The Long-Term Impact of Medicare's Introduction on Adult Mortality[*]

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Abstract

This paper estimates the shorter- and longer-term causal effects of the introduction of Medicare on the mortality rates and life expectancy of the program's early recipients, which remains largely unknown. We construct a new dataset of more than 18 million individuals observed in the 1940 census who can be linked to a death record in the FamilyTree database at FamilySearch. These data accurately describe the mortality profiles of the affected cohorts, as they closely match the cohort life tables recently produced by the Social Security Administration and include information on important personal characteristics that predict health and mortality. We develop three novel empirical strategies to estimate the long-term effects of Medicare. We find that Medicare increases life expectancy for men at age 55 by 0.74 years on average: the effect gradually increases from zero to roughly 2 years for the birth cohorts born 1885-1910 and then decreases and becomes negative for the 1915 birth cohort. We also find Medicare's effect on reduced mortality is larger for those with low socioeconomic status (SES). The results for women, however, are somewhat less robust, highlighting important sex differences that are poorly understood in the literature. We also find important differences in the effects of the program by SES.

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I. Introduction

Today, almost all countries in the world provide some form of health insurance. A primary aim, alongside financial protection, is to maintain population health by providing access to basic health care. Because the incidence and severity of medical problems increase with age, older people account for the largest portion of health insurance costs and may enjoy its largest benefits. Yet, estimating the causal effects of health insurance on the health of elderly recipients has proved difficult. One reason is the lack of variation that can identify these effects in countries with universal coverage, but even in the United States, where universal health insurance under Medicare begins at age 65, studies of health insurance typically can only find effects for policies that cover high-need groups and in settings with longer-run outcomes.

For instance, despite large increases in insurance coverage and health care use when Americans age into Medicare, neither Card et al. (2004) nor Polsky et al. (2009) find sharp declines in mortality at age 65 in the overall population. But among people admitted to hospitals for a subset of conditions that need urgent care, Medicare lowers one-year mortality rates (Card et al., 2009). Huh and Reif (2017) find that Medicare's prescription drug coverage lowers cardiovascular mortality rates among people age 65 and older by increasing drug spending among near-poor people. Historical studies of Medicare using population mortality data come to mixed conclusions. Finkelstein and McKnight (2008) find that the program's introduction in 1966 did not significantly reduce short-run mortality rates in regions with higher pre-Medicare uninsurance rates, while Chay, Kim, and Swaminathan (2010) find large reductions in elderly versus near-elderly mortality rates.[2]

None of these studies observes longer-term mortality rates, however, where Medicare's effects may be largest. Theoretical models of health (Dalgaard and Strulik, 2014; Grossman, 1972; Lleras-Muney and Moreau, 2022) as well as empirical studies (Leinonen et al., 2001; Levinsky and Schiff, 2021) document that health is a stock that is built slowly over time and that also deteriorates slowly. Therefore, the benefits of insurance, which provides access to preventive

[2] These findings echo patterns in the much larger empirical literature on public health insurance for non-elderly people. For instance, Medicaid, the public insurance program for the poor, reduces mortality rates for children and adults, but only when it covers those with very low incomes or high health needs (Borgschulte and Vogler, 2020; Currie and Gruber, 1996a, b; Goodman-Bacon, 2018, 2021; Graves et al., 2020; Miller et al., 2021; Wherry and Meyer, 2016). This pattern holds in three randomized evaluations as well, which find null effects on average adult mortality rates but reductions for lower-income, older, or less healthy people (Baicker et al., 2013; Finkelstein et al., 2012; Goldin et al., 2021; Newhouse et al., 1993). Moreover, except in cases where health insurance funds acute interventions that immediately save lives (Duggan and Evans, 2008; Goodman-Bacon, 2018), all of these effects take time to manifest.

care, chronic care management and diagnostic tools, may not materialize for many years, especially among relatively healthy people. Indeed, recent evidence tracking the impact of health insurance on children suggests that there are large long-term impacts of health insurance provision.[3] Whether this evidence applies to adults who are no longer growing is unknown. Moreover, these models show that while insurance can theoretically affect health across the population, it is only likely to change mortality for sub-populations near the death threshold.

This paper estimates the shorter- and longer-term causal effects of the introduction of Medicare on the mortality rates and life expectancy of the program's early recipients. Medicare, enacted in 1965, began providing health insurance for hospital stays and physician visits to (almost) all Americans over the age of 65 in July 1966. To evaluate this historic change, we construct a new dataset of more than 18 million individuals observed in the 1940 census who can be linked to a death record. These data closely match the Social Security Administration's cohort life tables, but they also include covariates that allow us to investigate heterogeneity in ways that are not possible using existing data sources. These data allow us to map causal effects on period/age mortality rates into a *long-run* outcome: cohort life-expectancy conditional on surviving to age 55.

We pre-specify three research designs that identify Medicare's average treatment effect on the treated under distinct identifying assumptions about how mortality would have evolved without Medicare. First, based on a theoretical model that implies a log-linear relationship between mortality and age, we estimate changes in the slope and intercept of the age profile of log mortality for each cohort after they gain Medicare. This interrupted time series (ITS) approach identifies Medicare's average treatment effect on the treated (ATT) by using the observed trend in log mortality at younger ages to estimate counterfactual mortality at older ages.

Second, we employ a difference-in-differences (DiD) design that compares mortality rates for cohorts before and after they gain Medicare to the change in mortality at the same ages for older comparison cohorts who did not have Medicare coverage. This DiD approach identifies

[3] Goodman-Bacon (2018, 2021) documents that the introduction of Medicaid in 1966 resulted in immediate reductions in US infant and child mortality followed by reductions in mortality and disability up to 50 years later. Other work also finds evidence that Medicaid expansions during childhood led to better self-reported health at later ages, fewer chronic diseases and related hospitalizations, and lower mortality (Brown et al., 2020; Currie et al., 2008; Miller and Wherry, 2019; Thompson, 2017; Wherry and Meyer, 2016; Wherry et al., 2018).

the same ATT parameter by using mortality changes for other cohorts to estimate counterfactual mortality trends (ie. a parallel trends assumption).[4]

Third, we estimate both the ITS and DiD designs by region to study how estimated ATT parameters vary with pre-Medicare private health insurance coverage rates, similar to Finkelstein and McKnight (2008). Under an additional assumption about the comparability of ATTs across regions, we can interpret this heterogeneity as the causal effect of incremental changes in population-level insurance coverage (Callaway et al., 2024). Relatedly, we generate predicted health insurance coverage at the individual level by combining covariates from our analysis dataset with a predictive model of health insurance coverage using pre-Medicare survey data. This generates a much larger amount of pre-existing variation in insurance rates than previously used.

To empirically ground our design choices and still conduct valid statistical inference, we use a random 20 percent of our sample to test identifying assumptions and make key specification choices. For example, our detailed microdata allows us to assess how Medicare's effects vary by SES, but we do not know a priori how best to measure SES in order to capture relevant heterogeneity and be able to detect it. Our training sample allows us to test different ways to measure SES in the 1940 Census without inducing pre-test bias. Similarly, we can condition our analysis on a range of 1940 covariates (as well as look for other types of heterogeneous effects), and we use our training sample to select covariates and choose the best conditional estimator (Sant'Anna and Zhao, 2020). We will publicly archive our full analysis plan (and code) and the evidence from our training sample that justifies it with the Open Science Framework before conducting our main analysis.[5] Results in this manuscript use training data only.

Our preliminary results show that Medicare increases life expectancy for men at age 55 by 0.74 years on average: the effect gradually increases from zero to roughly 2 years for the birth cohorts born 1885-1910 and then decreases and becomes negative for the 1915 birth cohort. We discuss the reasons for these declines. As predicted by theory and consistent with the empirical evidence for children, these increases in lifespan are driven by small impacts on mortality in the short term that become larger over time. The results for women, however, are

[4] [ONE SENTENCE ON DID METHODOLOGICAL CONTRIBUTION HERE]

[5] Prior to creating our analysis sample, we did estimate preliminary versions of our ITS design and regression versions of the DiD design. We committed to preregistration prior to refining these strategies and all subsequent choices about our analytical approach were chosen using only the training sample.

close to null and somewhat less robust, highlighting important sex differences that are poorly understood in the literature.

(ADD THE DID RESULTS AND THE HETERGENEOUS RESULTS BY SES)

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

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

II. Background

For decades prior to Medicare's passage as part of the Social Security Act amendments of 1965, poor health had been an increasingly burdensome problem for American seniors. Private health insurance, initially developed in the 1920s, exploded after World War II. The war gave millions of soldiers new experience with regular medical care, new military technologies found health care applications such as ultrasounds (Van Tiggelen and Pouders, 2003), the federal government massively subsidized hospital capacity (Chung et al., 2017), and labor negotiations during the war meant that employer-sponsored insurance offered major tax advantages (Thomasson, 2003). These developments were good for population health, but created an intractable affordability problem for seniors.

People over age 65 generally did not get insurance through employers or on the individual market. Individual insurance markets offered medically underwritten policies, making them prohibitively expensive to older people with high expected costs. Blue Cross Blue Shield tried to offer affordable plans through "community rating," but these plans suffered from adverse selection problems that pushed up premiums (Thomasson, 2004). Scattered survey data from the early 1960s suggest that little more than half of people over age 65 held any kind of health insurance policy. The share was 40 percent in one 1960 survey (Morgan et al., 1962), 55 percent in the 1963 Survey of Health Services Utilization and Expenditure (CHAS and NORC 1984), and between 43 and 68 percent (for hospital insurance) across regions in the 1963 National Health Survey (Finkelstein, 2007). Among Social Security beneficiaries in 1966, private insurance covered less than one-quarter of their medical costs; they paid the rest entirely out of pocket (Loewenstein 1971).

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 (Finkelstein and McKnight, 2008). A small number of low-income elderly people had their care financed by welfare departments through the Old Age Assistance program or a short-lived program called Medical Assistance for the Aged, which mainly operated in a handful of large states. These programs ultimately had a claim on the resources of a recipient's children and so many people were reluctant to accept welfare medical care. Charity providers often provided low quality care, had long wait times, and could refuse service or pursue aggressive collection practices (Stevens, 1989). Therefore, in the decades leading up to Medicare's passage medical care was improving and growing in cost, but the primary mechanisms by which prime-age Americans paid for that care were out of reach for older people. In response, many simply went without care and accepted the health consequences.

[MOVE CHRONOLOGICALLY TO JOHNSON ADMIN AND STRUCTURE]

In response to these conditions, the Johnson administration included Medicare as the signature component of the 1965 Social Security Act amendments. Medicare had two insurance components modelled after the Blue Cross Blue Shield plans that provided most insurance coverage to seniors. Part A provided compulsory hospital insurance for people over 65 with a $40 annual deductible and no cost sharing for stays under 60 days (Gornick et al., 1996). It is

funded by payroll taxes Individuals ages 65 and over are automatically enrolled in Part A. Part B offered optional medical and surgical insurance with 20 percent coinsurance for a $3 per month (Gornick et al., 1996). Part B operates like a standard insurance contract: individuals can choose to enroll in it and pay a premium to have services covered. Although individuals pay premiums, these were designed to only cover 50% of the cost of the program, and the rest is partly funded by general revenues.[6]

The program officially began on July 1, 1966. About 93% of individuals eligible for hospital insurance also enrolled in Part B (West, 1971). Medicare's introduction dramatically increased insurance coverage for the elderly; the percent of seniors with hospital insurance and surgical insurance dramatically increased from 50% in 1963 to 95% in 1968 (National Center for Health Statistics, 1963, 1968), though Finkelstein (2007) estimates an even larger 75 percentage point increase in "meaningful health insurance". It also significantly lowered the cost of health care for seniors. Surveys indicate that patients were paying about 47% of medical costs whereas the prior number was around 77% (West, 1971).

Surveys taken around the introduction of Medicare indicate that utilization increased immediately after its passage. For example, short stay hospital care for people 65 and older increased by 25% with greater increases among those aged 75 and over, Blacks, residents of the South, urban residents, and persons in one-member family units with low incomes (West, 1971). Previous research has shown that the introduction of Medicare was also associated with an increase in health care utilization and spending (Finkelstein, 2007), and with a substantial reduction in medical expenditure risk (Finkelstein and McKnight, 2008). Today this remains true: Individuals on Medicare use health care at much higher rates than those without (Card et al., 2008) (Card et al., 2008) and they have lower medica expenditure risk (Barcellos and Jacobson, 2015).

III. Data Sources and Data Quality

[6] Today the program has 2 other parts. 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, which was introduced in 1999. Finally in 2006 Medicare implemented a new voluntary insurance product (Part D) to cover prescription drugs expenditures.

III.A. Data Sources

Aggregate cohort tables. We use multiple sources of data to assess the impact of Medicare's introduction. The first source comes from the Social Security Administration (SSA), which recently developed *cohort* life tables for the nation, and which are freely available.[7] These tables track the mortality and life expectancy of men and women in the nation for cohorts born in 1900 and thereafter. These data are only available at the aggregate 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. Cohort tables by definition are based only on actual events for extinct cohorts, so they are accurate for those born before 1930, and based on partial projections for cohorts born thereafter. We focus attention on cohorts born 1900 to 1915.

