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The Effect of Health Insurance on Mortality: What Can We Learn from the Affordable Care Act Coverage Expansions?

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Abstract. A large literature examines the effect of health insurance on mortality. We add to this literature by exploiting quasi-experimental variation provided by the Affordable Care Act (ACA) expansions in health insurance coverage. We make two main contributions. First, using various differences in differences (DiD) and triple difference designs that compare mortality trends in Medicaid expansion and non-expansion states, in addition to other forms of identification using all ACA insurance expansions, we find no convincing evidence that ACA expansions have changed mortality for non-elderly adults. However, confidence intervals are large, thus our results should not be interpreted as evidence that health insurance has no effect on overall mortality for this age group. Second, we provide a simulation-based power analysis, showing that even the nationwide natural experiment provided by the ACA is underpowered to detect plausibly sized mortality effects in available datasets, and discuss data element and sample size needs for the literature to advance. Our power analysis, which applies pseudo-shocks in the pre-treatment period, can serve as a guide for other natural-experiment studies where assessing plausible effect sizes and exploring statistical power can inform research design and increase the validity of reported results.

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I. Introduction

The relationship between health insurance and mortality is at the center of much empirical inquiry in the health economics literature. Since the first rigorous study of this relationship through the RAND Health Insurance Experiment, researchers have studied this question using varying study designs and populations, finding mixed results on the existence and strength of any relationship; a recent literature review found over 200 studies published on the topic, (Gaudette et al., 2016). Many papers in this literature focus on mortality as an extreme, but readily measurable outcome. Most studies, including the RAND Experiment, studies of Medicare, and the more recent Oregon Health Insurance Experiment find no statistically significant impacts of health insurance on overall mortality for the general adult population (Levy and Meltzer 2008; Finkelstein and McKnight 2008; Finkelstein et al., 2012).[1]

Some more recent studies report mortality reductions from state or federal insurance expansions for adults (e.g. Sommers, Long, and Baicker, 2014). A separate literature finds health and mortality gains from health insurance for children (e.g., Currie and Gruber, 1996a,b; Wherry and Meyer, 2015; Brown, Kowalski, and Lurie 2017). Other studies examine the effect of insurance on non-physical health outcomes, such as mental health stress levels and financial health (e.g., Hu et al, 2016; Baicker et al. 2013).

The Affordable Care Act produced substantial insurance expansions for the low-income, non-elderly adult population (e.g. Kaestner et al., 2015; Wherry & Miller, 2016; Frean et al., 2017; Simon et al., 2017; Courtemanche et al., 2017). These expansions provide a new opportunity to study the link between health insurance and mortality, and to examine issues of statistical power for natural experiment studies of low-frequency outcomes. Our study examines this relationship using mortality microdata from 1999-2015. We use both difference-in-differences (DiD) and triple-difference/age discontinuity approaches to study the effect of state Medicaid expansions and the ACA in total on mortality. We exploit heterogeneity in assignment to "treatment" (health insurance) and potential treatment effect heterogeneity along several dimensions: healthcare

[1] These are studies of the effect on mortality of health insurance, not health care. For example, Finkelstein and McKnight (2008) observe that "part of the explanation for [finding no mortality effect could be that], prior to Medicare, elderly individuals with life-threatening, treatable health conditions sought care even if they lacked insurance, as long as they had legal access to hospitals."

amenable vs. non-amenable causes of death; specific major causes of death (cancer, heart disease, diabetes, and respiratory disease); and sociodemographic factors at the individual (gender, race/ethnicity, and education) and the county (baseline percent uninsured and percent in poverty) levels. Our triple-difference/age-discontinuity design compares the near-elderly (age 55-64) to the young-elderly (ages 65-74), who were already covered by Medicare. We focus on the near-elderly, both because they are more likely than younger persons to have health conditions for which healthcare is important for survival, and because limiting the age bands makes the above and below-65 groups more comparable. This follows the approach taken in the Finkelstein and McKnight (2008) study of Medicare, except their treatment group is our control group. We obtain similar results in analyses using broader age ranges (age 45-64, or all non-elderly adults). We do not find a statistically significant pattern of results consistent with Medicaid expansion causing mortality changes, but we also cannot rule out large effects in either direction. One reason for this "null result" is that our "first stage" is weak: the identifying variation (the relative change in uninsurance rates for Medicaid expansion versus non-expansion states) is a small fraction of the population. The increase in health insurance coverage attributable to Medicaid expansion depends on the population under study; it is only around 4% even when we hone in on low-educated populations and is just above 1% for the full sample of non-elderly adults. A second reason for failure to reject the null of no effect is a high level of "noise" - substantial background variation in mortality, and mortality trends, across states and demographic groups. A third reason is that mortality is a low-frequency outcome. We note too that any effects of health insurance on mortality are unlikely to emerge only over a short time frame.

Our second contribution is to propose that observational studies can often benefit from performing and reporting power analyses, and to use a simulation-based power analysis, in which we impose treatment effects of varying sizes on actual data during the pre-treatment period, and assess whether ACA expansion effects on mortality of plausible size can be detected with the available datasets. We conclude that even the nationwide natural experiment provided by the ACA is severely underpowered. It will be extremely challenging for a study such as ours to reliably detect effects of insurance coverage on mortality unless these data can be linked at the individual level to large-sample panel data. Such data could include information on health, income, and insurance status, which would allow the study to focus on subsamples with a larger first stage and/or a higher sensitivity of health and mortality to healthcare use. Even with such hypothetical

data, it is likely that only fairly large effects of health insurance on mortality could be reliably detected.

We estimate power using our pre-treatment period data (pre 2014) by first applying a pseudo-shock to health insurance rates at the beginning of 2012 as if the ACA expansion had occurred then. We choose pseudo-treated states at random, and then apply pseudo treatment effect (mortality shocks) of different sizes to the group of pseudo-treated states (by randomly removing deaths from our mortality data). We repeat this process 1,000 times. The repeated re-randomization of the set of pseudo-treated states effectively converts the non-parallel pre-treatment trends, for which we find evidence in the data, into additional noise, which reduces power but does not lead to bias. We then assess the likelihood that these pseudo shocks we introduce in 2012-2013 would be detected, using methods similar to our actual specifications. This approach (applying simulated treatment effects to actual data, drawn from a period when no effect should exist) can be applied to a wide-variety of research settings, including both structural and non-parametric work.

The minimum reduction in amenable mortality for all persons aged 55-64 years in expansion states, detectable at the 95% confidence level (two-tailed test), 80% of the time (a standard threshold for a study to be considered adequately power) as a result of a state expanding Medicaid is about 0.022. Together with a 0.012 first-stage this implies a roughly 200% drop in amenable mortality among the newly insured. The DD and triple difference models have similar power. Power does not improve when we examine vulnerable subgroups: non-parallel trends remain common and the gain in power from a higher first-stage and a higher base mortality rate is more than offset by smaller sample sizes.

To put into perspective the implied 200% minimum detectable effect of gaining insurance on the mortality of the newly insured, the introduction of sulfa drugs reduced maternal mortality by 24-36% (Thomasson and Treber, 2008; Jayachandran et al., 2012); Finkelstein and McKnight (2008) found no significant effect of the introduction of Medicare on mortality for those aged 65-74 years (point estimate after 5 years = -0.15%; 95% CI [-3.9%, +3.6%]); Card, Dobkin, and Maestas (2004) use an age-discontinuity design and find no reduction in mortality at age 65 (point estimate +0.5%, 95% CI [-3.3%, +4.3%]); the RAND Experiment found no significant overall effect of health insurance on mortality but found a 10% reduction in mortality for a subsample of persons with vulnerable health; and the Oregon Experiment found no significant effect, with a

point estimate of -13% but a wide 95% confidence interval (95% CI [-39%, +13%]).[2] Large effects are also unlikely because prior research finds that the uninsured already consume substantial healthcare -- about 80% as much as the insured (e.g., Black et al., 2017). Our prior expectation, considering the near-zero estimates and confidence intervals in the largest prior studies (Finkelstein and McKnight, 2004; Card, Dobkin and Maestas, 2004), the substantial healthcare consumed by the uninsured, the imperfect safety net that already covers some vulnerable populations (e,g, the elderly and the disabled), and the availability of emergency care regardless of insurance status (Card, Dobkin, and Maestas, 2009), were that any effect of the 2014 insurance expansion on mortality was unlikely to exceed 10% for the newly insured, and that any effect would likely appear only over time.

Combining this past literature with a power analysis perspective, we expect that if significant effects of expanding health insurance eligibility on general adult mortality are found these are likely to greatly overstate actual magnitudes, be false positives, or both. Reasons to distrust results from low-powered studies include: they may draw from the right tail of a probability distribution; failure to adequately balance treated and control units or address non-parallel trends; specification searches; and "file-drawer bias." McCrary, Christensen and Fanelli (2016) propose a minimum *t*-statistic around 3 to correct for file-drawer bias (insignificant results remaining unpublished) alone.

Power analyses are common in the design (*ex ante*) stage of a randomized trial; researchers use them to ensure that the trial does not "fail" to find a true effect due to inadequate sample size. They are rare, however, for DiD and other observational studies. Ioannidis et al. (2017) and McCloskey (1985) criticize the failure of economics researchers to conduct power analyses. We propose that DiD and other shock-based, observational studies with panel data would often benefit from assessing plausible effect sizes and conducting power analyses, ideally in an explicit "design stage" (with outcomes hidden; see Rubin, 2008). Conducting these analyses can reduce the chance of inadvertently publishing false positive results or results with inflated magnitudes (Button et al., 2013; Gelman and Carlin, 2014).[3]

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[2] Sulfa drugs: See Jayachandran et al. (2012), table 1. Medicare adoption: See Finkelstein & McKnight (2008), App. A. Oregon Experiment: See Finkelstein et al. (2012), Table IX. Medicare age discontinuity: see Card et al (2004) table 11, RAND experiment: see Brook et al. (1983) Table 7.

[3] We discuss below the limited prior examples we have found on use of a simulated power analysis in applied economics research; none involve imposing a simulated treatment effect on actual data.

For example, we find non-parallel trends between treated and control states; mortality among those aged 55-64 drops fairly substantially in treated states over 2009-2013 relative to control states (Figure 2). The triple difference design using persons aged 65-74 years as a within-state control group reduces this problem, but non-parallel trends remain for blacks and Hispanics (Figure 4). DD and triple difference regressions ignore these non-parallel trends. As a result, we find implausibly large, statistically significant effects of ACA expansion on mortality for blacks and Hispanics, in both DD and triple difference specifications. The power analysis and the parallel trends tests reduce the temptation to interpret these significant coefficients as robust results.

We note several limitations of our work. First, our analysis should not be interpreted as evidence that health insurance does *not* affect mortality or health, either overall or for particular diseases or subgroups. Second, studying mortality with ACA-induced variation in health insurance is marginal in three senses: (i) those previously uninsured (implying average lower demand for health insurance) may experience lower marginal gains from insurance than the already insured; (ii) emergency care and substantial healthcare access for vulnerable populations were already provided through prior policy interventions; and (iii) access to health insurance does not equate to access to healthcare, as even the uninsured consume substantial healthcare, and some insurance-induced healthcare could be at the "flat" (or even the downslope) of the marginal benefit curve. We also study a relatively short post-shock time frame, yet any effects of health insurance on mortality may appear only over a longer time frame. However, our simulations suggest that longer-term effects on mortality, with plausible effect sizes, cannot be reliably detected with currently available datasets; moreover, concern with non-parallel trends emerging in the treatment period increases as one moves further away from the shock. Thus, additional years of data, using existing sources, are unlikely to allow a convincing longer-term effect to emerge.

In Part II we summarize the prior literature on the relationship between health insurance and mortality. Part III provides an overview of the conceptual concerns that inform our analysis. The past literature presents a mixed picture. There is no consistent evidence for statistically significant effects of insurance on mortality for the general adult population. There are some effects for specific vulnerable populations such as those with HIV, but not for others, such as those with a disability. Part IV summarizes the ACA insurance expansions. Part V describes our data and presents summary statistics. Part VI summarizes our empirical approach. Part VII presents full-sample results.

In Part VIII, we search for evidence of heterogeneous effects for different subpopulations. Confidence intervals are large and no statistically convincing evidence points to detectable effects even in subsamples where we would expect effects to be more likely. Part IX presents our power analysis, highlights the limited sources of identifying variation and the risk of false positives, and assesses which data and sample sizes might provide adequate power. Part X concludes.

II. Prior Research

A. The Effect of Health Insurance on Health and Mortality

Our first contribution, on whether Medicaid expansion predicts lower mortality, fits into a large literature that examines the connection between health insurance and health status. This literature spans experimental and quasi-experimental settings, and examines morbidity and mortality, physical and mental health, elderly and non-elderly adults, pregnant women, children, infants, short- and long-run effects, and specific diseases and demographic subpopulations.

For our first aim, we focus on the effect of health insurance on mortality in the general adult population.[4] Historically, the first rigorous evidence on how health insurance affects health and mortality comes from the RAND Health Insurance Experiment (Brook et al., 1983; Keeler, 1985; Newhouse, 1993) which provided experimental exposure to varying degrees of insurance generosity; none of the study subjects was fully uninsured. Brook et al. (1983) found no significant overall effect on mortality for the full sample (of persons aged 14 to 61, followed for 3-5 years (point estimate -0.02; 95% CI [-0.05, +0.02]), but found 10% lower mortality for high-risk individuals who received generous insurance. The RAND HIE also found some improvements in blood pressure for low-income populations receiving generous insurance, but otherwise found limited evidence that generous insurance led to improved health.

Finkelstein and McKnight (2008) study Medicare's introduction in 1965, which remains the largest health insurance policy change in US history. Their first stage is around 75%, because

[4] In early research using a natural experiment, Currie and Gruber (1996a,b) find that Medicaid expansions in the late 1980s and early 1990s reduced infant mortality by 8% and all-cause child mortality by 5%. Currie and Gruber (1997) find that neonatal mortality improves when the mother resides close to a NICU unit. However, Howell et al (2010) find that the effects of Medicaid expansion on child and infant mortality are limited to accidental deaths, not disease-related deaths – a puzzling result, since emergency care regardless of insurance has been required since 1996 under the Emergency Medical Treatment and Active Labor Act (EMTALA) and was widely available pre-EMTALA. Wherry and Meyer (2015) examine the long-run impact of eligibility expansions for children using a regression discontinuity design and find lower mortality for nervous system diseases and cancer, rather than for accidents, among black but not white children. These studies, while pointing in different directions, suggest that there is important heterogeneity based on both cause of death and race.

private insurance for the elderly was uncommon pre-Medicare (Finkelstein, 2007). Finkelstein and McKnight (2008) find a 40% drop in out-of-pocket medical expenditures, but no discernible mortality effects over a 10-year period (point estimate after 5 years = -0.15%; 95% CI [-3.9%, +3.6%]). Finkelstein and McKnight observe that these results may be due to the fact that prior to Medicare, those with life-threatening but treatable conditions likely sought care even if they were uninsured.

