How much has growth of 'reasonable and necessary' medical treatments reduced U.S. mortality?

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Abstract

Only treatments that have been designated as "reasonable and necessary" (R&N) for the diagnosis or treatment of an illness or injury are covered by the U.S. Medicare program. Between 1994 and 2019, the number of R&N treatments that had ever been developed more than doubled, from 1210 to 2845.

Using publicly available data, we investigate the effect of R&N treatment growth on the mortality of Americans during the period 1999-2019. We employ a difference-in-differences (disease by year) research design: we measure attributes (the number and mean vintage) of the R&N treatments for a disease that had ever been developed by or before a year, and analyze their correlation with several measures of mortality from that disease in that year.

Mortality is more strongly related to the mean vintage of R&N treatments ever established than it is to the number of R&N treatments ever established, but estimates based on the two alternative innovation measures are qualitatively similar.

Our estimates indicate that the number of years of potential life lost before ages 85, 75, and 65 from a disease is significantly inversely related to the mean vintage of R&N treatments associated with the disease that had ever been established 0-4 or 0-6 years earlier. Mean age at death from a disease is significantly positively related to the mean vintage of R&N treatments associated with the disease that had ever been established 2-8 years earlier.

We also assess the impact that R&N treatment growth had on the mortality of different demographic groups, defined by education or race. Premature (before age 75) mortality of all education groups (people with 0-11 years, 12 years, or 13+ years of education) was reduced by R&N treatment growth. Innovation may have increased the longevity of more educated people sooner than it increased the longevity of less educated people, perhaps because more educated people have earlier access to innovations, but innovation appears to have had similar long-run (after 7 or 8 years) effects on the longevity of the three education groups.

Premature (before ages 85, 75, and 65) mortality rates of Black Americans is not significantly related to the mean vintage of R&N treatments that had ever been established 0-8 years earlier, but their mean age at death is significantly positively related to the mean vintage of R&N treatments that had ever established 5-8 years earlier. Innovation may have increased the longevity of black people later than it increased the longevity of other racial groups, perhaps because black people had later access to innovations, but the long-run (after 5-8 years) effect of innovation on the longevity of black people appears to be similar to its effects on the longevity of other racial groups.

During the period 1999-2019, age-adjusted rates of premature (before ages 85, 75, and 65) mortality declined by about 20%. We cannot reject the null hypothesis that in the absence of R&N treatment growth, premature mortality rates would not have declined during the period 1999-2019. Some factors other than biomedical innovation (e.g., the decline in smoking rates and the increase in educational attainment) probably contributed to the decline in mortality. However, other factors (the increase in obesity and chronic condition prevalence) probably contributed to an increase in mortality.

I. Introduction

Policymakers and economists recognize that longevity growth (or mortality reduction) is a major component of human development, or economic growth, broadly defined. Longevity growth is one of the three components of the Human Development Index (HDI) published by the United Nations Development Programme (2025).[1] Nordhaus (2003) argued that "to a first approximation, the economic value of increases in longevity in the last hundred years is about as large as the value of measured growth in non-health goods and services."

Many leading economists believe that at least one component of economic growth—GDP growth—is primarily driven by technological progress, which is generated by R&D investment. Jones (2002) presented a model in which long-run growth is driven by the discovery (via research effort) of new ideas throughout the world. His model built upon a large collection of previous research, including Romer (1990), Grossman and Helpman (1991), and Aghion and Howitt (1992), as well as earlier contributions by Phelps (1966), Shell (1966), Nordhaus (1969), and Simon (1986). When it awarded the 2025 Nobel Prize in Economic Sciences to three economists, the Royal Swedish Academy of Sciences (2025) declared that "sustained economic growth occurs when new technologies replace old ones as part of the process known as creative destruction."

The health sector of the U.S. economy is more research-intensive than the overall economy. According to Research America (2025), medical and health R&D as a percent of U.S. health spending was 5.13% in 2018, while, according to the National Center for Science and Engineering Statistics (2025), the ratio of U.S. R&D to GDP was 2.92%.

In this study, we will investigate the effect of technological progress, as embodied in new medical services, supplies, and equipment, on another major component of U.S. economic growth during the period 1999-2019:[2] the longevity growth (or mortality reduction) of Americans. In addition to assessing the impact that those innovations had on the mortality of all Americans, we will assess the impact that they had on the mortality of different demographic

[1] The HDI is the geometric mean of normalized indices of three variables: life expectancy at birth, mean years of schooling, and gross national income per capita.

[2] 1999 is the first year in which the ICD-10-CM cause of death classification was used. To avoid the impact of the COVID-19 pandemic, our sample period ends in 2019. From 2010 to 2019, the age-adjusted mortality rate declined by 4.3%. From 2019 to 2021, the age-adjusted mortality rate increased by 23%. See Appendix Figure 1.

groups, defined by education or race. All of the data we will use are publicly available on the internet.

We will employ a difference-in-differences (disease by year) research design: we will construct several measures of the medical treatments (items and services) officially designated as 'reasonable and necessary' (R&N) to treat a disease that had ever been developed by or before a year,[3] and analyze their correlation with several measures of mortality from that disease in that year. Medicare coverage is limited to treatments that are reasonable and necessary for the diagnosis or treatment of an illness or injury (and within the scope of a Medicare benefit category).

As shown in Figure 1, between 1994 and 2019, the number of R&N treatments used to treat at least one disease that had ever been developed more than doubled, from 1210 to 2845. As illustrated by Figure 2, the magnitude of the increase varied considerably across diseases. From 1994 to 2019, the number of R&N treatments associated with "malignant neoplasms of other and unspecified sites within the lip, oral cavity and pharynx" (C03-C09, C14.2-C14.8) increased by 66%, from 172 to 285, while the number of R&N treatments associated with "infections with a predominately sexual mode of transmission: other and unspecified syphilis" (A50-A51, A52.7-A52.9, A53) increased by 132%, from 170 to 395. We will estimate econometric models to test the hypothesis that there were larger reductions in mortality from diseases for which there was more growth in R&N treatments.

In the next section, we discuss the measurement of R&N treatments, by disease and year. In section III, we present our econometric model of mortality. Data sources and descriptive statistics are presented in section IV. Empirical results are presented in section V. Implications of the estimates are discussed in section VI. The final section provides a summary.

II. Measurement of 'reasonable and necessary' (R&N) treatments

There are three ways in which an item or service can be covered by Medicare. First, it can be covered under a National Coverage Determination (NCD): a determination by the Centers for Medicare & Medicaid Services (CMS) that a particular item or service is covered nationally

[3] During the period 2010-2023, data on the number of R&N treatments actually provided by Medicare are available. During that period, growth in the number of R&N treatments actually provided is strongly positively correlated across diseases with growth in the number of R&N treatments that had ever been developed.

under Medicare. Second, it can be covered under a Local Coverage Determination (LCD): a determination by one or more Medicare Administrative Contractors (MACs) that a particular item or service is covered on a contractor-wide basis in accordance with the "reasonable and necessary" standard. LCDs may be developed in the absence of an NCD or as a supplement to an NCD, as long as the LCD policy does not conflict with national policy. Third, in the absence of an LCD or NCD, claims are reviewed on a claim-by-claim basis by the MACs. In such cases, the MACs review the claim on an individual basis to ensure that all CMS requirements have been met prior to paying the claim.

Due to data limitations, our measures of R&N treatments, by disease and year, will be based solely on treatments covered by LCDs. Our R&N treatment measures are therefore subject to measurement error, but according to CMS (2025a), "most Medicare coverage decisions are made locally by the MACs."[4] Moneer et al (2024) also noted that "local coverage determinations…represent the vast majority of Medicare's coverage decisions for new technologies."

Our measures will be based on a CMS database (the "Current and Retired Articles" component of the Medicare Coverage Database) of treatments that have been designated by MACs as 'reasonable and necessary' to treat a disease (Center for Medicare and Medicaid Services (2025b, 2025c)). This database contains 575,664 links between 3404 medical items and services and 7344 (narrowly defined) covered disease (ICD-10-CM) codes. As shown in Appendix Table 1, these medical items and services fall into many categories and subcategories, e.g., molecular testing, imaging, chemotherapy, and cardiovascular procedures. Medical items and services are defined by Healthcare Common Procedure Coding System (HCPCS) codes (see Gutmann (2024)). HCPCS was established in 1978 by the Federal government to standardize the reporting of medical services, supplies, and equipment, essentially creating a unified system for medical billing across different healthcare providers and payers. While initially voluntary, the Health Insurance Portability and Accountability Act (HIPAA) of 1996 made the use of HCPCS codes mandatory for all healthcare transactions. Appendix Table 2 provides an illustration of the data for a single disease, Salmonella sepsis (A02.1). Thirty-one HCPCS codes

[4] The number of treatments (HCPCS codes) covered by LCDs (3404) is more than ten times the number covered by NCDs (200-300). According to the Medicare Coverage Database (Centers for Medicare and Medicaid Services (2025b)), as of 21 December 2024, there were 338 NCDs and 2324 LCDs.

(listed in order of year of HCPCS code establishment) are covered in at least one LCD for that disease.

We will analyze the impacts of two attributes of the R&N treatments for each disease in each year: the *number* (or variety) of treatments, and the mean *vintage* (year of code establishment) of treatments. Numerous authors have provided both theoretical and empirical support for the hypothesis that increased product variety is an important source of economic growth. Dixit and Stiglitz (1977, p. 297) argued that "less variety…entails some welfare loss." Judd (1985) and Romer (1990) modeled growth based on horizontal product innovations, using the Dixit-Stiglitz model of product variety. Segerstrom et al (1990) noted that most of the product-life-cycle literature has treated product innovation as being the introduction of greater variety. Grossman and Helpman (1991, p. 53) treat "technology-based growth as a process of generating an ever-expanding *variety* of horizontally differentiated products" [emphasis added]. Their consumption index exhibits *love of variety* over an infinite set of horizontally differentiated products, as in Dixit and Stiglitz. Utilizing panel data for 19 OECD countries, Funke and Ruhwedel (2001) found that an index of relative product variety was significantly positively correlated across countries with relative per capita income levels.

There is also theoretical and empirical support for the hypothesis that the *vintage* of technology or capital has a positive effect on economic growth. As noted by Jovanovic and Yatsenko (2012), in "the Spence–Dixit–Stiglitz tradition…new goods [are] of higher quality than old goods." In 1987, when the Royal Swedish Academy of Sciences (1987) awarded the Nobel Prize in Economic Sciences to Robert Solow for his contributions to the theory of economic growth, it noted that "the most important aspect of Solow's article was not so much the empirical outcome, but the method of analyzing 'vintage capital'."

