller health effects on higher-income or more-educated people. Higher-SES groups might stand to gain less in terms of their health from accessing Medicare because they had higher insurance rates prior to its introduction (Epstein and Murray 1967) and were in better health (Kitagawa and Hauser 1973, Braveman, Cubbin et al. 2010). On the other hand, conditional on having insurance high-SES people still visit specialists at higher rates (Braveman et al., 2010; Dunlop et al., 2000) and benefit more from medical advances because they appear to adopt these at higher rates (Glied and Lleras-Muney 2008). Thus, higher SES individuals might benefit more. The model reflects this ambiguity as well. More people in high-initial-health groups reach 65, but the survivors from a low-initial-health group are very positively selected, making it unclear which surviving group is healthier and would benefit more from Medicare.

In addition, a different but subtle issue arises when predicting life expectancy gains: groups who see the largest decreases in mortality rates may not see the largest increases in life expectancy if their mortality rates throughout the remaining ages remain more elevated than those of other groups (Dahl, Kreiner et al. 2024).[31] So even if groups with lower health see larger declines in mortality as a result of Medicare, they may not see larger increases in life expectancy because they still face higher mortality rates thereafter. We examine this question empirically.

The second reason these sub-group estimates are interesting is that they 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

[31] To gain intuition about this result consider the following example. Suppose that mortality rates λ are constant at every age and the time until death follows an exponential distribution so that life expectancy is given by $1/\lambda$. Suppose that in period 1 group A has a mortality rate $\lambda_a = 0.1$ and group B has a mortality rate of $\lambda_b = 0.2$, so that life expectancy is equal to 10 for group A and 5 and group B. Suppose now that mortality falls, more so for group B which has the initially higher rates. In the second period $\lambda_a = 0.06$ and $\lambda_b = 0.1$, so the decline in mortality is larger for B in absolute and relative terms (the decline for B is of 0.1 or 50%, whereas for A the decline is only of 0.04 or 40%). But the life expectancy of A is now 16.6 and that of B is 10. Group A sees an increase of 6.6 years whereas B only sees an increase of 5 years despite their larger mortality declines. This occurs because the mortality rates for B are higher than A's in period 2.

education or income groups. These conditional assumptions may be more plausible than the 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 educational levels increased substantially for the later birth cohorts in our sample and that education is associated with lower mortality rates in these cohorts as well (Goldin 1998; Lleras-Muney 2005; Lleras-Muney, Price et al. 2022; Yue, Ponce et al. 2023).

We start by considering effects by education levels. **Figure 10** plots the event-study estimates by sex and for the three education groups separately: those with less than middle school (0-7 years of schooling), those that graduated from middle school but not high school (8-11 years of schooling), and high school graduates (12+years of schooling). There correspond to 24, 47 and 29% of men in the population, and 21, 46 and 33% of women in the population (Goldin 1998). We find very little evidence of pre-trends for either sex in any education group, suggesting that our conditional assumptions hold as well.

For men we find evidence that Medicare lowers log mortality rates at an increasing rate over time for all three groups. But the effects (in logs) appear larger for lower education groups. Indeed, by year 9 the decline in the log of mortality is about -0.2 for men with 0-7 years of schooling, larger than then -0.15 decline we see for men with 12 or more years of schooling. **Table 3** shows that the implied effects on life expectancy does decrease with education levels from 1.1 years for those with less than primary education to 0.85 for high school graduates. But this is true only for the ITS approach. The DiD approach finds the opposite pattern with somewhat larger gains for the most educated group (1.05 for the most educated vs. 0.96 for the lowest education group). Furthermore, these differences are small, and the estimates are not statistically significant from each other, so on net the effects on life expectancy at age 65 do not appear to be meaningfully lower for the less educated despite their larger declines in mortality rates.

The same is true when we look at differences by income. **Figure 11** shows the results by income levels. The event studies show similar reductions in log mortality for men regardless of their income in 1940 though the reductions in mortality in fact appear somewhat *larger* for individuals from high income households. **Table 3** shows that on net however men from low-income and high-income households have very similar benefits from Medicare and just like in

the case of education, we cannot reject that the life expectancy gains are the same for both groups.

For women the event studies in **Figure 10** and **Figure 11** do not show impacts for any group except perhaps for those without primary schooling. **Table 3** shows that life expectancy gains are larger for the least educated women and for the poorer ones, but this is only true in the DiD estimates. The ITS estimates are insignificant in magnitude or even negative. Moreover, the confidence intervals for women are large so we cannot rule out that the estimates are identical across groups. So again, the results for women are inconclusive, with larger standard errors and disagreement across methods.

In sum, for men we find some evidence of larger declines in mortality rates for lower SES groups, but the gains in life expectancy at age 65 are very similar by education and income. The results for women by SES are not robust across methods and have larger standard errors.

VI. Discussion

VII.A Magnitudes

Our estimates for men show that Medicare obtained at age 65 increases life expectancy at age 65 by as much as two years. These are very large effects. **Figure A2** shows that life expectancy at age 65 rose by about 2 years for women and 1.5 years for men. So, the effects of Medicare that we estimate for men born in e.g. 1901 are larger than the gains observed across these 30 birth cohorts.

Are these magnitudes reasonable? Evidence from randomized trials suggests these are plausible. Heart disease was the main cause of death among men ages 65 and above in 1965. 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). So our results would be consistent with this evidence if a substantial share of men had benefitted from these innovations which came about precisely in the 1960s.

One way to assess the magnitude of our estimates is to compare them to the effects of other programs. Goodman-Bacon (2018) quantifies the effects of Medicaid's introduction and finds that it reduces the mortality rates of its youngest nonwhite recipients by about 30 percent. This setting differs from Medicare's introduction in several ways. Essentially all new Medicaid

children would otherwise have been uninsured, while about half of all elderly people had insurance before Medicare began. Therefore, the gain in insurance rates in our study is about 50 percent, but the other half received a subsidized version of a product they already owned. A naïve rescaling of our long-run effect on log mortality rate—about -15 percent for men—by the size of their coverage gains, 40-50 percentage point, implies an upper bound on Medicare's average effect on the mortality rates of the newly covered of -30 to -38 percent. This does not account for differences in the mortality rates of the previously uninsured, financial benefits to the always-insured, or any effects of Medicare on health system capacity that may have benefitted everyone. Thus, our results imply that for men Medicare's long-run effects on period mortality rates are at most as large as Medicaid's effect on young nonwhite children. An important takeaway is that the ATT estimates that form the basis of our results are not implausibly large when compared to Medicare enormous effect of insurance coverage.

Interestingly our mortality estimates are similar to the effects of Old Age Assistance (OAA) in the US. Galofré-Vilà et al. (2022) estimate that OAA lowered the mortality rates of the over 65 population by 30-40% within the first ten years, which is similar to what we find except that OAA targeted only the elderly poor whereas our estimates pertain to the entire over 65 population.

Finally, 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 (about $3,000 in today's dollars). If men enrolled in Medicare at age 65 lived for 16 years after age 65 (14 years + 2 gained), the total cost per enrollee's lifetime is about $54,000. Since beneficiaries gained about 2 years of life, it cost about 24,000 per year of life gained, substantially lower than the estimated statistical value of a year of life.[32] However, the cost of these life extensions is larger than the cost of similar life extensions resulting from programs that benefit younger populations, such as cash transfers to poor families or youth training programs.[33] Also note that today's cost per

[32] The statistical value of a year of life is estimated to be about $150,000, a middle range estimate across studies (Keller et al. 2021).

[33] Aizer et al (2016) find that giving about 30-50% of FPL in cash to poor families for a median duration of 3 years increased the LE of boys by 1.5 years. The cost of that was up to 50% of the FPL per year. The FPL today for a family of 5 is $37,650, so the total cost was $56,475 per family, or ~14,120 per kid (4 kids and a mom, assuming all money spent on kids). Thus, the cost of 1 year life extension was $9,412. Azier et al. (2024) find that training men ages 19-25 for about 1 year during the great depression in the CCC program increased lifespan for about 1 year. the cost of the program estimated to be about $23,000. Thus the estimated cost of a 1-year life extension was $23,000.

beneficiary is about $15,000, making the cost of a an extension of a year of life larger (about $120,000).

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.[34] 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.[35] 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 on comparing within-cohort age profiles and our findings align more closely with CKS.

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 (Callaway, 2024 #193). Because regional health insurance coverage was strongly correlated with factors that potentially mediate the effect of Medicare on mortality, such as income,

[34] 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.

[35] 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 $ATT_{LE}(g_t, g_a)$.

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.[36]

VII.C Discussing sex differences

Our results for men are consistent across approaches, particularly once we focus on the pre-1910 birth cohorts. For women our results vary wildly across methods and do not appear to be robust. We now discuss why.

A concern in using historical data is that women are typically tracked at lower rates than men. Because our matching is to family trees which contain both maiden and married last names, our ability to track women is very high. However, men are slightly more likely to be represented in our sample than men, relative to the 1940 census. We can assess the possible magnitude of the bias that this introduces by computing bounds (TBD).

One possibility is that women's health and life expectancy starts improving not when they become eligible for Medicare but when their (typically older) husband's do. In this case women would start benefitting from Medicare before age 65 which would pose a problem for all of our approaches: the slope of mortality will deviate from linear before age 65, which the SM would not rationalize in the absence of a shock. This would also affect the ITS approach by affecting the pre-Medicare slope. Similarly, it would affect the DiD approach. To investigate this, we re-estimate the model for women that were never married or who were not married in the 1940 Census. We still estimate null effects for this group (**Figure A.8**) ruling out this as an explanation for our results. A similar logic would suggest that the estimation might be biased if women below age 65 become eligible for Medicaid at large rates but we do not find evidence that effects are more consistently estimated in states where Medicaid was implemented later (**Table A.2**).

Another possibility is that many women experience the death of their husbands close to the age at which the become eligible for Medicare. Indeed we compute in our data that 50% of women become widows between the ages of 55 and 75. This correlated shock would significantly

[36] 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.

increase mortality rates at the same time that Medicare lowers them. Indeed if we estimate the model for widows we find significant declines in mortality. This can be seen in Figure

A final possibility is that the medical technologies available in 1966 were not particularly effective at treating women. Although cardiovascular disease was also the main cause of death of women over 65 at the time, the majority of studies on the prevention and treatment of cardiovascular disease at this time were conducted primarily on men and it was assumed that the results would apply to women. More recent research has shown this to be incorrect. As Thomas and Braus (1998) noted *"Women's health issues that did not concern reproduction received little attention and little research funding prior to 1986. This was particularly unfortunate in the area of heart disease, which was long considered to be a greater threat to men than to women. Researchers have found more recently that, while men typically develop heart disease earlier in life than women, women have a worse prognosis than men once they have had a myocardial infarction. Women also face a worse prognosis than men following surgical therapy for coronary artery disease (CAD)."* Other more recent work has confirmed these observations.

Why do results change for the most recent cohorts?

VII.D Using estimates to predict LE gains among current cohorts. (TBA) Also say something about external validity to today – mention our cohorts die up to 2023, so they experience not just medicare then but medicare as it has operated since its inception.

What if we lower eligibility to younger ages?Age 65 was chosen arbitrarily following the German model. Maybe have called for expanding Medicare. We can simulate the effects of doing this.

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

VII.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 between 1885 and 1905, and that they generated cohort-level changes in life expectancy of up to 2 years. 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 for men.

We do not find the same to be true for women for whom the estimates are more unstable. Whether this is the results of identification failures or not is unclear. However, it is also true that medical understanding of how to treat cardiovascular disease for women was inadequate in the 1960s perhaps explaining why we fail to find effects. These large sex differences necessitate further investigation, but they are not unique to our study.

Future work should also investigate effects on health. There are a number of valuable health interventions that improve the quality of life without extending life, particularly among older populations. These include palliative care, chronic pain management, hearing and vision aids hip replacement, and perhaps mental health interventions. Our estimates do not quantify these benefits.

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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 ($Cohort\ FE * EventAge$).

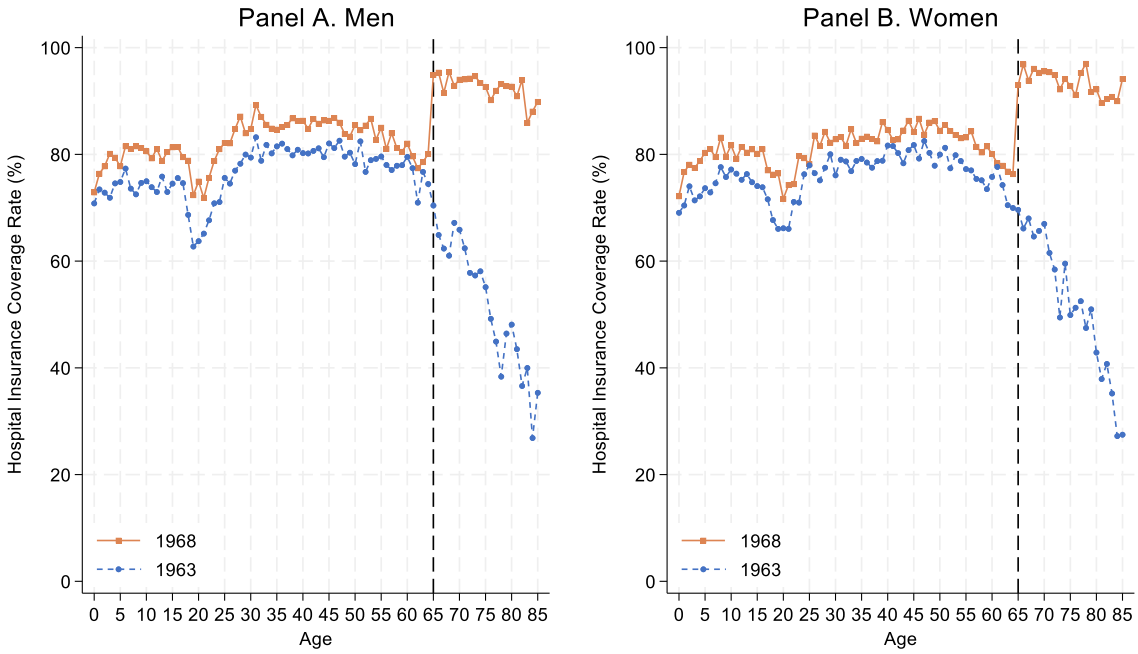
Table 3. Estimates of Medicare's average treatment effect on Life Expectancy at Age 65 overall, by education, and by income in 1940

	N	Mean Life expectancy at age 65	ITS Estimates	DiD Estimates
Panel A. Males				
All men	1,985,048	14.35	1.00	1.13
			(0.86, 1.15)	(0.80, 1.45)
Education				
0-7 years of schooling	465,407	13.57	1.11	0.96
			(0.89, 1.37)	(0.48, 1.47)
8-11 years of schooling	922,526	14.10	0.96	1.05
			(0.75, 1.18)	(0.61, 1.52)
12+ years of schooling	557,896	15.12	0.85	1.05
			(0.53, 1.24)	(0.25, 2.01)
Household total income in 1940				
Below Median (mean $284)	992,602	14.20	0.89	1.02
			(0.69, 1.11)	(0.55, 1.50)
Above Median (mean $2,389)	992,446	14.46	1.07	1.22
			(0.86, 1.31)	(0.76, 1.77)
Panel B. Females				
All women	1,712,115	18.74	-0.55	0.75
			(-0.85, -0.24)	(0.11, 1.34)
Education				
0-7 years of schooling	357,273	17.60	0.01	1.32
			(-0.53, 0.60)	(0.46, 2.24)
8-11 years of schooling	776,058	18.59	-0.45	0.34
			(-0.89, -0.01)	(-0.42, 1.09)
12+ years of schooling	547,351	19.62	-1.11	0.48
			(-1.76, -0.57)	(-0.88, 1.85)
Household total income in 1940				
Below Median (Mean $252)	856,132	18.55	-0.31	0.90
			(-0.75, 0.09)	(0.17, 1.67)
Above Median (Mean $1,830)	855,983	18.90	-0.76	0.55
			(-1.19, -0.32)	(-0.29, 1.47)

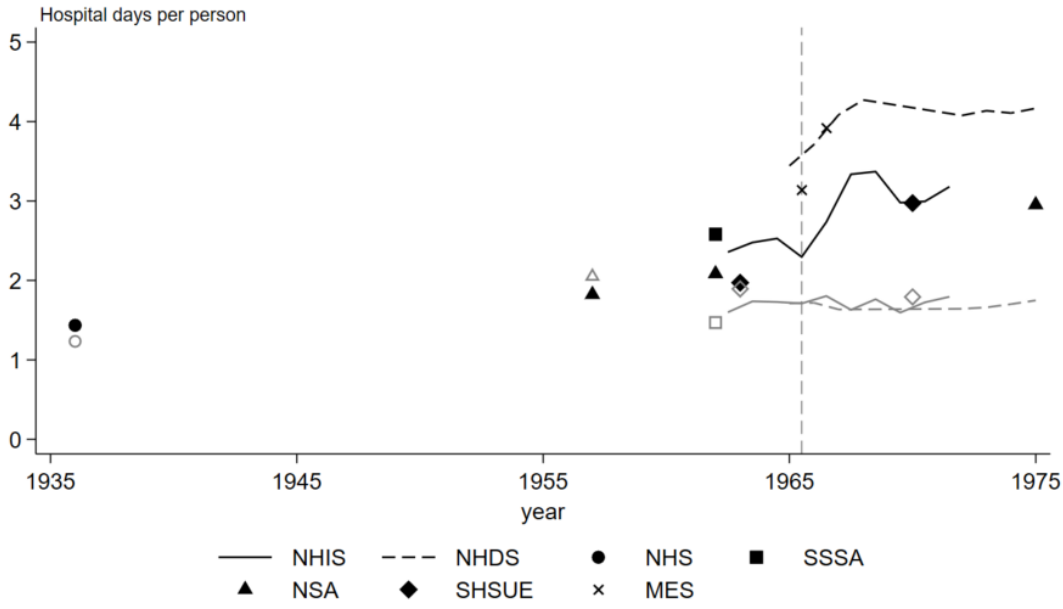
Notes: This table reports the estimated life expectancy gains associated with gaining access to Medicare. The Appendix contains the details of our computations. All estimates are produced using the CensusTree data. In parentheses are bias-corrected 95% confidence intervals produced by bootstrapping with 1,000 resamples. Mean Life expectancy at age 65 denotes weighted average cohort-specific life expectancy at age 65 using the number of people survived to Medicare eligibility age as weights.

