Effects of Medicare coverage for the chronically ill on health insurance, utilization, and mortality: Evidence from coverage expansions affecting people with end-stage renal disease

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Abstract:

I study the effect of the 1973 expansions of Medicare coverage among individuals with end-stage renal disease (ESRD) on insurance coverage, health care utilization, and mortality. I find that the expansions increased insurance coverage by between 22 and 30 percentage points, in models that include trends in age, with the increase explained by Medicare coverage, and increased physician visits by 25–35 percent. These expansions also decreased mortality due to kidney disease in the under 65 population by between 0.5 and 1.0 deaths per 100,000. Lastly, I provide evidence for two mechanisms that affected mortality: an increase in access to and use of treatment, which may be due to changes in insurance coverage; and an increase in entry of dialysis clinics and transplant programs.

Keywords: insurance | mortality | kidney disease | health | health insurance

Article:

1. Introduction

The United States has typically expanded public insurance programs by providing coverage to distinct demographic groups. For example, the introduction of Medicare and Medicaid in 1966 provided insurance coverage to people who were 65 and older or had low income. However, several expansions of these programs have defined eligibility based in part on the presence of medical conditions (e.g. long-term disabled, people with end-stage renal disease, pregnant women, and women diagnosed with breast or cervical cancer). By selecting on ill-health, the effects of a disease-specific insurance expansion on insurance coverage, health care utilization, and health outcomes may differ considerably from the effects of more broad-based expansions.

Previous studies of the Medicare and Medicaid programs have demonstrated that Medicare may reduce mortality (Card et al., 2009, Chay et al., 2017), increase health care utilization (Card

et al., 2008), and improve financial risk protection (Barcellos and Jacobson, 2015, Engelhardt and Gruber, 2011), while the introduction of state Medicaid programs reduced infant mortality (Goodman-Bacon, 2017). More recent evidence from an expansion of Medicaid for pregnant women demonstrates improvements in infant health outcomes (Currie and Gruber, 1996) and a related expansion affecting children improved their health and increased health care utilization (Currie and Gruber, 1996). A recent randomized study of the Oregon Medicaid program (Finkelstein et al., 2012) also demonstrated greater health care utilization and better self-rated physical and mental health among people randomized to receive Medicaid coverage, although there was no statistically significant difference in mortality.

There is, to my knowledge, no empirical evidence of the effects of three other disease-specific insurance expansions that provided insurance coverage for women with breast or cervical cancer, the long-term disabled, and people with end-stage renal disease (ESRD). In this paper, I examine the effect of a 1973 Medicare expansion that provided coverage to two groups of people: long-term beneficiaries of the Social Security Disability Insurance (SSDI) program and people who are undergoing dialysis or have received a kidney transplant due to having end-stage renal disease. The focus of this paper is the effect of the expansion on people with kidney disease who became eligible for Medicare coverage through either the ESRD route, if they were not already receiving SSDI payments, or due to SSDI receipt. Consistent with prior practice of the Medicare program itself, I consider both sets of enrollees as being enrolled in the ESRD program.1

These expansions are attractive to study for several reasons. First, the introduction of the program was, for the most part, unanticipated so that there is unlikely to be any significant anticipatory effects (Ball, 1973). Second, people with kidney disease tend to be in extremely poor health, so insurance is likely to have unusually large effects on health. Third, because for most people treatment was unaffordable prior to the expansion and insurance typically did not cover treatment (Rettig, 2011, Congressional Research Service, 1971), these results provide some insight into the welfare consequences of moral hazard induced spending since the bulk of any increase in utilization can be attributed to ex-post moral hazard. The ESRD program is also worthy of study on the basis of the size of the program. In 2015 the United States spent over $30 billion to treat 500,000 Medicare beneficiaries with ESRD, which represents 1% of all Medicare beneficiaries and 5% of Medicare spending. In other words, the ESRD program is almost as large as the entire Medicaid program in the state of Texas, which is the third largest Medicaid program (by spending) in the country.

In order to identify the effect of the ESRD program, I estimate triple-difference models that compare outcomes for people over 65, who were always eligible for Medicare coverage, versus those under 65, before versus after the expansion took effect, with versus without ESRD. However, due to the expansion of Medicare coverage to the long-term disabled, the triple difference estimate is biased. Hence, I also estimate difference-in-differences models that condition on having ESRD, which yields unbiased estimates as long as there is no selection into treatment, i.e. as long as ex-ante moral hazard is small. These two estimators will yield similar results as long as either the effect of Medicare eligibility is small in the non-ESRD group or the share of people eligible for Medicare coverage in that group is small.

In this paper I document three main facts about the ESRD program. First, I demonstrate that the ESRD expansion significantly increased insurance coverage among people under 65 years of age with kidney disease. Close to the traditional Medicare eligibility threshold of 65, I find a 22.6–29.6 percentage point increase in the probability of any insurance coverage among people with kidney disease. I find somewhat larger increases in Medicare coverage (26.0 and 33.9

percentage points, respectively), indicating that some people would have had insurance coverage in the absence of the expansion.

Second, I find that the ESRD expansion increased physician visits by 25–35 percent for people with kidney disease below 65 years of age. The increase in physician visits is consistent with my results on health insurance coverage and implies that a 10 percent increase in the share of the population with insurance increases physician visits by about eight percent. Because of the wording of the survey question that I use to assess physician visits, it is also possible that the increase in physician visits represents an increase in visits to, among other things, dialysis clinics.

Third, I document a significant reduction in mortality due to kidney disease of between two and seven log points, depending on specification and definition of kidney disease. I am able to replicate this finding in cross-national comparisons as well that allow me to control for innovations in kidney disease treatment across countries. My results imply that the program averted between 174 and 325 deaths per year for whites between 45 and 64 years of age (my estimation sample). Assuming that the entire change in mortality arose among people who gained insurance coverage, then my mortality and insurance results imply that the probability of dying in the coming year of kidney disease fell by 0.2–0.5 percentage points.

I am also able to provide evidence in support of two mechanisms by which the ESRD expansion affected health. First, the state-specific effect of the ESRD expansion on kidney disease mortality was larger in states that had more treatment facilities per capita in 1971. One interpretation of this result is that the presence of treatment facilities reduced mortality by increasing access to treatment. This interpretation is also consistent with the increase in physician visits. Second, I document an increase in the number of dialysis clinics and transplant programs per capita from 1971 to 1975 in states that had a higher under 65 mortality rate due to kidney disease, which is consistent with a demand side shock encouraging entry of new treatment facilities.

My mortality estimates also allow me to extrapolate to changes in survival and imply that the expansion saved between 2000 and 14000 life years, based on the change in survival for 45 year olds. This range encompasses some values where, using a value of $100,000 per statistical life year, the cost of the program are outweighed by the survival benefits. However, these estimates ignore other costs that the program imposes on society (e.g. increased disability insurance payments) but also ignores the value of spillover effects on to people 65 and older.

The remainder of the paper proceeds as follows. Section 2 provides background information on end-stage renal disease, discusses the role that the federal government has played in the treatment of ESRD, and describes the 1973 Medicare expansions that I study. Sections 3 describes the data that I use for my analyses and the empirical approach that I take, while |Sections 4 present my main results from the Medicare expansion in 1973. Section 5 presents potential mechanisms behind my results. Section 6 discusses the welfare implications of my results. Section 7 concludes.

2. Background

End-stage renal disease (ESRD) is the end result of a progressive decline in kidney function due to chronic kidney disease. Leading causes of ESRD and chronic kidney disease include chronic kidney disease include diabetes, hypertension, glomerulonephritis, polycystic kidney disease, kidney stones, urinary tract infections, and various congenital defects (National Kidney Foundation, 2009).2 The loss of kidney function that characterizes ESRD leads to a rapid buildup

in toxins and disregulation of potassium and sodium levels in the blood that, left unchecked, rapidly leads to death.

Treatment for ESRD emphasizes restoring or augmenting the body's ability to filter out toxins and maintaining electrolyte levels either by transplanting a functioning kidney from either a living or cadaveric donor or by externally filtering blood using a dialysis machine. There were significant scientific advances affecting both forms of treatment in the late 1950s through the 1960s. The first successful kidney transplant was performed in 1956 with the subsequent decade leading to slow, but steady, improvements in transplantation (Congressional Research Service, 1971) so that by 1971 there were 1172 kidney transplants performed (Rettig, 1976). Throughout this period, kidney transplantation was a costly procedure with the Congressional Research Service (1971) estimated that kidney transplantation had a nominal one-time cost of $10,000 to $20,000 ($59,000 to $117,000 in 2015) and maintenance costs of $1,000 per year ($5,900 in 2015).

Chronic dialysis, which is what is necessary to treat ESRD, was not feasible until 1960. Furthermore, at its inception, dialysis was extremely costly leading to rationing at the first dialysis clinic in the United States (Alexander, 1962). In July of 1972, there were 5786 living dialysis patients in the United States (Rettig, 1976, p. 200) and the Congressional Research Service (1971) estimated that the annual cost of dialysis was $15,000 in 1971 (nominal dollars, $85,000 in 2015 using the CPI-U).

Despite the availability of treatment modalities in the late 1960s and early 1970s, the Congressional Research Service (1971) reported that most health insurance plans did not cover treatment for ESRD.

During the 1960s, the federal government took an active role in promoting the diffusion of treatments for ESRD as well as funding research and development of new treatments. In 1963, the Veteran's Administration began to open dialysis clinics in its hospitals across the country and, by 1971, there were 40 dialysis facilities and 15 transplant programs open at VA and military hospitals across the U.S. Beginning in 1964, the National Institutes of Health started programs to study transplant immunology, which was intended to increase the number of successful kidney transplants. In 1965, the Public Health Service started the Kidney Disease Control Program, which provided start-up grants to open a dozen dialysis centers (Rettig, 1991). The federal government, through the Bureau of the Budget, also began examining the fiscal implications of the growth in ESRD and the advent of new methods to treat ESRD, although these discussions ultimately did not appear to have affected federal policy (for further discussion see Rettig, 1991).