III. Econometric model of mortality

Our assessment of the impact that R&N treatments had on the mortality of Americans will be based on the following two-way fixed effects model:

$$\ln(\text{mortality}_{dt}) = \beta_k \, \text{r\&n_measure}_{d,t-k} + \alpha_d + \delta_t + \varepsilon_{dt} \qquad (1)$$

where mortality_{dt} is one of the following variables:

$ypll85_{dt}$ = the number of years of potential life lost before age 85 due to disease d in year t (t = 1999, 2000, 2001,…, 2019)

$ypll75_{dt}$ = the number of years of potential life lost before age 75 due to disease d in year t

$ypll65_{dt}$ = the number of years of potential life lost before age 65 due to disease d in year t[5]

age_death_{dt} = the mean age at death from disease d in year t

n_deaths_{dt} = the number of deaths from disease d in year t

and $r\&n_measure_{d,t-k}$ is one of the following variables:

$\ln(n_r\&n_{d,t-k})$ = the log of the number of R&N treatments for disease d whose codes were established by year t-k (k = 0, 1, 2,…, 8)

$vint_r\&n_{d,t-k}$ = the mean vintage (year of code establishment) of R&N treatments for disease d whose codes were established by year t-k

$n_r\&n_{d,t-k}$ is calculated as follows:

$n_r\&n_{d,t-k} = \sum_h covered_{hd}\ established_{h,t-k}$

where

$covered_{hd}$ = 1 if HCPCS code h is covered for disease d in at least 1 LCD
= 0 if HCPCS code h is not covered for disease d in at least 1 LCD

$established_{h,t-k}$ = 1 if HCPCS code h was established by year t-k
= 0 if HCPCS code h was not established by year t-k

$vint_r\&n_{d,t-k}$ is calculated as follows:

$vint_r\&n_{d,t-k} = (\sum_h covered_{hd}\ established_{h,t-k}\ hcpcs_year_h) / (\sum_h covered_{hd}\ established_{h,t-k})$

where

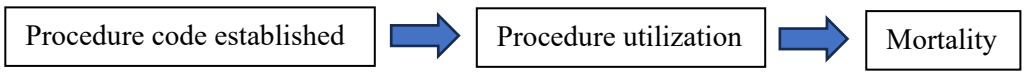
[5] The U.S. CDC's WISQARS Years of Potential Life Lost (YPLL) Report website permits one to specify age thresholds of 65, 70, 75, 80, and 85. The World Health Organization has used YPLL to measure disease burden in its Global Burden of Disease (GBD) and Global Health Estimates (GHE) reports for many years. In the 2010 GBD, the WHO used an age threshold of 86.01 years for all persons. In the current GHE, the WHO uses an age threshold of 91.93 years for all persons.

$hcpcs_year_h$ = the year in which HCPCS code h was established

In eq. (1), α_d is a fixed effect for disease d, and δ_t is a fixed effect for year t. The year fixed effects (δ_t's) in eq. (1) control for the effects of changes in aggregate demographic and macroeconomic variables (e.g. population size and age structure, GDP, educational attainment), to the extent that those variables have similar effects on mortality from different diseases.

Eq. (1) will be estimated by weighted least-squares. When $mortality_{dt} \neq age_death_{dt}$, the weight will be $\sum_t mortality_{dt}$. When $mortality_{dt} = age_death_{dt}$, the weight will be n_deaths_{dt}. Disturbances will be clustered within diseases.

Eq. (1) allows the effect on mortality of the number or vintage of R&N treatments ever established to be subject to a lag. There may be a lag for two reasons, which may be depicted as follows:



First, peak utilization of a procedure may occur years after the code for the procedure was established. We can obtain some evidence about the shape of the typical procedure "age-utilization profile"—the relationship between time since procedure code establishment and procedure utilization—using annual 2013-2022 CMS data (Center for Medicare and Medicaid Services (2025d)). Appendix Figure 2 shows the mean number of times each procedure was used, by age (number of years after code establishment) of procedure. Surprisingly, mean utilization of procedures is about half a million in the year that the code was established (and in each of the following 2 years). It rises to 700,000 by age 5 and then starts to decline.

A potential explanation for this is that the establishment of a HCPCS code may be a "lagging indicator": procedures may be used years before they are assigned HCPCS codes. For one important type of procedure—provider-administered drugs—we have evidence to support this hypothesis. Medicare contractors (e.g., Palmetto GBA (2025)) publish "NDC/HCPCS Crosswalks": listings of each National Drug Code (NDC) that is assigned to a HCPCS. From the Medical Expenditure Panel Survey (MEPS) Prescribed Medicine Files (Agency for Healthcare Research and Quality (2025)), we can obtain estimates of the number of prescriptions by NDC and year during the period 1996-2022. Our calculations reveal that 16% of MEPS prescriptions for NDCs linked to HCPCS codes were consumed in years prior to the year the HCPCS code

was established. This provides support for the hypothesis that procedures may be used years before they are assigned HCPCS codes.

The diseases identified in the LCDs are extremely detailed; 7344 distinct diseases are identified. The diseases identified in the mortality data are less detailed; 3914 distinct ICD-10-CM causes of death are identified in the 2019 mortality data. We will use the CDC's 358 cause of death classification (Centers for Disease Control and Prevention (2025a; page 24)) to aggregate the data and perform the analysis. External causes of mortality (*U01-*U03, V01-Y89) are excluded.

IV. Data sources

R&N treatment data. Data on $covered_{hd}$ (which indicates whether HCPCS code h is covered for disease d in at least 1 local coverage determination) were obtained from CMS' Current and Retired Articles database, which contains data on the most current, in effect (or future effective), and retired, Local Coverage Article versions. Data on $hcpcs_year_h$ (the year in which HCPCS code h was established) were obtained from the Restructured BETOS Classification System database (Center for Medicare and Medicaid Services (2025e)). Data on the number of services and Medicare expenditure, by HCPCS code and year (2013-2022), were obtained from the Medicare Physician & Other Practitioners - by Geography and Service database (Center for Medicare and Medicaid Services (2025d)).

Mortality Data were obtained from annual 1999-2019 Multiple Cause-of-Death Mortality files derived from the National Vital Statistics System of the National Center for Health Statistics. These files may be downloaded from the National Bureau of Economic Research (2025) website. Each record in the microdata is based on information abstracted from death certificates filed in vital statistics offices of each State and District of Columbia. Causes of death were coded according to the International Classification of Diseases, Tenth Revision from 1999 on. Each record contains the 358-cause recode of the underlying cause of death, the decedent's age at time of death, educational attainment, and race.

Appendix Table 3 displays 1999 and 2019 mortality and R&N treatment data for the top 38 diseases, ranked by number of deaths during 1999-2019.

V. Empirical results

Estimates of eq. (1) when $r\&n_measure_{d,t-k} = \ln(n_r\&n_{d,t-k})$ for different mortality measures are reported in Appendix Table 4. Estimates of eq. (1) when $r\&n_measure_{d,t-k} = vint_r\&n_{d,t-k}$ for different mortality measures are reported in Table 1 and plotted in Figure 3. The estimates based on the two alternative innovation measures are qualitatively similar, but mortality is more strongly related to the mean vintage of R&N treatments ever established than it is to the number of R&N treatments ever established, so henceforth we will focus on estimates of eq. (1) when $r\&n_measure_{d,t-k} = vint_r\&n_{d,t-k}$.

In panel A of Table 1 and Figure 3, the dependent variable is $\ln(ypll85_{dt})$: the log of the number of years of potential life lost before age 85 due to disease d in year t. $\ln(ypll85_{dt})$ is significantly inversely related to the mean vintage of R&N treatments ever established 0 to 4 years earlier. It is most strongly inversely related to the mean vintage of R&N treatments ever established one year earlier. The point estimate of β_1 indicates that a one-year increase in R&N treatment vintage reduced premature (before age 85) mortality one year later by 1.4%. Between 1999 and 2019, the weighted (by $ypll85_{dt}$) mean value of $vint_r\&n_{d,t-1}$ increased by 10.6 years (from 1987.4 to 1998.0). This implies that the 1998-2018 increase in vintage reduced premature (before age 85) mortality by 14.6% (= .0138 * 10.6). During the period 1999-2019, the age-adjusted rate of the number of years of potential life lost before age 85 per 100,000 population declined by about 20%.

In panel B of Table 1 and Figure 3, the dependent variable is $\ln(ypll75_{dt})$: the log of the number of years of potential life lost before age 75 due to disease d in year t. $\ln(ypll75_{dt})$ is significantly inversely related to the mean vintage of R&N treatments ever established 0 to 6 years earlier. It is most strongly inversely related to the mean vintage of R&N treatments ever established three years earlier. The point estimate of β_3 indicates that a one-year increase in R&N treatment vintage reduced premature (before age 75) mortality three years later by 1.7%.

In panel C of Table 1 and Figure 3, the dependent variable is $\ln(ypll65_{dt})$: the log of the number of years of potential life lost before age 65 due to disease d in year t. $\ln(ypll65_{dt})$ is significantly inversely related to the mean vintage of R&N treatments ever established 0 to 6 years earlier. It is most strongly inversely related to the mean vintage of R&N treatments ever

established three years earlier. The point estimate of β_3 indicates that a one-year increase in R&N treatment vintage reduced premature (before age 65) mortality three years later by 1.8%.

In panel D of Table 1 and Figure 3, the dependent variable is $\ln(age_death_{dt})$: the log of the mean age at death from disease d in year t. $\ln(age_death_{dt})$ is significantly positively related to the mean vintage of R&N treatments ever established 2 to 8 years earlier. It is most strongly related to the mean vintage of R&N treatments ever established five years earlier. The point estimate of β_5 indicates that a one-year increase in R&N treatment vintage increased mean age at death five years later by 0.23%. During the period 1999-2019, mean age at death increased by 1.38%.

In panel E of Table 1, the dependent variable is $\ln(n_deaths_{dt})$: the log of the number of deaths from disease d in year t. $\ln(n_deaths_{dt})$ is not significantly related to the mean vintage of R&N treatments ever established in year t or any of the previous 8 years. The innovation-induced reductions in the number of years of potential life lost before ages 85, 75, and 65 appear to be due to an increase in mean age at death from the disease rather than to a reduction in the number of deaths from the disease.

As noted above, we can assess the impact that innovation had on the mortality of different demographic groups, defined by education or race, by estimating eq. (1) using group-specific mortality data. Estimates of eq. (1) when $r\&n_measure_{d,t-k} = vint_r\&n_{d,t-k}$, by educational attainment of the decedent, are shown in Appendix Table 5 and plotted in Figure 4. Educational attainment is divided into three categories: 0-11 years, 12 years, and 13+ years of education. The left side of Appendix Table 5 (panels A, B, and C), and the top of Figure 4 (panel A), show the estimates when $mortality_{dt} = ypll75_{dt}$. For all three education groups, $\ln(ypll75_{dt})$ is significantly inversely related to the mean vintage of R&N treatments ever established 0 to 5 years earlier. This indicates that premature (before age 75) mortality of all education groups was reduced by medical innovation. Surprisingly, medical innovation appears to have had a larger (more negative) impact on the mortality of people with 12 years of education than it had on the mortality of people with either less or more education, although the estimated differences may not be statistically significant.

The right side of Appendix Table 5 (panels D, E, and F), and the bottom of Figure 4 (panel B), show the estimates by education group when $mortality_{dt} = age_death_{dt}$. Mean age at death of people with 12 or 13+ years of education is significantly positively related to the mean

vintage of R&N treatments ever established 0-8 years earlier, but mean age at death of people with 0-11 years of education is only significantly positively related to the mean vintage of R&N treatments ever established 7-8 years earlier. This may indicate that innovation increased the longevity of more educated people sooner than it increased the longevity of less educated people, perhaps because more educated people had earlier access to innovations. However, innovation appears to have similar had long-run (after 7 or 8 years) effects on the longevity of the three education groups.