Figure 1. Medicare and rates of insurance and health care utilization

a. Hospital insurance coverage before and after Medicare's passage



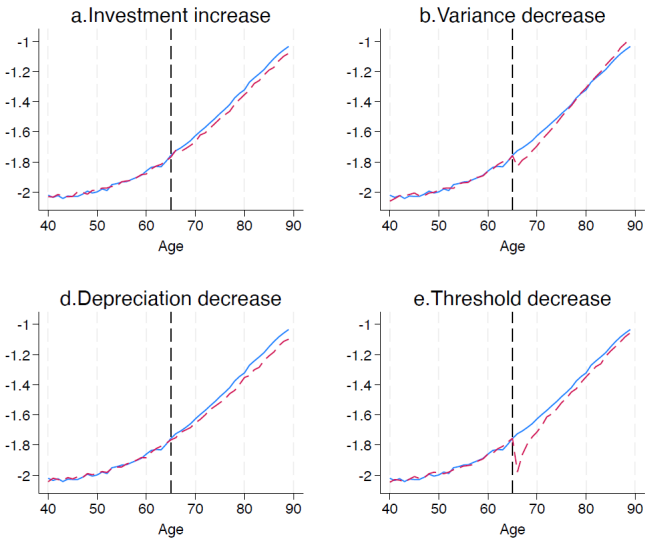
b. Hospital utilization trends for over and under 65 population



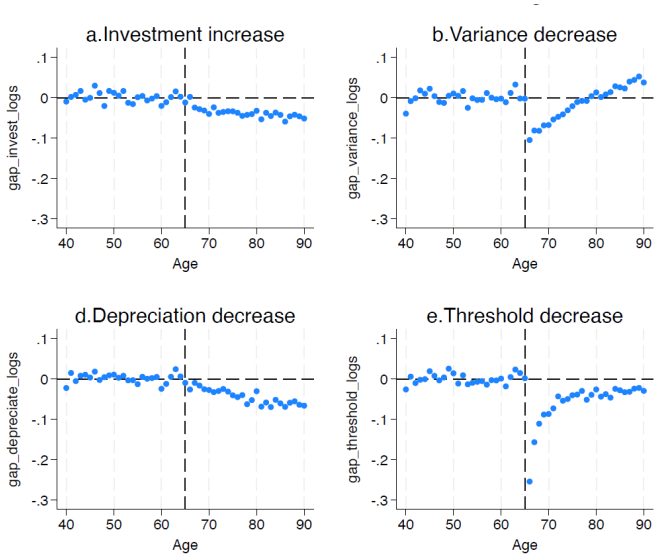
Notes: Figure 1.a. uses the National Health Interview Survey, a nationally representative survey of the ono-institutionalized population to document changes in hospital insurance rates between 1963 and 1968. Figure 1.b shows days of hospital care per person from several data sources for individuals ages 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

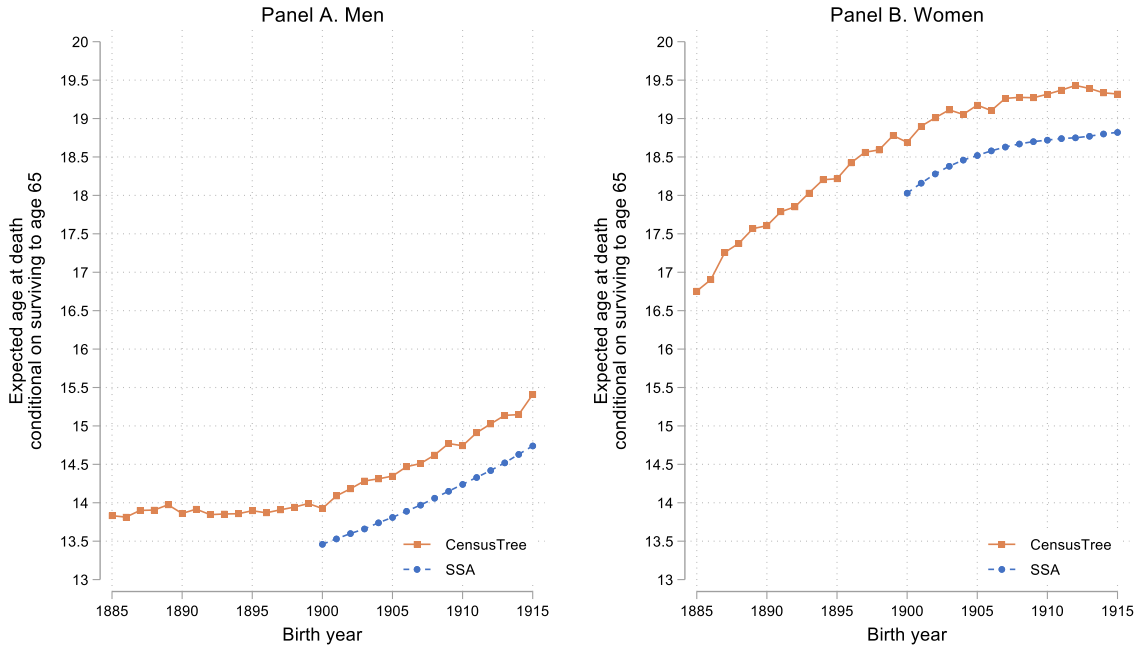


b. Effects of parameter changes at age 65 on log mortality



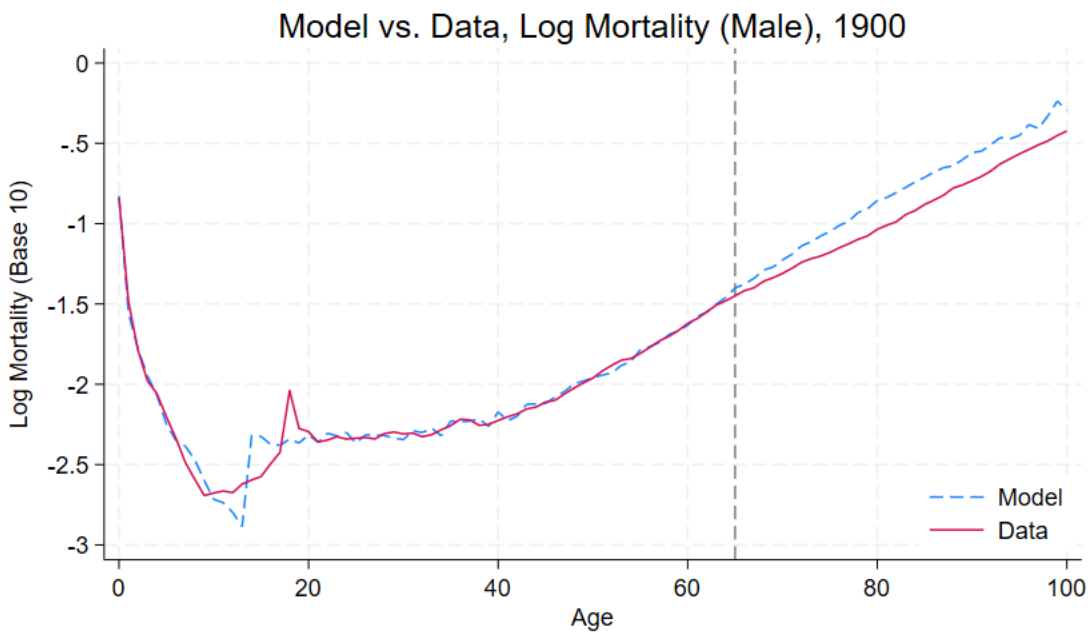
Notes: The figures show simulated results based on Lleras-Muney and Moreau (2022)'s unified model for cohort mortality, with parameter values for the baseline model given by $I = 0.4$, $\delta = 0.0006$, $\sigma = 1$, $\alpha = 1.7$, $\mu_0 = 0.9$, and an adolescent accident shock $\kappa = 0.008$. The 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. The expected age at death conditional on survival to age 65.



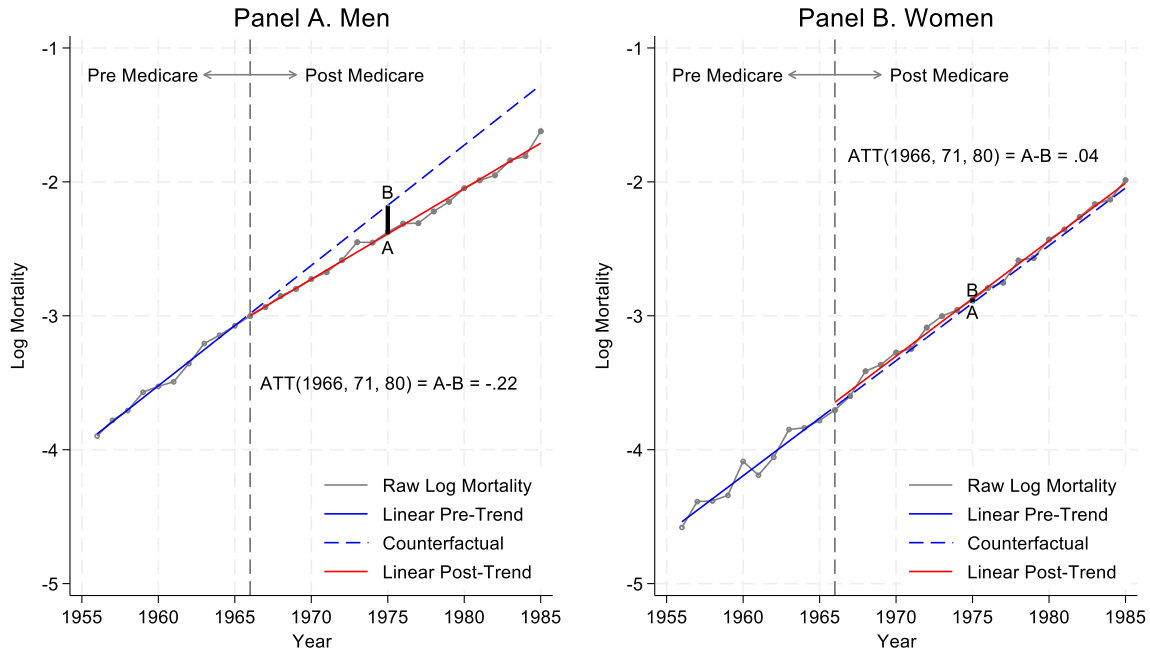
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort.

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



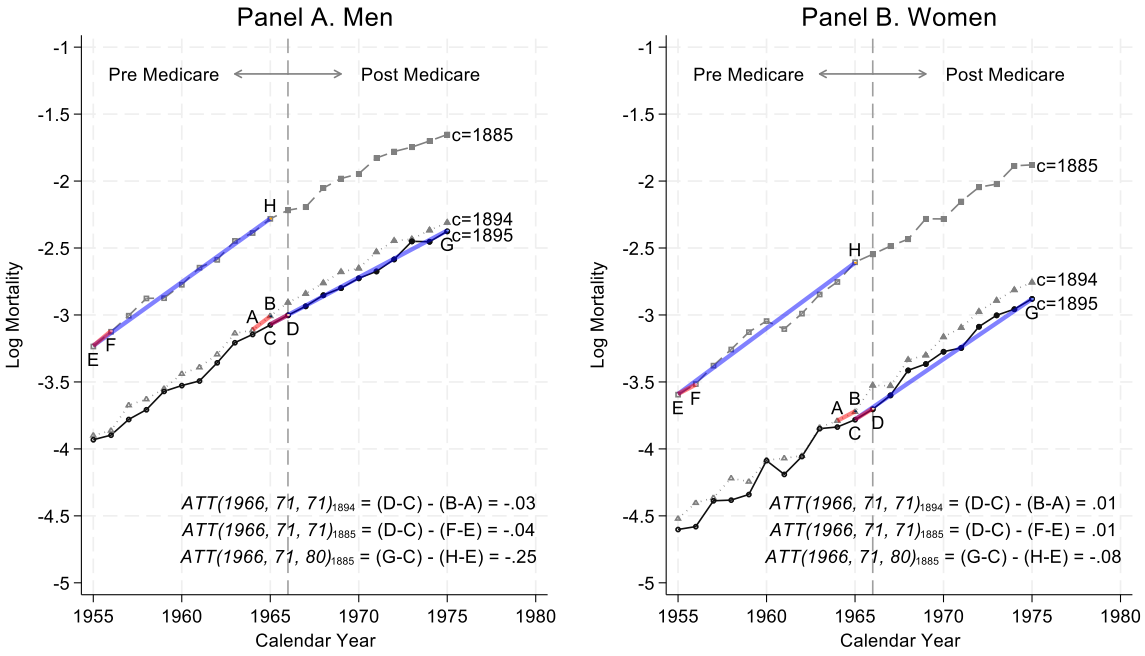
Notes: This figure demonstrate how the model can be used for identification using the Social Security Administration cohort tables (dashed blue line) starting at birth. We first estimate the model using data up to age 65. We then project mortality rates beyond age 65 using the estimated parameters (solid 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.

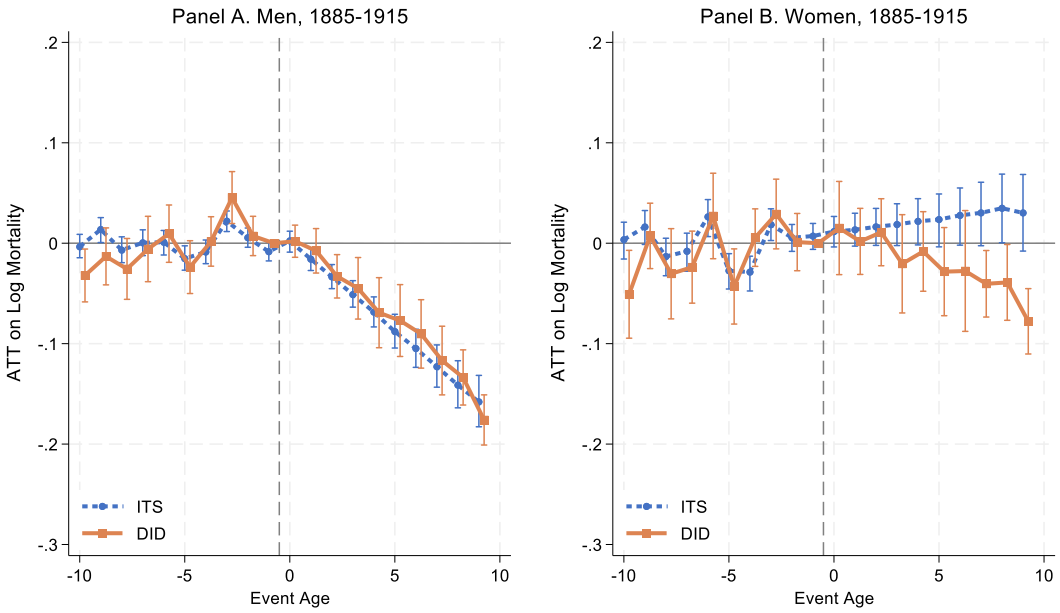
Figure 6. Identification of Medicare's effect on one cohort's log mortality rates in the difference-in-differences design