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

NIHS data. We use the restricted 1963 National Health Interview Survey (NIHS). The NIHS is a nationally representative survey of non-institutionalized households conducted to obtain

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

information about the health of the population. The NHIS also collected information on basic socioeconomic characteristics of individuals. Crucially, respondents were asked about whether the individual or the household head had health insurance coverage. Also, importantly for our study the restricted access data contains state of residence identifiers. We use these data to estimate hospital insurance rates by state and socioeconomic groups and to estimate the propensity of an individual to have insurance based on their observable individual and household level characteristics.

III.B Sample Construction and Sample Selection

We linked the 1940 full-count census which includes 132 million people to the Family Tree and 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 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. However, ages at death are only observed for a subset, for whom the information is available. Since racial/ethnic minorities are not well represented in our family tree data (Price et al., 2021), we further restrict our main analysis to whites.

The final data set includes 18,485,813 white individuals, accounting for 32.2% of the 1940 population in the white population born 1885-1915 that we targeted.[8] 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.

III.C 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. Our sample appears to be representative of the target population (whites) in the 1940 census in terms of year of birth, ethnicity, veteran status, and educational levels, as shown in **Table 1**.[9] However, it includes fewer females (46.3% in Census Tree versus 49.8% in the

[8] 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.

[9] Note, 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

1940 Census), more married individuals (84.2% versus 72.3%), fewer people born in the Northeast region (16.9% versus 24.3%), and fewer people who resided in Northeast in 1940 (17.8% versus, 30.5%). In fact, no existing linking methods produce samples that are consistently representative of the linkable population (Bailey et al., 2020).

However, we can re-weight the sample to make it representative and address potential selection bias (Bailey et al., 2020; Lleras-Muney et al., 2022; Yue et al., 2023a). Specifically, we estimate the probability, $p(X_i)$, of a successful match among the same population (e.g., Whites) between our sample and the 1940 full-count Census using a robit model as a function of the covariates available in 1940 census (Newson and Falcaro, 2023; Seaman and White, 2013).[10] We then reweight matched observations by $p(X_i)^{-1}$ (rescaled to sum to one within the matched sample) so their covariate distributions match the 1940 Census. As shown in **Table 1**, the weighted statistics are fairly comparable to those from the target population in the 1940 full-count census.

To assess the quality of death information in our data we compare the unweighted age-specific mortality rates in our data with those in the SSA cohort life tables. Since SSA life tables are for all races and only available for birth cohorts 1900 and onwards, we conduct the comparison for birth cohorts 1900-1915 from all races (although we do not use all races in our analysis) by sex. As shown in **Figure 1**, 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. This is likely occurring because our data include no racial minorities and fewer immigrants, groups who tend to have a higher mortality rate than whites. As a result, the expected age at death conditional on survival to age 40 computed using our data is larger than that in SSA life tables (**Figure A2**): For males, the gap is about 1.8 years for the 1900-1904

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.

[10] These are: sex (male, female), years of schooling (continuous), age (continuous), marital status (married and spouse present, married and spouse absent, separated, divorced, widowed, never married), number of children (continuous), labor force participation (yes, no), occupation (categorical), occupation income scores (continuous), wage and salary income (continuous), veteran status (yes, no, unknown), place of birth, and state of residence in 1940. We use a robit model to estimate the inverse probability weights (IPW) because it is more suitable to handle missing-at-random values without producing extremely large weights (Newson and Falcaro, 2023; Seaman and White, 2013). In addition, we further truncated IPW at the 99th percentile to stabilize the weights (Seaman and White, 2013).

cohorts and 1.3 years for the 1905-1915 cohorts. For females, the gap is about 2 years for the 1900-1904 cohorts and 1.5 for the 1905-1915 cohorts.

IV. Potential Effects of Medicare and Target Causal Parameters

Upon signing Medicare into law, President Johnson declared "no longer will older Americans be denied the healing miracle of modern medicine." There are many reasons to expect that this promise may have been fulfilled and that it led to lower mortality rates for people over age 65. This section discusses theoretical frameworks that describe Medicare's potential mortality effects in detail as well as empirical research on Medicare and mortality. We also define causal parameters that are our ultimate objects of interest.

IV.A. Theoretical Effects of Medicare

We start by simulating the potential effects of Medicare on lifetime mortality using the model developed by Lleras-Muney and Moreau (2022). In this model, individuals are endowed with an initial level of health *H,* which reflects parental health and genes, as well as *in utero* conditions, and is normally distributed in the population. Thereafter, health evolves at each age a as follows: $H_a = H_{a-1} - \delta a^{\alpha} + I + \varepsilon_a$. It can increase (decrease) if individuals receive positive (negative) health investments *I*; it deteriorates with age at an increasing rate (given by the aging function δa^{α}); and finally it receives stochastic shocks ε_a, which are also assumed to follow a normal distribution N(0, σ). Individuals die when their health falls below a certain threshold which is normalized to zero. 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).

Conceptually, Medicare may affect individuals in several different ways, each of which predicts different changes in the position and slope of their Gompertz curves. First, Medicare may affect the level of annual resources (I) that individuals have access to, starting at age 65. In principle Medicare is an insurance product that only shifts income across time periods. In practice however the program pays for (some) of its cost out of payroll taxes from individuals that are currently working, so the initial generations that we study essentially received a transfer. That transfer may increase health investments and thus health.

A second related reason is that Medicare lowers the variance of health resources individuals have by insuring them against the financial burden of health shocks (in addition to contributing through payroll taxes throughout life). Thus, Medicare may lower the variance of health shocks (σ) to the extent that they derive from income shocks. If Medicare increases the use of preventive care or leads to more timely diagnoses, it may have the same effect in a more direct way.

Third, Medicare may slow the rate of aging (δ) because it provides individuals with both continuous treatment and prevention. Finally, by raising the use or quality of acute medical care, Medicare may change the "threshold for dying" (0). This means that it would simply help keep people who are in bad health alive, potentially raising subsequent mortality rates by altering the population frailty.

The simulated effects of these changes on the log of the mortality rates of the population are shown in **Figure 2a**. Resource increases (labeled "investment increase" in panel a), as well as decreases in depreciation rates (panel d), do not result in immediate declines in mortality rates; rather, they result in a downward rotation of the curves, resulting in increasingly lower mortality rates over time as individuals age. Thus, these changes make the slope of the Gompertz curve flatter. By contrast, declines in the variance of health resources (panel b) do lower mortality immediately, and then they result in steeper slopes, similar to the effects of changing the threshold for dying. But over time a smaller variance also means higher mortality rates because while negative shocks are less negative, positive shocks as also less positive.

Finally note that changing the threshold (panel e) lowers mortality immediately at age 65 and thereafter, but the declines in logs are the largest at 65 and they become smaller as individuals age, similar to what a decline in accident rates would do (shown in panel c for completeness though it is not likely that Medicare would lower accident rates since these are exogenously given in the model).

The resulting "impulse responses" or time-varying effects that would be observed in the ideal experiment that compares the mortality of an affected and unaffected population are shown in **Figure 2b**. Figure 2b plots the evolution of the log gaps one would observe in an event study in each case scenario, under the assumption that the ideal counterfactual is available.[11] What

[11] We show the log gaps, but Appendix Figure X shows the results for gaps in levels.

these results suggest is that to estimate the long term effects of Medicare, we need to estimate what occurs to the intercept and the slope of the log mortality curve at age 65.

These findings guide our first approach to identification. Our objective will be to understand how Medicare affects the intercept and the slope of mortality after age 65. The results will identify long term effects and can be used to compute effects on life expectancy. They will also partially identify which mechanism is at play, that is why/how Medicare affects health and mortality.

IV.B. Empirical Evidence on Medicare and Mortality

Despite clear evidence of increases in health care use and financial security, prior studies on the introduction of Medicare have found mixed effects on the mortality rates of the elderly. Finkelstein and McKnight (2008) show that the decline in the mortality rates of 65-74 year olds began several years before Medicare's introduction, while that for the 55-64 the decline began slightly after. Thus they concluded that Medicare had no discernible impact on the elderly aggregate mortality rates in first ten years, even among the Black population. In contrast, in a working paper Chay et al. (2010) use finer age grouping and find sharp convergence between elderly and near-elderly mortality immediately after Medicare's introduction.

Studies using contemporary data also find ambiguous impacts on mortality. These studies identify the effects of Medicare by looking for discontinuities at age 65. Neither Polsky et al. (2009) nor Card et al. (2004) find declines in mortality at age 65 for the overall population, despite increases in utilization.[12] These findings mask substantial heterogeneity, however. Card et al. (2009) find a significant drop in 7-day mortality for severely ill patients admitted through emergency departments. Eligibility expansions of Medicare to people with disabilities (Andersen, 2018) and inclusion of prescription drug coverage (which had its largest effects of drug utilization for near poor people) significantly reduced mortality in the short run (Andersen, 2018; Dunn and Shapiro, 2019; Huh and Reif, 2017).

The current evidence has several limitations.

First and foremost, these studies only investigate short term impacts on mortality. As noted above theoretically it is not clear that Medicare would necessarily impact mortality rates

[12] Lichtenberg (2002) finds a significant break in mortality at age 65, but Card et al. (2009) argue that it is an artifact of the interval-smoothing procedures used to construct the data.

immediately: increased health resources or lower depreciation rates would only manifest in lower mortality rates over time. Some empirical evidence also suggests we should expect delayed impacts. Many Medicare-covered services are known to save lives (Yusuf et al., 1994) but these effects might not materialize within a short period. For example the MRFIT randomized intervention, which aimed at lowering deaths from cardiovascular disease by lowering cholesterol, lowering blood pressure, and quitting smoking had immediate effects on health and on earnings (Stephens Jr and Toohey, 2022), but mortality declines were not observed in the first seven years, and were only significant at the 10 and 15 year mark (Multiple Risk Factor Intervention Trial Research Group, 1982, 1990, 1996). Health insurance has also been shown to improve health behaviors and mental health in the short run (Baicker et al., 2013). These improvements could improve longevity, but they likely do so with a delay. Medicare improves access to many diagnostic tools and preventive services which could also benefit individuals with a delay. For example, colonoscopies are associated with lower incidence of colorectal cancer and lower mortality within ten years but not within two years (Niikura et al., 2017). Thus, it is possible that the long-term effects of Medicare will be larger than the short-term estimates suggest.

Second, historical studies so far have only used aggregate mortality data and cannot estimate the kinds of heterogeneous effects documented in recent studies.

The effects of Medicare likely vary by SES. For example, SES measures are strong predictors of having private health insurance (Card et al., 2008) and better health prior to reaching age 65 (Braveman et al., 2010). Therefore, those with lower SES may have larger health improvements from gaining Medicare (Card et al., (2004) Table 10). Even with equal access to health care, however, low-SES people still visit a specialist at lower rates (Braveman et al., 2010; Dunlop et al., 2000) and benefit less from medical advances because they appear to adopt these at lower rates (Braveman et al., 2010; Dunlop et al., 2000; Glied and Lleras-Muney, 2008). Thus, in the long run, high SES individuals might benefit more or less from increased access to health insurance through their better use of the health care system.

Medicare may also have very different effects across space. Differences in pre-Medicare insurance coverage are a natural reason (Finkelstein and McKnight, 2008), but aside from its coverage effects, the provider landscape to which it granted access varied widely across the country, especially in the 1960s. Even today large differences in Medicare spending across localities are not fully explained by differences in the underlying health of the population

(Finkelstein et al., 2016). Moreover, low-income seniors gained access to Medicaid coverage around the same time, but these benefits varied in their extent and timing across states (because Medicaid is a state-level program unlike Medicare).

Finally Medicare may have had different effects over time. The first cohorts to be eligible – the 1901 cohort which turned 65 in 1966 and older cohorts – did not make any contributions to the Medicare funds throughout their lifetimes but received benefits. But younger cohorts did. As a result, these cohorts may have had lower health prior to becoming eligible for Medicare. A second reason these effects could vary is that older cohorts accessed Medicare at older ages potentially resulting in greater or lower impacts.

The Medicare program itself evolved overtime providing yet another reason why its effects could vary across cohorts. In 1973, Medicare was offered to disabled individuals and to individuals with and those with end-stage renal disease, expanding the number of non-elderly individuals among beneficiaries (De Lew, 2000). In the 1970s and 1980s the services that were included were expanded to include more preventive services and hospice services. Most notably, in 1997, Medicare allowed managed care companies to provide services, and in 2006, prescription drug benefits were added. As a result of these changes, the benefits of the program likely evolved.

Finally, as the population became aware of the program there might have responded to its availability, even before turning 65. 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. Thus, behavioral responses on the part of consumers and suppliers may also result in different health effects – since delayed care is detrimental to health this anticipatory behavior could lead to lower benefits among more recent cohorts.