To assess whether innovation had different effects on the mortality of different racial groups, we also estimated eq. (1) using only black mortality data. These estimates are shown in Appendix Table 6. $vint_r\&n_{d,t-k}$ did not have a significant effect on any of the three premature mortality measures ($\ln(ypll85_{dt})$, $\ln(ypll75_{dt})$, $\ln(ypll65_{dt})$) for any value of k. The standard errors of the estimates in Table 1 and Appendix Table 6 are quite similar, so the insignificance of the estimates in Panels A, B, and C of Appendix Table 6 does not appear to be due to greater volatility of the black mortality data. Mean age at death of blacks is significantly positively related to the mean vintage of R&N treatments ever established 5-8 years earlier. The estimates may indicate that innovation increased the longevity of black people later than it increased the longevity of other racial groups, perhaps because black people had later access to innovations. However, the long-run (after 5-8 years) effect of innovation on the longevity of black people appears to be similar to its effects on the longevity of other racial groups.

VI. Discussion

Estimates of two versions of eq. (1)—including and excluding $r\&n_measure_{d,t-k}$ as a regressor—enable us to estimate how much biomedical innovation reduced mortality during the period 1999-2019. When $r\&n_measure_{d,t-k}$ is included in eq. (1), δ_{2019} is an estimate of the 1999-2019 change in mortality, holding constant $r\&n_measure_{d,t-k}$, i.e., in the *absence* of biomedical innovation. (δ_{1999} is normalized to zero.) When $r\&n_measure_{d,t-k}$ is excluded from eq. (1), δ_{2019} is an estimate of the 1999-2019 change in mortality in the *presence* of biomedical innovation. Changes in age-adjusted mortality *rates* can be computed by subtracting the corresponding log-change in age-adjusted population from the δ_{2019} estimates.

These calculations are shown for four mortality measures (when $r\&n_measure_{d,t-k}$ = $vint_r\&n_{d,t-k}$) in Figure 5. In each case, we use the lag for which the relationship is most significant. Panel A shows estimates of the post-1999 log change in the age-adjusted ypll85 rate in the presence and absence of the change in vint_r&n one year earlier. The growth (log-change) in the presence of biomedical innovation is -0.20 and is significantly different from zero. The growth in the absence of biomedical innovation is -0.05 and is not significantly different from zero. We cannot reject the null hypothesis that in the absence of biomedical innovation, as measured by the lagged change in vint_r&n, the age-adjusted ypll85 rate would not have declined during the period 1999-2019. The estimates for the age-adjusted ypll75 and ypll65 rates in Panels B and C are quite similar. As shown in Panel D, mean age at death was 0.98% higher in 2019 than it was in 1999 (controlling for disease fixed effects), and this increase was statistically significant. Our estimates indicate that, if vint_r&n had not increased between 1994 and 2014, mean age at death would have been 1.23% *lower* in 2019 than it was in 1999, although this estimated decline is not statistically significantly different from zero.

Some factors other than biomedical innovation probably contributed to the decline in mortality. The adult cigarette smoking rate declined from 23.5% in 1999 to 13.7% in 2018 (American Lung Association (2025)). The fraction of adults who had at least a bachelor's degree increased from 23.9% in 1997 to 34.2% in 2017 (American Council on Education (2025)). However, other factors probably contributed to an increase in mortality. From 1999-2000 through 2017-March 2020, US obesity prevalence increased from 30.5% to 41.9%, and the prevalence of severe obesity increased from 4.7% to 9.2%. Obesity-related conditions include heart disease, stroke, type 2 diabetes and certain types of cancer. These are among the leading causes of preventable, premature death (Centers for Disease Control and Prevention (2025b)). And between 2007 and 2018, the prevalence of 14 out of 21 chronic conditions among male Medicare beneficiaries increased, and the prevalence of 16 out of 21 chronic conditions among female beneficiaries increased (Center for Medicare and Medicaid Services (2025f)).

VII. Summary and conclusions

Only treatments that have been designated as "reasonable and necessary" (R&N) for the diagnosis or treatment of an illness or injury are covered by the U.S. Medicare program. Between 1994 and 2019, the number of R&N treatments that had ever been developed more than doubled, from 1210 to 2845.

Using publicly available data, we investigated the effect of R&N treatment growth on the mortality of Americans during the period 1999-2019. We employed a difference-in-differences (disease by year) research design: we measured attributes (the number and mean vintage) of the R&N treatments for a disease that had ever been developed by or before a year, and analyzed their correlation with several measures of mortality from that disease in that year.

Mortality is more strongly related to the mean vintage of R&N treatments ever established than it is to the number of R&N treatments ever established, but estimates based on the two alternative innovation measures are qualitatively similar.

Our estimates indicated that the number of years of potential life lost before ages 85, 75, and 65 from a disease is significantly inversely related to the mean vintage of R&N treatments associated with the disease that had ever been established 0-4 or 0-6 years earlier. Mean age at death from a disease is significantly positively related to the mean vintage of R&N treatments associated with the disease that had ever been established 2-8 years earlier.

We also assessed the impact that R&N treatment growth had on the mortality of different demographic groups, defined by education or race. Premature (before age 75) mortality of all education groups (people with 0-11 years, 12 years, or 13+ years of education) was reduced by R&N treatment growth. Innovation may have increased the longevity of more educated people sooner than it increased the longevity of less educated people, perhaps because more educated people have earlier access to innovations, but innovation appears to have had similar long-run (after 7 or 8 years) effects on the longevity of the three education groups.

Premature (before ages 85, 75, and 65) mortality rates of Black Americans is not significantly related to the mean vintage of R&N treatments that had ever been established 0-8 years earlier, but their mean age at death is significantly positively related to the mean vintage of R&N treatments that had ever established 5-8 years earlier. Innovation may have increased the longevity of black people later than it increased the longevity of other racial groups, perhaps because black people had later

access to innovations, but the long-run (after 5-8 years) effect of innovation on the longevity of black people appears to be similar to its effects on the longevity of other racial groups.

During the period 1999-2019, age-adjusted rates of premature (before ages 85, 75, and 65) mortality declined by about 20%. We cannot reject the null hypothesis that in the absence of R&N treatment growth, premature mortality rates would not have declined during the period 1999-2019. Some factors other than biomedical innovation (e.g., the decline in smoking rates and the increase in educational attainment) probably contributed to the decline in mortality. However, other factors (the increase in obesity and chronic condition prevalence) probably contributed to an increase in mortality.

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Figure 1
Number of codes for R&N treatments ever established, all diseases, 1994-2019

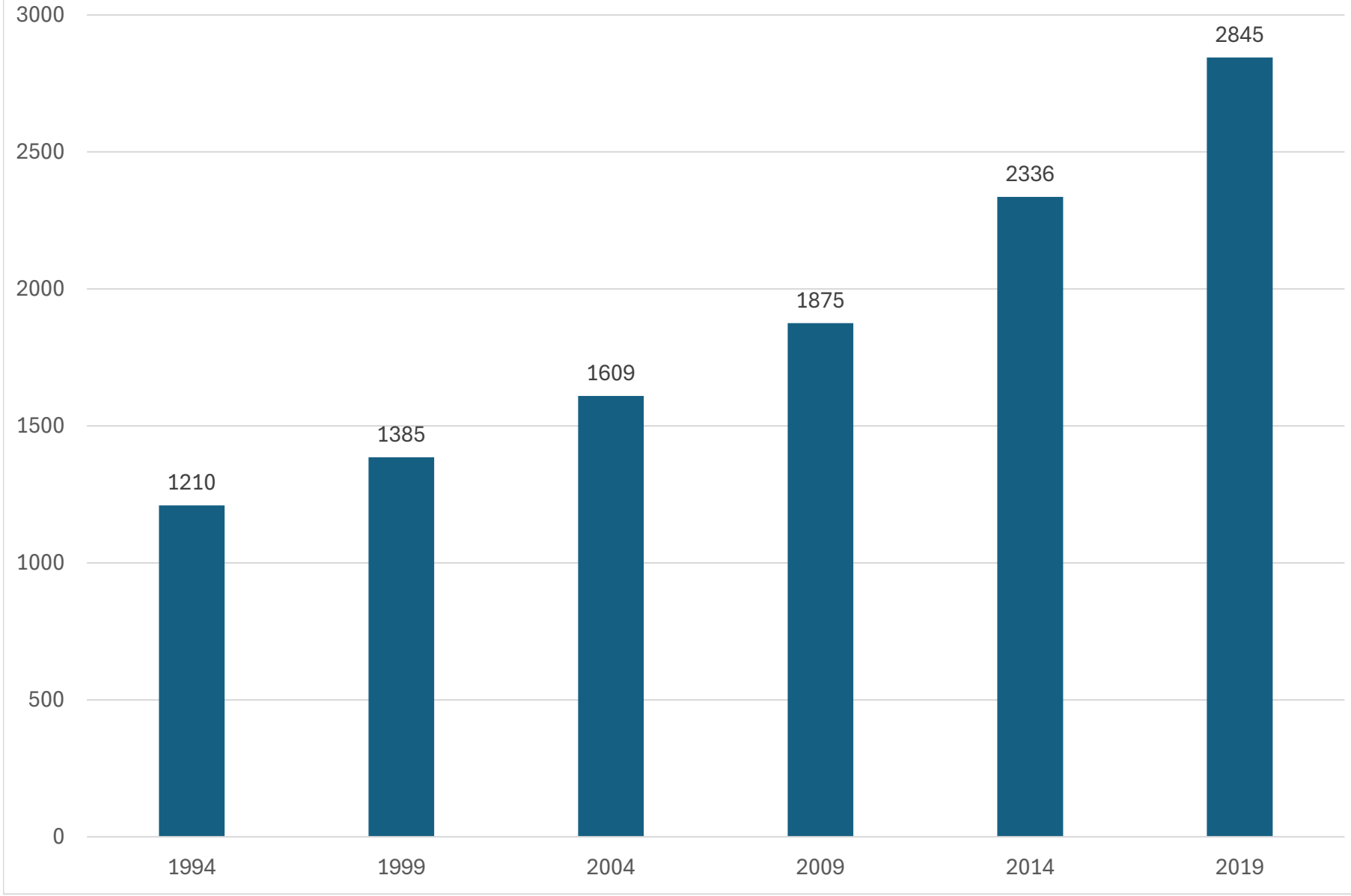


Figure 2

Number of codes for R&N treatments associated with five diseases ever established, 1994-2019