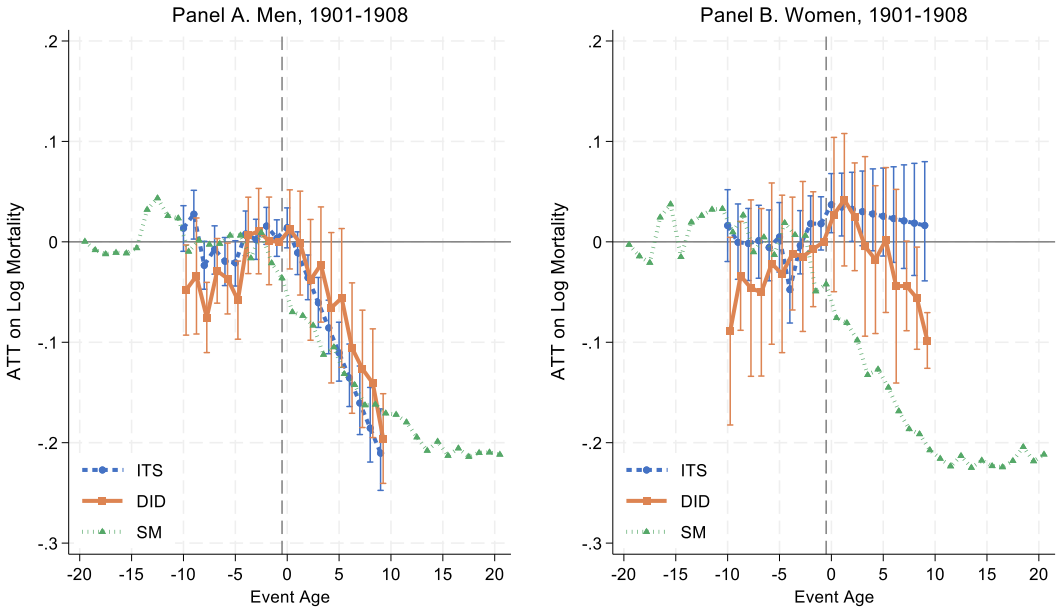
Notes: This figure highlights the estimator for $ATT(g_t, g_a, t)$ for the 1895 cohort as an example. We describe the treatment effect for the Medicare's introduction year ($t = 1966$) and 9 years after the introduction. This effect is identified by comparing the change in the (log) mortality rate before (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



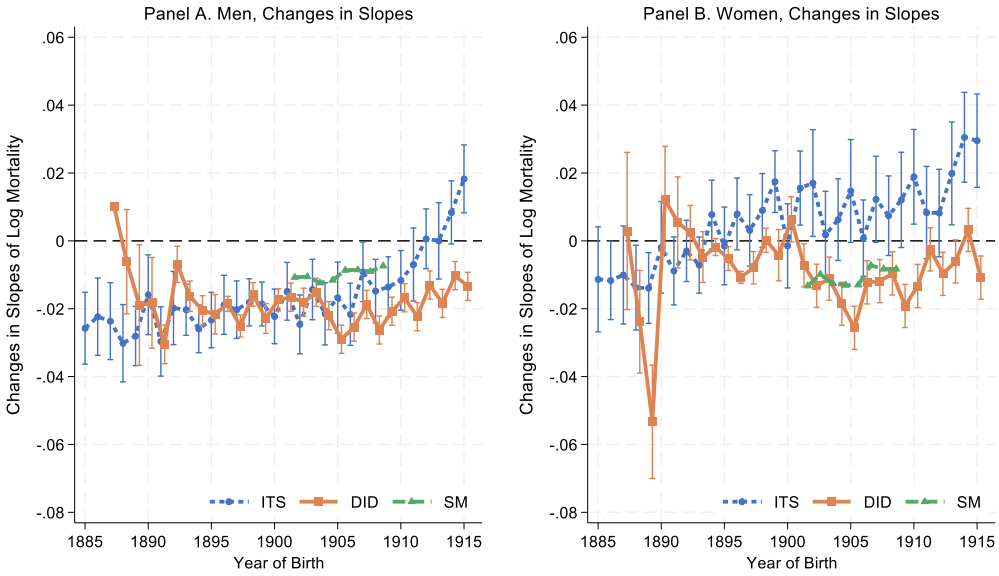
b-Structural Approach for selected cohorts



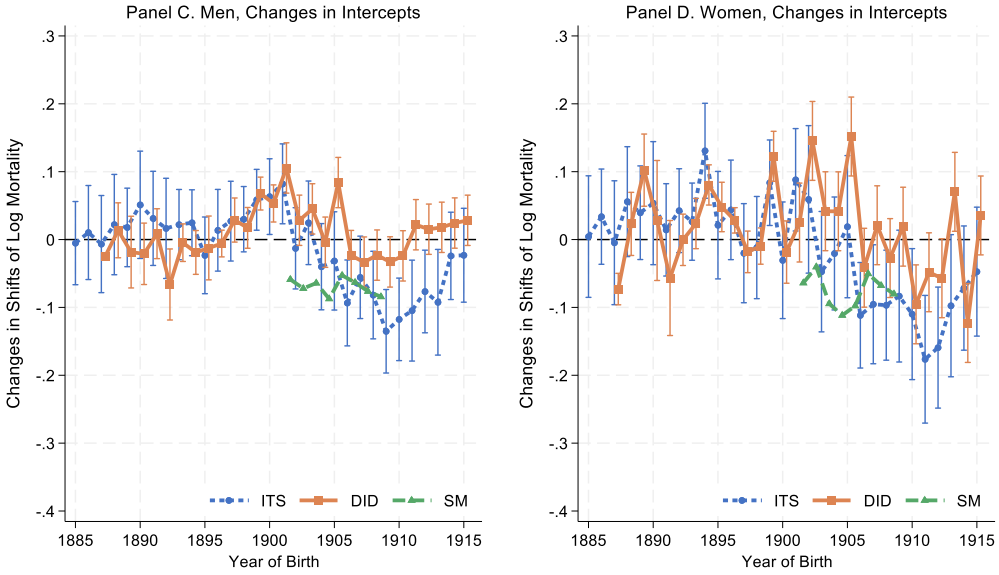
Notes: The figure 5a plots the estimates of $ATT(e)$ parameters from equation (Y) for men and women using both the ITS and DiD designs across all birth cohorts. Figure 5b reports the estimates for the subset of cohorts available in the SSA life tables for which we have complete cohort data from birth 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

a-Changes in slopes

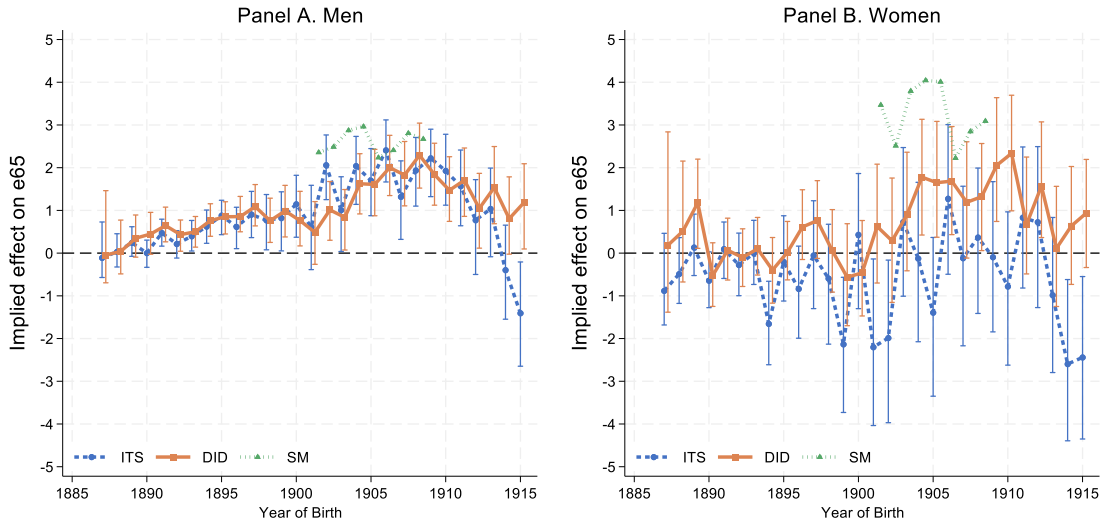


b-Changes in intercepts



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 65 by cohort



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

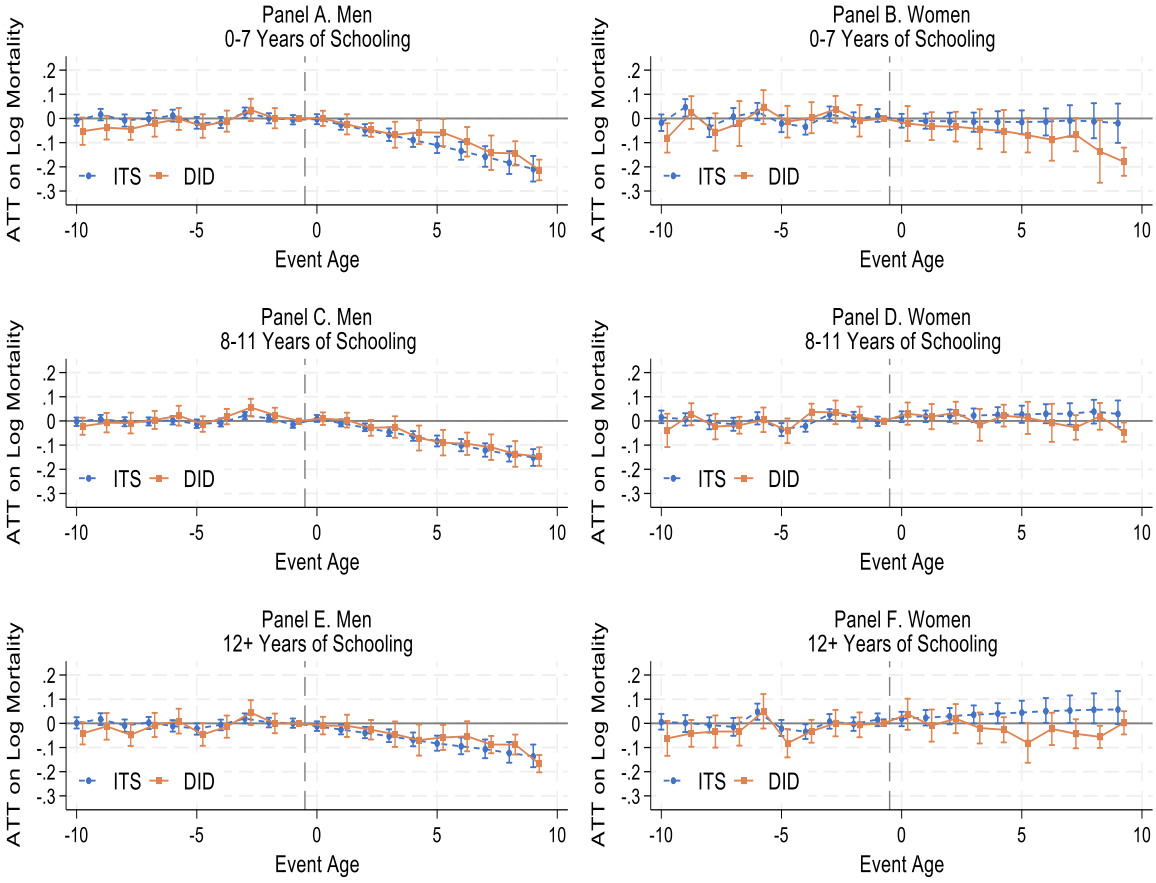


Figure 11. Estimates of Medicare's average treatment effect on log mortality rates by event-age and income group

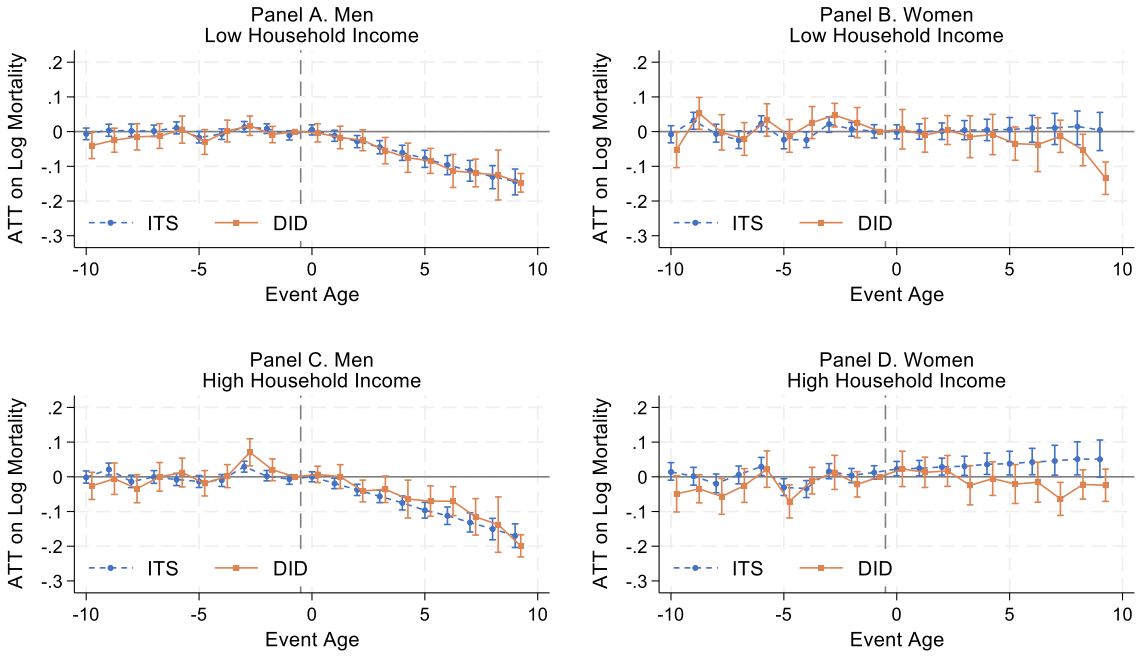
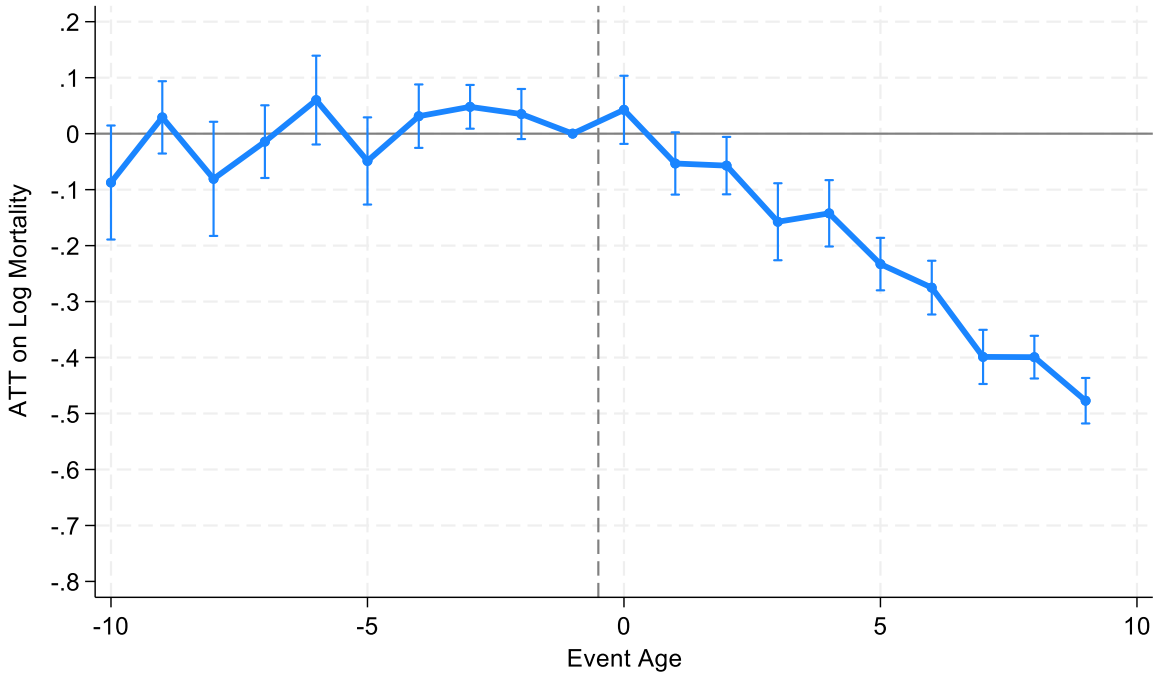


