The Impact of Medicare on Mortality

Samuel Arenberg[*] Seth Neller[†]

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Abstract

Medicare was created to bring older Americans the "healing miracle of modern medicine." Nonetheless, even six decades after its creation, the degree to which Medicare improves health and survival remains uncertain. Researchers have extensively studied the short-term impacts around the eligibility threshold of age 65, but the long-term effects that accrue slowly are not known. We address this open question by leveraging the fact that near-universal health-insurance coverage begins at age 65 in the United States—and *only* in the United States. Specifically, we estimate the impact of Medicare on mortality over long horizons using a synthetic-control approach, which generates a counterfactual from a blend of other countries that closely matches age-specific mortality rates in the United States up to age 65. Our results, illustrated by a sudden and persistent departure of the United States from that counterfactual at age 65, indicate that Medicare has extended life expectancy at age 65 throughout its history by about eight months on average. Critically, a similar impact is still seen today. The constituent causes of death point decisively toward medical care as the central mechanism. Gains arise almost solely from reductions in deaths considered amenable to timely and effective treatment. Our results imply that Medicare recoups a substantial portion of its trillion-dollar annual bill through previously unknown extensions in longevity. We conclude that lowering the age of Medicare eligibility would generate benefits exceeding the net costs to Americans.

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[*]Arenberg: University of Houston, email: sarenberg@uh.edu
[†]Neller: The University of Tennessee, Knoxville, email: sneller@utk.edu

1 Introduction

Central to the US healthcare system is Medicare, which first provided publicly financed health insurance to Americans age 65 and older in 1966. As he signed it into law, President Lyndon B. Johnson said of the program, "No longer will older Americans be denied the healing miracle of modern medicine." His oft-repeated declaration conveys an intent to grant access to new health-enhancing, perhaps life-saving, treatments.

Whether Medicare affects health, however, remains an open question 60 years after its creation. The earliest causal studies of mortality—a ubiquitous marker that captures both acute events and chronic processes—suggest that Medicare has little or no impact (McWilliams et al., 2007*a*; Card, Dobkin and Maestas, 2008; Finkelstein and McKnight, 2008; Card, Dobkin and Maestas, 2009; Polsky et al., 2009; Chay, Kim and Swaminathan, 2012). A critical limitation of these studies, however, is that they examine mortality over a short window, meaning they focus on more acute pathways. Causal studies of more cumulative mechanisms, which may be the dominant forces given that health accumulates and depreciates slowly, remain virtually absent (Goodman-Bacon et al., 2025). The implication is that the impact of Medicare on mortality is not yet known and could differ markedly from what the existing short-term evidence suggests.

The purpose of this paper is to characterize the missing long-run health dynamics that unfold over decades of aging. To do that, we conduct an international comparison of mortality profiles between the United States and other high and middle-income countries, where turning 65 carries no comparable policy significance. Specifically, we employ a synthetic-control approach. The method, from Abadie, Diamond and Hainmueller (2010), is designed to handle settings with a single treated unit. For our purpose, it generates a counterfactual from a blend of peer nations that matches the age-specific mortality rates in the United States prior to Medicare eligibility. The impact of Medicare on mortality, then, is estimated by the divergence between the United States and its "synthetic" counterpart at age 65 and into advanced ages. Crucially, this approach permits identification in recent decades, not just in the years surrounding Medicare's introduction, where existing prior causal work has necessarily been concentrated.

The method produces a counterfactual that, despite being determined only by mortality rates, demonstrates "balance" on other salient measures of health and healthcare. The counterfactual, for instance, has similar ratings of healthcare access and healthcare quality. Perhaps most instructive, rates of unmet medical need are similar prior to age 65—before those rates drop precipitously in the United States with Medicare eligibility. We emphasize, however, that the impact of Medicare on mortality need not operate solely through access to care. Medicare may improve quality of care, lower the out-of-pocket cost of care, or sim-

ply compel Americans to think differently about their health as they become eligible for a program that targets the "elderly" population.

The effect we uncover is substantial. Pooling years from the inception of the program in 1967 through 2019, we find that Medicare increases life expectancy, conditional on survival to age 65, by about eight months. For context, life expectancy at age 65 in the United States rose in that span, which was marked by unprecedented reductions in adult mortality, by about five years. Beneath the eight additional months of life, the constituent age-specific effects emerge quickly at age 65 and grow steadily until about age 80, at which point American mortality is almost 15 percent lower than under the constructed counterfactual. An effect with a similar shape is present whether we consider the earliest years of the program or more recent years. The impact is, however, smaller today (about six months) than it was at the outset of the program (about seven months) or at the end of the 20th Century, when it was largest (almost twelve months).

The results pass several falsification exercises. If we examine years *prior* to the introduction of Medicare, we find no significant separation between the United States and its counterfactual. If we generate a counterfactual using ages only through 54, the departure still emerges around age 65. We also rule out the possibility that employment-related changes could drive the result, as the United States does not depart meaningfully from peers in labor-force participation at age 65. If we apply other methods that are conceptually similar to synthetic control, we obtain similar estimates. We show that, when we cycle through all the countries in our data, assigning each of them "treatment" status, the estimates with the United States as the treated country are the largest, no matter the time period.

To understand mechanisms and further corroborate the plausibility of our findings, we also investigate cause of death. We first split deaths into two widely cited classes, "amenable" and non-amenable (Nolte and McKee, 2003). Amenable deaths are deaths that should not occur (or should be delayed significantly) in the presence of effective medical care. We indeed find that the effect loads onto amenable causes. This is true even though amenable causes comprise only about one third of deaths in our sample period. When we instead group deaths into several broad categories, we find that the reductions in all-cause mortality come largely from reductions in death due to cardiovascular diseases, the rate of which is down by about 25 percent in ages 80 through 84. Deaths from cancers are also down by an important margin. External causes of death, on the other hand, show no indication of a change at age 65. The apparent heterogeneity is consistent with medical advances of the last 60 years, as, for example, understanding of cardiovascular disease improved dramatically and early detection and effective treatment of cancer became feasible.

Although it is concentrated in certain causes of death, we show that the impact of Medicare is felt broadly within the United States. Consistent with the national nature of the pro-

gram, we find reductions in mortality among both men and women, among both white and Black people, and in every region of the country. The impact is not, however, even. We find that effects are larger for women than for men and larger for Black Americans than for white Americans. The implication is that Medicare is responsible for some of the female mortality advantage and for some of the convergence between Black and white mortality witnessed in recent decades.

We also consider a critical marker of health among the living. Specifically, we pull together rates of disability for a large set of countries, split out by type of difficulty. We see that the likelihood of reporting physical difficulties decreases significantly in the United States starting at the eligibility threshold. By advanced ages, the likelihood that someone reports difficulty with walking or with self-care, such as dressing or bathing, is substantially lower than the counterfactual rate. These influences on morbidity imply that Medicare increases, not just years of life, but quality of life.

Because calls to expand (and to curtail) Medicare are growing, we conclude with a cost-benefit analysis of changing the age threshold for eligibility. We estimate that lowering it to age 55 would increase life expectancy at the threshold by an additional five months (from six to eleven months), whereas raising it to age 75 would decrease the gains by four months (from six to two months). Lowering the age of eligibility would certainly increase government expenditure substantially. However, given that Medicare pays less than other insurers for many of the same goods and services, such an expansion would likely *decrease* total expenditure.

The impact of Medicare on mortality advances several literatures. First, it continues a recent wave of papers that credibly identify mortality effects of health insurance coverage, namely effects of expanding Medicaid eligibility (i.e., Miller, Johnson and Wherry, 2021; Wyse and Meyer, 2025), purchasing private insurance plans (i.e., Goldin, Lurie and McCubbin, 2021), and switching Medicare Advantage plans (i.e., Abaluck et al., 2021). Those papers redefined the consensus about the impact of health insurance on adult mortality, yet they capture only short-run effects for specific subpopulations in response to recent interventions. Our estimates reveal long-run dynamics that apply to the full population and span several decades.

Second, our findings add to a large and growing literature documenting the benefits of various social safety net and social insurance programs, many of which trace back to President Johnson's Great Society. Many components of the Great Society—Medicaid (e.g., Goodman-Bacon, 2018, 2021; East et al., 2023), food stamps (e.g., Bailey et al., 2024), and head start (e.g., Bailey, Sun and Timpe, 2021), for instance—have been rigorously examined from a long-run perspective. Medicare is an exception. To date, only a contemporaneous working paper, Goodman-Bacon et al. (2025), has endeavored to quantify the impact of Medicare on

life expectancy, a measure that would capture the life-course dynamics that are so difficult to identify. That paper, however, considers only the introduction of Medicare. The impact of Medicare in recent years, when much has changed, remains unknown. Our international approach informs through the present, and we forecast how US life expectancy might change if the age of eligibility for Medicare were changed.[1]

Third, we build on a body of causal work establishing a wide range of impacts, whether short-term or long-term, of Medicare itself. Those impacts, of course, include strong influences on healthcare delivery (e.g., Finkelstein, 2007; Duggan and Scott-Morton, 2010; Clemens and Gottlieb, 2017; Sacarny, Yokum and Zhang, 2022; Sacarny, Barnett and Olenski, 2023). Many of its other important impacts are financial (e.g., McWilliams et al., 2007*b*; Finkelstein and McKnight, 2008; Barcellos and Jacobson, 2015; Goldsmith-Pinkham, Pinkovskiy and Wallace, 2023; Gottlieb et al., 2025). The health impacts include the aforementioned studies of mortality (e.g., McWilliams et al., 2007*a*; Card, Dobkin and Maestas, 2008; Finkelstein and McKnight, 2008; Card, Dobkin and Maestas, 2009; Polsky et al., 2009; Chay, Kim and Swaminathan, 2012). They also include more recent studies on certain features of Medicare, especially access to prescription drugs and cancer treatment (e.g., Gowrisankaran, Town and Barrette, 2011; Afendulis, Chernew and Kessler, 2017; Huh and Reif, 2017; Andersen, 2018; Dunn and Shapiro, 2019; Kaestner, Schiman and Alexander, 2019; Myerson et al., 2020; Chandra, Flack and Obermeyer, 2024; Roberts et al., 2025; Horn et al., 2026). Our goal is to consolidate these benefits and the many yet to be documented, through life expectancy, into a single estimate of Medicare's beneficence. That estimate appears sufficient to offset a substantial portion, a third or more, of the now trillion-dollar annual cost of providing Medicare. Yet we also examine markers of morbidity and find reductions, for instance, in rates of physical difficulty, adding another impact to the literature and suggesting that Medicare grants, not just more years, but better ones.

Finally, we contribute to the expansive interdisciplinary literature on the determinants of mortality (e.g., Cutler, Deaton and Lleras-Muney, 2006). Though mortality was long governed by other factors, healthcare is now a critical determinant. Our estimates—substantial reductions in mortality driven by deferring deaths thought most amenable to modern medicine—provide a clear statement of that new regime. The impact of Medicare approaches the impact of the most significant scientific advances in recent centuries on adult longevity.

[1] With respect to Goodman-Bacon et al. (2025), in an appendix, we compare (and contrast), in detail, the subset of our results that overlaps with theirs.

2 Data

We rely primarily on mortality data from the Human Mortality Database ("HMD"), a publicly available resource that provides harmonized, high-quality mortality and population estimates for countries with reliable vital registration systems. The database compiles official death counts and population exposures from national statistical offices and applies standardized demographic methods to construct internally consistent life tables. Measures of mortality, such as age-specific probabilities of death, are reported by single year of age (from zero to extinction), calendar year (or year of birth), and sex. For our main analysis, when we pool mortality rates from 1967 through 2019, we have 40 countries on which to construct a synthetic control, a process we discuss in the next section. See Appendix Table A1 for a comprehensive list of countries that are part of our inquiry.

We augment the mortality data from the HMD with data from the World Health Organization Mortality Database ("WHOMD"), which similarly gathers national vital registration data, in two endeavors. First, we want to verify that there is no "treatment effect" in periods prior to the enactment of Medicare. The World Health Organization includes more countries in those (older) years, which greatly improves the fit of our synthetic control.[2] Second, the World Health Organization provides causes of death for a large number of countries. The Human Mortality Database has cause of death as well, but for fewer countries.[3] The causes are based on standard categorizations of underlying cause from the International Classification of Diseases (ICD). We focus on more recent years (1980 onward) to increase the number of countries available and to restrict attention to the most comparable coding regimes, ICD-9 and ICD-10.

Our study involves many other data sources in various supporting analyses. To pull greater detail on the United States, we draw on the American Community Survey, the Current Population Survey, the National Health Interview Survey, the Medical Expenditure Panel Survey, the Multiple Cause of Death files from the National Vital Statistics System, and the Centers for Medicare and Medicaid Services National Health Expenditure data. Abroad, we bring together data from the European Union Statistics on Income and Living Conditions, the European Health Interview Survey, IPUMS International, the Luxembourg Income Study, the Global Burden of Disease, the International Social Survey Programme, United Nations National Transfer Accounts, and several country-specific surveys, including the Taiwanese

[2] In the additional years from the World Health Organization, however, mortality rates are available only in five-year age intervals. Thus, analyses using older years are coarsened with respect to age. In years where there is overlap, the two sources have nearly identical mortality rates.

[3] In both sources, cause-specific mortality rates are available only in five-year age intervals. So, like analyses using older years, analyses using cause of death are coarsened. Again, where data exists in both sources, rates are very similar.

Social Change Survey and Russian Comprehensive Living Conditions.

This assembly of disparate data sources allows us, to the greatest extent possible, to characterize the United States and its synthetic counterpart along dimensions other than mortality. As noted, the number of available countries and the granularity of age vary by outcome and era, but coverage is sufficient at each stage of the analysis to support the comparisons we draw. Summary statistics for our three main outcomes, all-cause mortality, cause-specific mortality, and disability rates, appear in Appendix Tables A2 through A4, respectively. The sources for supporting analyses are introduced as they enter the paper, but a complete catalog, with exposition of data-access procedures and data-construction decisions, is provided in Appendix A.

3 Methods

Our international comparison is based on the synthetic-control method first formalized by Abadie, Diamond and Hainmueller (2010). The idea, in our context, is to find a weighted average of other countries, "donors," that matches the age-specific mortality rates of the United States prior to age 65. In practice, the counterfactual is generated by searching over the convex hull of possible weighted averages of donors—the set of all non-negative weight vectors summing to one—to find the combination that most closely reproduces the pre-65 mortality profile of the United States. Specifically, the optimal weights solve:

$$\text{argmin}_{\omega} \sqrt{\frac{1}{N_{pre}} \sum_{a=45}^{64} \left(y_{a,usa} - \sum_{c \neq usa} y_{a,c} \times \omega_c \right)^2} \quad \text{subject to} \tag{1}$$

$$\sum_{c} \omega_c = 1 \quad \text{and} \quad \omega_c \geq 0 \,,$$

where $y_{a,c}$ represents the log mortality rate for a given country c at a given age a. After estimating the weights ω, our age-specific treatment effects are given by:

$$\tau(a) = y_{a,usa} - \sum_{c \in W} y_{a,c} \times \hat{\omega}_c \,, \tag{2}$$

where W is the set of donor countries receiving positive weight.

Unless otherwise noted, the confidence intervals presented with our results are calculated using the placebo method outlined in Clarke et al. (2024). This method uses the variance across placebo treatments, wherein a given $\tau(a)$ is calculated for each donor unit in our sample, to obtain a sampling distribution for each $\tau(a)$. Specifically, with the set of all (non-US) countries, C, we calculate:

$$\hat{V}_{\tau_{usa}} = \frac{1}{|C|} \sum_{c \neq usa} \left(\tau_c - \frac{1}{|C|} \sum_{c \neq usa} \tau_c \right)^2 , \tag{3}$$

which is then used to construct standard errors and confidence intervals.

Synthetic control is a natural fit for our inquiry. The method was designed to handle scenarios with a single treated unit, as we have. Standard panel-data methods, including many difference-in-differences approaches, can be unreliable when there are one or few treated units (Abadie, 2021). Synthetic control holds an advantage over standard panel-data methods also because our outcome is mortality. Death at a given age might be influenced by mortality rates earlier in the life course through processes of scarring or selection (e.g., Bozzoli, Deaton and Quintana-Domeque, 2009). Consequently, even if it were to match US *trends* in mortality prior to age 65, a counterfactual with different *levels* of mortality might manifest a "treatment effect" with or without intervention. Standard panel-data methods rely only on demonstrations of parallel trends. Synthetic control will match the entire path of mortality, both level and trend as well as higher-order derivatives with respect to age.

Furthermore, our international comparison—for which synthetic-control methods are ideal—is perhaps the only way to empirically study the effects of Medicare on health across the entire population and across the life course, apart from its inception 60 years ago. However, estimates from Medicare's introduction may differ from its present-day effects. We will consistently show results for three different "eras" in addition to main results from pooling all years. Namely, we will break the "pooled" period, 1967 through 2019, into three similarly wide pieces: 2000 through 2019, the "recent" era; 1980 through 1999, the "middle" era; and 1967 through 1979, the "early" era.[4] For each era, we will generate a new counterfactual. To aggregate across years within each era, for a given country and age, we simply take the unweighted average of mortality rates from all years in the era. We restrict our attention to the countries available consistently throughout the era.

In principle, the optimal vector of weights might lie outside the convex hull spanning the donor pool. After all, the United States, for its standard of living, is an outlier in terms of longevity. However, our data are not limited to the wealthiest European nations, where mortality rates would be almost uniformly lower at the ages on which we match. We find good fits, and we show the optimal weights in Appendix Figure A1. We show them for the pooled years and for each era. As one illustration, in the pooled period, the three countries with the largest weights are Hungary at approximately 0.30, Chile at 0.25, and Israel at 0.15 (with the remaining 0.30 spread across several countries, including Iceland, New Zealand, South Korea, and Russia). In specific eras, Canada, Scotland, Spain, and Taiwan feature more

[4] We exclude 1966 because it is partially treated. Medicare passed in July of 1965 and took hold in July of 1966.

prominently. In several comparisons to follow, we will denote these optimal mixtures (for a given era) as ω-weighted averages.

Although these counterfactuals are constructed using mortality alone, the synthetic United States resembles the actual United States along several other dimensions of health. Once our main result is introduced, we will show, for instance, that rates of unmet medical need are similar (prior to age 65), as are reputable ratings of healthcare access and quality. We will also validate our main result through several falsification tests. We will consider a "placebo" era, 1950 through 1965, and show that no effect exists where no effect should exist (because Medicare did not exist). We will also show that capping the match window at age 54 (i.e., well before Medicare eligibility) leaves the separation intact at age 65. If the synthetic control were failing on unobservable dimensions, we would expect the United States to diverge as soon as the match window closes, at age 55. Instead, it tracks closely for another ten years, until age 65. We will also show that the "treatment effects" for other countries are consistently smaller than estimates for the true treated unit, the United States. We will follow these falsification tests with a battery of other robustness checks, including alternative methods that are similar to synthetic control and numerous restrictions on the donor pool.[5] Accordingly, we believe synthetic control yields a viable counterfactual for the path of mortality in the United States absent Medicare.

4 Results

4.1 Mortality profiles

We illustrate our main result—the impact of Medicare on mortality—in multiple ways. We first show, in Figure 1, the mortality profiles of the United States and its synthetic counterfactual in each era. In the left-hand column, we plot age-specific mortality rates—raw means for treatment and control—expressed as deaths per 1,000 on a log scale from age 45 through 85. Critically, the two profiles are nearly identical in every era prior to age 65, demonstrating that the synthetic control succeeds in combining peer countries in a way that matches the United States' trajectory prior to Medicare eligibility. At age 65, however, Americans break from their peers, again in every era, in the direction of becoming healthier than the counterfactual. Their advantage grows steadily with age. Since Medicare's inception, Americans age

[5] This list of checks on internal validity is not exhaustive. Hollingsworth and Wing (2022) lay out several recommendations for assessing threats to identification in a synthetic-control setup. We utilize their framework to provide a structured discussion of all the potential issues that we consider (and reject) in our setting in Appendix Table A5.

80 faced a 6.6 percent chance of dying over the next year. Absent Medicare, that likelihood would have instead been 7.7 percent.

We will formalize these differences shortly, but we first note another important feature of the mortality profiles, the exponential growth—or lack thereof—in risk with respect to age. In the right-hand column of Figure 1, we plot deviations from a log-linear (exponential) trend fit by ordinary least squares to pre-65 mortality rates. We do this unit-specific detrending for the United States, its counterfactual, as well as other high-income countries for perspective, and we recenter each series of residuals around the pre-65 average of those high-income countries, which is normalized to zero.[6] The residuals, in virtually every era and location, are approximately flat before age 65 but rise soon after. The implication is that mortality, broadly, accelerates faster than the prediction of Gompertz Law, the empirical regularity first documented two centuries ago that an individual's mortality risk rises exponentially throughout adulthood (Gompertz, 1825).[7] However, Gompertz Law, despite its name, is not a law, and population-level mortality rates can break log-linearity for many reasons (e.g., Vaupel, Manton and Stallard, 1979), including Medicare in the case of the United States.

We highlight this pattern, partly, to stress that the trajectory of the *synthetic* United States, despite defying a commonly held view of mortality, is in line with global norms. The most important finding in the right-hand column of Figure 1, however, is the *shape* of the departures from log-linearity. Clear from both columns, the United States becomes healthier than its counterfactual once it reaches the Medicare threshold. Less obvious but still very telling, the (actual) United States appears to be the only place where mortality rates, relative to trend, *drop* with age 65. This is seen most clearly in the early and middle eras or in the pooled period. Indeed, while eventual increases in mortality above extrapolation are a global phenomenon, immediate *reductions* in mortality appear to be a feature specifically of the United States, the only country in the world to condition health insurance coverage to people 65 and older.

The right-hand column of Figure 1 serves one more purpose. It illustrates the fact that the United States, among high-income countries, ranks as one of the worst in terms of mortality. As displayed in Appendix Figure A2, it is more comparable to countries deemed middle-income, which explains why the synthetic-control method places weight on countries that

[6] Countries were classified as "high-income" if they have been designated as such since the World Bank first published the distinction in 1987. Like we do with years in an era, we take means across countries among the high-income countries prior to generating residuals as outlined.

[7] It is possible that Gompertz Law doesn't take hold until a later age in many places or that the log-linear fit prior to 65 isn't perfect. Regardless, Gompertz Law is widely believed to hold firmly from about age 40 until very advanced ages, and we do not see that. Nor do we rely on it for identification. Based on these mortality profiles, methods that impose exponential growth in mortality risk may understate Medicare's impact or miss it entirely.

are not its closest economic peers.[8] This exercise reminds that income is not the dominant force on mortality. If it were, the United States would not be an outlier. Our results suggest that access to medical care is a far more important factor.

4.2 Main results

We formalize the impact of Medicare on mortality in Figure 2. Namely, we plot the percentage differences in age-specific mortality rates, $\tau(a)$, between the United States and its counterfactual and shade 95-percent confidence intervals around those differences.

Pooling all the years of Medicare's existence, in Panel A of Figure 2, we reiterate that the differences prior to age 65 are approximately zero. Again, the implied similarity in mortality rates is by design. The synthetic-control method finds a nearly exact match for the United States throughout midlife. Upon reaching the Medicare threshold, however, Americans diverge from counterfactual, becoming less and less likely to die at any given age. The protective effect grows in percentage terms steadily until about age 80, at which point mortality in the United States is about 15 percent below what is assumed absent Medicare.

In Panel A (and in every panel), we summarize these age-specific effects in two ways. We first report the arithmetic mean of $\tau(a)$ across ages 65 and older, which we denote the "ATT." This formulation has the advantage of being comparable across exercises, some of which necessarily involve less granularity in age or more restrictive top-coding of age. In the pooled period, that average is a reduction in mortality risk of about 10 percent (0.10 log-points). The associated 95-percent confidence interval on the ATT ranges from a reduction as large as 15 percent to a reduction still of 5 percent, meaning that we rule out not only null effects but modest ones.[9]

For a more intuitive aggregation, we run the results through a life table to generate life years saved. Specifically, we calculate life expectancy (conditional on survival to age 65) with mortality rates from the synthetic control and subtract the life expectancy given when deflating those rates by the pictured treatment effects. (This calculation includes ages beyond 85, which are not shown in our primary analysis to maintain comparability with other specifications where age top-coding is prevalent. Mechanically, age-specific probabilities of death must converge to unity with age, as mortality is currently inevitable, and the estimated dif-

[8] We also see from Appendix Figure A2 that the departures from Gompertz Law are common, not just among high-income countries, but among middle-income countries. We caution, however, that everything we show is from dual-source mortality data, meaning that deaths and exposures are collected separately. Single-source mortality data, which are rarely available, may not demonstrate the same patterns.

[9] Because the proper way to conduct inference on synthetic control is still an open subject of research, we will consider other methods for calculating confidence intervals. All results that obtain significance under our baseline method will also obtain significance under the alternatives.

ferences do return to zero.) The result in the pooled period, when people aged 65 would often die in their late 70s or early 80s, is 0.68 years of life.

Across its existence then, Medicare has extended the lives of older Americans by about 8 months. For context, during the 60 years since Medicare's passage, life expectancy at age 65 in the United States rose—in what was a uniquely active period for adult mortality worldwide—by about 5 years.

The magnitude of Medicare becomes clearer when set against the landmark episodes that shaped elderly mortality in the two centuries prior.[10] In each of these three spans, life expectancy at age 65 rose by just over a year: between 1750 and 1900 in Sweden (one of few countries with reliable records), in a period that captures many of the gains associated with the Second Agricultural Revolution and the discovery of germ theory; from 1900 to 1940 in the United States, when many cities would filter and chlorinate their water supplies and when several important vaccines would become available; and during the 1940s in the US, a decade marked by the proliferation of sulfa drugs and penicillin. Indeed, the impact of Medicare on mortality approaches the level of some of the most significant events of the epidemiological transition. We will return to how our estimates fit with the literature.

An important feature of our inquiry is that we can examine how the impact of Medicare has evolved over its tenure, as medical care has advanced and as private insurance has proliferated. Our focus is on Panel B of Figure 2, the recent era. It is most relevant from a policy perspective, given its proximity to the present. It is also the period when we have the most information available with which to assess outcomes that precede mortality, which we will do later in this paper.

In the recent era, we see a break that emerges decisively at age 65. The United States again gradually improves relative to counterfactual in percentage terms before leveling off around age 80. In the recent era (2000-19), the overall impact does appear to be muted, stabilizing at about 10 percent lower with life-years saved amounting to about 6 months. Medicare's impact is largest at the end of the 20th Century, in the middle era (1980-99), as seen in Panel C, where the increase in longevity is nearly 12 months. In the early era (1967-79), in Panel D, we find an impact of about 7 months. We must note, however, that our confidence intervals are insufficiently precise to make definitive statements about one era delivering statistically larger or smaller effects than the others. Consequently, we will not speculate as to the drivers behind the differences, but we *can* confidently state that Medicare ranks highly on historical scales in every era since its inception.

[10] These historical events had seismic effects on mortality at younger ages. We are deliberately comparing longevity gains conditional on survival to age 65. All figures are authors' calculations using the HMD or life tables from the Social Security Administration. They are not intended to be the causal impact of select events. They are time-series differences surrounding the events meant to convey the pace of progress.

4.3 Unmet need

To contextualize the main results, we provide some perspective on the state of healthcare in the countries that comprise our synthetic control, which, again, is a weighted-average of countries (with weights denoted ω) based *only* on mortality rates. In doing so, we will show that the counterfactual is similar to the United States in several salient measures of health inputs—prior to age 65. For these demonstrations, we will focus on the recent era (2000-19) and therefore on the synthetic control for the recent era. We focus on the recent era partly because data elements other than mortality are available for so few countries in earlier eras and partly because of its policy relevance.

We report "balance" in two important respects. We first consider unmet medical need, which Medicare is meant to ease. In Figure 3, we plot, by the available age groups, the fraction of people who report forgoing care for any reason in Panel A and for financial reasons specifically in Panel B.[11] We plot those fractions for the United States, its counterfactual (i.e., the ω-weighted mean), and the average of countries in the European Union for context. As shown in Panel A, the United States and its counterfactual report nearly identical rates of overall unmet need—until the percentage in the United States drops precipitously with the Medicare eligibility, falling to levels seen in Europe, where coverage is generally universal. As shown in Panel B, that reduction in unmet need comes entirely from erosion of a financial barrier. We reiterate that the similarity in unmet need (prior to age 65) occurs despite the counterfactual being determined only by mortality rates.

Our second demonstration of balance is on a host of related measures taken from the Global Burden of Disease ("GBD") or the International Social Survey Programme ("ISSP"). In Appendix Figure A4, we show the Healthcare Access and Quality Index (Panel A) and Healthcare Coverage Index (Panel B) from the GBD (2020) for the United States, its counterfactual, and the average of other high-income countries. We use the versions that reflect the working-age populations, and we z-score the values by country. The United States and its counterfactual, relative to high-income countries, are very similar in both indices. In Ap-

[11] We compiled this information from a wide variety of sources that are outlined beneath the figure and described in more detail in Appendix Table A1 and Appendix Section A. The concept of unmet need was chosen, rather than insurance rates, as it is more comparable across countries, many of which may not have health insurance play a major role in healthcare delivery. This concept is also highly relevant for our context, as Medicare reduces financial burden on both the uninsured and the *under*-insured. We demonstrate this point in Appendix Figure A3, which shows lower rates of unmet need and delayed care among the Medicare-eligible population than among the near-elderly who are insured. Additionally, because the insured population is much larger, there are more insured individuals *in total* reporting unmet need due to financial reasons than uninsured individuals, despite the disparity in rates (Panel B). As under-insurance due to high cost-sharing has been shown to increase mortality (e.g., Chandra, Flack and Obermeyer, 2024), the improvement in insurance quality is likely a key—and perhaps underappreciated—part of Medicare's benefits.

pendix Figure A5, we plot differences between the synthetic control and the United States (or other high-income countries) in responses to several questions about healthcare from the ISSP (again, z-scored) as well as an index of the various questions (the mean of the z-scores). Although there is variation in the components, these metrics tell a similar story that is captured by the aggregate index. The United States is much more like its counterfactual than other rich countries—prior to age 65. After age 65, these views of the healthcare system, as captured in the index, become decidedly more favorable in the United States, perhaps because of Medicare.

We take these two demonstrations of balance as evidence that the United States and its counterfactual have similar healthcare environments for people younger than 65. That landscape changes dramatically for Americans when they reach 65, but it remains unchanged for their peers abroad as they age, as it should in an appropriate counterfactual. At the same time, there is no reason to believe that access is the only channel through which Medicare may impact population health. It may improve quality or frequency of care, lower the cost among those already receiving care, or simply compel changes in health behaviors for people who will now frequently be labeled "older." We do not intend to separate the mechanisms, although the analyses to follow suggest the primary channels involve medical care. We intend only to establish—for the first time—that Medicare eligibility in the United States marks significant improvements in health seen at the population level in rates of mortality both historically and currently.

4.4 Monetary valuation

Monetizing the improvements provides some useful framing. Although the reductions in mortality are consistently large, the program has always been expensive. It now consumes about $1 trillion annually, and thus requires a large benefit to justify its cost. The additional 6 months of life in the recent era, adjusting the valuation of a statistical life-year from Cutler (2004*a*) for inflation, is worth about $80,000 per person. If we were to apply instead the value used by the U.S. Department of Health and Human Services, this benefit rises to almost $140,000 per person (Kearsley, 2024). The lifetime cost of providing Medicare, extrapolating recent age-specific cost figures from Kaiser Family Foundation (2022) alongside a life table, stands at about $240,000. The arithmetic, then, is that extensions in longevity recover at least one third of the total price.

Population health, however, is not the primary purpose of Medicare. The primary purpose, as President Johnson stated when he signed it into law, is financial. Finkelstein and McKnight (2008) have already estimated that Medicare recoups a substantial portion of its bill, as much as three quarters, by reducing exposure to risk. Three quarters is the high end

of an estimate based on the introduction of Medicare, when private insurance was weaker, but it raises the possibility that Medicare is paying for itself through a combination of risk reductions and health improvements. An analogous estimate from Barcellos and Jacobson (2015), based on a recent time period, suggests that Medicare now recoups "only" about 20 percent of its bill by lowering out-of-pocket costs. In that case, the mortality reduction we estimate for the first time in a modern period *exceeds* the insurance value of Medicare—at a time when healthcare costs have never been higher and when longevity gains appear smallest. Neither of these conclusions nor any in between are credible, however, if our result fails to withstand scrutiny.

5 Robustness

Placebo era

In a series of figures, we carefully consider the robustness of our main results. We first want to verify that, in years when Medicare did not exist, we find no treatment effect. We might find one if there are longstanding differences (predating Medicare) between the United States and other places that set in around age 65. The expected null result, however, is shown in Appendix Figure A6, where age-specific mortality rates track similarly between the United States and its counterfactual in the placebo era (1950-65). For this exercise, we use data from the World Health Organization Mortality Database (rather than the Human Mortality Database) as it has more complete coverage in the pre-Medicare era. The switch leaves us with only five-year age groups. Timing with respect to age is crucial, and we will not be able to unpack the placebo era in the ways we can unpack the other eras in the checks to follow. We also caution that the confidence intervals in the placebo era are wide. At ages 80 through 84, for instance, we cannot rule out the treatment effects seen in the periods when Medicare does exist.

Placebo threshold

We also want to stress *extensively* the location of the break, first to assess the validity of the counterfactual and then to connect the break more firmly to Medicare eligibility. By design, synthetic control matches mortality rates from age 45 through 64. Consequently, the United States, by construction, cannot freely separate from the counterfactual until age 65. One could imagine a scenario in which the synthetic control, despite matching American mortality rates for 20 years, is, in unobserved ways, a bad counterfactual for mortality thereafter. Accordingly, we redetermine the optimal weights using mortality rates only through age 54. If the

counterfactual were not suitable, one might expect the United States to "escape" the synthetic control as soon as it can, at age 55. In Figure 4, we find instead that the new path is very similar to the original, with the departure remaining close to 65.

One exception, however, is the early era. There, it seems that the United States does separate prior to age 65, as if the underlying characteristics of treated and untreated units differ in ways that precede Medicare ages. The implication is that we may not obtain an appropriate counterfactual in the earliest years of the program. We note, however, that the late 1960s opened a remarkably productive period in terms of adult longevity in several parts of the world. In Appendix Section B, we explain why those dramatic improvements make it difficult to find a suitable control group in the data we have. In that appendix, because we believe our international perspective provides important context, we also contrast our results from the early era with those from Goodman-Bacon et al. (2025), a contemporaneous study that leverages cohort variation *within* the United States to estimate the impact of Medicare's introduction on life expectancy. No matter the reason, the flaws in the early era, where the donor pool is thinnest, are instructive. This exercise instills confidence in the other eras because it suggests that the exercise has the capacity to detect problems when they exist.

Retirement age

We dig further into timing of the break with respect to age. Medicare eligibility is sharp at age 65. If the effect sets in just a year or two sooner or later, one might reasonably conclude, based on visual inspection, that Medicare is not driving the effect, but some other transition common among Americans in their early 60s, which is an eventful part of the life course. Although the counterfactual is matched through age 64 (and robust to age 54), we acknowledge that, in the pooled era for instance, one might place the onset of the treatment effect not at age 65, but at age 63. We have several remarks. First, underpinning that placement, age 62 appears elevated, like a "bump" in mortality. Second, there is noise associated with the age-specific estimates. For instance, our pooled sample shows similar bumps at ages 48 and 58. Third, the recent era, where there are fewer fluctuations, shows no bump at age 62. Nonetheless, there may be a meaningful event in the United States that is not witnessed (and therefore not matched) in other countries at age 62. In fact, 62 is the youngest age Americans can claim Social Security, and, as Fitzpatrick and Moore (2018) shows, many people retire at that age, and some of them die as a result.

We suspect that a causal retirement effect of this nature exists at age 62, but with an important caveat. Building on the result from Fitzpatrick and Moore (2018), we argue that the impact of retirement is "harvesting," which manifests as anomalously high risk at age 62 but also anomalously low risk soon after, as if retirement claims, specifically, lives that are

already on the brink. We make this case in Appendix Section C.1 and the accompanying Appendix Figure A7. Briefly, age 62 is anomalously high (and 64 anomalously low) only once the early retirement age became 62 in 1961 (i.e., not in the 1950s). Once age 62 ceases being an anomaly—coincidentally in the early 2000s after the study period in Fitzpatrick and Moore (2018) ends—age 64 also ceases being an anomaly.[12] Our conclusion is that age 63 is not the beginning of the departure between the United States and its synthetic control, despite opposing visual evidence in select eras. We view the beginning as age 65, as is seen clearly in our focal era, the recent era.

Labor force

In addition to pension-motivated retirement, we give labor-force participation and income dedicated attention. Medicare eligibility itself might induce retirement given the strong tie between employment and insurance in the United States.[13] We address these concerns directly in Appendix Figure A8. In Panel A, we consider labor-force participation, which comes from the Luxembourg Income Study ("LIS"). As we did with unmet need, we plot participation by age for the United States and its ω-weighted average of peers in the period for which the necessary data exist.[14] The two series are nearly parallel around age 65, with no clear evidence of a jump or kink at age 65. It therefore seems unlikely that changes in labor-force participation are driving the results we obtain.