In 1972 Congress, for the first time, passed a law expanding eligibility for Medicare coverage, with the expansion taking effect on July 1, 1973. Congress did so by declaring that two groups were eligible for coverage people who: had been eligible for Social Security Disability Insurance (SSDI) benefits for more than 24 months; or have received three, or more, months of renal dialysis with coverage extending up to twelve months after a person received a kidney transplant.

Neither component of the expansion was truly universal since in both cases, only individuals who were eligible for insurance under the Social Security program became eligible. Collectively, these two programs increased Medicare enrollment by 1.7 million people, of whom 6,371 were eligible solely due to the ESRD in the first year of the program. By 1978, there were almost 44,000 Medicare beneficiaries with ESRD, of whom almost 35,000 were under 65 years of age, with per capita spending of almost $65,000 (in 2015 dollars).

The ESRD component of the expansion (which includes long-term disabled with ESRD), which was initially expected to enroll 35,000 people and cost $1 billion (nominal) per year, rapidly

ballooned in size, covering more than 50,000 people and costing over $1 billion per year in 1979 (Table 1). In 2013, the ESRD program covered almost half a million people at a cost of $30 billion, which represents approximately 1% of Medicare enrollees and 5% of Medicare spending.

Table 1. Enrollment, spending, and utilization in the ESRD program

Year	Enrollment		Kidney Deaths		Spending		Utilization	
	Total	Under 65	Under 65	65 and Over	Total	Per enrollee	Transplants	Dialysis
1971			5335	7534				
1974	15993		4633	8949	1050.3	65673		
1975	22674	12702[a]	4540	9491	1545.6	68164		
1976	28941	14721[a]	4532	10597	2086.9	72110		
1977	35889	16514[b]	4345	11008	2449.2	68243		
1978	43482	34828	4498	11973	2804.6	64500		
1979	52636	43031	3761	11966	3126.4	59397	4189	45565
1981	61930	47520	3761	13703	3723.7	60127	4898	58924
1986	93197	59570	3914	17851	6786.7	63646	8948	90886
1991	142510	83443	3395	17963	9704.2	56844	10037	144175
1996	255578		3433	20869	14141.8	55333	12219	215557

Source—Greenbook (various years), Annual Statistical Supplement to the Social Security Bulletin (various years), Multiple Cause of Death files, 1971–1996

[a] Enrollees eligible solely due to ESRD.

Notes—Enrollment based on enrollment in Medicare Part A, expenditures are for Medicare Parts A and B. Spending data have been inflated to 2015 using the CPI for urban workers. Utilization data are the number of transplants and number of enrollees dialyzed, respectively. Kidney deaths are based on chronic coding only, see Appendix Table A; the coding of kidney deaths changed between 1978 and 1979.

3. Data and empirical framework

3.1. Data

I use data from a variety of sources to measure insurance coverage, health care utilization, and mortality in my main analyses as well as data on potential mechanisms and confounding factors. In this subsection, I describe each of these data sources.

3.1.1. Insurance coverage and health care utilization

The National Health Interview Survey asked respondents about insurance coverage in even numbered years beginning in 1968, although the specific wording and universe for various questions has changed over time. In 1968 the NHIS inquired about health insurance generically and did not differentiate between public and private coverage and it was not until 1978 that the NHIS inquired about Medicare coverage for people under 65 years of age. In the 1974 and 1976 waves of the survey individuals with only Medicare coverage were instructed to respond that they were uninsured. As a result, I present results using data from 1968, 1970, 1972, 1978, and 1980 for most insurance outcomes (I include data on private insurance coverage in 1974 and 1976). I define an individual as having private insurance coverage based on whether or not an individual reported having private hospital coverage (as in Finkelstein, 2007) and define Medicare coverage in a comparable manner.

The NHIS also included questions on the number of doctor visits in the prior year beginning in 1969, which I use to measure health care utilization. Because the NHIS questions refer to treatment received over the prior year, I omit people 65 years of age from the utilization analysis and all data from July 1973 through June 1974, the 12 months following the implementation of the Medicare expansion.

I use the condition inventory and the list of conditions that caused the interviewee to miss days from work or access health care services to construct indicators for the presence of kidney disease. The coding is based on the codes for the broad definition, but incorporating the NHIS omissions, listed in Appendix Table A. In total, out of 371,181 people in the NHIS, I identified 1644 people between 45 and 84 years of age with kidney disease using the broad definition. Despite the small sample size, the ESRD expansion is likely to have led to large changes in insurance coverage, hence I remain sufficiently powered to identify effects of the ESRD expansion on insurance coverage. For the utilization analyses, it is possible that I will be underpowered to detect effects if the increase in physician visits from the expansion is small.

3.1.2. Mortality

I use the Multiple Cause of Death files from the National Center for Health Statistics' (NCHS) for the years from 1968 through 1978 (United States Department of Health and Human Services, 2007, 2007). These data provide the state and county of residence, race, gender, age, underlying cause of death and all other diagnoses listed on the death certificate for all deaths in the United States, except in 1972, when the NCHS was only able to process half of the submitted death certificates.3 Preliminary analyses of the distribution of deaths by age indicated significant excess mass at five-year intervals of age for non-white individuals, which was also reported in Honoré and Lleras-Muney (2006), so I omit non-whites from my mortality analyses. I also drop deaths to non-U.S. residents since they were not eligible for the ESRD program.

I code each death as being a kidney disease death, or not, based on either the underlying cause of death, which the World Health Organization defines as "the disease or injury that initiated the train of events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury," or using any of the diagnosis codes listed on the death certificate. For each source of cause of death diagnosis codes, I defined a death as due to kidney disease using three sets of diagnosis codes. First, I defined a "narrow" definition of kidney disease, which did not restrict to only chronic disease, but is generally based on the "renal failure" codes in the ICDA-8. Second, I created a "chronic" definition by restricting the narrow definition to deaths due to chronic causes. Lastly, I created a "broad" definition, which was based on the codes used by the Kidney Disease Program in tracking kidney disease mortality (Kidney Disease Program, 1971). Appendix Table A lists the ICDA-8 diagnosis codes for the three cause of death groupings that I use.

I combine the mortality data with population data from the SEER program and the U.S. Census Bureau in order to adjust for changes in the size of the population over time, which also affects the expected number of deaths due to kidney disease. Because these data do not break out population figures for individuals 85 and over, I restrict my analysis to deaths to individuals who are 84 or younger.

3.1.3. Mechanisms and confounders

In my discussion of mechanisms and potential confounders, below, I rely on data from three other datasets. I collected data on the geographic distribution of treatment facilities in 1971 from the publication "Kidney disease services, facilities, and programs in the United States" (Kidney Disease Program, 1971), which lists treatment facilities by state. Based on the name of the facility, I also classified these facilities into Veteran's Administration/Military vs. civilian categories since access to the former may be restricted. Data on treatment facilities in 1975 came from the 1977 Annual Statistical Supplement to the Social Security Bulletin, which lists the number of hospital transplant programs, hospital-based dialysis clinics, and free-standing dialysis clinics by state.

I collected data on the share of people in an age-gender-state cell who receive income from either Social Security or the Supplemental Security Income program from the March CPS supplements for 1977–1979 (spanning 1976–1978).

3.2. Empirical approach

3.2.1. Identification

My data includes three sources of variation that I could use to identify the effect of the Medicare ESRD program on insurance coverage, health care utilization, and kidney disease mortality. First, there are differences over time in Medicare eligibility for individuals of the same age and disease status. Second, there are differences by age in eligibility for Medicare for individuals in the same year and disease status. Third, there are differences by disease status in eligibility for Medicare coverage for individuals in the same year and of the same age. In principle, these three sources of variation would justify a triple difference estimator assuming that potential outcomes between these groups satisfy a "parallel trends" assumption (Lee and Kang, 2006). However, in my setting the parallel trends assumption is unlikely to hold because the SSDI expansion means that there is partial takeup of Medicare coverage in one of the comparison groups. The structure of the problem, allows me to identify the source of any bias from these comparisons and identify a solution that leads to unbiased estimates of the intent-to-treat effect of the Medicare expansion on people with kidney disease.

To demonstrate the bias and identify situations in which it does not affect my results, let Y^e_{akt} denote the potential outcome for someone in age group a (a = 1 for people under 65) who has kidney disease if k = 1, in time period t (t = 1 in the post period), and is either eligible (e = 1) or ineligible (e = 0) for Medicare coverage. Assume that there is a probability αakt that a person is eligible for Medicare in each akt cell and define $Y_{akt} = \alpha_{akt} Y^1_{akt} + (1 - \alpha_{akt})\, Y^0_{akt}$. Ignoring the fact that some people 65 and older are not eligible for Medicare, Medicare program rules imply that $\alpha_{0kt} = 1$ for all $k, t \in \{0, 1\}$ and $\alpha_{1k0} = 0$ for $k \in \{0, 1\}$. Finally, because (almost) everyone with kidney disease is automatically eligible for Medicare coverage, but only some people without kidney disease are eligible for Medicare coverage, we also have $\alpha_{111} > \alpha_{101}$.

Then the triple-difference estimator can be written as:

$$\begin{aligned}\text{DDD} &= \alpha_{111}\left(Y^1_{111} - Y^0_{111}\right) - \alpha_{101}\left(Y^1_{101} - Y^0_{101}\right) \\ &+ \left[\begin{array}{l}\left(Y^0_{111} - Y^0_{110}\right) - \left(Y^1_{011} - Y^1_{010}\right) \\ -\left(Y^0_{101} - Y^0_{100}\right) + \left(Y^1_{001} - Y^1_{000}\right)\end{array}\right]\end{aligned}$$

The "parallel trends" assumption can be stated as the assumption that the terms in the large brackets in the previous equality vanish and that $\alpha_{101}\left(Y^1_{101} - Y^0_{101}\right) = 0$. While it is plausible that the second and third terms vanish, the $\alpha_{101}\left(Y^1_{101} - Y^0_{101}\right)$, which reflects the effect of the expansions on the disabled without ESRD, is unlikely to vanish. Therefore DDD is biased by partial takeup of treatment ($\alpha 111 < 1$) and the fact that some people without kidney disease are also treated ($\alpha 101 > 0$). However, the bias can be signed if one assumes that the sign of the treatment effect is the same regardless of kidney disease status, in which case the triple-difference estimate will be biased towards zero unless the treatment effect of eligibility for people without kidney disease is significantly greater than the treatment for people with kidney disease.