Figure 12: DiD estimates of the effects of Medicare among widows

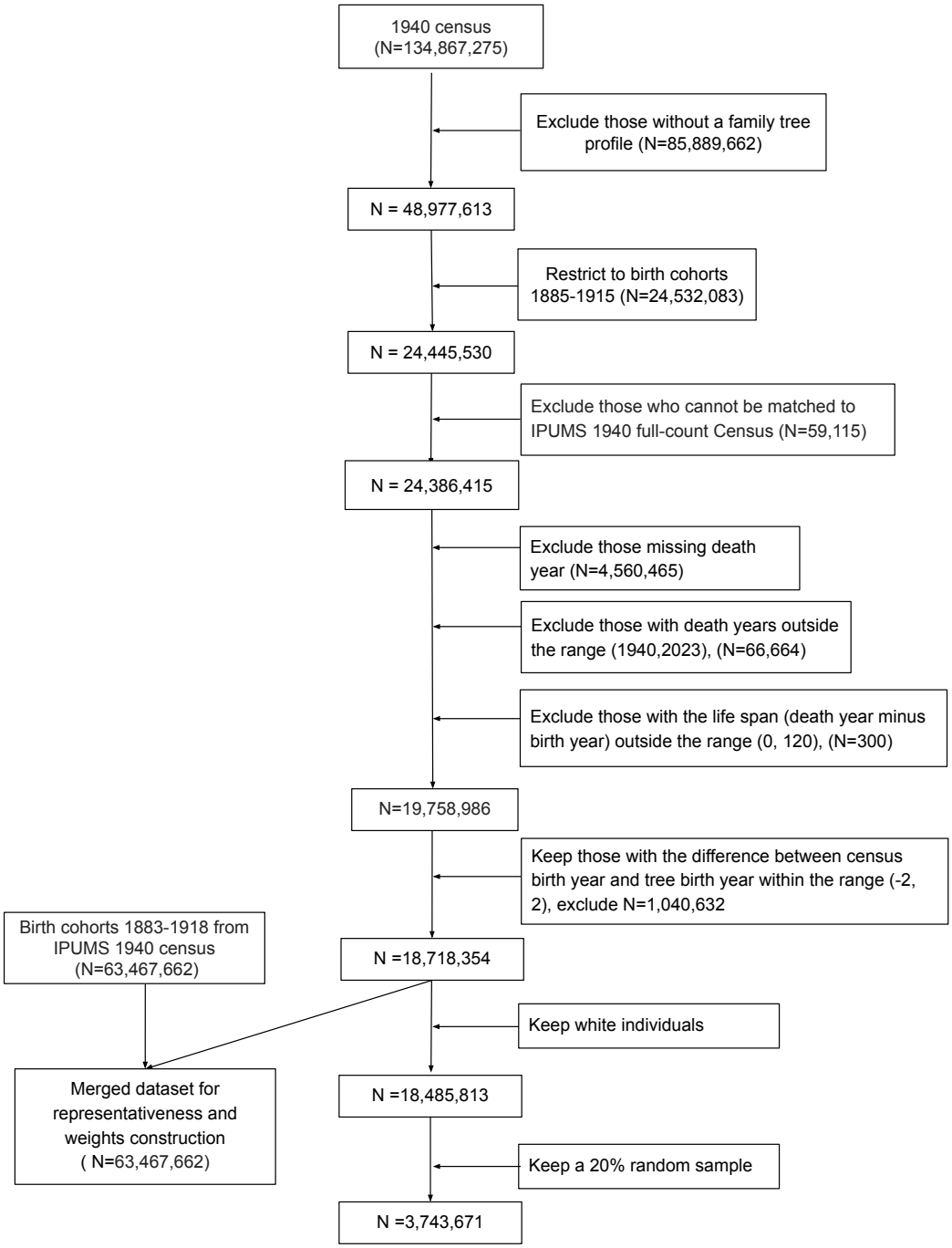


Note: We define widows as women whose husbands died before them. The comprise about 30% of the sample of women, though there are some women in the sample whose husband's death date cannot be observed and they are not included in these regressions.

Appendix

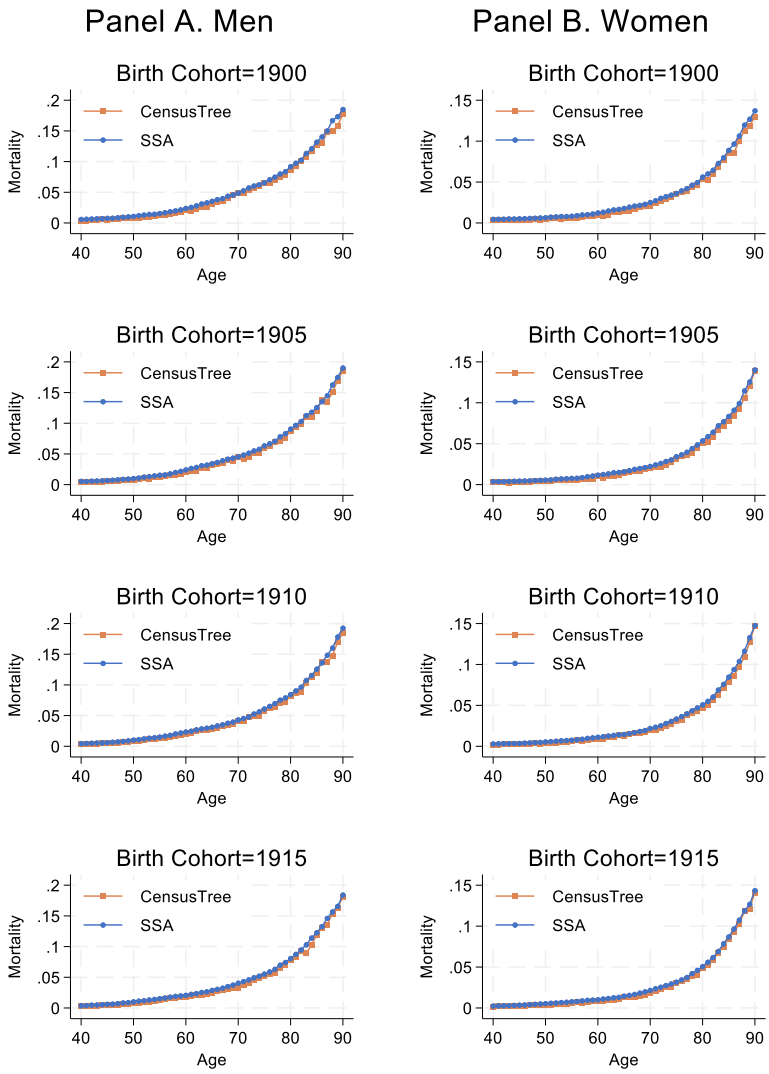
1. Figures
2. Tables
3. Computation of implied effect on ex65 based on the ITS analysis
4. Computation of implied effect on ex65 based on the DiD analysis (TBA)
5. Details on the estimation of the structural model (TBA)
6. Behavioral responses to Medicare (TBA).

Figure A1. Sample flowchart of Census Tree Sample



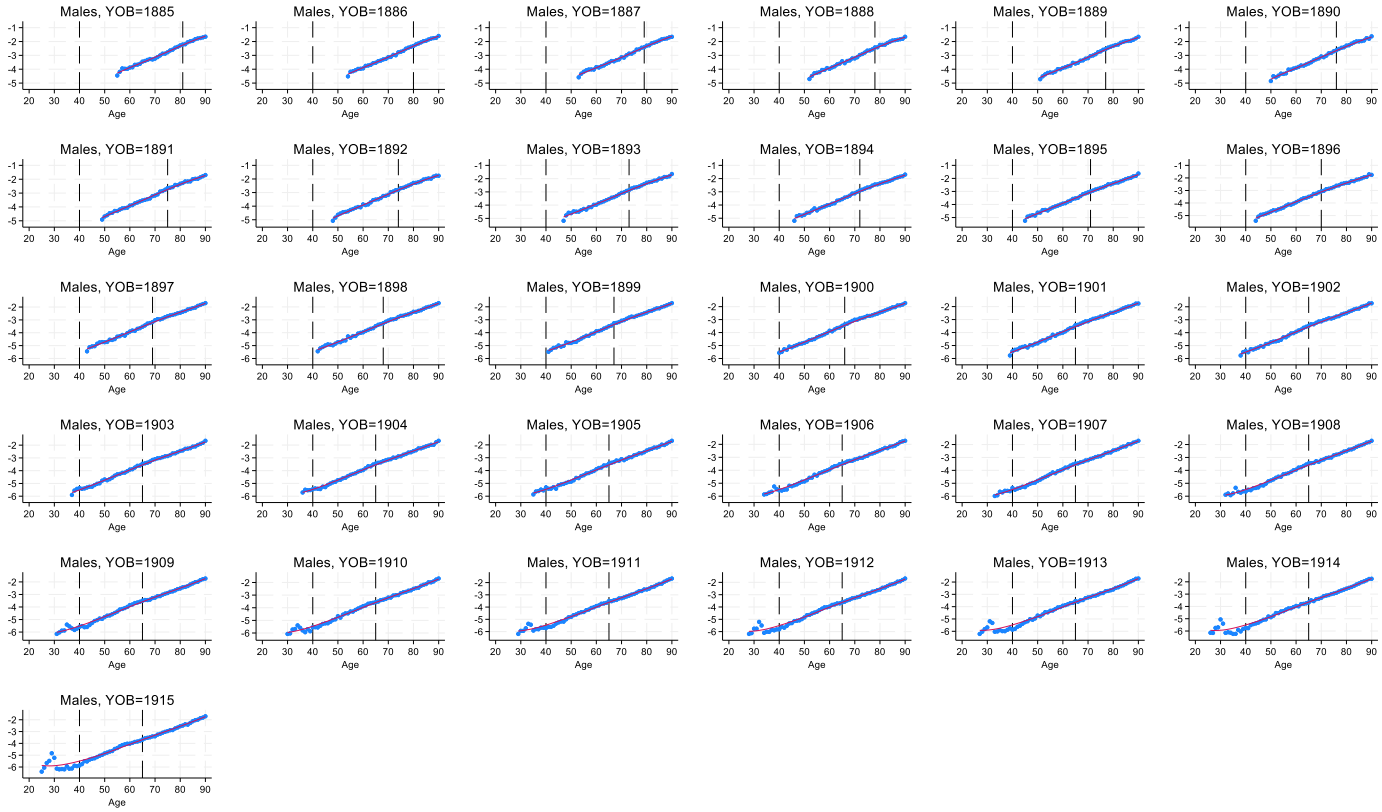
Notes: We rely on the year of birth recorded in the Family Tree in our analysis because the 1940 Census does not ask for birth year and only reports age. However, the birth year variable from Family Tree was scrapped because people would have had the chance to find birth records and make corrections. 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. Comparison of mortality between Census Tree and SSA life tables for selected cohorts



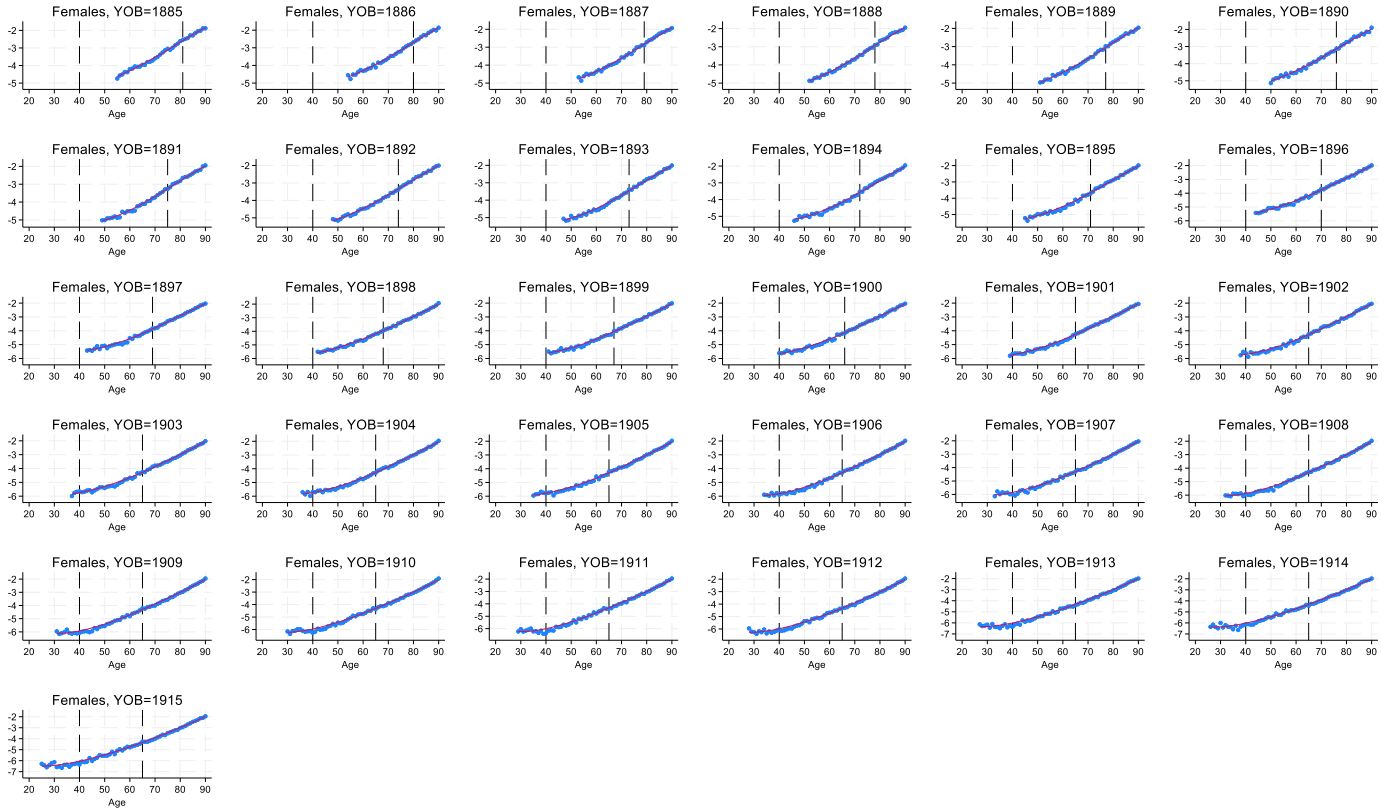
Notes: Shown are age specific mortality rate by cohort, sex, and data source. The CensusTree data represent a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. We compute age-specific mortality rate using the inverse probability 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 A3a. Gompertz curves for studied birth cohorts of white males. (updated)



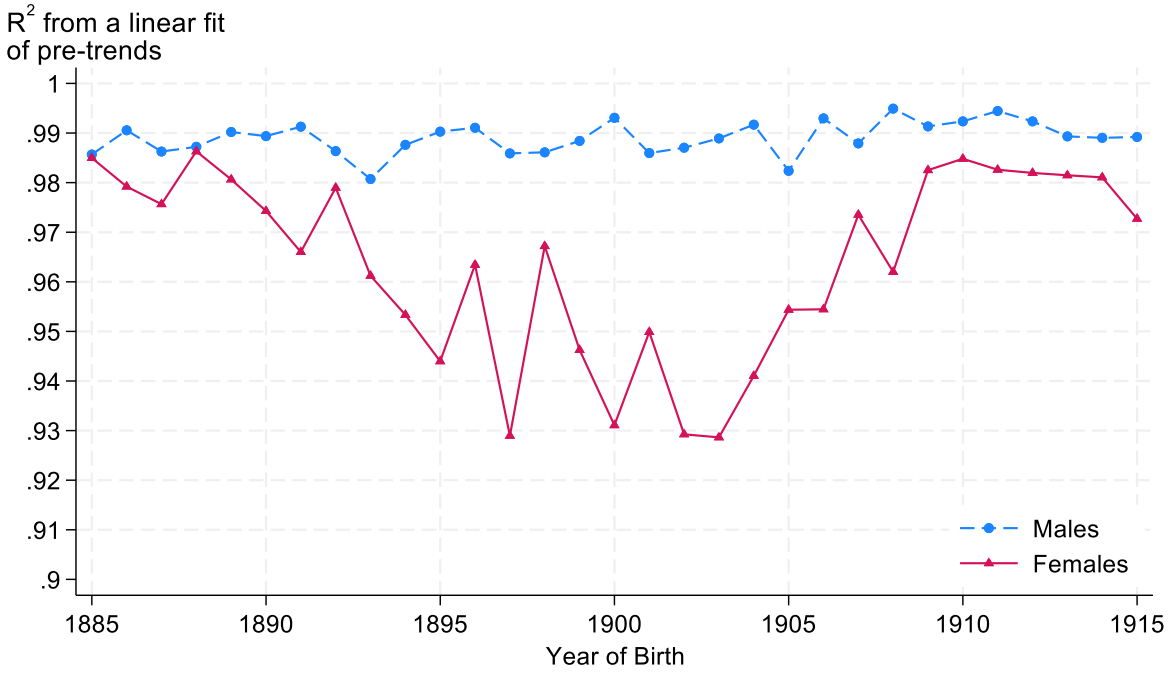
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. We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