Household income

In Panel B of Appendix Figure A8, we plot median household income again for the United States and its counterfactual. This income measure also comes from the Luxembourg Income Study, and it includes proceeds from work, pensions, cash transfers (such as social security), and the monetary value of other non-cash transfers (such as housing and food assistance).

[12] In Appendix Section C.1, we also show that rates of early Social Security claiming rise and fall in tandem with the anomalies.

[13] One might view these incentives to retire as a treatment effect. Though such an impact is theoretically possible, there has historically been little evidence of job lock due to Medicare (e.g., Fairlie, Kapur and Gates, 2016). What is perhaps more probable, but still unlikely, are employment choices related to Social-Security claiming. Such claiming could be concurrent with Medicare, as the full retirement age ("FRA") for Social Security was 65 for cohorts born in 1937 or prior. However, due to the presence of deferred retirement credits, which reward individuals for deferring past the FRA, there are not particularly strong incentives to retire at age 65—and certainly not strong in comparison to the minimum eligibility age of 62, which we addressed already.

[14] The LIS harmonizes work, income, and wealth data across a wide range of countries and time periods. We use information from 1980 onward. Only nine countries have any coverage prior to 1980. See Appendix Section A.3 for further discussion of these data.

We view this household measure of income therefore as the best available metric by which to appraise financial resources in adulthood. Like labor-force participation, median household income appears to trend similarly between treatment and control, albeit at different levels.

In Panel B, we also plot the tenth percentile of household income, a part of the income distribution where mortality might be more responsive to resources (e.g., Chetty et al., 2016). At that level of income, a split does emerge, with low-income families in the United States declining by approximately $2,000 less than those in the synthetic control (adjusted for inflation and purchasing power). That amount is certainly meaningful relative to the overall income levels of this group, but it is unlikely to be driving our effects, given what has been established about the relationship between income and health. Miller et al. (2024), for instance, conducted a randomized control that granted low-income adults $1,000 *per month* and found precise null effects on a wide variety of health outcomes two years later. A recent review on poverty and health by Lleras-Muney, Schwandt and Wherry (2025) states, "Studies analyzing the impact of material resources on adult health have found limited impacts."[15] Nonetheless, we also generate estimates while controlling for labor-force participation and income and find that our results are largely unchanged, as shown in Appendix Figure A9. We will return to this figure shortly.[16]

In a final note on income, we highlight an informative case study where later-life assistance to low-income individuals is particularly generous: Canada. As shown already in Appendix Figure A8, the tenth-percentile of household income in the United States flattens relative to trend around retirement ages, when earned income decreases but is partially offset by social security and other sources of retirement income. As displayed in Panel A of Appendix Figure A10, the same percentile of household income in Canada *jumps* by about $5,000 around age 65.[17] If resources among low-income individuals were indeed the main driver of our results, we should expect Canada then to also show large reductions in mortality at age 65.

[15] This review notes that such a conclusion may not hold over the long run. Schwandt and von Wachter (2023), for instance, find that early-career income shocks take two decades to manifest, which is at odds with the dynamics we find. Additionally, their effects are connected to health behaviors, namely substance abuse, which differs from what we will report on causes of death below. Moreover, studies considering the impacts of income in later life, such as the famous Social Security "notch," are not statistically significant or are modest when they are (Snyder and Evans, 2006; Noghanibehambari and Fletcher, 2026). This qualitative conclusion is reinforced by Schwandt (2018), who finds that a 10-percent decrease in wealth among retirees causes a 1-percentage-point decrease in two-year mortality, which is modest compared to what we find. Furthermore, recent work by Finkelstein et al. (2025) demonstrates that reductions in economic activity can lead to *decreases* in mortality. Again, if income were the central determinant of health, the United States would not be the outlier that it is.

[16] When controlling for variables, we utilize the method outlined in Clarke et al. (2024), where a two-way fixed-effects regression, $y_{ac} = X_{it}\beta + \mu_c + \theta_a + \varepsilon_{ac}$, is estimated using only the untreated units. Those estimates are then used to form a residual outcome, $\widetilde{y_{ac}} = y_{ac} - X_{it}\hat{\beta}$, which becomes the outcome in our baseline method.

[17] We reiterate that this value includes cash assistance and the monetized value of non-cash assistance, such as housing and food subsidies.

This, however, is not the case. As shown in Panel B of Appendix Figure A10, performing our synthetic control analyses with Canada (rather than the United States) as the treated unit yields a smaller and statistically insignificant effect. That reduction fully dissipates, as seen in Panel C, when controlling for income.

Sample restrictions

We provide a battery of robustness checks in Appendix Figure A9, focusing on the stability of the ATT (again, the average of post-65 estimates) as we make changes to our default approach. The figure notes themselves describe what each change entails, but we highlight two groups of them here. First, we restrict the donor pool in several different ways. The idea is to confirm that no one country or set of countries exerts outsized influence on the synthetic control. One at a time, for instance, we drop a country or an entire region of the globe from the sample.[18] In no era do any of these restrictions materially change our qualitative conclusion about the impact of Medicare on mortality. In the few cases where the *quantitative* conclusions change, the estimates become larger. This is true also when we restrict the donor pool only to OECD countries or only to high-income countries. Those estimates are perhaps most noteworthy. Even when compared only to the countries that are most frequently viewed as its peers, the United States shows substantial reductions in mortality.

Alternative methodologies

Second, we implement other methods that are similar to synthetic control, namely synthetic difference-in-differences from Arkhangelsky et al. (2021) and an interactive fixed effects model from Bai (2009). Those methods produce similar and larger estimates, respectively. We also consider alternative methods of inference, including a recently developed technique from Chernozhukov, Wüthrich and Zhu (2021, 2025) as well as bootstrapping. The confidence intervals from these methods are either similar to or narrower than those generated using our main method.

Permutation inference

On inference, we want to further demonstrate how extreme the United States is in terms of its divergence from counterfactual. In an exercise illustrated in Figure 5, we replicate our main result for other countries. Specifically, we drop the United States from the sample,

[18] Regions are based on the United Nations M49 designations, with select adjustments. Namely, we form a separate group for former Soviet states and for the Anglosphere, which appeared to have trends distinct from the groups to which they originally belonged.

assign status as the treated unit to every other country in the donor pool one at a time, and overlay those treatment effects on our main result. Critically, the United States constitutes the outer envelope in every era. That is, no pictured nation sees comparable reductions in mortality in any era since Medicare's inception. Appendix Figure A11 makes this point more directly by highlighting America's position in the histogram of permuted treatment effects (scaled ATTs).[19] This figure, however, reveals another noteworthy feature of our main result. Although the recent era demonstrates the smallest impact in terms of life-years saved, it is by far the most extreme era in relation to peers. This point is seen in the histograms by the distance between the United States and the mass of other nations. In at least one way then, the most current impact of Medicare is the most striking.

Returning to Figure 5, we have annotated select countries to provide perspective. We first annotate countries that demonstrate large divergence from their counterfactual, such as Lithuania in the recent era. None of these countries show large divergences consistently across all eras. We also annotate countries in the Anglosphere, as they might be reasonably viewed as the closest peers of the United States in multiple respects. Canada, which, as outlined, has particularly generous public assistance for older people, appears to show treatment effects. It still does not approach the magnitude of the reductions in the United States, nor do any of the other Anglo countries.[20] This form of permutation inference, however, is but one way to give context for the magnitude of our main results.

Alternative counterfactuals

Our main result is based on a singular optimum, ω, for the problem in Equation 1. We understand that there are many weights that would produce similarly excellent (though slightly worse) fits, and we want to compare those alternatives to the main result, especially the alternatives that are not just trivial perturbations of the original.[21] To discipline this exercise, we generate numerous synthetic controls by applying regularization and variable-selection

[19] Countries with poor matches, as indicated by root mean square prediction error ("RMSPE") in the pre-treatment period, were omitted from Figure 5. In Appendix Figure A11, we follow Kumar and Liang (2024) and include those countries but show the ATTs scaled by pre-period fit. The basis for this scaling is that it punishes "matches" that have pre-treatment trends or even high volatility, as those cases are more likely to spuriously generate large treatment effects.

[20] Figure 5 provides another interesting case study. If one suspects that retirement could drive our results, one might also expect to see Sweden stand out in the middle era. Throughout that time period, Sweden had a mandatory retirement age of 65 (Palme and Svensson, 1999). It does not stand out nor does it receive any weight in our counterfactual.

[21] Hollingsworth and Wing (2022) describes this problem as an issue of overfitting, and we give more background on the process underlying Appendix Figure A12 in Appendix C. The authors also provide a helpful roadmap of key assumptions in synthetic-control designs and standard threats to those assumptions. In Appendix Table A5, we reframe many of our robustness results in terms of this framework.

machine-learning techniques (i.e., Lasso and elastic net) to 1,000 randomly chosen 50-percent samples of the donor countries. The logged mortality rates of the selected countries are used as covariates in the applicable regression. The results of each iteration are displayed in Appendix Figure A12. The results are remarkably consistent with one another and with our main result. We view this consistency as a powerful demonstration: Virtually any control group we can construct that matches mortality rates in the United States prior to Medicare eligibility will yield a similar impact of Medicare on mortality.

Additional robustness

In Appendix C, we consider several other possible sources of confounding. We consider clinical guidelines that could involve age-65 cutoffs (e.g., screening practices) as well as take-up of other social programs that Medicare eligibility might trigger. We also consider models with additional control variables, including age-specific rates of smoking and obesity as well as rates of early-life mortality. Details of these exercises are provided in the appendix. Throughout, we find no evidence of a confound.

Comparable literature

We end our discussion of robustness with a comparison to four recent papers on the relationship between insurance and mortality, each of which took significant strides on the topic, redefining a consensus that insurance had little impact on mortality even in the short run.[22] The comparison we wish to draw is extremely difficult to draw, partially because our goal is to measure something new, a long-term impact on a general population. It is challenging also because the treatments are, as we'll outline, quite distinct. We endeavor nonetheless to offer some kind of benchmark for our findings.

For comparability, we will consider only the recent era and only our short-run impact, which we'll define as the average of treatment effects between ages 65 and 69. That figure is, roughly, a 4-percent reduction in annual mortality. The four papers in reference examine: eligibility for Medicaid (two papers), which is means-tested; the purchase of private plans, among people on the margin of purchasing one; and shifts from lower to higher quality Medicare plans, where shifts are induced by the termination of plans. Each of them, therefore, con-

[22] To be clear, these were not the first papers to find impacts of insurance status on mortality risk (e.g., McWilliams et al., 2004; Sommers, Baicker and Epstein, 2012; Sommers, Long and Baicker, 2014; Borgschulte and Vogler, 2020), but the totality of evidence was more mixed (e.g., Card, Dobkin and Maestas, 2008, 2009; Finkelstein and McKnight, 2008; McWilliams et al., 2007*a*; Polsky et al., 2009; Chay, Kim and Swaminathan, 2012). It is also worth noting that studying such a difficult question typically requires strong assumptions and often strains statistical power (Levy and Meltzer, 2008; Black et al., 2022).

siders a population more "vulnerable" than the general population we study. Accordingly, we expect that our short-run effect will be significantly smaller than theirs.

Miller, Johnson and Wherry (2021) find that the Medicaid expansion under the Affordable Care Act reduced annual mortality, almost immediately upon enactment, by about 9 percent among low-income adults ages 55 through 64. Wyse and Meyer (2025) revise this figure down, with even better data, to about half that magnitude. Goldin, Lurie and McCubbin (2021) find that nudges sent to randomly selected uninsured Americans reduced two-year mortality by about 10 percent among those nudged. Abaluck et al. (2021) find that moving Medicare Advantage enrollees to a plan that is one-standard-deviation higher in quality (defined by the risk-adjusted mortality rate of people on the plan) quickly reduces annual mortality among movers by about 23 percent.[23]

In sum, our priors, although necessarily diffuse, are consistent with the literature. The few papers capable of generating causal estimates with any precision, which examine more potent interventions, indeed generate stronger short run impacts. The contrast also demonstrates the importance of viewing Medicare from the long-term perspective we take. A modest short-term improvement—modest in relation to the literature—that is applied to the full population and is sustained over decades accumulates into substantial gains in life expectancy. Despite the robustness illustrated here, we concede that we cannot disentangle events that happen uniquely in the United States at age 65 from the influence of Medicare. Still, a significant and persistent improvement in the health of Americans relative to their peers around age 65 seems clear from our inquiry, and the set of possibilities appears narrow. To that end, we recall Figure 1. The United States does not just depart from its synthetic control. It appears to be the only country to deflect *downward* (healthier) at age 65 from its prior mortality trend. Given what has now been shown about the causal relationship between insurance and mortality, we would charge just as emphatically that Medicare ought to be regarded as the likeliest candidate, as the explanation to either be rejected or unpacked into constituent mechanisms by future research.[24] The investigation of causes of death to follow seems to corroborate that

[23] An alternative framing is to examine programs that have changed features of Medicare. Huh and Reif (2017) find a 2-percent reduction in the mortality from Medicare Part D. Andersen (2018) finds decreases in kidney disease mortality due to expanded coverage for end-stage renal disease. Additionally, in recent work focusing specifically on Medicare's idiosyncratic cost-sharing features, Chandra, Flack and Obermeyer (2024) and Roberts et al. (2025) demonstrate very clearly the impact of Medicare's expanded access to pharmaceuticals on mortality.

[24] The effects we identify, in a conservative interpretation, might be viewed as mortality impacts of all US social programs that target older adults. For further context on that idea, we note that, when combined, government-provided health insurance, cash assistance (such as Social Security, Supplemental Security, and disability income), and other non-cash assistance comprise approximately 97.0 percent of all such spending. As discussed previously, cash and non-cash assistance are very unlikely to yield the effects that we see, as the U.S. social safety net is not unusually generous, and our estimates do not respond strongly to adding controls for these variables. If one believes our treatment effect is operating primarily through government

position and point to certain pathways.

6 Causes

We now ask which deaths Medicare averts. All analyses to this point involve all-cause mortality, an unambiguous marker of population health. Underlying causes can be difficult to assign and interpret, but they offer a powerful diagnostic tool, one that illuminates potential mechanisms while simultaneously serving as a final robustness check. In Figure 6, we split causes of death several ways, but we first review a few caveats. Broadly, the cause-specific data are more limiting than the all-cause data. We will restrict attention to the period from 1980 onward. If we were to consider earlier years, we would have few countries from which to generate counterfactuals.[25] To maintain precision as we disaggregate deaths, we will pool the two eras in this span, the middle and recent eras. Also, we have only five-year age intervals for cause-specific mortality, so, in Figure 6, the horizontal axis is coarsened.[26] We recall lastly that, for each type of death (for each panel in Figure 6), we will find suitable synthetic controls using trends only in that category.

We first split causes into those that are "amenable" to healthcare and those that are not in Panels A and B, respectively. The delineation follows from Nolte and McKee (2003), who, in describing amenable deaths, state, "Deaths from certain causes should not occur in the presence of timely and effective health care." The delineation is certainly subjective, but, if Medicare is to lower mortality, one might expect amenable deaths to be most responsive. Critically, the distinction cuts through many broad categories. It is not simply internal versus external or cardiovascular versus all else, though we consider those classifications next. To illustrate, death from hypertensive heart disease (ICD-10 I11) is amenable. It is slow to develop, easy to diagnose, and potentially reversible (for instance, with statins). Death from cardiac arrest (I46), and its largest subcategory, "Cardiac arrest, cause unspecified" (I46.9), is not amenable. Though it is treatable, it is typically an emergent event where the under-

intervention other than Medicare and those assistance programs, that leaves approximately 3.0 percent of spending to find the confounder. Of this, care through the Veterans Health Administration ("VA") totals an additional 2.9 percent. Given that veterans are overwhelmingly male, if VA spending were responsible, we would expect the post-65 effects on the male population to be much larger than those for women. We will find that they are actually smaller. The remaining budget, which is mainly funding through the Older Americans Act, amounts to roughly $48 of spending per adult aged 65 and older. It is therefore extremely unlikely to serve as a serious confounder.

[25] We would also have to map ICD-7 and ICD-8 codes to ICD-9 codes, which is not straightforward given that ICD-9, adopted in the late 1970s, reflected a significant advance in the taxonomy of disease.

[26] This resolution is a property of both the HMD and the WHOMD. We use data from the WHOMD for our primary analysis because it has more complete coverage of countries with cause of death. However, we consider robustness to using the HMD in Appendix Figure A13 and find similar results.

lying causes are not clear and often attributable to lifestyle and genetics. Among cancers, death from cervical cancer (C53) is amenable. Modern medicine provides many avenues for screening and treatment (i.e., secondary and tertiary prevention). In contrast, death from lung cancer (C34) is not amenable. Avoidance is achieved instead through patient behavior, namely smoking cessation (i.e., primary prevention).

In Figure 6, we see that deaths from amenable causes (Panel A) decrease quickly and substantially, while those from non-amenable causes (Panel B) do not. Amenable causes are down almost 10 percent in ages 65 through 69 and down by almost 30 percent in ages 80 through 84.[27] The confidence intervals allow for effects of meaningfully different magnitude, but they clearly reject null results. Non-amenable causes remain virtually unchanged relative to the counterfactual through age 74. There are drops of about 5 percent by more advanced ages (and not sooner), but they are statistically indistinguishable from zero.

Critically, the impact of Medicare on mortality loads onto amenable causes of death despite the fact that amenable deaths account for only about one third of all deaths. This approximate ratio holds at ages younger than Medicare eligibility and in other countries, and we summarize mortality rates and shares by cause in Appendix Table A3. The reduction in mortality concentrating in causes that "should not occur in the presence of timely and effective healthcare" is consistent with Medicare—rather than some unobserved shock at age 65 or some unobserved difference between the United States and its synthetic control—as the driving force behind older Americans' improvement. If, for instance, the counterfactual group were in poor latent health relative to the United States all along, one might expect to see "reductions" in a wider variety of causes.

In Panel C through E of Figure 6, we partition causes of death into more objective categories, based on broad ICD-10 "chapters" that we order by frequency. In Panel C, we consider mortality associated with cardiovascular disease, which accounts for roughly 40 percent of all deaths among those age 65 and older. We find that mortality rates from cardiovascular disease fall by nearly 25 percent by ages 80 through 84, relative to the mortality rate we would have expected without Medicare.

In Panel D, we consider deaths from cancer, which comprise about one quarter of deaths ages 65 and older. Cancer mortality rates fall by about 10 percent by ages 80 through 84. Though statistically indistinct from zero, this estimate represents a striking reduction given

[27] Nolte and McKee (2003) intend their definition to apply only to deaths before age 75, partly inheriting convention from earlier efforts to classify causes as avoidable or not, (e.g., Rutstein et al., 1976; Charlton et al., 1983). Their premise was that amenable mortality was fundamentally about premature death and that deaths at or after age 75 do not qualify. We take a different view. Life expectancy (no matter the reference age) has increased substantially since their work, and, even in the 1970s, someone age 75 could expect to live more than a decade. However, even if we were to ignore older ages, mortality from amenable causes shows a statistically (and economically) significant reduction already by ages 70 through 74.

that cancer may have a strong genetic component and some cancers are tied to health behaviors, such as smoking, that are often set early in the life course. The confidence intervals, however, do contain zero, including the confidence interval around the ATT. At the same time, if we instead perform inference using methods developed by Chernozhukov, Wüthrich and Zhu (2021, 2025), our estimates achieve significance at the standard 5-percent level.

In Panel E, we consider respiratory illnesses, almost all of which are influenza, pneumonia, or chronic obstructive pulmonary disease ("COPD") in the periods and ages we study. Deaths from such illnesses appear to decrease, though not immediately. They are down by almost 20 percent for ages 80 through 84. However, the confidence intervals are wide, so wide that we truncated them for aesthetic purposes. Even the aforementioned alternatives for standard errors fail to achieve statistical significance at the 5-percent level at the most advanced ages. If the impact were genuine, we would caution anyway that respiratory illness, pneumonia in particular, is a common *final* cause of death and that final and underlying causes are commonly conflated. The practical implication is that a reduction in respiratory illness may reflect the decreases we have already shown. Again, the categories in Figure 6 are chosen because they are most common at ages 65 and older, not because of any priors we carry.

In Panel F, we consider external causes of death, most of which are unintentional accidents, such as falls and car accidents. One might therefore expect this kind of mortality to be less responsive to the aid that Medicare entails. The confidence intervals are again wide (regardless of method), but we see no indication of a change in external causes.[28]

These four categories (Panels C through F of Figure 6), while encompassing 82 percent of all deaths, are not the full partition of causes. The next four largest categories, comprising approximately 9 percent of deaths, are shown, for completeness, in Appendix Figure A14. We caution that there is even less precision on those (rarer) causes than is seen already, and we must resort to synthetic difference-in-differences to bring select causes back into the convex hull of the donor pool.[29] But we note here that we see little evidence of decreases. If anything,

[28] External causes are potentially—but not necessarily—a placebo test. Medicare could conceivably reduce their likelihood, for instance, through orthopedic interventions that improve balance and subsequently reduce falls. On the other hand, it could increase them if it improves health in a way that increases physical activity (or driving) altogether. We reiterate that cause-specific mortality can be difficult to interpret. In fact, we will soon show that self-reported rates of physical difficulty drop in the United States (relative to counterfactual) after age 65. The lack of a "corresponding" reduction in external mortality, almost half of which are falls, could be mechanical. The decreases in mortality from the causes we have already shown could result in deaths from less treatable causes.

[29] Synthetic difference-in-differences raises the probability of a pre-period match, as it de-means treatment and control units to net out unit-specific and age-specific effects prior to matching on pre-treatment trend. While this is desirable in some settings, we do not utilize it as our main method because it induces matches between units with different levels of mortality. As noted, if those level differences in mortality span the early part of the life course, concerns with selection and scarring arise (although we doubt they would arise sharply at

we see some increases, which could be consistent with displacement, wherein avoiding one death by one cause at an earlier age leads to mortality from another cause of death later in life. In fact, all the cause-specific dynamics we have shown reflect the net of Medicare's impact against such displacement.

The variation by cause of death that we document, overall, could be consistent with several different explanations, but it is at least consistent with advances in medicine over the last few decades. Cardiovascular disease, where the impact is largest, has been greatly diminished, in large part due to the development of improved medical practices and key pharmaceuticals (Cutler, 2004*a*). Cancer, where we see indications of a smaller but important impact, has seen remarkable improvements in early detection, rendering once-fatal conditions nonfatal. Respiratory illness is perhaps too varied (and too speculative based on confidence intervals) to interpret, but external causes, where a connection to adequate healthcare seems weakest, appear far less responsive than the other categories.

7 Heterogeneity

While the impact of Medicare is confined to select types of deaths, it is pervasive across demographics within the United States. We show here that the impact is present for both men and women, for both white and Black Americans, and in every region of the country. We also show, however, that the effect is uneven. This heterogeneity is displayed in Figure 7, focusing on the pooled period for parsimony.[30]

In Panel A of Figure 7, we split men and women. The Human Mortality Database includes sex-specific mortality rates for all years in every country, and we generate counterfactuals for each sex using only the mortality rates of the corresponding sex in other countries.

An effect is present for both sexes, and the effect builds with age for both. The impact of Medicare is, however, decidedly larger for women than for men. Women appear to gain almost 9 months while men gain about 5 months in life expectancy at age 65. Women typically live longer (about 19 versus 16 years conditional on survival to age 65 in the pooled period), but these effects are clearly not proportional. We refrain from a determination as to why women benefit more than men, but the heterogeneity is consistent with women utilizing more healthcare than men upon eligibility. We provide some evidence of that utilization pattern in Panel C of Appendix Figure A3, where we return to unmet need. Women show a larger reduction than men, about twice as large, around age 65. However, as with our main

age 65).

[30] We will, however, for completeness, show the effects by era in appendix figures. Sex, race, and geography are shown in Appendix Figures A15, A16, and A17, respectively.

result, the impact of Medicare must extend beyond those with unmet need. We also note that, if employment or income were driving the effects we've recorded, one might expect the reverse—larger changes for men than for women (e.g., Fitzpatrick and Moore, 2018; Chetty et al., 2016). No matter the reason, the difference we document implies that Medicare is partly responsible for the female mortality advantage—which has not always existed (Goldin and Lleras-Muney, 2019).

In Panel B of Figure 7, we split white and Black Americans. The Human Mortality Database does not provide race-specific mortality rates, so we instead use the Multiple Cause of Death ("MCOD") files from the National Vital Statistics System. The ways in which federal and state entities racialize people have changed over time, and, for much of the pooled period, they did not elicit "ethnicity," meaning we cannot separate people who would have been marked as Hispanic. That is, Panel B is based only on "race" categorized as Black, white, and non-white.[31] There is no reason to conceptualize an analog to the Black population in other countries. We treat the white and Black populations as distinct treated units and generate separate counterfactuals from the original donor pool of (whole) countries.[32]

In Panel B, we see that the series for white mortality obtains a close match, as differences from the counterfactual hover around zero prior to Medicare eligibility. The series for Black mortality does not. Its differences from counterfactual are uniformly positive. The reason is that no other country available through the entire sample period has mortality rates as high as Black Americans, prior to age 65.[33] Nonetheless, the dynamics around age 65 are similar, although estimates for Black Americans are clearly noisier given the relative sample sizes. For both, we see rapid and persistent decreases upon Medicare eligibility, indicating that Black Americans are not left out of the gains from Medicare. In fact, we see larger gains for them. Their implied life-years saved are about 12 months compared to about 7 months for white Americans, and their gain is on a shorter base. Over the period we examine, Black Americans could expect to live about 18 fewer months than their white counterparts conditional on survival to age 65. That gap has been closing, however. Given the relative treatment effects,

[31] Non-white consists of Black and "other," the latter of which comprise only about 3 percent of deaths, making it too small to study separately. Regarding the classification of ethnicity, we are able to separate non-Hispanic white individuals during our middle- and recent-eras, and we note that our all-ethnicity white ATTs differ by, at most, 0.01 from the non-Hispanic white estimates.

[32] Because they may be instructive, we show the optimal weights by race in Appendix Figure A18. Notably, weights assigned for white Americans are concentrated on higher-income countries with, for instance, Canada garnering the largest weight in the pooled period. For Black Americans, more weight is placed on Eastern European countries, which are among the lower-income countries in the HMD.

[33] If we relax the requirement that donors be present throughout a study period, New Zealand's Maori population, which ceased being a distinct entity in the HMD in 2008, reenters the sample. Perhaps telling, their inclusion brings the mortality rates of Black Americans back into the convex hull. In this case, our estimates are, as expected, approximately zero in the pre-period, and we obtain an ATT of 0.20.

we estimate that Medicare—which President Johnson purposefully designed to reduce racial discrimination by healthcare providers—is causing some of the convergence between Black and white Americans in elderly longevity.[34]

In Panel C, we consider geography, which we define by the four Census regions of the United States. Again, we rely on the Multiple Cause of Death data, and we match each region as its own treated unit (as we did with race). The impact of Medicare appears pervasive across space. Effects of similar magnitude exist in the Northeast, Midwest, South, and West. The estimates are not identical but not statistically distinguishable either. The biggest differences in mortality after age 65 appear to be, not within the United States, but *between* the United States and its peers.

8 Morbidity

Although our focus is on mortality, we wish to understand Medicare's influence on morbidity as well. It provides vital context for the increases we find in longevity. If Medicare mainly prolongs the lives of individuals who are ill, it may raise rates of morbidity, and the life-years it adds would be of low quality. If Medicare instead extends the lives primarily of individuals who are well, it may decrease morbidity, and the life-years it adds would be of higher quality. Morbidity is also a result on its own. Medicare may simply remedy or prevent certain ails.

Assessing morbidity, however, is difficult. Cross-country data on chronic conditions are widely available, but they are based on diagnosis, rather than an individual's underlying health. Because diagnoses depend on healthcare access—which rises sharply for Americans at age 65—a rise in chronic conditions upon Medicare enrollment would be neither unexpected nor indicative of harm.

Accordingly, we turn to self-reported questions about disabilities and functional limitations. Like chronic conditions, they carry large implications for quality of life, but, unlike chronic conditions, they are, for a given level of mortality, unambiguously bad. These measures of morbidity do present difficulties, however. Many so-called disability questions are subjective. Cross-country comparisons, for instance, may reflect differences in cultural norms as much as differences in health status.[35] To alleviate this concern, we focus on a set of ques-

[34] Medicare would "pay" providers through federal reimbursements. Under Title VI of the Civil Rights Act of 1964 (which President Johnson also passed), any provider that practiced racial discrimination would be ineligible for those payments.

[35] For example, according to the International Social Survey Programme, roughly 13 percent of American respondents ages 40 and older report being in "excellent" health, approximately three times more than the average of other high-income countries surveyed. In sharp contrast, life expectancy at age 40 in the United States is about 3 years *shorter* than it is in those high-income countries.

tions produced by the Washington Group on Disability Statistics ("WG") that were specifically designed for international comparability. These questions focus on objective limitations, such as trouble getting dressed or taking a bath. The exact text of these questions, along with information regarding the underlying data sources brought together for this analysis, is included in Appendix Section A.2. Summary statistics are included in Appendix Table A4.

In certain data sources, namely the European Union Statistics on Income and Living Conditions ("EU-SILC") Health Supplement and the European Health Interview Survey ("EHIS"), only ten-year age intervals were available in the public releases, which forced two adjustments to our methodology. First, we extended the pre-period back to age 35 to increase the number of periods for our matching procedure. Second, we utilized synthetic difference-in-differences, rather than traditional synthetic control, as it generally performs better with fewer pre-treatment matching periods. Throughout, we z-score each outcome (using the age-specific mean and standard deviation of untreated units), and, when appropriate, we combine similar measures into indices, as popularized by Kling, Liebman and Katz (2007).

In Figure 8, we partition difficulties into three categories: physical, cognitive, and sensory. Physical difficulty is defined by difficulty with mobility, which is framed as trouble walking or climbing stairs, or difficulty with self-care, which is typically framed as trouble dressing or bathing. Cognitive difficulty is defined as difficulty remembering, concentrating, or making decisions. Sensory difficulty is difficulty seeing or hearing, which is assessed with corrective lenses and hearing aids. We choose these aggregations as a balance between parsimony and preservation of the distinct natures of the disabilities, but we show the individual components in Appendix Figure A19.

In Panel A, we see that physical difficulty decreases substantially relative to counterfactual. For ages 75 and older, rates are down by about one standard deviation. Translating the magnitude, the prevalence of physical difficulty falls almost 30 percent relative to counterfactual.[36] In Panel B, we find a decrease in cognitive difficulty that is also large in magnitude but not one that can be distinguished from zero. In Panel C, we see that sensory difficulty is nearly unchanged, but the confidence intervals do not rule out meaningful changes.

Focusing on mobility difficulty, which is by far the most prevalent of the individual difficulties we consider, a straightforward calculation assigns an approximate dollar value to the reduction. We first use our age-specific reductions to estimate the number of limiting conditions avoided. We then multiply those counts by adjustments from Shaw, Johnson and Coons

[36] A one-standard-deviation change in the physical difficult index (post-65) is associated with a change in the rate of mobility difficulty of 8 percentage points and a change in the rate of self-care difficulty of 3 percentage points. When dividing by the corresponding means (from Appendix Table A4), we obtain a 29-percent decrease in mobility difficulty rates and a 28-percent decrease in self-care difficulty rates.

(2005) that translate health states into quality-adjusted life-years ("QALYs").[37] We further multiplied that figure by the expected number of life-years lived in the 65-through-74 and 75-plus age intervals and applied the inflation-adjusted value of a statistical life year from Cutler (2004*a*). The result is that improvements in mobility alone are worth about $30,000 per beneficiary. This valuation is equal to about 40 percent of the increase in longevity that Medicare provides. If we assume that longevity and quality gains are separable—which is a strong assumption but does provide an informative bound—we obtain an impact of Medicare worth $110,000 per person ($80,000 from longevity).

However, we report these monetary gains mostly for context and completeness. Given the coarseness of our data and the size of our confidence intervals (not to mention the difficulty in defining quality adjustments), we are hesitant to put much stock in any quantitative takeaways with respect to quality of life. Instead, we highlight two *qualitative* conclusions that surface in our analysis of morbidity. First, we rule out worsening physical health (as captured by difficulty with mobility and self-care). The implication is that Medicare is, perhaps partly, but not primarily, extending the lives of people who are in poor health. Again, if Medicare were rescuing only frail individuals, we would see *increases* in rates of physical difficulty, and we do not. Second, the effects are strongest where we expect them to be strongest if Medicare is indeed the mechanism underlying health improvements that set in around age 65 in the United States. There are many health problems that could both cause physical difficulty and subside (or never occur) with treatment—arthritis or stroke, to name just a couple. There is less capacity for healthcare to remedy sensory difficulties, some of which are set at birth, all of which are assessed *with* hearing aids or corrective lenses. We conclude that Medicare is increasing, not just lifespans, but healthspans.

9 Policy

Enrollment in Medicare has grown from nearly 20 million upon inception to 70 million today, and spending on Medicare now accounts for almost 4 percent of gross domestic product. Many are calling to expand the program further. President Joe Biden, for instance, proposed lowering Medicare's eligibility age to 60 during his final campaign (Kaiser Family Founda-

[37] To be precise, we use the EuroQol five-dimension instrument ("EQ-5D"), a widely used preference-based measure of quality of life that maps each health state onto a utility score anchored at zero (death) and one (full health); the difference between utility scores defines QALYs, the standard unit for comparing health across conditions. The five EQ-5D domains map closely onto the WG questions underlying our index, with the mobility and self-care dimensions corresponding almost perfectly. We apply the utility decrements from Shaw, Johnson and Coons (2005) corresponding to "some problems" (which they term "Level 2") on each dimension.

tion, 2021).[38] Others are pushing to curtail an already expensive program. Recent proposals have suggested raising the age threshold to 70 and then indexing it to life expectancy (Picchi, 2022).[39] If Medicare were to not only reduce risk but improve health outcomes as we see in this paper, the relative merits of such proposals would change substantially. Therefore, we close our inquiry by asking how many life-years might be gained or lost if Medicare's age-eligibility threshold were changed.

Quantifying the ramifications of these changes is not straightforward, however. Simply "shifting" our estimates, for example, forward to younger ages (e.g., applying our age-65 treatment effect to age 60, our age-70 treatment effect to age 65, and so on) would understate the impact of Medicare because treatment effects would deteriorate earlier than they should naturally. Conversely, shifting our estimates backward to older ages would overstate the impact because the deterioration in treatment effects would be artificially delayed.

To handle these kinds of issues, we impose additional structure on our treatment effects. We parameterize them with specific functional forms for what we term "exposure," the accumulating protective effect of Medicare eligibility, and "age fade," the background aging process that must eventually overwhelm any intervention. In some cases, we allow $\tau(a)$ to vary based on health stock, which is proxied by cumulative survival of the cohort to the reference age.[40] We consider multiple such functional forms to illustrate sensitivity (or insensitivity) to our structural choices. That variety is discussed further in Appendix Section D. We show our preferred specification in Equation 4:

$$\tau(a) = \underbrace{\beta}_{\text{Slope}} \times \underbrace{[S_a]^{\gamma}}_{\text{Survival}} \times \underbrace{\frac{1}{1+e^{-\phi(d-d_0)}}}_{\text{Exposure}} \times \underbrace{e^{-(a/\lambda)^k}}_{\text{Age fade}} \tag{4}$$

where a is age, years of exposure is represented by $d = \max\{0, a-65\}$, and S_a is cohort survival (i.e., the fraction of a cohort alive at age a). Our exposure function is logistic and is preferred because it models a reasonable pattern for health investments. Services such as prevention and screening may matter little immediately, but consistent access to care may exhibit compounding effects over time. However, the growing benefits from exposure cannot carry on indefinitely, and the bounded nature of the logistic function captures those dimin-

[38] There are also, of course, proposals of "Medicare for All," which have been suggested most famously by Senator Bernie Sanders. Evaluating this particular proposal is outside the scope of this analysis.

[39] We note, however, that life expectancy has been approximately stagnant in the United States for more than a decade.

[40] These data are obtained from the Social Security Administration, which publishes cohort life tables back to 1900.

ishing returns. The age-fade component is represented by a Weibull function that is used often in survival analysis and is suitable for our purposes.[41] We estimate the parameters for the model using nonlinear least squares. Because they are most pertinent to current debates, we consider only estimates from the recent era.

The results of this exercise are displayed in Figure 9. In Panel A, we plot our age-specific effect (dots), with the age horizon now extending to 100. The plotted lines represent the model fit (for age 65) and counterfactual impacts of Medicare if the eligibility threshold were changed to age 55, 60, 70, or 75. Analogous graphs for other parameterizations are displayed in Appendix Figure A20. We call attention to the fact that the parameterization fits our main result well.