Card, Dobkin, and Maestas (2004) exploit the age-65 discontinuity in coverage using more recent data from 1989-1998; they find no significant effect of turning 65 on population mortality (point estimate +0.5%, 95% CI [-3.3%, +4.3%]).[5] Their first stage is around 8% for the full sample (Table 3) and 14% for a low-education subsample. In a related study that speaks to possible mechanisms, Card, Dobkin, and Maestas (2009) find a drop in mortality at age 65 among those admitted to hospital through the ED for severe, non-deferrable reasons for which individuals would seek care at the ED whether insured or not: having insurance through Medicare increases treatment intensity by around 3% and results in a 1% absolute (20% relative) reduction in 7-day mortality and a 3% relative reduction in 1-year mortality.

Doyle (2005) studies a subpopulation with strong need for emergency medical care (victims of auto accidents who are alive when they reach the hospital) and finds higher adult mortality rates for uninsured persons in Wisconsin during 1992-1997. He finds that being uninsured increases in-hospital mortality by 39%, relative to other auto accident victims (1.5 more deaths per 100, relative to a mean of 3.8 deaths per 100) (point estimate 0.015, 95% CI [0.003, 0.027]), which he attributes to differences in treatment intensity, rather than pre-accident differences in health; in this sense, the paper also speaks to a specific channel involving in-hospital treatment intensity for emergency care for severe traumatic injury.[6]

Levy and Meltzer (2004, 2008) review the literature and conclude that, consistent with Finkelstein and McKnight (2008) and Card, Dobkin, and Maestas (2004), the literature presents evidence at most of modest health benefits from general adult health insurance expansions. They note potential exceptions for specific vulnerable populations, but conclude that "for most of the

[5] The overall mortality results are included in the 2004 NBER working paper but not later published papers (Card, Dobkin, and Maestas, 2008, 2009).

[6] Another example of health insurance affecting health among a uniquely vulnerable population is Goldman et al. (2001), who use state HIV policies and Medicaid generosity as instruments for insurance status; they find that 6-month mortality falls by 71% as a result of gaining insurance.

population at risk of being uninsured (adults of ages 19 to 50), we have limited reliable evidence on how health insurance affects health." (Levy and Meltzer 2008, p.404).

In addition to the RAND Experiment, two other randomized experiments deserve attention. Weathers and Stegman (2012) find no significant mortality effect for adults receiving Social Security Disability Insurance when they receive health insurance immediately rather than after the usual 2-year waiting period, even when given assistance in navigating the health insurance system (point estimate for odds ratio 1.28, 95% CI [0.71,1.85]. However, their sample of 2,000 persons is small, and thus confidence bounds are wide. They do find that those receiving insurance have higher self-reported health. The second recent experiment is the Oregon Experiment, involving Medicaid expansion for adults, administered through a lottery among those who applied. Finkelstein et al. (2012) and Baicker et al. (2013) find no statistically significant improvement in adult mortality or measures of physical health after 2 years. They do find increased healthcare use, increased diabetes detection and care (but not lower blood sugar levels), reduced financial strain, and less depression. Their first stage on health insurance coverage is strong at around a 25% relative rise in coverage for those in the treatment group; this difference shrinks rapidly, however, and is only half as large after 16 months. Their point estimate for mortality reduction is large, at -13%, but with a wide 95% CI [-26%, +13%]. Thus, both experiments find statistically insignificant effects for relatively vulnerable populations (the disabled for Weathers and Stegman (2012), and poor adults who signed up for the Medicaid lottery and later enrolled if eligible for the Oregon Experiment).

In contrast, several recent papers on insurance expansions for nonelderly adults (Sommers, Baicker, and Epstein (SBE), 2012; Sommers, Long, and Baicker (SLB), 2014; Powell, 2018; and McClellan, 2017) find large effects of health insurance on mortality rates. SBE (2012) considers Medicaid expansion for non-elderly adults in three states (Arizona, Maine, and New York) that expanded Medicaid in the early 2000s compared to neighboring non-expansion states; SLB (2014) and Powell (2018) consider the Massachusetts insurance expansion in 2006. McClellan (2017) considers the ACA mandate that requires employers to cover young adults under their parents' employment-based insurance policies until age 26. And finally, Dunn and Shapiro (forthcoming) considers the effect of Medicare Part D prescription drug coverage for elderly adults.

B. Power analyses and prior use of simulated power in economics research

Our second contribution focuses on the value of conducting and reporting a formal power

analysis in an observational study. We propose that power analyses for DiD studies with reasonably long panels be done using simulation, in which one imposes treatment effects of varying sizes on actual data during the pre-treatment period.

Ex ante power analyses, before research is carried out, are often used in randomized trial designs to assess feasibility and determine necessary sample size,[7] as well as in grant applications for observational studies.[8] However, even when performed at an early stage in a research project, power analyses are rarely reported in published research. It is rarer still to find simulated power analyses. The exceptions we found include Hannon et al. (1993) from bird ornithology, and Hsiang et al. (2015) and Croke et al. (2016) from economics. Of these only Hannon et al. (1993) modify observed data to discern power, while Hsiang et al. (2015) and Croke et al. (2016) create synthetic data that is designed to proxy for real world variables of interest.[9]

Some have argued that power analysis should not be done after results are available (Hoenig and Heisey, 2001; Senn, 2002); citing concerns that a lack of power will be used to justify insignificant findings, which could be due to lack of a treatment effect. Conversely, Gelman and Carlin (2014) point out in low-powered studies which *find* a statistically significant effect, the estimated effect size will often have the wrong sign or have magnitude far larger than the true effect; this implies a need for power analysis in studies which find significant effects.[10]

A growing literature documents the prevalence of underpowered studies in a number of fields, including neuroscience, psychology, medicine, and economics (Button et al., 2013; Maxwell, 2004; Ioannidis, 2005; Ioannidis et al., 2017). Related early work in this vein by economists includes the lament by McCloskey and Ziliak that power analyses are rarely conducted (McCloskey, 1985; McCloskey and Ziliak, 1996; Ziliak and McCloskey, 2004). De Long and

[7] For example, after making assumptions about the mean and sampling distribution of a potential treatment effect, a researcher designing an RCT could use a standard formula to estimate the minimum number of subjects needed to detect an effect of that size at a 5% significance level 80% probability – termed 80% power. This ex ante power analysis is helpful in ruling out study designs that are underpowered given realistic assumptions, and can allow researchers to assess the needed sample size, and to enhance power by changing the research design.

[8] The National Institutes of Health (NIH) require reviewers of grant applications to evaluate how statistical power has been addressed and advice to potential grant applicants is to aim for at least 80% power (NIH, 2016; Gerin et al. (2017).

[9] Hannon et al. (1993) are also the only researchers who conduct a power analysis on their own results, as we propose. Hsiang et al. (2015) and Croke et a. (2016) run power analysis on studies by others.

[10] Gelman (2018) and Button et al. (2013) note a technical concern: power analysis should not be based upon the estimated treatment effect size since noise in the estimated effect size will cause error in the estimated power; an estimated effect that exceeds the true effect would lead to estimated power that exceeds actual power.

Lang (1992) discuss the "file-drawer" problem). Ioannidis et al. (2017) estimate that the median statistical power in a large set of economics articles is 18%, which is far lower than the 80% standard used in experimental design. The authors determine power by relying on meta-analyses of these articles, and comparing the weighted effect size from the meta-analysis to a weighted standard error. Their approach, however, cannot be used to assess power in a single study.[11] In addition, Banerjee et al. (2015) review six randomized trials assessing microcredit and find that most suffer from low power due to a limited take up rate.

Single-study power analyses can be either closed form (based on an assumed data generating process) or simulation-based; the simulation can involve either artificial data (from an assumed data generating process) or actual data, to which a treatment effect is added. A study of bird nest visitation by Hannon et al. (1993), the earliest simulated power analysis we found, is similar in spirit to our own in that the authors apply a simulated treatment effect to actual data. The authors modify their outcome variable (nest visitation) using draws from the binomial distribution, gradually increasing (or decreasing) the probability of visitation. For each modified sample, they draw 50 bootstrapped samples, re-estimate their statistical model, and report power for each imposed effect size as the percentage of times the imposed effect is statistically significant among the bootstrapped samples.

In contrast, Hsiang et al. (2015) estimate power using synthetic data. They generate the dependent variable (likelihood of conflict) using a normal distribution with a fixed mean and standard deviation; they impose a treatment effect by varying the mean to indicate a "treatment effect." For each imposed effect size, they analyze the synthetic data using their preferred specification and report power as the percent of times a statistically significant result is found at the 95% confidence level. Croke et al. (2016) examine a meta-analysis done by Taylor-Robinson et al. (2015) on the impact of mass administration of deworming drugs on childhood health. Croke et al. (2016) demonstrate that the meta-analysis is under-powered by using a simulation similar to Hsiang et al. (2015).

An advantage of entirely synthetic data is that there will be no pre-treatment trends or treatment effect unless one is imposed. However, fully synthetic data involves accepting many

[11] Zhang and Ortmann (2013) and Gallet and Doubouliagos (2017) use similar approaches to estimate power for a series of related studies. Zhang and Ortmann report median power of 25% in experimental papers using the dictator game. Gallet and Doubouliagos report that 59% of studies examining the impact of healthcare spending on life expectancy have adequate power.

unrealistic assumptions, similar to those noted for closed form power analyses by Burlig et al. (2017); one must implicitly impose structure on the variance-covariance matrix, for which the true structure may not be known. For example, in a panel data setting, values could be autocorrelated across time, pre-treatment trends could be non-parallel in complex ways (as we find for our data), and unobserved covariates could predict both treatment and outcome. As Stigler (1977) points out, real data are rarely drawn from a "perfect distribution." Our approach, of applying a simulated treatment effect by modifying existing data during the pre-treatment period, does not guarantee a distribution centered around the null when we impose a zero treatment effect (the data can exhibit an "accidental" effect), but it preserves both the obvious and more subtle relationships present in the actual data that can affect power, and lets us take accidental effects into account in estimating power. We have yet to find a prior example of this approach other than Hannon et al. (1993).[12]

III. Conceptual Concerns

We study the end result (mortality) of a process that starts with policy changes to eligibility for free or subsidized health insurance. To assess the plausible magnitude of any treatment effect and the challenges in measuring that effect using available datasets, one must keep in mind the chain of causation between policy changes and health or mortality. Because large-scale datasets available to researchers do not adequately measure morbidity, many studies (including ours) focus on mortality. However, mortality records are generally not linkable at the individual level to other information, including information on pre-ACA insurance status (which one could use to exclude the always insured from the sample, thus increasing the first stage)[13] or income (which determines eligibility for Medicaid and subsidized private insurance).

Several concepts inform our analysis and the interpretation of our results. One is the existence of prior policies that provide vulnerable populations with health insurance, or with healthcare regardless of health insurance status. These include health insurance or healthcare for the elderly and disabled through Medicare or Medicaid; pregnant women through Medicaid; many low-to-middle-income children through the Children's Health Insurance Program; persons needing emergency care through the 1996 Emergency Medical Treatment and Active Labor Act

[12] Similar procedures are suggested in the online appendix of Burlig et al. (2017), § D.2 and by Gelman and Carlin (2014).

[13] An analogy: The Oregon Experiment achieved a 25% first stage because insurance was offered only to persons who were previously uninsured and had applied for the Medicaid lottery.

(EMTALA); persons with specific high-cost health conditions (AIDS through the 1990 Ryan White Act and end-stage renal disease under Medicare since 1972); those who suffer workplace or automobile injuries; and those with access to public hospitals, publicly supported clinics, or the charity care provided by nonprofit hospitals. Thus, further health insurance expansions will affect principally populations and medical conditions outside these groups.

A second concept that informs our analysis is selection into coverage for a new program, such as the ACA Medicaid expansion, including selection effects for both take-up of new coverage and crowd-out of other coverage. The less policymakers are practically or politically able to target groups likely to be uninsured and promote a high take-up rate, the less power studies like ours have to find detectable effects on health or mortality. For example, the ACA changes eligibility but does not directly provide insurance. As in any "intent-to-treat" (encouragement) experimental design, we can estimate a treatment effect only for the "compliers" with the encouragement. Multiple selection effects are possible, including that those who sign up: (i) may be more health-conscious in other ways; (ii) may have greater healthcare needs (e.g., Kenney et al., 2012); (iii) may be more likely to use additional healthcare once insured; and (iv) may be more compliant with medical advice than the "never-takers" who do not sign up. Thus, estimates for compliers may differ from those for never takers or always takers (the already insured).

Third, there could be substantial treatment heterogeneity even among the compliers, with health insurance improving health for some, but being neutral or even detrimental due to overtreatment (e.g., opioid addiction as an unintended effect of pain treatment). Yet the available data limits our ability to study specific subpopulations.

A fourth concern is heterogeneous health insurance quality. In many states, Medicaid insurance is considered to be of lower quality than commercial insurance (Polsky et al., 2015).

Fifth, health insurance is only one factor potentially affecting trends in health and mortality. Other factors can vary by age and ethnic group (e.g., Case and Deaton, 2015, find rising mortality in middle-age for less-educated whites, but not other groups), and by state (as we find below). Differing trends complicate any effort to define a suitable control group.

These concerns, taken together, highlight the complex relationship between health insurance and health outcomes, and anticipate the limitations of the available data and policy shocks.

IV. **Data**

We measure mortality using the confidential version of the Compressed Mortality File (CMF), which contains records on approximately 2.6 million deaths a year.[14] This dataset is compiled by the National Center for Health Statistics (NCHS) and contains individual death records from the National Death Index, with county-level geographic identifiers.[15] Other data in the mortality files include (1) race, ethnicity, and gender; (2) year of death; (3) age at death (which we collapse into 5yr-age groups, e.g., 55-59, 60-64, etc., because county population, which we use as the denominator for measuring mortality rates, is available only for these groups); and 4) primary cause of death (4 digit ICD-10 code). We use data from 2009-2013 as the pre-treatment period and 2014-2015 as the treatment period for our main DD analysis, but use longer periods going back to 1999 for selected analyses. We conduct county-level analyses, using county population (from the U.S. Census Bureau) as weights, to produce state-level and national estimates that are representatives of the respective populations. To examine the first-stage health insurance estimates that correspond to our mortality analyses, we use information on uninsurance rates from the Census Bureau's Small Area Health Insurance Estimates (SAHIE).[16]

V. **ACA Insurance Expansions and Identifying Variation**

In 2014, the two main insurance expansions under the ACA took place, with Medicaid expansions occurring in 27 states (including the District of Columbia) on or soon after January 1, 2014, and in three more states on or soon after January 1, 2015. "Standard" expansion included coverage for all non-elderly adults with family income less than 138% of the federal poverty level (FPL). Of these 30 expansion states, 10 had conducted significant expansions prior to 2014 and are not included in our main specifications. The "treated" states for our principal DD analyses are the remaining 20 "Full Expansion States"; the control group consists of the 21 "Non-Expansion States"—several of whom expanded Medicaid toward the end of or after our sample period. A number of other studies of Medicaid expansion also focus on the Full-Expansion States (e.g.,

[14] The public-use version of this data can be found at http://wonder.cdc.gov/mortSQL.html, but that version suppresses death counts in county-years with 10 or fewer deaths in any query.

[15] http://www.cdc.gov/nchs/data_access/cmf.htm#data_availability. We do not use data prior to 1999 because that is the first year in which death certificates began using ICD10 codes.

[16] Source: https://www.census.gov/data/datasets/time-series/demo/sahie/estimates-acs.html. SAHIE data is available for ages 50-64, rather than the 55-64 age group we study in our main analyses, but first-stage magnitudes should be similar.