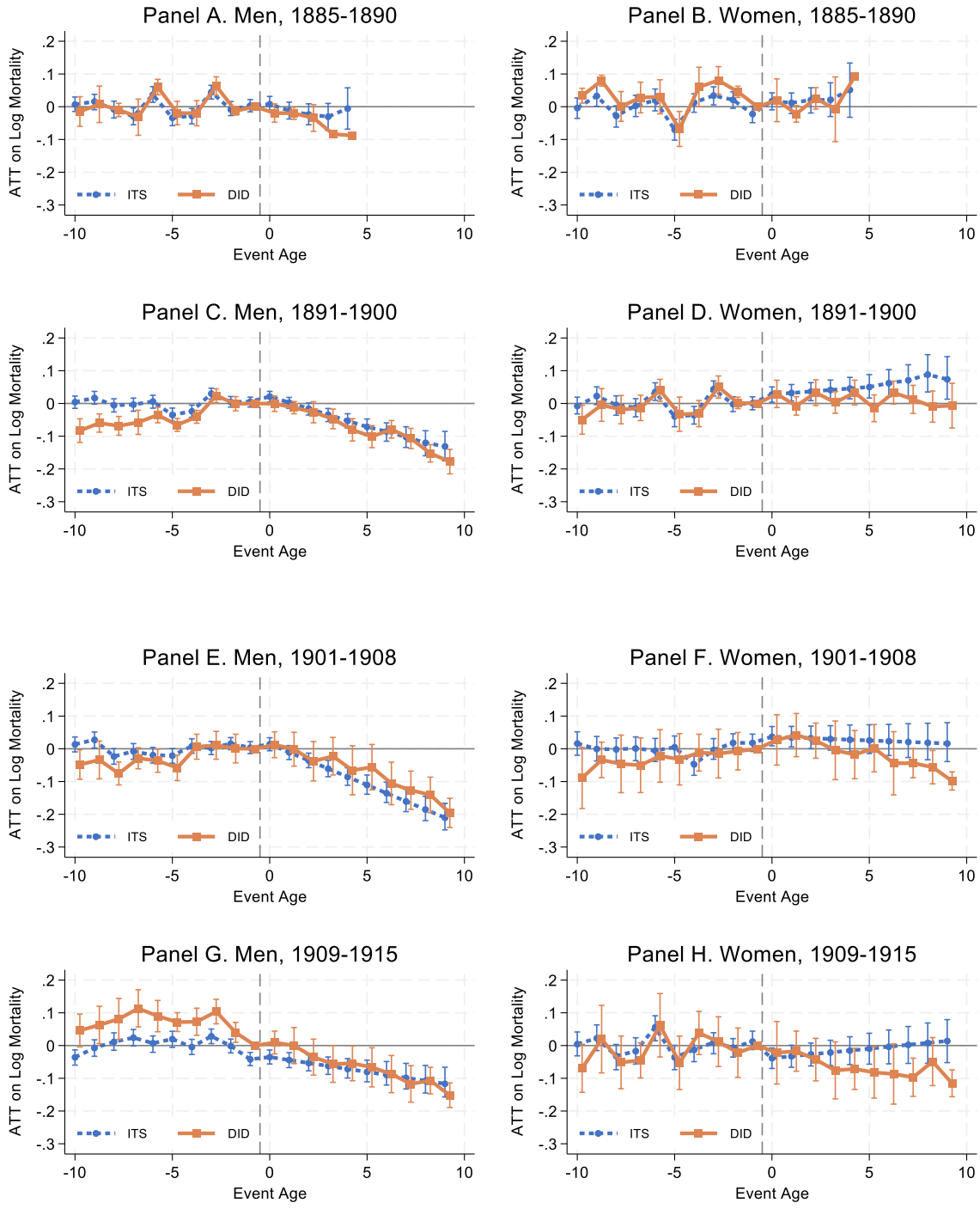
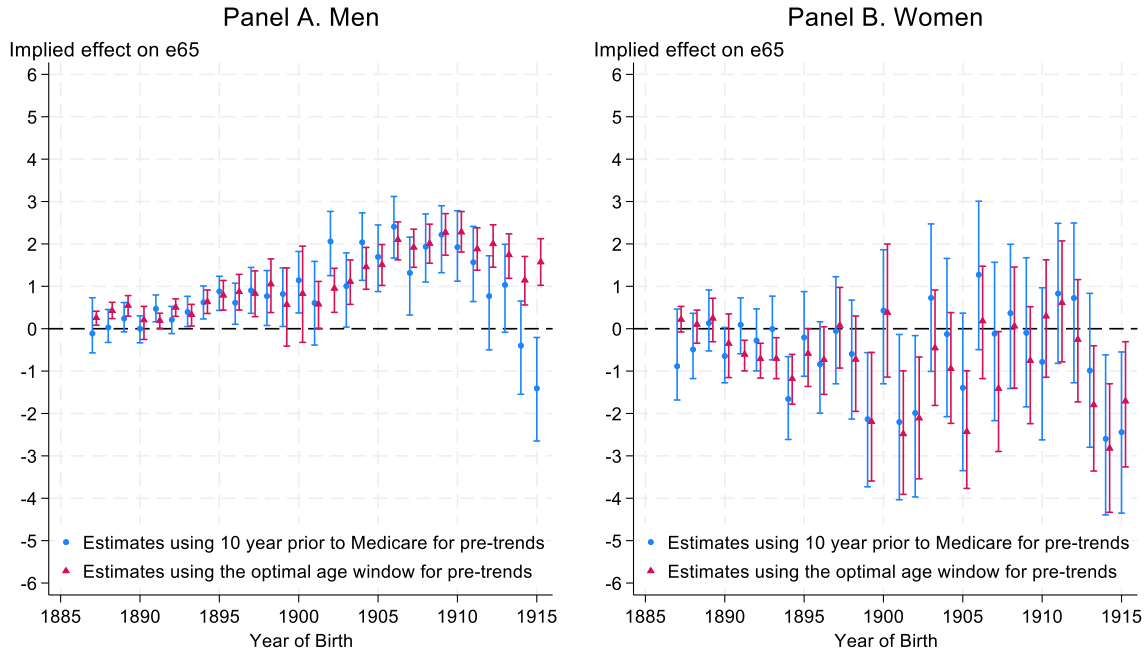
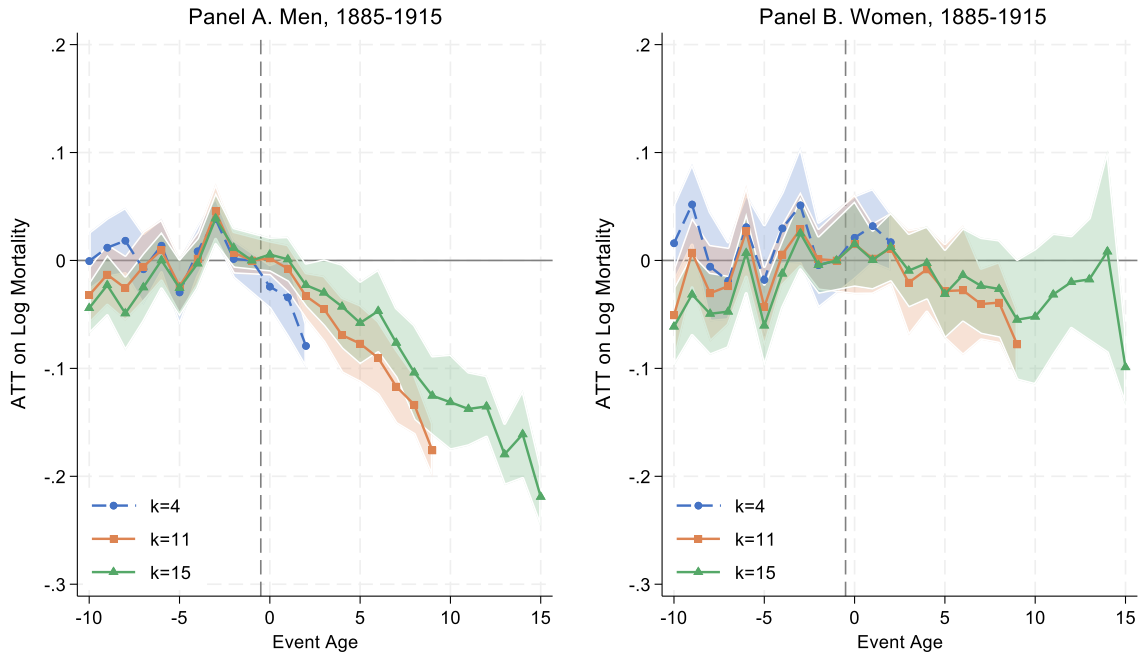


Figure A6. Implied effect of Medicare on life expectancy at age 55 using the ITS approach with the optimal age window for pre-trends



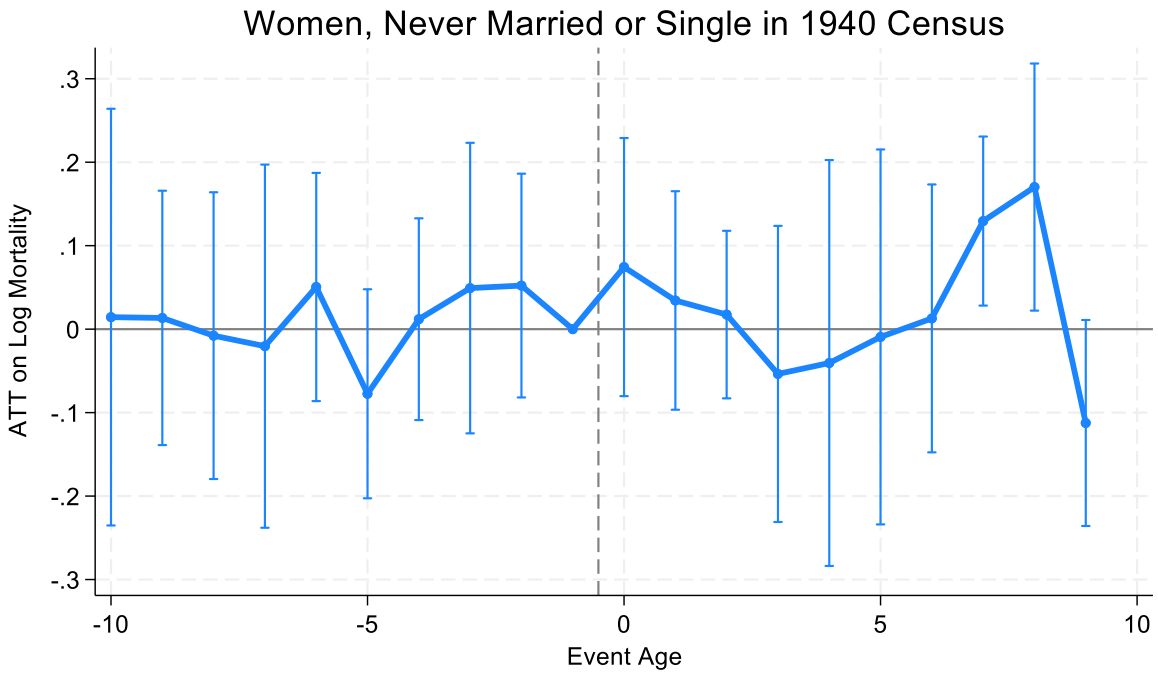
Notes: Shown are implied effect of Medicare's introduction on life expectancy at age 55 based on our interrupted time series (ITS) approach, by sex, birth cohort, and approach. All estimates are from a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count 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.



Notes: Shown are event-study estimates from the DiD approach with different restrictions on the comparison cohorts, defined by k. When $k = 4$, the comparison cohorts received Medicare no more than 3 years later than a treatment cohort. When $k = 11$, the comparison cohorts received Medicare no more than 10 years later than a treatment cohort. When $k = 15$, the comparison cohorts receiv Medicare no more than 14 years later than a treatment cohort, which used all available cohorts in our data.

Figure A8: Effect of Medicare on never married women or women that were single in the 1940 Census



Notes: Shown are event study estimates from the DiD approach with k=11. The analytic sample includes women who reported never married or single in the 1940 census. N= 149,106.

Figure AX. Event-Study Estimates from the Structural Model

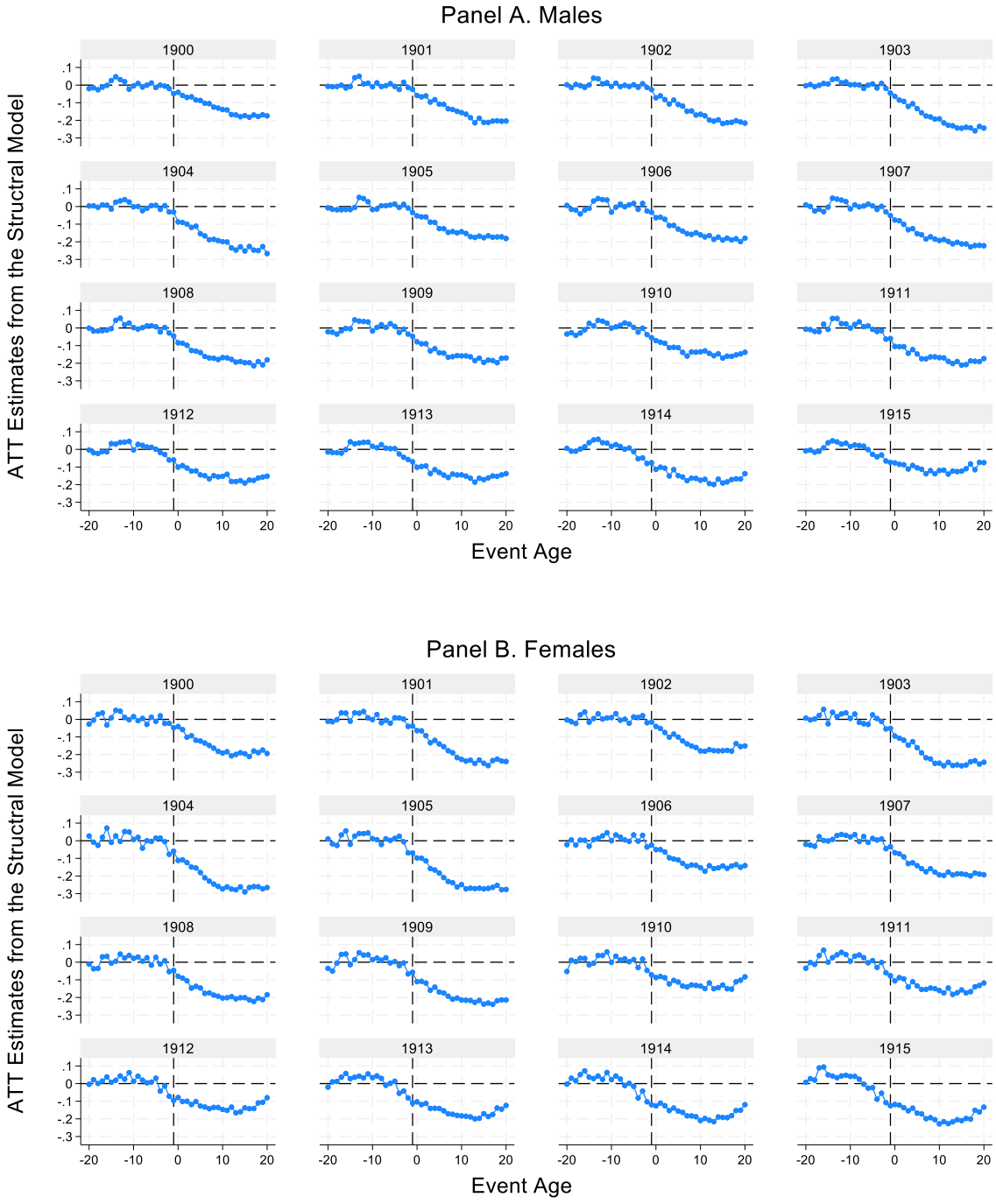
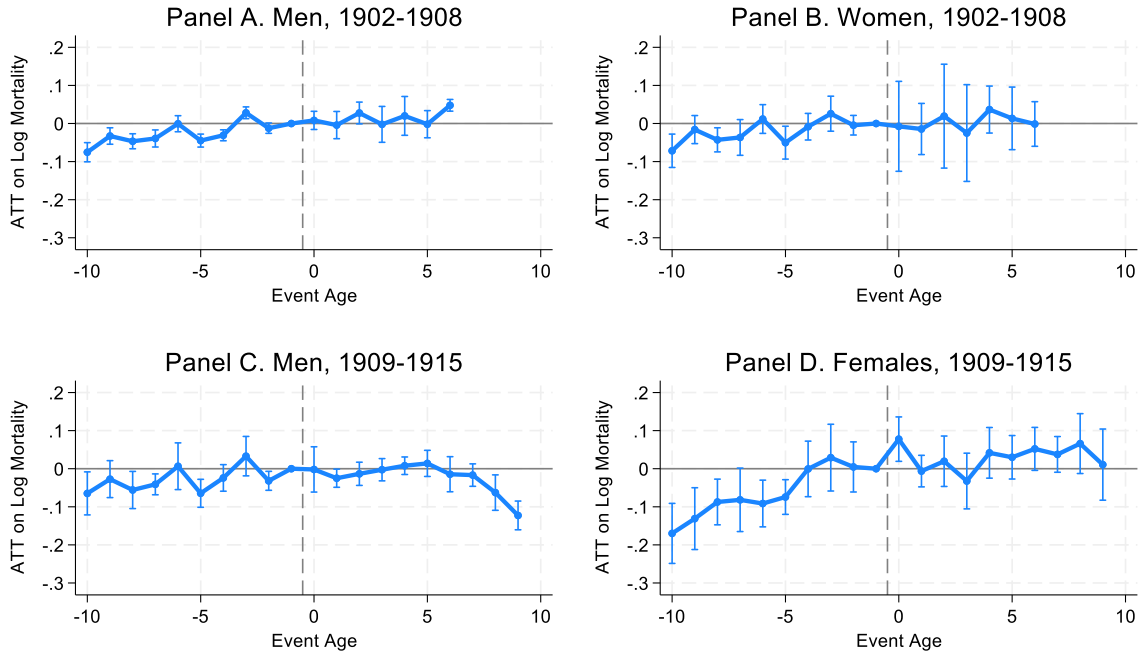
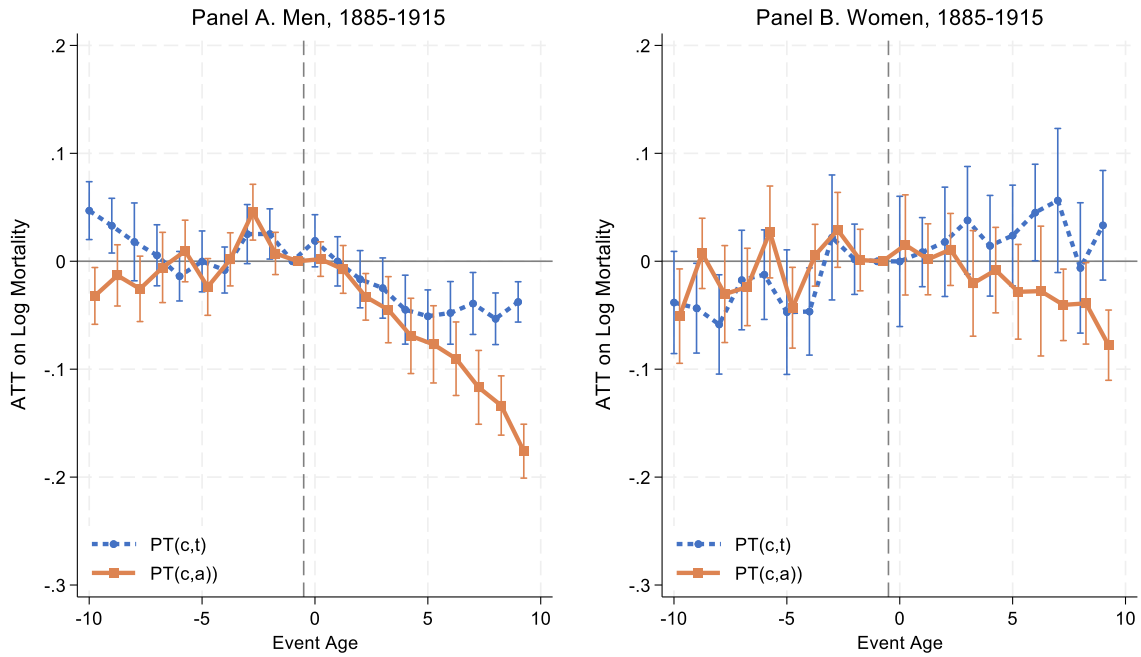