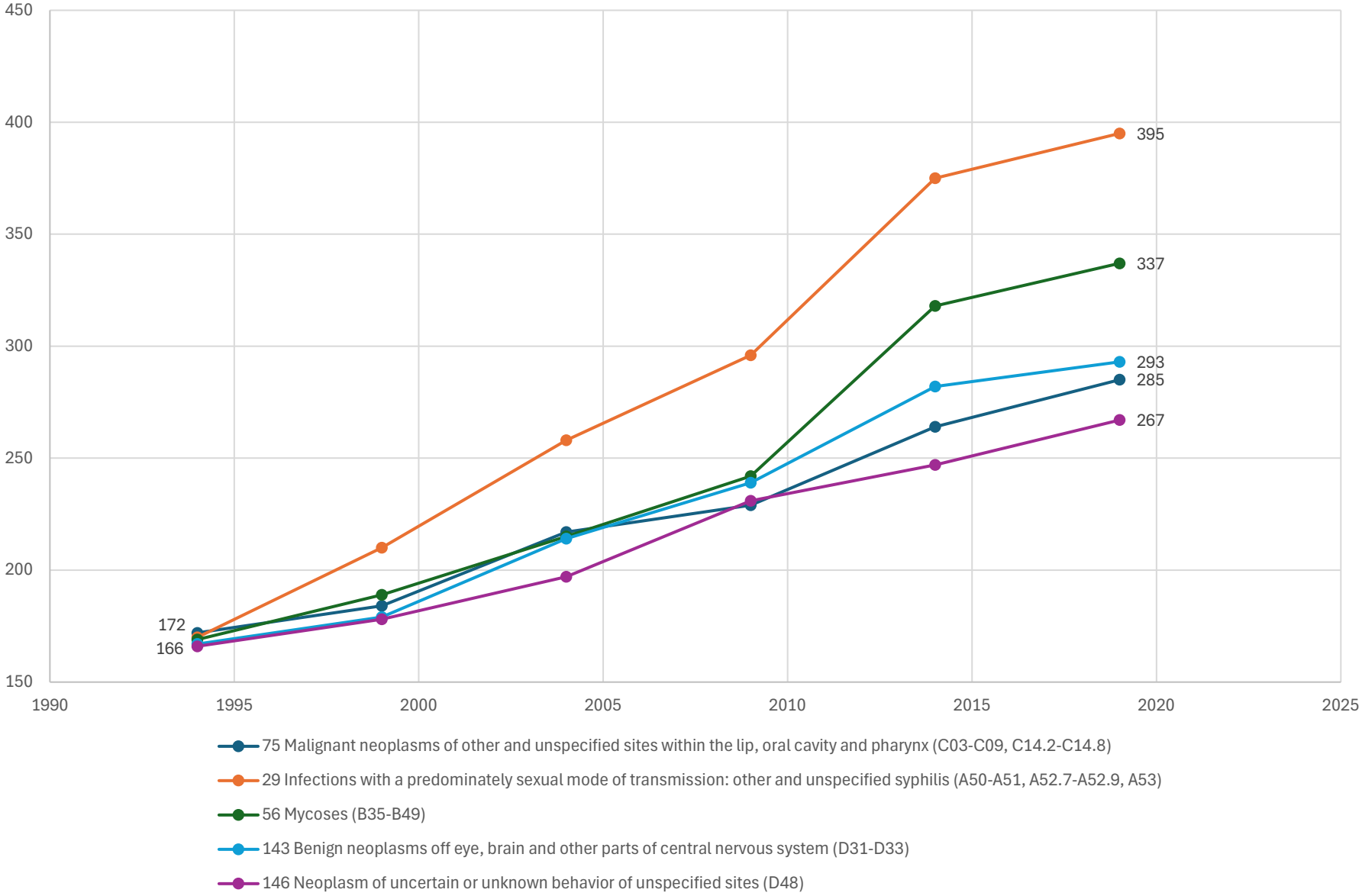
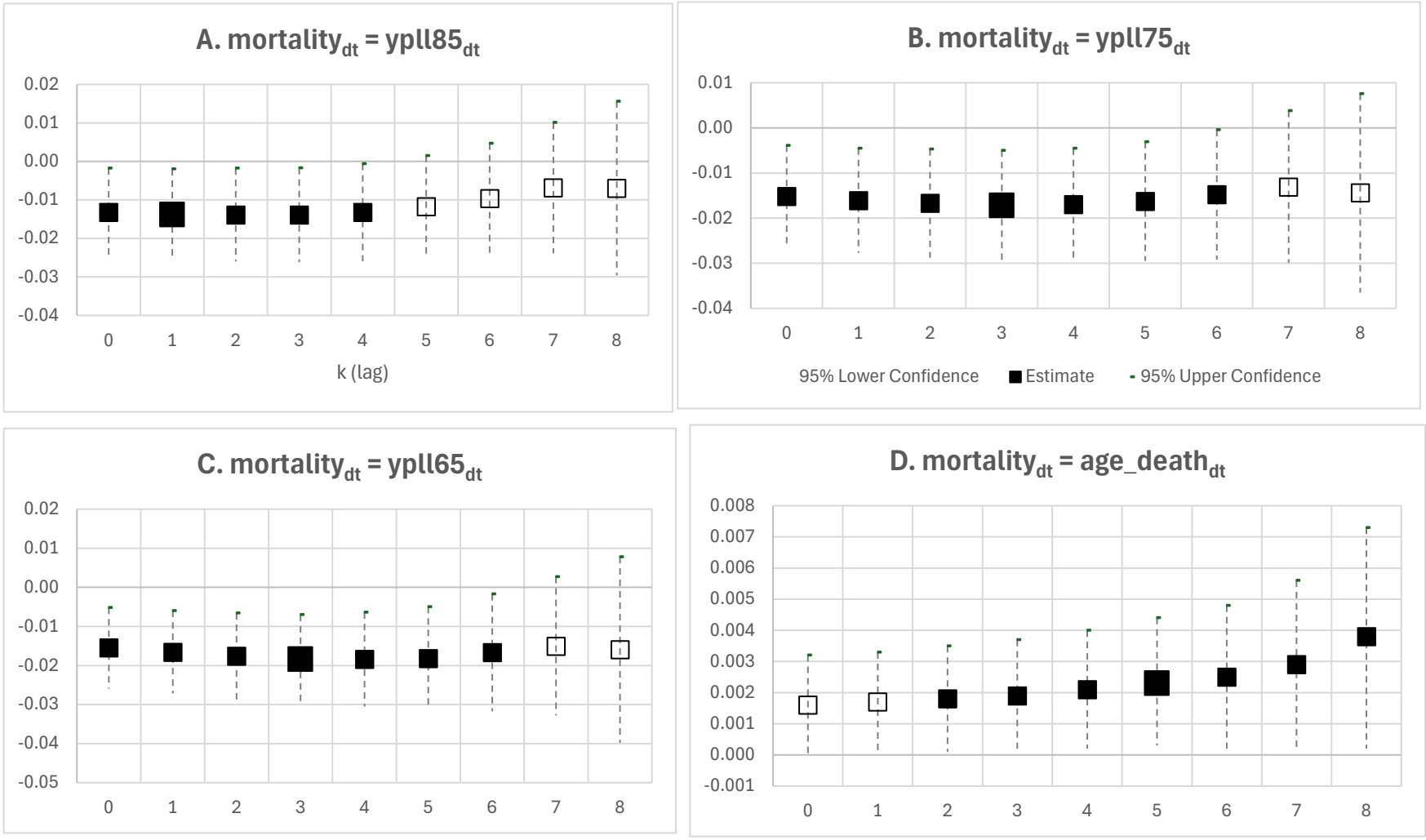


Figure 3

Estimates of β_k from model $\ln(mortality_{dt}) = \beta_k \, vint_r\&n_{d,t-k} + \alpha_d + \delta_t + \varepsilon_{dt}$



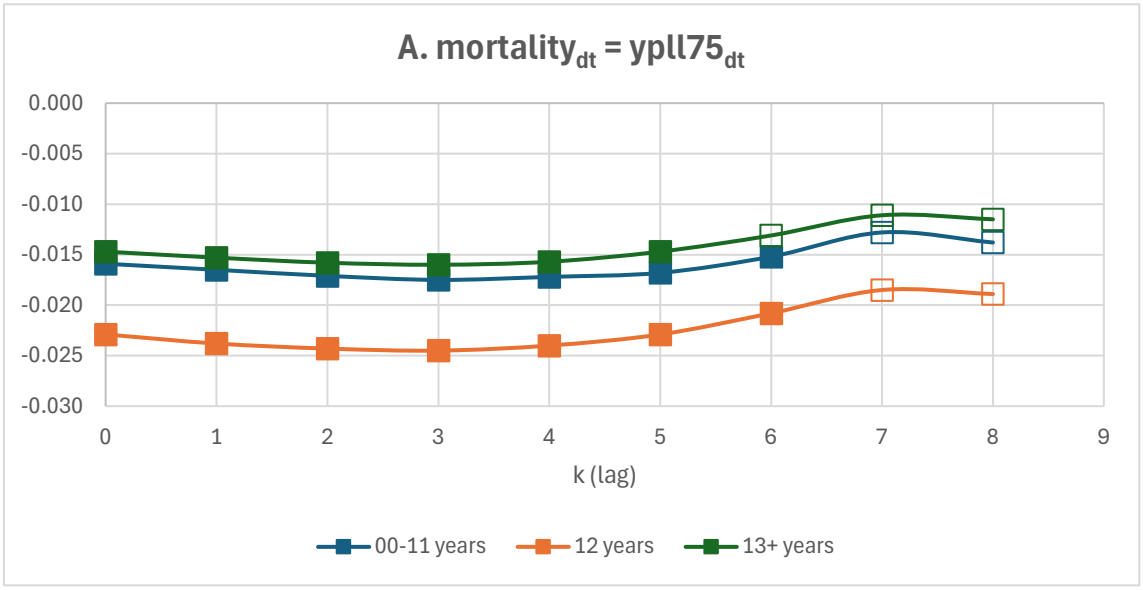
A solid marker indicates a statistically significant (p-value < .05) estimate.

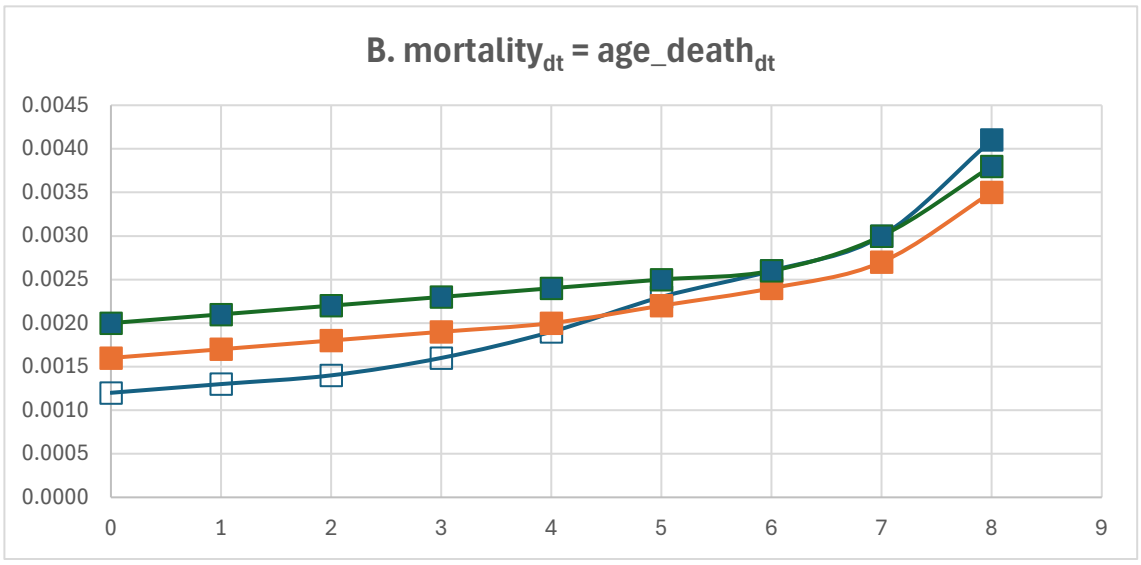
A hollow marker indicates a statistically insignificant estimate.