Wherry and Miller, 2016). Table 1 lists the states in each expansion group, as well as the change in percent uninsured in each state from 2013-2015 for persons between the ages of 50 and 64; Appendix Table A-1 provides additional details on each state's expansion status.

The second major way in which the ACA expanded coverage was by creating "marketplaces" with private insurance subsidies for those with income between 138% and 400% of the FPL in expansion states, and 100-400% of the FPL in Non-Expansion States and Wisconsin (which expanded Medicaid only to 100% of the FPL). Our study design exploits mainly variation in Medicaid expansion, but we also provide estimates that use both sources of variation provided by the ACA by comparing areas that received different shocks to uninsurance rates due to differing pre-ACA characteristics.

There is ample evidence that the proportion of uninsured adults fell, and that the sources of payment for hospitalizations shifted toward more Medicaid and less self-pay**.** However, the uninsured population fell in both Expansion and Non-Expansion States. As Table 1 shows, the population-weighted drop in uninsurance rates from 2013 to 2015 for the 50-64 age group averaged 6.1% in Full-Expansion States versus 5.2% in Non-Expansion States; the difference between the two groups is only 0.9%.[17]

This small difference in secular uninsurance declines between treatment and control groups poses a major challenge to any effort to use Medicaid expansion to estimate the effect of health insurance on mortality. Because the "first stage" of the encouragement design is only around 1% of the population, we consider particular subgroups who were more likely to be affected by Medicaid expansion, for whom we can also measure mortality. Even then, we face first stages of 5% or less.

Although the ACA unambiguously reduced uninsurance rates, causal effects on healthcare delivery appear more modest and uneven across types of care (e.g., Mazurenko et al., 2018). The Oregon Experiment found a 40% increase in ED visits among the newly Medicaid eligible (Taubman et al., 2014), and Ghosh et al. (2017) find that ACA Medicaid expansion predicts a

[17] Here, we use uninsurance rates for persons aged 50-64 as the closest available match in the Small Area Health Insurance Estimates (SAHIE) data on uninsurance rates to our principal treatment group of those aged 55- 64. The drop in uninsurance rates was somewhat larger for the entire adult population. See Appendix. If one weighs states equally, rather than by population, the drop in uninsurance rates is 6.4% versus 4.4% (a difference of 2.0%). But the apparent gain in first-stage strength is offset by greater reliance on small states, for which mortality rates are noisier; moreover, this approach answers a different question: 'how is the average US state affected', rather than 'how is the average newly insured person affected?'

nearly 20% increase in prescription drug use. In contrast, there is no evidence that the ACA Medicaid expansion led to a significant rise in ED visits in expansion states (Pines et al., 2016; Wherry and Miller, 2016). Both from this evidence and from prior studies of the effect of health insurance on mortality discussed above, we expect the effect of receiving health insurance on mortality during our study period to be modest.

VI. Empirical Approach

A. Effect of Health Insurance on Mortality

To investigate the effect of Medicaid expansion on mortality, we use several DD specifications: (i) a "simple DD" specification, which assumes a one-time change in mortality rates; (ii) a "leads-and-lags" model, which allows for a separate treatment effect in each year, both before and after Medicaid expansion, and lets us assess whether pre-treatment trends are parallel; and (iii) a "distributed lag" model, which allows the treatment effect to cumulate over the post-treatment period. Treatment is recorded in event time, relative to the year in which each expansion state expanded Medicaid. For states that expand on a date other than January 1 of year *t*, we treat year *t* as post-expansion if expansion occurred in the first half of the year; we treat year *t* as pre-expansion otherwise (see Table 1 for details). All models use county-level data, county and year fixed effects (FE), county population weights, and standard errors clustered at the state level.[18] The simple DD model is:

$$Y_{jt} = \alpha + \beta Post_{st} + \partial X_{jt} + \tau_t + \vartheta_j + \varepsilon_{jt} \qquad [1]$$

Here, *i* indexes individuals; *j* indexes county; *s* indexes state; t indexes time in years, the dependent variable; Y_{jt} is *ln*((deaths)/100,000 persons)+1); we add 1 to the mortality rate to avoid dropping county-years with zero deaths.[19] We limit the sample to Full- and Non-Expansion States to form a stronger comparison. The predictor variable of interest is Post =1 for Full Expansion States in post-expansion years (2014 and 2015 for the 17 states that fully expanded Medicaid in 2014; 2015 for the 3 states that expanded in 2015). The covariate vector X_{jt} includes the following county-level demographic characteristics: % male; % Black; % White, % Hispanic; % aged 0-19, 20-34,

[18] A small number of small, rural counties experienced boundary changes over the study period, which are reflected at different times in different datasets. To handle this problem, we merged some counties (see the Appendix for details).

[19] We use a log-linear model for convenience, so that the regression coefficients are interpretable as (approximate) fractional changes in mortality. We obtain similar results with a linear model, with Y_{jt} = (deaths)/100,000 persons.

35-44, 45-54, 55-64, 65-74, 75-84, and 85+; managed care penetration (Medicare Advantage beneficiaries as % of all Medicare beneficiaries); % disabled (% of Medicare beneficiaries receiving SSDI benefits); % in poverty; unemployment rate; median household income; mean per-capita income; % with diabetes; % obese; % physically inactive; % smokers; active practicing non-federal physicians/1,000 persons.[20] We convert all amounts to 2010 dollars.[21] In some specifications, we use a narrower set of covariates or no covariates, partly to assess whether our results are sensitive to including observable, time-varying, county-level factors, and also because expansion could affect some covariates. We include county and year fixed-effects ($\tau_t and\ \vartheta_{jt}$) in all models to control for potential unobserved covariates that vary across counties but are fixed over time, and for determinants of mortality that are constant across counties but vary over time.

Appendix Table A-2 provides a covariate balance table showing mean values for each covariate by state, averaged over the pre-reform period of 2010-2013. As expected, there are differences in a number of covariates. Expansion states differ from non-expansion states in a number of ways, including age structure (more weighted towards middle ages), race (more White), poverty (less poor), health status (less diabetes, more physical activity), health care access (more physicians per capita) and health insurance (less uninsured). To address covariate imbalance, we also implement an inverse propensity score weighting approach in which we compute ATT weights and use ATT*population weights.[22] Results with these weights, presented in the Appendix, are consistent with those we report in the text.

We principally study mortality due to healthcare-amenable causes (Nolte and McKee, 2003), but also provide some estimates for non-amenable and total mortality. The concept of

[20] We take population data from the Census Bureau at http://www.census.gov/popest/. We use mid-year inter-censal estimates for 1999 and 2001-2009, and post-census estimates for 2011-2015. We obtain physician counts (interpolating from adjacent years for 2009 due to missing data), unemployment rate, median household income, percent in managed care (interpolating from adjacent years for 2006-2007 due to missing data), and percent disabled from the Area Health Resource File (AHRF) at http://arf.hrsa.gov/. County per-capita personal income comes from the Bureau of Economic Analysis at http://www.bea.gov/regional/. County health variables comes from the Centers for Disease Control at https://www.cdc.gov/dhdsp/maps/atlas/index.htm.

[21] Source: www.bls.gov/cpi/. We use the annual average consumer price index for all urban consumers.

[22] To generate propensity scores, we average the covariates over the pre-treatment period (2009-2013). We then run a logit regression, which predicts whether a county is in a Full- or Non-Expansion State, using all variables in Table A1 to generate the fitted propensity p. ATT weights are calculated as (p/(1-p)).

amenable mortality seeks to capture deaths from conditions that are potentially preventable with timely care; examples include heart disease, stroke, cancer, diabetes, and infections.[23]

To study the time pattern of any apparent treatment effect, and to assess whether pre-treatment trends differ between Full- and Non-Expansion States, we use a leads-and-lags model in event time, with the first expansion year set to zero, following Equation (2):

$$Y_{jt} = \alpha + \sum_{k=-5}^{2}(\beta_k * D_{jt}^{k}) + \partial X_{jt} + \tau_t + \vartheta_j + \varepsilon_{jt} \quad [2]$$

Here, k indexes "event time" in years relative to Medicaid expansion. $D_{jt}^{k} = 0$ for Non-Expansion States for all t and k. For Full-Expansion States, $D_{st}^{k} = 1$ for the k^{th} year relative to the adoption year, and 0 otherwise. For states that expanded Medicaid on January 1, 2014, $D_{st}^{1} = 1$ for 2014 and 2 for 2015. Thus, β_1 provides the estimated population average treatment effect for the first expansion year, while β_{-1} is the estimated effect one year before adoption, and so on. We adjust the coefficients by subtracting β_{-3} from each, so that reported $\beta_{-3} \equiv 0$.

In the Appendix, we also report results from a "distributed lag" model, which allows the treatment effect to evolve during the post-reform period:

$$Y_{jt} = \alpha + \sum_{k=1}^{2}(\beta_k * D_{jt}^{k-lag}) + \partial X_{jt} + \tau_t + \vartheta_j + \varepsilon_{jt} \quad [3]$$

Here, the first treatment lag D_{jt}^{1-lag} equals 1 for Full-Expansion States beginning in the first expansion year, while D_{jt}^{2-lag} turns on in the second expansion year. Thus, the coefficient on D_{jt}^{1-lag} estimates the impact of expansion in the first expansion year, while the coefficient on D_{jt}^{2-lag} estimates the *additional* impact in the second expansion year after reform. One can then combine the lagged effects to obtain an overall treatment effect ($\sum_{k=0}^{2} \beta^k$) and accompanying *t*-statistic (using the lincom command in Stata). The principal difference between the leads-and-lags and distributed lag models is that the leads-and-lags model provides a coefficient and standard error for each year by itself, relative to a base year. In contrast, the distributed lag model provides estimates for annual incremental changes, starting from a pre-reform average; we then compute a "sum of coefficients" for the post-reform period.

[23] We implement the concept of amenable mortality using the ICD-10CM causes of death tabulated in Sommers, Long, and Baicker (2014), App. 1, last column. This definition is somewhat broader than the Nolte and McKee definition.

We find evidence from the event study model (described below) that states have differing mortality trends during the pre-treatment period, which casts doubt on the parallel trends assumption required for valid DD analysis. To address these sources of differing trends, we use a further source of within-state variation: mortality trends among those who are 65 or older (and thus always insured) can potentially control for the otherwise unobserved state-specific factors that generate non-parallel trends. We thus also use a triple-difference/age-discontinuity specification (similar to Finkelstein and McKnight, 2008), where the third difference is mortality among persons between the ages of 65 and 74, who are eligible for Medicare and should not be affected by Medicaid expansion, and limit the sample to persons between the ages of 55 and 74, thus comparing mortality trends for the 55-64 age group to those f7or 65-74 age group. The triple-difference specification, analogous to simple DiD, is:

$$Y_{jt} = \alpha + \beta Post_{st} * Under65_{st} + \beta Post_{st} + \beta Under65_{st} + \partial X_{jt} + \tau_t + \vartheta_j + \varepsilon_{jt} \quad [4]$$

Heterogeneity/Robustness

We also seek to strengthen the first stage (the fraction of county population that gains insurance due to Medicaid expansion) and to investigate potential heterogeneous treatment effects, by estimating a model that interacts the double difference with an indicator for counties with high uninsurance rates in 2013, prior to Medicaid expansion. High2013 indicator equals 1 for the counties with the highest uninsurance rates in 2013, such that together they contain 20% of the population of our treated and control states (or demographic subsamples), and 0 for the counties with the lowest uninsurance rates in 2013, containing another 20% of this population; we remove from the sample counties with moderate uninsurance rates (containing 60% of the U.S. population). We thus compare high-uninsurance counties to low-uninsurance counties. The regression equation is:

$$Y_{jt} = \alpha + \beta Post_{st} x High2013_j + \beta Post_{st} + \partial Xjt + \tau_t + \vartheta_j + \varepsilon_{jt} \quad [5]$$

We similarly compare counties with high poverty rates in 2013, containing 20% of the sample population, to counties with low poverty rates, also containing 20% of this population. This approach exploits variation from the ACA overall, rather than just the Medicaid expansion component.

We also estimate separate models for subsamples stratified on covariates that may predict uninsurance rates or response to health insurance, for which we also have mortality data: education, gender, and race/ethnicity. For example, lower-educated subgroups will have larger first stages

and higher mortality rates, and will (subject to the offsetting effect of reduced sample size) be more likely to produce detectable mortality changes.

B. Power Analysis

The power of a statistical test is the probability that the test will correctly reject a false null hypothesis, at a given confidence level. For a regression coefficient, power is normally taken to be the likelihood that the coefficient will be found to be significantly different from zero, at that confidence level. We conduct a simulation-based power analysis by artificially introducing treatment effects of different sizes into the data in the pre-treatment period, and then assessing (over 1,000 iterations) how often our DD and triple-difference regression models can detect these effects at the 90%, 95%, 99%, and 99.9% confidence levels (using two-tailed tests). The goal of this analysis is to determine the minimum effect of health insurance on amenable mortality that is reliably detectable with our data and research design.

The alternative of a closed-form power analysis requires fully modeling the data generating process, including parameterizing the error term for both variance and covariance terms, and is especially hard to construct with panel data in which observations can be correlated over time (Burlig et al., 2017). A simulation using entirely artificial data has similar problems. We therefore use simulation methods applied to real data. For example, our simulation approach builds in "noise" from non-parallel trends in the actual data; with a closed-form analysis we would have to model the level and form of these trends. Our use of regression weights and clustered standard errors further contributes to the difficulty in producing a tractable and credible form for an analytic power calculation. Simulation, applied to real data, avoids these challenges and lets us use the same research design and econometric specification as the main analysis (Burlig et al., 2017).

Our simulation proceeds as follows. We exclude all data from the post-treatment period and use data from 2007-2013 rather than the 2009-2015 period used in our actual analyses. We then do the following 1,000 times: we randomly assign a pseudo-expansion status to 20 of the 41 states in our final study (that either fully expanded or did not expand Medicaid). Thus, in each draw, 20 states are pseudo-treated and 21 are pseudo-control. In each case, we assume that the expansion occurred in 2012, giving us two years of post-expansion data for each pseudo-treated state.

For each randomly drawn set of pseudo-treated states, we impose a pseudo treatment effect of a reduction in amenable mortality (from 0% to 6%, in 0.25% increments) for all persons aged

55-64 living in a pseudo-treated state. We do this by randomly removing deaths from each pseudo-treated county-year using draws from a binomial distribution. For example, if a county-year has 100 healthcare-amenable deaths and the imposed treatment effect is 0.5%, we remove each death with probability 0.005. The expected number of remaining deaths is then 99.5, but the actual number must be a whole number and could be 100, 99, 98, etc. Each imposed treatment effect is randomly distributed across the pseudo-treated states and across counties in each state. Thus, as in this example, it is unlikely that any pseudo-treated county will have its mortality rate decrease by exactly 0.5%, but the pseudo-treated counties will still experience the imposed treatment effect on average (subject to sample variation).