Figure AX1. Falsification test using 1966 as the treatment for 1902-1915 cohorts



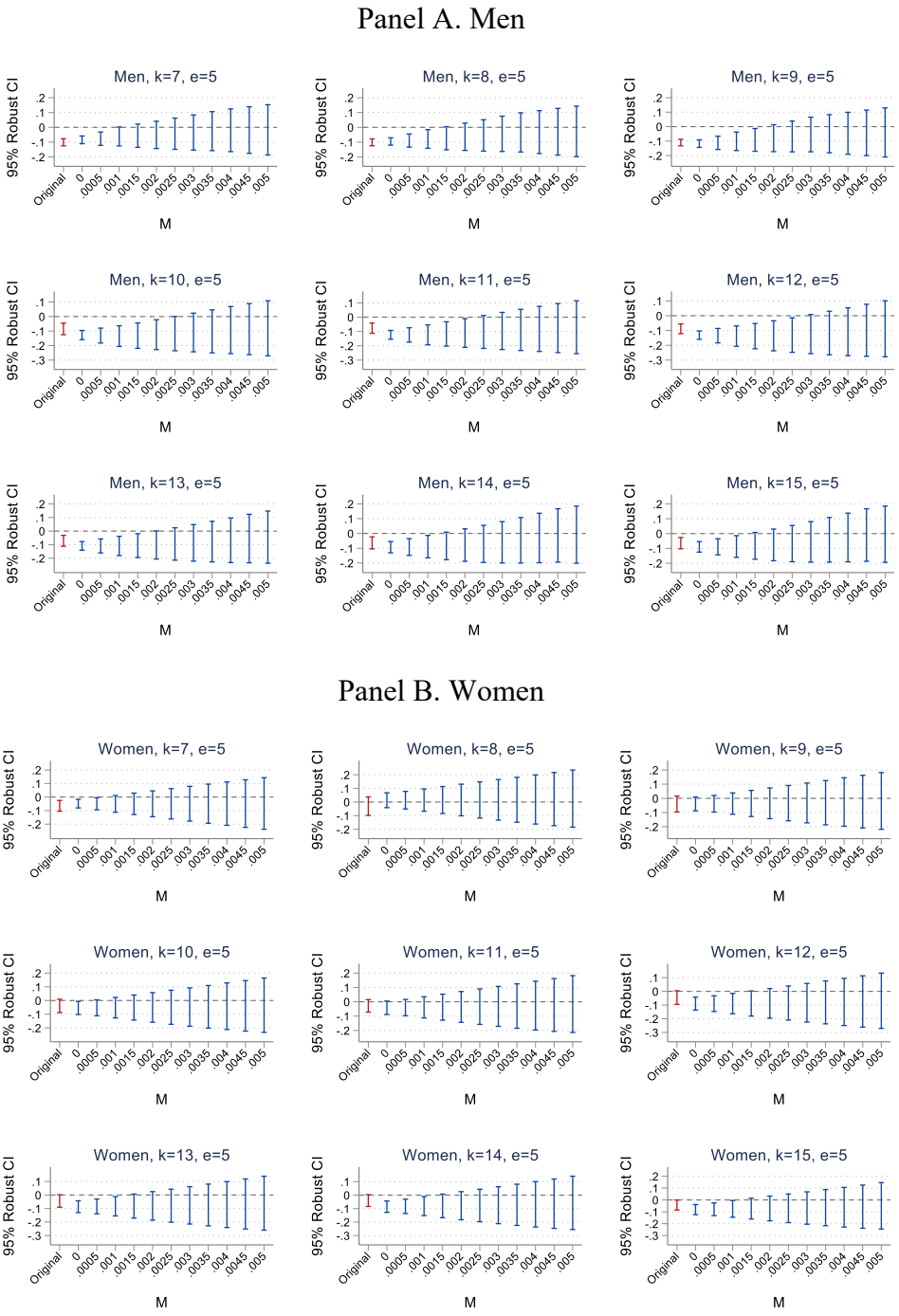
Notes: In this falsification test, we use aggregated log mortality data by sex and cohort but restrict to the pre-Medicare ages. We assume 1966 as the treatment year for those more recent cohorts (1902-1915) when they aged 51-64 and included older cohorts as controls.

Figure AX2. Difference-in-differences estimates with an alternative parallel trends assumption PT(C,T)



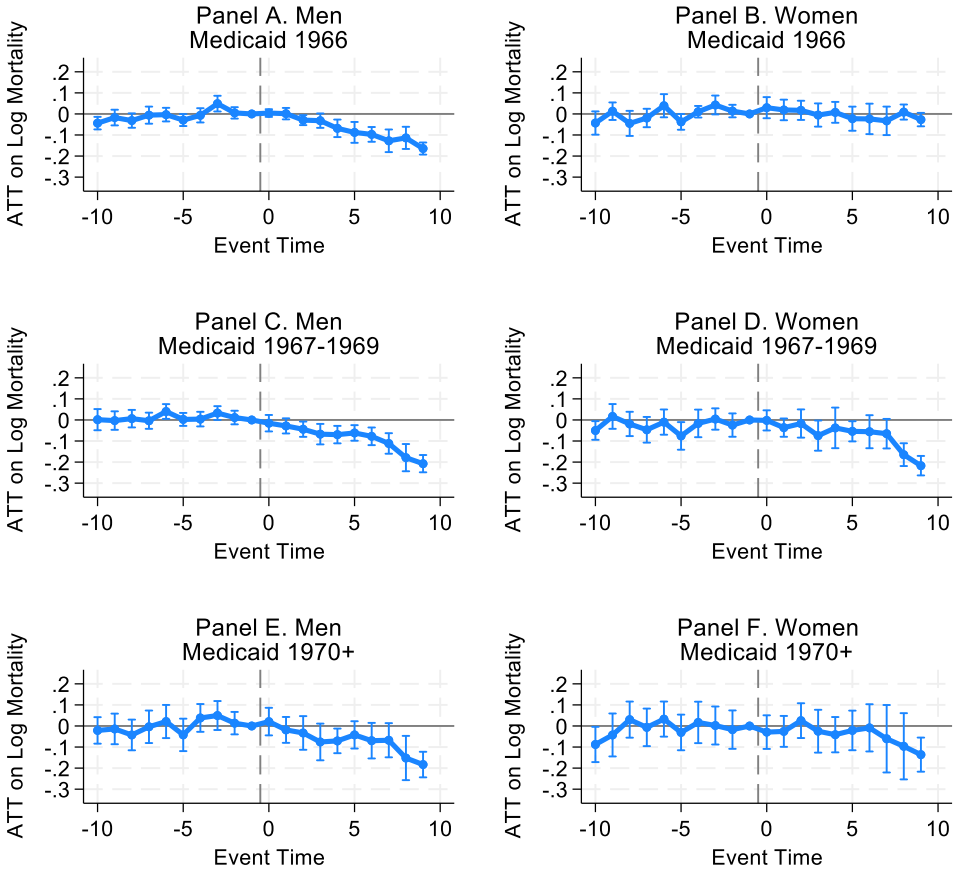
Notes: In this analysis, we use an alternative difference-in-differences (DiD) study design based on comparing changes in log mortality for a cohort (c) treated in year (t), to the change in log mortality over the same time periods for a comparison group of younger cohorts treated in later years; the parallel trend assumption is PT(c,t). In our main analysis, we compare changes in log mortality for a cohort (c) treated in year (t), to the change in log mortality over the same age for a comparison group of older cohorts treated at older ages; the the parallel trend assumption is PT(c,a).

Figure Ax3. Selection of k in the difference-in-differences study design using smoothness restrictions of Rombachan and Roth (2023).



Notes: In our difference-in-differences design (equation 8), we need to select the nearby cohorts for comparisons. To choose k, we conducted sensitivity analysis based on the smoothness restrictions of Rambachan and Roth (2023) using our 20% training sample. Using all cohorts, we estimate the sensitivity of ATT estimate at event age 5 for k∈{7,15}, and calculate the maximum period-to-period violation, Mbar, 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 maximize Mbar—the breakdown value for a significant effect. Mbar=0 denotes the ATT estimate at event age 5 based on a linear extrapolation of pre-trends.

Figure AX. CSDID Event study estimates by Medicaid rollout year



Notes: Shown are event-study estimates from our DiD approach, stratified by state's Medicaid rollout period. States that implemented Medicaid in 1966 include California, Connecticut, Idaho, Illinois, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Nebraska, New Mexico, New York, North Dakota, Ohio, Oklahoma, Pennsylvania, Rhode Island, Utah, Vermont, Washington, West Virginia, and Wisconsin. States that introduced Medicaid in 1967-1969 include Colorado, District of Columbia, Georgia, Iowa, Kansas, Missouri, Montana, Nevada, New Hampshire, Oregon, South Carolina, South Dakota, Tennessee, Texas, Virginia, and Wyoming. States that introduced Medicaid in 1970 or later include Alabama, Arizona, Arkansas, Florida, Indiana, Mississippi, New Jersey, and North Carolina. Note that Hawaii and Alaska were excluded from this analysis. Delaware is not included because of the lack of sample size in the 20% training data.

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

Cohort	I	Delta	Sigma E	Alpha	Mu H	Kappa	I Flu	Fit
1900	0.185857	0.000588	0.396904	1.537628	1.039128	0.003421	0.144972	0.610847
1901	0.198339	0.000598	0.41938	1.555101	1.12038	0.003362	0.159735	0.430616
1902	0.183633	0.000591	0.397618	1.547402	1.130027	0.003172	0.153828	0.356146
1903	0.210434	0.000406	0.437437	1.656926	1.158004	0.003092	0.158467	0.427097
1904	0.191934	0.000607	0.402983	1.553661	1.128296	0.002984	0.163471	0.42032
1905	0.220801	0.000871	0.440424	1.473545	1.129444	0.002509	0.171224	0.623492
1906	0.224981	0.000905	0.437892	1.46691	1.120221	0.002463	0.17443	0.598782
1907	0.216053	0.000883	0.422748	1.473351	1.144007	0.002485	0.1758	0.568277
1908	0.226071	0.001544	0.43202	1.353749	1.170935	0.002406	0.198678	0.705891
1909	0.249161	0.001175	0.458147	1.417157	1.196309	0.002029	0.188954	0.981687
1910	0.294168	0.001535	0.500254	1.369112	1.191308	0.001738	0.205908	1.564212
1911	0.295303	0.001548	0.501926	1.379879	1.264296	0.001885	0.21882	1.411769
1912	0.333774	0.001504	0.522993	1.386133	1.257879	0.001508	0.216238	1.574186
1913	0.409235	0.001876	0.571386	1.35188	1.248685	0.001204	0.237471	1.932289
1914	0.446752	0.002007	0.566278	1.341936	1.261894	0.00131	0.246893	2.812347
1915	0.597628	0.006902	0.658532	1.096046	1.280844	0.001115	0.344719	2.916599

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

Cohort	I	Delta	Sigma E	Alpha	Mu H	Kappa	I Flu	Fit
1900	0.23645	0.000781	0.460506	1.52048	1.159841	0.003888	0.226491	0.624191
1901	0.232774	0.00075	0.461915	1.541758	1.234128	0.003793	0.237543	0.584383
1902	0.238673	0.000828	0.474971	1.505343	1.256585	0.003449	0.226964	0.552024
1903	0.232687	0.001177	0.461778	1.448429	1.273879	0.003457	0.262017	0.585931
1904	0.24742	0.001238	0.476209	1.449087	1.257898	0.003373	0.275947	0.581653
1905	0.255561	0.001228	0.481759	1.453678	1.248246	0.003286	0.278741	0.796751
1906	0.286348	0.000527	0.529719	1.616036	1.258154	0.002694	0.219867	1.511147
1907	0.280935	0.000506	0.5205	1.632015	1.284576	0.002625	0.224806	1.259653
1908	0.279253	0.000492	0.51003	1.63705	1.296614	0.002502	0.222197	1.132342
1909	0.274534	0.00049	0.498866	1.637072	1.315042	0.002405	0.22097	1.015023
1910	0.24623	0.001383	0.454376	1.368538	1.29838	0.002172	0.221867	1.114322
1911	0.254475	0.00145	0.468089	1.372792	1.37414	0.002145	0.236615	1.167031
1912	0.237073	0.001329	0.443984	1.369906	1.378014	0.001831	0.212715	1.02339
1913	0.250072	0.001329	0.452407	1.383902	1.373497	0.001777	0.223532	1.203178
1914	0.256532	0.001528	0.466172	1.361596	1.417533	0.001635	0.235382	1.408449
1915	0.265532	0.001514	0.460021	1.364652	1.417365	0.001584	0.235489	1.820806

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 for the effect of Medicaid

	ITS	DiD
Panel A. Men		
Medicaid in 1966	0.94	1.11
	(0.75, 1.13)	(0.70, 1.55)
Medicaid in 1967-69	1.05	1.31
	(0.77, 1.30)	(0.75, 2.06)
Medicaid 1970	1.13	1.00
	(0.77, 1.57)	(0.11, 1.85)
Panel B. Women		
Medicaid 1966	-0.63	0.37
	(-1.03, -0.19)	(-0.37, 1.07)
Medicaid 1967-69	-0.48	1.80
	(-1.03, 0.16)	(0.80, 2.93)
Medicaid 1970	-0.25	1.16
	(-1.03, 0.52)	(-0.45, 2.89)

Notes: States that implemented Medicaid in 1966 include California, Connecticut, Idaho, Illinois, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Nebraska, New Mexico, New York, North Dakota, Ohio, Oklahoma, Pennsylvania, Rhode Island, Utah, Vermont, Washington, West Virginia, and Wisconsin. States that introduced Medicaid in 1967-1969 include Colorado, District of Columbia, Georgia, Iowa, Kansas, Missouri, Montana, Nevada, New Hampshire, Oregon, South Carolina, South Dakota, Tennessee, Texas, Virginia, and Wyoming. States that introduced Medicaid in 1970 or later include Alabama, Arizona, Arkansas, Florida, Indiana, Mississippi, New Jersey, and North Carolina. Note that Hawaii and Alaska were excluded from this analysis. Delaware is not included because of the lack of sample size in the 20% training data.

Table A.3: 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.1. Interrupted Time Series Approach

A.1.1 Selection of the pre-Medicare age window for the ITS analysis.

Since the ITS approach relies on the Gompertz-Makeham (GM) assumption that posits the log mortality is a linear function of age after a certain age (a_{c0}). However, a_{c0} is unknown and may vary across cohorts and sex. We use two methods to select a_{c0}.