Figure 4

MORTALITY BY EDUCATIONAL ATTAINMENT

Estimates of β_k from model $\ln(mortality_{dt}) = \beta_k\ vint_r\&n_{d,t-k} + \alpha_d + \delta_t + \varepsilon_{dt}$

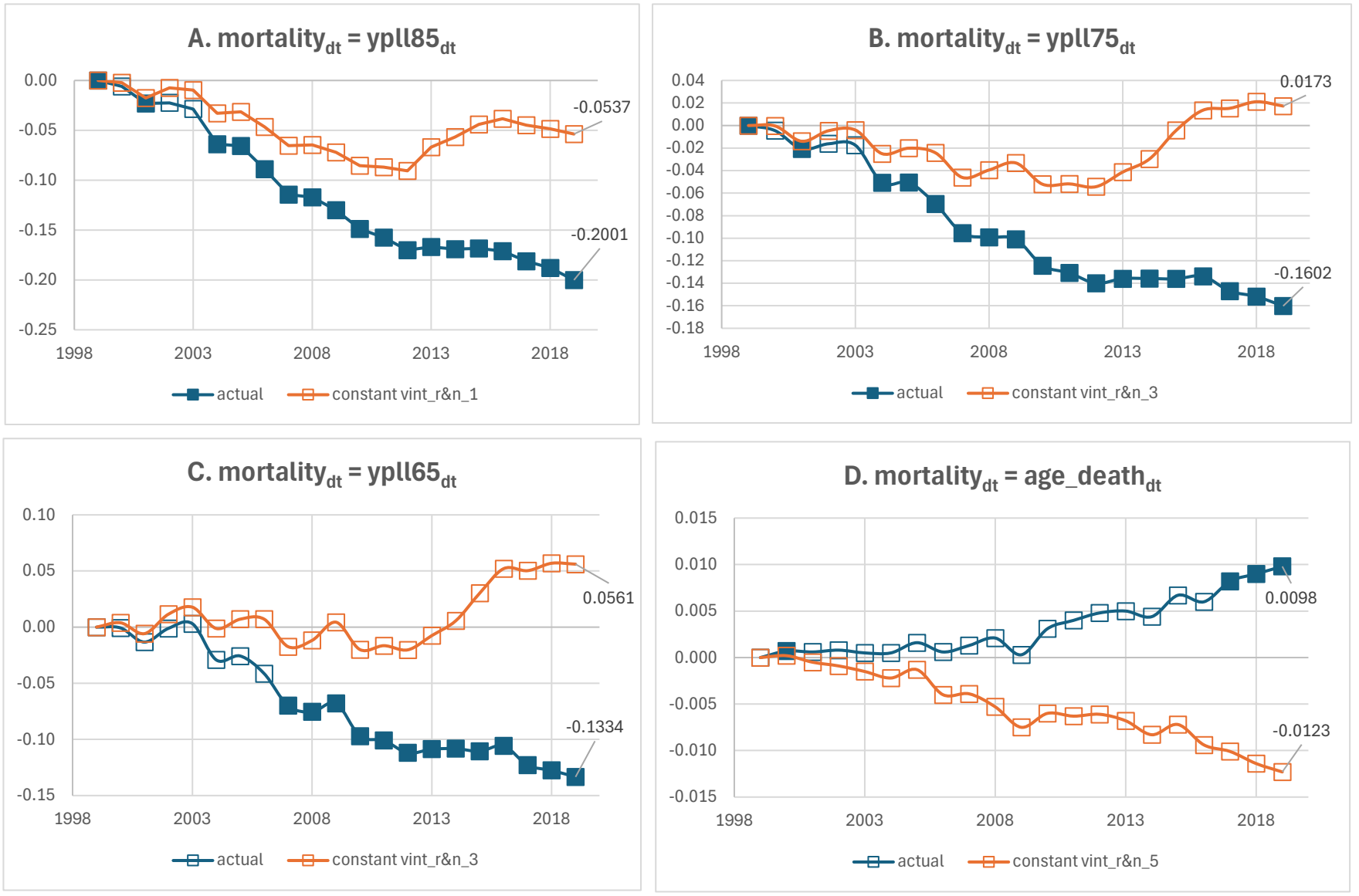




A solid marker indicates a statistically significant (p-value < .05) estimate.
A hollow marker indicates a statistically insignificant estimate.

Figure 5

Actual versus counterfactual (constant lagged vint_r&n) post-1999 change in mortality, 2000-2019



A solid marker indicates that the value in year t is significantly different (p-value < .05) from the 1999 value.

A hollow marker indicates that the value in year t is not significantly different from the 1999 value.

Table 1

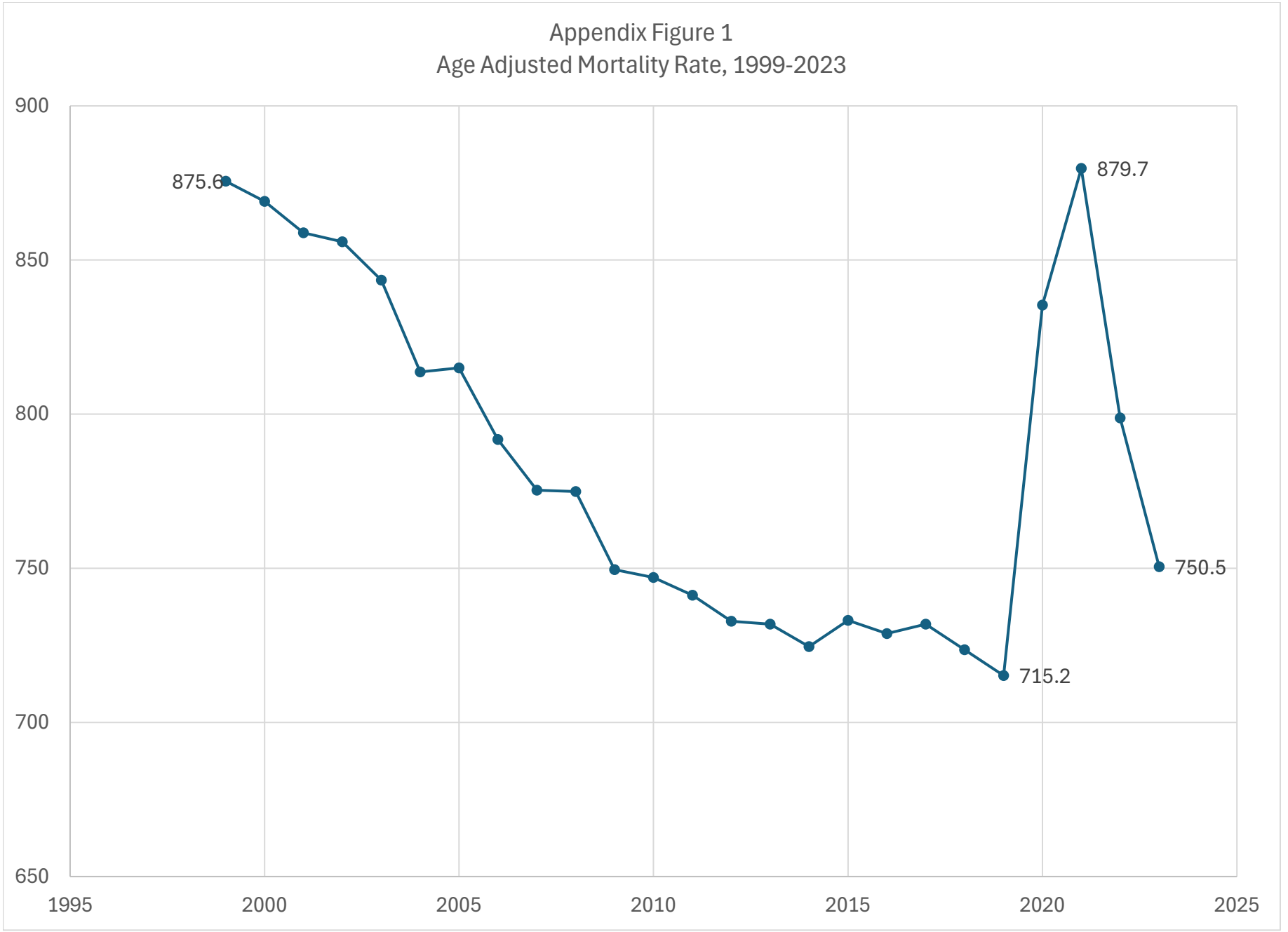
Estimates of β_k from model $\ln(\text{mortality}_{dt}) = \beta_k \text{ vint_r\&n}_{d,t-k} + \alpha_d + \delta_t + \varepsilon_{dt}$

k (lag)	Estimate	Std. Err.	Z	Pr > |Z|		k (lag)	Estimate	Std. Err.	Z	Pr > |Z|
	A. mortality$_{dt}$ = yll85$_{dt}$						**B. mortality$_{dt}$ = yll75$_{dt}$**			
0	**-0.0132**	**0.0058**	**-2.26**	**0.0238**		0	**-0.0152**	**0.0058**	**-2.64**	**0.0083**
1	**-0.0138**	**0.0060**	**-2.30**	**0.0216**		1	**-0.0161**	**0.0059**	**-2.71**	**0.0067**
2	**-0.0139**	**0.0062**	**-2.26**	**0.0241**		2	**-0.0167**	**0.0061**	**-2.73**	**0.0064**
3	**-0.0139**	**0.0063**	**-2.22**	**0.0261**		3	**-0.0172**	**0.0062**	**-2.76**	**0.0058**
4	**-0.0132**	**0.0065**	**-2.05**	**0.0407**		4	**-0.0170**	**0.0064**	**-2.66**	**0.0079**
5	-0.0118	0.0068	-1.73	0.0829		5	**-0.0163**	**0.0067**	**-2.42**	**0.0154**
6	-0.0097	0.0074	-1.32	0.1868		6	**-0.0148**	**0.0073**	**-2.02**	**0.0435**
7	-0.0069	0.0087	-0.80	0.4256		7	-0.0131	0.0086	-1.52	0.1280
8	-0.0070	0.0115	-0.61	0.5443		8	-0.0144	0.0113	-1.28	0.2000
	C. mortality$_{dt}$ = yll65$_{dt}$						**D. mortality$_{dt}$ = age_death$_{dt}$**			
0	**-0.0155**	**0.0053**	**-2.95**	**0.0032**		0	0.0016	0.0008	1.89	0.0583
1	**-0.0166**	**0.0054**	**-3.07**	**0.0021**		1	0.0017	0.0009	1.95	0.0511
2	**-0.0176**	**0.0056**	**-3.13**	**0.0018**		2	**0.0018**	**0.0009**	**2.05**	**0.0400**
3	**-0.0184**	**0.0058**	**-3.16**	**0.0016**		3	**0.0019**	**0.0009**	**2.16**	**0.0306**
4	**-0.0184**	**0.0062**	**-3.00**	**0.0027**		4	**0.0021**	**0.0010**	**2.21**	**0.0272**
5	**-0.0182**	**0.0067**	**-2.71**	**0.0067**		5	**0.0023**	**0.0011**	**2.22**	**0.0263**
6	**-0.0167**	**0.0077**	**-2.18**	**0.0294**		6	**0.0025**	**0.0012**	**2.09**	**0.0363**
7	-0.0151	0.0091	-1.67	0.0955		7	**0.0029**	**0.0014**	**2.04**	**0.0411**
8	-0.0160	0.0121	-1.32	0.1885		8	**0.0038**	**0.0018**	**2.09**	**0.0367**
	E. mortality$_{dt}$ = n_deaths$_{dt}$									
0	-0.0096	0.0088	-1.09	0.2777						
1	-0.0084	0.0087	-0.97	0.3331						
2	-0.0060	0.0087	-0.68	0.4945						
3	-0.0033	0.0094	-0.35	0.7228						
4	0.0011	0.0109	0.10	0.9170						
5	0.0070	0.0132	0.53	0.5953						
6	0.0131	0.0156	0.84	0.4025						
7	0.0229	0.0195	1.17	0.2400						
8	0.0328	0.0259	1.26	0.2060						