In the difference-in-difference estimate that restricts to people with kidney disease, there is no bias from the fact that people without kidney disease are partially treated. One can write this estimator as:

$$\text{DD}_k = \alpha_{111}\left(Y^1_{111} - Y^0_{111}\right) + \left(Y^0_{111} - Y^0_{111}\right) - \left(Y^1_{011} - Y^1_{010}\right)$$

Assuming that the parallel trends assumption holds, then DDk provides a scaled estimate of the causal effect of Medicare eligibility for people with kidney disease. In the DDk estimator, the parallel trends assumption implies that in the absence of the ESRD expansion, trends in mortality would have progressed along similar paths following the expansion for people over and under 65 years of age. While there are reasons to doubt this assumption, due to the fact that renal replacement therapy was generally more suited to younger, rather than older, people, it is unclear why there would be a sudden change at age 65, as would be required to bias my estimates.

It is tempting to also consider difference-in-difference models that compare people with and without kidney disease who are under 65, but such a model would be subject to the same biases as the triple difference model since some people without kidney disease also became eligible for Medicare coverage following the 1973 expansions.

3.2.2. Event study and difference-in-difference models

I first consider analyses that use age and time variation separately. To do so, I estimate event-study models of the form:

$$\begin{aligned}y_{\text{iatgd}} &= \beta_1^{65}\,\text{Kidney}_d + \beta_2^{65}\,\text{Post}_t + \beta_3^{65}\,\text{Kidney}_d \times \text{Post}_t \\ &+ \textstyle\sum_{a' \neq 65}\left(\beta_1^{a'}\,\text{Kidney}_d + \beta_2^{a'}\,\text{Post}_t + \beta_3^{a'}\,\text{Kidney}_d \times \text{Post}_t\right)\mathbf{1}_{[a=a']} \\ &+ X_{\text{ig}}\Gamma_1 + \tau_t + \alpha_a + \varepsilon\end{aligned} \qquad (1a)$$

and:

$$
\begin{aligned}
y_{\text{iatgd}} &= \beta_1^{1971}\,\text{Kidney}_d + \beta_2^{1971}\,\text{Under 65}_a + \beta_3^{1971}\,\text{Kidney}_d \times \text{Under 65}_a \\
&+ \textstyle\sum_{t' \neq 1971} \left(\beta_1^{t'}\,\text{Kidney}_d + \beta_2^{t'}\,\text{Under 65}_a + \beta_3^{t'}\,\text{Kidney}_d \times \text{Under 65}_a\right)\mathbf{1}_{[t=t']} \\
&+ X_{\text{ig}}\Gamma_1 + \tau_t + \alpha_a + \varepsilon
\end{aligned}
\tag{2a}
$$

Where y_{iatgd} is the outcome—type of insurance coverage, amount of health care utilization, or deaths per 100,000 people—for person i (I only have person-level data on insurance coverage), who belongs to age group a in time period t, where time is measured in half-year increments (although there is some abuse of notation), gender g, and cause of death d, Kidney_d is a dummy for deaths due to kidney disease, Post_t is a dummy for the ESRD period, which takes the value of 1 for time periods after July 1, 1973, Under65_a is an indicator that a is less than 65, X_{ig} is a vector of controls including fixed effects for each demographic group, τ_t and σ_a are year and age fixed effects, respectively. The coefficients $ß^a{}_i$ and $ß^t{}_i$ are the age or year-specific coefficients on kidney disease, the post period (or being under 65), and their interaction.

I then summarize the results of these event studies using a triple-difference estimator, which is subject to bias from the SSDI program, and a difference-in-differences estimator that is unbiased, but may also be less precise. The triple-difference model can be written as:

$$
\begin{aligned}
y_{\text{iatgd}} &= \beta_1\,\text{Kidney}_d + \beta_2\,\text{Post}_t + \beta_3\,\text{Under 65}_a + \beta_4\,\text{Kidney}_d \times \text{Post}_t \\
&+ \beta_5\,\text{Kidney}_d \times \text{Under 65}_a + \beta_6\,\text{Post}_t \times \text{Under 65}_a \\
&+ \beta_7\,\text{Kidney}_d \times \text{Post}_t \times \text{Under 65}_a + X_{\text{ig}}\Gamma_1 + \tau_t + \alpha_a + \varepsilon
\end{aligned}
\tag{3}
$$

And the corresponding difference-in-differences estimator is:

$$
\begin{aligned}
y_{\text{iatgd}} &= \alpha_1\,\text{Post}_t + \alpha_2\,\text{Under 65}_a + \alpha_3\,\text{Post}_t \times \text{Under 65}_a + X_{\text{ig}}\Gamma_1 + \tau_t + \alpha_a \\
&+ \varepsilon
\end{aligned}
\tag{4}
$$

The previous discussion of identification in this setting implies that $|\alpha 3| \geq |\beta 7|$, assuming that treatment effects in the ESRD expansion are comparable in size, or larger, than treatment effects of the SSDI expansion.

Standard errors for all models are clustered on age and time, unless otherwise specified, using clus_nway.ado (Kleinbaum et al., 2013, Cameron et al., 2011). I cluster on age to be consistent with the recommendations in Lee and Lemieux (2010) and Lee and Card (2008). I cluster on time based on recent results in Hausman and Rapson (2017).

4. Effect of the Medicare expansion

4.1. Health insurance

I first consider the effect of the Medicare expansion on health insurance coverage among people with kidney disease. Fig. 1 presents an event study of the change in any insurance coverage (panel A) and Medicare coverage (panel B) using the triple-difference version of equation 1b.4 Prior to the Medicare expansion, the probability that an individual with kidney disease had any form of insurance coverage was increasing from 1968 to 1970, but stable from 1970 to 1972. However following the expansion there was no appreciable increase in insurance coverage, on average, for people with kidney disease. Medicare coverage, by contrast, increased significantly by 1978 with the bulk of the increase in Medicare coverage happening at older ages (panel D). Conversely, the ESRD expansion appears to have increased coverage somewhat for people close to the age 65 cutoff, but there was also a noticeable increase in insurance coverage for people around 40 years of age (panel C).

The results in Fig. 1 provides some support for the "parallel trends" assumption underlying differences-in-differences estimators. The fact that there was an increase in the point estimates from 1968 to 1970 for the probability of having any insurance is concerning, but this trend does not continue into 1972. I find no indication of a time trend in Medicare coverage. By age, panels C and D demonstrate that insurance coverage for people over 65 was not appreciably affected by the Medicare expansion.

Consistent with the event study in Fig. 1, I find no evidence that the ESRD expansion increased insurance coverage among people with kidney disease (Table 2, column 1). However, there was a 23–30 percentage point increase in coverage for people close to the age 65 cutoff (column 2). The increase in insurance coverage in models with age trends is slightly smaller than the increase in Medicare coverage (columns 3 and 4), which is consistent with either a degree of crowd-out or "doubling-up" of private and public insurance coverage. I find only modest evidence of a decrease in private insurance coverage associated with the ESRD expansion, but a large decrease in the share of people who reported only private insurance coverage. This final change–the reduction in reports of only private insurance–is indicative of people using both Medicare and private insurance coverage simultaneously. This kind of doubling up of insurance coverage provided additional benefits to people with ESRD since private insurance plans at the time typically did not cover dialysis or renal transplantation, hence adding Medicare coverage represented a significant improvement in insurance coverage for people with kidney disease.

Comparing the DDD and DD coefficients provides support for the fact that the Medicare expansion's effect on people without kidney disease appears to bias my DDD estimates towards zero for having any insurance coverage and for Medicare coverage. This bias is what one would expect if the Medicare expansion also affected insurance coverage for some people without kidney disease, in this case by providing coverage to the long-term disabled without kidney disease.