Once we have introduced the artificial shocks, we run the DD model in eqn. (1) and save the regression coefficient and standard error. The percentage of times a result is found to be statistically significant for a given effect size and significance level is the power for that effect size and significance level; a common threshold for a study to be deemed adequately powered is 80% power at a 95% confidence level. We similarly assess power using the DDD model in eqn. (4). In addition to statistical power, we also report three measures based upon Gelman and Carlin (2014) that inform the plausibility of any significant results obtained, given the study's underlying power: the percentage of times a significant, estimated treatment effect has the wrong sign (opposite from the imposed effect; that is, a *higher* mortality in expansion states); in the subset of cases where a significant effect is found, the mean ratio of the estimated treatment effect to the true (imposed) effect (the exaggeration ratio); and the percentage of significant treatment effect estimates that have the correct sign and an exaggeration ratio below 2 (which we term a "believable" coefficient).

VII. Full-Sample Results

We present full-sample results in this section, principally for adults aged 55-64 some limited results for adults in a broader 45-64 age group. See the Appendix for similar results for all non-elderly adults. In Part VIII, we conduct alternative analyses to assess the effects of ACA-induced insurance variation on mortality, focusing on vulnerable subgroups or particular causes of death, which might be more conducive to producing statistically detectable effects.

A. Univariate Graphical Evidence

In Figure 1, we display trends in amenable mortality for the four state groups, for the full time period with available data (1999-2015). We aggregate data to the state-group level using

population weights, and show amenable mortality rates per 100,000 persons aged 55-64; Appendix Figure A-1 shows data for persons aged 18-64. Several features of Figure 1 are important. First, there are substantial differences in mortality rates across the state groups, although these are smaller between our principal comparison groups—the Full-Expansion vs. Non-Expansion States.

Second, Figure 1 shows clear evidence of non-parallel pre-treatment trends. Unless these differences are absorbed by the regression covariates (for our data, we show below they are not) or by our third difference (they partly are), any DD analysis is suspect. More specifically, over 2010-2015, mortality continues to decline in the Mild-Expansion and Substantial-Expansion states, but levels off in the Full-Expansion States and rises in the Non-Expansion States. If one simply compares the post-treatment average difference in mortality rates for Non-Expansion versus Full-Expansion States to a similar post-treatment average difference—as a simple DD regression does—it would appear that Medicaid expansion has a large, immediate effect in reducing mortality. In fact, mortality rates for these two state groups diverge principally over 2010-2013. There is little additional divergence during 2014-2015. The simple DD coefficient would be misleading, because it ignores the non-parallel pre-treatment trends. One value of the power analysis presented below is to protect against finding spurious significance due to non-parallel trends; the power simulation during the pre-treatment period treats pre-treatment trends as a source of additional noise, which reduces power.[24]

[FIGURE 1 about here]

B. Covariate Balance

Appendix Table A-2 provides a covariate balance table showing means, and the normalized difference in means, between Full- and Non-Expansion states for the pre-expansion period of 2009-2013. There are meaningful differences between the two state groups on a number of covariates, as well as on mortality (our principal outcome; see Figure 1) and uninsurance rates. In light of these differences, we also rerun the analyses reported below with ATT*population weights instead of population weights. Results are similar to those we present; see the Appendix. We use population-weighted results as our main specification, as they are more transparent.

[24] A common robustness check, which provides some protection against DD results being driven by non-parallel, pre-treatment trends, is to add linear unit-specific trends to a DD regression. This can be effective in some cases, but requires a long pre-treatment period to estimate the linear trends and assumes a simple parametric (linear) form for those "trends."

C. Leads-and-Lags Regression Results

We turn next to leads-and-lags graphs, using equation (3). Figure 2, Panel A, provides annual point estimates and 95% CIs over 2004-2015, for amenable mortality among persons aged 55-64. There is, as expected, strong evidence for non-parallel pre-treatment trends, with relative mortality improving in Full-Expansion States over 2007-2013. There is also no evidence of a change in relative mortality in the first two expansion years. In Appendix Figures A-3 and A-4, we provide leads-and-lags graphs for total mortality and non-amenable mortality, these also show no evidence of a significant treatment effect.

[FIGURE 2 around here]

The likelihood of finding credible evidence of causal effects weakens further when we compare the coefficient magnitudes in Figure 1 to plausible effect magnitudes for the full populations of the treated states, given the small first stage shown in Table 1. Based on the prior research discussed in Part II, even a 10% effect of health insurance on mortality within two years would be large. Yet a 10% reduction in mortality for the treated (newly insured), with a roughly 1% first-stage (percent of the population treated), implies an average mortality reduction for all persons aged 55-64, and thus a DD coefficient of 0.001 (0.1%). It is apparent from Figure 1 that this reduction would be undetectable; it would be far lower than the annual 95% CIs, and far lower than year-to-year relative changes in mortality in the pre-treatment period, which can be up to 20 times as large (0.02 from year -2 to year -1).

If we take 0.02 as the minimum detectable effect with one year of data and 0.001 as a large but perhaps plausible effect size coefficient, Figure 2 suggests that our study is underpowered by a factor of 20 (equivalently, the ages 55-64 population needs to be 400 times larger). Adding one or two more years of data (which should be possible in the near term) would help, but would not be adequate to overcome this issue. We present a formal power assessment below, which is consistent with this qualitative discussion.

In Figure 2, Panel B, we present a similar figure for amenable death rates for those aged 65-74 to provide background for our triple-difference regression estimates. There is again evidence of non-parallel trends, with mortality dropping in Full- versus Non-Expansion states in the pre-treatment period. This suggests that the third difference (where we use 65-74 year olds as a within-state control) can limit the non-parallel trends we saw in Figures 1 and 2A.

Figure 2, Panel C provides triple-difference leads-and-lags results: annual point estimates and CIs are for Full- versus Non-Expansion States and for the 55-64 versus 65-74 age groups. Non-parallel trends are muted, but standard errors are larger than in Panel A. Moreover, there are still large year-to-year swings in relative mortality in the pre-treatment period, with a jump of around 0.02 from 2006 to 2007, and a similar jump from 2009-2010. Figure 2C shows dips in relative mortality in Full-Expansion States in 2014 and 2015, but the magnitude is both much larger than the plausible causal effect of around 0.001 and too small to be statistically convincing, given the year-to-year variation we observe in the pre-treatment period.

We considered an alternate DD specification that compares persons aged 55-64 to those aged 65-74 in the same state, but concluded that inference would be unreliable due to strongly non-parallel pre-treatment trends (rising relative mortality for those aged 55-64). See Appendix. The triple difference specification appears to be the best available in limiting the extent of non-parallel pre-treatment trends; it remains suspect, however, because it depends on non-parallel trends in the double differences tending to offset each other in the pre-treatment period, with no basis for confidence that they would continue to do so in the treatment period.

D. Synthetic Control Results

We also sought to assess whether we could obtain a better match between treated and control states, and thus tighter confidence bounds, using synthetic control methods. We used two approaches. In the first, we combined the Full-Expansion States into a single treated unit and used usual synthetic control methods (Abadie, Diamond, and Hainmueller, 2010)[25] to construct a synthetic match using the Non-Expansion States as donor states. We report results in Figure 3.

[FIGURE 3 around here]

The synthetic control approach minimizes the difference between the pre-treatment mortality rates of the treated states and a weighted combination of the Non-Expansion States. However, the maximum difference between the two series is still sizeable, at around 0.02 in 2007. Moreover, visually, a large gap arises in 2013. Thus, this approach fails to create a close enough match in 2013 for this method to produce a satisfying solution to our concern with non-parallel

[25] We used code for this approach from Soni (2016).

trends. We were not persuaded that, for our data, the synthetic control approach is an improvement over the triple-difference design.[26]

We also considered an extension of the synthetic control strategy, following Xu (2017). Xu's "generalized synthetic control (gsynth)" method generates a separate synthetic control for each full-expansion state, drawn from the non-expansion states. One can then conduct DD analyses on the resulting treated and control units, and obtain analytical standard errors (which the original method does not provide). This procedure does not allow for weighting different units. We therefore only discuss state-level results.[27] While we cannot exactly replicate our triple difference models using the gsynth method, we constructed an approximation, by using as the treated units each treated state's 55 to 64 year olds, and as the donor pool both every non-expansion state's 55 to 64 year olds and every state's (expansion or not) 65 to 74 year olds. We present results in Appendix Figure A-13. Similar to the simpler synthetic control method presented above, there is a large drop in amenable mortality in Full-Expansion States in 2013; mortality in expansion states then rebounds in 2014. The poor pre-period fit is even more pronounced with county-level data, and is driven by small counties, which have highly varying death rates and are hard to fit even with a large donor pool. We concluded that the gsynth approach cannot be reliably applied to our data

E. DD and Triple-Difference Regression Results

We next turn to regression analysis. Table 2 shows results from DD regressions, following eqn. [1], with county and year FE and county population weights, separately for our principal treatment group (ages 55-64) and the placebo group (ages 65-74). It also shows triple-difference results, following eqn. [4]. While both DD and triple-difference specifications are suspect because of parallel trends problems, non-parallel pre-treatment trends are less severe for the triple difference; thus we focus on those results and show the DD results principally for comparison. We show separate results for healthcare-amenable mortality, non-amenable mortality, and total mortality. Even-numbered columns include the covariates noted above. We present results for the 55-64 age group both because we expect the effects of health insurance to be higher for this

[26] A further concern with the synthetic control approach is that it gives zero weight to most donor states and assigns positive weights to several very-low-population states (Alaska, Maine, Wyoming) that do not otherwise seem good matches for the Full-Expansion States. Appendix Table A-8 shows the weights on each donor state.

[27] Although we could not directly use population weights within Xu's method, we simulate doing so by repeatedly running his procedure on bootstrapped datasets with draws weighted by population. Results, with both state-level and county-level data, were similar to those we discuss in the text.

group than for younger persons, and because we need to study a limited age band to pursue the triple-difference approach.[28] In the Appendix we estimate DD models that include younger ages for the treated population, with similar results. We caution that these regressions assume flat pre-treatment trends, but we in fact observe a declining trend. Given this trend, DD results will be biased toward finding a post-expansion drop in mortality.

In Table 2, in regressions with covariates, we find a statistically significant 2.1% post-expansion fall in amenable mortality for those aged 55-64, with no significant change in non-amenable mortality. However, in addition to assuming parallel trends, these results are fragile. First, the coefficient on the Full-Expansion dummy is far too large to be credible. Given our roughly 1.2% first stage, it implies an impossible 175% (2.1%/1.2%) reduction in amenable mortality among those who gain health insurance. Second, for the placebo group (ages 65-74) and the placebo-outcome (non-amenable mortality), we observe a large, statistically significant *rise* in mortality. Third, the triple-difference decline in mortality is far smaller, at 0.7% (although still implausibly large) and is not close to statistical significance. Note too that the standard errors for amenable mortality are around 0.007 with covariates and rise to 0.009 in the triple-difference specification. This implies a minimum detectable effect of around 0.014 to 0.018, which implies a 120-150% drop in amenable mortality for compliers. This is further evidence the research design is severely underpowered.[29]

[TABLE 2 around here]

[28] In Appendix Table A21, we consider a version of this where we examine Gompertz style mortality curves, that is the mean health care amenable death rate per 100,000 by single year of age. We report a curve for both expansion and non-expansion states, both before and after expansion (2014). Difference across time (pre-2014 to post-2014 for non-expansion states; and pre-expansion to post-expansion in expansion states) illustrate that the death rate of each single year of age in expansion states have reduced relative to each analogous group in non-expansion states. The differences across age groups (55-64 v 65-74) illustrate that this improvement was not limited to those eligible for Medicaid. That is, the improvement occurred for Medicare enrollees as well. Thus even with disaggregated data by age, we do not find conclusive evidence of a Medicaid expansion impact on the mortality rate for the near elderly (55-64).

[29] If we expand the age range for the treated group to 45-64 instead of 55-64, the insignificant negative triple-difference point estimate in Table 2 switches sign; see Table 4. Moreover, by broadening the age range, we weaken the logic behind using mortality for persons aged 65-74 as a third difference, yet we need the third difference to address non-parallel pre-treatment trends.

VIII. Is There Evidence of Heterogeneous Effects?

We next investigate whether the discouraging conclusion for the general adult population—no evidence of a statistically significant effect, and far too little power to detect an effect of plausible magnitude—would change if we focused on subgroups that are more likely to be affected by the ACA health insurance expansion. These subgroups can potentially provide a stronger first stage, a stronger second stage, or both. However, moving to subgroup analysis also reduces sample size. We consider subgroups based on gender, race/ethnicity, education, specific cause of death, and county poverty and baseline uninsurance rates.

Our data has limitations for all subgroups except gender. For race and ethnicity, we can obtain estimates of the first stage (change in uninsurance rates) only at the state level, not the county level, due to limitations of the SAHIE data. The DD design does not explicitly use the first stage, but it is central to assessing what coefficient magnitudes are reasonable. For education, population data is available only for broad age groups (45-64 and 65+; 5-year average). For analysis by prior insurance status and by income, we observe percent uninsured and percent below 138% of the FPL threshold for full ACA expansion at the county*year level, but cannot directly study these subsamples because the mortality data does not contain information on income or insurance.

A. Variation Between Demographic Groups

We begin in Figure 4 with leads-and-lags graphs of the triple differences in amenable mortality for samples subdivided on gender and on race/ethnicity (white non-Hispanic, black non-Hispanic, and Hispanic). Most post-expansion point estimates are insignificant. The exception is non-Hispanic Blacks, who show a post-expansion drop in mortality. However, for this subgroup, we observe non-parallel pre-treatment trends even with the triple-difference specification; the post-expansion drop in mortality could merely reflect continuation of those trends. Also, the first stage for non-Hispanic Blacks is not greatly different from that for the population as a whole (Table 3). Thus, the point estimates in Figure 4 (around -0.05) are not possible as true effects of Medicaid expansion. There is milder evidence of a drop in mortality in event year +1 (second expansion year) for Hispanics, but here too there is a drop; the point estimate for 2015 (around -0.10) is too large to be possible, let alone plausible.

[Figure 4 around here]

We turn next to DD and triple-difference regression results for amenable mortality for these subsamples, starting with demographic subsamples in Table 3. The "all" row in Table 3 is the same as in Table 2. The first column of Table 3 shows the first-stage change in uninsurance rates for Full- versus Non-Expansion States, in percent, for persons aged 50-64 (the closest available age match to our main treatment sample). All first stages are small; the largest is for Hispanics at 2.4% (not significant). The hope that we might attain greater power for, say, Blacks or Hispanics, who were more likely to be affected by Medicaid expansion, fails. The loss in power due to reduced sample size outweighs the gains due to modestly higher first stages.

In Table 3, a number of the DD coefficients in column (2) are significant and negative, but significance disappears in the triple-difference specification except for non-Hispanic Blacks and Hispanics. However, as noted above, both of these estimates are suspect due to non-parallel pre-treatment trends and implausibly large point estimates.

[TABLE 3 around here]

B. Variation Based on Education

In Figure 5, we show leads-and-lags graphs for the triple difference in amenable mortality for subsamples stratified on education. Low education predicts poverty and hence eligibility for Medicaid expansion; it may also affect the mortality response to the "treatment" of obtaining Medicaid. Recall that for these subsamples, we study persons aged 45-64, and the triple difference compares these persons to all persons age 65+. We present leads-and-lags graphs for elementary school only; partial high school without graduating; high-school graduate; and some college. There is no evidence of a post-expansion decline in mortality for any subgroup, including the less-than-high-school groups.