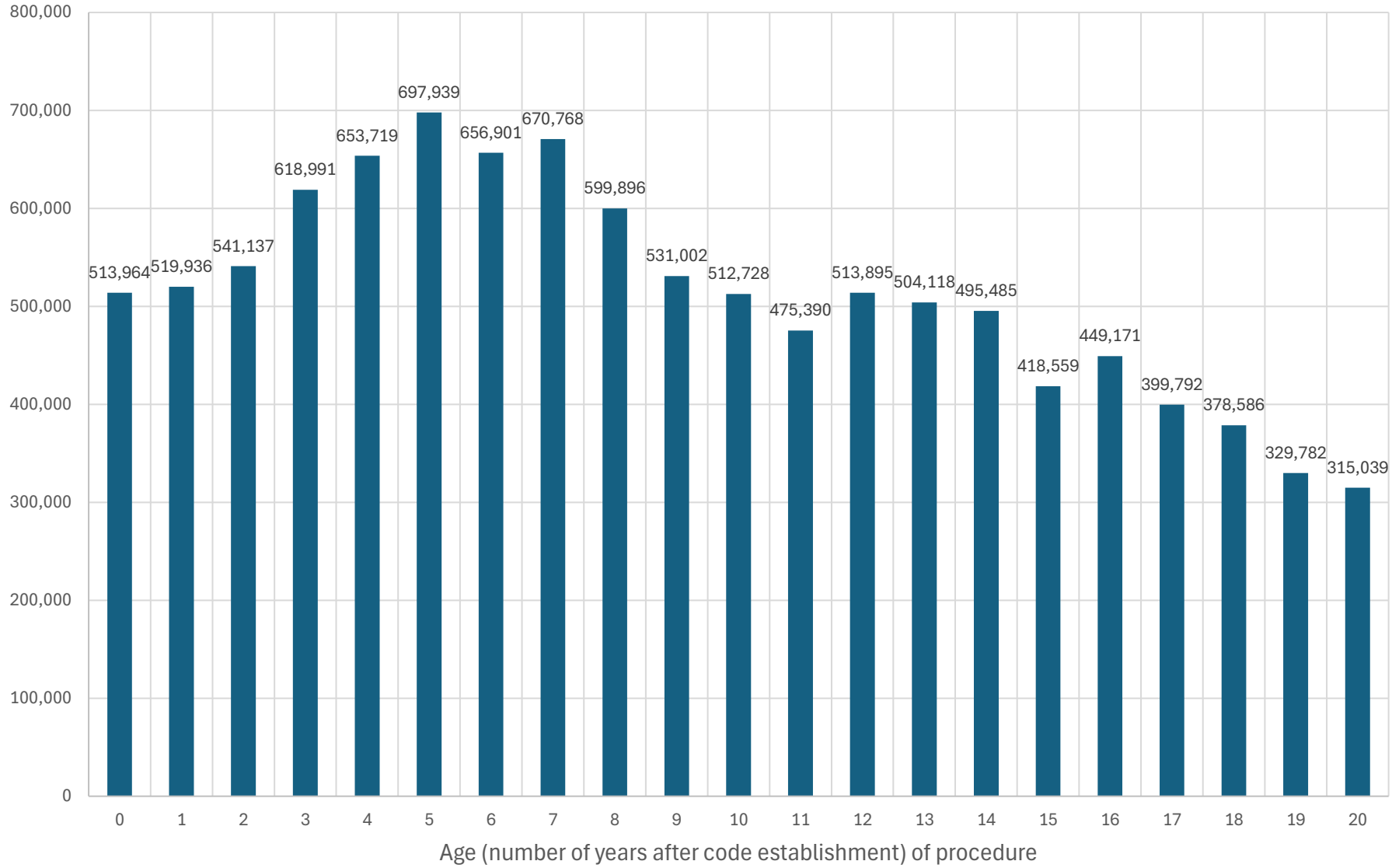
Each estimate is from a separate model.

Estimates in bold are statistically significant (p-value < .05).

Appendix Figure 1
Age Adjusted Mortality Rate, 1999-2023



Appendix Figure 2
Mean number of times each procedure was used,
by age (number of years after code establishment) of procedure



Appendix Table 1

Number of R&N items and procedures by category and subcategory

Anesthesia	**47**
Anesthesia	47
Durable Medical Equipment	**27**
Drugs Administered Through DME	5
Medical/Surgical Supplies	3
Orthotic Devices	3
Other DME	16
Evaluation and Management	**108**
Behavioral Health Services	56
Care Management/Coordination	2
Critical Care Services	2
E&M - Miscellaneous	15
Home Services	15
Observation Care Services	2
Office/Outpatient Services	4
Ophthalmological Services	12
Imaging	**761**
CT Scan	84
Imaging - Miscellaneous	11
Magnetic Resonance	94
Nuclear	136
Standard X-ray	308
Ultrasound	128
Other	**36**
Vision, Hearing, and Speech Services	36

Procedure	**1062**
Breast	42
Cardiovascular	110
Digestive/Gastrointestinal	176
Eye	61
Hematology	1
Musculoskeletal	281
Other Organ Systems	106
Skin	209
Vascular	76
Test	**1324**
Anatomic Pathology	26
Cardiography	66
General Laboratory	131
Molecular Testing	821
Neurologic	102
Pulmonary	37
Pulmonary Function	35
Test - Miscellaneous	106
Treatment	**446**
Chemotherapy	42
Dialysis	11
Injections and Infusions (nononcologic)	249
Physical, Occupational, and Speech Therapy	66
Radiation Oncology	31
Spinal Manipulation	9
Treatment - Miscellaneous	38

Appendix Table 2
HCPCS codes (listed in order of year of HCPCS code establishment) covered in at least one LCD for A02.1 Salmonella sepsis

year_hcpcs	hcpcs	long_description
1982	93005	ELECTROCARDIOGRAM, ROUTINE ECG WITH AT LEAST 12 LEADS; TRACING ONLY, WITHOUT INTERPRETATION AND REPORT
1982	93000	ELECTROCARDIOGRAM, ROUTINE ECG WITH AT LEAST 12 LEADS; WITH INTERPRETATION AND REPORT
1982	93010	ELECTROCARDIOGRAM, ROUTINE ECG WITH AT LEAST 12 LEADS; INTERPRETATION AND REPORT ONLY
1982	93320	DOPPLER ECHOCARDIOGRAPHY, PULSED WAVE AND/OR CONTINUOUS WAVE WITH SPECTRAL DISPLAY (LIST SEPARATELY IN ADDITION TO CODES FOR ECHOCARDIOGRAPHIC IMAGING); COMPLETE
1984	93040	RHYTHM ECG, 1-3 LEADS; WITH INTERPRETATION AND REPORT
1984	93041	RHYTHM ECG, 1-3 LEADS; TRACING ONLY WITHOUT INTERPRETATION AND REPORT
1984	93042	RHYTHM ECG, 1-3 LEADS; INTERPRETATION AND REPORT ONLY
1984	87188	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; MACROBROTH DILUTION METHOD, EACH AGENT
1984	87190	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; MYCOBACTERIA, PROPORTION METHOD, EACH AGENT
1984	87186	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; MICRODILUTION OR AGAR DILUTION (MINIMUM INHIBITORY CONCENTRATION [MIC] OR BREAKPOINT), EACH MULTI-ANTIMICROBIAL, PER PLATE
1984	87181	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; AGAR DILUTION METHOD, PER AGENT (EG, ANTIBIOTIC GRADIENT STRIP)
1984	87184	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; DISK METHOD, PER PLATE (12 OR FEWER AGENTS)
1988	87187	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; MICRODILUTION OR AGAR DILUTION, MINIMUM LETHAL CONCENTRATION (MLC), EACH PLATE (LIST SEPARATELY IN ADDITION TO CODE FOR PRIMARY PROCEDURE)
1989	93312	ECHOCARDIOGRAPHY, TRANSESOPHAGEAL, REAL-TIME WITH IMAGE DOCUMENTATION (2D) (WITH OR WITHOUT M-MODE RECORDING); INCLUDING PROBE PLACEMENT, IMAGE ACQUISITION, INTERPRETATION AND REPORT
1990	93325	DOPPLER ECHOCARDIOGRAPHY COLOR FLOW VELOCITY MAPPING (LIST SEPARATELY IN ADDITION TO CODES FOR ECHOCARDIOGRAPHY)
1990	93321	DOPPLER ECHOCARDIOGRAPHY, PULSED WAVE AND/OR CONTINUOUS WAVE WITH SPECTRAL DISPLAY (LIST SEPARATELY IN ADDITION TO CODES FOR ECHOCARDIOGRAPHIC IMAGING); FOLLOW-UP OR LIMITED STUDY (LIST SEPARATELY IN ADDITION TO CODES FOR
1993	93313	ECHOCARDIOGRAPHY, TRANSESOPHAGEAL, REAL-TIME WITH IMAGE DOCUMENTATION (2D) (WITH OR WITHOUT M-MODE RECORDING); PLACEMENT OF TRANSESOPHAGEAL PROBE ONLY
1993	93314	ECHOCARDIOGRAPHY, TRANSESOPHAGEAL, REAL-TIME WITH IMAGE DOCUMENTATION (2D) (WITH OR WITHOUT M-MODE RECORDING); IMAGE ACQUISITION, INTERPRETATION AND REPORT ONLY
1997	93317	TRANSESOPHAGEAL ECHOCARDIOGRAPHY FOR CONGENITAL CARDIAC ANOMALIES; IMAGE ACQUISITION, INTERPRETATION AND REPORT ONLY