In this study we will investigate heterogeneity across cohorts, locations and SES status.

IV.C Defining Causal Effects of Medicare on Mortality

The objects of interest in this paper are causal parameters that describe Medicare's effect on mortality rates for different cohorts, at different ages/times, and for different sub-groups.

Person i belongs to a birth cohort $C_i = c \in (1885,1915)$. We observe deaths in periods $t = 1940, \ldots, 2020$, but we only keep observations for which people are ages $a \in (40, 90)$. Ages, periods, and cohorts are related by the identity: $a = t - c$.

For our purposes, Medicare policy has two dimensions, its starting year, $t^* = 1966$, and its minimum age of eligibility, $a^* = 65$. Units from cohort c become treated by Medicare at time $G_t(c) = max\{c + a^*, t^*\} = max\{c + 65,1966\}$ which occurs when they are age $G_a(c) = G_t(c) - c = max\{a^*, t^* - c\} = max\{65,1966 - c\}$. This structure reflects the fact that the only people first treated at ages older than 65 necessarily got Medicare in 1966, and the only people first treated after 1966 were necessarily treated at age 65.[13]

The values g_t and g_a define potential outcomes (Rubin 1974, Robins 1986): $Y_{it}(g_t, g_a)$. We use $Y_{it}(\infty)$ to denote potential outcomes in the absence of Medicare. We initially assume that Medicare does not affect mortality prior to cohorts gaining eligibility (either through individual anticipation or spillover effects) so that $Y_{it}(g_t, g_a) = Y_{it}(\infty)$ whenever $t < G_t(C_i)$ or, equivalently $a < G_a(C_i)$. We relax this assumption below.

The basic building block causal parameter in this paper is the log average treatment effect on the treated in period t for units who gained Medicare eligibility in period g_t and age g_a:

$$\begin{aligned} ATT(g_t, g_a, t) = \log(E[Y_{it}(g_t, g_a) | G_t(C_i) = g_t, G_a(C_i) = g_a]) \\ - \log(E[Y_{it}(\infty) | G_t(C_i) = g_t, G_a(C_i) = g_a]) \end{aligned}$$

Because the policy variables g_t and g_a fully determine a unit's a cohort, this notation implicitly defines ATT parameters for all cohorts in all periods.[14] Note that because our main outcome is mortality, only the surviving members of cohort c are at risk of death in period t. Expectations implicitly condition on survival.

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[13] If $G_t(c) > 1966$ then $c + 65 > 1966$ which implies that $65 > 1966 - c$ and thus $G_a(c) = 65$. Similarly, if $G_a(c) > 65$ then $65 < 1966 - c$ which implies that $1966 > c + 65$ and thus $G_t(c) = 1966$.

[14] $G_t(c)$ and $G_a(c)$ pin down a unit's cohort $g_c(g_t, g_a) = \begin{cases} t^* - g_a & if\ g_t = t^* \\ g_t - a^* & if\ g_t > t^* \end{cases}$. Different sets of $ATT(g_t, g_a, t)$s correspond to the three notions of time: age, period, and cohort. A collection of $ATT(g_t, g_a, t)$s for many periods traces out the time-varying (or age-varying) effects of Medicare on the single cohort treated at time g_t and at age g_a. A collection of $ATT(g_t, g_a, t)$s in period t for a cohorts/ages treated at different ages g_a traces out average treatment effects at a point in time across cohort/age distribution. For example, Card et al (2004) plot survey-based health outcomes by age for a given calendar year. Finally, a collection of $ATT(g_t, g_a, t)$s at age a for a range of cohorts/periods treated at different times g_t traces out average treatment effects of Medicare on age-a mortality. Figure 2, for instance, in Finkelstein and McKnight (2008) plots the time series of mortality for the "young elderly" (65-74) and the "near elderly" (55-64).

Our link to the 1940 Census also allows us to target conditional ATT parameters for subgroups by SES (education or income) and geography, denoted by X_i: $CATT(g_t, g_a, t; X_i)$. Finally, ATT (or CATT) parameters on period/age mortality for a given cohort can be used to calculate average treatment effects on life expectancy starting at age 55. This quantity equals the difference between observed life expectancy conditional on living to age 55 minus counterfactual life expectancy defined by subtracting the estimated ATT parameter from period mortality rates; see Appendix section 1 for details. These target parameters answer the main causal questions in the paper: what were Medicare's (heterogeneous) effects on longevity?

Identification of these parameters requires a strategy to estimate counterfactual log mortality rates that would have been observed had Medicare not been enacted: $\log(E[Y_{it}(\infty)|G_t(C_i) = g_t, G_a(C_i) = g_a])$. We construct these counterfactuals in three ways: using each cohort's pre-Medicare mortality trends, using other cohorts' observed mortality trends, and using mortality trends in geographic areas affected differently by Medicare. We present both the methods and results from each of these approaches in the following three sections.

V. Interrupted Time Series Analysis of Gompertz Curves

Our first identification strategy relies on that log mortality rate without Medicare are linear in age after a certain point (a_{c0}):

Assumption 1. Linearity of untreated log mortality rates in age

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

$$\log(E[Y_{it}(\infty)|G_t(c) = g_t, G_a(c) = g_a]) = \alpha_c + \beta_c(a - a_{c0})$$

This is a standard Gompertz-Makeham model is also implied by the model in section IV.A (Lleras-Muney and Moreau 2022) where α_c denotes the age-independent level of mortality and β_c denotes the impact of age on mortality. Log-linearity fits observed mortality rates very well across a wide range of time periods and populations: **Figures A3a and A3b** plot these curves for our data and show that linearity appears to be a reasonable restriction.

Under our no anticipation assumption, α_c and β_c can be estimated from separate regressions by cohort of log mortality on a constant and age using data from before the age of Medicare

eligibility, $g_a(c)$. This leads to simple estimates of counterfactual mortality rates at post-Medicare ages: $\hat{\alpha}_c + \hat{\beta}_c(a - a_{c0})$. In practice, we also impose a linear model for post-Medicare log mortality rates and estimate the change in the slope and intercept of log mortality for each cohort (and by sex) after age $g_a(c)$ using a simple trend-break regression specification:

$$\log(E[Y_{it}|G_t(C_i) = g_t, G_a(C_i) = g_a]) = \alpha_c + \beta_c(a - a_{c0}) + \delta_c 1\{a \geq g_a\} + \theta_c 1\{a \geq g_a\}(a - g_a) + \varepsilon_{ca}$$

In this framework, $ATT(g_t, g_a, c + a) \approx \delta_c + \theta_c(a - g_a)$. (The approximation comes from modeling post-Medicare log mortality as a linear function.) Moreover, **Figure 2** not only suggests that the shift and trend-break parameters δ_c and θ_c fit the theoretical mortality profiles well, they also provide information about *how* Medicare affected mortality.

This interrupted time-series (ITS) approach is simple to implement, but requires use to choose a range of ages, a_{c0} to a_{max}, over which we believe log mortality satisfies Assumption 1. There are controversies over whether there is a late-life mortality deceleration after age 80. We include data up to age $a_{max} = 90$, similar to Chetty et al. (2016) and Fletcher et al. (2023). The log of mortality at younger ages, however, is non-linear, which can have a large impact on the estimated trend before age 65 (β_c) and generate bias in our estimates.[15]

To address this non-linearity problem, we implement two approaches. The first is a simple arbitrary rule: we use ten years of pre-Medicare eligibility data for all cohorts to estimate the age profile of mortality before Medicare kicks in. This approach is simple and treats all cohorts equally. However, it does not make the best use of the data since we potentially ignore useful information that can be used to better estimate the pre-treatment slopes.

Alternatively, 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). We use mortality data prior to the Medicare eligibility age search for the optimal break age by varying b in the following specification:

[15] 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 perfectly for males with an R^2 over 0.98 for all included birth cohorts. The model fit for females is perfect 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.

$$\log(E[Y_{it}|G_t(c) = g_t, G_a(c) = g_a]) = \mu_c + \rho_c(a - 40) + \lambda_c 1\{a \geq b\}(a - b) + \varepsilon_{ca}$$

We conduct the analysis for three cohort groups: 1901-1915 cohorts, 1891-1900 cohorts, and 1891-1900 cohorts, which got Medicare at age 65, ages 66-75, and age 76-81, respectively. Within each cohort group, we estimate this model for values of b between $max(40, 1940 - c) + 5$ to $max(65,1966 - c) - 6$, which ensures we have at least five data points to measure trends before or after the break age. For example, we search for the age breaks at age 45-59 using mortality data from age 40 to 64 for birth cohorts born 1901-1915, who gained Medicare at age 65. For each iteration, we evaluate the model's fit using five-fold cross-validation methods, using our Census Tree individual-level micro data, to compute the mean squared error (MSE). The b with the smallest MSE defines the lower bound, a_{c0}, of our ITS estimation sample for cohort c.

To summarize the impact of Medicare on total life expectancy, we compute the implied life expectancy at age 55 using the estimated age-specific period mortality with Medicare and then again using the projected mortality rates that would have occurred without Medicare (setting δ_c and θ_c to zero).[16] The difference between the two gives us an estimate the implied effect of Medicare on life expectancy. We then use a bootstrapping approach with 1,000 resamples to obtain standard errors and 95% confidence intervals.

We estimate the impact of Medicare for each cohort separately – thus, we can assess if the effects of Medicare are constant over time. We also compute the overall effect for the 1885-1915 cohorts as the weighted average of cohort-specific effect using the cohort-specific population size at the Medicare eligibility age (essentially the population in each cohort in 1966) as weights. We conduct this estimation with the SSA cohort tables and with our individual-level data to further assess the quality of our Census Tree data. Then using our data, we conduct the analysis by educational level, household income, and state.

V.A. Findings from the Interrupted Times Series of Gompertz Curves

Results for Men. We find no statistically significant changes in the intercept (there is a small increase, but it is not statistically significant) but statistically significant decreases in the slope of log mortality for the 1885-1905 cohorts (Panel A, **Figure 4**). Through the lens of the model,

[16]We use the standard life table method to compute life expectancy (Preston et al., 2000). We assume that mortality rates continue to be linear after age 90 to close the life table.

this suggests that, for the oldest cohorts of men, Medicare is equivalent to increasing annual investments in health or lowering depreciation/aging rates.

Starting around 1905 we observe a change in the pattern: the intercept becomes negative and statistically significant. The change in the slope on the other hand goes from negative to positive. Through the lens of the model, for these more recent cohorts Medicare is equivalent to lowering the variance of resources or the threshold for dying.

Table 2 shows the estimated changes in the slopes and the intercepts if we pool cohorts into 5 year age-groups. If we pull all the cohorts together, we estimate a decline in the slope (-0.015) and the intercept (-0.023) of the age profile of the log of mortality. But the results differ by cohort. For the earliest cohorts (1885-1900) the intercept increases (0.022) when cohorts become eligibility, and the slope decreases (-0.023) every year after Medicare eligibility. The change in intercept is small and insignificant for the 1901-1905 cohorts but the slope change remains negative (-0.018). For the 1906-1910 cohorts the change in the intercept and the slope are both negative. Finally for the 1911-1915 cohorts the intercept change is large negative (-0.064) and the slope change is positive and small (0.004).

It is not clear why the results change across cohorts. One possibility is that the effects for the most recent cohorts are underestimated due to changes in Medicare. As noted earlier beginning in 1973, Medicare was expanded to individuals under age 65 with disabilities or end-stage renal disease. Individuals from the two high-risk groups born after 1907 would become eligible for Medicare before age 65, which could flatten our estimation of pre-trends in log mortality and then lead to underestimated effect on e55 from our interrupted time series analyses. Another possibility is that the effects of Medicare changed overtime as discussed earlier. We revisit these conclusions later in the paper.

Based on the changes in intercepts and slopes of Gompertz curves, we calculate that for men, Medicare increased e55 by 0.74 (95% CI: 0.62 to 0.86) on average (**Table 3**): the effect gradually increases from zero to roughly 2 years for the birth cohorts born 1885-1910 and then decreases and becomes negative for the 1915 birth cohort (Panel A, **Figure 5**). For context, life expectancy conditional on survival to age 55 rose from about 33 years to 34.2 years in the census tree data (**Figure A2**) – our estimates imply that Medicare accounts for 50-66% of this increase.

The pattern of results is very similar if we re-estimate the model using the national data from SSA. Using the optimal age window for pre-Medicare linear trends of log mortality, we also find similar results except that the implied effects on e55 are around 1.5 years for cohorts 1911-1915 (**Figure A5**). Thus the results for the most recent cohorts of men – those born after 1910 are sensitive to the choice of widow used for estimation. But the effects are positive and increasing for the older birth cohorts. Thus, the results for men suggest that Medicare had large impacts on life expectancy, but these effects varied in magnitude and nature across cohorts.