[Figure 5 around here]

In Table 4, we show regression results by education level. The first row shows full sample results. These differ from Table 2 due to the broader age range that we use due to data limitations. Note that in the preferred triple-difference specification, the point estimate is now positive (higher mortality) and insignificant, and that Medicaid expansion predicts a significant drop in mortality for the elderly (a placebo group). Both results cast further doubt on whether an effect of Medicaid expansion on mortality can be reliably detected.

The first column shows the relevant first stages. The first stage is around 4% for persons without a high school degree, but drops to 1.6% for high school graduates with no college, and to only 0.5% for persons with some college**.** However, the non-high-school graduates are only 12% of the 45-64 age group, so the power gained from a stronger first stage is largely offset by loss of sample size.

The first row shows full sample results. The second through fifth rows show effects for the four education groups, starting with the lowest group, those with only elementary school completion, while the other rows show successively higher education categories. All DD and triple-difference point estimates are insignificant, consistent with the leads-and-lags graphs in Figure 5. The point estimate for three of the four education groups, including the least educated, are positive (opposite from predicted).

[Table 4 around here]

C. Variation by Primary Cause of Death

In Table 5, we present effects specific to HIV as well as the top 4 causes of death: cancer, diabetes, cardiovascular causes, and respiratory illnesses. The Appendix includes corresponding leads-and-lags graphs. All of these causes are within the broad category of amenable mortality. First-stage estimates are not available with our data, because we lack data on Medicaid insurance takeup among those with specific diseases. However, Soni et al. (2018a, 2018b) use a DiD design based on Medicaid expansion and report a 2.4% first stage among persons with cancer diagnoses and a 6.4% increase in early-stage cancer diagnoses. Diabetics could plausibly benefit more strongly from Medicaid expansion given the negative correlation between income and diabetes prevalence and evidence from the Oregon Medicaid Experiment that gaining Medicaid insurance predicts increased diabetes diagnosis (Baicker et al., 2013). HIV is another specific condition, for which health insurance has predicted lower mortality in previous studies (Goldman et al., 2001). However, both DD and triple-difference coefficients are insignificant for all causes of death.

[Table 5 around here]

D. Variation by Pre-ACA Uninsurance and Poverty Rates

We turn next to an effort to exploit pre-ACA uninsurance rates and poverty levels. We cannot measure the second stage (mortality by individual income and insurance status) from the mortality data, so we address this source of heterogeneity indirectly at the county level. The DD

specification is the same as above; the third difference for is high-versus-low pre-ACA uninsurance rates in counties. We compare "treated" high-uninsurance counties (the counties with the highest pre-ACA uninsurance rates, defined so that they together contain 20% of the U.S. population) to "control" counties with the lowest pre-ACA uninsurance rates, also containing 20% of the U.S. population; we drop all other counties. This is similar to the analysis in Finkelstein and McKnight, 2008, exploiting pre-Medicare variation in insurance levels, and Courtemanche et al 2017 for the ACA. The third difference for high-vs-low poverty counties is similar: high-poverty counties (the counties with the highest poverty rates, together containing 20% of the US. population) versus low-poverty counties (counties with the lowest poverty rates, also containing 20% of the U.S. population); we drop all other counties. These comparisons rely on all ACA-induced sources of health insurance expansion, rather than Medicaid expansion alone.

We present leads-and-lags graphs for amenable mortality in Figure 6. Neither graph shows evidence of a treatment effect. Both graphs show signs of a pre-treatment trend toward lower mortality in high-uninsurance counties (over 2009-2013) and high-poverty counties (over 2010-2013), which does *not* continue in the post-expansion period and indeed reverses for the high-uninsurance counties.

[Figure 6 around here]

We present regression estimates in Table 6, for the full sample and for demographic subsamples. Data are sufficient to let us compute first-stage estimates only for the full sample and for male and female subsamples. The first stage rises to 2.4% for the by-uninsurance subsample but to only 1.6% for the by-income subsample, but the sample size is, by construction, only 40% of the full sample. There is no evidence of significant effects of Medicaid expansion on mortality. For the full sample, the coefficients for both subsamples are insignificant, and the coefficient for high-vs-low uninsurance rates is positive (opposite from predicted). For demographic subsamples, six of the 14 coefficients are positive; only two are significant, one of those is positive, and the only significant negative coefficient is barely significant and yet of implausible magnitude.[30]

[Table 6 around here]

[30] In the Appendix, we use all counties and estimate continuous versions of the comparisons in Table 6 between high and low uninsurance (or poverty) counties, again with insignificant results.

E. Heterogeneous Effects: Summary

In sum, our search for evidence of a significant effect of Medicaid expansion on mortality for particular subgroups comes up empty. Most regression coefficients are insignificant. When significance is found (for Non-Hispanic Blacks and for Hispanics, in Table 3), there are other factors that cut against a causal interpretation, including non-parallel pre-treatment trends and coefficients of implausible magnitudes given the weak first stages. We are also wary of assigning too much importance to statistically significant results in particular specifications given the number of estimates we produced, although we did not conduct formal Bonferroni type p-value adjustments.

IX. Power Analysis

We return to our conceptual framework of the chain of events by which insurance expansions may affect mortality, and discuss the conditions under which studies of the ACA using death certificate data could establish a connection between health insurance and mortality.

A. An Illustrative Example

Suppose first that out of 100,000 individuals aged 55-64, half became newly insured. By how much would the likelihood of death within 2 years have to change for us to find that change to be statistically significant? The annual amenable mortality rate in this group is around 600 per 100,000 per year (Appendix Table App-1), if insurance were to reduce the probability of death by 25% among the newly insured, then insuring 50,000 individuals among 100,000 individuals would reduce the expected number of annual deaths by 75 (0.5*0.25*600) to 525. In expectation, a DD regression should show a 25% reduction in mortality rate.[31] But there will also be random variation in mortality. If mortality events are independent, the expected standard deviation (σ) of mortality/100,000 persons will be around 24,[32] and the expected *t*-statistic will be 3.07.

Now assume that there is random "external" variation in state-level mortality rates, with a standard deviation of around 2% per year (±12 deaths per year. As we show below, this is a reasonable level for our data. If this source of variance is independent of that due to health

[31] The expected coefficient in a regression, such as those we run, with *ln*(mortality rate +1) as the dependent variable should be around -0.22

[32] This uses the standard formula for the variance of a binomial distribution with probability Var = n*p(1-p). For n 100,000 and p = .006, Var = 596 and $\sigma = Var^{0.5} = 24.42$.

insurance, expected total variance will be 596 (from random mortality events) + 144 (from external variation) = 740, expected standard deviation will be 28 and the expected t-statistic will be 2.76 – lower but not dramatically so.[33] The large effect of health insurance swamps the additional "noise" from other sources of variation in mortality.

Now assume that the background noise remains the same, but only 5% of the population is treated, and the mortality reduction for the newly insured is 10% instead of 25%. The expected population average treatment effect is now a reduction in the mortality rate of 3 (0.05*0.1*600) to 597. The standard deviation in the number of expected deaths remains the same, so the expected *t*-statistic will be only 3/28 = 0.11. To bring this *t*-statistic up by a factor of, say, 20 to 2.2, one might initially imagine we would need a sample 400 times as large – 40 million people.

However, as sample size increases, the variance in mortality rate due to independent mortality events falls by the usual factor of $n^{1/2}$. With a hypothetical sample of 40 million, the variance in the mortality rate (per 100,000 persons) would be 594/20 ≈ 30. But the variance due to external state-level mortality shocks will not fall and will dominate expected total variance, which will be 30 + 144 = 174; implying expected ($\sigma = 13.2$; $t = 0.23$).

This, in a nutshell, is the power problem we face. With a weak first stage, and a moderate second stage, even a very large sample cannot overcome the confounding effect of external variation in mortality rates. If that external variation is independent across states, then having more treated and control states will help but only somewhat. For example, if we had 20 treated states and 20 control states, all of equal size, the combined external variance for both groups would be (144/20) + (144/20) = 14.4; expected total variance would be around 44, implying expected ($\sigma = 6.64$, $t = 0.45$). If the treatment effect of health insurance on mortality were felt immediately then more years of data would help, but only somewhat, given that state-level mortality shocks are likely to persist over time. For example, 3 years of data, variance due to random arrival of deaths would fall to $29.7/(3^{1/2}) = 17.1$, but if state shocks are persistent, total expected variance will be 17.1 + 14.4 = 31.5; implying expected ($\sigma = 5.62$; $t = 0.53$). Having a first stage lower than 5% -- as we do -- will only exacerbate matters.

Thus, this example illustrates that a full-sample effect size on the order of a 0.5% reduction in mortality (hence an expected regression coefficient around - 0.005 in the log-linear specification

[33] Variances due to independent sources add so $Var_{tot} = 596 + 124 = 740$, and $\sigma_{tot} = Var_{tot}^{0.5} = 27.56$.

we use) will not be detectable. Our power analysis formalizes this intuition, and shows that for plausible effect sizes, the effect of ACA Medicaid expansion on mortality is too small to be captured using death certificate data, unless that data can be linked to income data and insurance data, thus permitting a much larger first stage. We also show below that given lower power, one should be cautious in interpreting any statistically significant results from studies such as ours, even if parallel trends assumptions appear satisfied.

B. Available First-Stages

An initial question for our power analysis is what first stage one could realistically achieve with better data. Our full-sample first stage is similar to that in ACA Medicaid expansion studies.[34] From SAHIE data, the first stage for low-income, Medicaid-eligible adults (income < 138% of FPL) is around 5%. We also saw above that the first stage for low-educated adults is around 4%.[35] Thus, around 5% is likely as large a first stage as one can achieve without linked individual data on some combination of income, family status (children at home), pre-expansion insurance, and mortality.[36] ACA-derived insurance gains were somewhat smaller among the near elderly (on whom we focus) than among younger adults, perhaps because the near-elderly have greater healthcare needs and greater income, which led many to obtain insurance pre- ACA.[37]

We present power calculations below for the aged 55-64 population (around 29M persons, 14M in treated states), and also for our triple-difference specification. The first stage for the closest population for which we have data, persons aged 50-64, is around 1.2% (Table 3). A 10% reduction in mortality for the newly insured, as large a near-term effect as we consider plausible, thus corresponds to a 0.012% reduction in mortality for all persons in this age group. The upper

[34] Long et al (2014), using data from 2013-2014, find a 5.8% drop in uninsurance in expansion states vs 4.8% in non-expansion states, between 2013 and 2014, implying a 1.0% first stage. Smith and Medalia (2015) find a 3.4% reduction in uninsurance for all persons aged 0-64 in expansion states vs 2.3% in non-expansion states, hence a 1.1% first stage.

[35] Kaestner et al. (2015) estimate a similar 3% first-stage for low-educated adults, age 19-64.

[36] Wherry and Miller (2016), use income data from the National Health Interview Survey to isolate persons with incomes < 138% of FPL and find a 7% relative increase in insurance rates from 2010 to 2H2014 low-income persons aged 19-64; compare the 5% increase from 2013 to 2014 we find using SAHIE data. Simon et al. (2017) combine income data with childless status and find a 10% increase for childless adults age 19-64, with incomes < 100% of FPL and no children at home in 2014-2015, relative to a 2010-2013 baseline.

[37] Appendix Figure A-25, reproduced from the American Community Survey (ACS), shows the ACA-related change in uninsurance rates by age.

end of the 95% CIs from Finkelstein and McKnight (2008) and card, Dobkin and Maestas (2004) imply an even lower mortality decline, bounded at 0.004%.

To put these numbers in context, Medicaid expansion led to around 170,000 people gaining health insurance in Full-Expansion States (0.0012 * 14.1M). If the mortality of the newly insured would have been similar to all persons in this age range but for Medicaid expansion, about 0.6% would have died each year (about 1,000 persons), and a 10% reduction in mortality would save around 100 lives annually. We cannot directly measure the relative mortality of the uninsured with our mortality data, but Black et al. (2017) provide evidence from the Health and Retirement Study that mortality for uninsured persons in the HRS population (initial age 50-61, so similar to the group we study) was similar to mortality for insured persons.[38]

The power challenge is to find statistically significant evidence for a fall in mortality of 100 persons (or less), in a combined treated and control population of around 29M, with 170,000 annual deaths. As we show below, that challenge cannot be met without individual level data on personal characteristics (income, family status, pre-ACA insurance), sufficient to greatly increase the first stage, linked to mortality data. Even with that data (not currently available), one would need a very large sample of newly insured persons and similar controls.

C. Full Sample Power Simulation Results

To investigate the minimum effect that our main DD and triple-difference specifications can detect, we perform the power exercise outlined in Section VI B. Figure 7 illustrates the results from our power simulation, using the amenable mortality rate for all persons aged 55 to 64 as the dependent variable. The simulation uses data from 2007-2013, and a pseudo-shock applied on January 1, 2012, to states chosen at random from our actual treated and control states.

Panel A shows DD results and Panel B shows triple-difference results, using the same regression models as in Table 2. The DD results indicate that to achieve 80% statistical power (finding a significant effect at least 80% of the time), the minimum detectable population average

[38] Black et al. (2017), Table 2 calculates mortality differences in the manner most appropriate for these comparisons; the uninsured (aged 50-61) have higher mortality than the privately insured, but lower mortality than the publicly insured, leading to similar overall mortality between insured and uninsured over two- and four-year observation periods. To put these estimates in the context of prior literature, Galea et al 2011 reports that mortality for poor non-elderly adults is 75% higher than for the non-poor but does not report mortality differences for poor uninsured vs poor insured, which is the relevant comparison for our study. Kronick (2009) finds a 1.20 mortality hazard ratio for the uninsured versus the privately insured over a 14-year followup period after controlling for income (but does not compare the uninsured to the publicly insured).

treatment effect size at the 95% confidence level is a mortality reduction of 2.20% for the DD simulation, and 2.16% for the triple-difference simulation. Below, we focus on the triple-difference results, which we prefer because they are less subject to concern with non-parallel trends. A 2.16% fall in overall amenable mortality, given the roughly 1.2% first stage, implies that Medicaid expansion would have to reduce the average amenable mortality rate of all newly insured persons by (.0216)/(.012) = 180%. If we apply a stricter significance standard, to account for specification error, specification searches, and file-drawer bias, the minimum detectable effect will be substantially higher – Figure 7 also shows power curves for the 99% and 99.9% and confidence levels.

The minimum detectable effect can also be framed in terms of lives saved. The 2.16% reduction in mortality needed for 80% power and 95% confidence translates into about .0216 * 14.1M * .006 = 170,000 = 1,827 annual deaths – almost 20 times the maximum plausible effect.

[Figure 7 about here]

The power analysis assumes that the underlying mortality rate of the newly Medicaid insured is similar to other persons aged 55-64. The actual rate could be higher (the newly insured tend to be low income, and thus higher mortality), or lower (the disabled are already insured, those in poor health could be more likely to already have insurance, and the first stage is lower for men, who have higher mortality rates than women), but is unlikely to be radically different. By comparison, Finkelstein et al. (2012, Table IX) study a likely lower-income, less-healthy population (persons who applied for the Oregon Medicaid expansion lottery), and report annual total mortality for the controls of 0.008, which is similar to the average total mortality rate we find for persons aged 55-64 in both Full-Expansion and Non-Expansion States. Power is also similar if we weight states equally, rather than by population; this increases the first stage to around 2%, but increases noise by giving more weight to smaller states.