The simplest approach is to use ten years of pre-Medicare eligibility data ($a_{c0} = g_a - 10$). An visual examination of the Gomperz curves by sex and cohort, shown in Figures 3A and 3B, suggest the linearity assumption is plausible about 10 years prior to the Medicare eligibility age.

As a robustness check, 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). To do so, we pool data for three cohort groups by sex based on the Medicare eligibility ages. As shown in Table AX, 1901-1915 cohorts became eligible for Medicare at age 65, 1891-1900 cohorts eligible for Medicare at ages 66-75, and 1885-1890 cohorts eligible for Medicare at ages 76-81.

For each sex and cohort group, we select the best-fitting pre-Medicare trend break using five-fold cross-validation. We use five-fold cross-validation to get a measure of fit for the following two-slope linear model:

$$y_{ca} = \beta_0 + \alpha_0 a + \alpha_1 (a - a_{break}) + u_{ca}$$

Where y_{ca} is log mortality for cohort group c at age a. a_{break} denotes the potential age for a_{c0}. $\hat{\alpha}_1$ will be used for our counterfactual in the ITS analysis. Because the regression is at the cohort group-level, to implemented the cross-validation approach, we split the data at individual level using our micro data. Specifically, we sort the data and put aside the first 20% data as the test sample and the remaining 80% as training sample. We then aggregate the test and training samples to cohort groups level log mortality by age. The training sample is used to fit the model for a given break age (a_{break}) and the testing sample is used to compute the out-of-sample root mean squared error $RMSE(a_{break})$. We then repeat the procedure five times, putting aside then next 20% of the data each time. We then compute the *average* out-of-sample root mean $\overline{RMSE}$ across the five folds.

We vary a_{break} over the range of potential break ages defined in Table AX; for example, ages 45-56 for the 1901-1915 cohort group, and obtain $RMSE(a_{break})$ at potential break age. The break age (a^*_{break}) with the smallest $\overline{RMSE}$ will be considered as the identified break age, as listed in Table AX.

As shown in Figure AX, our ITS estimated Medicare effects on life expectancy at age 65 are not sensitive to the choice of the age from the two approaches. In our main analysis, we decided to use the 10-year prior to Medicare to measure the pre-trends in log mortality.

Table AX. Medicare eligibility ages and the selection window of breaks by cohort groups.

	Cohort Groups		
	1901-1915	1891-1900	1885-1890
Medicare eligibility age	65	66-75	76-81
Pre-Medicare ages	40-64	50-65	55-75
Range of potential break ages (a_{break})	45-56	55-60	60-70
Identified break ages (a^*_{break})	46 (Men) 52 (Women)	59 (Men) 56 (Women)	70 (Men) 69 (Women)
Define break age as 10-year prior to Medicare (main analysis)	55	56-65	66-71

A.1.2. ITS event study estimates ($\widehat{ATT}^{ITS}(e)$).

We first compute $\widehat{ATT}^{ITS}(g_t, g_a, a)$ for each birth cohort at each age ranging from 10 year prior to Medicare to age 90, using on equation (7) as described in V.B.2 Interrupted Time Series in the main text.

Although the ITS approach allows us to estimate $\widehat{ATT}^{ITS}(g_t, g_a, a)$ at each event age from -10 to 9, our DiD approach can only estimate $\widehat{ATT}^{ITS}(g_t, g_a, a)$ for later event age for more recent cohorts; we shall explain cohort composition for the DiD event-study estimates below. To further make ITS event study estimates ($\widehat{ATT}^{ITS}(e)$) comparable to the those from the DiD approach, we use the same cohort composition at each event age for aggregation. Specifically, for each event-study estimates $\widehat{ATT}^{ITS}(e)$, we compute the weighted average of $\widehat{ATT}^{ITS}(g_t, g_a, a)$ across included birth cohorts using the number of people survived to the Medicare eligibility age in each cohort as weights.

As shown in the table below, we include all 1886-1915 cohorts for event ages from -10 to 0. We exclude the 1885 cohort because we cannot compute $\widehat{ATT}^{DID}(g_t, g_a, a)$ due to a lack of comparison group. We include $\widehat{ATT}^{ITS}(g_t, g_a, a)$ from 1887-1915 cohorts for $\widehat{ATT}^{ITS}(e = 1)$,

and loss one cohort for each later event age, and only include $\widehat{ATT}^{ITS}(g_t, g_a, a)$ from 1895-1915 cohorts for $\widehat{ATT}^{ITS}(e = 9)$.

To obtain the 95% confidence interval of $\widehat{ATT}^{ITS}(e)$, we use a bootstrapping procedure with 1000 resamples based on our individual level data. We report the bias corrected 95% confidence interval in the main text.

Table AX+1. ITS birth cohort composition over event age.

Event Age (e)	Birth cohorts included in aggregating ATTs for event study estimates $\widehat{ATT}^{ITS}(e)$
-10 to 0	1886-1915
1	1887-1915
2	1888-1915
3	1889-1915
4	1890-1915
5	1891-1915
6	1892-1915
7	1893-1915
8	1894-1915
9	1895-1915

Notes: The cohort composition used to compute aggregated $\widehat{ATT}^{ITS}(e)$ at each event age mimic that used in the DiD approach.

A.1.3. ITS estimates on changes in intercepts and slopes of log mortality

We run the following equation separately for each birth cohort to estimates the changes in intercepts at event age 0 and changes in slopes of log mortality after Medicare.

$$y_{ca} = \beta_0 + \beta_1 EventAge_c + \beta_2 Post_c + \beta_3 Post_c * EventAge_c + \epsilon_{ca}$$

Where y_{ca} is log mortality for cohort c at age a. $EventAge_c$ is the relative age to Medicare's eligibility age for cohort c, defined as: $age - \max(65, 1966 - birthyear)$. $Post_c$ is a dummy variable equals to 1 for post-Medicare ages ($EventAge_c \geq 0$) and equals to 0 for pre-Medicare ages ($EventAge_c < 0$). Similarly, we restrict the age window from 10-year prior to Medicare eligibility age to age 90.

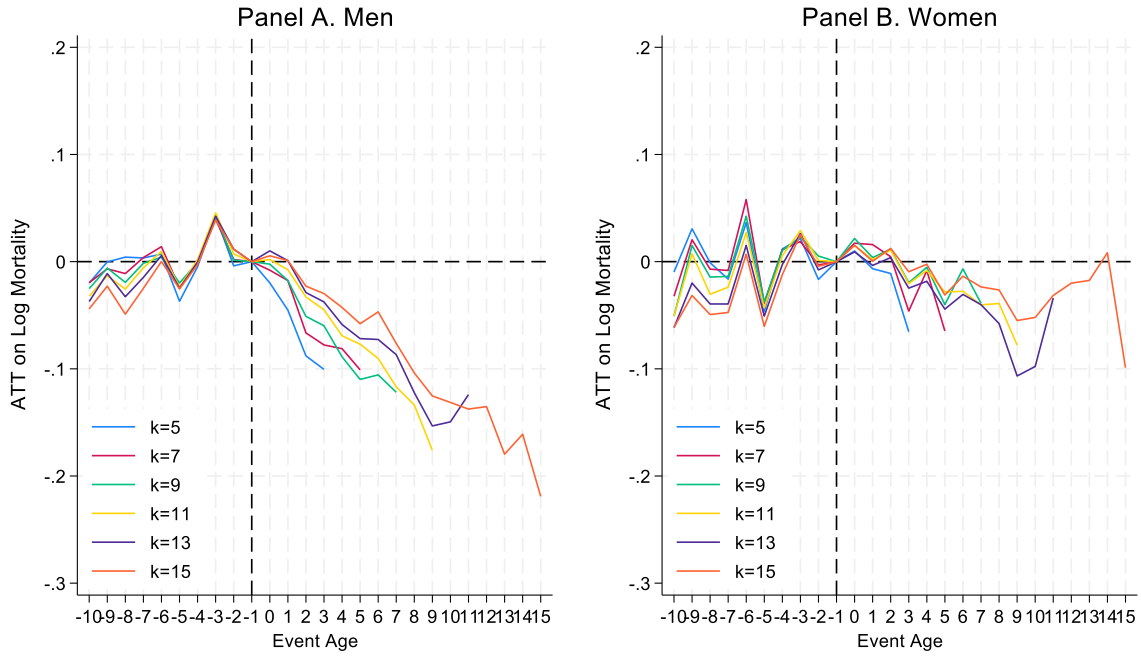
β_2 captures changes in the intercept (or level) of log mortality at the Medicare eligibility age or $EventAge_c = 0$. β_3 measures changes in slopes of log mortality after Medicare: Post-Medicare slope minus pre-Medicare slope.

Section A.2. Difference-in-Differences (DiD)

Section A2.1. Restrict the comparison cohorts in the DiD approach

As described in *V.B.3 Difference-in-Differences Design*, we restrict the set of comparison cohorts so that we only assume parallel trends for those who gain eligibility close to the Medicare eligibility age (g_a). We modified the Callaway and Sant'Anna (2021) estimator by adding the choice of k to the Stata command `csdid` (Callway, Stan'Anna, Rios-Avila, 2021), which restricts the comparison birth cohorts to those who gain eligibility close to the treatment cohorts Medicare eligibility age.

As illustrated in the following figure, a smaller k allows us to use fewer and closer cohorts as comparisons, make the pre-trends in mortality are more parallel, but yields a shorter-term estimate on log mortality. For example, when *k=4,* we can only obtain the event-study estimates up to event age 2. When *k=11*, event-study estimates are available up to event age 9. As described in the main text, we use various approaches to choose the optimal k including Rambachan and Roth (2023).



In our main analyses, we set $k = 11$, so we require the comparison cohorts received Medicare no more than 10 years than the treatment cohort. For example, to compute DiD estiamtes for the 1901 cohort that became eligible for Medicare at age 65, the comparison group includes all preceding cohorts up to 1891 cohort that gained Medicare at age 75.

In Tables AXa and AXb, we describes the exact cohort groups used as comparison for each cohort across the event age. Overall, we use different cohorts as comparison for cohorts received Medicare care at various ages (g_a) and for post-Medicare event study estimates.

Table AXa. Comparison birth cohorts used for each treatment cohort across event age.

Event Age	Treatment Cohort (Medicare eligibility age)								
	1886 (80)	1887 (79)	1888 (78)	1889 (77)	1890 (76)	1891 (75)	1892 (74)	1893 (73)	1894 (72)
(-10, 0)	1885	1885-1886	1885-1887	1885-1888	1885-1889	1885-1890	1885-1891	1885-1892	1885-1893
1	NA	1885	1885-1886	1885-1887	1885-1888	1885-1889	1885-1890	1885-1891	1885-1892
2	NA	NA	1885	1885-1886	1885-1887	1885-1888	1885-1889	1885-1890	1885-1891
3	NA	NA	NA	1885	1885-1886	1885-1887	1885-1888	1885-1889	1885-1890
4	NA	NA	NA	NA	1885	1885-1886	1885-1887	1885-1888	1885-1889
5	NA	NA	NA	NA	NA	1885	1885-1886	1885-1887	1885-1888
6	NA	NA	NA	NA	NA	NA	1885	1885-1886	1885-1887
7	NA	NA	NA	NA	NA	NA	NA	1885	1885-1886
8	NA	NA	NA	NA	NA	NA	NA	NA	1885
9	NA	NA	NA	NA	NA	NA	NA	NA	NA

Notes: NA indicates that we do not have the required comparison birth cohorts to compute DiD estimates. In parentheses are Medicare eligibility age for each birth cohort. The table cells show the birth cohorts used as comparisons.

Table AXb. Comparison birth cohorts used for each treatment cohort across event age.

Event Age	Treatment Cohort (Medicare eligibility age)						
	1895 (71)	1896 (70)	1897 (69)	1898 (68)	1899 (67)	1900 (66)	1901-1915 (65)
(-10, 0)	1885-1894	1886-1895	1887-1896	1888-1897	1889-1898	1890-1899	1891-1890
1	1885-1893	1886-1894	1887-1895	1888-1896	1889-1897	1890-1898	1891-1899
2	1885-1892	1886-1893	1887-1894	1888-1895	1889-1896	1890-1897	1891-1898
3	1885-1891	1886-1892	1887-1893	1888-1894	1889-1895	1890-1896	1891-1897
4	1885-1890	1886-1891	1887-1892	1888-1893	1889-1894	1890-1895	1891-1896
5	1885-1889	1886-1890	1887-1891	1888-1892	1889-1893	1890-1894	1891-1895
6	1885-1888	1886-1889	1887-1890	1888-1891	1889-1892	1890-1893	1891-1894
7	1885-1887	1886-1888	1887-1889	1888-1890	1889-1891	1890-1892	1891-1893
8	1885-1886	1886-1887	1887-1888	1888-1889	1889-1890	1890-1891	1891-1892
9	1885	1886	1887	1888	1889	1890	1891

Notes: In parentheses are Medicare eligibility age for each birth cohort. The table cells show the birth cohorts used as comparisons. Note that the Medicare eligibility age for 1901-1915 cohorts are (generally) at age 65, thus we use the same birth cohorts as comparisons.

Section A2.2. DiD event-study estimates ($\widehat{ATT}^{DID}(e)$)

We use the post-estimation command (`estat event`) after `csdid` to obtain the event-study estimates. However, it is worth noting that because we conduct the DiD analysis at the birth cohort level, all birth cohorts are equally weighted when we aggregate $\widehat{ATT}^{DID}(g_t, g_a, a)$ to $\widehat{ATT}^{DID}(e)$.

Alternatively, we can compute $\widehat{ATT}^{DID}(e)$ as the weighted average of $\widehat{ATT}^{DID}(g_t, g_a, a)$ using the number of people survived to the Medicare eligibility age in each cohort as weights. As shown in Tables Xa and Xb, the cohort composition is different across post-Medicare event ages. For example, we can estimate $\widehat{ATT}^{DID}(g_t, g_a, a)$ at evet age 0 for 1886-1915 cohorts, but can only obtain $\widehat{ATT}^{DID}(g_t, g_a, a)$ at event age 9 for 1895-1915 cohorts.

To compute $\widehat{ATT}^{DID}(e)$, we manually compute $\widehat{ATT}^{DID}(g_t, g_a, a)$ for each birth cohort across ages, and then calculating the weighted average at each event age (e) across included birth cohorts. Specifically, for one birth cohort such as $c = 1900$, we compute $\widehat{ATT}^{DID}(1966,66,75)$ at event age 9 ($\widehat{ATT}_c^{DID}\ e = 9$) using equation (9) in the main text:

$$\widehat{ATT}^{DiD}(1966,66,75) = \left(\bar{q}_{1900,75} - \bar{q}_{1900,65}\right) - \left(\bar{q}_{1890,75} - \bar{q}_{1890,65}\right)$$

we compute $\widehat{ATT}^{DID}(e = 9)$ as the weighted average of $\widehat{ATT}^{DID}(e = 9)$ across 1895-1915 birth cohorts. We use a bootstrapping procedure with 1000 resamples based on our individual level data to obtain the 95% confidence interval.

The results are displayed in Figure X.

Section A2.3. DiD estimates on changes in intercept and slope

In the main text *V.B.4 Estimating aggregated parameters*, we use the OLS formula for the intercept and slope, shown in equations (10) and (11), to compute changes in intercept and slope for each cohort as linear combinations of $\widehat{ATT}^{DID}(g_t, g_a, a)$ using post-estimation commands following `csdid` analysis among 1885-1915 birth cohorts. Specifically, the post-estimation allows us to compute changes in intercept ands slope for 1887-1900 cohorts that gained Medicare at various ages and have $\widehat{ATT}^{DID}(g_t, g_a, a)$ estimates across at least two ages. Since the 1901-1915 cohorts become eligible for Medicare at the same age of 65, we need to run separate `csdid` analysis with 1885-1900 cohorts (as comparisons) and one of the

1901-1915 (as treatment) at each run. The advantage of this post-estimation approach is that we leverage the variance-covariance structure from the `csdid` approach to produce both the point estimates of changes in intercept and slope, along with the corresponding standard errors. We use this method to estimate DiD estimates on changes in intercept and slope in Figure 8.

However, an important limitation of the post-estimation approach is that the high computation cost because we have to run the `csdid` regression 16 times: once for the 1885-1900 cohorts, and once for each of the 1901-1915 cohorts. As a result, it requires a long runtime (~10 mins) to compute the cohort-specific changes in intercept and slope for the 1885-1915 cohorts. We need these estimates to impute the implied effect of Medicare on life expectancy at age 65 (ex65). When we bootstrap the ex65 effect, we have to compute these cohort specific estimates on intercept and slope for each of our 1000 resamples. It thus requires substantial computation resources and extended run time. However, we only need to point estimates on changes in intercept and slope.

To improve the computational efficiency, we use an alternative approach to compute changes in intercept and slope for each cohort. For each cohort, we first compute the post-Medicare $\widehat{ATT}^{DID}(g_t, g_a, a)$ based on equation (9) in the main text using comparison cohorts listed in appendix Table Xa and Xb. We then run a regression of $\widehat{ATT}^{DID}(g_t, g_a, a)$ on event age restricting to the post-Medicare period (event age $a - g_a \geq 0$) for each birth cohort:

$$\widehat{ATT}^{DID}(g_t, g_a, a) = \alpha + \beta(a - g_a) + \epsilon$$

Where $(a - g_a)$ denotes the event age, $\hat{\alpha}$ captures the change in intercept and $\hat{\beta}$ captures the change in slope.