Results for women. These results are much less stable, robust and imprecise for women (Panel B, **Figure 4**). We find that after the Medicare eligibility age, there are decreases in slopes of log mortality for the oldest cohorts (1885-1894) but increases for all other cohorts (1895-1915). Results on changes in levels of log mortality for women follow similar patterns as those for men but they are not precisely estimated: the most consistent pattern we estimate is that the intercept appears to fall and the slope appears to rise in the for the 1905-1915 cohorts. However the figure shows that these estimates are different in the SSA data. The results are similarly unstable if we use the optimal estimation widow. As **Figure 5** shows, the implied Medicare' effects on e55 are not distinguishable from zero for most cohorts, and they are negative for many cohorts.

Heterogeneity. For men **Table 3** shows that, using the restricted age window, the Medicare's effect on e55 is ~0.9 among those with less than high school degree, which is larger than that for those that graduated from high school (~0.6). Similarly, they are greater for men in households with below median incomes in 1940. The results are consistent with prior research showing that a-these groups were less likely to already have health insurance and thus were more affected by the introduction of Medicare, b- these groups are poorer and likely in worse health to begin with. However, these differences by education or income are not statistically significant. We also consider geographic variation in these effects. Figure 9 shows the results. For men the effects vary significantly across states with larger benefits in the East and smaller effects in the West. For women most estimates are negative.

In sum. The ITS approach suggests that Medicare resulted in substantial benefits for men, but not women, though these varied across cohorts, SES and space. The greatest limitation of these results is that we rely on the per-Medicare slopes to construct a counterfactual. We now re-estimate these using the alternative event study estimation approach.

VI. Event-Study with Staggered Adoption of Treatment

VI.a. Empirical Approach and identification assumptions

An alternative approach to estimating a given cohort's counterfactual mortality rates is to use information on mortality from adjacent cohorts who were not on Medicare at a given age. This motivates a difference-in-differences (DiD) design in which older cohorts observed in the years before Medicare began provide the counterfactual evolution of mortality.

Consider identification of the ATT for the 1901 cohorts in their first year of Medicare eligibility, which was 1966 when they were age 65:

$$ATT(1966,65,1966) = E[Y_{i1966}(1966,65) - Y_{i1966}(\infty)|G_t(c) = 1966, G_a(65) = 65]$$

The unobserved counterfactual is $E[Y_{i1966}(\infty)|G_t(c) = 1966, G_a(65) = 65]$, the untreated mortality rate in 1966 (or, equivalently at age 65) for the 1901 cohort. Because Medicare was universal, we do not observe untreated mortality for any 65-year-olds in 1966. We do observe untreated mortality at age 65 for older cohorts, who gained Medicare at later ages. These mortality rates necessarily come from years before 1966, though, and so are not a valid counterfactual if mortality rates are trending over time. If, however, the change in mortality between ages 64 and 65 for older cohorts equals the change in untreated mortality rates for the 1901 cohort—a parallel trends assumption—then $E[Y_{i1966}(\infty)|G_t(c) = 1966, G_a(65) = 65]$ is identified.

Our identifying assumption imposes parallel trends between pairs of cohorts and pairs of ages (Dinas and Stoker, 2014):

$$E\left[Y_{ic+a}(\infty) - Y_{ic+g_a-1}(\infty)\middle|G_t(C_i) = g_t, G_a(C_i) = g_a\right] = E[Y_{ic+a}(\infty) - Y_{ic+g_a-1}(\infty)|G_t(C_i) = a + k]$$

The parallel trends assumption means that the change in mean untreated potential outcomes between $g_a - 1$ and a for cohorts treated at the time g_t at age g_a is the same as the (observed) change in mean untreated potential outcomes for older cohorts treated at age $a + k$. Under the assumption, $ATT(g_t, g_a, t)$ is identified by

$$ATT(g_t, g_a, t) = \left(E\left[Y_{it} - Y_{ig-1}\middle|G_t(C_i) = g_t, G_a(C_i) = g_a\right]\right) - \left(E\left[Y_{it} - Y_{ig-1}\middle|G_t(C_i) = t + k\right]\right)$$

We apply the Callaway and Sant'Ana (2021) approach to estimate $ATT(g_t, g_a, t)$ and compute DiD estimates by defining cohorts as the cross-sectional unit, age as the time variable, and

$G_a(C_i)$ as the treatment variable. We assess the parallel trends assumption based on the event-study graph and conduct several analyses to further assess and strengthen the assumption. Again, using our data, we can conduct the analysis by cohort, educational level, household income, and state to investigate if the effects of Medicare vary. To compute the effect of Medicare on life expectancy we project the estimated effects into old ages.

Because all individuals over the age of 65 became eligible in 1966 we can construct many such counterfactuals allowing us to fully trace out the impact of Medicare over many years. This approach is only valid if all the cohorts share the same age profiles (identical slopes though not intercepts). For example, $\widehat{ATT}(1901, 65, 1966)$ is depicted in **Figure 3**. In this identification strategy, the 1900 birth cohort provides the counterfactual line for the 1901 cohort, for ages 64-65. The 1900 cohort at age 64-65 was not treated in 1966 when Medicare was introduced. But the 1901 was treated in 1966 at age 65. The identification assumption here is that the line for the 1900 cohort is an appropriate counterfactual for the 1901 line.

This identification assumption seems plausible when using the 1900 cohort, but less so for cohorts born much earlier. For example, to obtain Medicare's effect on mortality 15 years (event time 15) after its introduction, this approach compares the log mortality trends over age 64-80 among cohorts 1901-1915 who gained Medicare at age 65 to the mortality trend over the same age period for the 1885 cohort who gained Medicare at age 81.

The main limitation of this identification strategy then is that to estimate long term effects it must use older and older cohorts as counterfactuals. We address this limitation in our next approach by leveraging within-cohort variation in health insurance coverage before Medicare.

VI.b. Event-Study Estimates

Our event-study estimates based on $ATT(g_t, g_a, t)$ are presented in **Figure** 7 pooling the results across all cohorts.

For males, we do not find an immediate effect the first year after Medicare, but the ATT effect of Medicare on mortality grows over time (**Panel A Figure 7**).[17] For example, the ATT estimate is -0.1 at event time 9, which suggests that Medicare is associated with 0.1 decrease in log

[17] Note that the dynamics of the event-study estimates also reflect changes in the composition of birth cohorts. For example, we identify Medicare's effect at event time 0 for the 1886-1915 cohorts but can only identify the effect at event time 15 for the 1901-1915. We address this issue later.

mortality (or about 10% reduction in mortality rate) nine years after being eligible for Medicare. These results are consistent with a model in which Medicare's effect on mortality for males is operating through increased health investment and lowered depreciation/aging rates.

The event study assumptions appear to be met. The ATT estimates for unaffected ages are close to zero but with a slight increase pre-trend, which would increase our estimated ATTs (Dobkin et al., 2018).

These event study estimates for men are in fact entirely consistent with the pooled ITS estimates shown in column 1 of Table 3. These suggest a small decline in the slope of -0.023 which is within the confidence interval of the first-year effect of Medicare in the event study. The ITS estimates a decline in the log of the mortality rate of 0.015 per year after Medicare eligibility, which suggests a decline in log mortality of 0.15 after 10 years which is also exactly what we observe in the event study. Thus the results for men are very similar in magnitude and nature across both estimation strategies if we pool all cohorts together.

The event study results for women (Panel B, Figure 7) are smaller and not statistically distinguishable from zero (except that the ATT estimate at the event time 15). Thus both the ITS approach and the event-study approach fail to find benefits for women.

V.B.2. Investigating Differential Pre-Trends and Heterogeneous Effects

Our results in **Figure 8a** show that for males with 0-7 years of schooling, Medicare has a large effect over time. The estimates for males with 8-11 years are similar. The estimates are smaller for males with 12+ years of schooling similar to what we found in the ITS approach. But the standard errors are large and we cannot reject the estimates are the same. The results by income (Figure 8b) also show increasing benefits of Medicare over time, but not statistically significant differences by income levels. As was the case in the ITS analysis, we also observe significant heterogeneity in space (Figure 9)

For females, there are no systematic and statistically significant declines in mortality in any of the groups we look at, except perhaps for the lowest income group.

[Further analysis to be added here – compute e55 effects and compare ITS and ES estimates. Correlate with other state characteristics]

VII. Leveraging Variation in Private Health Insurance before Medicare's Introduction

We leverage geographic variation in the incidence of private health insurance in 1963 following the work of Finkelstein (2007) and Finkelstein and McKnight (2008). We start by using the data from Finkelstein and McKnight (2008), namely the fraction of individuals with private health insurance in eleven sub-regions. To improve on these estimates, we use the restricted 1963 National Health Interview Survey which provides state of residence identifiers to estimate private health insurance (e.g., hospital insurance rate) rates at the state level. We then assess if the impact of Medicare is larger in areas with larger uninsured populations. We start by assessing this by relating the estimated impacts of Medicare at the region level in the previous two sections with the insurance rates.

We then estimate individual level regressions that estimate the impact on life expectancy directly leveraging this geographic variation and controlling for additional individual covariates. This design leverages differences in the exposure to Medicare in the spirit of the recent analyses of the introduction of Medicaid (Goodman-Bacon, 2021). Specifically, we estimate the following model:

$$y_{ijs} = \alpha + \sum_{k=1877}^{k=1915} \beta_k \, PrivateInsuranceRate_s \mathbf{I}(j = k) + X_{ijs}\eta + \gamma_j + \varepsilon_{ijs}$$

where y_{ijs} denotes the longevity of individual i in birth cohort j from state s. $PrivateInsuranceRate_s$ denotes the state (subregion) level private health insurance rate. The omitted category is $k = 1876$. $\mathbf{I}(j = k)$ is an indicator for each birth cohort. β_k captures the Difference-in-Differences estimates of Medicare's effect on longevity by comparing birth cohorts that gained Medicare coverage at different ages (first difference) in areas with higher or lower private insurance coverage rates for older adults before Medicare's introduction (second difference). The second difference helps account for secular trends in longevity across birth cohorts. We will also consider robust estimates for DiD analyses with a continuous treatment (Callaway et al., 2024).

Alternatively, we use individual-level differences in the probability of having private health insurance in 1963 as a source of identification, similar to Hausman (2016)'s analyses of the 1936 veterans' bonus (Hausman, 2016). First, we use the information on the 1940 census to predict whether individuals have private health insurance by combining SES information in the

1940 census with models estimated from the 1963 National Health Interview Survey or Survey of Health Services Utilization and Expenditure. We then check to see whether these predictions match the geographic variation in Finkelstein and McKnight's data. This allows us to use the individuals' propensity to have private health insurance for identification (replacing $PrivateInsuranceRate_s$ with *individual-level* predicted probabilities of having private health insurance). Alternatively, we estimate effects for subgroups that were more or less likely to benefit from Medicare, such as the Amish, coal workers, veterans, and those who lived in Mississippi Delta that lack of Medicare-certified (racially desegregated) hospitals in the late 1960s (Figinski and Troland, 2020; Finkelstein and McKnight, 2008; Rohrer and Dundes, 2016).

[Analysis to be completed]

V. Conclusions

In contrast to prior studies, using novel data and new identification approaches, we find that the introduction of Medicare resulted in large reductions in mortality and significant life expectancy gains among men but not women. However, these gains for men do not appear to materialize immediately but instead emerge overtime. It is not clear why we estimate no benefits for women.

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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)
	Statistics w/o IPW	Statistics w/ IPW	Percent of 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: we linked our Census-Tree data to the 1940 full-count census to construct the inverse probability of matching weights (IPW). We restricted the analyses to whites.