Panel B of Figure 9 displays the gains (or losses) in life expectancy *relative to* the current eligibility threshold of age 65. We calculate that lowering or raising the Medicare cutoff by five years would result in 0.21 life-years gained (age 60) or 0.20 life-years lost (age 70) per person. These estimates are stable across our different parameterizations. Based on the 2020 Census, at present, about 4 million people age into Medicare every year. Our preferred parameterization suggests then that moving the eligibility age by five years would result in 840,000 life-years saved (age 60) or 800,000 life-years lost (age 70). Evaluating more dramatic adjustments, we caution that the strength of the assumptions increases, but our preferred parameterization suggests that moving eligibility up to age 55 would save 1.68 million life-years per cohort, while moving it back to age 75 would sacrifice 1.44 million life-years. In our most conservative estimates, these numbers fall to 1.16 million life-years saved and 1.24 million life-years lost, respectively (as seen in Appendix Figure A21).

Of course, any discussion about modifying the age of Medicare eligibility will consider the costs of doing so. Given that Medicare's average spending for an age-66 beneficiary is approximately $10,000 (Kaiser Family Foundation, 2022), reducing the eligibility age by even five years would surely have an immense impact on federal outlays.[42] If one assumes that individuals age 60 to 64 cost 95 percent as much as their age-66 counterparts, which is the ratio of expenditures we found in the Medical Expenditure Panel Survey, reducing the eligibility threshold to age 60 would result in approximately $200 billion in additional federal spending per cohort.

Whether this cost exceeds its benefit depends, in part, on the value of a statistical life-year ("VSLY"). Updating the oft-used $100,000 estimate, for instance, from Cutler (2004*a*) to 2023 dollars, for comparability, gives a value of approximately $160,000. With this valuation,

[41] More information on the parameters is provided in Appendix Section D.

[42] We elect not to use the figure from age 65 because it may be a particularly expensive age, as pent-up demand is satisfied.

lowering Medicare eligibility to age 60 would provide roughly $130 billion in benefits. If one were to instead use the VSLY from Kearsley (2024), the benefit rises to about $230 billion, easily exceeding the additional government expenditure. That is to say nothing of decreases in morbidity.

However, this hypothetical ignores the fact that individuals covered through public insurance (typically) do not require insurance from other sources, reducing the fiscal burden on themselves, their employers, their state governments, and other federal insurance programs. To understand how overall consumption of healthcare changes upon Medicare eligibility, we assembled spending data by age across several countries and performed a synthetic difference-in-differences analysis around the threshold.[43]

As shown in Appendix Figure A22, per-person spending decreases slightly relative to the counterfactual at age 65. This reduction in spending associated with Medicare should not be viewed as surprising. The prices paid by non-governmental insurers have long been recognized as the primary culprit for elevated health spending in the United States (e.g., Anderson et al., 2003; Anderson, Hussey and Petrosyan, 2019), and Medicare reimbursement is much lower than private insurance payments for the same services. Wallace and Song (2016) show clearly that costs per beneficiary decrease substantially when individuals age onto Medicare and that the decrease is solely attributable to prices. This finding, although easy to lose against the backdrop of a trillion-dollar annual bill, is critical for policymakers. It suggests that, although expanding Medicare would increase government spending substantially, it would likely *reduce* overall spending.

We wish to stress, however, that these conclusions are drawn from partial-equilibrium effects, which (purposefully) hold many things constant that would not be held constant in the event of broad reform. This is a serious limitation, given Medicare's immense influence on healthcare in the United States. Expanding Medicare, for instance, has been shown to have meaningful general-equilibrium effects on healthcare provision, such as hospital access (e.g., Finkelstein, 2007) and drug innovation (e.g., Lakdawalla and Sood, 2009). However, while reducing the Medicare eligibility age would likely increase the quantity of healthcare consumed, it is not clear whether the increase in quantity would offset lower prices received by providers. If expanding Medicare were to threaten the profit margins of healthcare providers, they may respond by reducing access or reducing quality (e.g., Cutler, 1998; Wu and Shen, 2014). Accordingly, government prices might need to rise in order to stabilize the market after any meaningful decrease in the age of Medicare eligibility.

[43] We utilize the SDID method, as per-capita spending in the United States is the highest in the world and thus is not within the convex hull of donor units. However, simply demeaning (log) spending as of age 64 and performing a standard synthetic-control analysis yields similar results. Appendix Section A.4 provides more detail.

On the other hand, *increasing* the age threshold could have ripple effects through private insurance markets. It is likely that some individuals would remain on private insurance, perhaps due to job lock (e.g., Johnson, Davidoff and Perese, 2003; Dague, DeLeire and Leininger, 2017; Wettstein, 2020), while many retirees would need to purchase insurance through exchanges (under the Affordable Care Act, or "ACA"). Increasing the number of older (i.e., higher average-cost) enrollees in these private insurance markets could push prices up and coverage down, even among people too young for Medicare (Dickstein et al., 2015; Orsini and Tebaldi, 2017; Tebaldi, 2025). This cascade could be particularly salient in the ACA marketplace, where the degree of age-based price discrimination is capped, inducing a potentially large increase in the degree of adverse selection. Conversely, reducing the eligibility age could have the opposite effect by lowering prices and reducing adverse selection.

All told, we stress that general-equilibrium considerations could dampen—or amplify—either the costs or the benefits that we have documented throughout this paper. Even so, our results suggest that there are realistic cases in which Americans live longer without spending more.

10 Discussion

The creation of Medicare in 1965 ushered in a new regime of healthcare provision in the United States, but it also marked the end of a long legislative road. The idea of universal coverage was first considered in earnest by President Harry S. Truman at the end of World War II, a time when many other wealthy countries would nationalize healthcare. These proposals were robustly debated by economists (e.g., Campbell and Campbell, 1952; Falk, 1952; Netzer, 1952) but also met staunch resistance, including strong objections from the American Medical Association (Alsan and Neberai, 2026). Though President Johnson had hoped to bring the "healing miracle of modern medicine" to all, in order to garner the support needed to pass Medicare through Congress, he restricted its benefits to older Americans.

From the compromise, however, comes an opportunity to understand whether Medicare improves the health of its beneficiaries as President Johnson laid out 60 years ago. Ever since, the United States has been the only country in the world to begin universal coverage at age 65. Exploiting the uniqueness, we compared Americans to their international peers and found that their mortality rates improve sharply relative to those peers upon eligibility. Critically, a clear and large advantage holds from the program's earliest years to today, and it is driven by reduction in deaths thought most preventable in the presence of proper healthcare. The impact of Medicare on mortality is large enough to dramatically change the welfare implications of one of the world's largest social-insurance programs.

Though understanding the past and present benefits of Medicare is our central contribution, the *future* of Medicare is also of immense national interest. Upon its passage, President Johnson repeatedly voiced an expectation that Medicare, through subsequent acts of Congress, would eventually extend to the entire population (Califano, 2008). That wish has not come to fruition, but calls to expand the program have not subsided and have perhaps intensified in recent years. We therefore use our findings to forecast the change in life expectancy that the United States might witness under younger and older ages of eligibility. We reason, under defensible assumptions, that expansions of Medicare would extend the lives of Americans without increasing what they already spend in aggregate on the healing miracle of modern medicine.

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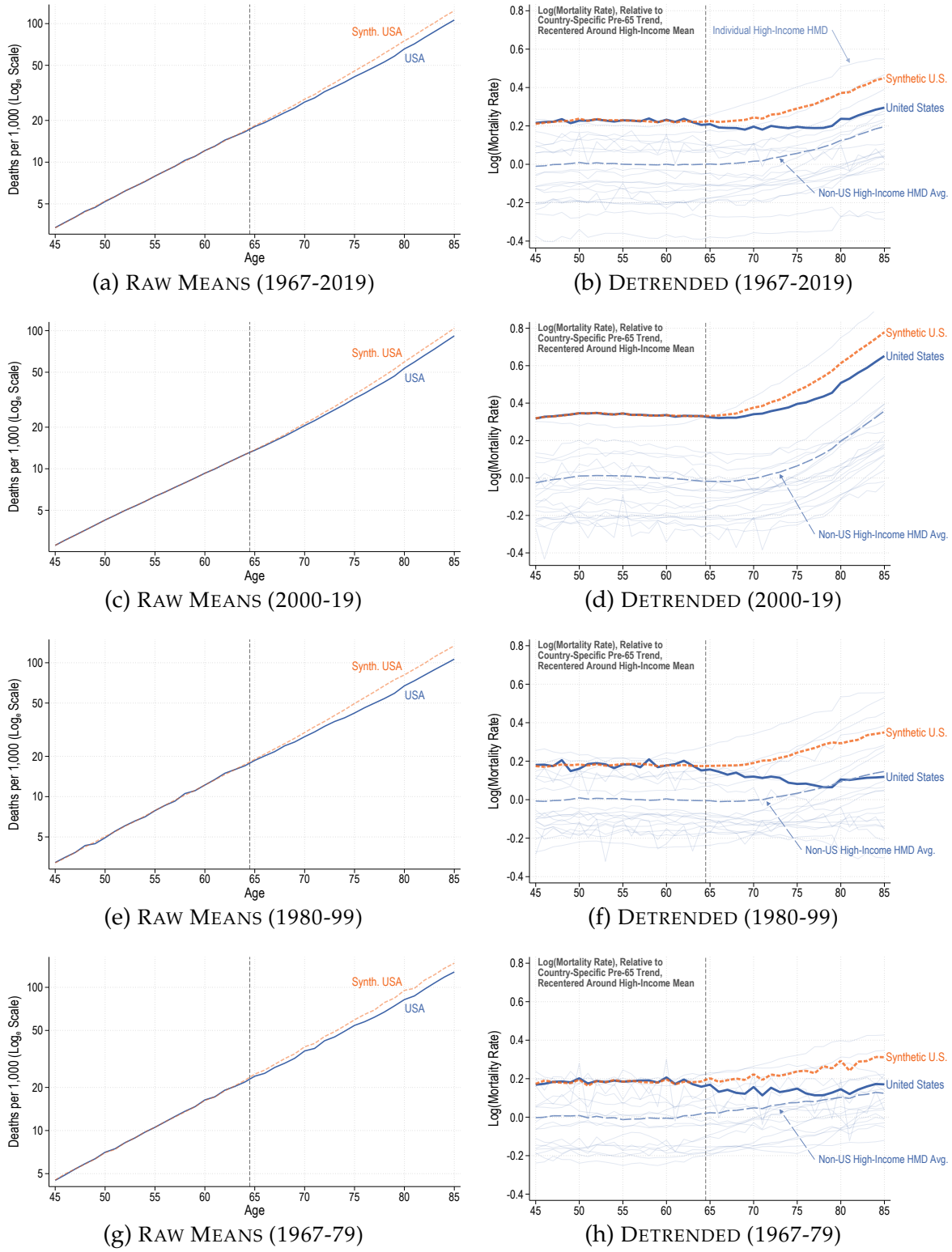
Figure 1: THE UNITED STATES' MORTALITY DEPARTS FROM ITS SYNTHETIC COUNTERFACTUAL AND FROM GLOBAL TRENDS AFTER AGE 65



(a) RAW MEANS (1967-2019) (b) DETRENDED (1967-2019)

(c) RAW MEANS (2000-19) (d) DETRENDED (2000-19)

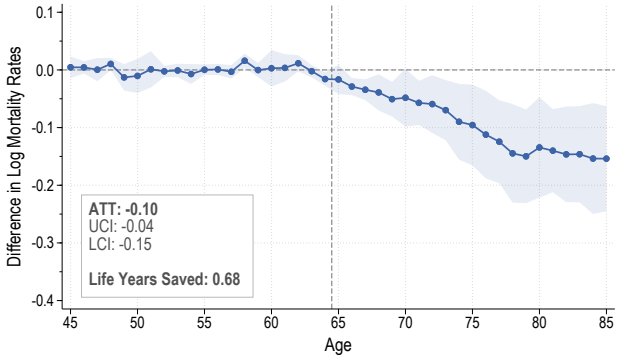
(e) RAW MEANS (1980-99) (f) DETRENDED (1980-99)

(g) RAW MEANS (1967-79) (h) DETRENDED (1967-79)

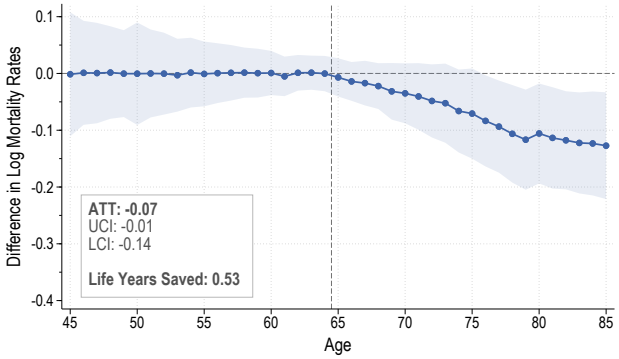
Notes: The purpose of this figure is to display the trajectories of the log mortality in the United States and its synthetic counterpart. The left column presents raw means, while the right column presents adjusted means for the U.S., its synthetic counterpart, and other high-income countries within the HMD sample, which are detrended accord to each nation's pre-65 trend and recentered relative to the (non-U.S.) high-income country mean. For consistency, countries were classified as "high-income" if they have been designated as such since the World Bank began publishing these classifications in 1987.

Source: Author calculations using the Human Mortality Database (2026)

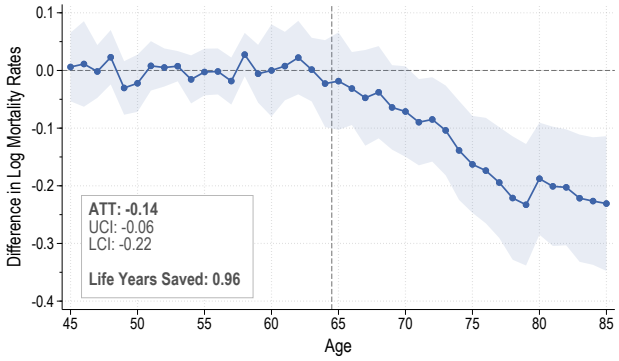
Figure 2: MEDICARE DECREASES MORTALITY IN EVERY ERA



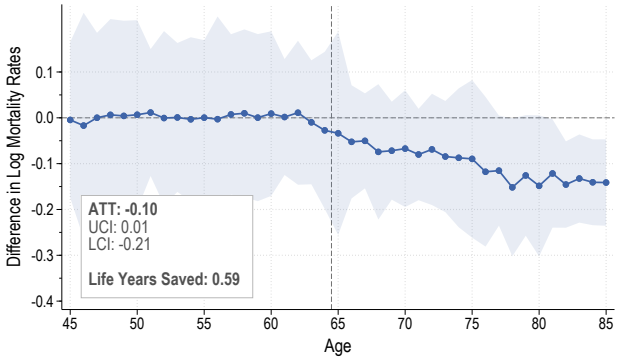
(a) POOLED (1967-2019)



(b) RECENT ERA (2000-19)



(c) MIDDLE ERA (1980-99)

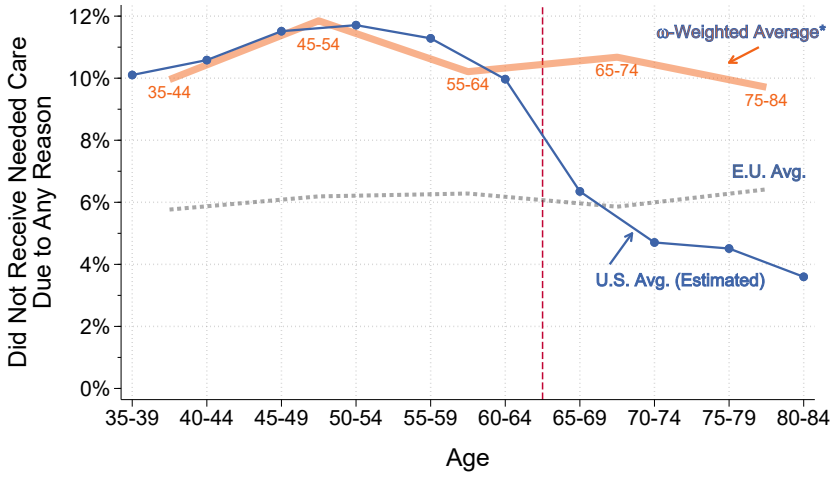


(d) EARLY ERA (1967-79)

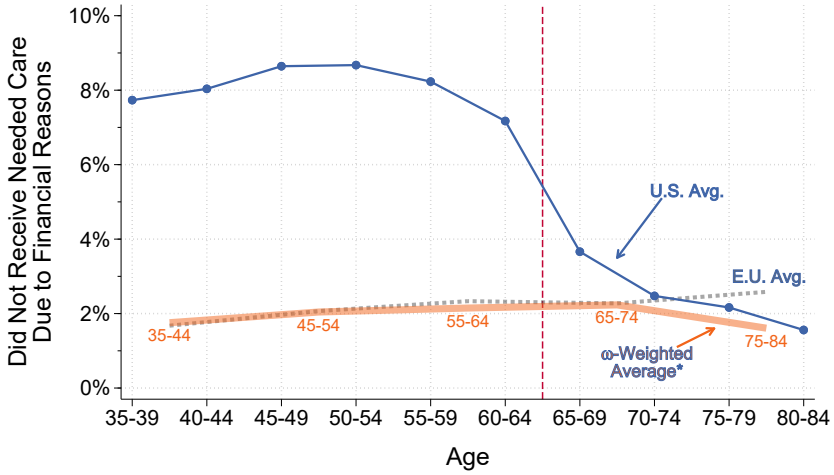
Notes: This figure demonstrates the way in which mortality in the United States departs from synthetic cohorts during the ages 65-85. Within the figure, the point represent estimated differences from the synthetic control group, with 95% confidence intervals calculated using methods from Clarke et al. (2024), discussed in Section 3. Life years saved were calculated by plugging estimates into a life-table. See Section 4 for further discussion.

Source: Author calculations using the Human Mortality Database (2026).

Figure 3: THE U.S. AND SYNTHETIC CONTROL HAVE SIMILAR LEVELS OF OVERALL UNMET PRIOR TO AGE 65, AFTER WHICH U.S. ACCESS IMPROVES



(a) UNMET NEED DUE TO *Any* REASON



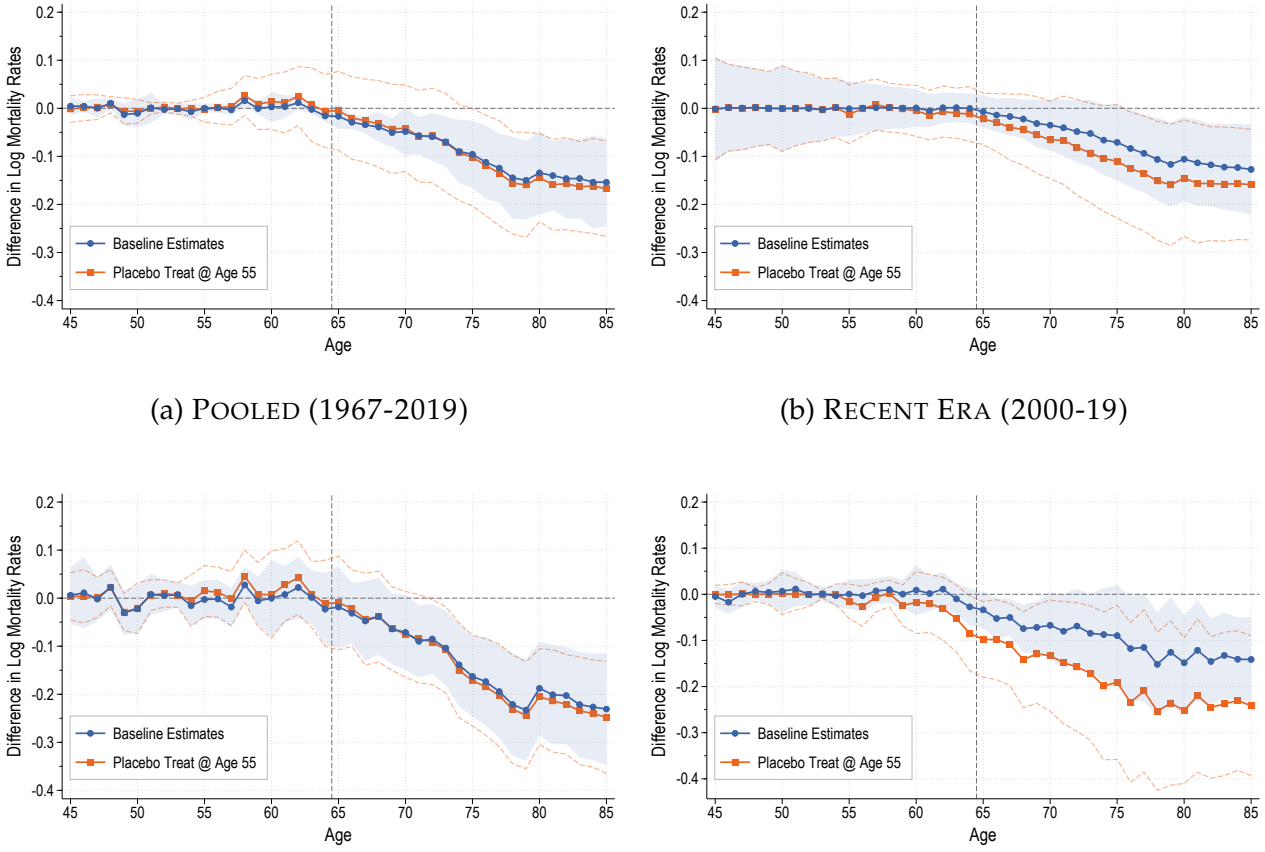
(b) UNMET NEED DUE TO *Financial* REASONS

Notes: This figure demonstrates the way that financial and overall unmet need evolves as individuals age into Medicare.

*The line labeled "ω-Weighted Average" is constructed by using the synthetic control weights from the recent-era *mortality* analysis (Figure 2, Panel B) and applying them to unmet need values across countries. Weights differ slightly as they were upweighted to reflect incomplete coverage of countries in the unmet need sample (91.3% of total weights for the 2000-19 era). See text for more details and Appendix Section A.1 for information on data construction.

Source: Author calculations using the National Health Interview Survey (Blewett et al., 2025*b*), Medical Expenditure Panel Survey (Blewett et al., 2025*a*), European Union Statistics on Income and Living Conditions (Eurostat, 2026*b*), Taiwanese Social Change Survey (Institute of Sociology, Academia Sinica, 2011), Russian Comprehensive Survey of Living Conditions (Rosstat, 2018), and Korean Health Interview Survey estimates from Chung (2022).

Figure 4: A PLACEBO 'TREATMENT' AT AGE 55 GIVES SIMILAR RESULTS FOR THE POOLED, RECENT, AND MIDDLE ERAS



(a) POOLED (1967-2019)

(b) RECENT ERA (2000-19)

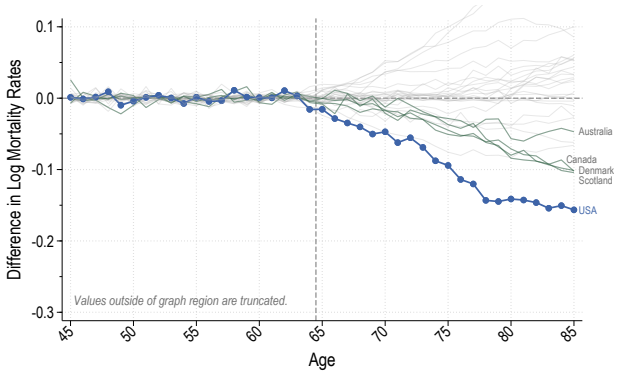
(c) MIDDLE ERA (1980-99)

(d) EARLY ERA (1967-1979)

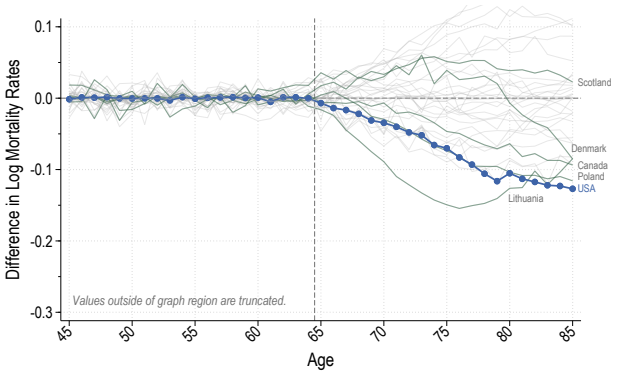
Notes: This figure demonstrates the robustness of utilizing a placebo treatment age of 55, wherein our synthetic control group is matched on ages 45 through 54, and effects are permitted to deviate from 55 onward. As displayed in the figure, the results from age 55-64 are highly similar to our primary specification, except potentially the early era, where they diverge slightly (and in a statistically indistinct manner) immediately before the age-65 eligibility date. See Section 5 for further discussion.

Source: Author calculations using the Human Mortality Database (2026).

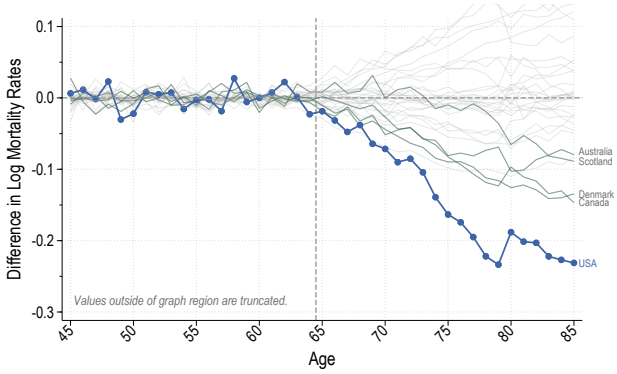
Figure 5: THE UNITED STATES' TREATMENT EFFECTS EXCEED THOSE FROM OTHER COUNTRIES



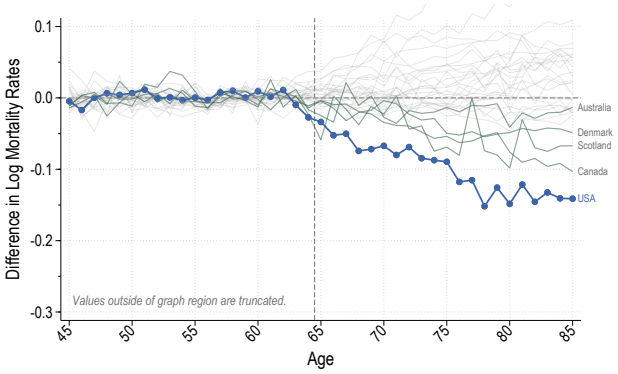
(a) POOLED (1967-2019)



(b) RECENT ERA (2000-19)



(c) MIDDLE ERA (1980-99)

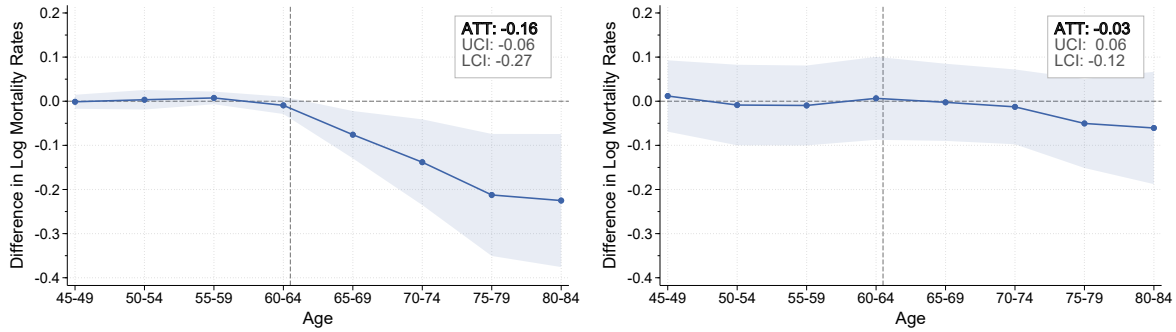


(d) EARLY ERA (1967-79)

Notes: The purpose of this figure is to display trajectories of other countries when assigned placebo treatment status. Placebo countries with large deviations in the pre-period (more than twice the median pre-treatment RMPSE) were omitted from the graph. Highlighted countries–i.e., those with darker lines and labels–include the top-three closest to the United States in the overall analysis, as well as those with notable trends in a given era. Light lines represent other placebo estimates.

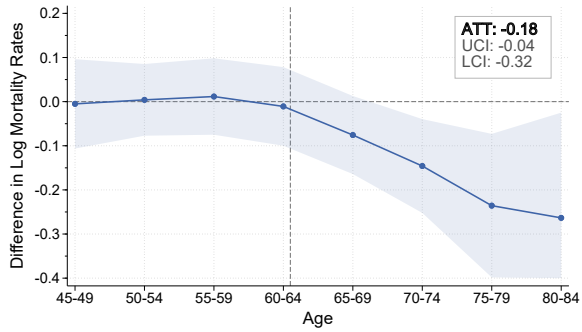
Source: Author calculations using the Human Mortality Database (Human Mortality Database, 2026)

Figure 6: MEDICARE DECREASES MORTALITY FROM TREATMENT-AMENABLE CAUSES AND CARDIOVASCULAR DISEASE, BUT NOT FROM NON-AMENABLE OR EXTERNAL CAUSES

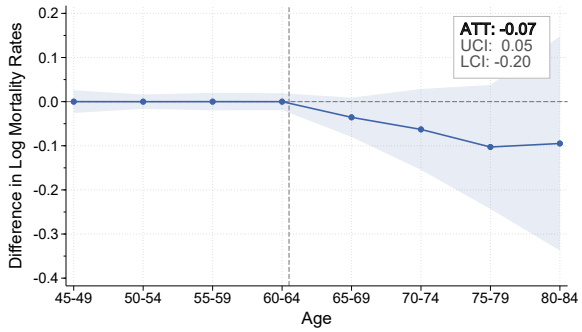


(a) DEATH FROM AMENABLE CAUSES

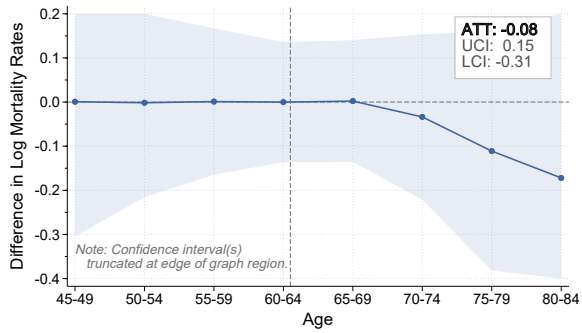
(b) DEATH FROM NON-AMENABLE CAUSES



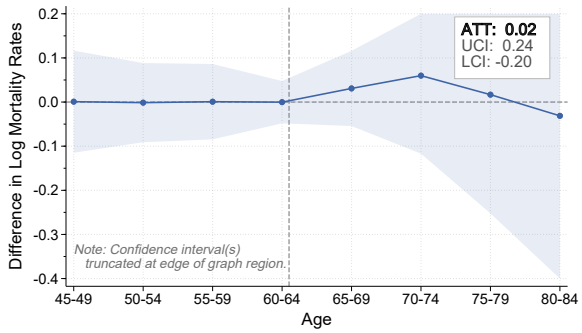
(c) CARDIOVASCULAR



(d) CANCER



(e) RESPIRATORY

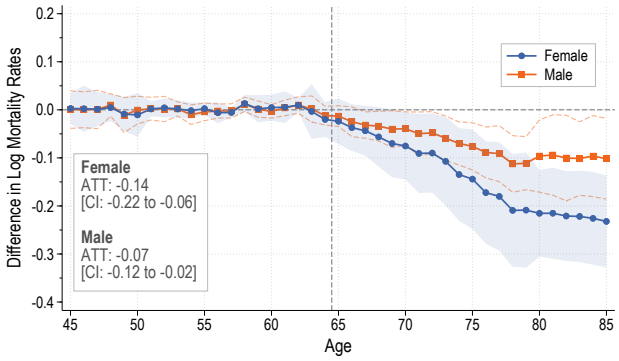


(f) EXTERNAL

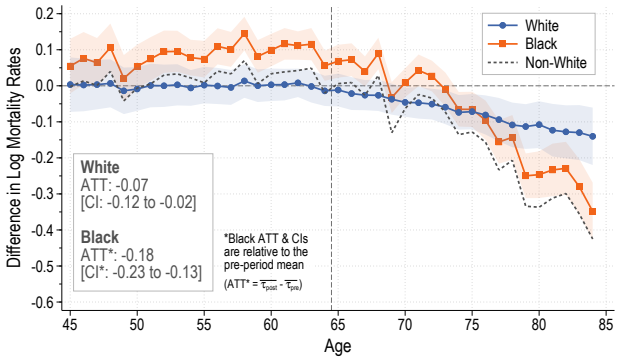
Notes: This figure demonstrates This figure demonstrates the impact of Medicare on mortality by cause of death. The top row breaks out mortality by "amenable" and "non-amenable" causes, where amenable are causes of death associated with conditions that should be responsive to healthcare treatment (Nolte and McKee, 2003, 2011). The middle and bottom rows illustrate Medicare's impact on mortality from the top-three causes of death among U.S. adults aged 65 and older, plus external causes. Together, these four causes comprise 82.0% of all mortality. Note that confidence intervals in Panels E and F are truncated at the edge of the graph region to preserve a common y-axis across all figure panels.

Source: Author calculations using the WHO Mortality Database (World Health Organization, 2026) and list of treatment-amenable conditions from Nolte and McKee (2003, 2011).

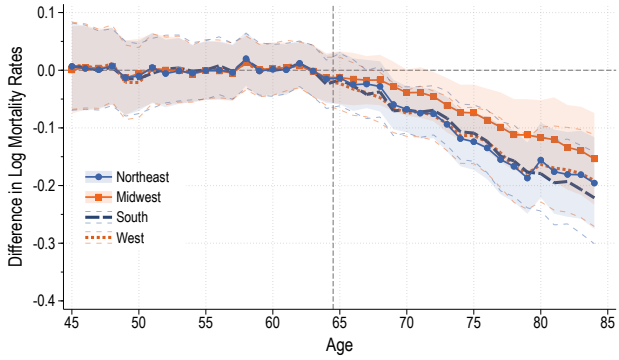
Figure 7: MEDICARE'S IMPACT IS PERVASIVE ACROSS SEX, RACE, AND REGION



(a) GENDER (1967-2019)



(b) RACE (1967-2019)

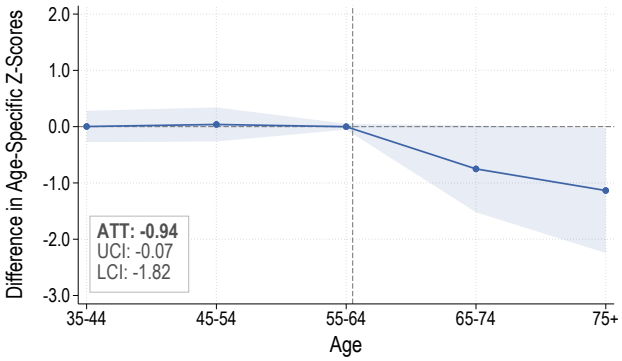


(c) CENSUS REGION (1967-2019)

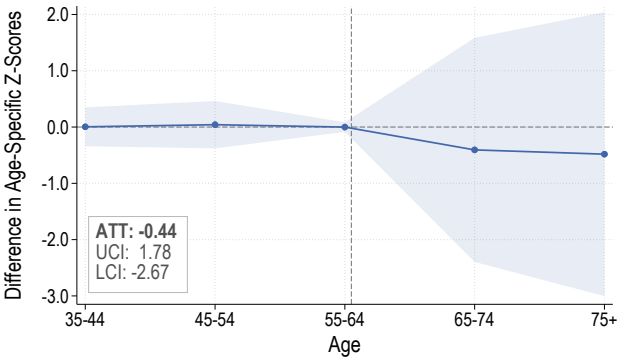
Notes: The purpose of this figure is to explore heterogeneity by gender, race, and U.S. region. Analyses using race and region were obtained by matching mortality specific to that U.S. sub-group with *overall* mortality of other countries. For each analysis, the race- or region-specific mortality was the only treated unit.In the figure, 'White' refers to all individuals identifying as white, regardless of Hispanic ethnicity. The estimates for Black individuals are presented alongside non-white, but are outside the convex hull for donor countries. However, the pre-post difference is nearly identical to the non-white ATT. See Appendix Figure A15 for more detail regarding gender, Appendix Figure A16 for more detail regarding race, and Appendix Figure A17 for more detail on U.S. Census Region.

Source: Author calculations using the U.S. Multiple Cause of Death Data (Vital Statistics) and Human Mortality Database (2026).