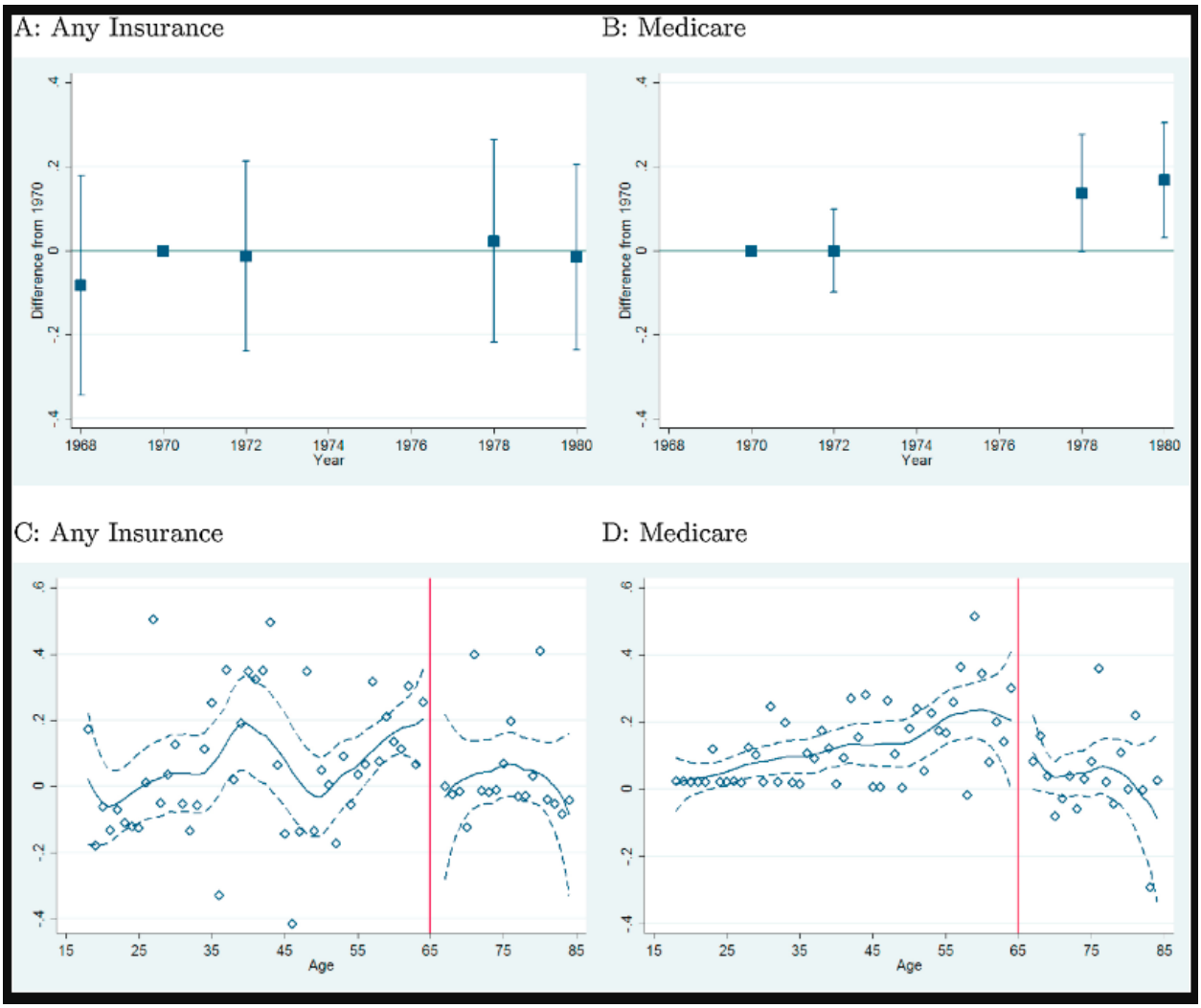

Fig. 1. Changes in insurance coverage from the Medicare expansion. Source—National Health Interview Survey, even number years from 1968 to 1980, excluding 1974 and 1976. Notes—Sample in panels A and B restricted to people between 45 and 84 years of age; panels C and D use everyone between 18 and 84 years of age. Points in panels A and B are year-by-under 65 years of age-by-kidney disease coefficients from a regression of insurance status on year fixed effects (omitted 1970), an under 65 indicator, an indicator for kidney disease, and all two- and three-way interactions. Panels C and D present point estimates for years of age interacted with a post dummy (after 1973) and kidney disease from regressions of insurance status on age fixed effects, post, kidney disease, and all two- and three-way interactions; smoothed line is local polynomial estimate where estimates are weighted by the inverse of their standard errors and dashed lines are 95% confidence intervals of the local polynomial estimate. Confidence intervals in panels A and B based on covariance matrix that is clustered on age, while panels C and D use heteroskedasticity robust standard errors. Kidney disease defined using the "broad" definition (see appendix Table A).

Table 2. Effect of the ESRD program on health insurance and health care utilization.

	Any insurance		Medicare		Any private		Only private		Doctor visit	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
DDD	0.021	0.226*	0.144*	0.260+	−0.084	0.056	−0.243*	−0.205*	0.181**	0.266+
	(0.068)	(0.082)	(0.052)	(0.126)	(0.081)	(0.108)	(0.081)	(0.071)	(0.053)	(0.159)
Agg. effect[a]	5409	57732	36866	66329	−19614	12893	−62070	−52429	411298	632898
Avg. effect[b]									2.28	3.50
N	147669	147669	118375	118375	188071	188071	118375	118375	371181	371181
DD	0.073	0.296**	0.193**	0.339*	−0.164*	−0.006	−0.238**	−0.210*	0.255**	0.353*
(0.069)	(0.092)	(0.056)	(0.131)	(0.076)	(0.123)	(0.069)	(0.088)	(0.069)	(0.163)	(0.069)
Agg. effect[a]	18582	75690	49223	86510	−38148	−1487	−60815	−53623	645823	939183
Avg. effect[b]									3.23	4.70
N	890	890	695	695	1084	1084	695	695	2055	2055
Age trends	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Means										
65+, Without kidney disease										
Pre	0.96	0.93	0.55	0.04	5.05					
Post	0.98	0.93	0.66	0.04	4.89					
65+, With kidney disease										
Pre	0.92	0.91	0.43	0.04	10.35					
Post	0.97	0.93	0.47	0.02	9.43					
<64, Without kidney disease										
Pre	0.82	0.00	0.82	0.82	3.87					
Post	0.89	0.04	0.84	0.81	3.89					
<64, With kidney disease										
Pre	0.72	0.00	0.76	0.76	9.95					
Post	0.85	0.22	0.64	0.50	11.28					

[a]Aggregate effect of the Medicare expansion on insurance coverage and annual number of physician visits for people between 45 and 64 years of age with kidney disease in the post period.

[b] Average individual effect of the Medicare expansion on the number of physician visits for people between 45 and 64 years of age with kidney disease in the post period. Source—Author's analysis of the National Health Interview Survey from 1968 to 1980. Notes—Dependent variable is indicated by the column group title. Kidney disease is defined using the "Broad" definition of kidney disease (see Appendix Table A). DDD is the triple-difference coefficient from the interaction of a dummy for being under 65 years of age, a dummy for the second half of 1973 or later, and a dummy for having kidney disease; DD is the difference-in-difference coefficient from a sample with kidney disease. Models include year, age, gender, and race fixed effects along with all one-, two-, and, if appropriate, three-way interactions of under 65, post, and kidney disease; models with age trends also include additional interactions with age-65. Sample restricted to individuals between 45 and 84 years of age; columns (9) and (10) also exclude people 65 years of age and data from July 1 1973 to June 30 1974. Estimates are from OLS regressions in columns (1)–(8) and Poisson in columns (9) and (10). Standard errors clustered on age in round brackets. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

The row labeled "Agg. Effect" presents the average aggregate effect of the Medicare expansion on insurance coverage using the triple difference or difference-in-difference coefficient, as appropriate. The average aggregate effect is the average of the annual total of the sampling weight for people between 45 and 64 years of age with kidney disease multiplied by the DDD or DD coefficient and indicates how many people with kidney disease gained or lost insurance coverage as a result of the Medicare expansion. These results indicate that as few as 5400 people or as many as 58000 people with kidney disease gained insurance coverage as a result of the expansion, although only the higher estimate is based on a statistically significant coefficient. Using data on Medicare coverage, I find a large increase in coverage of between 37000 and 66000 people having Medicare coverage. My estimates for Medicare coverage are large and are, in fact, larger than what Medicare trustees reported for the total number of people with ESRD, whether they became eligible solely due to having ESRD or because they were disabled. The fact that my implied increase in Medicare coverage is larger than the estimate from Medicare trustees should not be surprising since, in order to have sufficient data, I am applying a far more relaxed definition of kidney disease than is used by Medicare itself. The aggregate effect estimated using the DD coefficient is consistently larger than the estimate from the DDD estimate, which is what I had hypothesized based on the fact that some people without kidney disease were also gaining access to Medicare coverage.

As a specification check, online appendix Table B1 presents results from "donut" regressions that exclude people within five years of turning 65. These donut estimates are, in general, consistent with my main specifications, particularly for models without age trends.

Consistent with the event studies, column 9 of Table 2 demonstrates that the ESRD expansion was associated with a 18–25 percent increase in physician visits in models that do not control for age trends. Models that do control for age trends (column 10) yield larger estimates, which is consistent with the age profile of the change in physician visits from panel B of Fig. 1. In aggregate, my DDD estimates indicate that there were an additional 400000–600000 physician visits per year among people with kidney disease, or an additional 2.3–3.5 visits per person with kidney disease ("Avg. Effect" row).5

As was the case with my insurance estimates, my difference-in-difference results are generally comparable, although larger, than my triple-difference estimates and this extends to the aggregate and average effects of the expansion on doctor visits.

These results are essentially unchanged in donut regressions (online appendix Table B1, columns 9 and 10).

4.2. Health care utilization

Fig. 2 presents triple difference estimates for the number of physician visits. The estimates in panel A are extremely noisy, both in terms of the standard error, but also in the point estimate itself, with relatively large amount of variation in the point estimate both before and after the expansion took effect. However, visually it appears that there may have been an increase in physician visits after, versus before, the Medicare expansion for people with kidney disease. Panel B demonstrates that any increase in physician visits affected virtually all ages below 65 years of age and there was essentially no effect on physician visits for people over 65 years of age.