Table 2: Pooled estimates of the effects of Medicare on the log of mortality using interrupted time series methods

	Cohorts 1885-1915	Cohorts 1885-1900	Cohorts 1901-1905	Cohorts 1906-1910	Cohorts 1911-1915
Panel A. Men					
EventAge	0.086***	0.090***	0.087***	0.087***	0.073***
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)
Post	-0.023***	0.022***	0.004	-0.097***	-0.064***
	(0.007)	(0.007)	(0.014)	(0.014)	(0.016)
Post*EventAge	-0.015***	-0.023***	-0.018***	-0.014***	0.004*
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)
Cohort FE	Yes	Yes	Yes	Yes	Yes
Cohort FE*EventAge	Yes	Yes	Yes	Yes	Yes
Panel B. Women					
EventAge	0.085***	0.086***	0.078***	0.082***	0.078***
	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)
Post	-0.030***	0.021**	0.020	-0.099***	-0.110***
	(0.009)	(0.009)	(0.020)	(0.019)	(0.021)
Post#c.EventAge	0.008***	-0.001	0.011***	0.010***	0.019***
	(0.001)	(0.001)	(0.003)	(0.003)	(0.003)
Cohort FE	Yes	Yes	Yes	Yes	Yes
Cohort FE*EventAge	Yes	Yes	Yes	Yes	Yes

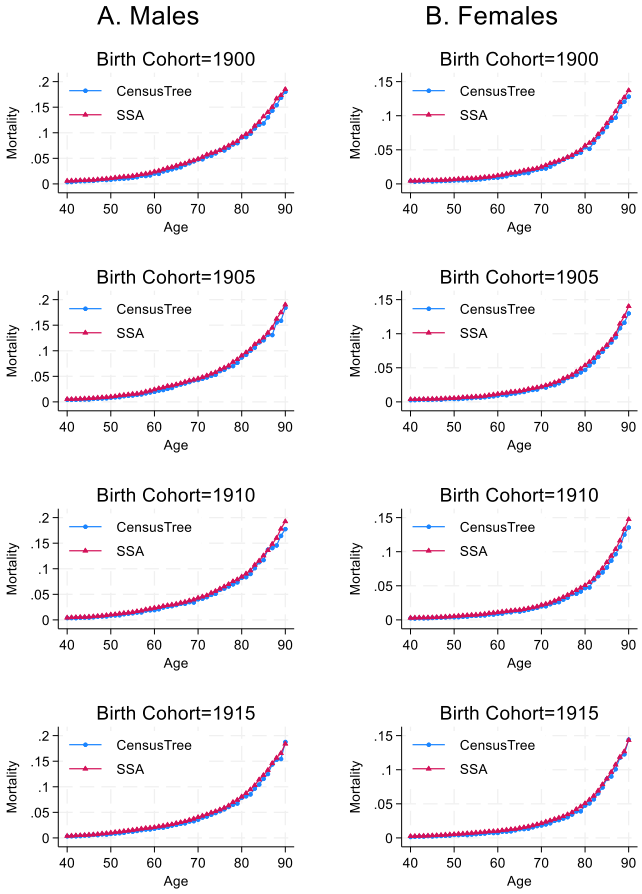
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. We 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: Effects of Medicare on Life Expectancy at Age 55 by SES

	ITS		DiD	
	ATT on life expectancy at age 55 (s.e.)	95% Confidence Interval	ATT on log mortality (s.e.)	95% Confidence Interval
Panel A. Males				
All men	0.741		-0.078	(-0.100, -0.057)
	(0.063)	(0.619, 0.862)	(0.011)	
Education				
0-7 years of schooling	0.876		-0.100	(-0.133, -0.067)
	(0.098)	(0.699, 1.084)	(0.017)	
8-11 years of schooling	0.714		-0.065	(-0.093, -0.037)
	(0.082)	(0.557, 0.871)	(0.014)	
12+ years of schooling	0.575		-0.052	(-0.091, -0.014)
	(0.147)	(0.289, 0.856)	(0.019)	
Household total income in 1940				
Less than or equal to Median	0.778		-0.074	(-0.106, -0.043)
	(0.087)	(0.620, 0.957)	(0.016)	
Above the Median	0.683		-0.080	(-0.105, -0.054)
	(0.085)	(0.526, 0.874)	(0.013)	
Panel B. Females				
All women	-0.658		-0.018	(-0.051, 0.015)
	(0.140)	(-0.932, -0.374)	(0.017)	
Education				
0-7 years of schooling	-0.023		-0.068	(-0.131, -0.005)
	(0.244)	(-0.466, 0.500)	(0.032)	
8-11 years of schooling	-0.540		0.005	(-0.031, 0.040)
	(0.194)	(-0.912, -0.138)	(0.018)	
12+ years of schooling	-1.326		-0.028	(-0.077, 0.021)
	(0.276)	(-1.911, -0.807)	(0.025)	
Household total income in 1940				
Less than or equal to Median	-0.423		-0.039	(-0.076, -0.003)
	(0.188)	(-0.767, -0.002)	(0.018)	
Above the Median	-0.866		0.005	(-0.038, 0.047)
	(0.202)	(-1.247, -0.469)	(0.022)	

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 compute the implied effects on life expectancy at age 55 based on the estimated changes in slopes and levels of log mortality from the interrupted time series (ITS) approach. The average effect on life expectancy at age 55 (ex55) is a weighted average of implied effect on ex55 across 1885-1915 birth cohorts using the number of people when there were eligible for Medicare in each cohort as weights. We use a bootstrapping approach with 1,000 resamples to obtain the standard error and 95% confidence intervals. For the DiD estimates, we use Callaway and Sant'Anna (2023) for the average treatment effect on the treated (ATT) estimation and aggregation; the post-Medicare aggregated ATT is reported.

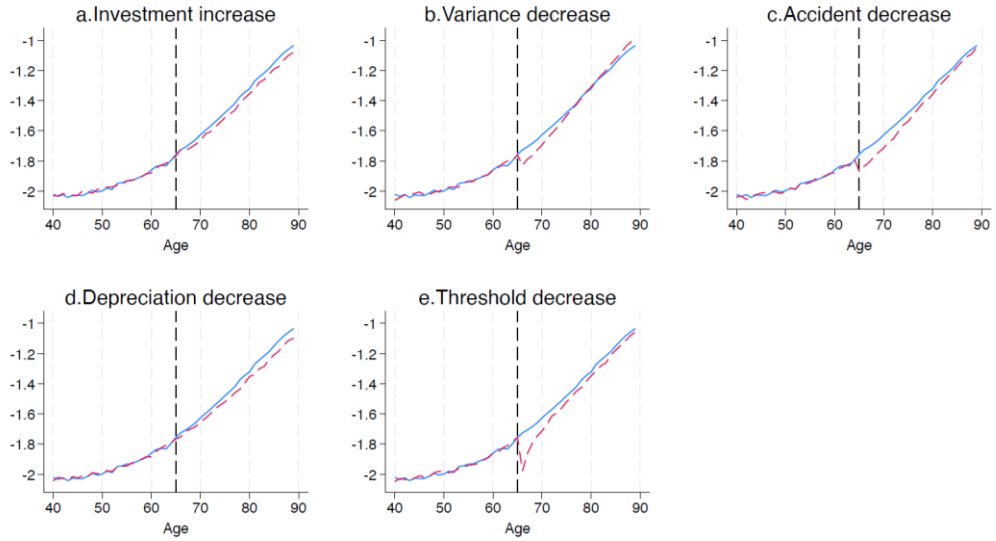
Figure 1. Comparison of mortality between Census Tree and SSA life tables for selected cohorts



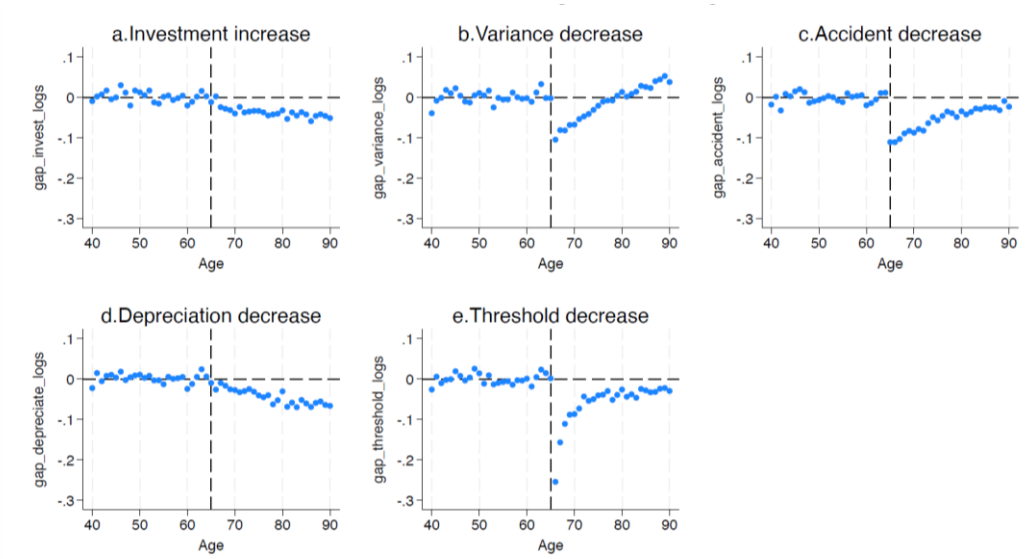
Notes: Shown are age specific mortality rate by cohort, sex, and data source. The CensusTree data represent a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. We compute age-specific mortality rate using the inverse probability 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 2. Simulated changes of Gompertz curves after permanent unanticipated shocks at age 65

a. Effects of the log of mortality

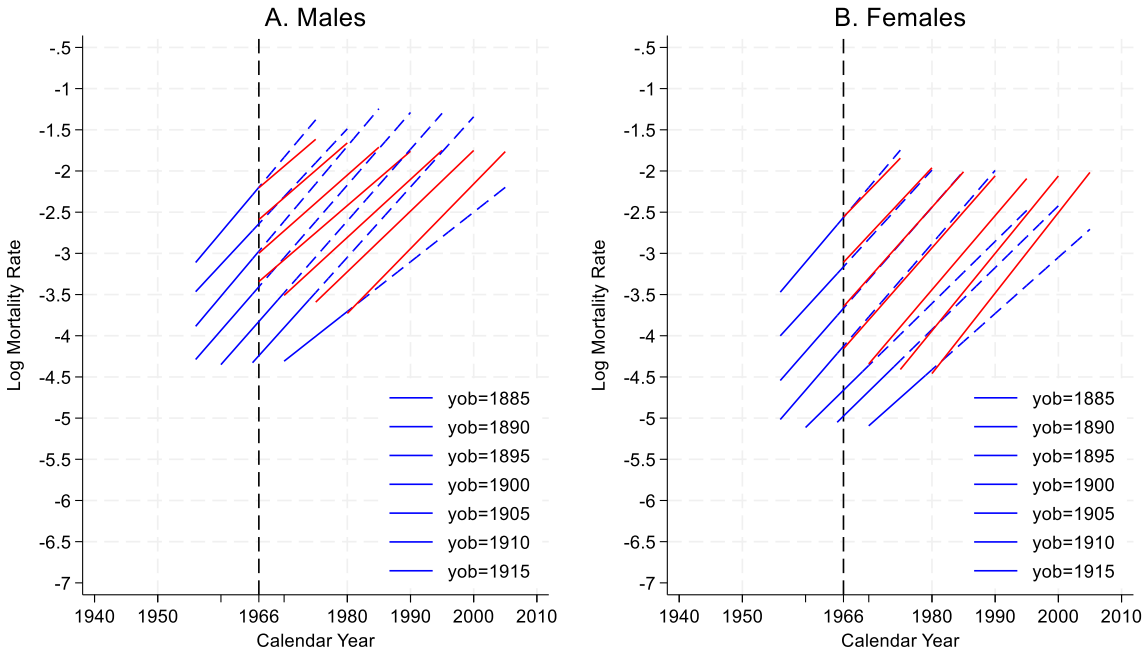


b. Effects on the gap in log mortality between affected and unaffected populations



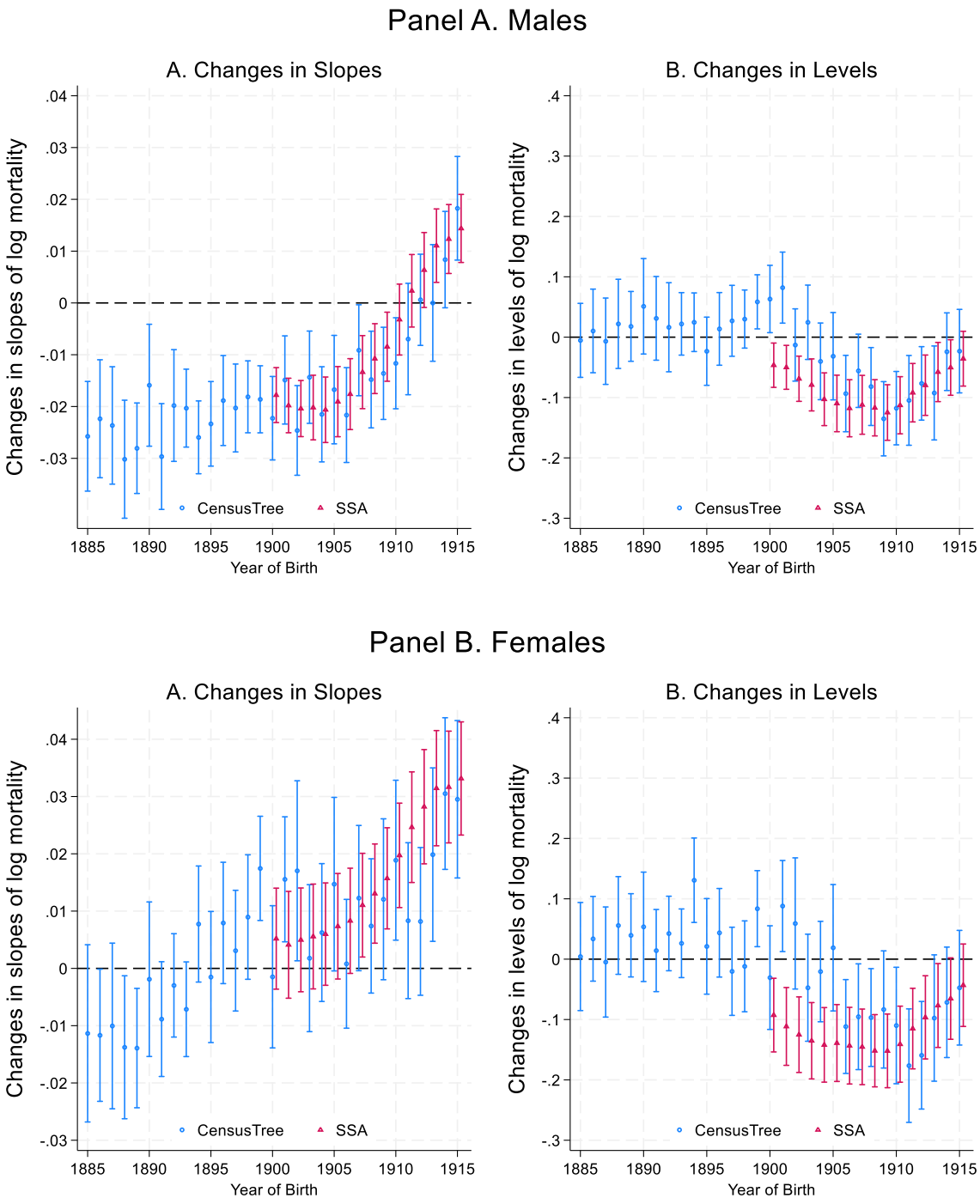
Notes: The figures show simulated results based on Lleras-Muney and Moreau (2022)'s unified model for cohort mortality, with parameter values for the baseline model given by $I = 0.4$, $\delta = 0.0006$, $\sigma = 1$, $\alpha = 1.7$, $\mu_0 = 0.9$, and an adolescent accident shock $K=0.008$. The threshold 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. Estimated effects of Medicare on the log of mortality for selected birth cohorts.