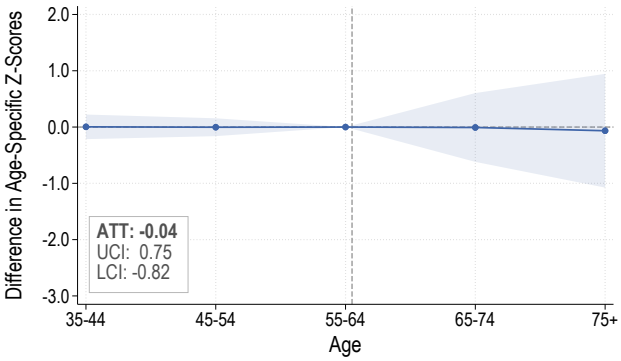
Figure 8: MEDICARE REDUCES MORBIDITY



(a) DIFFICULTY W/ PHYSICAL FUNCTIONS (INDEX)



(b) COGNITIVE DIFFICULTIES

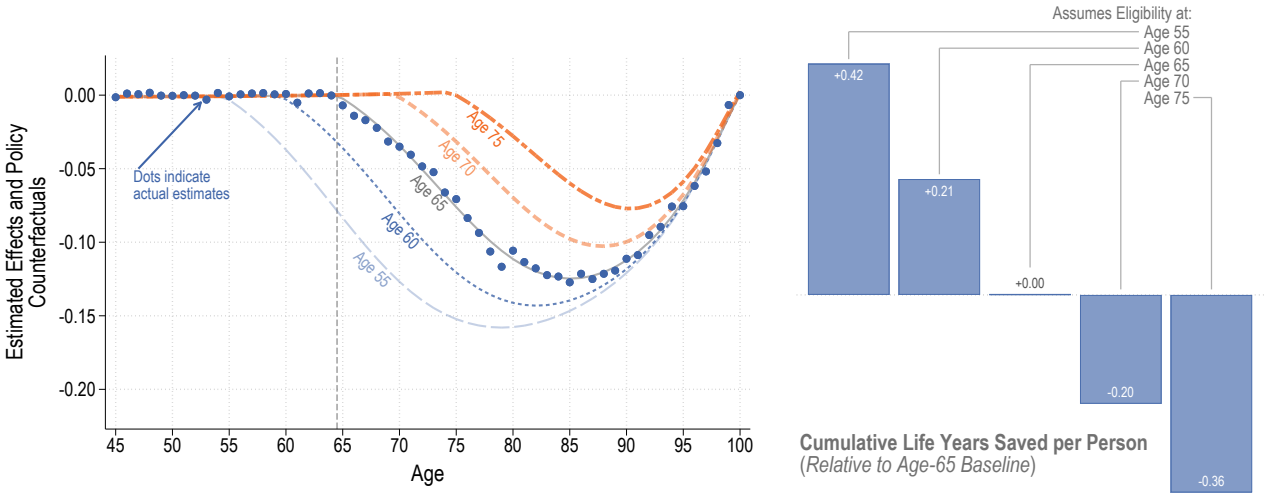


(c) SENSORY DIFFICULTIES (INDEX)

Notes: This figure demonstrates Medicare's impact on activities of daily living, which are captured by (A) a physical functions index, which aggregates difficulties with mobility and self-care, (B) cognitive difficulties, defined as remembering/concentrating/making decisions, and (C) sensory and/or cognitive, which aggregates difficulty seeing and hearing, even with corrective lenses or hearing aids, respectively. See Appendix Figure A19 for analysis on each sub-component of the index, using z-scored and logged incidence rates. Within the figure, ten-year bins are utilized, as that is the granularity available in the Eurostat/EHIS data. (These data were also top-coded at age 75.) Due to the small number of pre-periods, the pre-period was extended back to age 35 and synthetic difference-in-differences was used to estimate these effects.

Source: Author calculations using the American Community Survey (Ruggles et al., 2025*b*), European Union Statistics on Income and Living Conditions (Eurostat, 2026*b*), European Health Interview Survey (Eurostat, 2026*a*), and harmonized data from IPUMS International (Ruggles et al., 2025*a*).

Figure 9: MEDICARE'S IMPACT PREDICTED UNDER DIFFERENT ELIGIBILITY THRESHOLDS



(a) MODEL FIT AND COUNTERFACTUAL ESTIMATES (PREFERRED)

(b) COUNTERFACTUAL SUMMARY (PREFERRED)

Notes: The purpose of this figure is to display the results of our counterfactual analysis when using our preferred model to parameterize our recent-era estimates. Within the Figure, Panel A displays our original synthetic control estimates (indicated by dots), alongside the parameterized model fit. The other lines in the figure use the estimated parameters to indicate the mortality path under four different counterfactual scenarios–namely if Medicare eligibility were changed to age 55, 60, 70, or 75, respectively.

Panel B represents the life-years saved (or lost) under each of the scenarios, *relative* to current policy of Medicare eligibility at age 65. See Appendix Figure A20 for a recreation of Panel A under different modeling scenarios and Appendix Figure A21 for the relative life-years saved under those scenarios.

Source: Author calculations using Social Security Cohort Life Tables and the Human Mortality Database (2026).

Appendix

A Data Construction

A.1 Unmet Need Data

Data to derive the percentage of individuals with unmet medical needs were gathered from several sources, which are detailed below:

United States

Within the United States, we obtained primary data for unmet need due to *financial* reasons from the 2008-19 National Health Interview Survey (Blewett et al., 2025*b*). Unfortunately, the NHIS does not contain a question asking whether an individual has unmet medical need across *all* reasons. For this we utilized the Medical Expenditure Panel Survey ("MEPS") (Blewett et al., 2025*a*), which asks respondents questions pertaining to both financial and unmet need. However, questions in the MEPS have been revised multiple times over the sample period, resulting in a high level of variation. For instance, during the 1997-2001 period, overall and financial unmet need averaged 10.0% and 7.1%, respectively, among 45-64 year olds. These responses (for financial need) were generally in-line with those in the NHIS (5.5%). However, for the period from 2002-2017, these averages discontinuously plummeted to 2.5% and 1.9%, respectively, while the NHIS rate rose to 7.8%, making the MEPS no longer comparable to other surveys or previous results.

Accordingly, in order to estimate overall unmet need during the period, we used the ratio of financial need to unmet need in the MEPS during the 1997-2001 period, when these responses were comparable to the NHIS figure. We then applied this ratio to the financially focused unmet need question in the NHIS to obtain an estimated amount for overall unmet need. Formally, the estimated overall unmet need for an age (a) by year (y) cell is:

$$\text{Estimated Overall Unmet Need}_{ay} = \frac{\frac{1}{5}\sum_{i=1997}^{2001} \text{Overall MEPS}_{a,i}}{\frac{1}{5}\sum_{i=1997}^{2001} \text{Financial MEPS}_{a,i}} \times \text{Financial NHIS}_{ay}$$

Euro-Area Countries

For EU-area countries, we obtained unmet need information from published tables of the European Union Statistics on Income and Living Conditions ("SILC") (Eurostat, 2026*b*). Pub-

lished tables from the SILC are available from 2008 onward and present ages in ten-year increments, which is why the comparison series is presented with this degree of financial detail.

Russia

For Russia, we obtained unmet need information using microdata from the Comprehensive Survey of Living Conditions, obtained from Rosstat (2018), the Russian Statistical Service. While other published papers suggest key variables are available back to at least 2012, attempts to download the 2012 and 2014 survey microdata were met with errors indicating technical issues on behalf of the Russian-government website. Accordingly, only the 2016 and 2018 data were used.

Taiwan

For Taiwan, data was obtained from the 2011 Taiwanese Social Change Survey ("TSCS") (Institute of Sociology, Academia Sinica, 2011). We were unable to locate any other surveys within our sample period that provided unmet need (financial and overall), disaggregated by type and age.

South Korea

For South Korea, by-age estimates were obtained from Chung (2022), who derived them from the 2014-2018 Korean Health Interview Survey.

A.2 Difficulties and Limitations Data

Our measures regarding difficulties and limitations focused specifically on countries that utilize questions in the style of those proposed by the Washington Group on Disability Statistics ("WG"). Specifically, these questions are formatted in the following ways:

- Mobility: *"Does this person have serious difficulty walking or climbing stairs?"*
- Self-Care: *"Does this person have difficulty dressing or bathing?"*

- Cognition: *"Because of a physical, mental, or emotional condition, does this person have serious difficulty concentrating, remembering, or making decisions?"*
- Seeing: *"Is this person blind or does he/she have serious difficulty seeing even when wearing glasses?"*
- Hearing: *"Is this person deaf or does he/she have serious difficulty hearing?"*

We chose these specific measures, because they:

1. Are not tied to labor force participation (e.g., "Do you have a condition preventing you from working at a job or business"), as such questions would not apply to many adults past retirement age;
2. Are not tied to country-specific rules regarding social safety net participation; and
3. Maximize comparability due to the consistency of the language across surveys.

United States

U.S. data comes from the American Community Survey (Ruggles et al., 2025*b*), for the years 2008-19.

Euro-Area

Data for European countries were obtained from published tables of the European Health Interview Survey (2014, 2019) and the SILC Health Module (2022). Both surveys were obtained from the Eurostat website and present data in ten-year age bins, which is why that was chosen for our analysis.

It should be noted that, while the SILC has asked questions about disability dating back to its inception, the wording in these questions was closer to Global Activity Limitation Indicator, and is not comparable to the Washington-Group-style questions asked in the United States.

Non-European Union Countries

Additional data for non-EU countries was obtained from IPUMS International (Ruggles et al., 2025*a*), which harmonized Washington-Group-style data from the following countries (years): Brazil (2000, 2010), Mauritius (2011), Mexico (2020), Vietnam (2009, 2019), South Africa (2011, 2016), Suriname (2012), and Trinidad and Tobago (2011).

Robustness to Variations in Donor Pool

It should be noted that our results presented in Figure 8 are robust to using *only* countries that overlap with the Human Mortality Database, which are listed in Appendix Table A1. Namely, using a donor pool of only HMD countries yields a reduction of 0.87 ($p < 0.05$) to the physical difficulty index discussed in Section 8, comparable to the reduction of 0.94 in our main specification. Likewise, the HMD-only analysis yields a statistically insignificant decrease in cognitive difficulties and a near-zero impact on the sensory index, again mirroring our main results.

A.3 Labor Force Participation and Income Data

We obtained cross-country data for household income and labor force participation from the Luxembourg Income Study (2026) ("LIS"), which harmonizes income and labor surveys for a number of high- and middle-income countries. These data were supplemented with additional country-years from IPUMS International (Ruggles et al., 2025*a*). Because the quality and comprehensiveness of the cross-country labor data is substantially lower in the years before 1980, we opted not to harmonize and apply data for those years. One particularly appealing feature of the LIS data is that the household income variable also includes monetized values of other non-cash transfers (such as housing and food assistance), which allows it to capture the true degree of support enjoyed by older adults in each country.

Age Heaping/Bucketing. Country-era cells that did not provide single-year ages or had unusual age heaping were dropped from the dataset. For purposes of our analysis, a country

was considered to have bucketing or age-heaping if:

$$\frac{\frac{1}{N_{\text{mod}=0}} \sum_a obs_a \times I(\text{mod}(a,5)=0)}{\frac{1}{N_{\text{mod}\neq 0}} \sum_a obs_a \times I(\text{mod}(a,5)=0)} > 2,$$

where $N_{\text{mod}=0}$ and $N_{\text{mod}\neq 0}$ are the number of divisible and indivisible by five, respectively. In other words, country-era cells were dropped if there were at least twice as many observations in years divisible by five, on average, than those not divisible by five. We supplemented this by manually dropping observations with heaping at other ages, or which exhibited other unusual data patterns.

Canada. An exception to the rule discussed above is Canada, which exhibited heaping/bucketing in the recent era. However, due to Canada's importance as a comparative case study (see, for instance, Appendix Figure A10 and the related in-text discussion), we performed further data-cleaning procedures to make it suitable for analysis.

Specifically, we obtained the 2011 Canadian Census from IPUMS International, which provides microdata with single-year ages. We then used these data to interpolate within age-group dynamics for the recent-era LIS as follows:

$$\widetilde{LIS}_{a,2000-19} = \frac{IPUMS_{a,2011}}{\overline{IPUMS}_{g(a),2011}} \times \overline{LIS}_{g(a),2000-19},$$

where a represents age and $\overline{X}_{g(a)}$ represents the average over an age group ($X = \{LIS, IPUMS\}$). Note that, despite covering slightly different time frames and being derived from different underlying sources, the correlations between the LIS and the IPUMS International grouped averages were higher than 0.95 in all cases, and typically greater than 0.99.

Top-Coding. An additional challenge to utilizing the LIS data is that many surveys top-code age within our treatment window, particularly during the recent era. The youngest (and most common) age for top-coding was age 80, which applied to nine different country-era cells. Rather than limit our analysis to younger ages–which makes comparing estimates difficult–we opted to extrapolate older-age labor force participation and income using the synthetic control method, designating the 'treated' time as the age immediately before top-coding and using non-top-coded countries as donor units. To achieve the best fit, the pre-period for this exercise was limited to age 70. In each case, the synthetic control fit was

extremely strong (with correlations between the synthetic fit and observed values exceeding 0.99 across all variables). Due to the nature of this exercise and the synthetic control method in general, this extrapolation procedure will not affect estimates for ages under 80, where the impact of labor force participation is likely to be the greatest.

Units Used in Final Analysis. The countries/regions used in the final analysis are: Austria, Belgium, Bulgaria‡, Canada, Switzerland, Chile‡, Czech Republic, Germany‡, Denmark, Spain, Estonia‡, Finland‡, France, England Wales, Northern Ireland‡, United Kingdom, Scotland, Greece, Hungary‡, Ireland, Iceland‡, Israel†, Italy, Japan‡, Republic of Korea‡, Lithuania‡, Luxembourg, Netherlands, Norway, Poland, Portugal†, Russia‡, Slovakia, Sweden, and Taiwan.

†Indicates country/region is only present for the middle-era analysis (1980-99).

‡Indicates country/region is only present for the recent-era analysis (2000-19).

A.4 Healthcare Spending and Consumption Data

Data on spending by age were gathered from several sources, which we detail below:

United Nations National Transfer Accounts ("NTA"). The NTA focuses on allocating country-level consumption spending across various age groups to capture the degree to which transfers occur across the life cycle. This dataset was used for the following countries: Australia, Austria, Canada, Chile, Finland, France, Germany, Hungary, Israel, Italy, Japan, Poland, Russia, Slovenia, Spain, South Korea, Sweden, and Taiwan.[1]

Estimates for the United States were obtained from the Centers for Medicare & Medicaid Services ("CMS") National Health Expenditure Data ("NHE"), which allocates personal health consumption across several ages. However, these age groups still tend to be large—from ages 16 to 44, ages 45 to 64, and ages 65 to 84. To allocate spending across the life cycle, we utilized age-by-spending profiles from the Medical Expenditure Panel Survey (Blewett et al., 2025*a*) and applied it to the NHE estimates. Specifically for a given age (a),

[1] To be clear, this is the set of NTA countries that overlap with the HMD sample.

year (y), and NHE age group (g):

$$Spending_{ay} = \frac{MEPS_{ay}}{\sum_{a \in g} MEPS_{ay}} \times NHE_{yg},$$

which is to say that we applied the age-specific percentage of spending within a given age group, derived from the MEPS, to the NHE estimates. This was performed both for the NHE age groups and to NHE spending overall.

It should be noted that this is an expenditure-based measure, rather than a consumption-based measure as in the NTA. However, comparisons with the NTA-based measures reveal them to be nearly identical. (Moreover, adjusting U.S. spending based on a ratio of overall expenditure to overall consumption generates estimates that are essentially identical.)

Countries Not Included in the NTA. To supplement our sample of countries, we also integrated estimates of spending-by-age published by Eurostat (2016). These estimates are presented in five-year age bins, which is what we utilized in our overall analysis. These countries are: Czechia, Germany, Finland, Hungary, Lithuania, Latvia, the Netherlands, Slovenia, and Sweden.[2]

Other Adjustments. All spending amounts, regardless of source, were adjusted to be in 2010 PPP- and inflation-adjusted U.S. dollars.

B Reconciling our Results with Goodman-Bacon et al. (2025)

In this section, we consider a contemporaneous and complementary working paper by Goodman-Bacon, Lleras-Muney, Price, and Yue (henceforth, "GLPY"). This paper utilizes several complementary methods, combined with a novel mortality-linked Census Tree dataset, to examine the initial effects of Medicare's introduction. While the paper's overall conclusions—that Medicare's introduction saved a substantial number of life-years—generally agrees with our own, it deserves special discussion for two reasons.[3]

[2] There is some overlap between these countries and those in the NTA, but this list supplements with country-years that may not be present in the NTA data.

[3] Discussing a working paper (rather than a final, published work) in this level of detail is perhaps unusual. However, we believe that, given the working paper's quality—namely, the fact that it addresses a crucial question using novel data and innovative methodology—that it will be highly influential and is therefore

First, GLPY finds their life-saving effects are largely concentrated among men, with mixed evidence for women. Specifically, when using interrupted time-series ("ITS") estimates, they find an increase in mortality for women (a decrease in life-expectancy), while their staggered difference-in-differences specification finds decreased mortality for women, though slightly in smaller magnitude than the male-specific estimates. This contrasts with the findings in our paper, where we find the early-era effects for women are actually *larger* than men, though not in a statistically distinguishable manner (Appendix Figure A15).

Second, in contrast to our paper, GLPY utilize variation only from within the United States, which holds attractive features in relation to our international approach. Specifically, their staggered cohort difference-in-differences framework (henceforth, "CDID") allows them to address US-specific age shocks, assuming that these age shocks are consistent over time. The CDID estimates closely agree with those from ITS for men, a fact that increases confidence in both methods, but as noted above, the two methods generate divergent qualitative conclusions for women.

Taken together, these facts generate two questions: first, why does our paper find effects for women, while GLPY finds mixed effects? Second, why are the magnitudes of our paper generally smaller than those found by GLPY, particularly for men? These questions are addressed in the two subsections, below.

B.1 The Divergent Post-65 Mortality Patterns for Men and Women

As emphasized by Figure 1, a country's mortality patterns do not evolve in isolation—they are subject to global patterns that relate to disease, nutrition, and medical technology. These factors may influence population health among men and women in different ways—something that is clearly shown by Appendix Figure A23, Panels A and B. Within the figure, we plot the detrended and recentered logged mortality for the 1902-15 cohorts by gender, as compared to gender-specific mortality of other countries within the HMD.[4] Two patterns emerge from these panels.

worth an in-depth exploration. For purposes of our discussion, we refer to the draft dated October 23, 2025, which is available here.

[4] These cohorts were chosen as the 1902 cohort was the earliest to be fully exposed to Medicare, while the 1915 cohort is the upper bound used in GLPY's main sample.

First, as displayed in Panel A, female mortality, whether in the United States or abroad, markedly exceeded the pre-65 Gompertz trend. This suggests that there were global factors among other countries that could act as potential confounders in an ITS design, leading GLPY to estimate an increase in mortality due to the roll-out of Medicare. However, as is clearly shown in Panel A, while the United States' logged mortality is pulled above trend by these secular factors, it is far less affected than other countries, suggesting that Medicare played a major role in offsetting global trends.

Male mortality, shown in Panel B, displays a similar story, but in reverse. Here non-U.S. countries are, on average, meaningfully below the Gompertz trend. However, as before, the U.S. shows a larger trend break than its global peers, but the overall magnitude of the effect detected by an ITS design is likely to be contaminated by secular trends causing a divergence from Gompertz law, and not solely the impact of Medicare.

The potential issues with using an ITS design are more formally addressed in Panels C and D of Figure A23. To create this figure, we estimated interrupted time series models using methods analogous to GLPY, applying them to the 1885-1915 cohorts in countries other than the United States.[5] Within the figure, each thin line represents the estimates corresponding to an individual HMD country, while the connected points represent a simple average across all estimates. Consistent with the descriptive evidence provided in Panel A, the ITS estimates for women (Panel C) show a positive treatment effect, the same as GLPY's ITS estimates for women—although the international estimates show a positive pretrend. The estimates in Panel D also mirror the descriptive results (in Panel B), as the male-specific ITS show a meaningful downward trend, with the treatment effect roughly half as large as that shown in GLPY.

This, however, conjures another question: if the male-specific ITS estimates in GLPY are driven, in part, by global factors other than Medicare, then why do they generally agree with GLPY's CDID estimates? After all, the CDID estimates should, in theory, capture secular departures from the Gompertz trend by netting out age-effects—age effects which were derived from cohorts that lived through the pre-65 period before Medicare's inception. We address

[5] To be clear, we estimated an ITS design on each country-by-cohort group, then aggregated those estimates to the country level. The 1885-1915 cohorts were used, as those are the cohorts used by GLPY for their ITS analysis.

this in the following sub-section.

B.2 The Role of Period Factors

As noted above, GLPY use a staggered cohort difference-in-differences implementation, whereby they utilize aging patterns from pre-1902 cohorts, who did not have access to Medicare during the entirety of their post-65 adulthood, to account for non-linear age effects that might result in departures from Gompertz' law. The estimates for this exercise are therefore resistant to the critiques of the section above. They could, however, be confounded by period factors that change the age-mortality relationship, particularly the gradient relevant to older ages.

To ascertain if there are international period factors that could influence treatment effects, we estimate a cohort difference in differences model, using techniques developed by Callaway and Sant'Anna (2021). This procedure, which is again applied to the 1885-1915 cohorts, is similar in spirit to the methods used by GLPY, though we do not aggregate our ATTs in exactly the same fashion, since the estimates here are presented only for illustrative purposes. Specifically, we estimate the CDID separately for each country using Stata's `csdid` command and aggregating to the event-time level. The results of these country-specific CDIDs are displayed as thin lines in Appendix Figure A24, and the simple average across all country-level estimates are shown in the darker connected lines.

As indicated by Panel A, applying this technique appears to generate a treatment "effect" suggesting a decrease in mortality for men. This placebo effect appears in the simple average, and manifests for every country as of ten years post-treatment. Strikingly, the magnitude and age dynamics of the estimates are similar to the male-specific ITS estimates in Appendix Figure A23. In a way, this mirrors GLPY, whose ITS and CDID results are also very similar to each other in magnitude and age dynamics, although those magnitudes are roughly twice as large as what we find here, suggesting that Medicare is making an impact beyond the secular trend.

While the male-specific ITS and CDID estimates converge, the female-specific CDID estimates are starkly different from those recovered from the ITS method. This, again, mirrors GLPY, who find increases in female mortality from their ITS specification and reductions in

female mortality from their CDID specification. Again, their CDID estimates are roughly twice as large as what we find here.

It is worth asking: What period-specific factors could be causing such large, global effects? A prime candidate is the Cardiovascular Revolution, which started in the 1960s and brought about decreases in heart-related deaths over many years. Indeed, by 2000, many countries' cardiovascular death rates were half (or less) of their 1950s- and 1960s-era peak (Cutler, 2004*b*). While steady long-term trends may not be an issue for CDID methods, sharp structural breaks in period effects, such as the one brought about by the cardiovascular revolution, can bias estimates.

B.3 Summarizing Remarks

In this section, we have pointed out global phenomena that may be influencing GLPY's estimates of Medicare's impact, specifically the upward-biasing of the female-specific ITS results, and the downward-biasing of the male-specific ITS and CDID results.[6] We want to emphasize that, because these trends are derived from other countries, they do not necessarily represent the true secular trend present in the United States over the time period in which Medicare was being adopted. However, the pervasiveness of these global factors—not just on average, but across multiple countries—suggests that they may be greatly influencing the U.S. results.

We also wish to emphasize that the historic worldwide changes during this period make finding *any* control group exceptionally difficult. Indeed, even when using our synthetic control approach, which specifically seeks to net out the effects of these global trends, our early-era estimates do not convincingly pass the placebo cutoff tests, displaying a distinct downward trend during the ages of 55-64 when we relax the pre-period matching procedure and allow it to do so (see Figure 4). This suggests that our estimates and GLPY's estimates regarding the adoption of Medicare may be overstated due to secular trends, albeit to different degrees. Reassuringly, we find no such issues with our later eras or overall results, where the U.S. convincingly passes the aforementioned placebo-cutoff test, which is to say our synthetic

[6] While we do not explicitly comment on GLPY's third method, which utilizes a model-driven approach based on Lleras-Muney and Moreau (2022), we expect that the factors laid out in this sub-section will affect those results in a similar manner. This is especially likely given that the structure of the model has health stock decaying in a similar fashion to what one would expect based on Gompertz' Law.

control group is successful at netting out the effect global trends from our estimates.

C Additional Robustness

C.1 Further Evidence that Retirement is Not Driving Our Effects

As discussed in Section 5, one may reasonably view the departure between the United States and its counterfactual as commencing at age 63 (rather than age 65), which could point to a mechanism other than Medicare. We listed, at the time, several reasons that we do not hold that view. Perhaps most importantly, the break in the recent era occurs very sharply at age 65. For the other eras, the inclination to place the break at age 63 is compelled by a "bump" at age 62. Specifically, there are, in some eras, anomalous spikes in mortality at age 62 followed by what appears to be a persistent downward trend thereafter. It is our belief that the age-62 spikes are the result of retirement, an effect in line with that established in Fitzpatrick and Moore (2018). Going beyond what that paper established, we believe, furthermore that the spike at age 62 reflects "harvesting"—a short-term mortality increase among vulnerable individuals followed by lower-than-expected mortality—and that harvesting gives the appearance of the decline preceding Medicare eligibility. We justify our stances in Appendix Figure A7.

As in our analysis of Figure 1 (righthand column), in Appendix Figure A7, we show deviations from log-linearity between ages 45 and 64 in the United States, but we plot the age-specific deviations over time. Each age in the age range is its own time series, but we highlight ages 62, 63, and 64. These three series appear to trend together in the 1950s but disperse suddenly in the 1960s, with age 62 being persistently high risk and age 64 being persistently low risk. The presence of the cohort of 1900, which receives enormous heaping in death certificates, makes it very difficult to ascertain the start of the dispersal. However, it looks to be in the early 1960s and is seen clearly by 1965 when the heaping (and displacement) has cleared. Critically, the threshold for drawing on Social Security became 62 in 1961. So, this pattern—shape and timing—is consistent with a spike in mortality upon retirement that harvests from the two subsequent years of life. The three focal series reconverge around 2000. Harmoniously, the spike is no longer present in the recent era.

Reiterating our conclusion, the bump at age 62 is a genuine result of retirement, but it should not be viewed as the beginning of the departure between the United States and its synthetic control. Rather, it is a coincident anomaly in select years. If one were to "smooth out" the retirement spike over two years, the departure would occur at age 65, just as it does in the recent era.

C.2 Evidence that Results are Not Biased by Overfitting and that Our Results Are Robust to Using Many Permutations of the Donor Pool

Two potential issues that could bias our estimates include the possibility of (1) overfitting when selecting our counterfactual group and (2) that results are driven by idiosyncratic features of one or more of the selected control countries, rather than a true effect of Medicare. The first concern, as noted in Hollingsworth and Wing (2022), would bias our results if the synthetic control group is matched to idiosyncratic noise, rather than the true latent factors that drive mortality. The second would take the form of omitted variables bias, where there is some event within the control group that is not properly accounted for.

We note that potential issues with overfitting are somewhat mitigated by the fact that we pool mortality over many years, therefore averaging out idiosyncratic shocks to age-specific mortality, and that idiosyncratic control group issues are substantially addressed via our leave-out procedures described in Section 5 and displayed in Appendix Figure A9. Nonetheless, we perform additional robustness tests to address both concerns by drawing 1,000 random 50% samples of the donor population and then forming a synthetic control group using regularization techniques—namely Lasso and elastic net.[7] We combine these two features—estimating effects on numerous variations of our donor pool and applying regularization techniques—as Lasso and elastic net are particularly good at prediction. They are therefore able to match pre-trend, even in smaller samples.

The results of this analysis are displayed in Appendix Figure A12, where the estimates

[7] Lasso is the suggestion put forth by Hollingsworth and Wing (2022), as it will throw out control units that do not meaningfully contribute to predicting pre-treatment mortality. However, we also use elastic net, which combines Lasso and ridge regression, as it performs well when there is significant correlation among variables—which is the case among country-level mortality, as shown in Figure A12. Moreover, it may be desirable to simply downweight units (as elastic net and ridge regression do) rather than throw them out entirely (as Lasso does) when all units contain useful signals about global and secular trends.

from each sample are plotted with 10% opacity. As shown in the figure, the regularization techniques do an excellent job of matching pre-period trends. More importantly, the post-65 estimates all clearly mirror our main estimates displayed in Figure 2, both in shape and overall magnitude, with relatively little dispersion of treatment effects.

Taken together with the other evidence presented in this paper, this strongly suggests that any synthetic control group capable of mirroring the United States' pre-65 mortality trends will give extremely similar treatment effects. This is, in our view, extremely reassuring. Consider, for instance, the alternative explanation that the United States' stark trend break in mortality is driven primarily by social security (or U.S.-specific income sources in retirement). If that were true, then this feature of the United States would have to be extraordinary relative to (1) our primary control group and (2) all our leave-out estimates and (3) our estimates using exclusively high-income or OECD countries as donors and (4) the 1,000 other samples drawn from this exercise.

Such a circumstance seems unlikely for most government programs and other factors with potential to affect population-level health. However, it does *not* seem unlikely for Medicare, given that the U.S. is famously an outlier among high-income (and many middle-income) countries for its lack of comprehensive health coverage, a circumstance that changes sharply when a cohort turns 65.

C.3 Treatment Guidelines

One may be concerned about age 65 from a clinical perspective. For instance, certain guidelines, such as cancer screenings or risk scores, may contain age-65 thresholds. On one hand, if those thresholds are set at age 65 because so many people are reliably insured, we would view that as part of the treatment effect. On the other hand, if there are more anatomical or biological bases for the cutoff, these guidelines could be viewed as a confound. However, heuristics matter only if age-65 thresholds are common in the United States and not common elsewhere. There is no central body compiling international guidelines, making it difficult to know if these possibilities are true. Yet, in Appendix Figure A25, we provide some evidence that 65 is not ubiquitous and no more common in the United States than elsewhere.

We created this figure by first searching the US Preventative Services Task Forces's list

of high quality screening recommendations (U.S. Preventive Services Task Force, 2025). For each screening recommendation that spanned into older adulthood, we then searched for international guidelines that were roughly equivalent, with special focus on obtaining treatment guidelines in Russia and Taiwan, as these are the two largest donors for the recent period and are also two countries that we believed, ex-ante, might not adhere to Anglo or European norms in terms of screening and treatment.[8] We conclude that the United States is not an outlier relative to the comparison countries listed, nor does it appear that Medicare is driving sweeping changes in medical heuristics.

[8] The data sources used for this exercise are:

Ebell, Mark H., Thuy Nhu Thai, and Kyle J. Royalty. 2018. "Cancer Screening Recommendations: An International Comparison of High Income Countries." *Public Health Reviews, 39: 7.*

Ekoe, Jean-Marie, Ronald Goldenberg, Pamela Katz, and Diabetes Canada Clinical Practice Guidelines Expert Committee. 2018. "Screening for Diabetes in Adults." *Canadian Journal of Diabetes*, 42(Suppl. 1): S16–S19.

Health Promotion Administration, Ministry of Health and Welfare, Taiwan. 2024. "Healthy Taiwan - Expanding Cancer Screening Starting in 2025: Your Health is the Government's Responsibility." `https://www.hpa.gov.tw/`.

Hsieh, Chi-pan. 2022. "Health Screenings for Adults." Published March 10, 2022. Accessed May 4, 2026. `https://www.pcchh.com.tw/uplo ad/download/202209281408530.pdf`.

Kalmykova, Zilya Askhatovna. 2025. "Modern Diagnostics of Diabetes Mellitus." Published December 8, 2025. Accessed May 4, 2026. `https://www.gmsclinic.ru/blog/sovremennaya-diagnostika-sakharnogo-diabeta`.

Kanis, J. A., Cyrus Cooper, Rene Rizzoli, and J.-Y. Reginster. 2019. "European Guidance for the Diagnosis and Management of Osteoporosis in Postmenopausal Women." *Osteoporosis International*, 30(1): 3–44.

Ministry of Health of the Russian Federation. 2021. "On Approval of the Procedure for Conducting Preventive Medical Examination and Dispensary Examination of Certain Groups of the Adult Population." `https://normativ.kontur.ru/document?documentId=477456&moduleId=1`.

Ministry of Health of the Russian Federation. 2025. "Abdominal Aortic Aneurysms." `https://diseases.medelement.com/disease/`.

Mortensen, Martin Bødtker, and Børge Grønne Nordestgaard. 2018. "Comparison of Five Major Guidelines for Statin Use in Primary Prevention in a Contemporary General Population." *Annals of Internal Medicine*, 168(2): 85–92.

National Institute for Health and Care Excellence. 2017. "Type 2 Diabetes: Prevention in People at High Risk." `https://www.nice.org.uk/guidance/ph38/chapter/recommendations`.

Royal Australian College of General Practitioners. 2025. "Defining and Diagnosing Type 2 Diabetes." In Management of Type 2 Diabetes: A Handbook for General Practice.

U.S. Preventive Services Task Force. 2026. "A & B Recommendations." Accessed May 4, 2026. `https://www.uspreventiveservicestaskforce.org/uspstf/recommendation-topics/uspstf-a-and-b-recommendations`.

C.4 Other Controls and Result-Aggregation Methods Not Discussed in the Main Text

In this section, we discuss a variety of other robustness tests not discussed at-length in the full text, each of which is given its own subsection, below.

C.4.1 Control Variables: Infant Mortality-by-Age (Appendix Figure A9)

Infant mortality and old-age mortality have many common latent factors, including sensitivity to environmental factors and overall health system quality. Accordingly, we include infant-mortality-by-age controls—specifically, the interaction of logged infant mortality rates with age—to address the possibility of omitted variable bias that might not be addressed by our matched control group in the baseline specification. As shown in the figure, results are highly similar to our baseline.

C.4.2 Control Variables: Obesity + Smoking (Appendix Figure A9)

As discussed elsewhere in the text, the greatest candidates for potential sources of bias come from ways in which the U.S. sharply differs from essentially all other countries that would not manifest until later in life. Two of these potential factors include obesity and smoking. The United States has more obesity than other large, high-income countries, and, in contrast, has lower rates of smoking than many of its peers and countries in the selected control group. If the relative impacts of these two factors meaningfully differ in later life from the baseline, then they could potentially bias our results. As displayed in the figure, there is no such bias, though inclusion of these controls does reduce precision.

Aside from the evidence provided by directly controlling for these factors, we again point to our amenable/non-amenable results (Figure 6) as evidence against obesity and smoking driving the overall reduction in mortality that we find. Amenable factors do not include deaths from diseases that can be traced back to lifestyle factors, such as lung cancer, and thus if something like relatively low smoking rates were driving our results, we would expect an opposite-signed pattern in that exercise.

C.4.3 Aggregation Method: Estimating Individual Years Using Period and Cohort Approaches (Appendix Figure A26)

For our main results, we aggregate mortality by age across many years into "eras," keeping only countries that are consistently observed throughout. The advantage of this approach is that it reduces variance, increases precision, and reduces the probability that our synthetic control estimates will be matched on idiosyncratic noise, rather than true latent factors. However, to ensure that this isn't driving our results, we re-estimated our results for individual years and plotted each coefficient trajectory, along with the average across all those years and the average confidence intervals across all years.

This was done in two ways. First, in Panel A, we aggregate our estimates using period data, as we do in the main text. The estimates that we calculate are highly similar to our main results. Second, in Panel B, we estimate effects using cohort effects, where the x-axis is the years relative to Medicare eligibility, to account for the fact that the pre-1901 cohorts did not receive Medicare at age 65.

As shown in the figure, the overall results are largely the same, though far more imprecise, as using cohort mortality typically relies on older data, which may not be as reliable and limits the number of available synthetic control units, based on when potential donors established their vital statistics infrastructure.

C.4.4 Additional Control Variables: Cumulative Cohort Survival (Appendix Figure A27)

A related exercise considers whether cumulative survival to age 65 could be driving our results. Unfortunately, there are meaningful data limitations, as the United States did not have a national vital statistics system until 1933. However, the Social Security Administration ("SSA") does have cohort survival estimates back to 1900. We do, however, caution that this data is imputed from a subset of states reporting mortality data and decennial censuses and is therefore of lower quality than the data in the HMD.

To account for cumulative survival, we perform the procedure detailed below:

1. We estimated the relationship between cumulative cohort survival and logged mortality rates using all non-U.S. units in the sample to avoid contamination. Specifically, we

estimate:

$$y_{acy} = \mu_c + \lambda_a + \beta \cdot survival_{cb} + \sum_a \gamma_a \cdot survival_{cb} \times \mathbb{I}(age = a) + \varepsilon_{acy},$$

where, a indicates an age, c indicates country, y indicates year of observation, and $b = y - a$ indicates the cohort. The variable *survival* indicates cumulative survival *to* either age 45 (the beginning of our synthetic control period) or to age 65 (the treatment time). We emphasize that this is survival to the beginning of an age, and could be phrased as survival *through* ages 44 and 64, respectively.

2. Using our estimates from above, we form residualized values, $\widetilde{y}_{acy} = y_{acy} - \hat{\beta} \times survival_{cb} + \sum_a \hat{\gamma}_a \times survival_{cb} \times \mathbb{I}(age = a)$ and calculate the country-by-age-by-era averages. Only the recent era (2000-19) is used, as it is the only one with the necessary data from the SSA.