Section A2.4. DiD estimation with a difference PT assumption

Describe based on Bacon's write up on the DiD approach based on the PT(c,t) assumption.

A.3. ITS and DiD implied effect on life expectancy at age 65 (ex65).

We use cohort specific changes in intercept and slope of log mortality to compute the implied effect on life expectancy at age 65 separately for each cohort. Using the 1905 men cohort as an example, we describe the approach we use to compute the implied effect on life

expectancy at age 65 based on changes in slopes and levels of Gompertz curves after Medicare eligibility age.

The Gomperz curve for the cohort is illustrated in Figure A. It is consistent with the Gompertz Law of Mortality that log mortality is linear in age. 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 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 (solid 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 ages 35-100, we compute the life expectancy at age 65 (**ex65_nopolicy**).

Second, to compute the life expectancy with Medicare (**ex65_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. Alternatively, we can use the estimated changes in intercept and slope to construct the post-Medicare log mortality based on the pre-trend. 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 (solid grey line) and the predicted log mortality after Medicare (solid 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 65 (**ex65_policy**).

The implied effect of Medicare on life expectancy at age 65 is calculated as: $ex65_policy - ex65_nopolicy$. We then use a bootstrapping approach with 1,000 resamples of our microdata to obtain the standard error and 95% confidence intervals.

Figure A. Gompertz curve of 1905 white male cohort

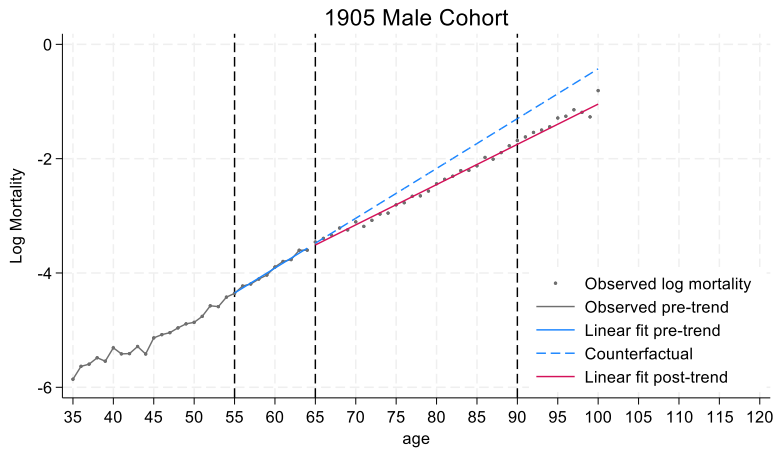
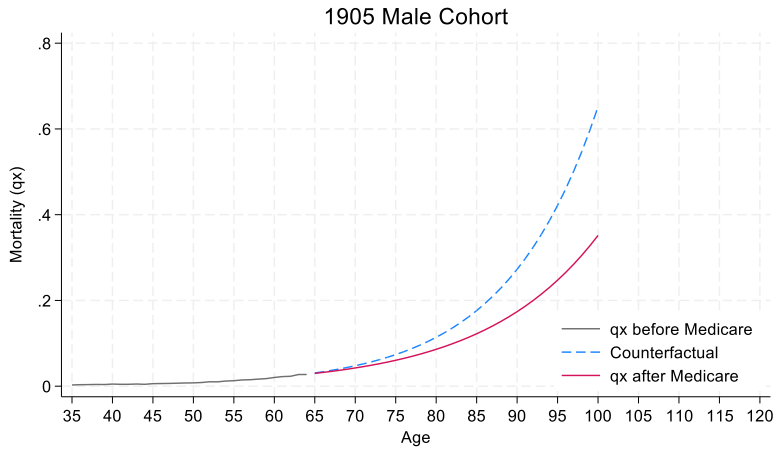


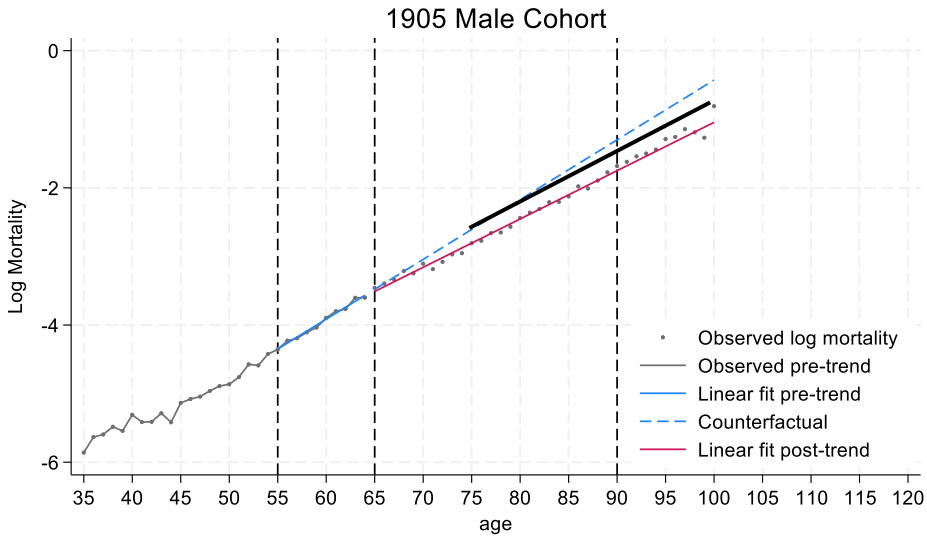
Figure B. Mortality (qx) with and without Medicare



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.

There are two important issues worth noting. First, prior work has shown that the Gompertz curve does not fit the mortality data well at older ages, e.g., after age 90. Second, the Medicare effect may become stagnant after certain ages. Our structural estimation shows that the Medicare effect becomes stable 10 years after Medicare coverage. We assess the robustness of our estimates on these two issues.

(Discuss how)



Section A.5. Details on the structural estimation

We estimate the structural model using the same methods as Lleras-Muney and Moreau (2022). The parameters are estimated by Simulated Method of Moments (SMM) in MATLAB. The method proceeds as follows. We take an initial guess about the parameters and simulate the resulting population data, mortality rates and associated survival curves. We then compute the difference between the actual and the predicted survival rate for each age. We iterate over the parameter space until we find the parameters that minimize the sum of squared errors in the survival rate The criterion function is the unweighted sum of squared errors:

$$Q = \sum_{t=0}^{T} \left(S_t^d - S_t^m\right)^2$$

where S_t^d is the population surviving to age t in the data, and S_t^m is the population surviving to age t in the model. S_t^i are expressed relative to an initial population of 100.

Because the process is sensitive to the choice of initial parameters, we follow the literature and iterate over the process by employing different optimization routines. We first use MATLAB's *fminsearch* routine, a downhill simplex method. Once the process converges, we use the results as starting values and re-estimate the parameters using Powell (1964)'s conjugate direction method. Once Powell's method converges we use the results as starting values for *fminsearch* again. Because of the high level of sensitivity to the initial parameters, we also manually searched over the parameter space using our familiarity with how

parameter guesses impact the shape of the survival curve to find a reasonable starting point for the optimization routine.

Section A.6. Behavioral responses to Medicare.

Section A.7. Spillovers

Section V defined potential outcomes only in terms of a cohort's own Medicare treatment variables, which implicitly ruled out spillover effects from *other* cohorts' Medicare coverage. In fact, because Medicare was so large its introduction did have widespread effects on providers (Finkelstein 2007) and medical device patenting (Clemens and Olsen 2021). To acknowledge these SUTVA violations, we write person i's potential age at death as a function of her own Medicare treatment variables and a variable s that represents generic spillovers:

$$Y_i(g_t, g_a, s)$$

We also modify the no anticipation assumption to account for the fact that any spillovers begin in calendar year 1966:

Assumption NA-s. No anticipation with spillovers

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

$$Y_i(g_t, g_a, s) = Y_i(\infty, 0) \;\; if \;\; c + Y_i(g_t, g_a) < 1966$$

$$Y_i(g_t, g_a, s) = Y_i(\infty, s) \;\; if \; c + Y_i(g_t, g_a, s) \geq 1966 \;\; and \;\; Y_i(g_t, g_a) < g_a$$

Which is equivalent to:

$$q_{c,a}(g_t, g_a, s) = q_{c,a}(\infty, 0) \quad if \quad c + a < 1966$$

$$q_{c,a}(g_t, g_a, s) = q_{c,a}(\infty, s) \quad if \quad 1966 \leq c + a < g_t \;\; .$$

This assumption states that prior to 1966 we observe untreated potential mortality rates without spillovers—$q_{c,a}(\infty, 0)$—and between 1966 and g_t we observe untreated potential mortality rates with spillovers— $q_{c,a}(\infty, s)$. Treated potential mortality rates necessarily involve spillovers—$q_{c,a}(g_t, g_a, s)$—because the fact that some people have Medicare is what generates the spillovers.

i. Causal Parameters

This notation defines several new average causal parameters. The average treatment effect on the treated target parameter from section V now also includes spillovers:

$$ATT(g_t, g_a, s, a) = q_{c,a}(g_t, g_a, s) - q_{c,a}(\infty, 0)$$

This parameter compares mortality rates with direct coverage *and* spillovers to counterfactual mortality rates with no Medicare coverage whatsoever; not for the treated cohort nor for any other cohorts. Since potential outcomes are a non-separable model this quantity does not distinguish between effects that arise purely because of own coverage, purely because of spillovers, or because the two interact.

We also define a direct effect of Medicare coverage as follows:

$$ATT^d(g_t, g_a, s, a) = q_{c,a}(g_t, g_a, 0) - q_{c,a}(\infty, 0)$$

This parameter compares mortality rates with direct coverage but no spillovers, which would occur if only one cohort were on Medicare, to untreated potentially mortality rates.[38]

Finally, we define average spillover effects on treated (AST) and untreated (ASU) cohorts:

$$AST(g_t, g_a, s, a) = q_{c,a}(g_t, g_a, s) - q_{c,a}(g_t, g_a, 0)$$
$$ASU(g_t, g_a, s, a) = q_{c,a}(\infty, s) - q_{c,a}(\infty, 0)$$

Both parameters measure average causal effects of spillovers, s, but differ as to whether the focal cohort is itself covered by Medicare. The non-separability of $Y_i(g_t, g_a, s)$ means that $AST(g_t, g_a, s, a)$ does not necessarily equal $ASU(g_t, g_a, s, a)$. Moreover, these definitions imply that:

$$ATT(g_t, g_a, s, a) = ATT^d(g_t, g_a, s, a) + AST(g_t, g_a, s, a)$$

The average treatment effect on the treated of Medicare eligibility is the sum of direct effects and spillover effects on the treated.

ii. Identification and estimation

With spillovers, the identification strategies we use in the main text all identify $ATT(g_t, g_a, s, a)$ under their respective assumptions—that is, our parameters include spillover effects. There is no way to use cross-cohort variation alone to separately identify their direct and spillover components. We can, however, apply the same type of assumptions to younger cohorts observed after Medicare began but prior to gaining Medicare themselves.

This requires no substantive change to the ITS analysis. We make the same Gompertz-Makeham assumption ($q_{c,a}(\infty, 0) = \alpha_c + \beta_c(a - a_{c0})$) but estimate α_c and β_c using data from before 1966 instead of before g_a. Spillover estimates using this approach simply equal $\bar{q}_{c,t-c} - \hat{\alpha}_c + \hat{\beta}_c(t - c - a_{c0})$) and can be estimated for periods $1966 \leq t < G_t(c)$.

[38] We abstract here from within cohort spillovers. "Direct" refers to a cohort's treatment, not individual's treatment.

Estimating spillovers using DiD requires a similar modification to the parallel trends assumption and estimator:

Assumption PT-s. Parallel Trends across cohorts and ages for spillovers

For $a < 65$ and $t < G_t(c)$, the change in untreated log mortality rates for cohort c between ages $a = t - c$ and $1965 - c$, equals the change in log untreated mortality rates between the same ages for older cohorts, j. These cohorts must be old enough to reach age $t - c$ prior to Medicare ($j + t - c \leq 1965$), and we only make this assumption for cohorts born up to k years before c ($c - j \leq k$). Therefore, given c and t, comparison cohorts are $j \in \{c - k, 1965 - (t - c)\}$:

$$q_{c,t-c}(\infty, 0) - q_{c,1965-c}(\infty, 0) = q_{j,t-c}(\infty, 0) - q_{j,1965-c}(\infty, 0)\,.$$

These deaths occur in periods t and 1965 for cohort c, and periods $j + t - c$ *and* $j + 1965 - c$. The restriction on the gap between cohorts ensures that the comparison group is only observed prior to Medicare and thus experiences no direct or indirect treatment.

Now consider a simple DiD comparison between cohorts c and j between 1965 and a post-Medicare period $t \geq 1966$:

$$\begin{aligned}
&\left(q_{c,t-c} - q_{c,1965-c}\right) - \left(q_{j,t-c} - q_{j,1965-c}\right) \\
&= \left(q_{c,t-c}(\infty, s) - q_{c,1965-c}(\infty, 0)\right) - \left(q_{j,t-c}(\infty, 0) - q_{j,1965-c}(\infty, 0)\right) \\
&= \left[q_{c,t-c}(\infty, s) - q_{c,t-c}(\infty, 0)\right] + \\
&\quad \left(q_{c,t-c}(\infty, s) - q_{c,1965-c}(\infty, 0)\right) - \left(q_{j,t-c}(\infty, 0) - q_{j,1965-c}(\infty, 0)\right) \\
&= ASU(G_t(c), G_a(c), s, t - c)
\end{aligned}$$

The second line substitutes potential for observed outcomes, the third line adds and subtracts counterfactual mortality for cohort c, $q_{c,t-c}(\infty, 0)$, and the fourth line uses Assumption PT-s and the definition of the average spillover effect on the untreated, $ASU(g_t, g_a, s, a)$.

Appendix Figure AX1 reports estimates based on this kind of DiD approach. We observe the 1902-1915 cohorts treated by spillovers but not directly covered by Medicare. They spend progressively longer periods of time exposed to *others'* Medicare coverage. The 1902 cohort potentially experiences spillovers for just one year—when they are 64 years old in 1966—while the 1915 cohort experiences 14 years of spillovers—from 1966 to 1979 when they are 51 to 64 years old. Comparison cohorts are born between

We find no evidence of spillover effects on younger people using either approach. The point estimates are small and not statistically significant for the younger and for the older subsets of these spillover cohorts.

There are two reasons why our analysis should capture the timing of Medicare's likely spillovers. First, Finkelstein (2007) finds immediate and large changes in hospital capacity, aggregate utilization, and entry. We find no health effects among the cohorts who without Medicare who may have gone to these new and larger hospitals. Moreover, even if Medicare's most important spillovers took time to materialize, our estimates for the 1908-1915 cohorts cover up to seven years after Medicare, a reasonable time frame over which to expect spillover mortality effects.

It is also possible that Medicare's spillovers were small for people without Medicare coverage. To benefit from additional provider capacity or technology investments, one must be able to afford to consume hospital care. In other words, spillover effects operating through health system capacity benefit insured people ($AST(g_t, g_a, s, a) \neq 0$), but not insured people ($ASU(g_t, g_a, s, a) = 0$). In fact, panel B of Figure 1 shows that no change in the time-series of hospital utilization for people under 64. Cross-cohort variation cannot speak to this argument.

A final interpretation of the results in Figure AX1 relates to the plausibility of our PT assumption. The estimates equal zero either because Assumption PT-s holds and $ASU(g_t, g_a, s, a) = 0$, or because violations of Assumption PT-s offset $ASU(g_t, g_a, s, a)$. We view the second interpretation as less plausible than the first since it implies a close alignment in magnitudes between a causal parameter and a bias term that arises only after 1966 and holds across as many as 7 years and in two cohort sub-groups. Under this interpretation Figure AX1 not only shows that spillovers on uncovered people are small, but also that these cohorts did not experience sharp changes in their mortality rates or Gompertz curves after 1966. To the extent that their experience reflects the path of untreated log mortality for the cohorts who did gain Medicare in 1966, this result supports our main PT assumption by ruling out calendar year effects on $q_{c,a}(\infty)$.[39]

[39] If $ASU(g_t, g_a, s, a) = 0$, then the path of observed mortality rates around 1966 for not-yet-eligible cohorts provides an additional way to measure changes in *untreated* log mortality. This can be used to motivate a distinct DiD design that compares mortality changes in calendar time for cohorts who gain Medicare to the same calendar time changes for *younger* comparison cohorts who have not yet aged into the program. The implication of Figure AX1 is that this design, based on contemporaneous comparisons across ages rather than same-age comparisons across time periods, also identifies $ATT(g_t, g_a, s, a)$. In results available upon request we show that these two DiD approaches (younger comparisons and older comparisons) yield nearly identical results within about 6 years of Medicare.