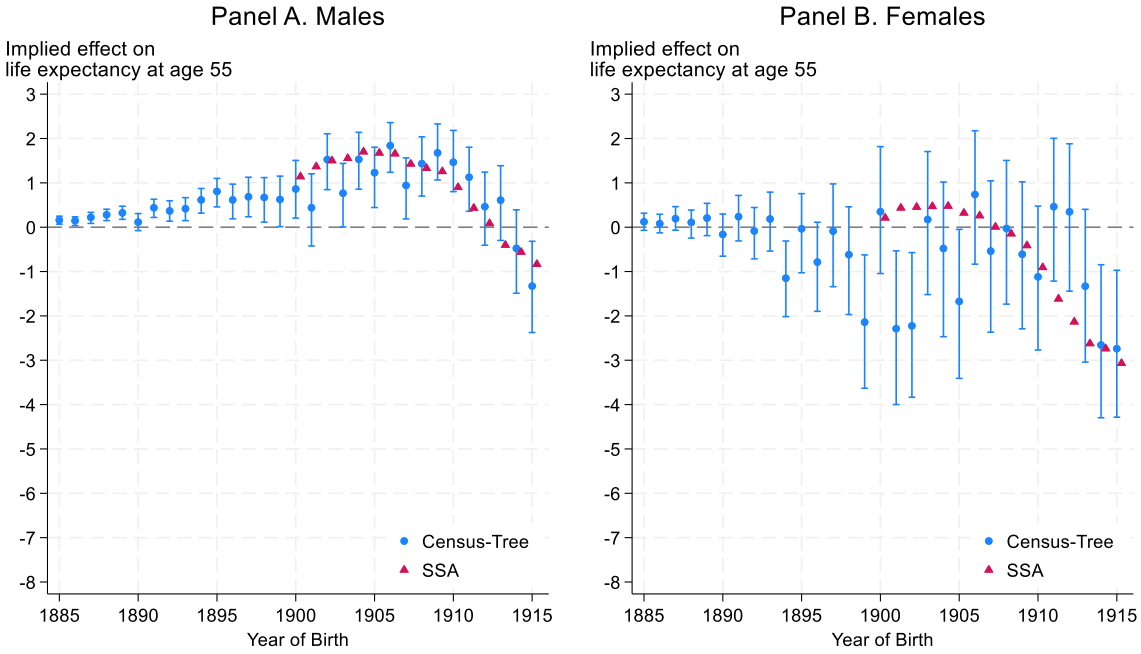
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. The figures show the log mortality (Gompertz curves) over the age window ranging from 10-year prior to Medicare eligibility age to 90, by sex and birth cohort. 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 4. Effects of Medicare on the Intercept and Slope of Mortality



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. The blue circle 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 red triangle estimates are derived from the Social Security Administration life tables, which are available starting from the 1900 cohort onwards.

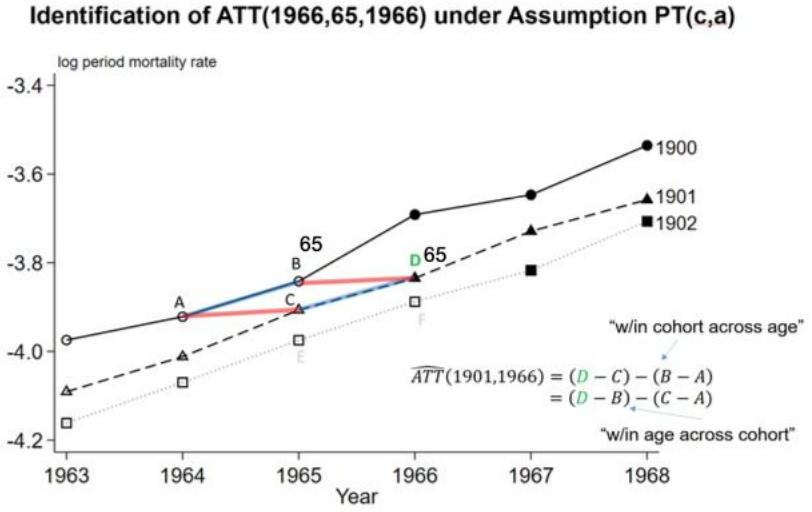
Figure 5. Implied effect of Medicare on life expectancy at age 55 using the ITS approach.



Notes: Shown are the implied effects of Medicare's introduction on life expectancy at age 55 based on our interrupted time series approach by sex, birth cohort, and data source. The blue circle 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. The red triangle estimates are derived from the Social Security Administration life tables, which are available starting from the 1900 cohort onwards. 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 6. Identification of the Average Treatment on the Treated (ATT) under the parallel trends assumption.

a. One-year treatment effect.

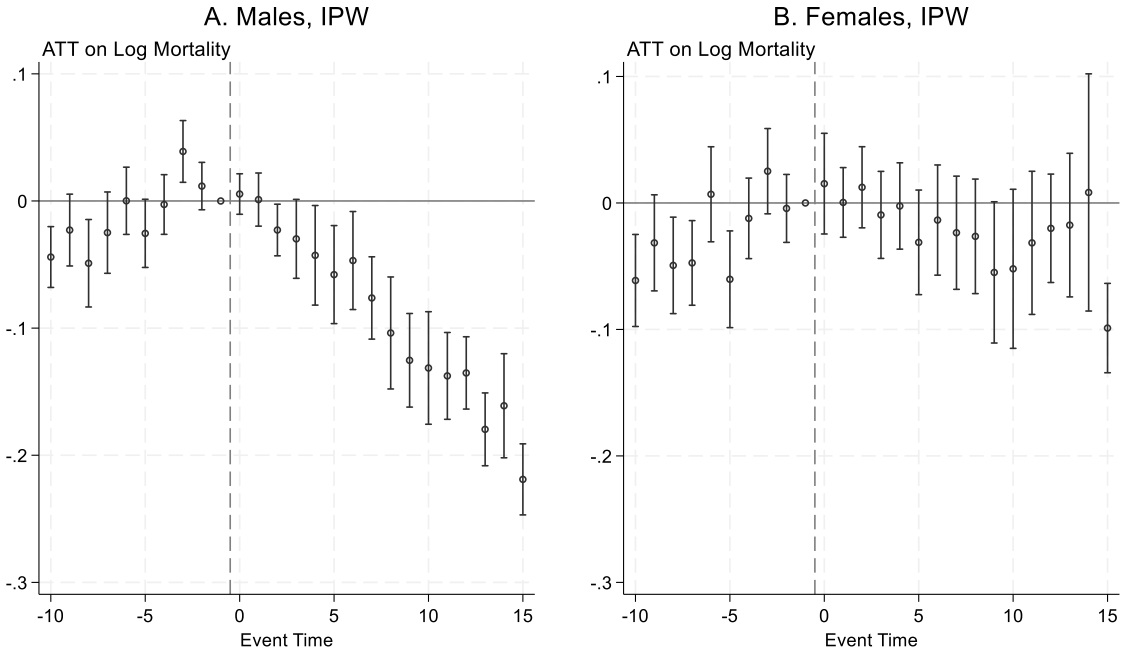


b. Ten-year treatment effect.

(Placeholder for the ten-year treatment effects)

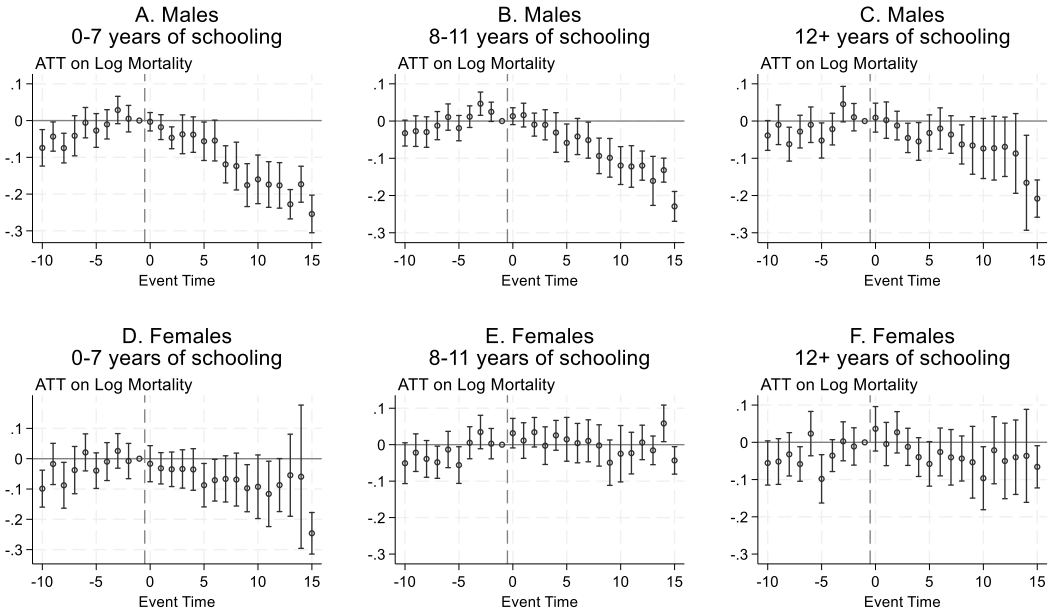
Notes: This figure highlights the estimator for $ATT(g_t, g_a, t)$. In panel a, we describe how the treatment effect for the first year. This effect is identified by comparing the change in the (log) mortality rate before (C) and after treatment (D) (at ages 65 and 64) for the 1901 cohort, to the change for the 1900 cohort 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 1900 cohort between ages 64 and 65 serves as a counterfactual for the change in mortality for the 1901 cohort because they would have evolved in parallel if the 1901 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 64. The implied estimate of the effect of Medicare in the first year in this figure is given by [(-3.83) – (-3.91)]- [(-3.84)-[-3.92]]= -0.008.

Figure 7. Event-study estimates by sex



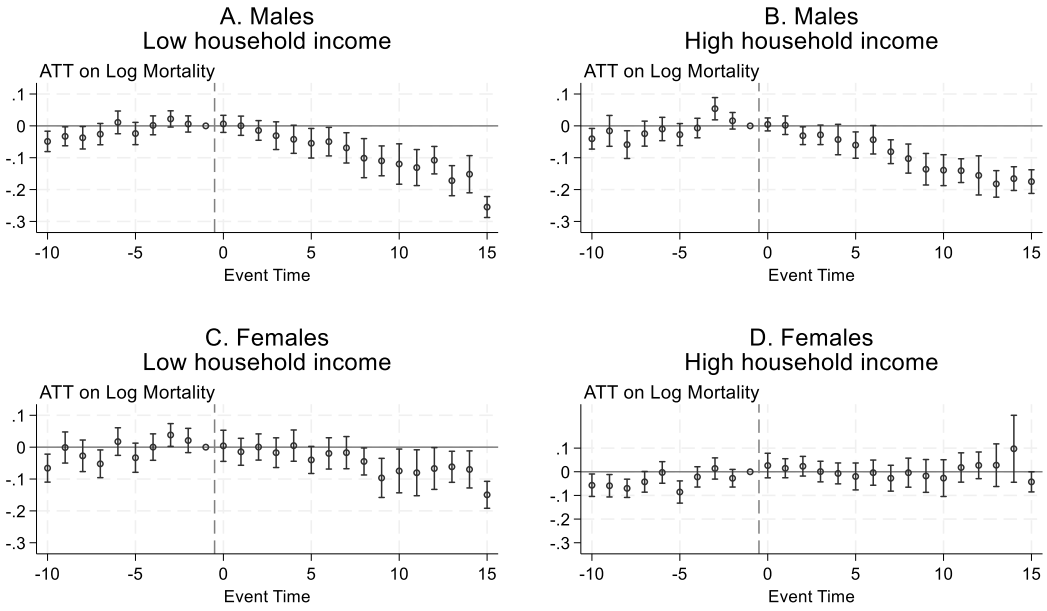
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex and birth cohort. We use Callaway and Sant'Anna (2023) for the average treatment effect on the treated (ATT) estimation and aggregation. The event time on the x-axis denotes the year relative to the Medicare eligibility age.