3. We then perform our synthetic control procedure, using the average of our residualized values.

4. Inference was conducted by repeating Steps 1-3, above, with all other countries in the sample. We then used those placebo treatment effects to calculate our variance, as discussed in Section 3.

The results of this exercise are displayed in Appendix Figure A27, which shows separate estimates using survival to age 45 and survival to age 65. (We do not use survival past age 65, because it would be effectively controlling for an outcome variable.) As shown in the figure, our estimates are slightly larger than baseline estimates and well within the confidence intervals.

C.5 Further Evidence that the U.S. Social Safety Net is Not Driving Our Effects

Although income alone seems insufficient to explain the result we find, it is possible that access to social programs could change suddenly with respect to age in the United States. It is even possible that Medicare itself introduces people to certain forms of public assistance

(e.g., via woodwork effects). As with changes in income at the bottom of the distribution, any explanation involving the safety net must be able to scale to the population. That said, there are many safety net programs, and they target more vulnerable populations by construction. So, in Appendix Figure A28 we show how take-up of various programs changes as a function of age in the United States. Specifically, we plot the total dollar value of various programs collected in successive waves of the Current Population Survey Annual Social and Economic Supplement ("CPS ASEC"), which ask about varying sets of social programs during different time-periods. As shown in the figure, we see no indication of a discontinuous change or trend break in the dollar amounts of assistance received.

Perhaps just as importantly, we call attention to the magnitude of the y-axis–individuals aged 65 and over receive roughly $200 of assistance per capita during the post-2000 regimes. It is unlikely that government programs of this magnitude could explain a meaningful portion of what we find.

D Treatment Effect Parameterization

The purpose of this section is to discuss alternative functional forms for our treatment effect parameterization, including considerations that were attempted and discarded due to poor fit or unrealistic patterns. The parameter fit and counterfactual treatment paths for our preferred and alternative models are presented in Appendix Figure A20. The overall life-years saved/lost estimates associated with these models are presented in Appendix Figure A21.

At the end of this appendix section, we also discuss different weighting schemes during our estimation procedure, which reduce reliance on estimates further away from the cutoff. Estimates corresponding to other weighting schemes are presented in Appendix Figure A29.

Model Components

Survival: $f(S_a)$

Survival measures encompass the cumulative survival of the cohorts in our sample, and are taken from the SSA data discussed in Appendix Section C.4.4. Including these measures is useful because survival can proxy for the health stock of individuals, and it is possible that

returns on health investment vary based on that stock.

However, there are also reasons *not* to include cumulative survival controls. First, they are imperfect proxies for the underlying health distribution–which is one reason why we continue to include the "age-fade" term along side survival measures, as cohort survival is insufficient to handle aging dynamics by itself. Second, they are subject to measurement error due to interpolation, as discussed in Appendix Section C.4.4. While the benefits of inclusion likely outweigh the downsides (which is why survival rates are included in our preferred estimates), we do omit them in some of our models to test sensitivity.

Exposure: $g(d)$**, where** $d = \max\{0, 65 - age\}$

As discussed in Section 9, we selected a logistic exposure function as it models a reasonable pattern for health investments. With this function, marginal returns are increasing at first, but eventually the additional exposure starts to diminish (with the parameter d_0 representing the inflection point between increasing and decreasing marginal returns). Critically, the logistic function is also bounded.

Nonetheless, we also considered a power function, d^ρ. Such a function is appealing in its simplicity, with one fewer parameter to estimate. It also nests convex, linear, and concave returns to exposure, based on the estimated value of ρ. Nevertheless, it is not bounded, and thus the overall modeled treatment effect only reverses when the age-fade becomes relatively stronger. The impacts of this modeling decision can be seen in Appendix Figure A20, Panels C and D, where the overall treatment effect is notably more dramatic than our preferred specification.

We also considered a modified power function that explicitly bounded the treatment effect, $\min\{\bar{d}, d^\rho\}$. However, in practice, the estimates from this model yielded counterfactuals that reached the $\bar{d}$ value no matter what. For example, if $\bar{d} = 0.125$, the age-55 counterfactual gradually reached that threshold over the course of 25 years, or at a rate of roughly 0.005 log points per year. The age-75 counterfactual, on the other hand, reached the maximum within 5 years, or at a rate of 0.025 log points per year, five times the slope of the earliest counterfactual. This stark difference in slopes seemed unlikely to represent a realistic pattern, and thus we did not include this specification among the models presented in this paper.

Age-Fade: $h(a)$

The primary function used across all specifications is a Weibull survival function: $e^{-(a/\lambda)^k}$. We considered other functional forms, including a Gompertz survival function, $e^{-\eta(e^{bx}-1)}$, and including only cohort survival (discussed above), but those specifications did not converge to 0 at age 100, which should mechanically occur based on our life table assumptions. These deviations (typically) led to inflated estimates for counterfactual policies and are therefore not included among the models presented in this paper.

Alternative Weighting

As discussed in Section 9, our life-years saved estimates utilize differences between the United States and its synthetic control group from age 65 through age 100. However, a valid concern is that our synthetic control group may not be valid when calculating treatment effects at very advanced ages, given that the match occurred based on mortality patterns from ages 45 to 64.

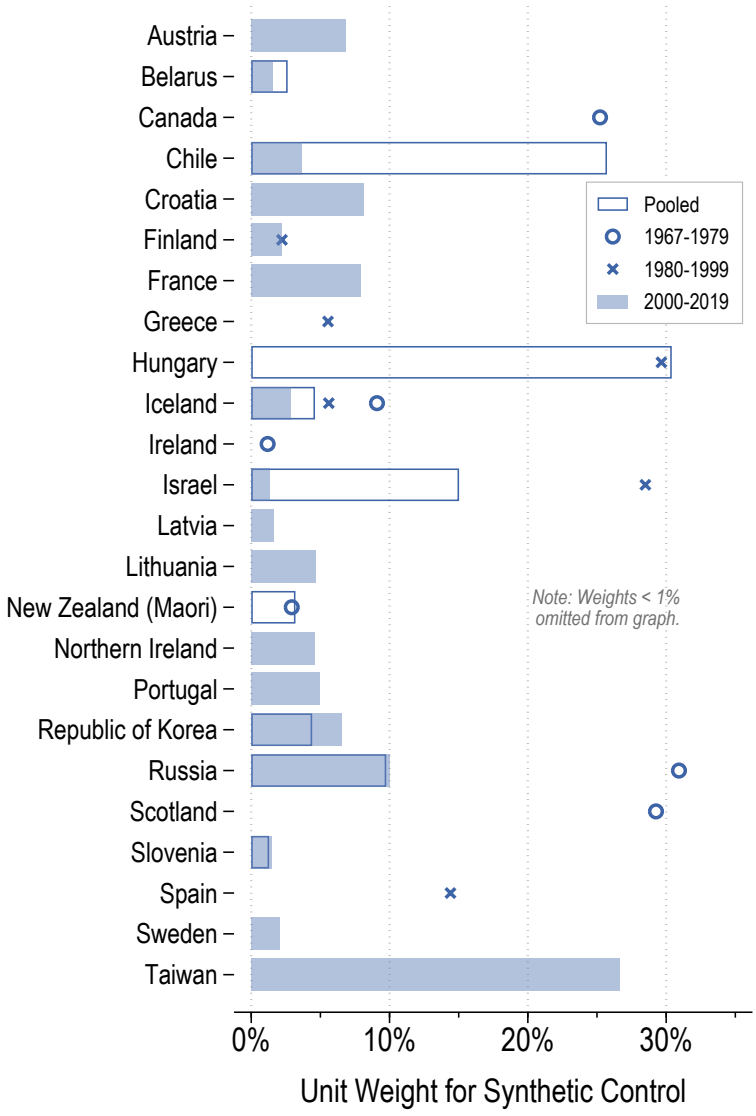
To address this concern, we fit our parameters to these treatment effects while down-weighting estimates from later ages. This was done in two ways.

The first alternative weighting method gives full weight to ages 45 through 85 (the main window used to display our results), *no* weight to estimates corresponding to ages 86 through 99, and full weight to age 100. The reason that age 100 is attributed full weight is that treatment effects must mechanically be equal as of that date, since our life-expectancy calculations assume full mortality at that age.

The second method uses triangular weighting, where full weight is given to observations closest to age 65, and that weighting decreases with distance from the Medicare eligibility cutoff, until it reaches zero after age 85. The exception to this is, again, age 100, which receives full weight for the reasons noted above.

In both scenarios, the estimated policy counterfactuals are extremely similar to our baseline. For our preferred scenario, the largest difference is 0.05 life years. Across all estimates and counterfactual policies, the average difference was 0.003 life years and the average *absolute* difference was 0.028 life years.

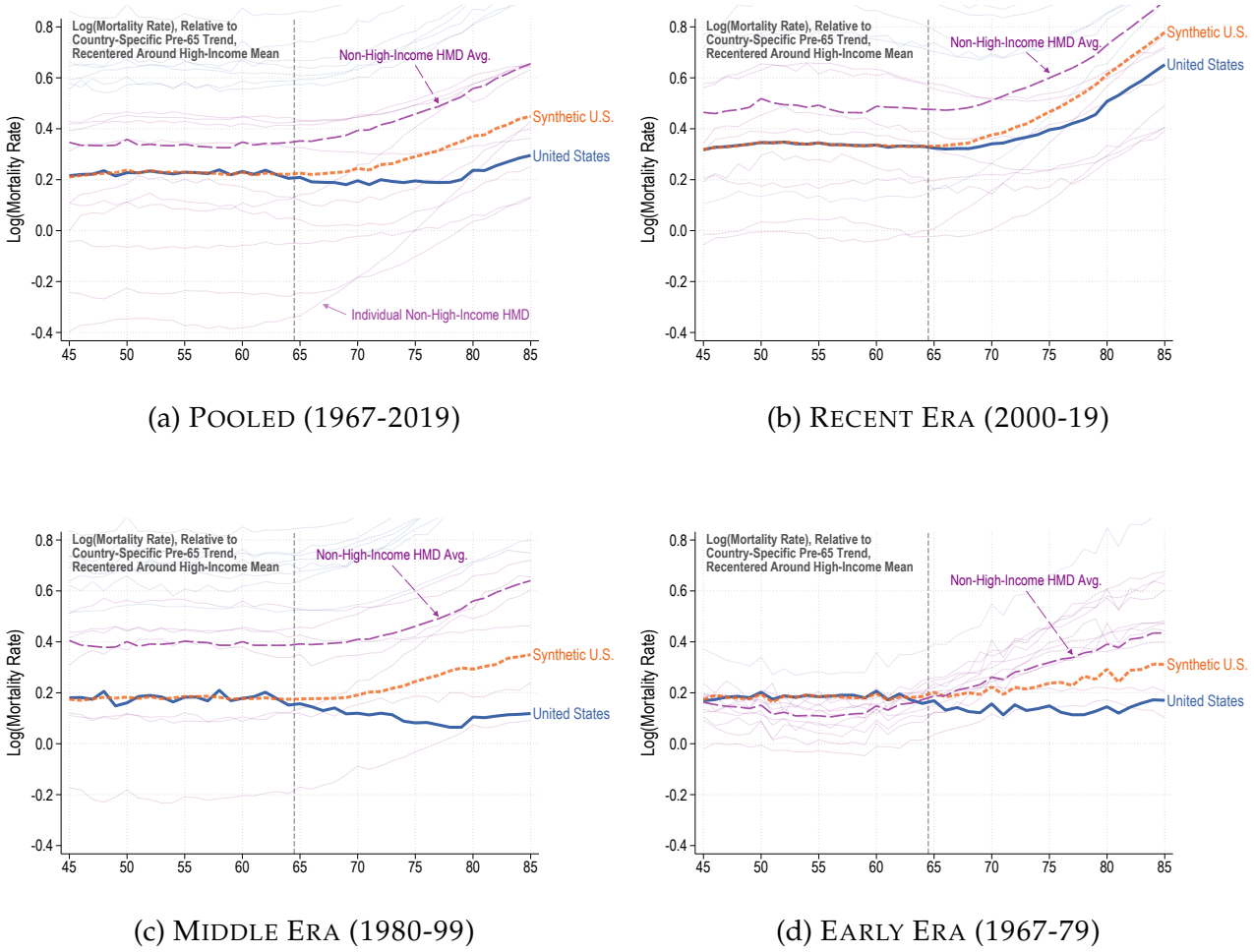
Figure A1: UNIT WEIGHTS FOR SYNTHETIC UNITED STATES



Notes: The purpose of this figure is to display unit weights for our synthetic control analysis on mortality (Figure 2).

Source: Author calculations using the Human Mortality Database (Human Mortality Database, 2026)

Figure A2: THE UNITED STATES' PRE-65 MORTALITY TRENDS AND LEVELS ARE COMPARABLE TO NON-HIGH-INCOME COUNTRIES, BUT DEPART AFTER AGE 65.



(a) POOLED (1967-2019)

(b) RECENT ERA (2000-19)

(c) MIDDLE ERA (1980-99)

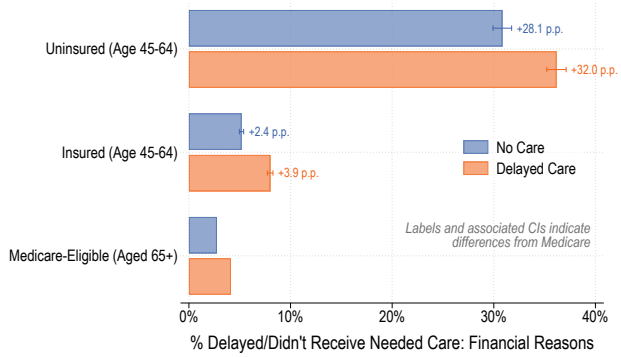
(d) EARLY ERA (1967-79)

Notes: The purpose of this figure is to recreate the analysis displayed in the right column of Figure 1, replacing high-income countries with the non-high income countries present in the Human Mortality Database. To maintain comparability with Figure 1, all countries remain re-centered around the *high-income* average, and the y-axis remains consistent. Series exceeding the maximum value of the original axis have been truncated.

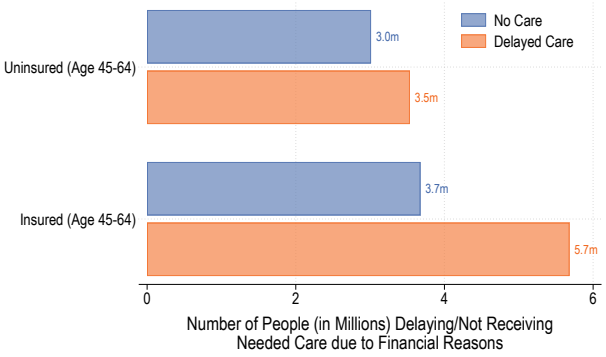
As in the original figure, countries were classified as "high-income" if they have been designated as such since the World Bank began publishing these classifications in 1987. Accordingly, this figure presents all countries who were ever classified as something *other* than high-income. (All HMD countries in this figure, except Ukraine, have been classified as at least middle-income since creation of these classifications.)

Source: Author calculations using the Human Mortality Database (Human Mortality Database, 2026)

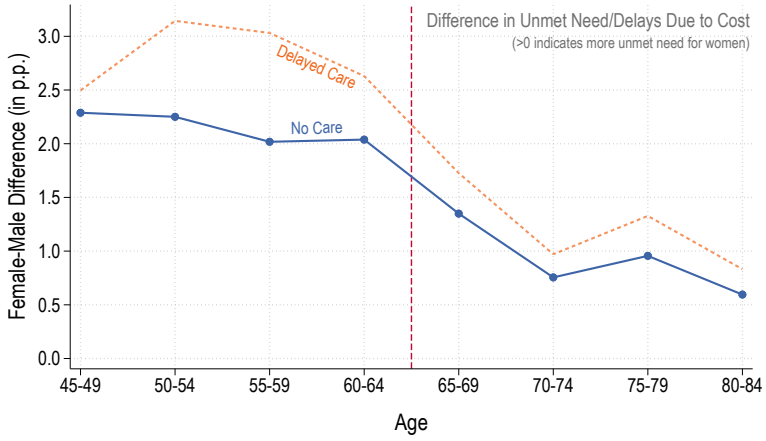
Figure A3: DETAILS OF UNMET NEED DUE TO FINANCIAL REASONS IN THE UNITED STATES



(a) RATES OF UNMET/DELAYED MEDICAL NEEDS (RAW AND RELATIVE TO MEDICARE)



(b) POPULATION W/ UNMET/DELAYED MEDICAL NEEDS (AGES 45-64)

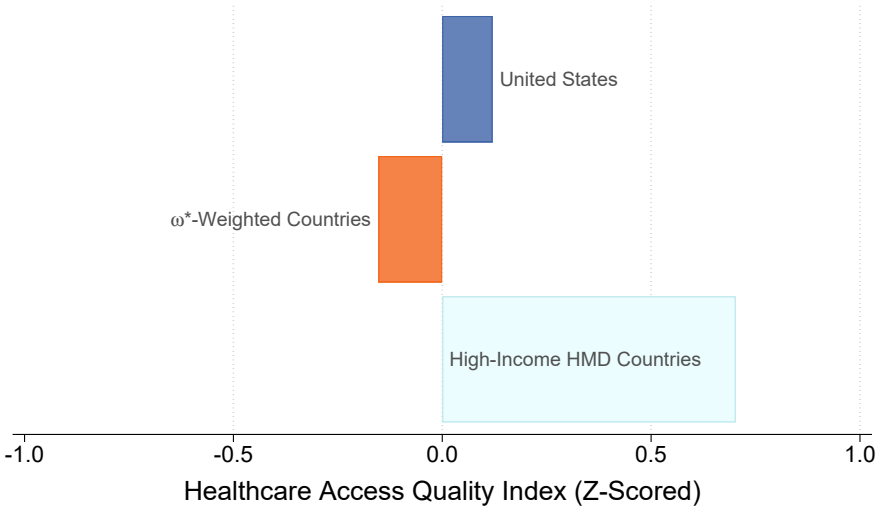


(c) FEMALE-MALE DIFFERENCE IN UNMET/DELAYED NEED DUE TO FINANCIAL REASONS

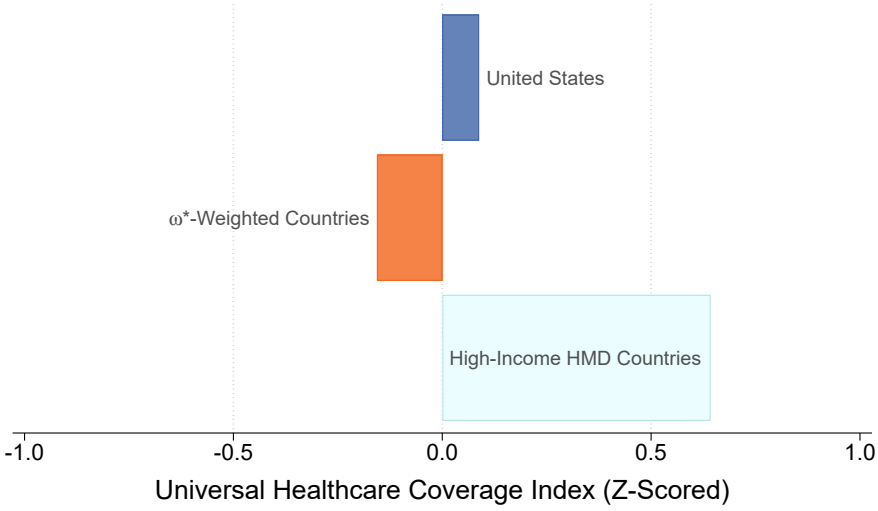
Notes: The purpose of this figure is to illustrate two primary points. First, as demonstrated by Panel A, insured individuals actually make up the majority of those with unmet medical needs due to cost considerations, a somewhat counterintuitive fact given their lower *rates* of unmet need (Panel B). However, this is case simply due to the relative size of the insured group vs. the uninsured. Panel B also demonstrates the the levels of unmet need for the 65+ group are lowest of all, suggesting that Medicare reduces financial barriers for the previously uninsured and previously insured populations alike. Second, as shown in Panel C, Women have higher levels of unmet need than men, both in terms of delaying care and foregoing care altogether. However, this gap shrinks markedly upon entry into Medicare, suggesting that women may obtain larger benefits relative to men.

Source: Author calculations using the National Health Interview Survey (Blewett et al., 2025*b*).

Figure A4: SYNTHETIC CONTROL GROUP USED IN MORTALITY ANALYSIS HAS SIMILAR MEASURES OF COVERAGE AND HEALTHCARE QUALITY



(a) HEALTHCARE ACCESS AND QUALITY INDEX



(b) UNIVERSAL HEALTHCARE COVERAGE INDEX

Notes: The purpose of this figure is to demonstrate the comparability of the United States' Healthcare system to the Synthetic United States constructed by applying the weights from our Recent-Era mortality analysis (ω). As shown in the figure, the United States' Healthcare system is much closer to the synthetic group than it is to other high-income countries, though it achieves a slightly better rating. This higher rating, however, is somewhat expected, given that the evaluation occurs across all age groups, and the U.S. departs sharply in terms of unmet need for those covered by Medicare (see Figure 3).

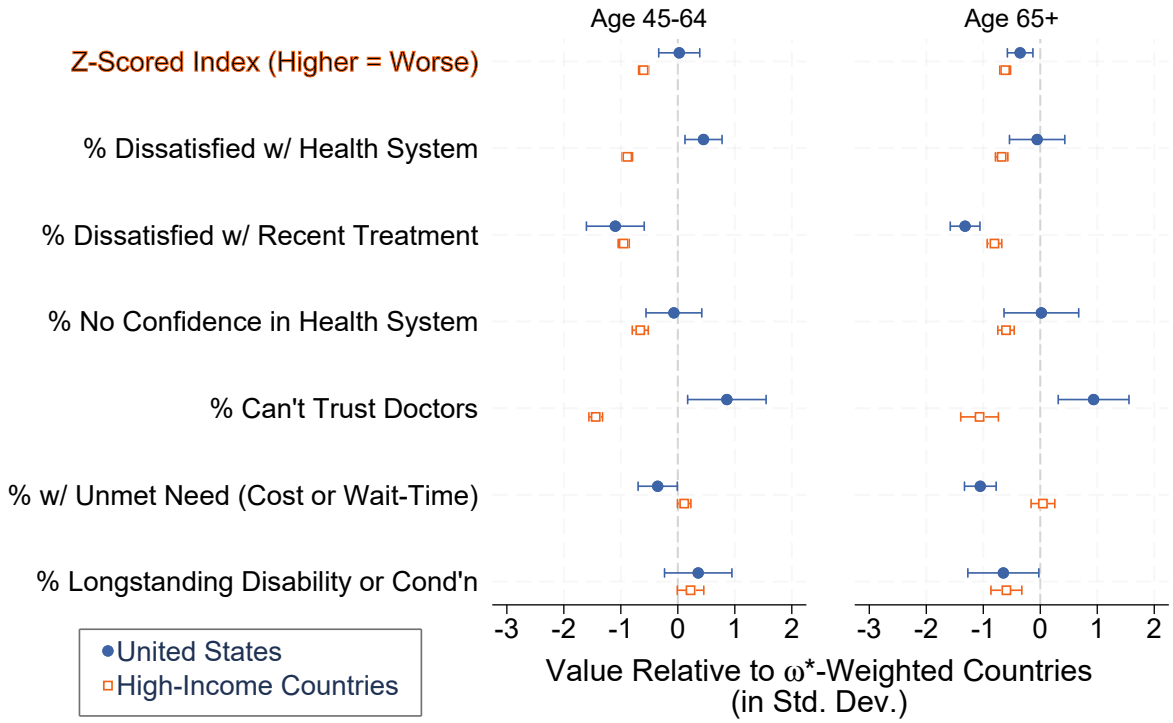
As not all donor countries were included in the Global Burden of Disease ("GBD") data, we upweighted according to the following formula:

$$\omega_i^* = \frac{\hat{\omega}_i}{\sum_{c \in GBD} \hat{\omega}_c}$$

where $\sum_{c \in GBD} \hat{\omega}_c = 99.7\%$. See Figure 3 for further discussion.

Source: Author calculations using Global Burden of Disease (Global Burden of Disease Collaborative Network, 2020, 2025).

Figure A5: SYNTHETIC CONTROL GROUP USED IN MORTALITY ANALYSIS HAS SIMILAR ISSP HEALTHCARE-RELATED MEASURES



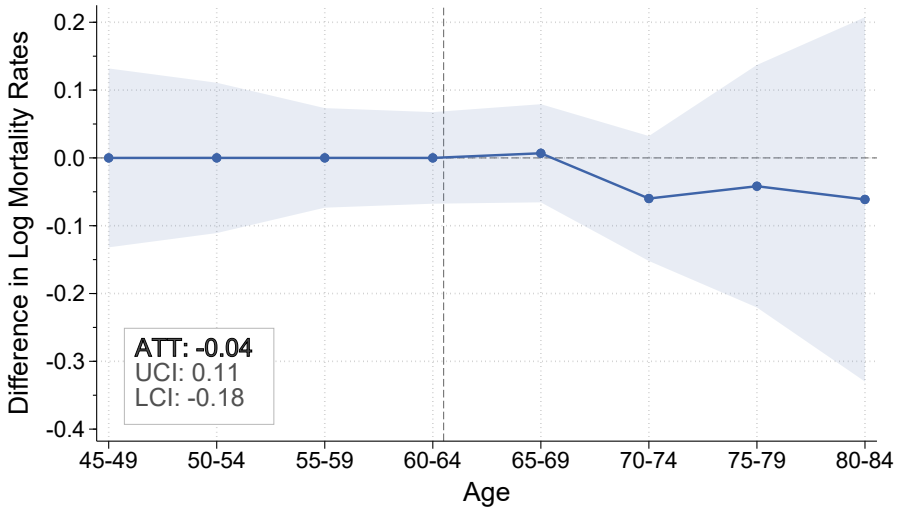
Notes: The purpose of this figure is to demonstrate the comparability of the characteristics pertaining to health and healthcare in the United States' to those of the Synthetic United States constructed by applying the weights from our Recent-Era mortality analysis (ω). As shown in the figure, the U.S. characteristics are much closer to the synthetic group than it is to other high-income countries. The top line, "Z-Scored Index" is an average of the other measures in the figure, all of which are themselves z-scored. The high-income countries in the figure are based on World Bank classifications, and include only those also included in the HMD (for comparability). The comparison group is the group of countries that serve as donors for our recent-era mortality analysis. As not all donor countries were included in the International Social Survey Programme ("ISSP"), we upweighted according to the following formula:

$$\omega_i^* = \frac{\hat{\omega}_i}{\sum_{c \in ISSP} \hat{\omega}_c}$$

where $\sum_{c \in ISSP} \hat{\omega}_c = 56.9\%$. See Figure 3 for further discussion.

Source: Author calculations using International Social Survey Programme data (ISSP Research Group, 2024).

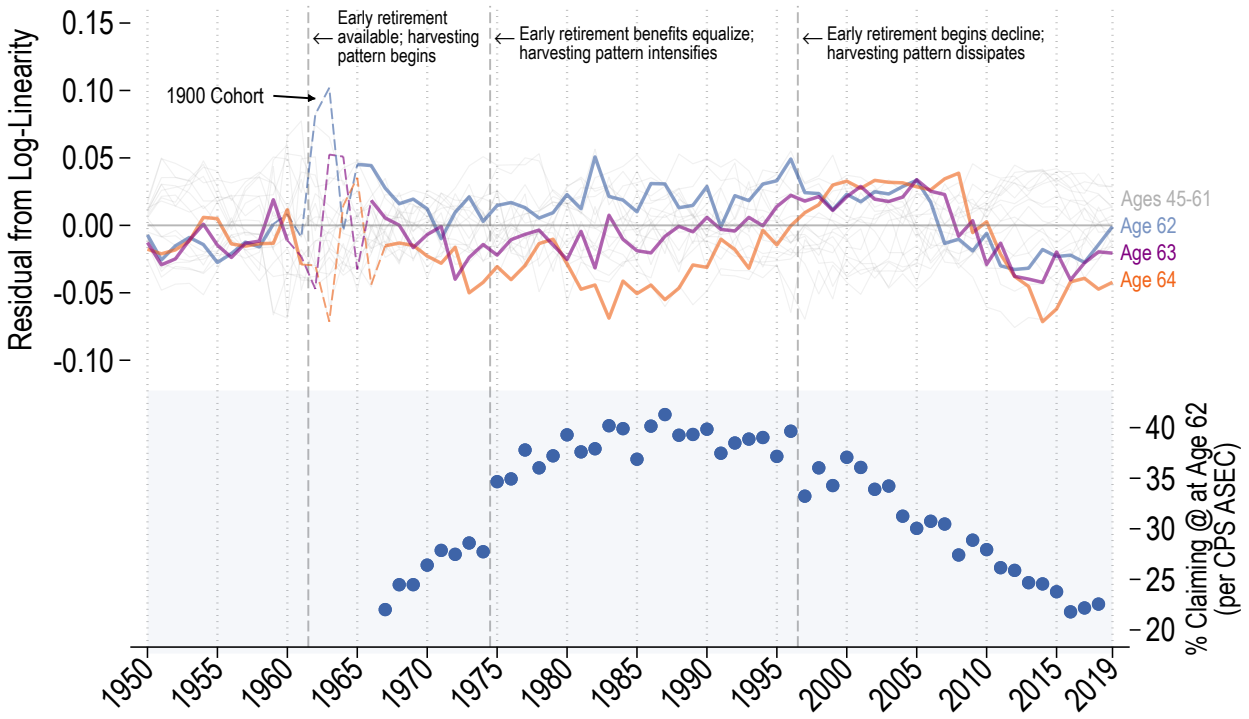
Figure A6: THERE IS NO SIGNIFICANT DEPARTURE FROM THE PRE-65 MORTALITY TREND DURING THE YEARS PRIOR TO MEDICARE'S IMPLEMENTATION



Notes: This figure demonstrates the difference in between the U.S. and its synthetic counterpart during the years from 1955-1965, which were prior to Medicare's implementation. Data from the World Health Organization Mortality Database, which groups deaths into five-year age-bins but has better coverage during this time period, was used for this exercise.

Source: Author calculations using the WHOMD (2026).

Figure A7: Decreased Mortality Just Prior to the Age-65 Cutoff Is Likely Due to Harvesting Related to Retirement Effects

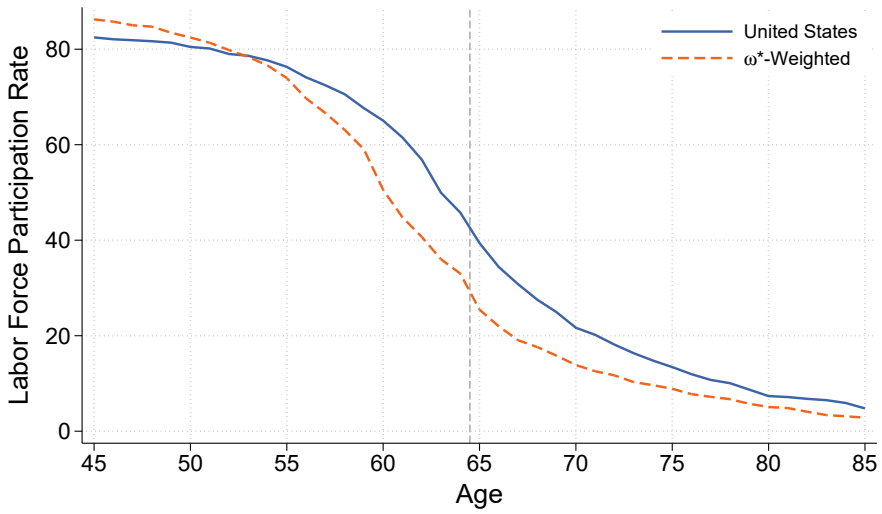


Notes: The purpose of this figure is to address pre-Medicare decreases in ages 63 and 64, which are likely related to the "harvesting" of deaths connected to retirement spikes at age 62.

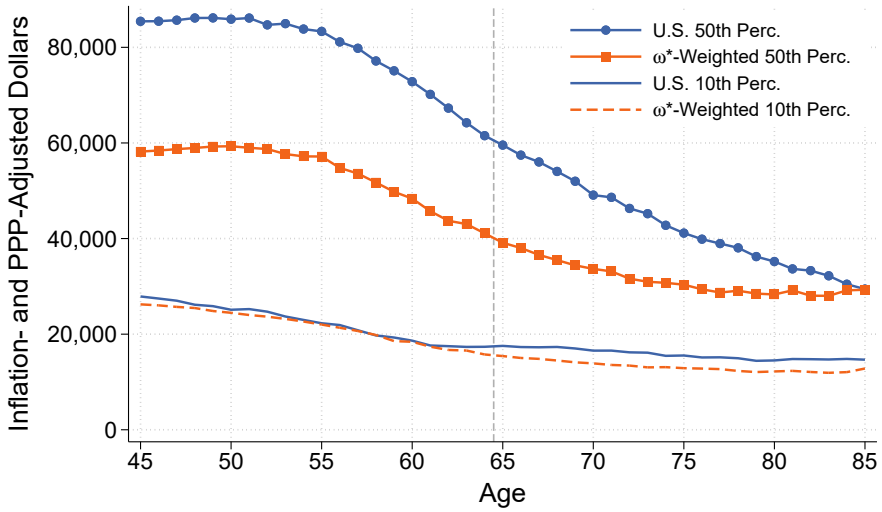
The top half of the figure displays residuals of age-specific U.S. mortality relative to a linear trend, fit on each year. The blue, purple, and orange lines represent key ages of 62, 63, and 64, respectively. Light gray lines represent all other ages for context. The bottom half of the figure displays, for context, the percentage of population receiving *any* social security income, back to the earliest year in which this data is available per the Current Population Survey Annual Social and Economic Supplements ("CPS ASEC"). See Appendix Section C for more detail.

Source: Author calculations using Human Mortality Database (2026) data and the Current Population Survey Annual Social and Economic Supplements (Flood et al., 2025).

Figure A8: Synthetic Control Group Used in Mortality Analysis Has Similar Trends in Labor Force Participation and Income



(a) Labor Force Participation



(b) Median and Low-Income

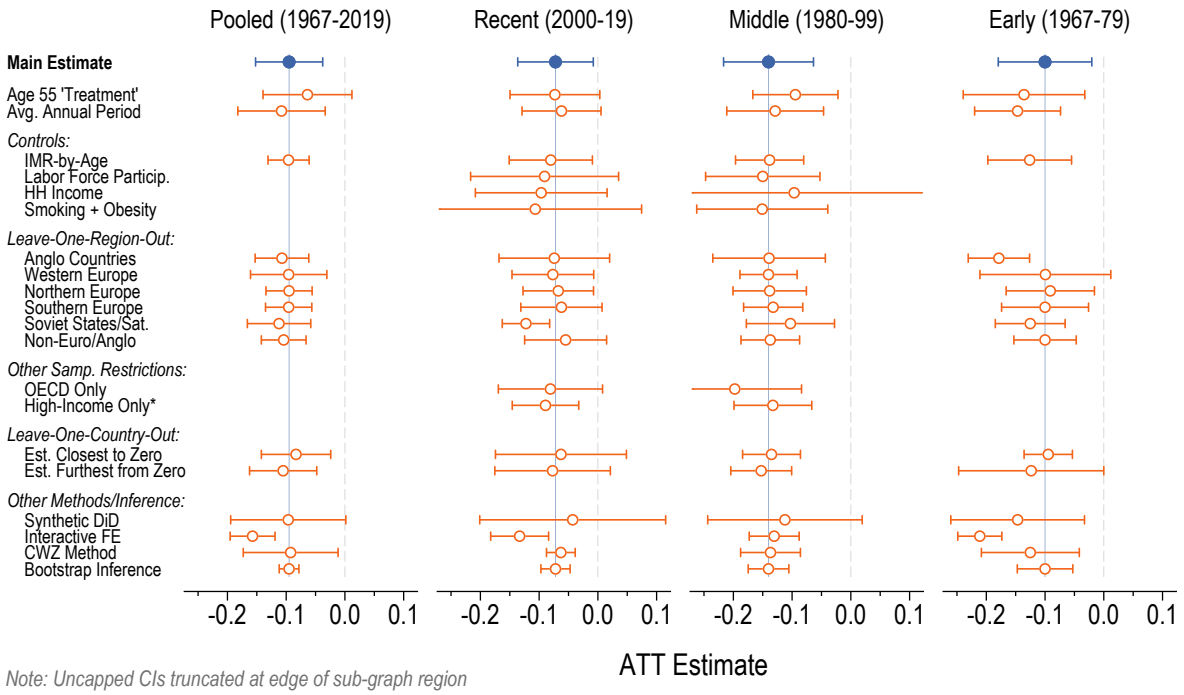
Notes: The purpose of this figure is to illustrate the underlying trends in labor force participation and household income for the United States and its synthetic counterpart, which is formed by applying the weights from our Recent-Era mortality analysis (ω). As show in the figure, while the United States generally differs in levels, these variables tend to be moving in parallel across the age-65 cutoff, when U.S. mortality diverges from the synthetic control group. As not all donor countries were included in the underlying datasets, we upweighted according to the following formula:

$$\omega_i^* = \frac{\hat{\omega}_i}{\sum_{c \in Data} \hat{\omega}_c}$$

where $\sum_{c \in Data} \hat{\omega}_c = 79.7\%$. See Figure 3 for further discussion.