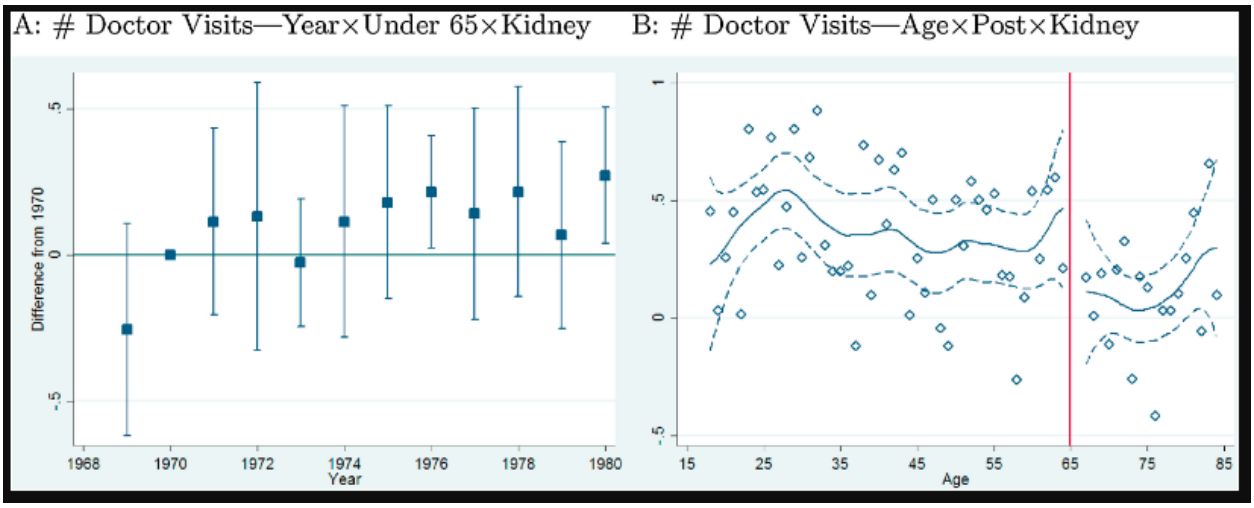

Fig. 2. Event study estimates of changes in health care utilization. Source—National Health Interview Survey, 1969–1980. Notes—Sample in panel A restricted to people between 45 and 84 years of age; panel B uses everyone between 18 and 84 years of age, but excludes observations from the second half of 1973 and the first half of 1974. Points in panel A are year-by-under 65 years of age-by-kidney disease coefficients from a regression of doctor visits on year fixed effects (omitted 1970), an under 65 indicator, an indicator for kidney disease, and all two- and three-way interactions. Panel B presents point estimates for years of age interacted with a post dummy (after 1973) and kidney disease from regressions of doctor visits on age fixed effects, post, kidney disease, and all two- and three-way interactions; smoothed line is local polynomial estimate where estimates are weighted by the inverse of their standard errors and dashed lines are 95% confidence intervals of the local polynomial estimate. Estimates are from Poisson regressions. Confidence intervals in panel A based on covariance matrix that is clustered on age, while panel B uses heteroskedasticity robust standard errors. Kidney disease defined using the "broad" definition (see appendix Table A).

4.3. Mortality effects

4.3.1. Comparisons within the United States

Fig. 3 plots event studies for kidney disease mortality using the underlying cause of death, where the event studies are based on triple-difference estimates. Panel A indicates that there was a reduction in mortality due to kidney disease in 1973 and visually, this reduction was larger than the potential downward trend in kidney disease mortality prior to 1973. Panel B demonstrates that there was a strong age trend in the mortality change following the ESRD expansion, which justifies focusing on specifications that include age trends.

In triple difference models based on equation (3) and difference-in-differences estimates based on equation (4) I find that the ESRD program reduced mortality from kidney disease by 36.3–37.3 log points in a model that does not include age trends. This estimate is, at first blush, implausibly large and reflects the age trends seen in panel B of Fig. 3. Including age trends (column 2) yields smaller estimates of a 7.3–7.9 log point reduction in mortality. In models with age trends, I also find that the DD estimate is larger in magnitude (although not significantly so) than the DDD estimate, which is what one would expect from the lower level of Medicare eligibility among people without kidney disease.

Because there are many potential diagnoses that may indicate a death due to kidney disease, in columns (5) and (6) I present results using the "broad" definition of kidney disease. The broad

results are qualitatively similar and also indicate a reduction in mortality based on kidney disease as the underlying cause of death, but not when kidney disease is defined using both underlying and contributing cause of death codes.

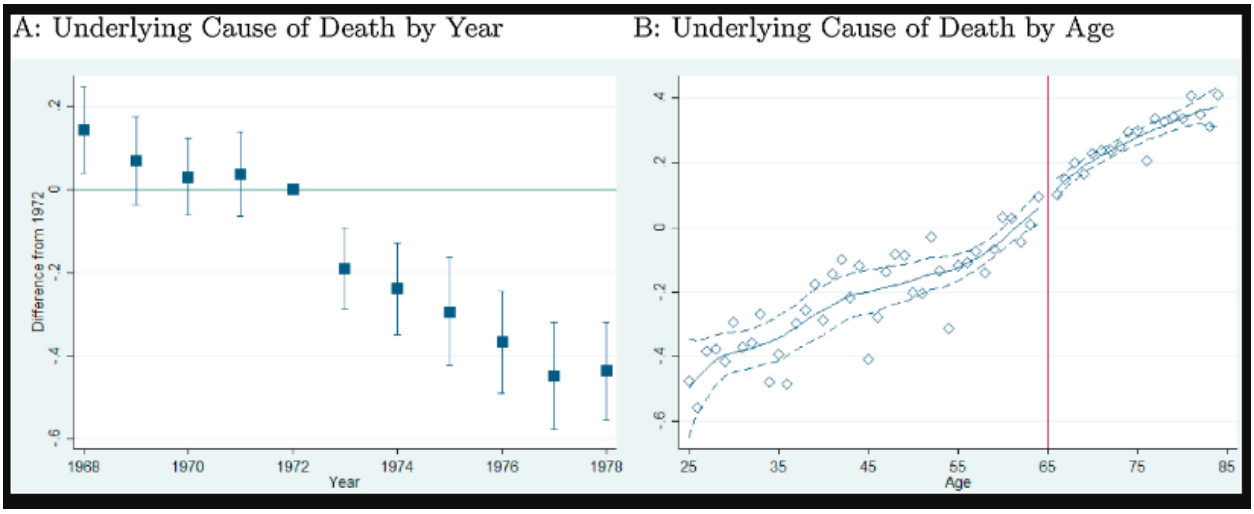

Fig. 3. Event study estimates of the ESRD program and mortality. Source—Author's analysis of multiple cause of death files, 1968–1978. Notes—Sample in panel A restricted to deaths to people between 45 and 84 years of age; panel B uses everyone between 25 and 84 years of age. Points in panel A are year-by-under 65 years of age-by-kidney disease coefficients from a regression of the cause-age-gender-time period mortality rate on on year fixed effects (omitted 1972), an under 65 indicator, an indicator for kidney disease, and all two- and three-way interactions. Panel B presents point estimates for years of age interacted with a post dummy (after 1973) and kidney disease from regressions of the cause-age-gender-time period mortality rate on age fixed effects, post, kidney disease, and all two- and three-way interactions; smoothed line is local polynomial estimate where estimates are weighted by the inverse of their standard errors and dashed lines are 95% confidence intervals of the local polynomial estimate. Estimates are from Poisson regressions. Confidence intervals in panel A based on covariance matrix that is clustered on age, while panel B uses heteroskedasticity robust standard errors. Kidney disease defined using the "narrow" definition (see Table A). Cause-age-gender-time period cells weighted by population.

Lastly, the ESRD program, in particular, was targeted at people with chronic kidney disease, so in columns (7) and (8) I restrict my definition of kidney disease to people who died of chronic kidney disease, based on the codings in appendix Table A. The chronic estimates indicate that the Medicare expansion was associated with a reduction in deaths due to chronic kidney disease, although three of the estimates are only significant at the ten percent level.

Table 3 also presents the change in the mortality rate and the implied number of deaths averted, based on population data from 1973 and the average mortality rate due to kidney disease before the Medicare expansion. Under the narrow definition and without age trends, the Medicare expansion appears to have reduced the mortality rate by 2.0–2.3 deaths per 100,000 for a total of between 800 and 900 fewer deaths per year due kidney disease. However, these estimates are significantly narrowed in models that include age trends (columns 2 and 4) to a reduction of 0.5–0.8 deaths per 100,000 or 170–320 fewer deaths. The fact that there is a greater reduction in the number of deaths using both underlying and contributing causes of death, versus just underlying causes of death, indicates that at least part of the reduction in mortality that I observed in column (2) is not due to a change in coding practices in which kidney disease was less likely to be listed as an underlying cause of death, but more likely to be listed as a contributing cause of death.

Table 3. Poisson estimates of the effect of the ESRD program on mortality.

	Narrow definition				Broad definition			
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**
A: Ages 45-84								
DDD	−0.373**	−0.073*	−0.059**	−0.021*	−0.067**	−0.010	−0.057*	−0.020+
	(0.049)	(0.029)	(0.007)	(0.010)	(0.011)	(0.009)	(0.027)	(0.012)
Δ in mortality rate	-2.0	-0.5	-2.3	-0.8	-0.8	-196	-114	-270
Δ in # deaths	-772	-174	-891	-323	-311	-196	-114	-270
DD	-0.363**	-0.079*	-0.048**	-0.025+	-0.073**	-0.015	-0.064+	-0.024+
	(0.053)	(0.035)	(0.014)	(0.014)	(0.022)	(0.011)	(0.034)	(0.014)
Δ in mortality rate	-2.0	-0.5	-1.9	-1.0	-0.9	-0.7	-0.3	-0.9
Δ in # deaths	-755	-189	-723	-388	-337	-288	-128	-331
B: Age 45-60 and 70-84								
DDD	-0.446**	-0.148**	-0.080**	-0.066**	-0.144**	-0.040**	-0.134*	-0.062**
	(0.052)	(0.049)	(0.006)	(0.017)	(0.021)	(0.011)	(0.053)	(0.021)
DD	-0.437**	-0.150*	-0.068**	-0.065*	-0.145**	-0.040+	-0.136*	-0.062*
	(0.056)	(0.066)	(0.015)	(0.026)	(0.043)	(0.021)	(0.069)	(0.026)
Age trends	No	Yes	No	Yes	Yes	Yes	Yes	Yes
Underlying only?	Yes	Yes	No	No	Yes	No	Yes	No
Meanl annual kidney disease mortality rates (per 100,000)								
Ages 45-64								
1968-1973H1	6.6	6.6	40.0	40.0	13.8	52.0	5.6	36.5
1973H2-1978	5.4	5.4	37.3	37.3	8.9	45.3	4.6	32.6
Ages 65-84								
1968*1973H1	28.0	28.0	251.0	251.0	100.4	368.1	24.4	238.2
1973H2-1978	32.4	32.4	241.1	241.1	76.7	323.2	27.4	218.6

Source—Author's analysis of Multiple Cause Mortality Files for 1968–1978.