"Power" also has peculiar properties, in the situation we face, where plausible effect sizes are small relative to those one can reliably detect. This implies both that: (i) the estimated effect is likely to greatly exceed the true effect; and (ii) there is an important risk that the estimated effect has the wrong sign (opposite from truth). Gelman and Carlin (2014) therefore recommend reporting two measures of plausibility in addition to power, the wrong-sign-likelihood and the exaggeration-ratio. Ioannides et al. (2017) report evidence that much economics research and thus prone to these concerns. We illustrate these problems in Figure 8.

In Figure 8, Panel A, we show the ratio of the magnitude of the estimated effect (when found to be statistically significant) to the "true" magnitude, imposed in the simulation. For population effect sizes under 1% (recall that a 10% mortality reduction for the newly insured implies a population effect around 0.1%) the exaggeration ratio is high – an effect which is large enough to be statistically significant is likely to be far from truth. In Panel B we show the proportion of statistically significant results that have the wrong sign. This proportion is also appreciable for the smaller population effect sizes. As we increase the imposed population effect size, the wrong-sign problem shrinks, and is negligible for effect sizes s above 1%; the exaggeration ratio also shrinks, but more slowly.

[Figure 8 around here]

As we discussed in Section A, one important source of "noise," captured in the power simulations but assumed away in DD regressions, is non-parallel mortality trends across states. We illustrate that concern in Figure 9. For this figure, we use a DD model, continue to use data from 2007-2013, apply a pseudo-shock to amenable mortality on January 1, 2012, but this time to one state at a time, treating all others as controls. We show a scatter plot of the DD estimates for each state of the change in amenable mortality, from regressions otherwise similar to those used for Table 2, versus *ln*(state population in 2012). We also superimpose a regression line showing the best linear fit between the point estimates and *ln*(population).

It is apparent from Figure 9 that for single states, it is common to find pseudo-treatment effects of 2% or more, with a fair number of states showing pseudo-effects of 4% or more, and Montana and Mississippi showing pseudo-effects around 6%. There is also a tendency for larger states to have better mortality trends than smaller states over 2012-2013, shown by the negative slope of the best-fit line.

[Figure 9 around here]

D. Power for Vulnerable Subgroups

We also conducted power analyses for the subsamples considered in Tables 3 and 4, and report results in the Appendix. Power is generally similar to, or lower than, that shown in Figure 7. Smaller sample size, which reduces power, offsets the effect of the modestly larger first stages, which are all we can achieve. And the effect of non-parallel trends, in reducing power, remains.

E. What Data Would Be Needed for Reasonable Power?

We turn in this section to a different question – what combination of a stronger first stage and a reduction in amenable mortality for the newly insured would be detectable with reasonable power, if we could use a richer dataset, with data on mortality linked to data on income and family status (to determine eligibility for expanded Medicaid coverage) and pre-ACA insurance status (to exclude the always-insured from the sample). This hypothetical data would improve the first stage and bring it toward (or even above) the 5% one could obtain by studying only adults with incomes < 138% of FPL, or the 10% in Simon et al. (2017) for childless adults with incomes < 100% of FPLs. We consider our preferred triple-difference design.

In this scenario, we imagine that we have can identify in each county both a treated subsample and a similar control subsample, both aged 55-64. For example, if the treated subsample is childless adults with income < 138% of FPL, the within county control subsample could be childless adults with incomes from 138% to 250% of FPL. We assume hypothetical first stages varying from 1% to 15% and hypothetical second stages varying from 0% to 10%. For, say, a 5% first stage and a 10% second stage, we assign "insurance due to Medicaid expansion" to 5% of the persons in a "5% first stage" subsample of each expansion county, and then remove 10% of the amenable mortality deaths from the treated persons in this subsample (thus applying an overall mortality reduction to the subsample of .005). We again use data from 2007-2013 and a pseudo-treatment at Jan. 1, 2012, and assess whether we could detect this mortality effect if we did not know which specific individuals within this subsample would have gained insurance due to this pseudo-treatment. Since the treated and control samples are drawn at random from the same county and age range, they have the same expected mortality rates, by construction.[39]

We assume that with the hypothetical data, (i) researchers can identify the subsample members, and (ii) *all* effects of Medicaid expansion on uninsurance rates are concentrated in the subsample we consider. Thus, in our 5% first stage/10% second stage example, we assume that the entire Medicaid-expansion-related relative drop in uninsurance –170,000 persons in Full-

[39] For small subsamples, there are many county-years with zero deaths in smaller counties. The log transform we use (y_{it} = *ln*((deaths)/100,000 persons)+1) can produce substantial bias when there are many zero-death observations but most non-zero death rates are large (because we multiply the fractional rate by 100,000), which can lead to underestimating statistical power. We therefore use a linear model in conducting power analysis for specifications that examine small sub-groups, and thus have many county-level observations with zero deaths.

Expansion States -- comes from this subsample. This defines the subsample size at 170,000/.05 = 3.4M treated persons, and a similar number of controls.

In Figure 10, we show power curves only for the 95% significance level. We vary (i) the assumed first stage (we show curves for 1%, 3%, 5%, 10%, 15%, and 20% first stages) and (ii) the imposed mortality reduction for the newly insured (from 0% to 10%) for the 5% significance level. With this hypothetical richer data, we need a smaller number of avoided deaths to be able to reliably detect a treatment effect. For example, with a 10% first stage, we could reliably detect mortality reductions of 2.6% or more in this subsample, or around 1,693 annual deaths. This is only slightly below the number of deaths we could detect in the full sample; thus, this hypothetical study remains severely underpowered. Recall that with a 10% second stage, we expect around 100 fewer annual deaths among those who actually gain insurance.

F. Implications of Power Analysis for Other Studies

While our exact simulation approach for understanding the minimum detectable effect is specific to our dataset and research design, a similar approach can be used in many other studies. We offer here four examples of why we believe power analyses such as ours, including an assessment of the minimum detectable effect and whether that effect size is plausible, can be broadly valuable in shock-based research.

First, our power analysis can be usefully compared to the results in Finkelstein et al. (2012), who study the Oregon Health Insurance Experiment. With a sample of 75,000 people and a roughly 30% first stage among people who took the trouble to sign up for the Oregon Medicaid lottery, who were randomly offered Medicaid or assigned to control, they found a large point estimate for the near-term effect of receiving Medicaid on mortality of around 13%, but a *t*-statistic only around 0.5. This implies that their study was undersized, even for that large point estimate, by a factor of around 16 – they would need a sample of 1.2M people to reliably find a 13% effect – and a sample of 8M people to find a 5% effect.

Second, our analysis of power to detect the effect of health insurance on non-elderly adult mortality has direct implications for prior DD studies of the effect of insurance expansions on adult mortality. We provide a back of the envelope calculation here, for example for SLB (2014), who report a statistically significant near-term decline in adult mortality following the "Romneycare" health insurance expansion in Massachusetts in 2006. Massachusetts has a moderate sized population (6.55M in 2017; 14th among all states). Kolstad and Kowalski (2012) find a first stage

insurance gain of 5.6%. The DD effect estimate in SLB – a 4.5% drop in amenable mortality by two years after reform –implies an extremely large 80% drop in amenable mortality for compliers.

To assess power, we build on Kaestner's (2016) replication of SLB (2014), in which he finds that their results are insignificant, using randomization inference to estimate confidence intervals.[40] We used Kaestner's code to compute the minimum effect size in their analysis with $p < .05$ (95% confidence). This minimum effect is 6.9%. The minimum detectable mortality decline for the newly insured, implied by this minimum effect size, is 6.9%/5.6% = 123%.

In two more examples, we turn to recent work by two of us, in separate projects. Soni et al. (2018a) report that Medicaid expansion predicts a 2.4% relative drop in the fraction of people with cancer who are uninsured. They cannot measure the drop in uninsurance among those with undiagnosed cancer, whose baseline uninisurance rate is likely higher. Soni et al. (2018b) report a 6.4% increase in diagnoses of early-stage cancer, but do not discuss plausible effect sizes or minimum detectable effects. What first stage would be needed among those with undiagnosed cancer to make a 6.4% increase in early diagnoses plausible? Meanwhile, a back of the envelope calculation using their reported 95% CI suggests a standard error ~2% which implies a minimum detectable effect ~4%.

Pines et al. (2016) find no evidence that Medicaid expansion predicts a significant increase in ED visits; their point estimate is a 0.6% drop in expansion states, relative to non-expansion states. They do not discuss the first stage (the relative drop in ED visits by uninsured persons), but from their Appendix, one can determine that the first stage is around 6.7%. Twice their standard error is .018, and .018/.067 = 0.27. This implies that if the only reason for change in ED visit rates were gaining insurance, the 95% CI around their point estimate implies a [-36%, +18%] change in ED visits by the newly insured. There is still no evidence of a higher visit rate by the newly insured, and the upper end of the 95% CI is still well below the +40% point estimate from the Oregon Health Insurance Experiment, but it one cannot rule out a fairly large increase in ED visits by the newly insured.

X. Discussion

In this paper, we examine the relationship between mortality and health insurance, principally using the DD research design used in many prior ACA studies. This design exploits

[40] We thank Robert Kaestner for providing his Stata code, which we used in our analysis.

the natural experiment created by variation between those states that expanded Medicaid insurance and those that did not. We also exploit variation that results from counties having varying uninsurance or poverty levels prior to 2014. We focus on persons aged 55-64 years, whose mortality rates are the most likely to be affected by health insurance. We study effects of the first two years after expansion by type of mortality (healthcare amenable vs non amenable), demographics (gender, race, and ethnicity), education level, cause of death, and residence in counties most likely to gain from the ACA expansion).

We find no convincing evidence of an ACA-induced decline in mortality in Medicaid expansion states. Instead, results are mixed; there are often non-parallel pre-treatment trends, and standard errors are far too large to allow detection of effects of plausible sizes. We confirm lack of power through a formal, simulation-based power analysis.

While it is possible that effects could materialize with more time, lengthening the study period would increase likelihood that other sources of variation, including cross-border moves, the instability of insurance status over time, and the underlying causes of the non-parallel pre-treatment trends we observe, will pose challenges for credible causal inference. Moreover, our power analysis implies that an extra year or three would still be insufficient to attain reasonable power, given plausible effect sizes.

We end with a discussion of the data needed to push forward the literature on the health outcome effects of health insurance. Large-scale data sets that include individual-level data on income insurance, and health status (aside from mortality) are essential. Income and prior insurance information would permit a substantially larger first stage. Health data would provide a more sensitive second stage, and might also permit analysis limited to health-vulnerable subpopulations, provided that these subpopulations still provide reasonable sample sizes. At the same time, given the power concerns we identify, studies of the health effects of health insurance should include efforts to assess the first stage, estimate reasonable magnitudes for treatment effects, and conduct a power analysis. These steps should improve researchers' ability to assess the plausibility of reported results in two senses –they should prevent apparently significant results from arising by chance (the usual meaning of a "false positive") and make it less likely that researchers will report estimates many times larger than true effects.

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Table 1. Full Expansion; Substantial Expansion; Mild Expansion, and No-Expansion states, and % Uninsured for Selected Years

Table shows expansion status of each state (including D.C.). For Full Expansion states and No-Expansion states, table shows expansion date if other than Jan. 1, 2014. For "substantial" and "mild" expansion states, table shows year of significant prior Medicaid expansion. Summary rows give either equal weight to all states in each expansion group, or population weight, as indicated. See Appendix Table A-1 for additional details and sources for each state's expansion status.

State	Expansion Date	% uninsured (age 50-64)			change in % unins.
		2013	2014	2015	(2013-2015)
Full Expansion		**13.4**	**9.4**	**7.0**	**-6.4**
Pop. weighted		**12.9**	**9.3**	**6.8**	**-6.1**
Arizona[1]		17.6	13.1	9.8	-7.8
Arkansas[2]		16.5	11.3	8.7	-7.8
Colorado[3]		13.6	9.2	7.2	-6.4
Illinois		14	9.8	6.6	-7.4
Indiana	Feb 2015	12.9	11	8.9	-4.0
Iowa[4]		7.7	5.9	4.3	-3.4
Kentucky		14.4	7.5	5.3	-9.1
Maryland		10	7.2	5.1	-4.9
Michigan	Apr 2014	11.4	8.2	5.7	-5.7
Nevada		19.8	14.3	10.9	-8.9
New Hampshire	Aug 2014	11.6	9.7	6.8	-4.8
New Jersey[5]		13.1	10.8	8	-5.1
New Mexico		19	15	11.3	-7.7
North Dakota		9.6	6.9	5.9	-3.7
Ohio		12.3	8.3	6.4	-5.9
Oregon[6]		15.3	9.6	6.7	-8.6
Pennsylvania	Jan 2015	9.5	7.7	5.7	-3.8
Rhode Island		11.2	6.2	4.5	-6.7
Washington[5]		13.1	8.2	6.2	-6.9
West Virginia		14.5	8.9	5.9	-8.6
Substantial Expansion		**10.3**	**7.1**	**5.2**	**-5.1**
Pop. weighted		**15.0**	**10.3**	**7.0**	**-8.0**
California[5]	2010	18.1	12.3	8.2	-9.9
Connecticut[5]	2010	9.7	6.2	5	-4.7
Hawaii[7]	1994	7.3	5.5	4.4	-2.9
Minnesota[5]	2010	7.3	5.1	3.6	-3.7
Wisconsin[8]	2009	9.1	6.6	5	-4.1
Mild Expansion		**7.6**	**5.7**	**4.4**	**-3.2**
Pop. weighted		**8.6**	**6.8**	**5.3**	**-3.2**
Delaware[9]	1996	9.5	7.2	5.5	-4.0
Dist. of Columbia[5]	2010	6.7	5.2	3.5	-3.2
Massachusetts[10]	2006	3.5	3	2.6	-0.9
New York[11]	2001	10.4	8.2	6.4	-4.0
Vermont[12]	1996	7.9	5	4.1	-3.8
No Expansion		**15.7**	**13.1**	**11.3**	**-4.4**
Pop. weighted		**17.6**	**14.6**	**12.4**	**-5.2**
Alabama		13.4	11.6	10.2	-3.2
Alaska	Sep 2015	19.1	17.2	15.2	-3.9

State	Expansion Date	% uninsured (age 50-64)			change in % unins.
		2013	2014	2015	(2013-2015)
Florida		22	17.9	14.1	-7.9
Georgia		18.2	15.2	13.1	-5.1
Idaho		17.1	12.9	11.3	-5.8
Kansas		11.8	9.7	8.2	-3.6
Louisiana	Jul 2016	17.8	15.6	12.1	-5.7
Maine		12.5	11	8.5	-4.0
Mississippi		18.8	15.4	13.1	-5.7
Missouri		13.3	10.3	9.1	-4.2
Montana	Jan 2016	18	14.4	13.2	-4.8
Nebraska		10.7	8.8	6.9	-3.8
North Carolina		15.8	12.6	10.8	-5.0
Oklahoma		18.1	15.6	14.1	-4.0
South Carolina		17.1	13.7	11.5	-5.6
South Dakota		11.3	9.3	9.3	-2.0
Tennessee		15	12.7	10.8	-4.2
Texas		21	17.4	15.5	-5.5
Utah		13	11.3	9.8	-3.2
Virginia		12.3	10.8	8.8	-3.5
Wyoming		13.3	12.7	10.9	-2.4
National		**13.5**	**10.4**	**8.3**	**-5.1**
Pop. weighted		**14.6**	**11.2**	**8.8**	**-5.8**

Table 2: DD and Triple-Difference Estimates: Effect of Medicaid Expansion on Mortality

County-level regressions, with county and year FE and population weights, of *ln*((mortality/100,000 persons)+1) over 2009-2015 on full-Expansion dummy (=1 for Full-Expansion States in expansion years; 0 otherwise), and covariates (same as in Figure 2, used in even-numbered regressions. Third difference (regressions (5)-(6)) is ages 55-64 versus aged 65-74. Standard errors use state clusters. *.**, *** indicates statistical significance at the 10%, 5%, and 1% levels, respectively; significant results at 5% level or better in **boldface**.