Figure 8a. Event-study estimates by sex and educational level



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, birth cohort, and education/income group. We use Callaway and Sant'Anna (2023) for the average treatment effect on the treated (ATT) estimation and aggregation. The event time on the x-axis denotes the year relative to the Medicare eligibility age.

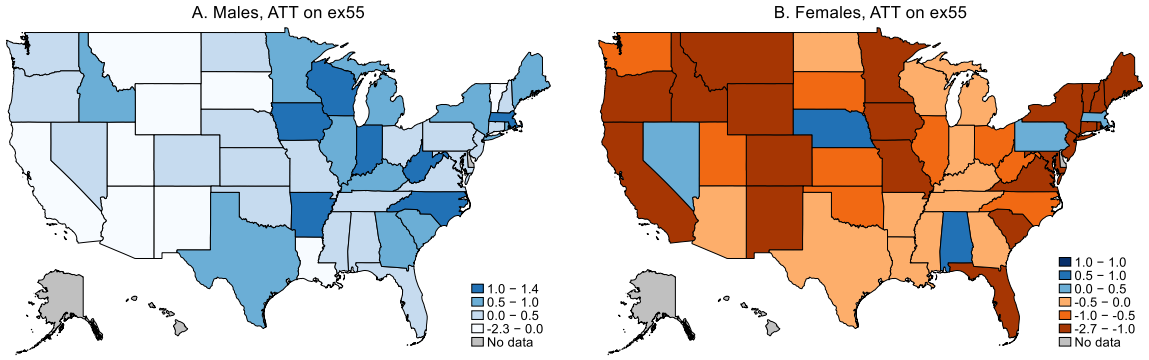
Figure 8b. Event-study estimates by sex and household total income in 1940



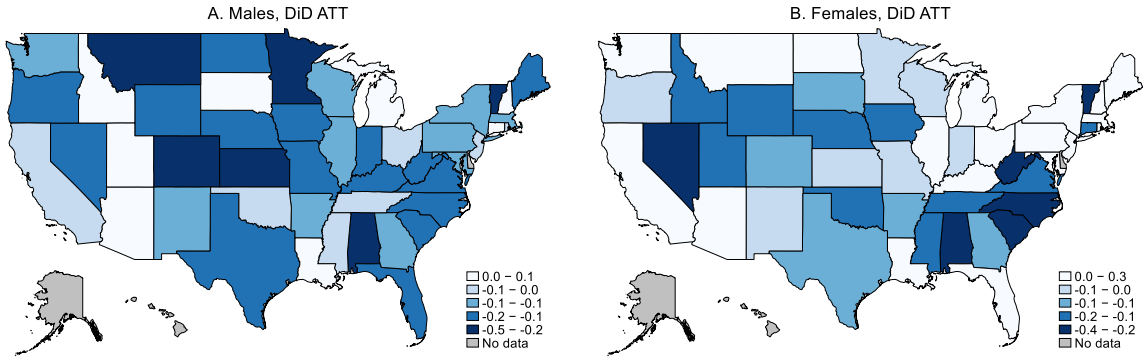
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, birth cohort, and education/income group. We use Callaway and Sant'Anna (2023) for the average treatment effect on the treated (ATT) estimation and aggregation. The event time on the x-axis denotes the year relative to the Medicare eligibility age.

Figure 9. Effects of Medicare on Life Expectancy at Age 55 and Log Mortality by state

a. Implied effect on ex55 from ITS by state



b. Aggregate event-study estimates on log mortality by state



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, birth cohort, and state. We compute the implied effects on life expectancy at age 55 based on the estimated changes in slopes and levels of log mortality from the interrupted time series (ITS) approach. The average effect on life expectancy at age 55 (ex55) is a weighted average of implied effect on ex55 across 1885-1915 birth cohorts using the number of people when there were eligible for Medicare in each cohort as weights. We use a bootstrapping approach with 1,000 resamples to obtain the standard error and 95% confidence intervals. For the DiD estimates, we use Callaway and Sant'Anna (2023) for the average treatment effect on the treated (ATT) estimation and aggregation; the post-Medicare aggregated ATT is reported.

Appendix

Section 1. Computation of implied effect on ex55 based on the ITS analysis

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

Figure A. Gompertz curve of 1905 white male cohort

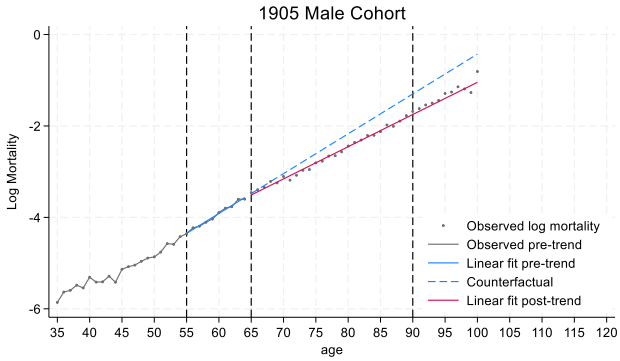
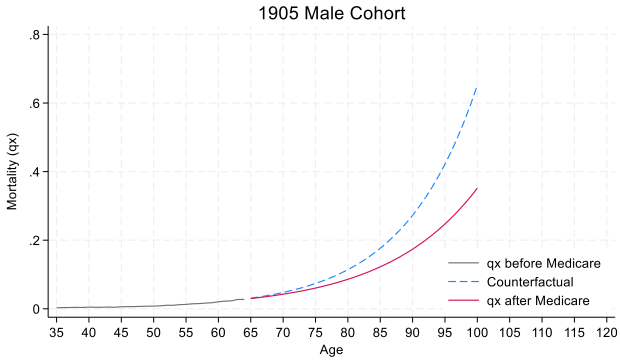


Figure B. Mortality (qx) with and without Medicare



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

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

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

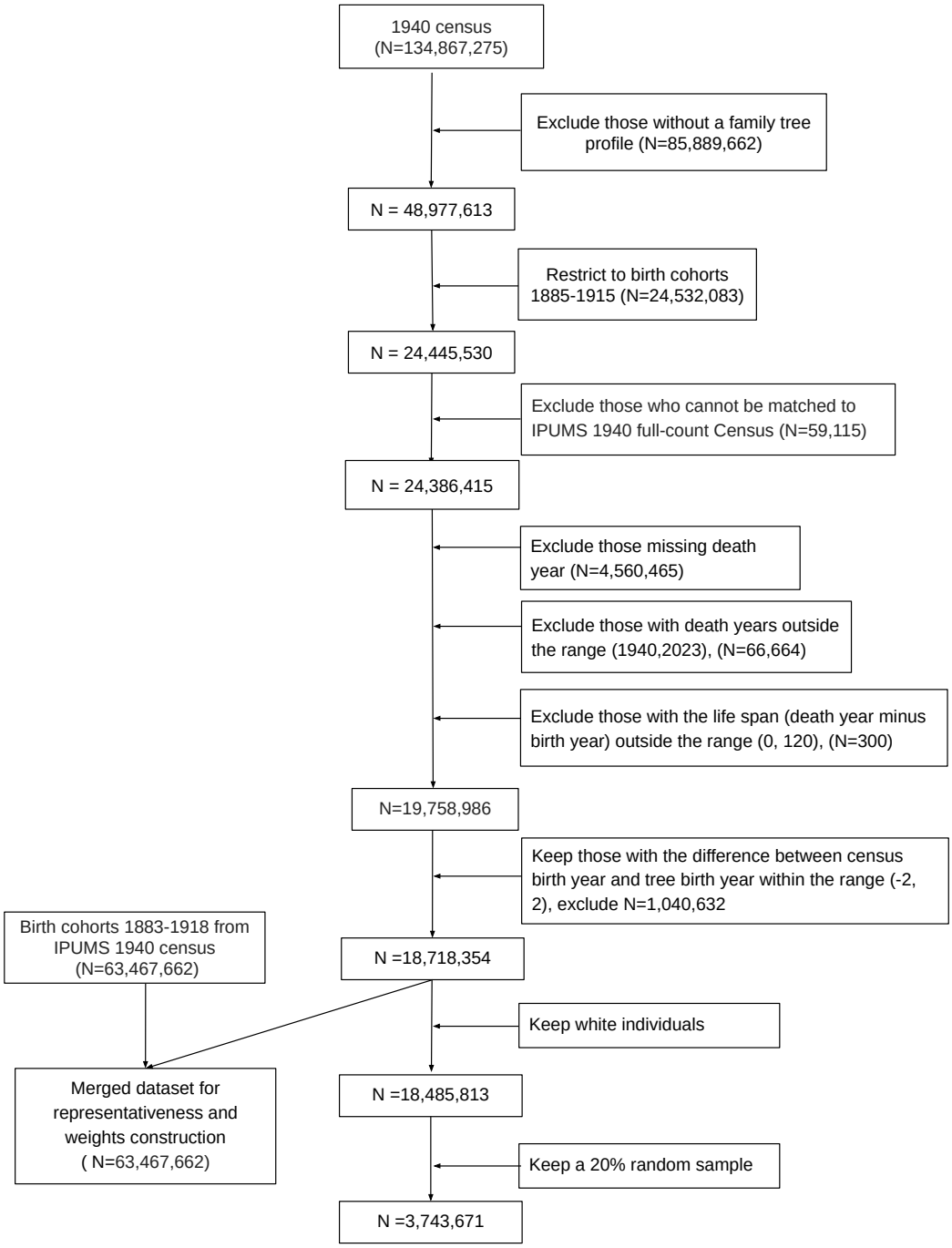
Second, to compute the life expectancy with Medicare (**ex55_policy**), we first fit a linear regression of log mortality on age for post-Medicare period (age 65 to age 90) and then predict log mortality for ages 65-100. The predicted log mortality for post-Medicare period is the red solid line in Figure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

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

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

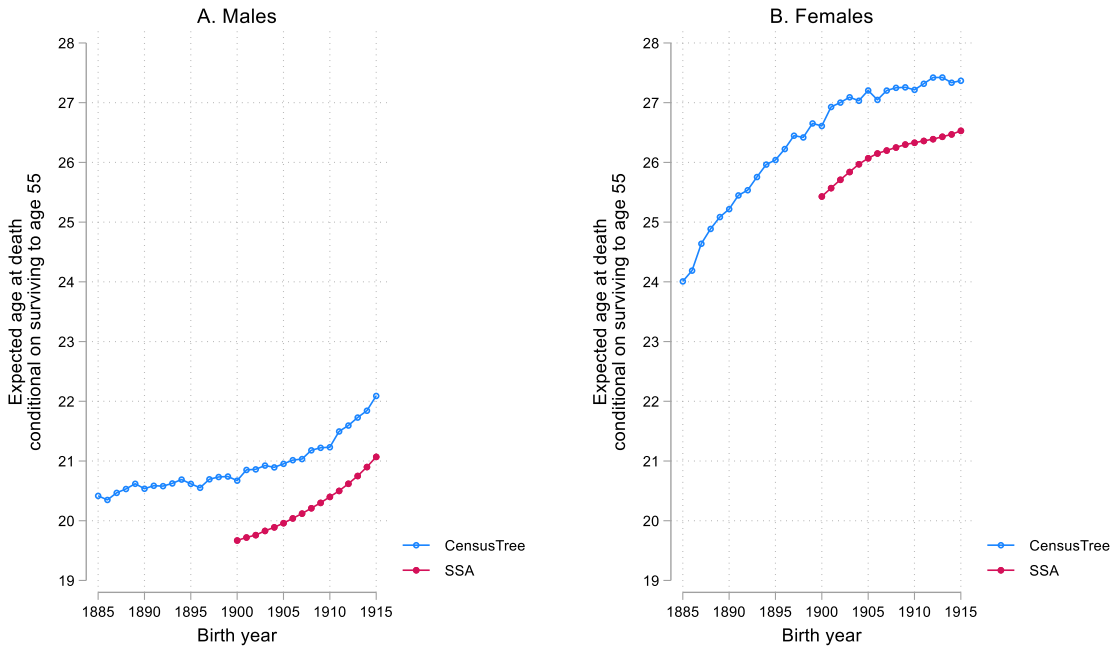
Section 2. Computation of implied effect on ex55 based on the DiD analysis