Notes—Dependent variable is the mortality rate in the age-time-gender-cause of death cells, where time is measured in half-year increments. Definitions of kidney disease based on codes in Table A. DDD is the triple difference coefficient for being under 65, in the post expansion period, with kidney disease; DD is the difference-in-differences coefficient for being under 65 and in the post expansion period using a sample that is restricted to deaths due to kidney disease.

Change in mortality rate is calculated as the exponentiated coefficient minus 1 multiplied by the pre-period mortality rate; change in number of deaths is the change in the mortality rate multiplied by the population between 45 and 64 years of age in 1973. Models that do not restrict to underlying causes of deaths also define a death as due to kidney disease if kidney disease is either an underlying or a contributing cause of death. All models include time fixed effects (measured in six month increments), age fixed effects, an indicator for female, and all possible interactions of an indicator for being under 65, a post period dummy, and, where appropriate, an indicator for deaths due to kidney disease. Models with age trends also include interactions with age minus 65 in addition to the under 65, post, and kidney disease interactions. Sample is restricted to deaths to whites between 45 and 84 years of age in panels A; panel B excludes deaths to people between 61 and 69 years of age. Estimates are from Poisson regressions, standard errors two-way clustered on age and time in round brackets.

Repeating the change in mortality analysis for both the broad and chronic definitions yields two interesting results. First, the vast majority of the reduction in kidney disease deaths is arising from fewer deaths due to chronic disease, particularly when using both underlying and contributing causes. Second, while there is a larger reduction in mortality under the broad definition when I only look at underlying cause of death codes, I actually find that there were fewer deaths averted under the broad definition than the narrow definition when I use both underlying and contributing causes of death.

The fact that there are fewer deaths averted using underlying and contributing cause of death codes versus just the underlying cause of death codes under the broad definition is unexpected. One might expect that under a more relaxed definition of kidney disease mortality the opposite would occur. The break in the pattern between underlying and underlying and contributing mortality does not reflect a change in coding practices since there were no relevant changes in the coding manuals published by the National Center for Health Statistics during this time period. However, what appears to be happening is that there is essentially no change in the number of deaths that list one of the more tenuously related cause of death codes as a contributing cause of death. Given the set of broad cause of death codes, it is not surprising that there is a smaller change in the number of deaths that list, for example, hypertension as a contributing cause of death.

The mortality reductions in panel A of Table 3 can be combined with the estimated change in insurance coverage from Table 2 to infer how large an effect insurance coverage may have on kidney disease mortality. Using either the increase in insurance coverage from column (2) or the increase in Medicare coverage from column (4) implies that there was a reduction of 0.4 percentage points (any insurance) or 0.33–0.35 percentage points (using the increase in Medicare coverage) in the probability of dying from kidney disease associated with insurance coverage. These estimates are about three times larger than the local average treatment effect of Medicaid coverage estimated by the Oregon Health Insurance Study (column (3) of Table IX in Finkelstein et al., 2012).

Alternatively, one can compute the elasticity of mortality with respect to insurance coverage. From Table 2, the percentage change in insurance coverage is 0.31 based on the DDD estimate for any insurance coverage and 0.41 using the DD estimate. Using the point estimates in Table 3 yields an elasticity of mortality using the narrow, underlying definition of −0.23 using the DDD and −0.19 using the DD estimate, which is twice as large as the elasticity from the Oregon Health insurance Study.6

There are two main threats to the validity of my results that are unique to mortality data. First, there is a reverse “harvesting” effect, in which people who would have died of kidney disease in the absence of the program are able to survive until they turn 65 after the program. The implication of this kind of harvesting is that the mortality rate among people 65 and older will be overstated. I am able to test for this possibility by re-running my underlying models while excluding people between 60 and 70 years of age (panel B). In these donut regressions, my results are essentially unchanged and, in fact, my estimated mortality reductions become larger. This is inconsistent with reverse harvesting, which would predict that the mortality reductions would be smaller in magnitude when I exclude people between 60 and 70 years of age.

The second threat is that people who do not die of kidney disease will die of something else. This “competing risk” effect is well known in economics and epidemiology and cannot be resolved without imposing assumptions on the processes that determine mortality (Honoré and Lleras-Muney, 2006). The bias due to competing risks is similar to the bias from harvesting, but

now it is the mortality rate due to non-kidney causes that is inflated. Notably, competing risks can only bias my estimates if there is, in fact, an effect of the Medicare expansion on kidney disease mortality. In the absence of such a reduction, there is no reason to expect to find a competing risk bias. I can address the bias from competing risks by restricting my data to deaths due to kidney disease, in other words the DD results are not subject to competing risks. My DD results demonstrate that any bias from competing risks is small since my DD estimates are, in general, larger in magnitude than the DDD estimates (which is also the relationship one would expect to hold if the treatment effect of Medicare eligibility was of the same sign for people with and without kidney disease).

The online appendix presents results from a log-linear OLS specification, which are qualitatively similar (Table B2). The online appendix also provides robustness tests of the triple and double-difference results by varying the range of ages included (online appendix Fig. A1) and varying the age and time controls that are included (Table B3).

In the online appendix (Table B4), I also consider the potential confounding effect of the introduction of the Supplemental Security Income (SSI) program in 1974. The SSI program provides cash transfers to low-income people who are aged, blind, or disabled and, in most states, also provides access to Medicaid coverage. To test if the SSI program is confounding my estimate of the effect of the SSDI program, I interacted the triple difference coefficients with (demeaned) shares of people in an age-gender-state cell who reported either Social Security or SSI income in the March CPS from 1977 to 1979 (covering years 1976 to 1978). In a separate specification, I interacted the triple difference coefficients with indicators for two factors that states can use to discourage enrollment in Medicaid–using more stringent eligibility criteria and requiring a separate Medicaid application. I find no evidence that these interactions are statistically significant using either the narrow or chronic definitions of kidney disease, indicating that the SSI program is not driving the differential mortality reduction for kidney disease, relative to other causes of death. However, I do find that more stringent Medicare criteria and separate Medicare applications are associated with increase mortality under the broad definition of kidney disease, indicating that there was an increase in mortality from these other cause of death codes.

4.3.2. Comparisons with other OECD countries

Fig. 4 plots event-study estimates of the change in kidney disease mortality in the United States, relative to other OECD countries by either year (panel A) or age (panel B). Over time, there is a pronounced reduction in kidney disease mortality for people under 65 in the United States in 1973 that was not observed in other countries. However, there is also some evidence of a trend in kidney disease mortality in the United States towards fewer people under 65 dying from kidney disease, although with one exception, all of the confidence intervals before 1972 include 0. Despite the possible violation of the parallel trends assumption, there is still evidence of a substantial reduction in kidney disease mortality beginning in 1973. Results by age (panel B) are also suggestive of a reduction in kidney disease mortality, although there appears to be a reduction in mortality among 65–70 year olds, relative to people 70 and older, in the data as well.

Going from the event-study estimates in Fig. 4 to triple and quadruple difference results, I find that the ESRD program was associated with a four to eight log point reduction in mortality from kidney disease, depending upon the specification and sample (Table 4). This reduction in mortality is robust to including country fixed effects, interacting country fixed effects with either kidney disease or an indicator for 1974 or later (the post dummy takes the value 0.5 in 1973), and

including year-by-kidney disease indicators, which accounts for innovations in the treatment of kidney disease. These results are also similar in magnitude to my results using the narrow definition of kidney disease and underlying cause of death codes in the US mortality data, which is the most comparable specification.

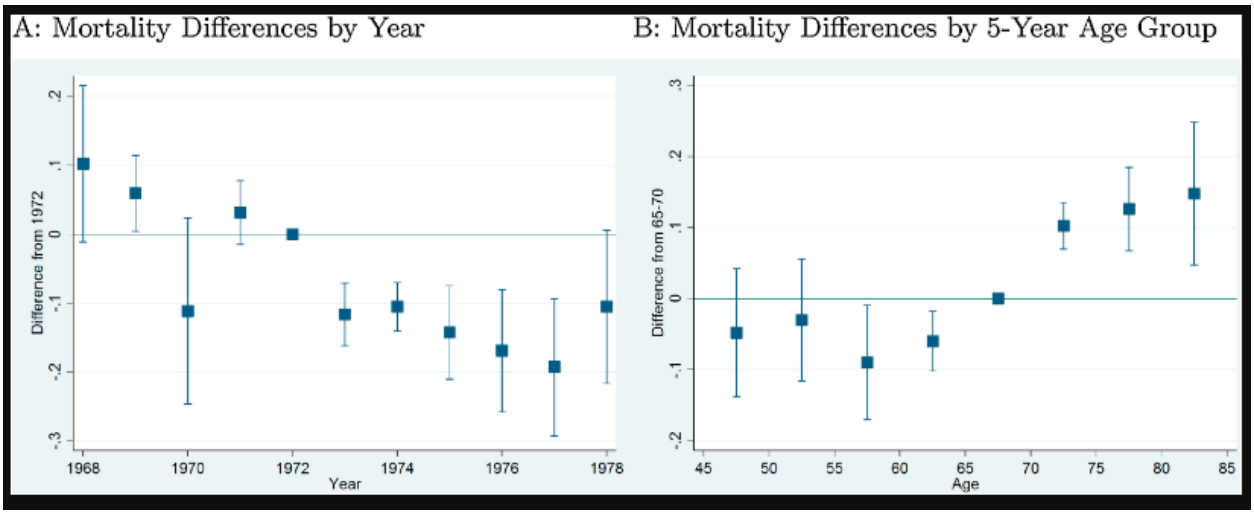

Fig. 4. Cross-country event study estimates of the ESRD program and mortality. Source—Author's analysis of the World Health Organization Mortality Database for 1968 through 1978. Notes—Sample restricted to deaths to people between 45 and 84 years of age. Points in panel A are year-by-under 65 years of age-by-kidney disease-by-United States coefficients from a regression of the cause-age-gender-year-country mortality rate on year fixed effects (omitted 1972), an under 65 indicator, an indicator for kidney disease, and an indicator for the United States and all two-, three-, and four-way interactions. Panel B presents point estimates for years of age interacted with a post dummy (after 1973), kidney disease, and United States from regressions of the cause-age-gender-year-country mortality rate on age fixed effects, post, kidney disease, United States, and all two-, three-, and four-way interactions. Estimates are from Poisson regressions. Confidence intervals are clustered by country, cause-age-gender-year-country cells weighted by population.