Source: Author calculations using Luxembourg Income Study data and harmonized data from IPUMS International (Ruggles et al., 2025*a*).

Figure A9: Findings are Robust to a Variety of Specifications, Sample Constructions, Control Variables, and Methods



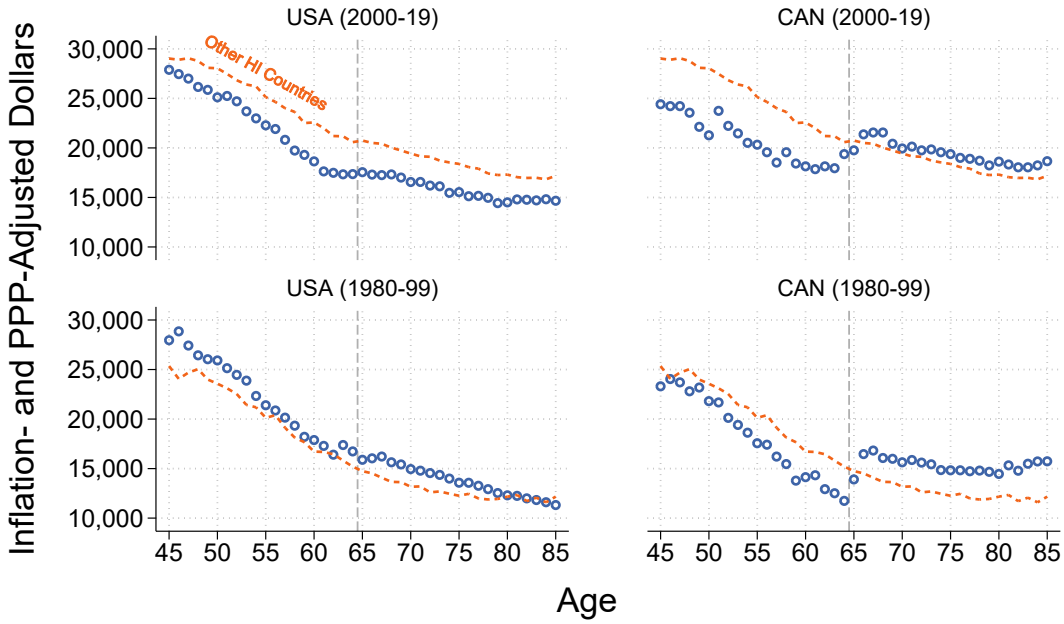
Notes: The purpose of this figure is to demonstrate robustness of our main results. Each line/section of the graph is described briefly below.

- **Age 55 'Treatment'**: represents the average of the post-65 coefficients and confidence intervals when the treatment time for the synthetic control estimator is set to age 55. As demonstrated by Appendix Figure A26, the effect continues to manifest along approximately the same trajectory as when the treatment date is set at age 65.
- **Avg. Annual Period**: represents the average ATT coefficients and confidence intervals when each year from 1967-2019 was estimated separately. See Appendix Figure A26 for display of average and individual-year trajectories by age.
- **Controls**: represents our estimates when including various control variables. For each set of variables, we utilize the method outlined in Clarke et al. (2024), where a two-way fixed-effects regression, $y_{ac} = X_{it}\beta + \mu_c + \theta_a + \varepsilon_{ac}$ using only the untreated units. Those estimates are then use to form a residualized outcome, $\widetilde{y_{ac}} = y_{ac} - X_{it}\hat{\beta}$, which is then used as the basis for our synthetic control analysis.

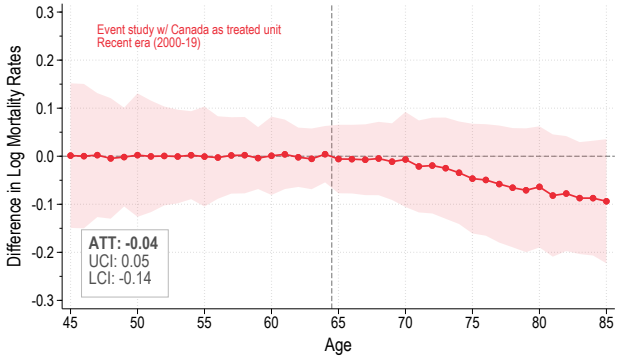
  IMR-by-Age is included as infant mortality is a benchmark for overall health system quality and is also sensitive to period shocks that might affect older adults. This control was formed by interacting interacting log(infant mortality rates) with age, as disease environment and health system quality are likely to have heterogeneous effects over the life-cycle. **Labor Force Participation** and **HH Income** were included to capture impact of economic factors, we included controls for labor force participation and household income (both at the median and the 10th percentile) for each country-by-age-by-era cell. **Smoking and Obesity** covariates were included to capture the extent to which other health risk factors may contribute to our results. These represent the percentage of population that was obese, as well as an estimate of lifetime cigarettes smoked, both of which were obtained at the country-by-age-by-era level.
- **Leave-One-Region-Out**: represents our analyses when the listed region is *omitted* from the set of potential donors. Non-U.S. "Anglo" countries include AUS, CAN, GBR, and NZL. Soviet States/Satellites include any country that was formerly part of the Soviet Union (BLR, EST, LVA, LTU, RUS, UKR) or one of its satellite states (BGR, CZE, SVK, ROU, HUN, POL, and East Germany). All other European countries were classified according to the United Nations M49 designation.
- **Other Samp. Restrictions**: represents our analyses when *limiting* our sample to certain groups. **OECD Only** limits potential donors to countries that were part of the OECD for the entire era. Note that estimates are only presented for the recent and middle eras, as the early and pooled eras did not have a strong pre-65 fit. **High-Income Only*** limits to countries that were classified by the World Bank as high-income during the entire era. Note that the World Bank has only included such classifications since the 1980s, and thus, these estimates are only available for the middle and recent eras. *The recent-era synthetic control estimate and confidence intervals have been adjusted to recenter the pre-period around zero, as U.S. mortality is outside the convex hull of donor countries. However, the trend is parallel to zero prior to age 65, suggesting this adjusted value is comparable to other estimates.
- **Leave-One-Country-Out**: displays estimates from an exercise where we drop one country from the potential donor pool. The estimates presented are the two most extreme from this exercise–one where the point estimate is closest to zero (smallest absolute value), and the other where it is furthest (largest absolute value).
- **Other Methods/Inference**: displays our results when choosing a method different than synthetic control or changing our strategy for inference. **Synthetic DiD** estimates effects using the synthetic difference-in-differences method developed by Arkhangelsky et al. (2021) using the `sdid` package discussed in Clarke et al. (2024). **Interactive FE** estimates effects using the interactive fixed effects methods developed by Bai (2009) and applied using the `fect` package discussed in Liu, Wang and Xu (2024). Inference was calculated using jackknife, as that was more conservative. Estimates on the **CWZ Method** line utilized newly developed synthetic control estimation and inference methods outlined in Chernozhukov, Wüthrich and Zhu (2021, 2025). Finally, **Bootstrap Inference**: estimates confidence intervals using a bootstrap method, rather than the placebo inference used throughout the rest of this paper.

Source: Author calculations using Human Mortality Database (2026), WHO Mortality Database (World Health Organization, 2026), Luxembourg Income Study, harmonized data from IPUMS International (Ruggles et al., 2025*a*), Global Burden on Disease (2025), World Bank, and OECD.

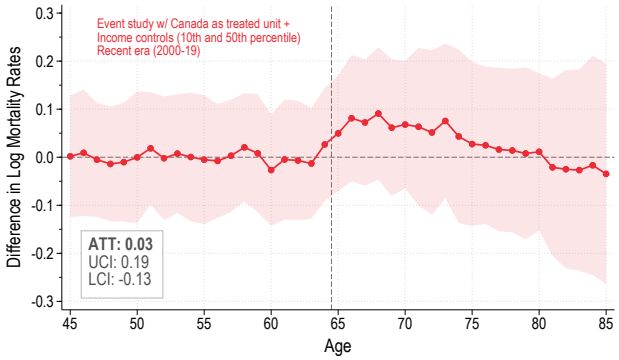
Figure A10: CANADA'S POST-65 REDUCTION IN MORTALITY LIKELY DRIVEN BY SHARP CHANGE IN SOCIAL SAFETY NET FOR LOW-INCOME HOUSEHOLDS



(a) 10TH-PERCENTILE OF HH INCOME: U.S. AND CANADA



(b) SYNTHETIC CONTROL W/ CAN AS THE TREATED UNIT (2000-19)

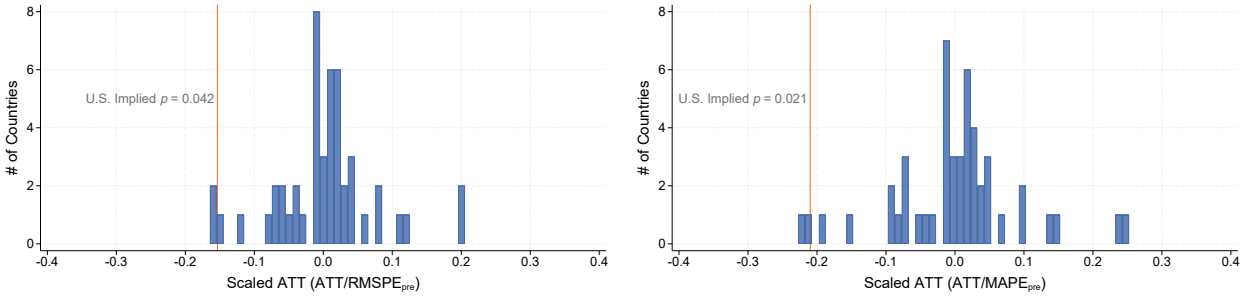


(c) SYNTHETIC CONTROL W/ CAN AS THE TREATED UNIT, CONTROLLING FOR INCOME (2000-19)

Notes: The purpose of this figure is to further examine Canada, which is consistently one of the countries exhibiting negative reductions in mortality after the age of 65 (See Appendix Figure 5). This is likely because low-income Canadians experience a sharp increase in income when they reach age 65, relative to the United States and other high-income countries (Panel A). This hypothesis is further confirmed by Panels B and C, which show that the reduction in post-65 mortality among Canadians disappears when income controls for the 10th and 50th percentile of income–i.e., the same controls used in Appendix Figure A9–are added. (Performing the same exercise for the Middle Era yields qualitatively similar results, which are available upon request.) Notably, application of these same controls to the United States does not nullify the treatment effect, supporting the hypothesis that the impacts we identify are attributable to Medicare, rather than later-life income from Social Security or some other source.

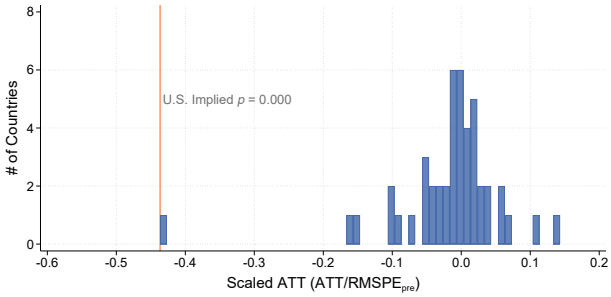
Source: Author calculations using data from the Human Mortality Database (2026), Luxembourg Income Study, and IPUMS International (Ruggles et al., 2025*a*).

Figure A11: Results are Statistically Significant According to Placebo-Inference Measures

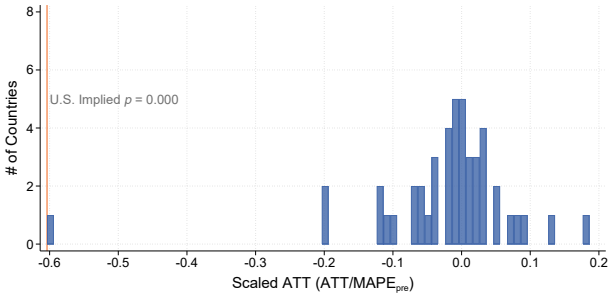


(a) RMPSE-Scaled ATTs (1967-2019)

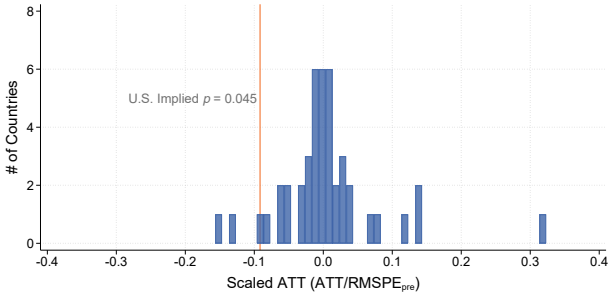
(b) MAPE-Scaled ATTs (1967-2019)



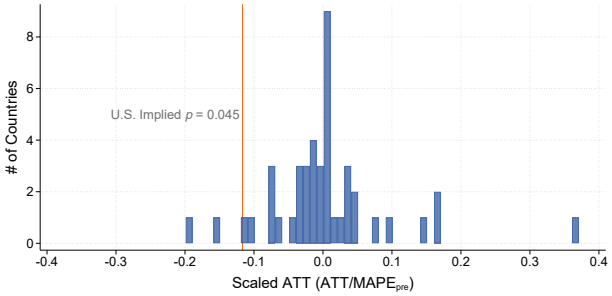
(c) RMPSE-Scaled ATTs (2000-19)



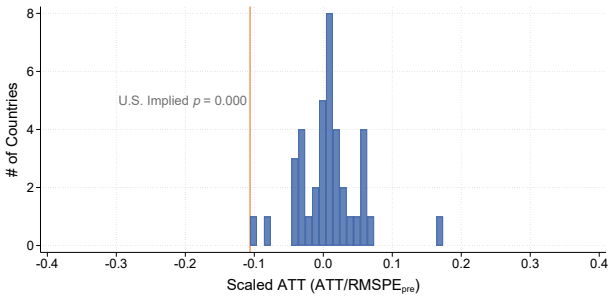
(d) MAPE-Scaled ATTs (2000-19)



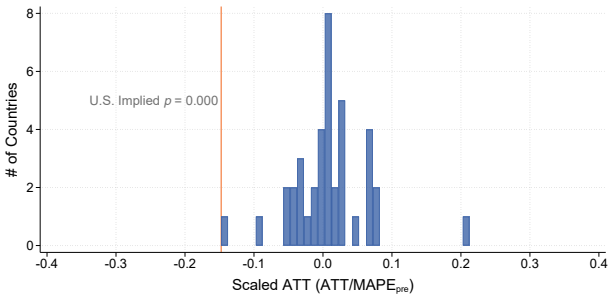
(e) RMPSE-Scaled ATTs (1980-99)



(f) MAPE-Scaled ATTs (1980-99)



(g) RMPSE-Scaled ATTs (1967-79)



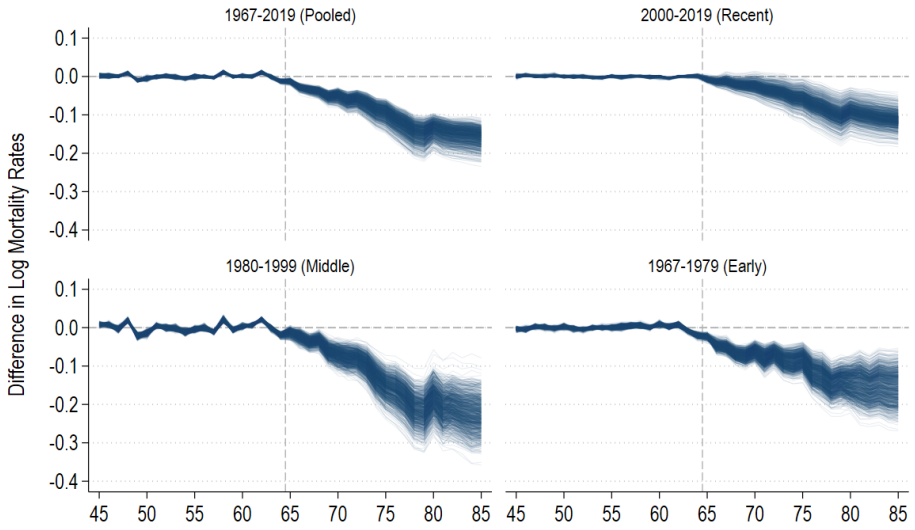
(h) MAPE-Scaled ATTs (1967-79)

Notes: The purpose of this figure is to illustrate results of placebo inference. Each unit in the figure is an estimate, generated by assigning treatment to a different country during the period. The average treatment effects from this exercise as then scaled by a measure of pre-period fit, to penalize units that do not have good synthetic control matches. In the left column, the measure of pre-period fit is root mean squared prediction error ("RMSPE"). In the right column, the measure is mean absolute prediction error ("MAPE"), which penalizes outliers less, but also has a lower "reward" for units with exceptional pre-period fit.

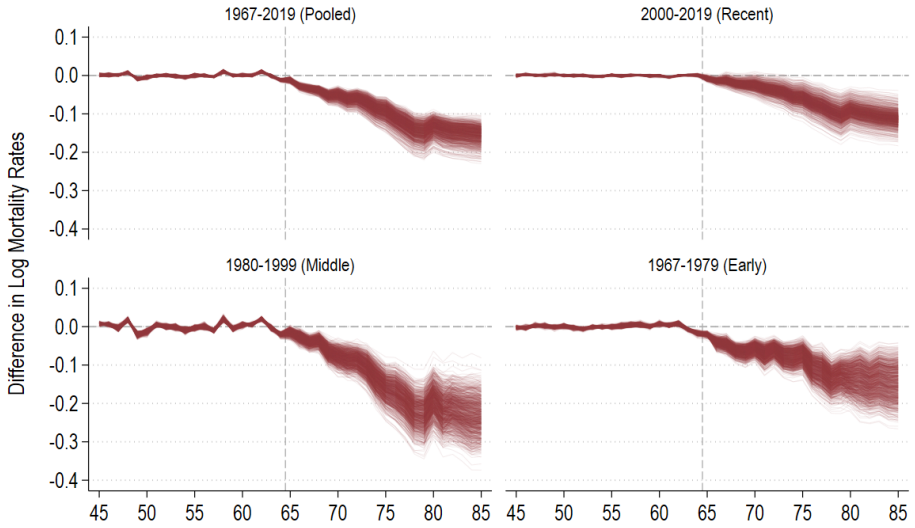
The p-values listed in the figure are one-sided estimates of $Pr(ATT^{scaled}_{placebo} < ATT^{scaled}_{usa})$. In all specifications, the U.S. achieves statistical significance of at least 95%.

Source: Author calculations using the Human Mortality Database (2026).

Figure A12: Robustness When Applying Lasso and Elastic Net to Combinations of the Donor Pool



(a) Lasso-Selected Synthetic Control Group



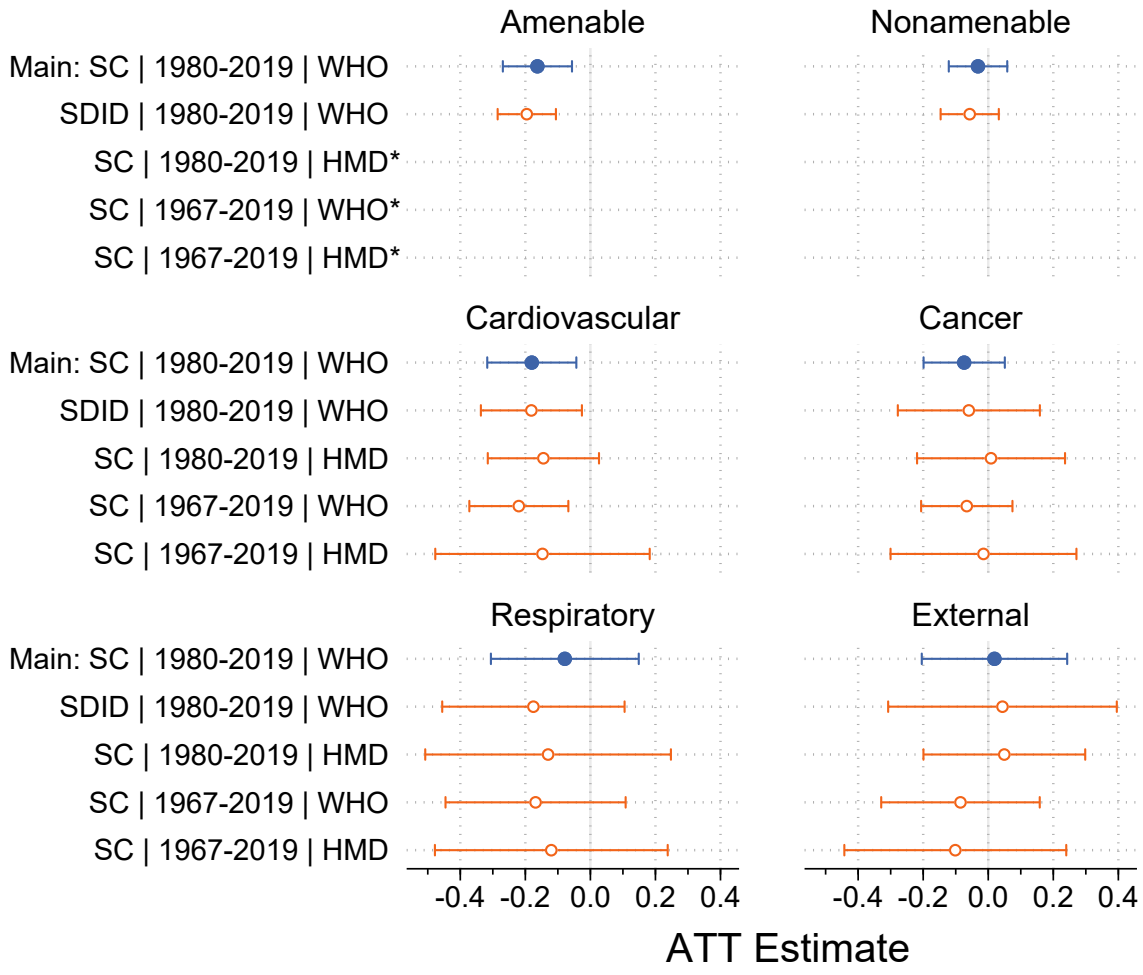
(b) Elastic Net-Selected Synthetic Control Group

Notes: The purpose of this figure is to demonstrate robustness along two dimensions. First, we utilize two regularization / sample-selection methods to select our control group, in the spirit of Hollingsworth and Wing (2022), to address concerns about overfitting when there are more potential donor units than pre-treatment periods. While Hollingsworth and Wing (2022) utilize Lasso, we also include a version using elastic net (with $\alpha = 0.5$), it often performs better than Lasso in settings with highly correlated predictors. (Country-level mortality, as shown in the righthand column of Figure 1, is highly correlated, as the age-mortality gradient is determined by both global trends and underlying biological process.

We combine this exercise with another procedure to address potential overfitting and increase confidence that a particular choice of synthetic control group is not driving our results. Namely, we estimate the Lasso/elastic-net regressions using 1,000 random 50-percent samples of our donor pool and plot each result with 10% opacity. As shown in the figure, every randomly selected donor group gives a similar effect as our main analysis.

Source: Author calculations using the Human Mortality Database (2026).

Figure A13: Main Cause of Death Results are Robust to Different Estimation Methods and Data Sources

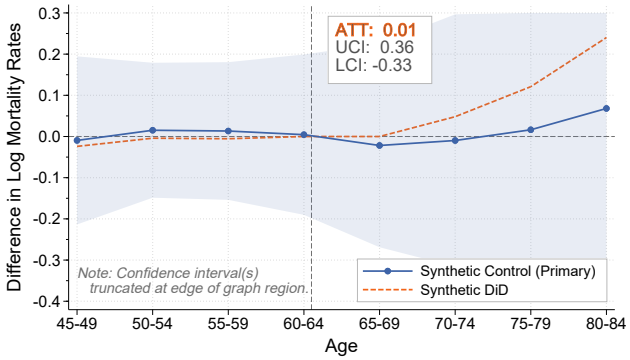


Notes: This figure demonstrates robustness of our cause-of-death results to different methods, eras, and datasets. Within the figure, 'WHO' indicates that the WHO Mortality Database was used, while 'HMD' indicates that the Human Mortality Cause of Death Database was utilized.

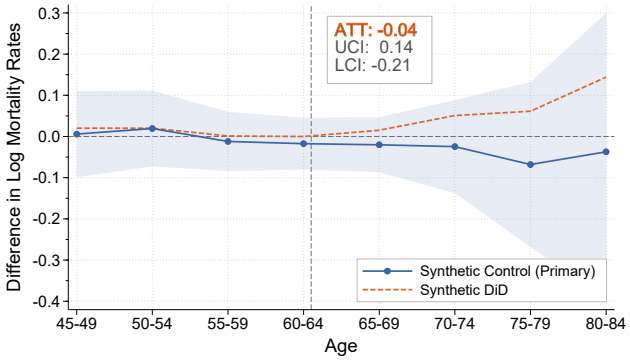
*Amenable and non-amenable estimates do not exist for the full period, as ICD-9 classifications only became commonplace around 1980. We do not repeat this analysis using the HMD, because only 11 countries in the database have sufficient cause-of-death detail during the 1980-2019 period.

Source: Author calculations using the WHO Mortality Database (World Health Organization, 2026), Human Cause-of-Death Data series (2026), and list of treatment-amenable conditions from Nolte and McKee (2003, 2011).

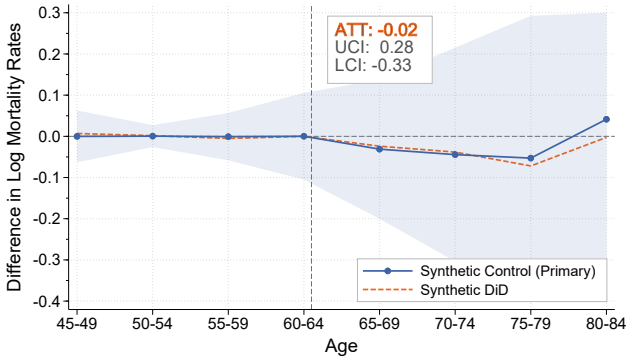
Figure A14: Medicare's Impact on Less Common Causes of Death



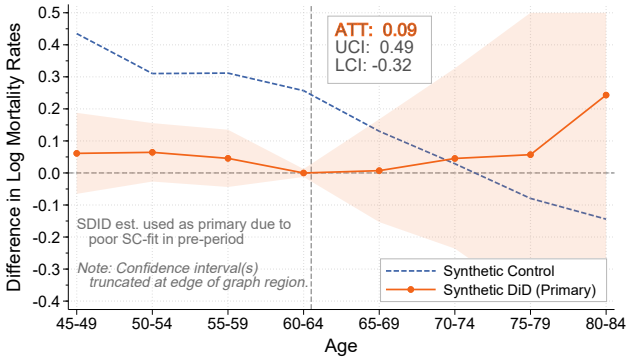
(a) Diseases of the Nervous System



(b) Diseases of the Digestive System



(c) Diseases of the Genitourinary System

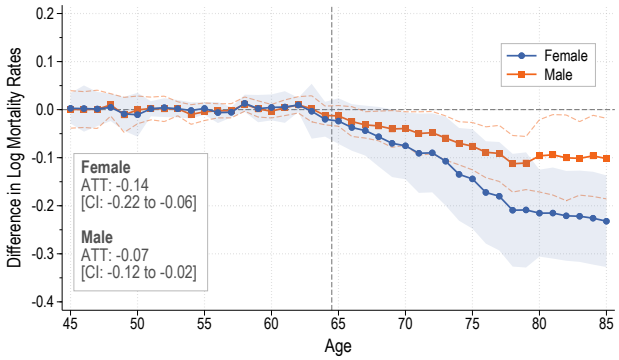


(d) Diseases of the Blood

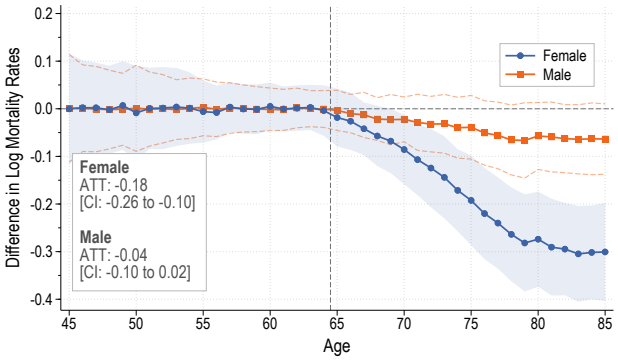
Notes: This figure demonstrates the impact of Medicare on mortality for additional causes of death, which are the four most common causes that weren't included in Figure 6. Together, these four causes comprise approximately 9.3% of all deaths in the United States among those aged 65 and older. Within the figure, both synthetic control and synthetic difference-in-differences estimates are presented, though the ATT is only presented for synthetic control. Panel D is an exception to this, because of the poor pre-period synthetic control fit–in this case, the ATT for SDID is presented.

Source: Author calculations using the WHO Mortality Database (World Health Organization, 2026) and list of treatment-amenable conditions from Nolte and McKee (2003, 2011).

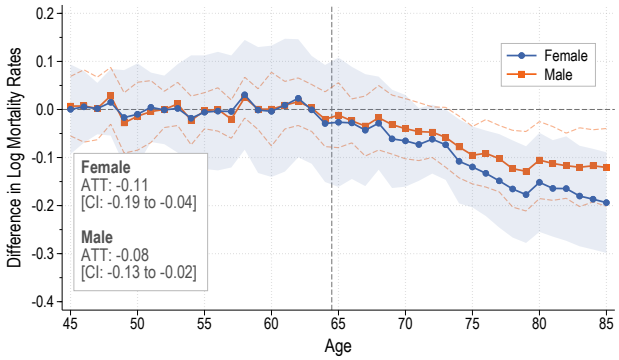
Figure A15: MEDICARE HAS A LARGER IMPACT ON FEMALE MORTALITY



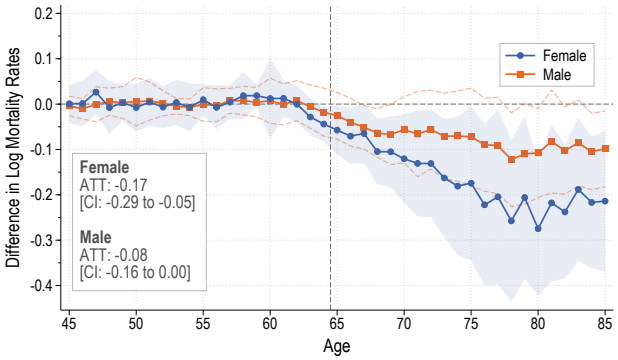
(a) POOLED (1967-2019)



(b) RECENT ERA (2000-19)



(c) MIDDLE ERA (1980-99)

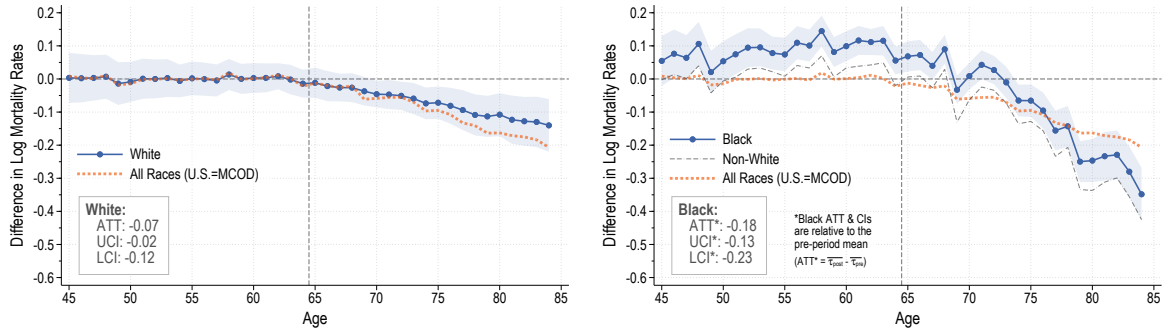


(d) EARLY ERA (1967-79)

Notes: The purpose of this figure is to demonstrate the differential effects of Medicare by sex.

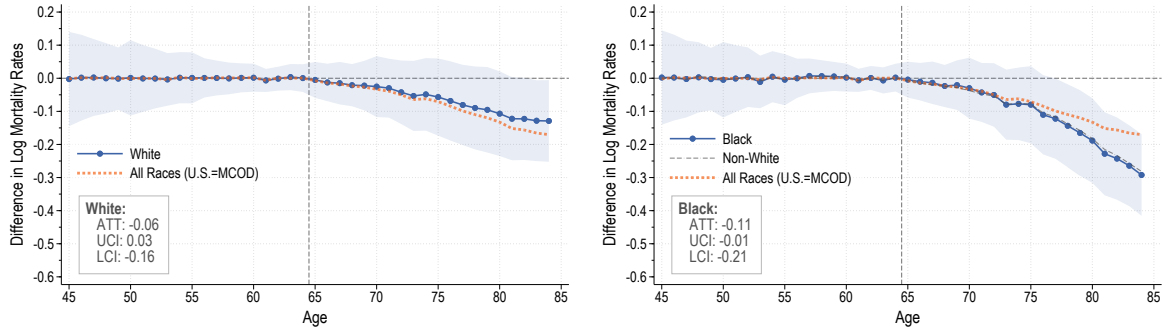
Source: Author calculations using the Human Mortality Database (2026).

Figure A16: Medicare Has a Larger Impact on Black Individuals



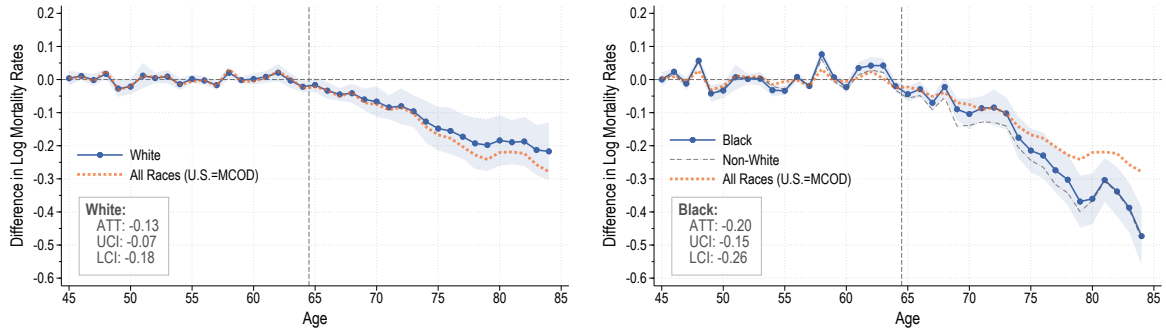
(a) White (1967-2019)

(b) Black (1967-2019)



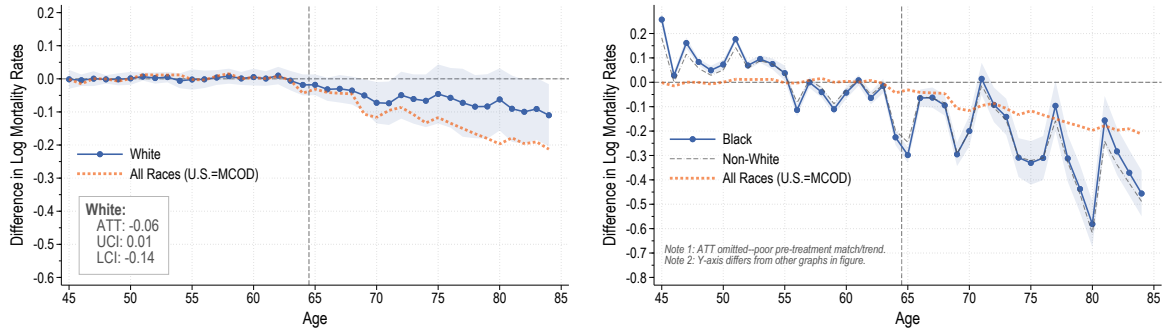
(c) White (2000-19)

(d) Black (2000-19)



(e) White (1980-99)

(f) Black (1980-99)



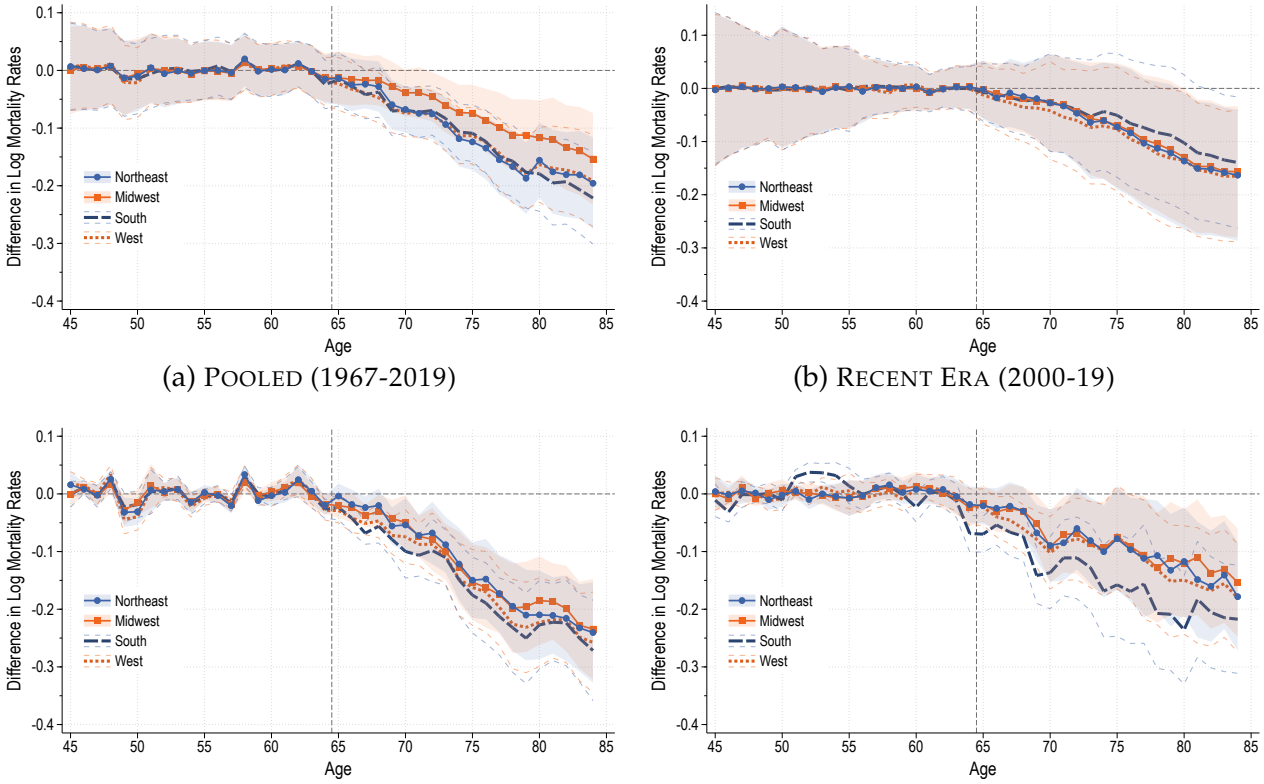
(g) White (1967-79)

(h) Black (1967-79)

Notes: The purpose of this figure is to display effects by race, where estimates are derived by matching race/ethnicity-specific mortality in the United States with *all-race* mortality of other countries. (To be clear, the race-specific mortality of the United States is the only treated unit in each analysis and are therefore analyzed separately.) In the figure, 'White' refers to all individuals identifying as white, regardless of Hispanic ethnicity. (Ethnicity was not widely available in the MCOD until the middle of our sample period. However, when estimating our later eras using only non-Hispanic white individuals, our estimates remain extremely similar.) Estimates for Black individuals are presented alongside non-white, and are statistically indistinguishable in all cases except for one–the pooled period. In that case, the mortality for Black individuals is outside the convex hull for donor countries, but the pre-post difference is nearly identical to the non-white ATT.