	DD 55-64 years		**DD 65-74 years**		**Triple diff.**	
	(1)	(2)	(3)	(4)	(5)	(6)
Healthcare Amenable Mortality						
Full Expansion Dummy	**-0.021****	**-0.021*****	-0.010	-0.004	**-0.012****	-0.003
	(0.010)	**(0.007)**	(0.007)	(0.005)	**(0.006)**	(0.006)
Full Expansion Dummy x Age 55-64 Dummy					-0.007	-0.007
					(0.010)	(0.009)
Non-amenable Mortality						
Full Expansion Dummy	0.007	0.002	**0.022****	**0.021****	0.019*	**0.026*****
	(0.011)	(0.010)	**(0.011)**	**(0.010)**	(0.010)	**(0.009)**
Full Expansion Dummy x Age 55-64 Dummy					-0.011	-0.013
					(0.013)	(0.012)
All Mortality						
Full Expansion Dummy	-0.012	**-0.014****	-0.002	0.002	-0.004	0.004
	(0.009)	**(0.006)**	(0.005)	(0.004)	(0.005)	(0.005)
Full Expansion Dummy x Age 55-64 Dummy					-0.006	-0.007
					(0.009)	(0.008)
County Population Weights	Yes	Yes	Yes	Yes	Yes	Yes
Year and County FE	Yes	Yes	Yes	Yes	Yes	Yes
Covariates	No	Yes	No	Yes	No	Yes
Observations	19,656	19,656	19,656	19,656	39,312	39,312

Table 3: DD and Triple-Difference Estimates: Different Demographic Groups (ages 55-64)

First column shows annual averages over 2009-2015 for number of deaths and population in millions. Of the full sample (28.5M people), 14.1M were in expansion states. Second column shows mortality rate for persons aged 55-64 for indicated groups. Third column shows first-stage DD estimates of change in uninsurance rates (in percent) from 2013 to 2015 for indicated demographic subsamples, for persons aged 50-64, from regression of percent uninsurance on Full Expansion dummy, with state and year FE and county population weights, using state-level SAHIE data (best available), and same covariates as the DD and triple difference regressions. Remaining columns show coefficients from DD or triple difference regressions on Full-Expansion dummy or, for triple difference column, full-expansion dummy * age 55-64 dummy, from county-level regressions with county-and year FE and population weights, similar to Table 2, for *ln*((amenable mortality/100,000 persons)+1) over 2009-2015. Standard errors use state clusters. *.**, *** indicates statistical significance at the 10%, 5%, and 1% levels, respectively; significant results at 5% level or better in **boldface**.

Demographic Subsamples	**Ann. Deaths (Pop. in M)** **(1)**	**Mortality rate** **(2)**	**First stage (%) 50-64 yrs** **(3)**	**DiD 55-64 yrs** **(4)**	**DiD 65-74 yrs** **(5)**	**Triple diff.** **(6)**
All Amenable	172,598	604.69	**1.174****	**-0.021*****	-0.004	-0.007
	(28.5)		**(0.487)**	**(0.007)**	(0.005)	(0.009)
Male	104,534	759.98	0.738	-0.017*	-0.007	0.003
	(13.7)		(0.498)	(0.009)	(0.007)	(0.011)
Female	68,063	460.26	**1.078****	**-0.028*****	-0.004	-0.012
	(14.8)		**(0.519)**	**(0.009)**	(0.009)	(0.012)
White (Not Hispanic)	128,511	592.40	**0.938****	**-0.015****	-0.007	-0.007
	(21.7)		**(0.440)**	**(0.007)**	(0.006)	(0.009)
Black (Not Hispanic)	31,793	931.06	1.424	**-0.038****	0.021	**-0.056*****
	(3.4)		(0.817)	**(0.017)**	(0.014)	**(0.019)**
Other	3,527	269.61	-	-0.099	-0.010	-0.081
	(1.3)		-	(0.065)	(0.048)	(0.087)
Hispanic	8,848	397.78	2.444	**-0.162*****	-0.068	**-0.072****
	(2.2)		(1.344)	**(0.056)**	(0.049)	**(0.034)**
Not Hispanic	163,750	622.18	-	**-0.021*****	-0.003	-0.008
	(26.3)		-	**(0.007)**	(0.005)	(0.008)
Pop. Weights			Yes	Yes	Yes	Yes
Covariates			Yes	Yes	Yes	Yes

Table 4: DD and Triple-Difference Estimates: by Educational Attainment (ages 45-64)

First column shows annual averages over 2009-2015 for number of deaths and population in millions. Second column shows mortality rate for persons aged 55-64 for indicated groups. Third column shows first-stage DD estimates of change in uninsurance rates (in percent) from 2013 to 2015 for indicated education-levels, for persons aged 45-64, from regression of percent uninsurance on Full Expansion dummy, with county and year FE and county population weights. Remaining columns show coefficients from DD or triple difference regressions on Full-Expansion dummy or, for triple difference column, full-expansion dummy * age 45-64 dummy, from county-level regressions with county and year FE and population weights, similar to Table 2, for *ln*((amenable mortality/100,000 persons)+1) among persons with indicated education levels, over 2009-2015. Standard errors use state clusters. *.**, *** indicates statistical significance at the 10%, 5%, and 1% levels, respectively; significant results at 5% level or better in **boldface**.

Education Subsample	**Ann. Deaths (Pop. in M)** **(1)**	**Mortality Rate** **(2)**	**First stage (%) 45-64 yrs** **(3)**	**DiD 45-64 yrs** **(4)**	**DiD 65+ yrs** **(5)**	**Triple diff.** **(6)**
All Amenable	251,302	422.29	**1.109****	-0.012	**-0.016*****	0.011
	(59.5)		**(0.546)**	(0.008)	**(0.006)**	(0.010)
Elementary School	14,921	571.81	**4.269****	0.057	0.008	0.058
	(2.6)		**(2.146)**	(0.051)	(0.058)	(0.046)
High School Incomplete	33,490	761.29	**3.792****	-0.022	-0.020	-0.008
	(4.4)		**(1.614)**	(0.062)	(0.063)	(0.037)
High School Complete	109,260	604.11	**1.617****	-0.025	-0.034	0.010
	(18.1)		**(0.749)**	(0.042)	(0.039)	(0.016)
Some College	86,379	251.00	0.493	-0.016	-0.021	0.014
	(34.4)		(0.468)	(0.035)	(0.030)	(0.013)
Population Weights			Yes	Yes	Yes	Yes
Covariates			Yes	Yes	Yes	Yes

Table 5: DD and Triple-Difference Estimates: by Cause of Death (age 55-64)

First column shows annual averages over 2009-2015 for number of deaths and population in millions. Second column shows mortality rate for persons aged 55-64 for indicated groups. Remaining columns show coefficients from DD or triple difference regressions on Full-Expansion dummy or, for triple difference column, full-expansion dummy * age 45-64 dummy, from county-level regressions with county and year FE and population weights, similar to Table 2, for *ln*((amenable mortality/100,000 persons)+1) among persons with indicated primary cause of death, over 2009-2015. Standard errors use state clusters. *.**, *** indicates statistical significance at the 10%, 5%, and 1% levels, respectively; significant results at 5% level or better in **boldface**.

By Cause of Death	**deaths (pop. In M)**	**DiD 55-64 yrs**	**DiD 65-74 yrs**	**Triple diff.**
	(1)	(2)	(3)	(4)
All Amenable	172,598	**-0.021*** **	-0.004	-0.007
	(28.5)	**(0.007)**	(0.005)	(0.009)
Cancer	86,733	-0.002	0.006	-0.004
	(28.5)	(0.007)	(0.006)	(0.009)
Diabetes	14,186	-0.036	0.014	-0.022
	(28.5)	(0.025)	(0.030)	(0.024)
Cardiovascular	69,718	-0.019	-0.004	-0.001
	(28.5)	(0.011)	(0.010)	(0.012)
Respiratory	16,129	-0.029	-0.020	-0.011
	(28.5)	(0.020)	(0.014)	(0.021)
HIV	1,279	-0.038	0.015	-0.050
	(28,5)	(0.037)	(0.038)	(0.060)
Pop. Weights		Yes	Yes	Yes
Covariates		Yes	Yes	Yes

Table 6: Triple Difference Estimates: Separating Counties by Baseline Health Uninsurance or Poverty Levels (age 55-64)

First column shows annual averages over 2009-2015 for number of deaths and population aged 55-64 in millions, for sample of high-versus low- uninsurance counties. Second and fourth columns column shows full-sample and by gender first stages; we lack the data to compute first stages for the other subsamples. Remaining columns show coefficients from triple difference, county-level regressions with county and year FE and population weights, similar to Table 2, over 2009-2015, for full sample and indicated demographic subsamples. Third difference in column (3) is between the counties with the highest uninsurance rate in 2013, containing 20% of the U.S. population, and the counties with the lowest uninsurance rate in 2013, containing 20% of the U.S. population. Third difference in column (5) is similar but is between the counties with lowest versus highest poverty rates in 2013. Standard errors use state clusters. *.**, *** indicates statistical significance at the 10%, 5%, and 1% levels, respectively; significant results at 5% level or better in **boldface**.

	(1)	(2)	(3)	(4)	(5)
Demographic Subsamples	**Deaths (pop. in M)**	**First Stage**	**Triple diff. Uninsurance**	**First Stage**	**Triple diff. Poverty**
All Amenable	65,642	**2.338*** **	0.025	**1.623** **	-0.010
	(11.8)	**(0.586)**	(0.024)	**(0.736)**	(0.019)
Male	40,387	**1.511*** **	-0.018	**1.380** **	**-0.044** **
	(5.7)	**(0.586)**	(0.036)	**(0.660)**	**(0.022)**
Female	25,770	**2.769*** **	**0.070** **	**1.809** **	0.030
	(6.1)	**(0.626)**	**(0.030)**	**(0.803)**	(0.027)
White (Not Hispanic)	50,852		-0.009		-0.028
	(9.0)		(0.032)		(0.020)
Black (Not Hispanic)	11,783		-0.048		0.017
	(1.4)		(0.063)		(0.050)
Other	1,504		-0.167		-0.043
	(0.5)		(0.165)		(0.152)
Hispanic	3,331		0.689		0.028
	(0.9)		(0.595)		(0.112)
Not Hispanic	60,321		0.020		-0.018
	(10.3)		(0.024)		(0.019)

Figure 1. Time Trends in Amenable Mortality for Persons Aged 55-64

Figure shows amenable mortality rate for persons age 55-64 for Full-Expansion, Substantial Expansion, Mild Expansion, and Non-Expansion States, over 1999-2014, using county population weights. State groups are defined in Table 1. Vertical line separate pre-expansion from expansion period.

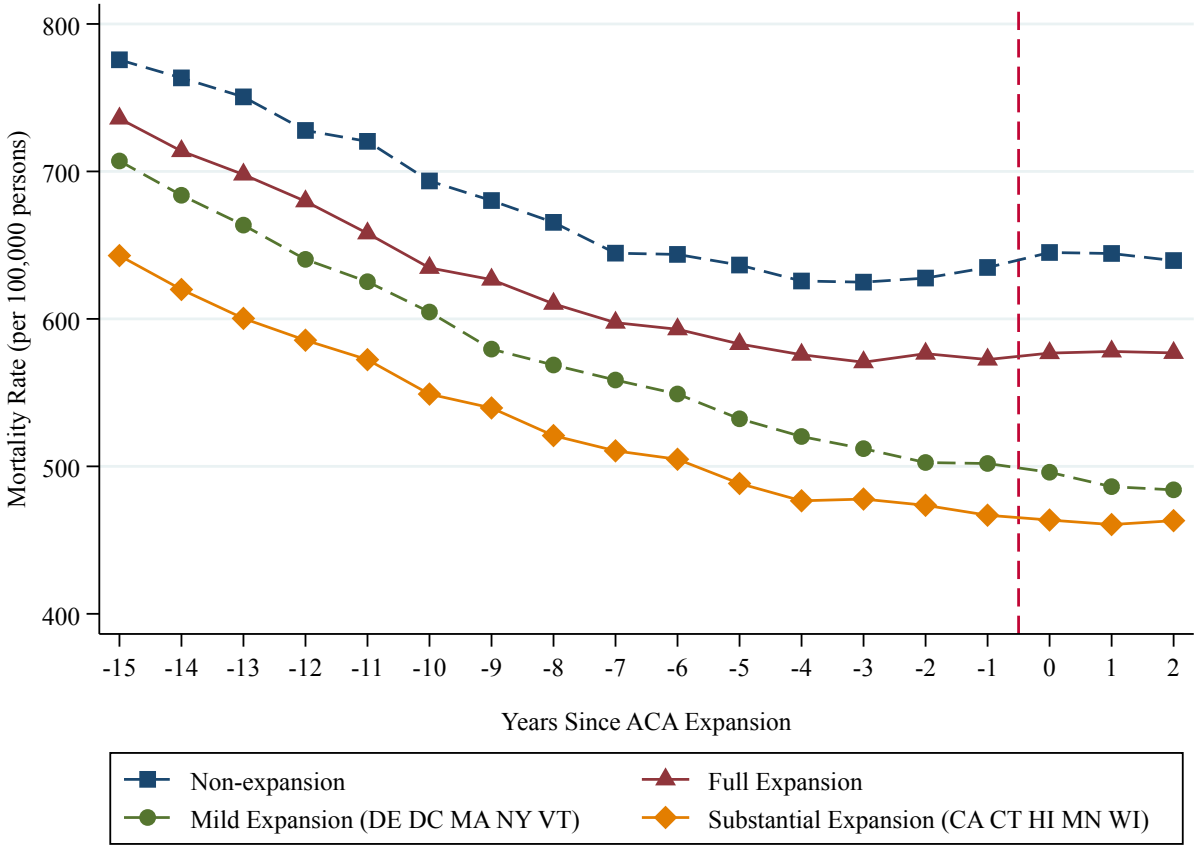
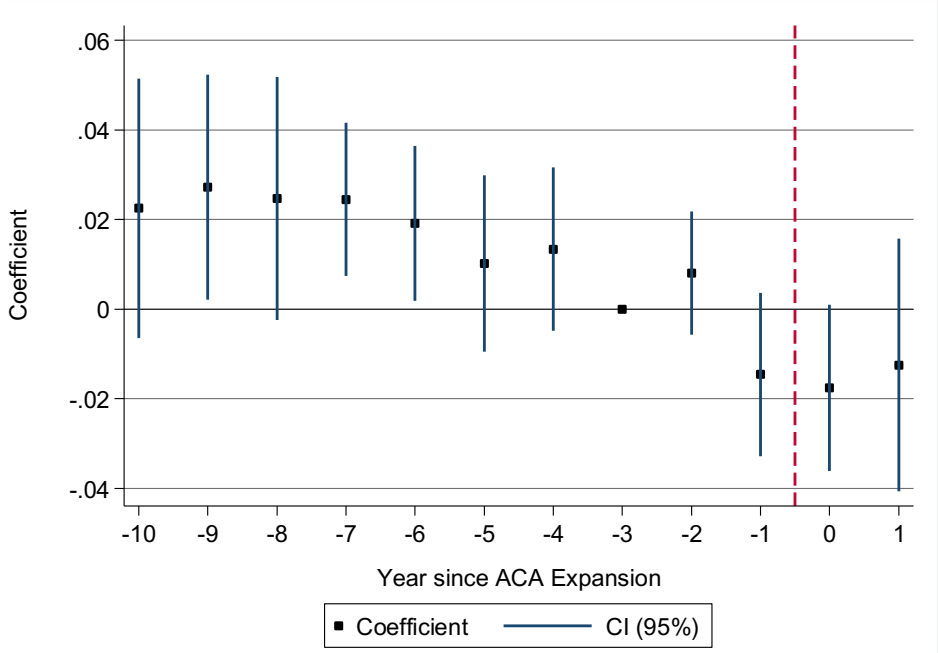