5. Mechanisms

The ESRD expansion may have affected health through two classes of mechanisms. First, by lowering the cost of accessing treatment, health insurance may have increased demand for renal replacement services (dialysis and kidney transplantation), which would have been otherwise unaffordable. This mechanism implies that there may be an "access motive" to purchase health insurance in the sense of Nyman (1999, 2003, 1999) and, in essence, reflects the fact that the Medicare expansion provided a large in-kind transfer from healthy people to those with ESRD.

The second class of mechanisms relate to changes in the supply of renal replacement services. The expansion did not merely shift the demand curve outward, but it also guaranteed payment for treatment services, which reduced the risk of investing in renal replacement services. In much the same way that the original introduction of Medicare stimulated entry by hospitals and increased technology adoption (Finkelstein, 2007), the ESRD expansion may have increased adoption and entry of renal replacement services across the country.

Table 4. Cross-country estimates of the effect of the ESRD program on kidney mortality.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
DDDD	-0.064**	-0.077**	-0.064**	-0.076**	-0.057**	-0.064**	-0.058**	-0.065**
	(0.023)	(0.024)	(0.024)	(0.024)	(0.020)	(0.023)	(0.020)	(0.023)
DDD	-0.048*	-0.060**	-0.045*	-0.048*	-0.039*	-0.040*		
	(0.023)	(0.022)	(0.020)	(0.021)	(0.017)	(0.018)		
Only members before 1969	No	Yes	No	Yes	No	Yes	No	Yes
Country FE			X	X	X	X	X	X
Country interactions					X	X	X	X
Year-by-kidney							X	X

Source—Authors' analysis of the World Health Organization Mortality Database for 1968 through 1978, covering the United States and OECD Member States at any point in time.

Notes—Coefficients are point estimates from Poisson regressions using the mortality rate per 100,000 in each country-year-gender-age group-cause of death cell as the dependent variable. DDDD is the coefficent on the four-way interaction of a dummy for the United States, an indicator for the post period, a dummy for deaths due to kidney disease, and a dummy for being 45–64 years of age; DDD is the corresponding coefficient in models that restrict to deaths due to nephritis. All models include year, age, and gender fixed effects and trends in age-65, where age in each cell was recentered by 2.5 years. Country Interactions are two-way interactions of country fixed effects with dummies for kidney disease and post. Sample restricted to individuals between the ages of 45 and 84 and years in which the country used the ICD-8 coding regime.

Estimates are from Poisson regressions, cells weighted by population, standard errors clustered on country in parentheses.

+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 5. In-state treatment capacity and mortality reduction.

	Narrow definition			Chronic only		
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
A: Base model						
DDD	-0.077**	-0.076*	-0.078*	-0.060*	-0.058+	-0.061+
	(0.028)	(0.031)	(0.033)	(0.028)	(0.031)	(0.032)
x Log dialysis clinics						
Per Capita in 1971	-0.065**		-0.093**	-0.062*		-0.086*
	(0.023)		(0.033)	(0.025)		(0.037)
x Log transplant programs						
Per Capita in 1971		-0.008	0.061		-0.009	-0.057
		(0.060)	(0.063)		(0.058)	(0.065)
B: Including indicators for VA treatment facilities						
DDD	-0.080**	-0.077*	-0.081*	-0.063*	-0.059*	-0.064*
	(0.029)	(0.031)	(0.032)	(0.029)	(0.029)	(0.031)
x Log dialysis clinics						
Per Capita in 1971	-0.067**		-0.082*	-0.065*		-0.076*
	(0.025)		(0.037)	(0.027)		(0.037)
x Log transplant programs		-0.016	0.044		-0.017	0.037
Per Capita in 1971		(0.058)	(0.065)		(0.057)	(0.064)

Source—Author's analysis of Multiple Cause Mortality Files for 1968–1978 and the publication "Kidney Disease Services, Facilities, and Programs in the United States" (Kidney Disease Program, 1971).

Notes—Dependent variable is the mortality rate in the state-age-time-gender-cause of death cells, where time is measured in half-year increments. Definitions of kidney disease based on codes in Table A. DDD is the triple difference coefficient for being under 65, in the post expansion period, with kidney disease; models with interactions of DDD with either dialysis clinics or transplant programs also include all two- and three- way interactions of dialysis clinics or transplant programs with under 65, post, and the kidney disease indicator. Models also include indicators for having no dialysis clinics or transplant programs in a state; panel B also includes indicators for the presence of VA dialysis clinics and transplant programs (also interacted with DDD). All measures of dialysis clinics and transplant programs have been demeaned. All models include state, time, and age fixed effects, an indicator for female, and age trends interacted with under 65, post, and kidney disease.

Sample is restricted to deaths to whites between 45 and 84 years of age. Estimates are from Poisson regressions, standard errors three-way clustered on state, age, and time in round brackets; each state weighted by its total population.

+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

5.1. Access to care

In order to test if access to care was an important contributor to the reduction in mortality associated with the ESRD expansion, I augmented Eq. (3) with interactions between the triple-difference variables and measures of the number of dialysis clinics and transplant programs per capita. Table 5 presents the results of this analysis. Panel A demonstrates that living in a state with more dialysis clinics in 1971 was associated with a significantly larger decline in kidney disease mortality, but there was no effect of living in a state with a transplant program. These results persist, even after I include indicators for the presence of Veteran's Administration dialysis clinics and transplant programs (panel B).

The lack of evidence that transplant programs affect local mortality is not surprising since transplant programs require fewer visits than dialysis clinics. Therefore patients may be willing to travel long distances in order to get a kidney transplant, meaning that the number of programs in a state is not the most relevant metric affecting their survival.

The implication of these results is that either the ESRD program increased the number of dialysis clinics in states that already had a large number of clinics, relative to population, or that the program facilitated access to the existing clinic network. In the next subsection, I test if the number number of dialysis clinics per capita after the expansion increased more in areas with more dialysis clinics per capita in 1971, or if there was greater entry in areas with fewer dialysis clinics per capita.

5.2. Entry of treatment facilities

The ideal data with which to test the entry hypothesis would involve regressing the change in treatment facilities on the number of people for whom dialysis or kidney transplantation was appropriate. However, such data are not available. Instead, I use the mortality rate due to kidney disease as a proxy. The idea behind this proxy is that in areas with a higher mortality rate there are likely to be more people for whom treatment is appropriate. Therefore, to test the entry hypothesis, I estimate the following model:

$$\begin{aligned}\ln E\left[\frac{y_s^{1975}}{\text{pop}_s^{1975}}\right] &= \alpha_0 + \alpha_1 \ln\left(\frac{y_s^{1971}}{\text{pop}_s^{1971}}\right) + \alpha_2 \ln \text{Mort}^{\text{Pre}}_{s,<65} \\ &+ \alpha_3 \ln \text{Mort}^{\text{Pre}}_{s,\geq 65} + \alpha_4 \mathbf{1}\left[y_s^{1971} = 0\right]\end{aligned} \tag{5}$$

Where the model is estimated as a Poisson regression, s denotes the state, superscripts refer to the year to which the data refer, y^t_s is either the number of dialysis clinics or transplant programs in state s at time t, pop^t_s is the population in state s and year t, and $\text{Mort}^{\text{Pre}}_{s,\,g}$ is the average annual kidney disease mortality rate from 1968 to 1971 using the "narrow" definition with deaths to attributed kidney disease based on the underlying cause of death codes in state s for age group g (either under 65 or 65 and older).7 α1 tests if the measure of treatment programs in a state is converging across the country depending on whether or not the elasticity of 1975 treatment capacity with respect to 1971 treatment capacity is greater than, less than, or equal to one. α2 and α3 test if treatment capacity is responsive to the burden of disease in the area since areas with a greater burden of disease will have a higher mortality rate due to kidney disease. A priori one would expect $\alpha 2 > 0$ and $\alpha 3 \cong 0$ as indicators that the Medicare expansion, since it affected people under 65 years of age, encouraged entry.

Table 6 demonstrates that the number of dialysis clinics (columns 1–3) was converging over time since the coefficient on 1971 treatment capacity is less than one. In other words, states with comparatively few dialysis clinics, relative to population, in 1971 experienced a more rapid rate of increase than did states with more dialysis clinics per capita in 1971. Furthermore, there is

Table 6. Entry of dialysis and transplant facilities.

	Per 100,000 in 1975					
	Dialysis			**Transplant**		
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Long per capita						
Dialysis clinics	0.316*	0.356**	0.379**			
	(0.127)	(0.090)	(0.091)			
Transplant programs				0.710**	0.577**	0.551**
				(0.154)	(0.108)	(0.123)
Log kidney disease mortality rate						
Under 65		0.367+	0.288			
		(0.197)	(0.222)			
65 and over			0.320			
			(0.284)			
Constant	-0.381	-0.693*	-1.421	-0.500	-1.615**	-0.805
	(0.260)	(0.292)	(1.054)	(0.437)	(0.581)	(1.770)

Source—Author's analysis of Multiple Cause Mortality Files for 1971, the publication "Kidney Disease Services, Facilities, and Programs in the United States" (Kidney Disease Program, 1971) and the 1977 Social Security Bulletin. See text for details.

Notes—Independent variables are measured in 1971, kidney disease mortality rate averaged from 1968 to 1971. Kidney disease mortality defined using the "narrow" definition and underlying causes of death. Models also include indicators for no facilities of a given type in 1971. Estimates from Poisson models, robust standard errors in parentheses.