Figure A1. Sample flowchart of Census Tree Sample



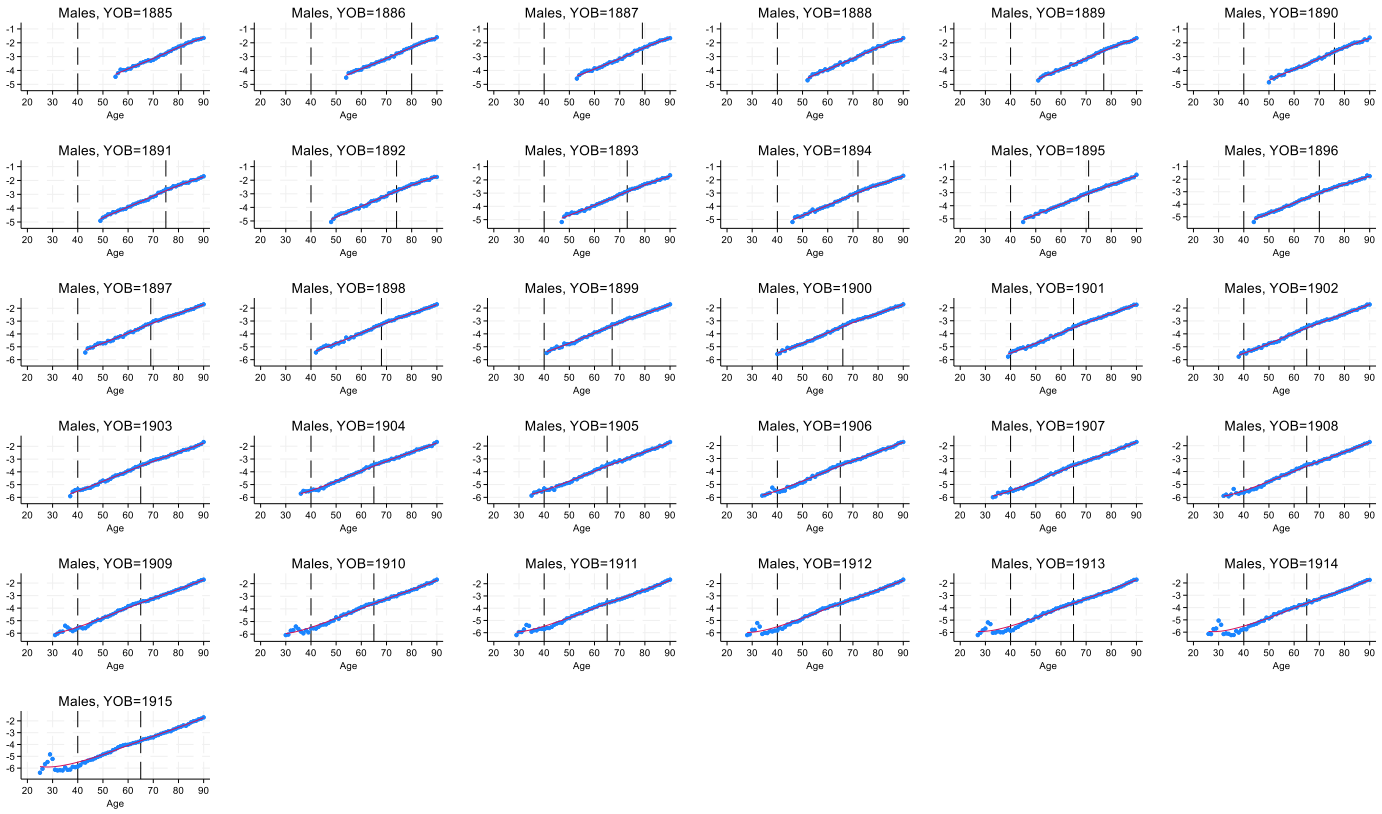
Notes: We rely on the year of birth recorded in the Family Tree in our analysis because the 1940 Census does not ask for birth year and only reports age. However, the birth year variable from Family Tree was scrapped because people would have had the chance to find birth records and make corrections.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

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



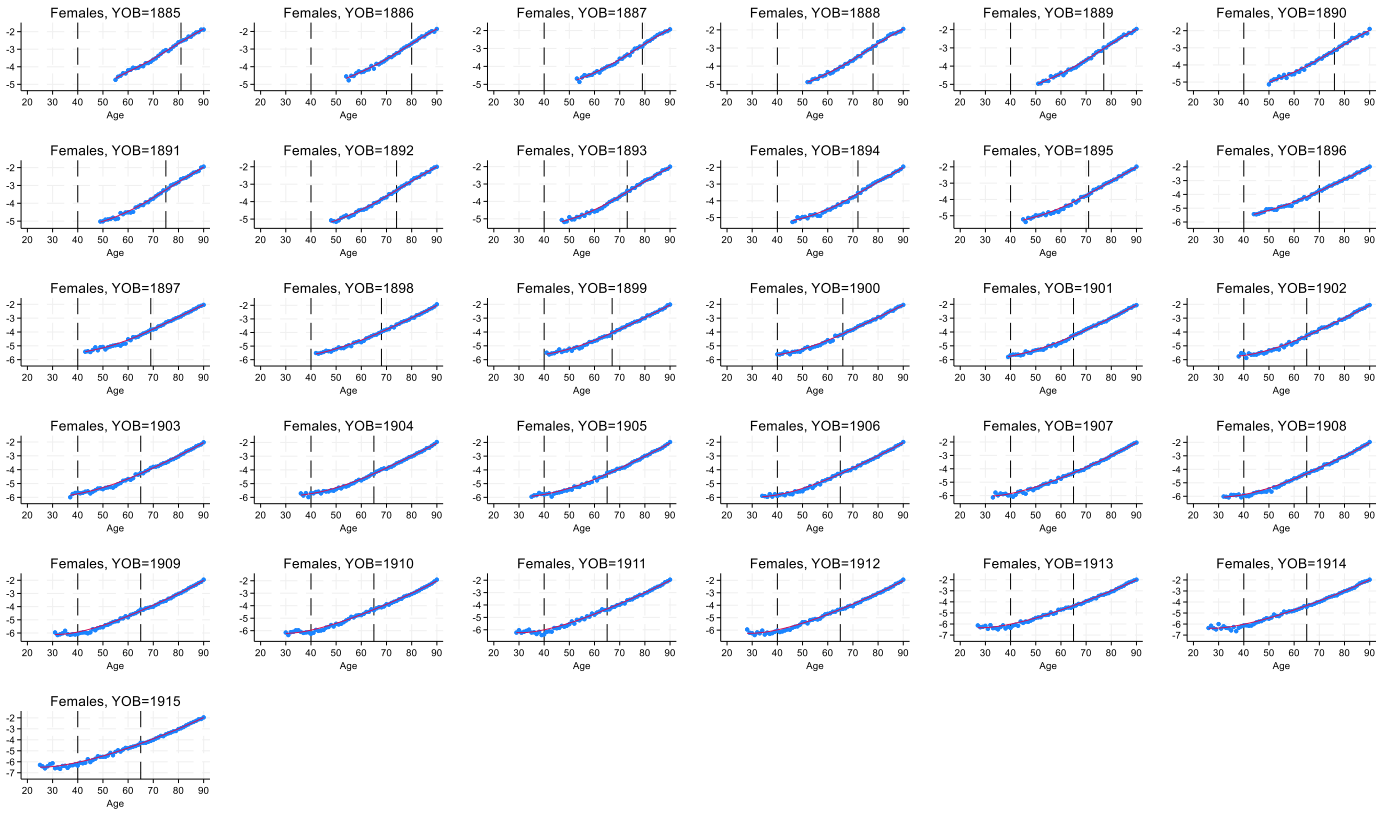
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort.

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



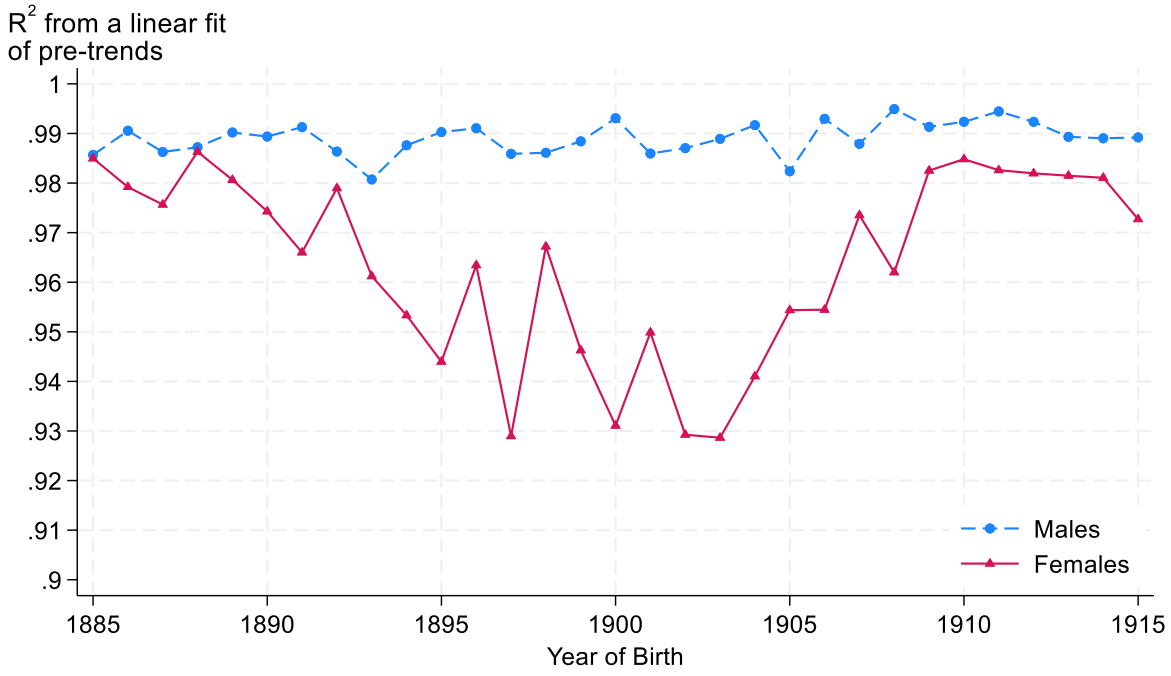
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. We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

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



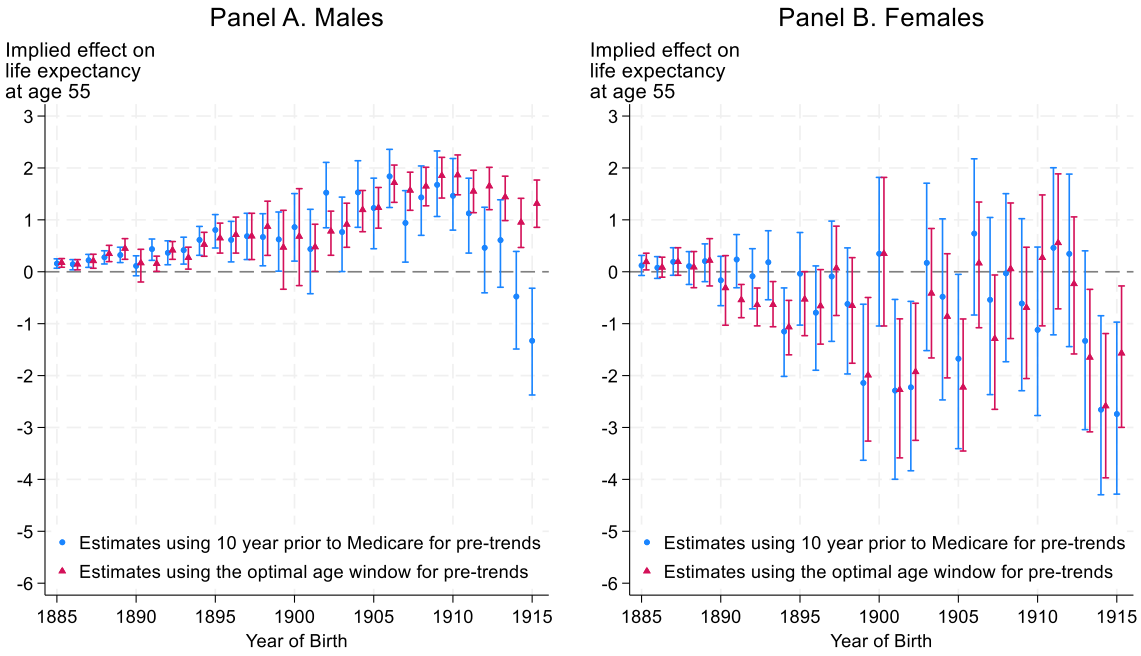
Notes: Analytic data include a 20% random sample of whites born 1885-1915 from our Census Tree data that links the 1940 full-count census to Family Tree. Using the Census Tree sample, we compute age-specific mortality rate using the inverse probability weights, separately by sex, and birth cohort. We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

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



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

Figure A5. 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.