Appendix Table 2
HCPCS codes (listed in order of year of HCPCS code establishment) covered in at least one LCD for A02.1 Salmonella sepsis

year_hcpcs	hcpcs	long_description
1997	93316	TRANSESOPHAGEAL ECHOCARDIOGRAPHY FOR CONGENITAL CARDIAC ANOMALIES; PLACEMENT OF TRANSESOPHAGEAL PROBE ONLY
1997	93315	TRANSESOPHAGEAL ECHOCARDIOGRAPHY FOR CONGENITAL CARDIAC ANOMALIES; INCLUDING PROBE PLACEMENT, IMAGE ACQUISITION, INTERPRETATION AND REPORT
2001	93318	ECHOCARDIOGRAPHY, TRANSESOPHAGEAL (TEE) FOR MONITORING PURPOSES, INCLUDING PROBE PLACEMENT, REAL TIME 2-DIMENSIONAL IMAGE ACQUISITION AND INTERPRETATION LEADING TO ONGOING (CONTINUOUS) ASSESSMENT OF (DYNAMICALLY CHANGING)
2001	87185	SUSCEPTIBILITY STUDIES, ANTIMICROBIAL AGENT; ENZYME DETECTION (EG, BETA LACTAMASE), PER ENZYME
2006	76377	3D RENDERING WITH INTERPRETATION AND REPORTING OF COMPUTED TOMOGRAPHY, MAGNETIC RESONANCE IMAGING, ULTRASOUND, OR OTHER TOMOGRAPHIC MODALITY WITH IMAGE POSTPROCESSING UNDER CONCURRENT SUPERVISION; REQUIRING IMAGE POSTPROC
2006	76376	3D RENDERING WITH INTERPRETATION AND REPORTING OF COMPUTED TOMOGRAPHY, MAGNETIC RESONANCE IMAGING, ULTRASOUND, OR OTHER TOMOGRAPHIC MODALITY WITH IMAGE POSTPROCESSING UNDER CONCURRENT SUPERVISION; NOT REQUIRING IMAGE POST
2008	C8926	TRANSESOPHAGEAL ECHOCARDIOGRAPHY (TEE) WITH CONTRAST, OR WITHOUT CONTRAST FOLLOWED BY WITH CONTRAST, FOR CONGENITAL CARDIAC ANOMALIES; INCLUDING PROBE PLACEMENT, IMAGE ACQUISITION, INTERPRETATION AND REPORT
2008	C8925	TRANSESOPHAGEAL ECHOCARDIOGRAPHY (TEE) WITH CONTRAST, OR WITHOUT CONTRAST FOLLOWED BY WITH CONTRAST, REAL TIME WITH IMAGE DOCUMENTATION (2D) (WITH OR WITHOUT M-MODE RECORDING); INCLUDING PROBE PLACEMENT, IMAGE ACQUISITION
2008	C8927	TRANSESOPHAGEAL ECHOCARDIOGRAPHY (TEE) WITH CONTRAST, OR WITHOUT CONTRAST FOLLOWED BY WITH CONTRAST, FOR MONITORING PURPOSES, INCLUDING PROBE PLACEMENT, REAL TIME 2-DIMENSIONAL IMAGE ACQUISITION AND INTERPRETATION LEADING
2015	87505	INFECTIOUS AGENT DETECTION BY NUCLEIC ACID (DNA OR RNA); GASTROINTESTINAL PATHOGEN (EG, CLOSTRIDIUM DIFFICILE, E. COLI, SALMONELLA, SHIGELLA, NOROVIRUS, GIARDIA), INCLUDES MULTIPLEX REVERSE TRANSCRIPTION, WHEN
2015	93355	ECHOCARDIOGRAPHY, TRANSESOPHAGEAL (TEE) FOR GUIDANCE OF A TRANSCATHETER INTRACARDIAC OR GREAT VESSEL(S) STRUCTURAL INTERVENTION(S) (EG, TAVR, TRANSCATHETER PULMONARY VALVE REPLACEMENT, MITRAL VALVE REPAIR, PARAVALVULAR RE
2022	93319	3D ECHOCARDIOGRAPHIC IMAGING AND POSTPROCESSING DURING TRANSESOPHAGEAL ECHOCARDIOGRAPHY, OR DURING TRANSTHORACIC ECHOCARDIOGRAPHY FOR CONGENITAL CARDIAC ANOMALIES, FOR THE ASSESSMENT OF CARDIAC STRUCTURE(S) (EG, CARDIAC C

Appendix Table 3

1999 and 2019 mortality and R&N treatment data for the top 38 diseases, ranked by number of deaths during 1999-2019

	n_deaths	n_deaths		yll75		age_death		n_r&n		vint_r&n	
358 Cause Recode (ICD-10-CM codes)	**1999-2019**	**1999**	**2019**	**1999**	**2019**	**1999**	**2019**	**1999**	**2019**	**1999**	**2019**
215 All other forms of chronic ischemic heart disease (I20, I25.1-I25.9)	4,423,540	255,317	186,567	823,600	610,454	79.3	79.9	144	291	1990.0	2000.0
93 Of trachea, bronchus and lung (C33-C34)	3,248,862	152,235	139,814	1,088,439	811,992	70.0	72.3	105	229	1986.2	1999.4
211 Acute myocardial infarction (I21-I22)	2,914,111	199,772	104,543	932,348	610,926	76.1	74.6	115	213	1989.8	1998.9
267 Other chronic obstructive pulmonary disease (J44)	2,561,334	99,622	144,869	307,373	511,028	76.9	77.1	136	205	1987.9	1995.0
175 Organic dementia (F01, F03)	1,864,109	28,712	115,125	9,951	50,236	86.8	87.2	82	184	1988.4	1999.9
189 Alzheimer's disease (G30)	1,738,728	44,543	121,539	20,356	53,644	85.1	86.6	113	189	1987.6	1996.8
159 Diabetes mellitus (E10-E14)	1,574,335	68,457	87,795	444,144	645,569	72.7	71.7	234	435	1988.2	1998.2
238 Stroke, not specified as hemorrhage or infarction (I64)	1,495,939	91,121	53,818	163,806	154,125	81.8	80.7				
214 Atherosclerotic cardiovascular disease, so described (I25.0)	1,325,964	72,150	66,459	371,576	501,329	76.2	72.1	1	1	1984.0	1984.0
230 Congestive heart failure (I50.0)	1,182,324	50,833	65,803	79,452	139,744	83.4	83.3	0	1	.	2011.0
257 Pneumonia due to other or unspecified organisms (J16,J18)	1,056,692	57,642	39,123	157,423	165,789	81.9	78.8	86	124	1987.0	1993.7
228 Conduction disorders and cardiac dysrhythmias (I44-I49)	946,871	39,973	56,886	191,077	250,278	77.4	78.4	161	326	1988.6	1999.4
327 Renal failure (N17-N19)	926,348	34,583	50,534	148,826	237,298	77.1	76.6	109	183	1988.9	1997.5
81 Colon (C18)	917,942	48,463	40,710	284,574	298,696	73.4	71.9	92	277	1987.1	2004.1
104 Malignant neoplasm of breast (C50)	877,158	41,557	42,826	432,312	396,333	67.7	69.3	74	291	1986.2	2006.1
125 Other malignant neoplasms of other and unspecified sites (C76-C80, C97)	855,158	43,894	43,506	311,321	274,485	71.5	72.9	326	621	1987.3	1998.1
88 Of pancreas (C25)	764,739	29,113	45,956	194,367	290,714	71.6	71.9	68	266	1986.6	2006.0
23 Septicemia (A40-A41)	751,592	30,434	38,380	170,397	245,501	75.4	73.8	95	150	1987.0	1995.0
207 Hypertensive heart disease (I11)	730,565	22,774	55,581	150,628	386,128	74.9	75.0	98	179	1989.3	1998.3

Appendix Table 3

1999 and 2019 mortality and R&N treatment data for the top 38 diseases, ranked by number of deaths during 1999-2019

	n_deaths	n_deaths		yll75		age_death		n_r&n		vint_r&n	
358 Cause Recode (ICD-10-CM codes)	**1999-2019**	**1999**	**2019**	**1999**	**2019**	**1999**	**2019**	**1999**	**2019**	**1999**	**2019**
194 All other diseases of nervous system (G10-G14, G23-G25, G31, G36-G37, G43-G44, G47-G72, G81-G98)	651,709	17,949	65,572	209,878	346,098	66.2	77.1	365	723	1987.7	1998.4
113 Of prostate (C61)	618,636	31,747	31,673	72,001	90,218	78.5	78.5	66	247	1985.9	2005.5
278 All other diseases of respiratory system (J80-J84,J93-J98)	614,044	22,425	40,745	119,112	185,295	74.8	76.3	175	253	1987.5	1993.9
239 Other cerebrovascular diseases and their sequelae (I67, I69)	612,038	29,530	44,901	51,116	90,581	82.5	83.4	256	491	1988.0	1998.2
236 Intracerebral and other intracranial hemorrhage (I61-I62)	578,363	25,496	28,179	172,279	169,764	72.6	74.5	80	146	1988.5	1997.5
227 Cardiomyopathy (I42)	506,725	27,167	20,389	246,647	185,673	70.3	70.8	123	242	1989.5	1999.3
188 Parkinson's disease (G20-G21)	479,226	14,596	35,327	12,716	35,517	81.4	81.9	152	261	1987.8	1997.3
129 Non-Hodgkin's lymphoma (C82-C85)	438,561	22,831	20,316	172,117	109,555	70.8	74.4	199	337	1987.5	1997.4
273 Pneumonitis due to food and vomit (J69.0)	373,081	15,245	19,192	36,475	67,631	81.7	80.0	1	1	1984.0	1984.0
206 Essential (primary) hypertension (I10)	349,862	9,920	23,089	36,645	105,577	79.3	78.2	61	119	1988.9	1997.3
300 Fibrosis and cirrhosis of liver (K74)	344,866	13,879	20,230	167,741	212,661	64.5	66.3	78	130	1988.0	1996.8
298 Alcoholic liver disease (K70)	344,684	11,971	24,176	241,047	444,014	55.2	56.9	81	133	1987.8	1996.5
85 Liver (C22.0, C22.2-C22.9)	320,165	9,847	20,190	92,753	156,621	68.0	69.6	0	1	.	2019.0
225 Nonrheunatic aortic valve disorders (I35)	310,449	12,218	16,141	25,205	30,206	82.3	84.5	103	186	1989.5	1998.4
118 Of bladder (C67)	302,861	11,916	16,812	47,796	57,232	76.0	77.9	55	130	1985.9	2000.8
109 Of ovary (C56)	300,898	13,641	13,469	116,141	106,071	69.2	70.1	57	235	1986.3	2006.3
293 Vascular disorders and obstruction of intestine without hernia (K55-K56)	299,166	13,747	14,674	49,234	66,530	78.3	77.2	94	181	1987.5	1998.4
122 Of brain (C71)	297,989	12,487	16,909	194,836	211,735	60.8	64.3	73	226	1987.1	2003.2
77 Of esophagus (C15)	294,546	11,923	15,985	100,916	122,814	68.8	69.8	78	157	1986.2	1998.9