Source: Author calculations using the U.S. Multiple Cause of Death Data (Vital Statistics) and Human Mortality Database (2026).

Figure A17: MEDICARE HAS A CONSISTENT EFFECT ACROSS CENSUS REGIONS



(a) POOLED (1967-2019)

(b) RECENT ERA (2000-19)

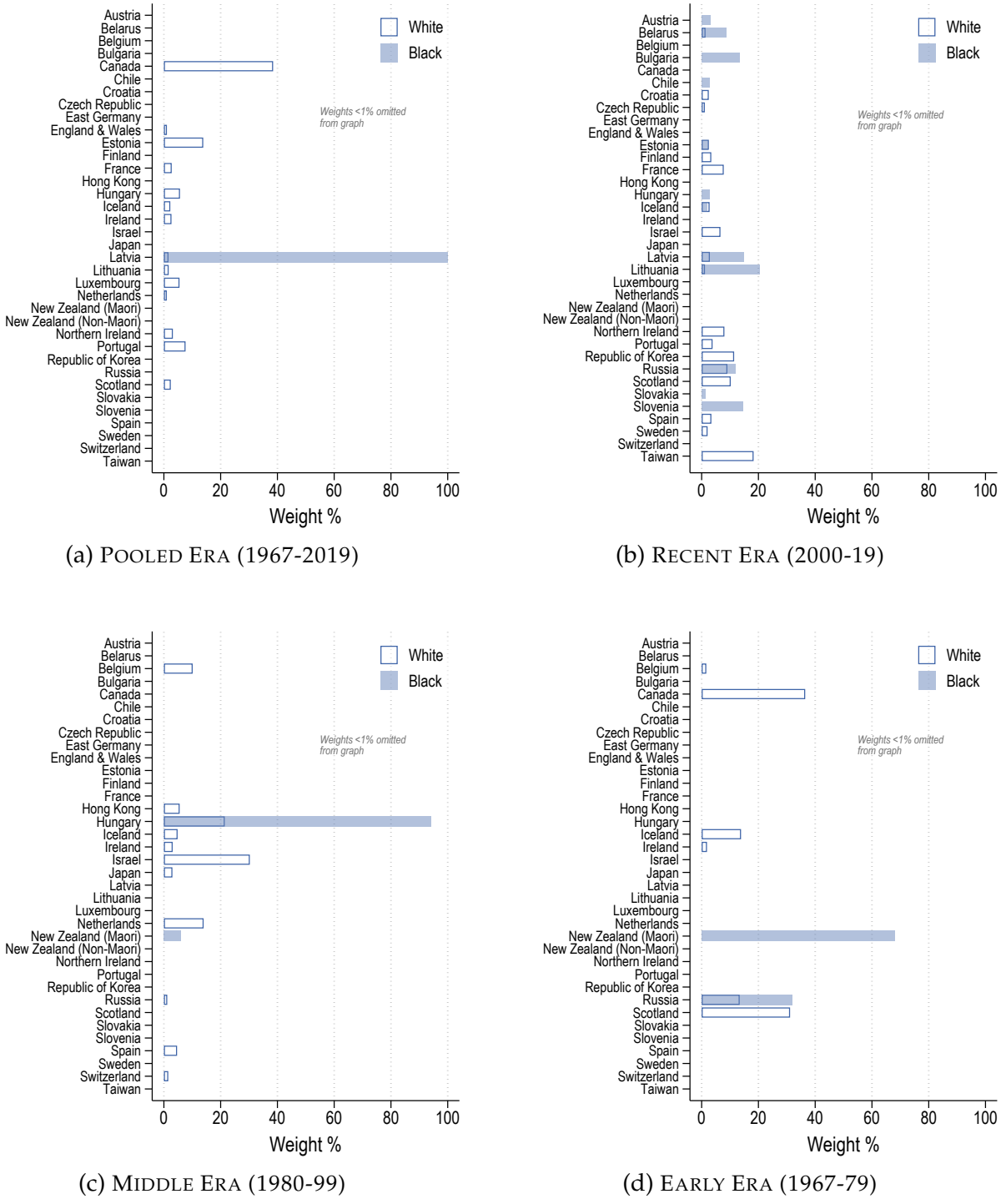
(c) MIDDLE ERA (1980-99)

(d) EARLY ERA (1967-79)

Notes: The purpose of this figure is to display effects by U.S. Census Region, where estimates are derived by matching region-specific mortality in the United States with *overall* mortality of other countries. (To be clear, the region-specific mortality of the United States is the only treated unit in each analysis and are therefore analyzed separately.) All region-specific estimates are statistically indistinguishable from each other.

Source: Author calculations using the U.S. Multiple Cause of Death Data (Vital Statistics) and Human Mortality Database (2026).

Figure A18: Synthetic Control Weights by Race



(a) Pooled Era (1967-2019)

(b) Recent Era (2000-19)

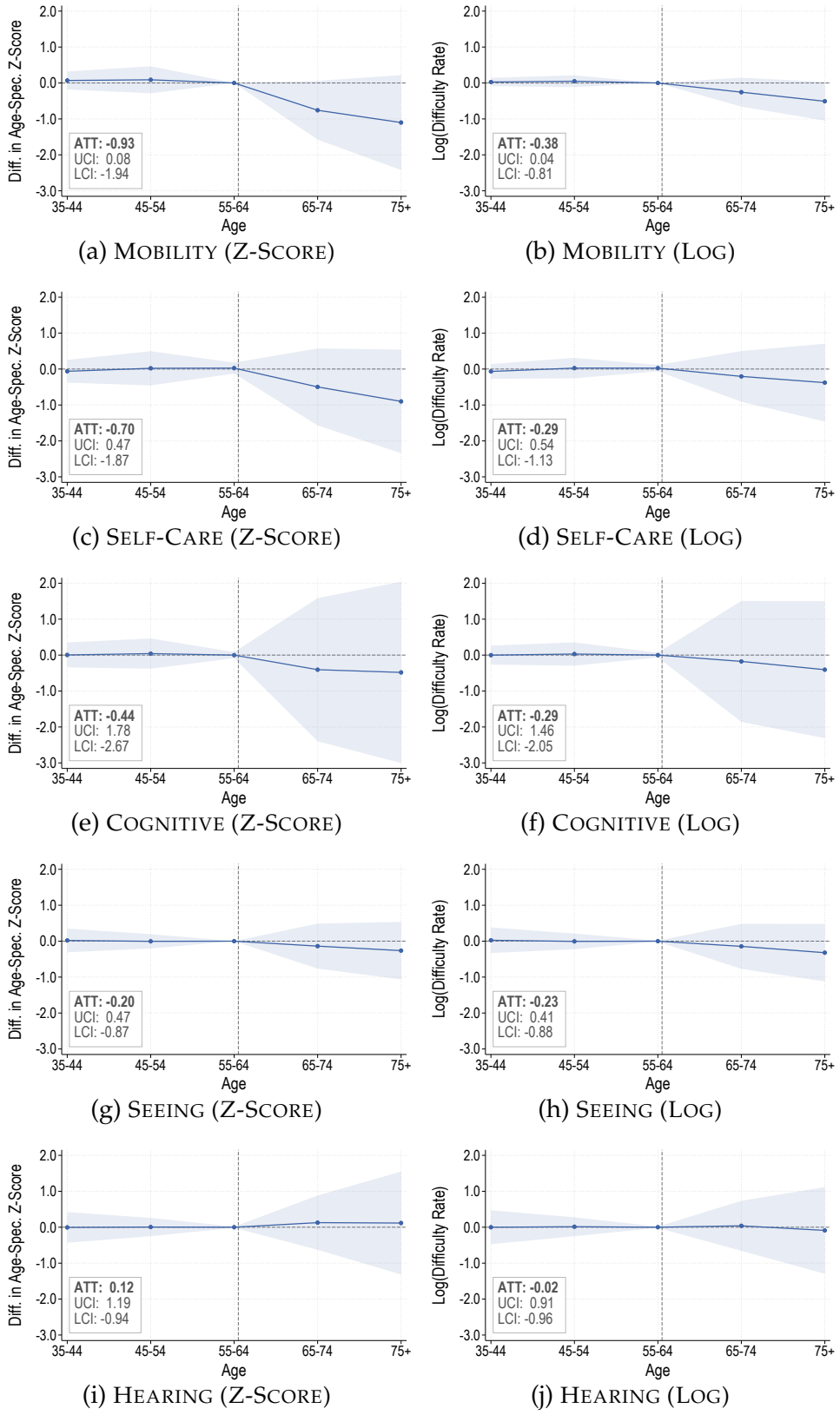
(c) Middle Era (1980-99)

(d) Early Era (1967-79)

Notes: The purpose of this figure is to display weights by race. See Appendix Figure A16 for more detail.

Source: Author calculations using the U.S. Multiple Cause of Death Data (Vital Statistics) and Human Mortality Database (2026).

Figure A19: MEDICARE'S IMPACT ON INDIVIDUAL MORBIDITY MEASURES



(a) MOBILITY (Z-SCORE) (b) MOBILITY (LOG)

(c) SELF-CARE (Z-SCORE) (d) SELF-CARE (LOG)

(e) COGNITIVE (Z-SCORE) (f) COGNITIVE (LOG)

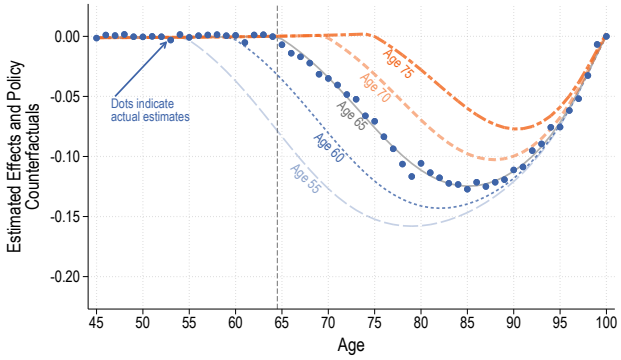
(g) SEEING (Z-SCORE) (h) SEEING (LOG)

(i) HEARING (Z-SCORE) (j) HEARING (LOG)

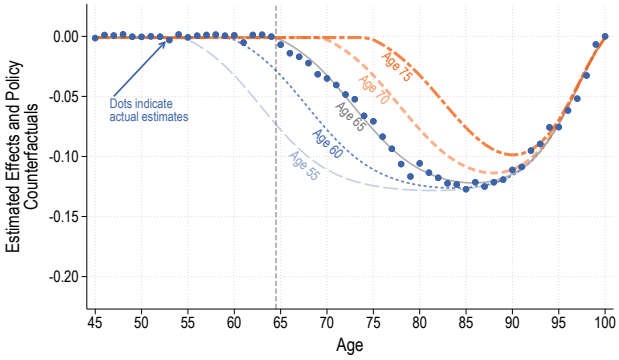
Notes: The purpose of this figure is to display the components of our z-scored summary measures in Figure 8 (left column) and their logged counterparts (right column). See Appendix Table A4 for further information to contextualize results.

Source: Author calculations using the American Community Survey (Ruggles et al., 2025*b*), European Union Statistics on Income and Living Conditions (Eurostat, 2026*b*), European Health Interview Survey (Eurostat, 2026*a*), and harmonized data from IPUMS International (Ruggles et al., 2025*a*).

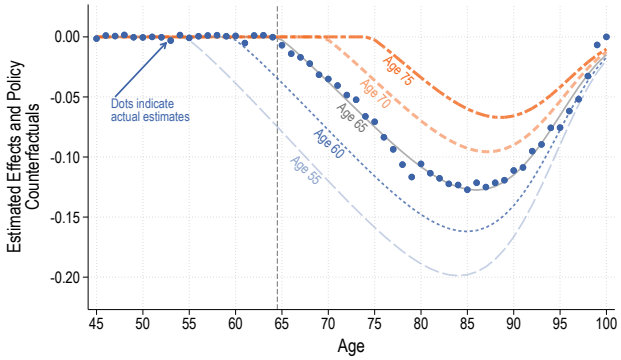
Figure A20: Estimated Counterfactual Treatment Effects of Medicare Eligibility at Different Ages



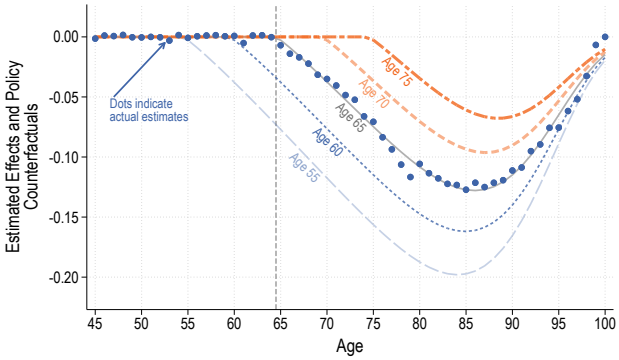
(a) Exposure: Logistic
Age-Fade: Weibull
Cumulative Survival: Yes



(b) Exposure: Logistic
Age-Fade: Weibull
Cumulative Survival: No



(c) Exposure: Power-Function
Age-Fade: Weibull
Cumulative Survival: Yes

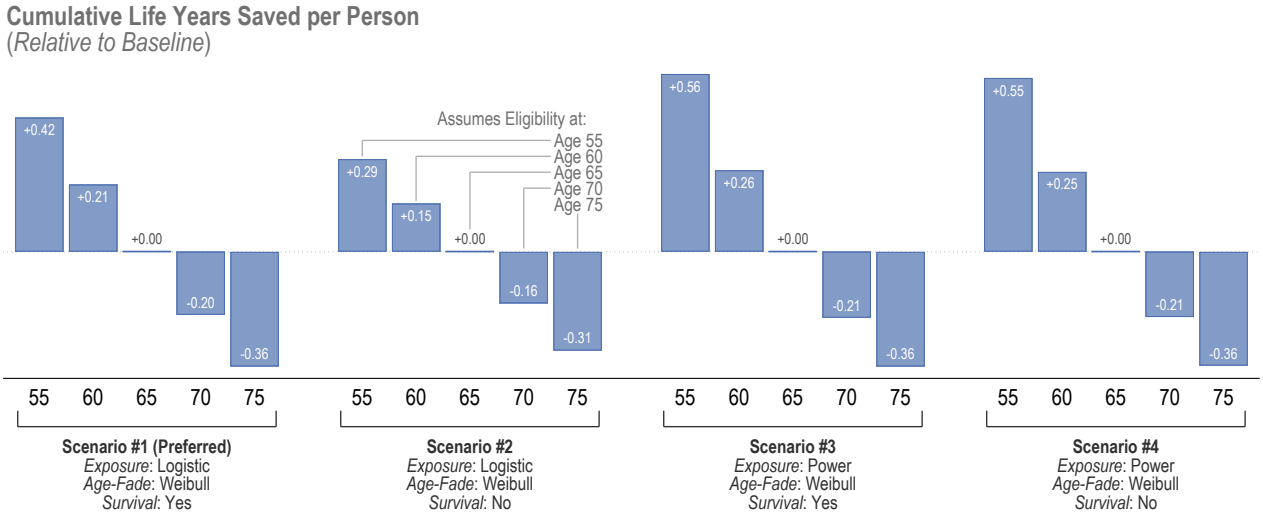


(d) Exposure: Power Function
Age-Fade: Weibull
Cumulative Survival: No

Notes: This figure demonstrates how the parameterization of recent-era treatment effects fits under different assumptions, and the resultant patterns for different counterfactual scenarios. Within the figure, dots indicate actual estimates (derived from applying the synthetic-control-derived weights, ω), while lines indicate the model fits, assuming Medicare eligibility at a given age. See discussion in Section 9 for more details.

Source: Author calculations using the Human Mortality Database (2026).

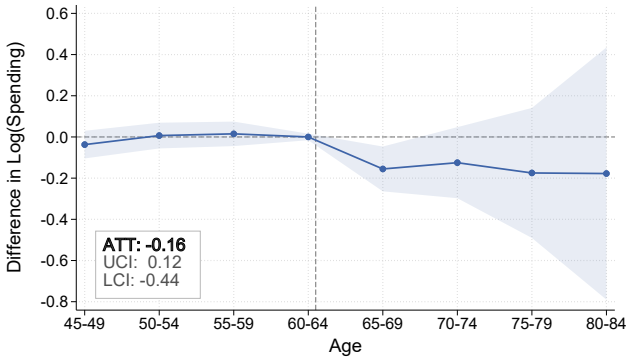
Figure A21: Extrapolating Effects Under Different Policy Counterfactuals



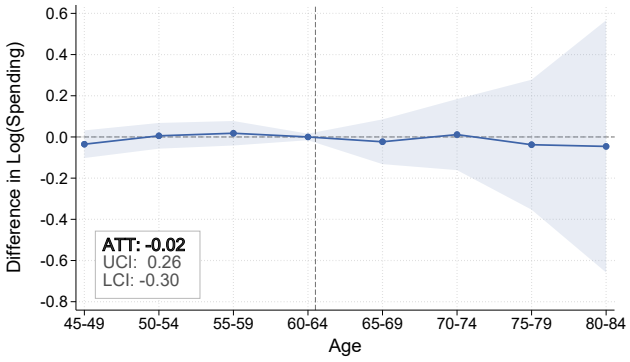
Notes: The purpose of this figure is to display the results of our counterfactual analysis. Each group of four bars represents a different scenario, which differ in their assumptions regarding the function form of Medicare exposure, the age-related fade-out of effects, and whether the effect parameterization takes into account cumulative cohort survival. These specific assumptions are detailed underneath the bar graph.

Source: Author calculations using the Human Mortality Database (2026).

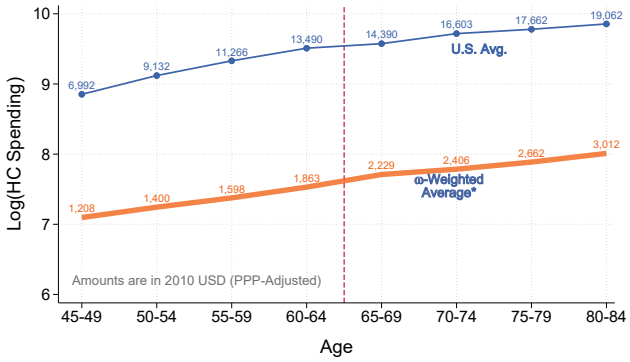
Figure A22: U.S. Healthcare Spending Does Not Rise After Age 65, Relative to Counterfactual



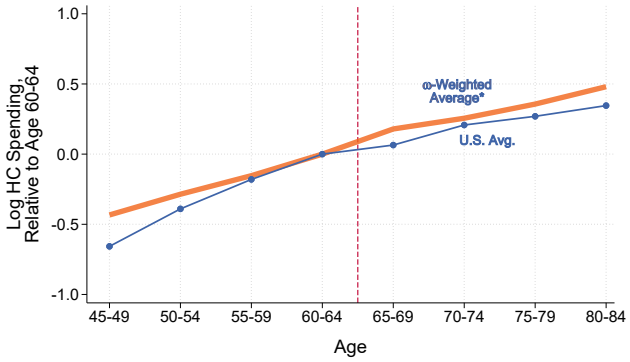
(a) Healthcare Consumption (U.S. Allocated within Overall NHE)



(b) Healthcare Consumption (U.S. Allocated within NHE Age Groups)



(c) U.S. Healthcare Consumption vs. Mortality Synthetic Control Group



(d) U.S. Healthcare Consumption vs. Mortality Synthetic Control Group (De-meaned)

Notes: This figure demonstrates the path of U.S. healthcare spending/consumption, relative to other countries. The top rows demonstrate our point estimates from a synthetic difference-in-differences estimation of Medicare's effect on log healthcare spending. (Synthetic difference-in-differences, rather than synthetic control, was used as the U.S. spends more money on healthcare than any other country.) Panel A calculates consumption-by-age by allocating National Health Expenditures ("NHE") to age groups using relative spending in the Medical Expenditure Panel Survey ("MEPS"). Panel B performs an analogous exercise, except that it allocates spending within the NHE's existing age groups (which cover ages 45-64 and 65-84, respectively).

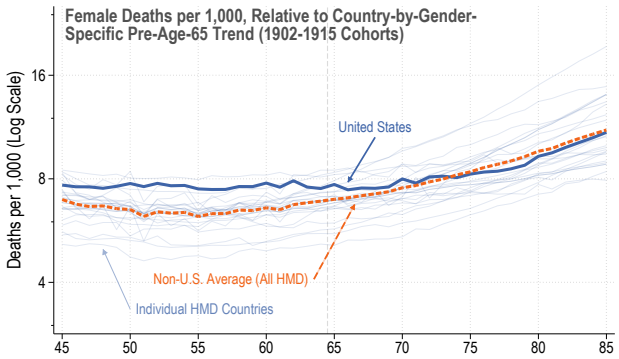
The lower row compares health spending in the United States and its synthetic counterpart, which is formed by applying the weights from our Recent-Era mortality analysis (ω). As shown in Panels C and D, the United States generally differs (substantially) in levels, and its trend flattens following the age-65 cutoff, suggesting that U.S. old-age healthcare spending slows relative to its synthetic counterpart. As not all donor countries were included in the underlying datasets, we upweighted according to the following formula:

$$\omega_i^* = \frac{\hat{\omega}_i}{\sum_{c \in \mathcal{C}} \hat{\omega}_c}$$

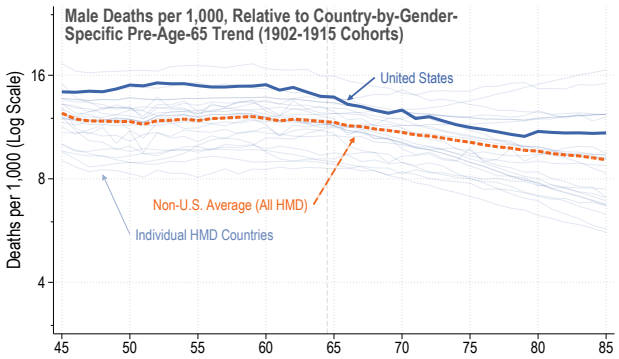
where $\mathcal{C}$ is the set of donor countries and $\sum_{c \in \mathcal{C}} \hat{\omega}_c = 75.7\%$. See Figure 3 for further discussion on this methodology.

Source: Author calculations using CMS National Health Expenditures Data, the Medical Expenditure Panel Survey (Blewett et al., 2025*a*), UN National Transfer Accounts data, and estimates from Eurostat (2016).

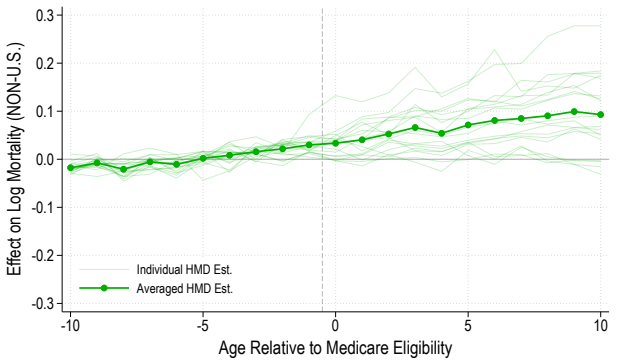
Figure A23: Other Countries Experienced Deviations from Pre-Medicare Trends in Log Mortality



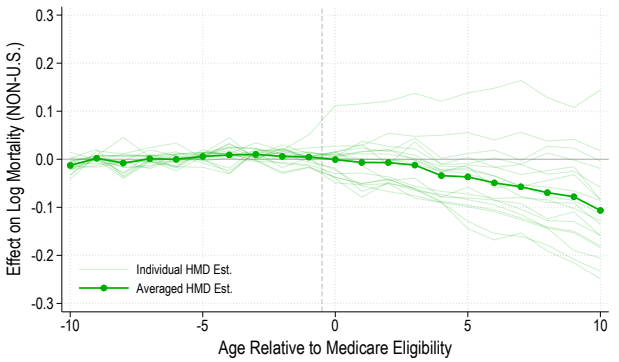
(a) Deviations from pre-65 Gompertz Trend: Female Mortality



(b) Deviations from pre-65 Gompertz Trend: Male Mortality



(c) Interrupted Time Series Estimates: Female

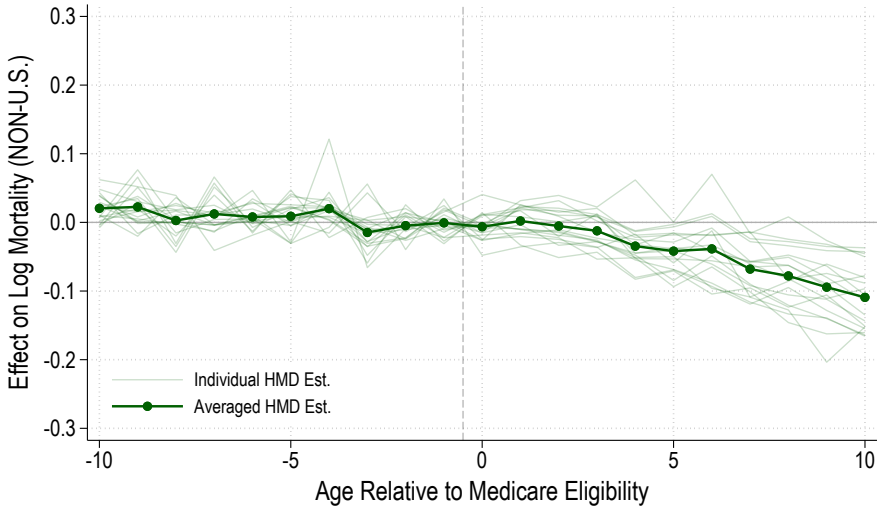


(d) Interrupted Time Series Estimates: Male

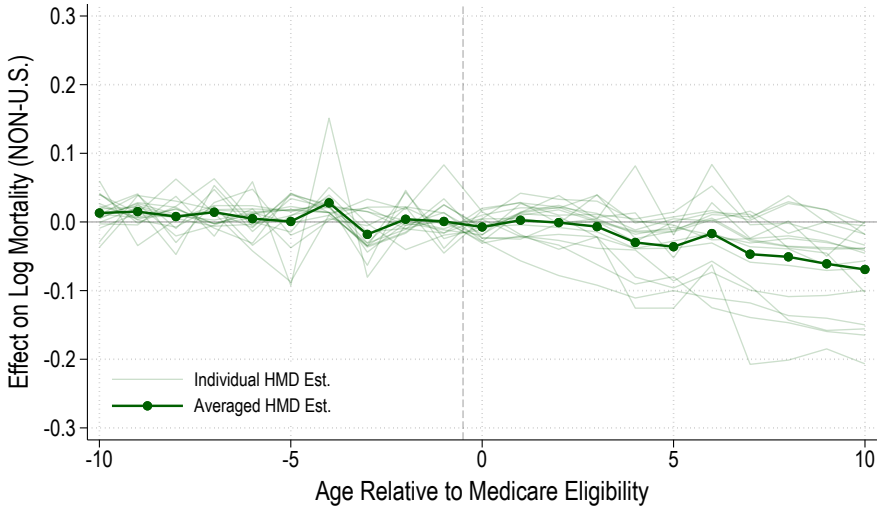
Notes: This figure demonstrates gender-specific trends in cohort mortality for the first cohorts to fully experience Medicare (Panels A and B). It also displays gender-specific estimates from applying the interrupted time-series ("ITS") method from Goodman-Bacon et al. (2025) to countries *other* than the United States (Panels C and D). Countries were included in the ITS analysis if they were in the HMD and had complete coverage for the 1885-1915 cohorts, which match those used in the primary sample of Goodman-Bacon et al. (2025). See Appendix Section B for further discussion.

Source: Author calculations using Human Mortality Database (2026) data.

Figure A24: OTHER COUNTRIES SHOW "TREATMENT EFFECTS" WHEN USING A COHORT DIFFERENCE-IN-DIFFERENCES FRAMEWORK



(a) COHORT DID: MALE

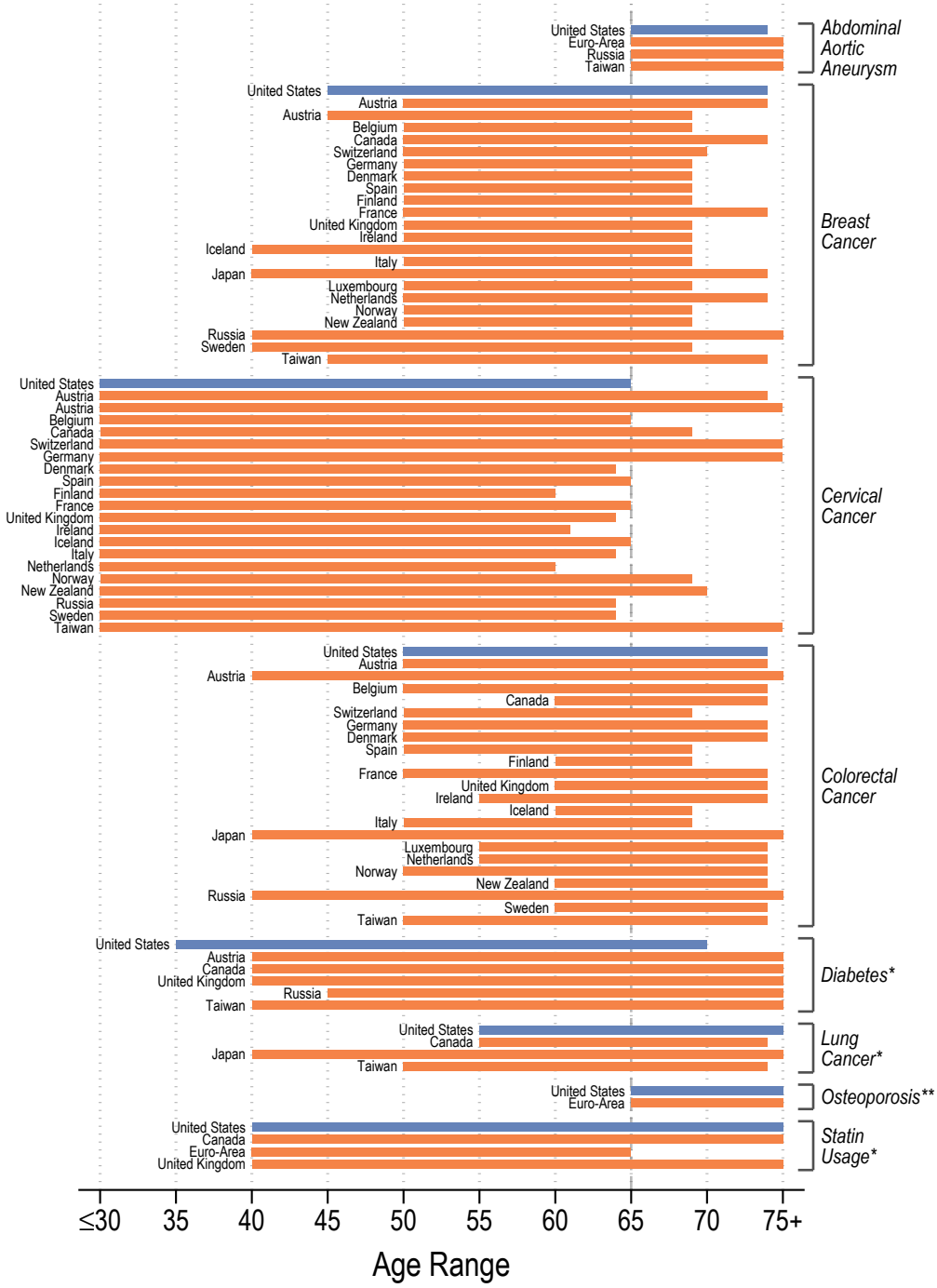


(b) COHORT DID: FEMALE

Notes: This figure displays gender-specific estimates from applying the cohort difference-in-differences ("CDID") method from Goodman-Bacon et al. (2025) to countries *other* than the United States. Countries were included in the CDID analysis if they were in the HMD and had complete coverage for the 1885-1915 cohorts, which match those used in the primary sample of Goodman-Bacon et al. (2025). See Appendix Section B for further discussion.

Source: Author calculations using Human Mortality Database (2026) data.

Figure A25: Screening and Preventative Treatment Guidelines in the United States Do Not Appear to Be Affected by Medicare and Are in Line with Other Countries

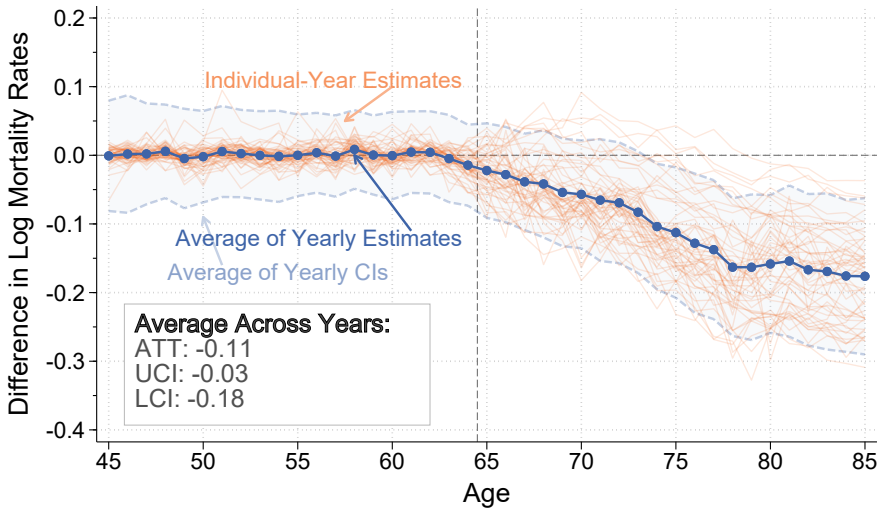


Notes: The purpose of this figure is to demonstrate how the United States compares to other countries in terms of screening guidelines. To create this figure, we gathered screening procedures that are highly recommended by the U.S. Preventive Services Task Force (2025) and searched for the presence of screening recommendations in other countries. We performed a targeted search for Russian and Taiwanese recommendations, as these are the two largest donors for the recent period and are also two countries that we believed, *ex-ante*, might not adhere to Anglo/European norms in terms of screening and treatment.