+p < 0.1, *p < 0.05, **p < 0.01.

some evidence that mortality among people under 65 served to increase the number of clinics in a state in 1975, which is consistent with the Medicare expansion encouraging entry of new dialysis clinics, although the mortality effect disappears when I also include mortality among people 65 and over.

Column 4–6 demonstrate that the pattern of convergence was weaker for transplant programs, but that there was a substantially larger effect of under 65 mortality on the entry of transplant programs than for dialysis clinics.

The fact that there was more rapid convergence for dialysis clinics than transplant programs is consistent with differences in how these two forms of treatment are used. Dialysis clinics require that patients return frequently for treatment since the typical treatment regimen may include as many as five treatments per week, as a result proximity to a dialysis clinic is important, hence one would expect to see a large increase in dialysis clinics. On the other hand, kidney transplant programs require fewer visits so that patients may be willing to travel long distances in order to get a kidney transplant, meaning that there is less need for a uniform distribution of transplant programs across the country.

6. Welfare implications

These results provide some insight into the welfare consequences of the Medicare expansion among people with kidney disease, specifically the productivity of moral-hazard induced care. One typically thinks of moral-hazard induced care as inefficient since it is care that the consumer was unwilling to pay for at the offered price (Pauly, 1968). However, one can recast this framework in terms of the marginal health product of health care and a consumer's willingness to pay for a unit of health. In this framework, a consumer's willingness to pay for health care is decreasing because either the marginal health product is decreasing or her valuation of a unit of health is decreasing. Assuming that a person's value of a unit of health is fixed (or at least unlikely to change significantly) then the downward slope of demand curves for health care (and the resulting welfare losses from moral hazard) come from the decreasing marginal product of health care.

In this paper, I provide suggestive evidence of an increase in utilization of dialysis facilities. First, I find an increase in self-reported physician visits in the NHIS, which includes dialysis care. Second, the reduction in kidney disease mortality was larger in areas with more dialysis clinics in 1971. Collectively, these results suggest that the increase in dialysis clinic visits had a positive marginal health product. Whether or not this health impact was large enough to eliminate the welfare cost of the increase in consumption depends on the size of the health improvement.

I can quantify the size of the health improvement by computing the change in survival associated with the program and, from there, calculating the number of life years saved due to the program. In order to estimate the survival gains, I begin by computing the age- and gender-specific average mortality hazard due to kidney and non-kidney causes in the pre period. In order to compute counterfactual survival, I then multiply the mortality hazards due to kidney disease for people under 65 years of age by the exponentiated triple difference or difference-in-difference coefficient from the models in Table 3. In order to combine these mortality hazards into a single hazard that I can use to calculate survival, I assume that latent survival durations from kidney and non-kidney causes were independent so that the mortality hazard at age a is the sum of the cause specific mortality hazards at age a.8 I compute survival from age 45 as the sum of the cumulative survival probabilities9 and compute the differences from the survival durations implied by the pre-period mortality rates. I then convert these differences, which are representative of the effects on

a 45 year old, into population-level estimates by multiplying by the population of white 45 years olds in 1973, which yields an estimate of the number of life years saved by the Medicare expansion's effect on kidney disease mortality and, therefore, the productivity of the induced health care utilization.

Table 7 presents the results of this analysis. In column (2), which reports results using the narrow definition and with age trends, in the first panel I report that life expectancy from age 45 is 18.36 years up to age 65 and almost 29 years to age 85 (I am unable to calculate subsequent mortality hazards since I do not have denominator data for people 85 and older). Using the triple-difference coefficients there is almost no change in survival—life expectancy rose by 0.001 years to age 64 and by 0.002 years to age 84. However, these estimates are for the entire population while only a small minority actually has kidney disease. When I scale these estimates by the number of white and non-white 45 year olds, I find that the expansion saved between 2200 and 5100 life years, depending on the age cutoff used. I find larger savings using the difference-in-difference estimate to construct the counterfactual mortality rates. Applying a value of $100,000 to a life year, the results in column (2) imply that the mortality benefits of the Medicare expansion due to changes in kidney disease mortality were worth between $220 million and $670 million per year. Spending on this population in a single year was around $750 million indicating that the program cannot be justified solely based on its effects on kidney-related mortality.10 However, using some of my more relaxed specifications (e.g. including contributing causes of death) implies that the value of the life years saved may exceed $1 billion, indicating that it is possible that this expansion yielded benefits in excess of costs, assuming that each life year was worth $100,000 and that other costs associated with the program (e.g. spending on other services) are not too large.

Across most of the remaining specifications, I find evidence of an increase in survival, with estimates using underlying causes of death and age trends indicating that the expansion saved between 2500 and 14000 life years; using contributing cause of death codes as well yields a somewhat broader range, though the increase is not as dramatic as with the narrow definition of kidney disease.

My welfare analysis does not consider the effect of these expansions on the incidence of ESRD. In essence, I am assuming that there is no “ex-ante” moral hazard (Ehrlich and Becker, 1972). However, this perspective is also consistent with my analysis not including the value that these expansions provide against the risk of developing ESRD. Notably these two omissions act in opposite directions–ex-ante moral hazard would tend to decrease the welfare benefit of the program, while the insurance value of protection against a previously uncovered risk would increase the welfare benefit of the program.

Table 7. Impact of the ESRD program on life expectancy at age 45.

	Narrow definition				Broad definition		Chronic only	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Actual								
Survival to age 64	18.36	18.36	18.36	18.36	18.36	18.36	18.36	18.36
Survival to age 84	28.83	28.83	28.83	28.83	28.83	28.83	28.83	28.83
DDD counterfactual								
Surivival to age 64	18.36	18.36	18.36	18.36	18.36	18.36	18.36	18.36
Difference from actual	0.003	0.001	0.003	0.003	0.002	0.002	0.001	0.002
Life years saved	7235	2189	7641	7883	4618	5045	1291	5117
Survival to age 84								
	28.84	28.83	28.84	28.84	28.84	28.84	28.83	28.84
Difference from actual	0.007	0.002	0.008	0.007	0.005	0.005	0.001	0.005
Life years saved	17671	5085	19546	17451	11025	11201	3043	11655
DD Counterfactual								
Survival to age 64	18.36	18.36	18.36	18.36	18.36	18.36	18.36	18.36
Difference from actual	0.003	0.001	0.003	0.005	0.003	0.005	0.001	0.004
Life years saved	7074	2974	6200	12429	6080	11234	1999	9415
Survival to age 84								
	28.84	28.84	28.84	28.84	28.84	28.84	28.83	28.84
Difference from actual	0.003	0.001	0.003	0.005	0.006	0.010	0.002	0.009
Life years saved	17278	6699	15857	26992	14152	24274	4500	20687
Age trends	No	Yes	No	Yes	Yes	Yes	Yes	Yes
Underlying only?	Yes	Yes	No	No	Yes	No	Yes	No

Source—Author's analysis of Multiple Cause Mortality Files for 1968–1978.

Notes—Based on estimates from models presented in Table 3. “DDD Counterfactual” uses DDD estimates from Table 3 and “DD Counterfactual” uses DD estimates. Counterfactual survival is based on multiplying kidney-specific mortality hazard by the relevant coefficient for cells under age 65 and then computing survival as the sum of the cumulative survival probabilities by age (see text for details). Life years saved based on population of white and non-white 45 year olds.

7. Conclusions

In this paper, I estimated the causal effect of the 1973 Medicare expansions affected people with kidney disease. In aggregate the expansion increased insurance coverage and physician visits for people with kidney disease. I also document a significant reduction in mortality due to kidney disease that was robust to a variety of specification checks and alternative definitions of kidney disease.

I identify two mechanisms for my results. The first mechanism is that the increase in insurance coverage provided access to treatment that was otherwise unavailable (Nyman, 1999, 1999, 2003). Consistent with this mechanism, I find larger reductions in kidney disease mortality for people under 65 in areas that had more dialysis facilities in 1971. An important implication of this mechanism is that there is a large liquidity effect in the demand for medical care, in which case the welfare loss from moral hazard may be significantly reduced.

I also find evidence in support of a second, supply-side, mechanism by which the Medicare expansion lead to increased entry of transplant programs and, to a lesser degree, dialysis clinics. Specifically, I find that having a higher mortality rate due to kidney disease among people under 65 betweenn 1968 and 1971 is correlated with having more dialysis clinics and transplant programs per capita in 1975.

My results contribute to a large literature on the effects of public insurance programs (Currie and Gruber, 1996, 1996; Finkelstein, 2007, Finkelstein et al., 2012, Finkelstein and McKnight, 2008, Cutler and Gruber, 1996, Gruber and Simon, 2008, Goodman-Bacon, 2017)(e.g. Currie and Gruber, 1996a,b; Cutler and Gruber, 1996; Finkelstein, 2007; Finkelstein and McKnight, 2008; Finkelstein et al., 2012; Goodman-Bacon, 2017; Gruber and Simon, 2008). However, a distinctive feature of my results, relative to others in the literature, is that the program that I study conditions coverage on being in poor health. As a result, the benchmark for evaluating this program is somewhat different than for other insurance expansions since an effect on mortality that may seem large among a population that was not selected on the basis of ill health, may be much more plausible in the context of a program that explicitly conditioned eligibility on people having an expected survival of days or weeks following diagnosis with ESRD.

Appendix A.

Appendix A. ICD codes for kidney disease, by ICD revision.

	ICD-7 (1968 NHIS)	ICDA-8 (1968–1978)	ICD-9 (1979–1980 NHIS)
Narrow definition:			
Chronic kidney disease	592–594,792	582–584, 593.2, 792	582–589
Acute kidney disease	590–591	580–581, 593.1	580–581, 584
“Broad” definition			
Other diseases of urinary system	600–609	590–599	590–599
Hypertension	442,446	403–404	403–404
NHIS omissions	604–609	594–599	594–599

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