Appendix Table 4

Estimates of β_k from model $\ln(\text{mortality}_{dt}) = \beta_k \ln(\text{n_r\&n}_{d,t-k}) + \alpha_d + \delta_t + \varepsilon_{dt}$

k (lag)	Estimate	Std. Err.	Z	Pr > |Z|		k (lag)	Estimate	Std. Err.	Z	Pr > |Z|
	A. $\text{mortality}_{dt} = \text{yll85}_{dt}$						B. $\text{mortality}_{dt} = \text{yll75}_{dt}$			
0	**-0.1769**	**0.0853**	**-2.07**	**0.0380**		0	**-0.1991**	**0.0827**	**-2.41**	**0.0160**
1	**-0.1821**	**0.0889**	**-2.05**	**0.0405**		1	**-0.2096**	**0.0861**	**-2.43**	**0.0149**
2	-0.1803	0.0928	-1.94	0.0521		2	**-0.2156**	**0.0895**	**-2.41**	**0.0160**
3	-0.1784	0.0979	-1.82	0.0684		3	**-0.2184**	**0.0926**	**-2.36**	**0.0183**
4	-0.1691	0.1073	-1.57	0.1153		4	**-0.2147**	**0.0986**	**-2.18**	**0.0295**
5	-0.1465	0.1194	-1.23	0.2200		5	-0.1990	0.1075	-1.85	0.0643
6	-0.1261	0.1318	-0.96	0.3385		6	-0.1800	0.1175	-1.53	0.1256
7	-0.1032	0.1484	-0.70	0.4869		7	-0.1577	0.1314	-1.20	0.2302
8	-0.1449	0.1855	-0.78	0.4348		8	-0.1764	0.1600	-1.10	0.2703
	C. $\text{mortality}_{dt} = \text{yll65}_{dt}$						D. $\text{mortality}_{dt} = \text{age_death}_{dt}$			
0	**-0.1978**	**0.0732**	**-2.70**	**0.0069**		0	0.0202	0.0118	1.71	0.0873
1	**-0.2108**	**0.0771**	**-2.73**	**0.0062**		1	0.0217	0.0123	1.77	0.0770
2	**-0.2214**	**0.0815**	**-2.72**	**0.0066**		2	0.0235	0.0127	1.85	0.0643
3	**-0.2261**	**0.0858**	**-2.64**	**0.0084**		3	0.0250	0.0131	1.91	0.0563
4	**-0.2224**	**0.0937**	**-2.37**	**0.0177**		4	0.0269	0.0141	1.91	0.0561
5	**-0.2088**	**0.1051**	**-1.99**	**0.0470**		5	0.0288	0.0155	1.86	0.0630
6	-0.1858	0.1182	-1.57	0.1160		6	0.0297	0.0171	1.73	0.0829
7	-0.1562	0.1344	-1.16	0.2451		7	0.0317	0.0196	1.62	0.1051
8	-0.1493	0.1616	-0.92	0.3553		8	0.0322	0.0223	1.45	0.1483
	E. $\text{mortality}_{dt} = \text{n_deaths}_{dt}$									
0	-0.1205	0.1273	-0.95	0.3441						
1	-0.0872	0.1374	-0.63	0.5258						
2	-0.0369	0.1576	-0.23	0.8146						
3	0.0077	0.1896	0.04	0.9678						
4	0.0758	0.2354	0.32	0.7474						
5	0.1624	0.2839	0.57	0.5672						
6	0.2239	0.3201	0.70	0.4844						
7	0.3033	0.3632	0.83	0.4038						
8	0.2350	0.4027	0.58	0.5595						

Each estimate is from a separate model.

Estimates in bold are statistically significant (p-value < .05).

Appendix Table 5

MORTALITY BY EDUCATIONAL ATTAINMENT

Estimates of β_k from model $\ln(\text{mortality}_{dt}) = \beta_k \text{ vint_r\&n}_{d,t-k} + \alpha_d + \delta_t + \varepsilon_{dt}$

k (lag)	Estimate	Std. Err.	Z	Pr > |Z|		k (lag)	Estimate	Std. Err.	Z	Pr > |Z|
	0-11 years of education									
	A. mortality$_{dt}$ = yll75$_{dt}$						**D. mortality$_{dt}$ = age_death$_{dt}$**			
0	**-0.0159**	**0.0056**	**-2.83**	**0.0046**		0	0.0012	0.0009	1.36	0.1724
1	**-0.0165**	**0.0058**	**-2.83**	**0.0047**		1	0.0013	0.0009	1.37	0.1698
2	**-0.0171**	**0.0060**	**-2.83**	**0.0046**		2	0.0014	0.0010	1.46	0.1436
3	**-0.0175**	**0.0062**	**-2.83**	**0.0046**		3	0.0016	0.0010	1.61	0.1081
4	**-0.0172**	**0.0063**	**-2.72**	**0.0066**		4	0.0019	0.0011	1.76	0.0782
5	**-0.0168**	**0.0067**	**-2.53**	**0.0113**		5	0.0023	0.0012	1.95	0.0509
6	**-0.0152**	**0.0072**	**-2.11**	**0.0346**		6	0.0026	0.0013	1.95	0.0513
7	-0.0128	0.0082	-1.57	0.1164		7	**0.0030**	**0.0015**	**2.00**	**0.0454**
8	-0.0138	0.0103	-1.34	0.1808		8	**0.0041**	**0.0017**	**2.33**	**0.0198**
	12 years of education									
	B. mortality$_{dt}$ = yll75$_{dt}$						**E. mortality$_{dt}$ = age_death$_{dt}$**			
0	**-0.0229**	**0.0069**	**-3.29**	**0.0010**		0	**0.0016**	**0.0008**	**2.08**	**0.0372**
1	**-0.0238**	**0.0072**	**-3.31**	**0.0009**		1	**0.0017**	**0.0008**	**2.15**	**0.0318**
2	**-0.0243**	**0.0074**	**-3.28**	**0.0010**		2	**0.0018**	**0.0008**	**2.24**	**0.0252**
3	**-0.0245**	**0.0075**	**-3.28**	**0.0010**		3	**0.0019**	**0.0008**	**2.34**	**0.0190**
4	**-0.0240**	**0.0077**	**-3.12**	**0.0018**		4	**0.0020**	**0.0009**	**2.38**	**0.0175**
5	**-0.0229**	**0.0080**	**-2.88**	**0.0040**		5	**0.0022**	**0.0009**	**2.39**	**0.0170**
6	**-0.0208**	**0.0084**	**-2.47**	**0.0134**		6	**0.0024**	**0.0011**	**2.20**	**0.0281**
7	-0.0185	0.0096	-1.93	0.0534		7	**0.0027**	**0.0013**	**2.15**	**0.0318**
8	-0.0189	0.0123	-1.54	0.1241		8	**0.0035**	**0.0016**	**2.22**	**0.0262**
	13+ years of education									
	C. mortality$_{dt}$ = yll75$_{dt}$						**F. mortality$_{dt}$ = age_death$_{dt}$**			
0	**-0.0147**	**0.0056**	**-2.62**	**0.0087**		0	**0.0020**	**0.0007**	**2.85**	**0.0044**
1	**-0.0153**	**0.0058**	**-2.63**	**0.0086**		1	**0.0021**	**0.0007**	**2.94**	**0.0033**
2	**-0.0158**	**0.0061**	**-2.60**	**0.0094**		2	**0.0022**	**0.0007**	**3.05**	**0.0023**
3	**-0.0160**	**0.0062**	**-2.58**	**0.0099**		3	**0.0023**	**0.0007**	**3.17**	**0.0015**
4	**-0.0157**	**0.0065**	**-2.41**	**0.0160**		4	**0.0024**	**0.0008**	**3.15**	**0.0016**
5	**-0.0147**	**0.0070**	**-2.11**	**0.0352**		5	**0.0025**	**0.0008**	**3.02**	**0.0025**
6	-0.0131	0.0077	-1.69	0.0908		6	**0.0026**	**0.0009**	**2.80**	**0.0052**
7	-0.0111	0.0091	-1.22	0.2223		7	**0.0030**	**0.0011**	**2.64**	**0.0082**
8	-0.0115	0.0126	-0.91	0.3612		8	**0.0038**	**0.0016**	**2.39**	**0.0170**

Each estimate is from a separate model.

Estimates in bold are statistically significant (p-value < .05).

Appendix Table 6

MORTALITY OF BLACKS

Estimates of β_k from model $\ln(\text{mortality}_{dt}) = \beta_k \text{ vint_r\&n}_{d,t-k} + \alpha_d + \delta_t + \varepsilon_{dt}$

k (lag)	Estimate	Std. Err.	Z	Pr > |Z|		k (lag)	Estimate	Std. Err.	Z	Pr > |Z|
	A. $\text{mortality}_{dt} = \text{yll85}_{dt}$						**B. $\text{mortality}_{dt} = \text{yll75}_{dt}$**			
0	-0.0048	0.0059	-0.82	0.4134		0	-0.0060	0.0063	-0.95	0.3447
1	-0.0049	0.0059	-0.82	0.4137		1	-0.0061	0.0063	-0.97	0.3330
2	-0.0045	0.0061	-0.73	0.4657		2	-0.0061	0.0065	-0.94	0.3479
3	-0.0040	0.0062	-0.65	0.5173		3	-0.0061	0.0066	-0.92	0.3557
4	-0.0020	0.0062	-0.32	0.7497		4	-0.0045	0.0067	-0.66	0.5078
5	0.0009	0.0063	0.15	0.8826		5	-0.0022	0.0069	-0.32	0.7515
6	0.0046	0.0069	0.67	0.5016		6	0.0011	0.0077	0.14	0.8896
7	0.0088	0.0081	1.09	0.2778		7	0.0042	0.0091	0.46	0.6433
8	0.0112	0.0109	1.03	0.3047		8	0.0049	0.0122	0.40	0.6866
	C. $\text{mortality}_{dt} = \text{yll65}_{dt}$						**D. $\text{mortality}_{dt} = \text{age_death}_{dt}$**			
0	-0.0088	0.0070	-1.27	0.2041		0	0.0017	0.0011	1.54	0.1247
1	-0.0090	0.0068	-1.31	0.1886		1	0.0018	0.0011	1.60	0.1106
2	-0.0091	0.0071	-1.29	0.1959		2	0.0020	0.0012	1.71	0.0867
3	-0.0094	0.0072	-1.30	0.1934		3	0.0022	0.0012	1.86	0.0624
4	-0.0075	0.0074	-1.03	0.3052		4	0.0024	0.0012	1.95	0.0512
5	-0.0052	0.0076	-0.69	0.4878		5	**0.0027**	**0.0013**	**2.04**	**0.0410**
6	-0.0016	0.0086	-0.18	0.8551		6	**0.0029**	**0.0015**	**1.99**	**0.0469**
7	0.0014	0.0104	0.14	0.8919		7	**0.0033**	**0.0016**	**2.02**	**0.0432**
8	0.0023	0.0145	0.16	0.8711		8	**0.0044**	**0.0020**	**2.23**	**0.0260**
	E. $\text{mortality}_{dt} = \text{n_deaths}_{dt}$									
0	-0.0039	0.0088	-0.45	0.6540						
1	-0.0029	0.0087	-0.34	0.7369						
2	-0.0007	0.0086	-0.08	0.9365						
3	0.0018	0.0089	0.20	0.8415						
4	0.0064	0.0097	0.66	0.5075						
5	0.0126	0.0111	1.14	0.2549						
6	0.0193	0.0127	1.52	0.1295						
7	0.0290	0.0155	1.87	0.0622						
8	**0.0404**	**0.0202**	**2.01**	**0.0449**						

Each estimate is from a separate model.

Estimates in bold are statistically significant (p-value < .05).