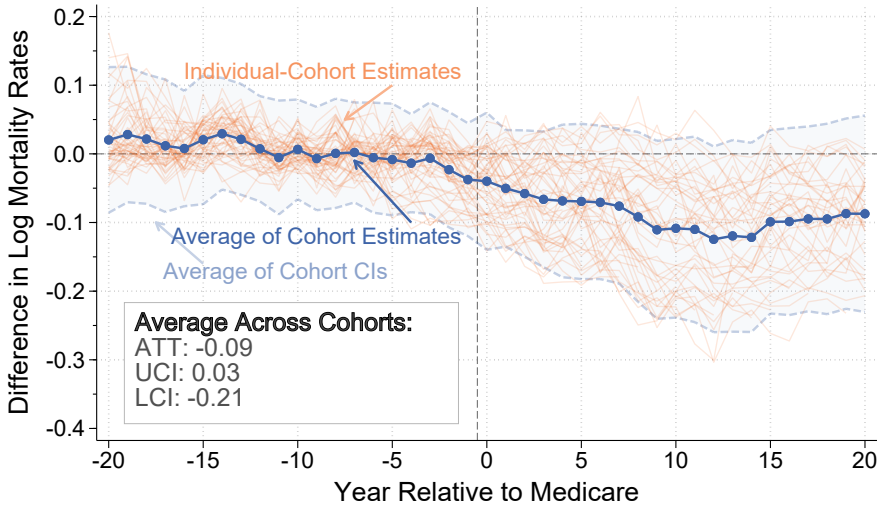
*Indicates screening guidelines are for high-risk groups.
**Indicates screening guidelines in Euro-area are more targeted than in the United States.

Source: Author calculations using the sources detailed in Appendix Section C.3.

Figure A26: Estimating Effects by Year and Cohort and then Pooling Those Estimates Gives Similar Results



(a) Estimates by Year



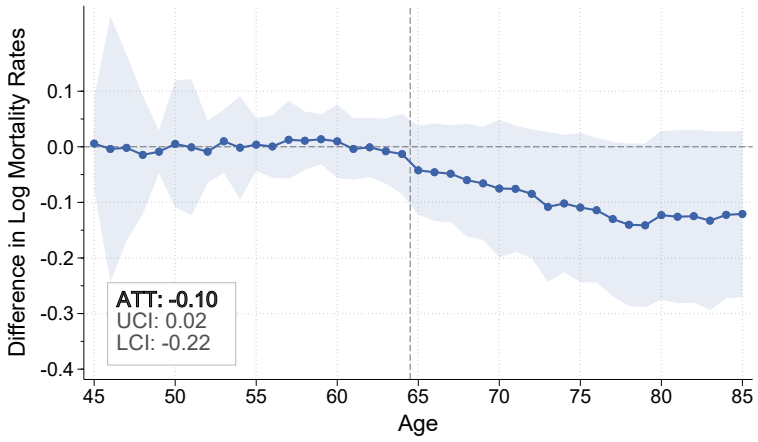
(b) Estimates by Cohort

Notes: This figure demonstrates the impact of separately estimating the effects of Medicare in individual years, rather than pooling into eras (Panel A), and also applies a similar concept to cohorts (Panel B). Within the figure, estimates for each year/cohort are presented via the thin orange lines, whereas the average of all those years/cohorts is presented via the darker connected dots. Confidence intervals are averages of yearly confidence intervals and are therefore conservative, relative to those calculated for pooled eras. (Also note that, with very few exceptions, individual years have large confidence intervals, making recovery of year-specific effects infeasible.)

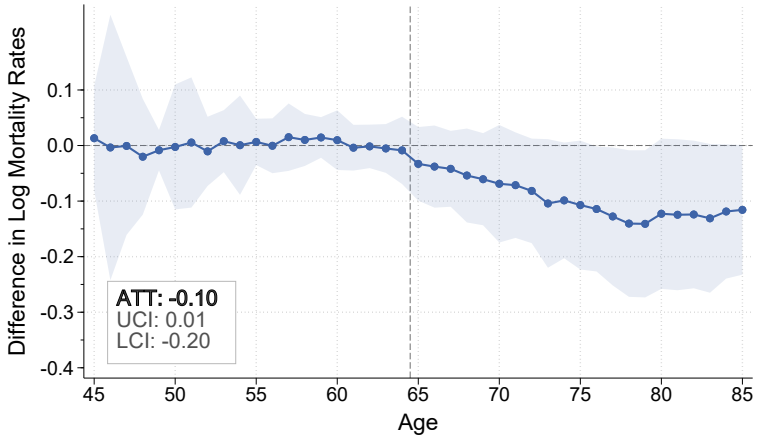
Panel B presents results from the 1885-1956 cohorts, where the x-axis is in *relative time* to account for the fact that early cohorts entered Medicare after age 65. For those born in 1902 and later, relative time equal to their age, minus 65.

Source: Author calculations using Human Mortality Database (2026).

Figure A27: CONTROLLING FOR CUMULATIVE COHORT SURVIVAL DOES NOT MEANINGFULLY AFFECT ESTIMATES



(a) SURVIVAL TO AGE 45 (THROUGH AGE 44)



(b) SURVIVAL TO AGE 65 (THROUGH AGE 64)

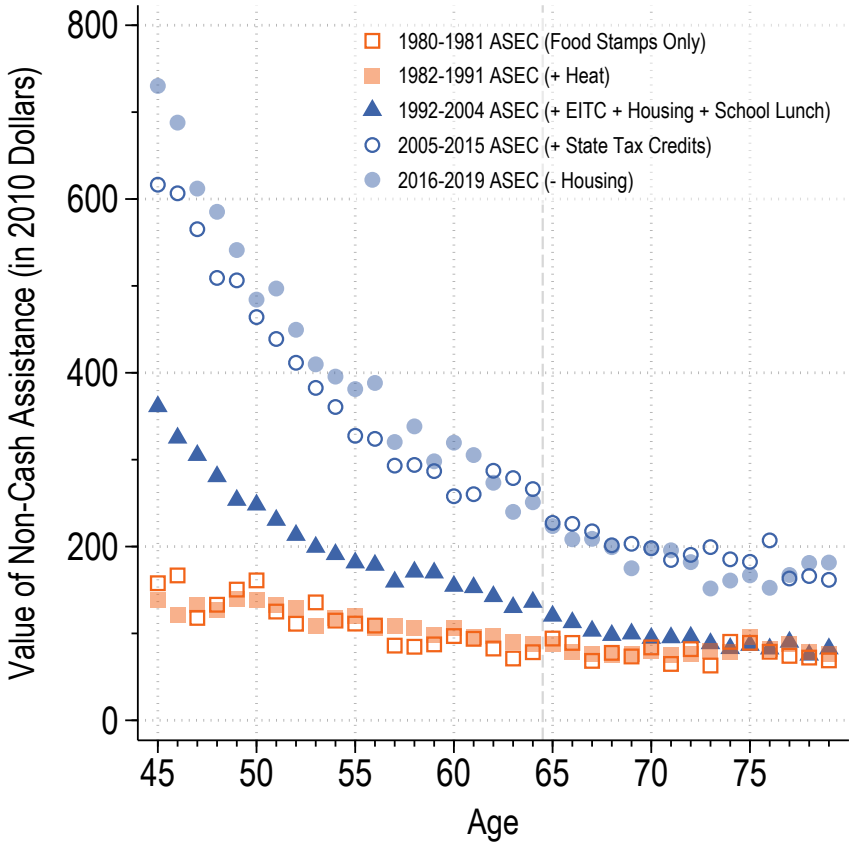
Notes: The purpose of this figure is to demonstrate how estimates for the recent-era change when controlling for cumulative cohort survival. The estimates focus exclusively on the recent era, as data for the 1915 cohort–the oldest cohort contributing to estimates in that era–are available via Social Security Life Tables. However, to perform a similar analysis for the middle or early eras would require complete cohort mortality back to the 1895 and 1882 cohorts, respectively, which is not available.

Controlling for mortality was done in a method similar to that outlined in the notes to Appendix Figure A9, except performed on the country-*year*, within a regression framework that allowed the impacts of cumulative cohort survival to vary by age. Cumulative survival *to* age 45 and 65 were chosen, as that encompasses the period prior to our synthetic control matching period and the treatment period, respectively. (Cumulative survival as of each age was not used, as it would effectively be controlling for an outcome when applied to ages 65 through 85.)

Once residualization for each country-year was performed, the outcomes were aggregated to the country-era level for our synthetic control analysis. Inference was performed using the method outlined in Clarke et al. (2024), modified to account for regression adjustment at the country-year level.

Source: Author calculations using the Human Mortality Database (2026) and Social Security Administration life cohort tables.

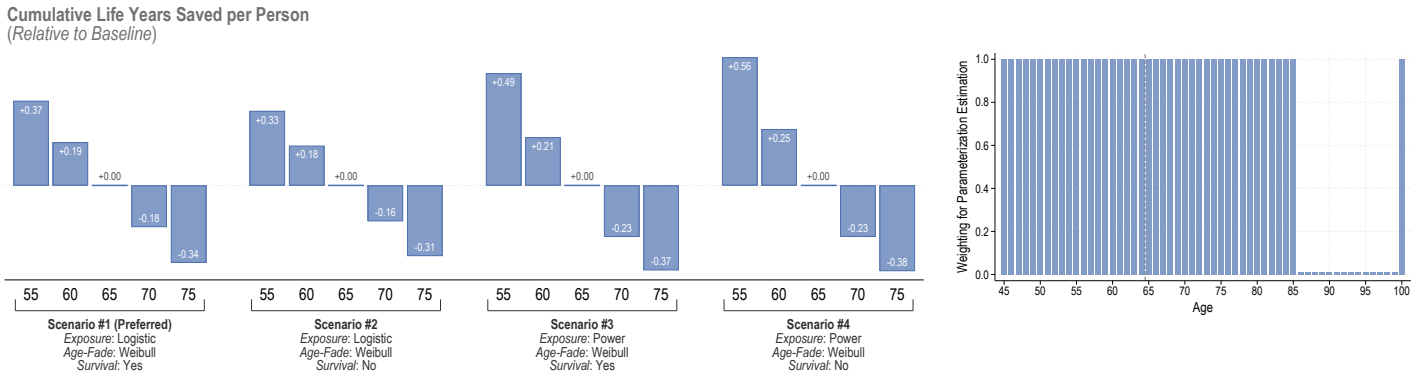
Figure A28: NON-CASH SUPPORT DOES NOT CHANGE SHARPLY AS U.S. ADULTS ENTER MEDICARE-ELIGIBLE AGES



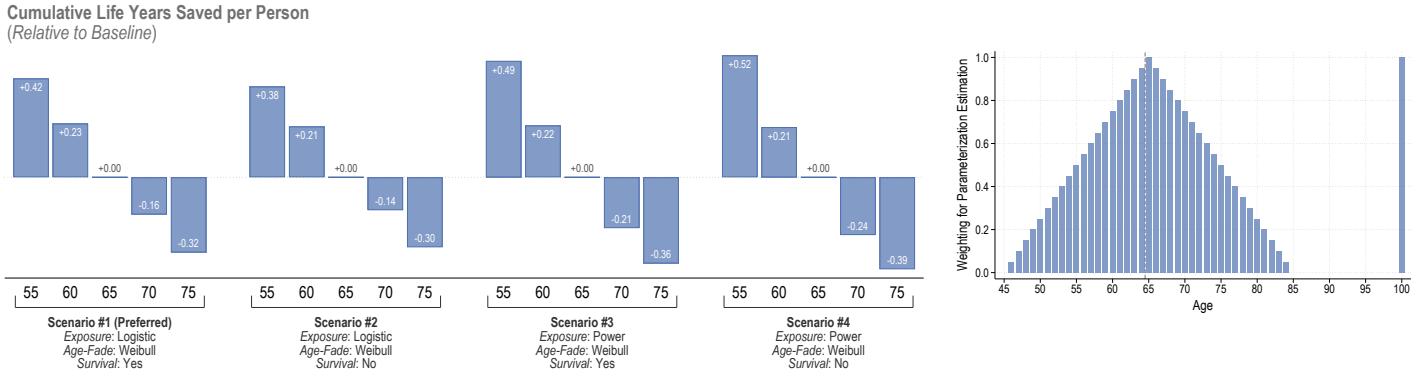
Notes: This figure demonstrates the way in which non-cash assistance within the United States evolves as individuals age. Within the figure, series are broken up by different time-periods, based on the variables tracked by the CPS ASEC during those years. All figures have been inflation-adjusted to 2011 dollars

Source: Author calculations using the Current Population Survey Annual Social and Economic Supplements (Flood et al., 2025).

Figure A29: Estimated Counterfactual Predictions with Different Weighting Assumptions



(a) Counterfactual Policy Estimates (Zero Weighting Post-85)



(b) Counterfactual Policy Estimates (Triangular Weighting)

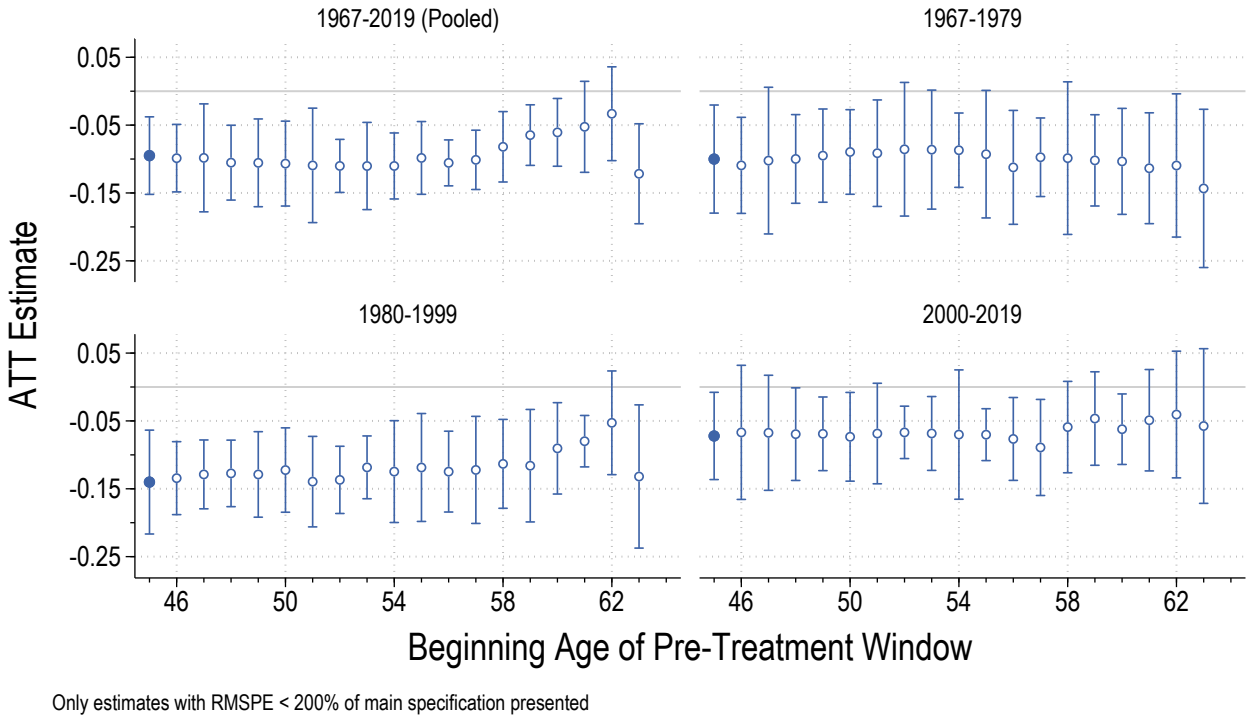
Notes: The purpose of this figure is to illustrate how our counterfactual policy estimates are affected by different weighting assumptions. In Panel A, we down-weight all ages after 85 to zero, in order to account for the fact that these estimates were outside our primary estimation window. A key exception to this is age 100, where the weight returns to 1. This is because mortality is assume to be complete at that age, and therefore treatment effects between treatment and control are mechanically identical (and thus estimated with extremely high confidence).

Panel B performs a similar exercise, except that weights shrink relative to age 65, so that the model prioritizes matches of treatment effects near the eligibility date, with less emphasis further away, when estimates may become less reliable. The exception to this is, once again, age 100, for the reasons noted above.

See Figure 9 for more detail and baseline estimates, which closely mirror those detailed in this figure.

Source: Author calculations using the Human Mortality Database (2026).

Figure A30: Our Estimates Are Not Sensitive to Choice of Pre-Treatment Matching Window



Notes: This figure demonstrates the way our estimates are affected when we change the pre-treatment window used to form our synthetic control group. As demonstrated from the figure, all estimates meaningfully overlap with those from our main specification.

Source: Author calculations using the Human Mortality Database (2026).

Table A1: Data Sources by Country and Analysis

		Understanding Synthetic U.S.		Robustness				Supporting Analyses and Heterogeneity			
Country	**Main**	**Unmet Need[R]**	**Healthcare Measures[R]**	**Placebo Era (1955-65)**	**Labor Force / Income[MR]**	**Smoking / Obesity[MR]**	**Survival[R]**	**Cause of Death[MR, 6]**	**Race/ U.S. Region**	**Morbidity[R, 7]**	**Spending[R, 7]**
United States	HMD	NHIS/MEPS	GBD, ISSP	WHOMD	LIS	GBD	SSA	WHOMD, HCD	MCOD	ACS	NHE/MEPS
Australia	HMD		GBD, ISSP	WHOMD		GBD		WHOMD	HMD		NTA
Austria	HMD	SILC	GBD	WHOMD	LIS + IPUMSI	GBD		WHOMD	HMD	EHIS, SILC-HC	NTA
Belarus	HMD		GBD			GBD			HMD		
Belgium	HMD	SILC	GBD	WHOMD	LIS	GBD		WHOMD	HMD	SILC-HC	
Bulgaria	HMD	SILC	GBD		LIS[R]	GBD		WHOMD	HMD	EHIS, SILC-HC	
Canada	HMD		GBD	WHOMD	LIS + IPUMSI	GBD		WHOMD, HCD	HMD		NTA
Chile	HMD[R]		GBD	WHOMD	LIS[R]	GBD		WHOMD	HMD[R]		NTA
Croatia	HMD[R]	SILC	GBD, ISSP			GBD			HMD[R]	EHIS, SILC-HC	
Czech Republic	HMD	SILC	GBD, ISSP		LIS	GBD		HCD	HMD	EHIS, SILC-HC	Eurostat
Denmark	HMD	SILC	GBD, ISSP	WHOMD	LIS	GBD	HMD[4]	WHOMD	HMD	EHIS, SILC-HC	
Estonia	HMD	SILC	GBD		LIS[R]	GBD		HCD	HMD	EHIS, SILC-HC	
Finland	HMD	SILC	GBD, ISSP	WHOMD	LIS[R]	GBD	HMD[4]	WHOMD	HMD	EHIS, SILC-HC	NTA, Eurostat
France	HMD	SILC	GBD	WHOMD	LIS	GBD	HMD[4]	WHOMD, HCD	HMD	EHIS, SILC-HC	NTA
Germany[1]	HMD	SILC	GBD	WHOMD	LIS[R]	GBD			HMD	EHIS	NTA, Eurostat
Greece	HMD[MR]	SILC	GBD	WHOMD	LIS + IPUMSI	GBD		WHOMD	HMD[MR]	EHIS, SILC-HC	
Hong Kong	HMD[MR]			WHOMD				WHOMD	HMD[MR]		
Hungary	HMD	SILC	GBD	WHOMD	LIS[R]	GBD		WHOMD	HMD	EHIS, SILC-HC	NTA, Eurostat
Iceland	HMD	SILC	GBD	WHOMD	LIS[R]	GBD	HMD[4]	WHOMD	HMD	EHIS, SILC-HC	
Ireland	HMD	SILC	GBD	WHOMD	LIS	GBD		WHOMD	HMD	EHIS, SILC-HC	
Israel	HMD[MR]		GBD, ISSP		LIS[M]	GBD		WHOMD	HMD[MR]		NTA[5]
Italy	HMD	SILC	GBD, ISSP	WHOMD	LIS	GBD	HMD[4]	WHOMD	HMD	EHIS, SILC-HC	NTA
Japan	HMD		GBD, ISSP	WHOMD	LIS[R]	GBD		WHOMD, HCD	HMD		NTA
Latvia	HMD	SILC	GBD			GBD		WHOMD, HCD	HMD	EHIS, SILC-HC	Eurostat
Lithuania	HMD	SILC	GBD		LIS[R]	GBD		HCD	HMD	EHIS, SILC-HC	Eurostat
Luxembourg	HMD	SILC	GBD		LIS	GBD		WHOMD	HMD	EHIS, SILC-HC	
Netherlands	HMD	SILC	GBD, ISSP	WHOMD	LIS	GBD	HMD[4]	WHOMD	HMD	SILC-HC	Eurostat
New Zealand[3]	HMD		GBD	WHOMD		GBD		WHOMD	HMD		
Norway	HMD	SILC	GBD, ISSP	WHOMD	LIS	GBD	HMD[4]	WHOMD, HCD	HMD	EHIS, SILC-HC	
Poland	HMD	SILC	GBD, ISSP		LIS	GBD		WHOMD, HCD	HMD	EHIS, SILC-HC	NTA[5]
Portugal	HMD	SILC	GBD	WHOMD	IPUMSI[M]	GBD			HMD	EHIS, SILC-HC	
Republic of Korea	HMD[R]	Chung (2022)	GBD		LIS[R]	GBD			HMD[R]		NTA
Russia	HMD	Rosstat	GBD, ISSP		LIS[R]	GBD		WHOMD, HCD	HMD		NTA[5]
Slovakia	HMD	SILC	GBD, ISSP		LIS	GBD			HMD	EHIS, SILC-HC	
Slovenia	HMD[MR]	SILC	GBD, ISSP			GBD			HMD[MR]	EHIS, SILC-HC	NTA, Eurostat
Spain	HMD	SILC	GBD	WHOMD	LIS	GBD	HMD[4]	WHOMD, HCD	HMD	EHIS, SILC-HC	NTA
Sweden	HMD	SILC	GBD	WHOMD	LIS	GBD	HMD[4]	WHOMD, HCD	HMD	EHIS, SILC-HC	NTA, Eurostat
Switzerland	HMD	SILC	GBD, ISSP	WHOMD	LIS + IPUMSI	GBD	HMD[4]	WHOMD	HMD	SILC-HC	
Taiwan	HMD[MR]	TSCS	GBD, ISSP		LIS	GBD			HMD[MR]		NTA
Ukraine	HMD		GBD			GBD		HCD	HMD		
United Kingdom[2]	HMD	SILC	GBD	WHOMD	LIS	GBD	HMD[4]	WHOMD, HCD	HMD		

Notes:

[1] East, West, and Combined in HMD
[2] England/Wales, North Ireland, Scotland, and U.K. in HMD
[3] Māori, Non-Māori, and Total Available in HMD
[4] Must have mortality back to 1915 to be included in analysis.
[5] Omitted from SDID analysis due to data limitations
[6] Only countries overlapping with HMD sample listed. HCD also includes Moldova and WHOMD/HCD include Romania
[7] Only countries overlapping with HMD sample listed. See Appendix Section A for more information.

[R] Recent-era only (2000-19)
[M] Middle-era only (1980-99)
[MR] Middle & recent only (1980-2019)

Abbreviations:

ACS = American Community Survey (IPUMS USA)
EHIS = European Health Interview Survey
GBD = Global Burden of Disease
HMD = Human Mortality Database
HCD = HMD Cause-of-Death Files
IPUMSI = IPUMS International
ISSP = International Social Survey Programme
LIS = Luxembourg Income Study
MCOD = Multiple Cause of Death Data
MEPS = Medical Expenditure Panel Survey
NHE = CMS National Health Expenditures
NHIS = National Health Interview Study
NTA = United Nations National Transfer Accounts
SILC = E.U. Survey of Income and Living Conditions
SILC-HC = E.U. SILC Healthcare Supplement
TSCS = Taiwanese Social Change Survey
WHOMD = WHO Mortality Database

Table A2: SUMMARY STATISTICS BY AGE (UNITED STATES)

Age	Mortality per 100				Log(Mortality per 1,000)			
	Pooled (1967-2019)	Recent (2000-19)	Middle (1980-99)	Early (1967-79)	Pooled (1967-2019)	Recent (2000-19)	Middle (1980-99)	Early (1967-79)
45	0.3	0.3	0.3	0.4	-5.7	-5.9	-5.7	-5.4
46	0.4	0.3	0.4	0.5	-5.6	-5.8	-5.6	-5.3
47	0.4	0.3	0.4	0.5	-5.5	-5.7	-5.6	-5.2
48	0.4	0.4	0.4	0.6	-5.4	-5.6	-5.4	-5.1
49	0.5	0.4	0.4	0.6	-5.4	-5.5	-5.4	-5.1
50	0.5	0.4	0.5	0.7	-5.3	-5.5	-5.3	-5.0
51	0.6	0.5	0.6	0.7	-5.2	-5.4	-5.2	-4.9
52	0.6	0.5	0.6	0.8	-5.1	-5.3	-5.1	-4.8
53	0.7	0.5	0.7	0.9	-5.0	-5.2	-5.0	-4.7
54	0.7	0.6	0.7	1.0	-4.9	-5.1	-5.0	-4.6
55	0.8	0.6	0.8	1.1	-4.8	-5.1	-4.8	-4.6
56	0.9	0.7	0.9	1.1	-4.8	-5.0	-4.8	-4.5
57	0.9	0.7	0.9	1.3	-4.7	-4.9	-4.7	-4.4
58	1.0	0.8	1.1	1.4	-4.6	-4.8	-4.6	-4.3
59	1.1	0.9	1.1	1.5	-4.5	-4.8	-4.5	-4.2
60	1.2	0.9	1.2	1.6	-4.4	-4.7	-4.4	-4.1
61	1.3	1.0	1.3	1.7	-4.3	-4.6	-4.3	-4.1
62	1.4	1.1	1.5	1.9	-4.2	-4.5	-4.2	-4.0
63	1.5	1.2	1.6	2.0	-4.2	-4.4	-4.1	-3.9
64	1.7	1.3	1.7	2.2	-4.1	-4.4	-4.1	-3.8
65	1.8	1.4	1.9	2.4	-4.0	-4.3	-4.0	-3.7
66	1.9	1.5	2.0	2.5	-3.9	-4.2	-3.9	-3.7
67	2.1	1.6	2.2	2.8	-3.9	-4.1	-3.8	-3.6
68	2.3	1.7	2.4	3.0	-3.8	-4.1	-3.7	-3.5
69	2.5	1.9	2.6	3.2	-3.7	-4.0	-3.7	-3.4
70	2.7	2.1	2.8	3.6	-3.6	-3.9	-3.6	-3.3
71	2.9	2.2	3.0	3.7	-3.5	-3.8	-3.5	-3.3
72	3.2	2.4	3.3	4.2	-3.4	-3.7	-3.4	-3.2
73	3.5	2.7	3.6	4.5	-3.4	-3.6	-3.3	-3.1
74	3.8	2.9	3.9	4.9	-3.3	-3.5	-3.2	-3.0
75	4.1	3.2	4.2	5.4	-3.2	-3.4	-3.2	-2.9
76	4.5	3.5	4.6	5.7	-3.1	-3.3	-3.1	-2.9
77	4.9	3.9	5.0	6.2	-3.0	-3.3	-3.0	-2.8
78	5.3	4.2	5.4	6.7	-2.9	-3.2	-2.9	-2.7
79	5.8	4.7	5.9	7.4	-2.8	-3.1	-2.8	-2.6
80	6.6	5.3	6.7	8.2	-2.7	-2.9	-2.7	-2.5
81	7.1	5.9	7.3	8.7	-2.6	-2.8	-2.6	-2.4
82	7.9	6.6	8.1	9.7	-2.5	-2.7	-2.5	-2.3
83	8.7	7.3	8.9	10.7	-2.4	-2.6	-2.4	-2.2
84	9.7	8.2	9.7	11.8	-2.3	-2.5	-2.3	-2.1
85	10.6	9.2	10.6	12.8	-2.2	-2.4	-2.2	-2.1

Notes: This table details U.S. mortality rates across ages and eras, in order to provide context for our main results.

Source: Author calculations using the Human Mortality Database (2026).

Table A3: SUMMARY STATISTICS BY AGE AND CAUSE OF DEATH (UNITED STATES)

			Cause of Death			
Age	**Amenable**	**Non-Amenable**	**Cardiovascular**	**Cancer**	**Respiratory**	**External**
			Panel A: Deaths per 10,000			
45 - 49	9	27	10	10	1	7
50 - 54	15	41	17	18	3	6
55 - 59	23	62	28	30	5	6
60 - 64	35	94	45	46	10	6
65 - 69	53	138	70	67	17	6
70 - 74	85	207	113	91	30	8
75 - 79	138	309	188	117	50	11
80 - 84	245	491	339	150	82	17
			Panel B: Deaths as a Percentage of Total (Age-Specific)			
45 - 49	25%	76%	27%	27%	4%	18%
50 - 54	26%	74%	31%	32%	5%	12%
55 - 59	27%	74%	33%	35%	6%	7%
60 - 64	27%	73%	35%	36%	7%	5%
65 - 69	28%	73%	36%	35%	9%	3%
70 - 74	29%	71%	39%	31%	10%	3%
75 - 79	31%	69%	42%	26%	11%	2%
80 - 84	33%	67%	46%	20%	11%	2%

Notes: This table details U.S. mortality rates across age-groups and causes, in order to provide context for our cause-of-death results.

Source: Author calculations using the WHO Mortality Database (World Health Organization, 2026) and list of treatment-amenable conditions from Nolte and McKee (2003, 2011).

Table A4: MORBIDITY BY AGE AND TYPE (UNITED STATES)

Age	Difficulty per 100 Persons				
	Mobility	**Self-Care**	**Cognitive**	**Seeing**	**Hearing**
Panel A: Rates of Difficulty					
25 - 34	1.7	0.9	3.7	1.1	1.0
35 - 44	3.4	1.4	3.9	1.4	1.4
45 - 54	7.0	2.5	5.1	2.5	2.5
55 - 64	12.1	3.8	5.9	3.3	4.6
65 - 74	16.4	5.2	5.9	4.4	9.2
75+	35.6	17.2	16.7	10.6	23.0
Panel B: Logged Rates of Difficulty					
25 - 34	0.5	-0.1	1.3	0.1	0.0
35 - 44	1.2	0.3	1.4	0.3	0.3
45 - 54	1.9	0.9	1.6	0.9	0.9
55 - 64	2.5	1.3	1.8	1.2	1.5
65 - 74	2.8	1.6	1.8	1.5	2.2
75+	3.6	2.8	2.8	2.4	3.1

Notes: This table details U.S. difficulty rates across age-groups and type, in order to provide context for Medicare's impact on difficulties and limitations in daily living. The descriptions of each limitation are described across surveys as follows:

- Mobility: Difficulty Walking, Climbing Stairs
- Self-Care: Difficulty Dressing, Bathing
- Cognitive: Difficulty Remembering, Concentrating, and/or Making Decisions
- Seeing: Difficulty Seeing, even with Glasses
- Hearing: Difficulty Hearing, including Deafness, Even with Hearing Aids

Source: Author calculations using the American Community Survey (Ruggles et al., 2025*b*).

Table A5: REFRAMING RESULTS OF OUR ROBUSTNESS ANALYSIS TO DIRECTLY ADDRESS THREATS TO IDENTIFICATION LAID OUT BY HOLLINGSWORTH AND WING (2022)

Threats to Validity	Discussion of Threat
Spillover & Anticipation Effects	While there is evidence that individuals defer demand until after they reach Medicare, most of it pertains to non-emergent procedures, such as hip and knee replacements (e.g., Card, Dobkin and Maestas, 2008; Schwartz et al., 2019). Increases in post-Medicare utilization tend to be as a result of unmet need, as displayed in Figure 3, rather than life-saving care that would affect mortality and affect our synthetic control estimates.
Structural Breaks	If there are structural breaks *prior* to the treatment date, but during the training period, then synthetic control estimates could be biased, as the counterfactual group was created to match the pre-break values, which are no longer appropriate. However, this does not appear to be an issue in our setting. As displayed in Figure 1, Gompertz' law still largely holds for the pre-age-65 period, and there is no evidence of structural break with the U.S. trend. Moreover, as displayed in Figure 4, when we limit our training period to ages 45-54, they still largely match the remaining pre-treatment time periods, particularly for the pooled, recent-era, and middle-era samples. If structural breaks were occurring, the synthetic control training on the younger ages only would deviate meaningfully from pre-treatment values. A similar test, shown in Appendix Figure A30, leaves out *earlier* ages from the training sample, progressively limiting the ages used for the synthetic control match to those closer to the age-65 cutoff. If a structural break occurred during the pre-treatment period, we would expect these estimates to change drastically as the matching window shrinks, as the smaller-window estimates would ostensibly be more likely to be based on the post-break latent factors. However, we instead note that our estimates are consistent, further assuaging concerns regarding pre-treatment structural breaks biasing our results.
Omitted Variable Bias	If there are time-varying factors that affect the United States' post-65 mortality, while *not* affecting the synthetic control countries in the same manner (or in the same way), our estimates could clearly be biased. This bias could also occur if donor unit(s) were subject to time-varying factors that did not affect the United States, such as idiosyncratic post-65 policies for influential donor units. However, given that our results are incredibly robust to the choice of different combinations of donors–as shown in Appendix Figures A9 and A12–the likelihood of an idiosyncratic shock to the donor pool leading to meaningful bias is vanishingly low, as it would by necessity need to be a shock to virtually *all* potential donors units other than the United States. Accordingly, any omitted variable bias would stem from (a) U.S.-specific characteristics or policy that (b) strongly affects post-65 mortality but *not* pre-65 mortality. We address such possibilities in several ways, most of which are discussed in Section 5. A great deal of our focus is dedicated to the impacts of labor force and income. These two items receive special attention, as confounders would need to have a pervasive, population-level impact to create the strong impacts we detect, and retirement (with its related changes in income) is one of the only factors that could plausibly meet that criteria. As noted in Section 5, our effects are materially unchanged when accounting for these factors. (Contiued on next page...)

Table A5: Reframing Results of Our Robustness Analysis to Directly Address Threats to Identification Laid Out By Hollingsworth and Wing (2022) (Continued)

Threats to Validity	Discussion of Threat
Omitted Variable Bias (Continued)	In our estimation, perhaps the only possible change that is (a) U.S.-specific, (b) post-65-specific, and (c) sufficiently systemic to induce population-level mortality shifts is if Medicare's influence on the U.S. healthcare system caused medical providers to shift screening, treatment, and diagnoses to align with Medicare eligibility. (E.g., if cancer screenings and preventative statin use all started at age 65, as opposed to ages used in other countries.) Even if this were to occur, it would be along Medicare's causal pathway–and therefore considered to be a mechanism, rather than confounder–but we consider it here as it would affect the way in which we interpret our results and consider counterfactuals. As shown in Appendix Figure A25, the U.S. does not appear to have stark differences in key preventative treatment and screening guidelines, relative to other countries.
Dormant Factors	In a similar vein, synthetic control estimates may be biased if there are dormant latent factors that only manifest after the age-65 cutoff. In our context, this could occur if there were health-related factors that did not (fully) manifest until older adulthood, and therefore could not be used for matching in the pre-treatment period. One such example could be health complications due to obesity and/or smoking, which might not manifest until older adulthood. There are several features of our results and robustness tests that address this. First, specifically relating to deaths pertaining to issues like obesity and smoking, we note that our results are much stronger for healthcare-amenable causes of death (Nolte and McKee, 2003), which focus on deaths that should be avoided for those with good healthcare access, rather than causes of death that are often brought on by health behaviors. Notably, lung cancer, chronic lung disease, and artery-related heart diseases–all of which are related to smoking in particular–are excluded from this measure. Additionally, we note the trend break in mortality appears early in the post-treatment (and directly at age 65 for the recent era), suggesting that uncontrolled-for dormant factors are not to blame. Finally, our robustness check limiting our synthetic control match to ages near to the cutoff (Appendix Figure A30) is another piece of evidence against dormant factors, as it matches on ages where those factors are more likely to have manifested.
Overfitting	The last threat is that of overfitting–that is, if the synthetic control is matched on noise or idiosyncratic shocks, it may not appropriately capture latent factors common to the treated and control groups. We address this when using our placebo age-55 'treatment', which constitutes a sort of 'out-of-sample' prediction exercise. We also address it by applying Lasso for selection of our synthetic control group, as recommended by Hollingsworth and Wing (2022), the results of which are shown in Appendix Figure A12.

Notes: The purpose of this table is to discuss specific threats to identification of synthetic control designs detailed in Hollingsworth and Wing (2022) and, as applicable, map these threats to areas of our paper where those concerns are addressed. See in-table discussion and text for more details.