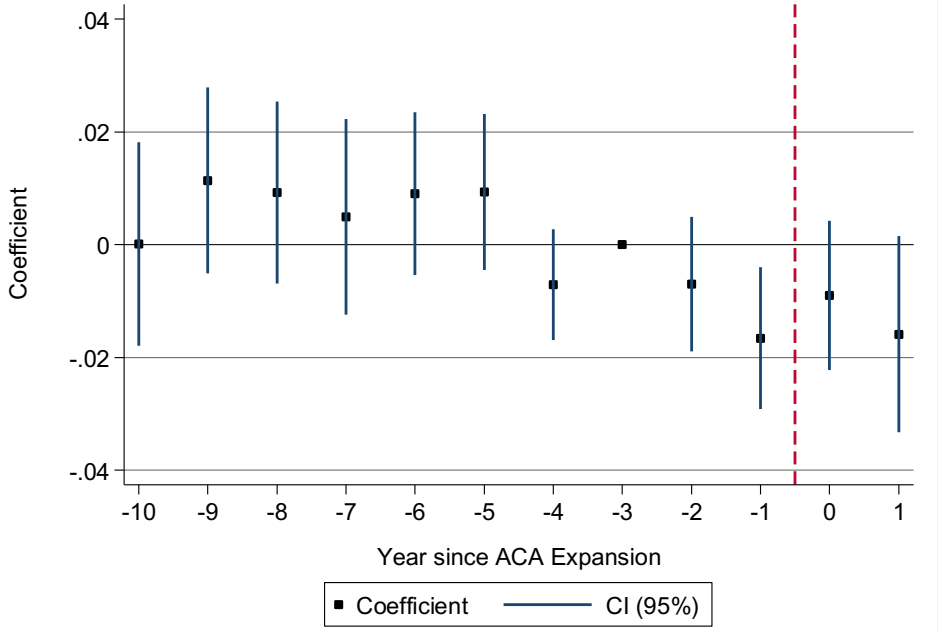
Figure 2. Leads-and-Lags Results for Ages 55-64 and 65-74, Amenable Mortality

Graphs from leads and lags regressions of *ln*((amenable mortality/100,000 persons)+1) for Full-Expansion States versus control group of Non-Expansion States, over 2004-2015. Covariates are listed in paper. Regressions include county and year FE, and county-population weights. y-axis shows coefficients on lead and lag dummies; vertical bars show 95% confidence intervals (CIs) around coefficients, using standard errors clustered on state. Coefficient for year -3 is set to zero.

Panel A. Amenable Mortality for Ages 55-64



Panel B. Amenable Mortality for Ages 65-74



Panel C. Triple difference. Leads and lags graphs for amenable mortality for persons age 55-64 in Full-Expansion States, relative to (i) persons age 65-74 in Full-Expansion States, and (ii) persons age 55-64 in Non-Expansion States.

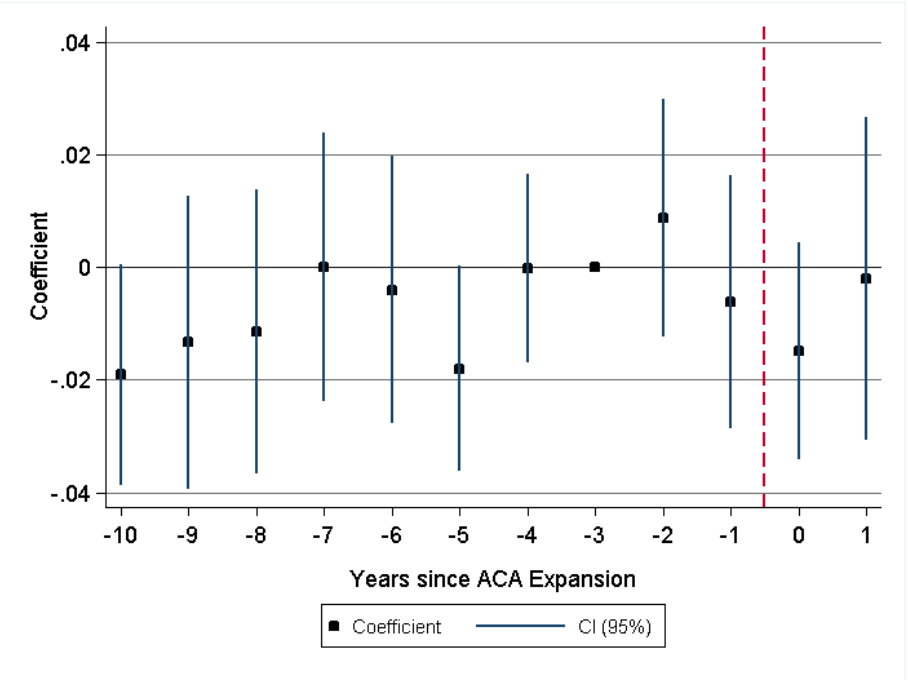


Figure 3. Synthetic Control Results for Near-Elderly Amenable Mortality

Synthetic control results for *ln*((amenable mortality/100,000 persons)+1) for Full-Expansion States (treated as a single treated unit) versus synthetic control drawn from Non-Expansion States, over 1999-2015. Covariates for constructing donor pool are same as in Figure 2, plus uninsurance rate in 2013. The y-axis shows *ln*((amenable mortality/100,000 persons)+1) for Full-Expansion States, combined into single treated unit (using population weights), and their synthetic control. Vertical dotted line separates pre-expansion from expansion period.

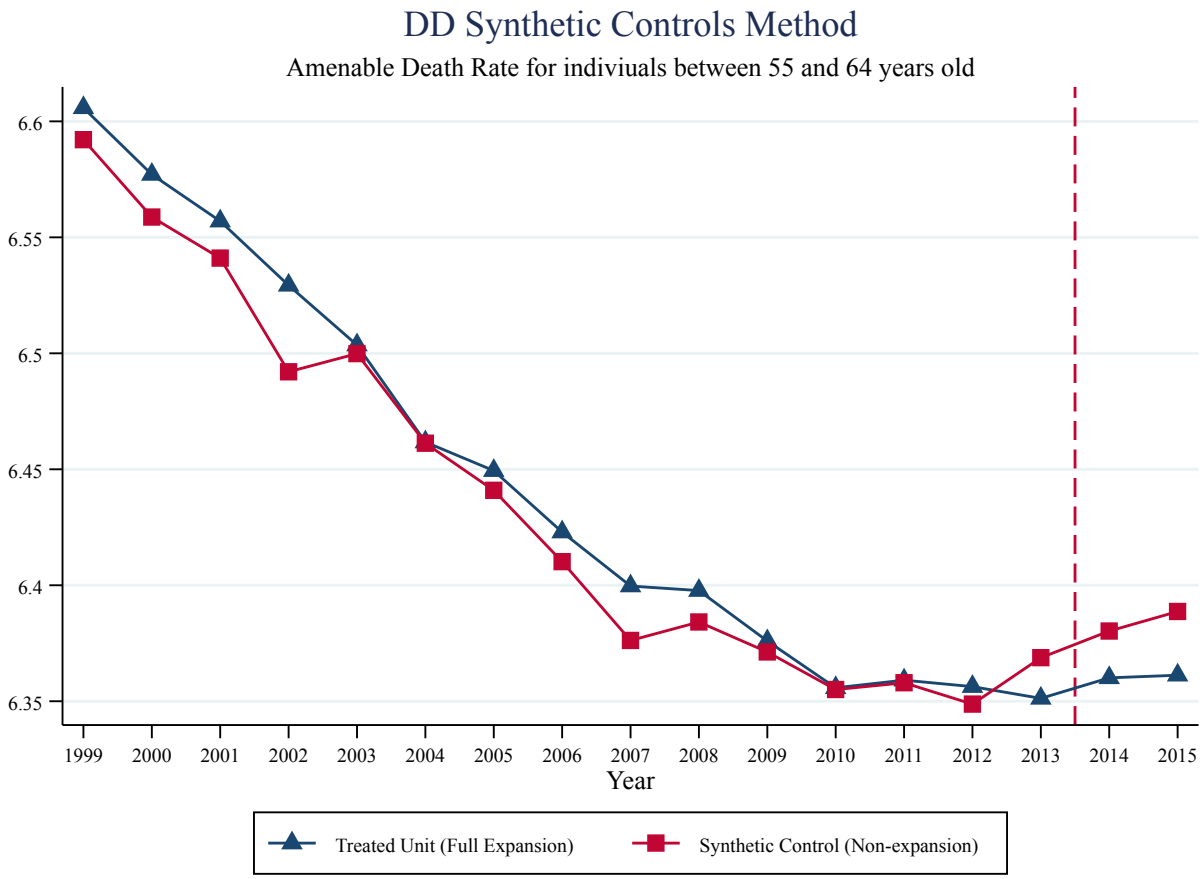


Figure 4. Triple Difference Leads-and-Lags Graphs: Demographic Groups

Graphs from leads and lags regressions of triple differences for indicated subsamples, of *ln*((amenable mortality/100,000 persons)+1) for persons aged 55-74, in Full-Expansion States versus No-Expansion States, over 2004-2015; the third difference is age 55-64 versus age 65-74. Covariates are same as in Figure 2. Regressions include county and year FE, and county-population weights. y-axis shows coefficients on lead and lag dummies; vertical bars show 95% CIs around coefficients, using standard errors clustered on state. Coefficient for year -3 is set to zero.

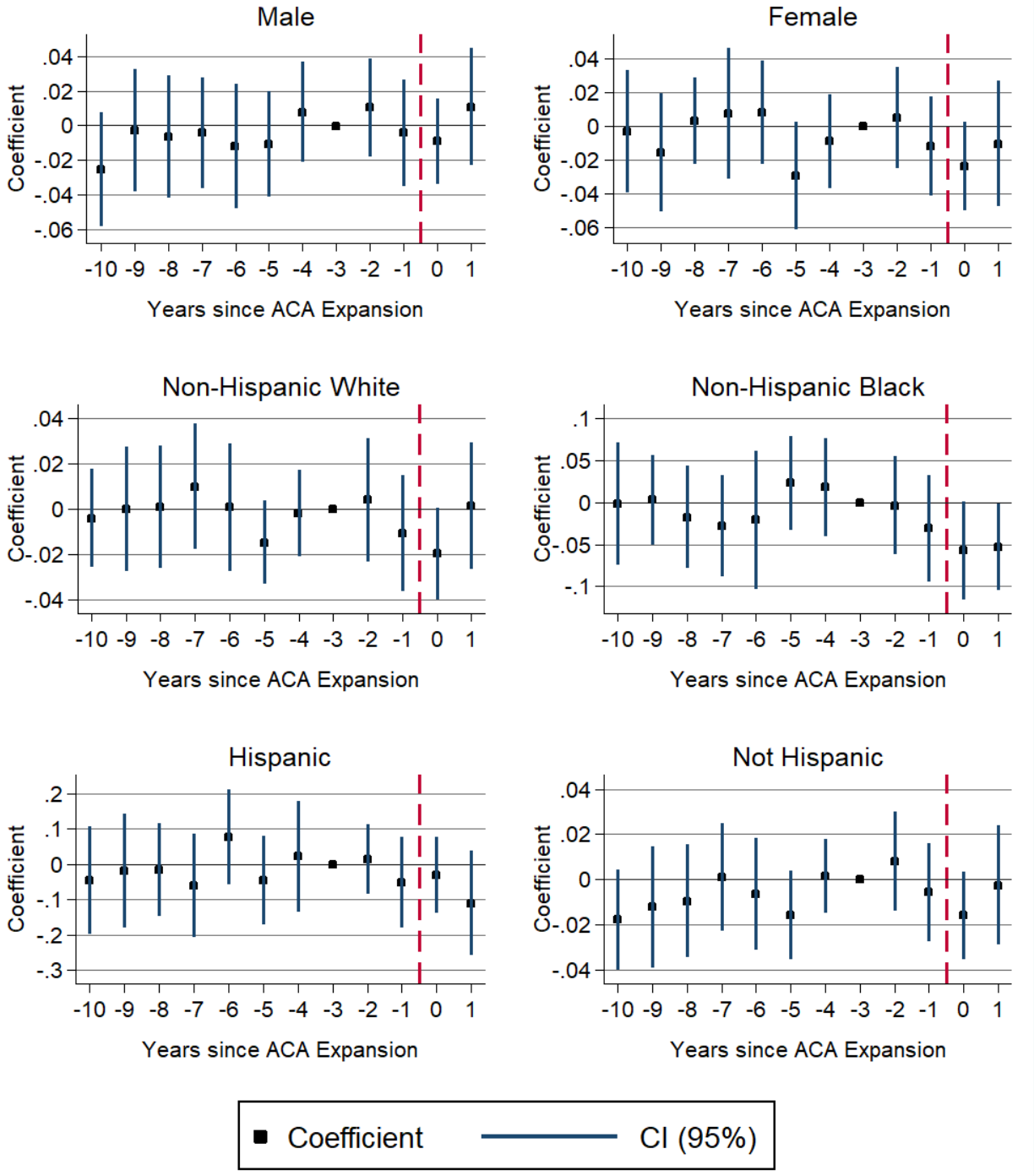


Figure 5. Triple Difference Leads-and-Lags Graphs: By Education Level

Graphs show leads and lags regressions of triple differences for indicated subsamples, of *ln*((amenable mortality/100,000 persons)+1) for persons aged 45+, in Full-Expansion States versus No-Expansion States, over 2004-2015; the third difference is age 45-64 versus age 65+. Covariates are same as in Figure 2. Regressions include county and year FE, and county-population weights. y-axis shows coefficients on lead and lag dummies; vertical bars show 95% CIs around coefficients, using standard errors clustered on state. Coefficient for year -3 is set to zero.

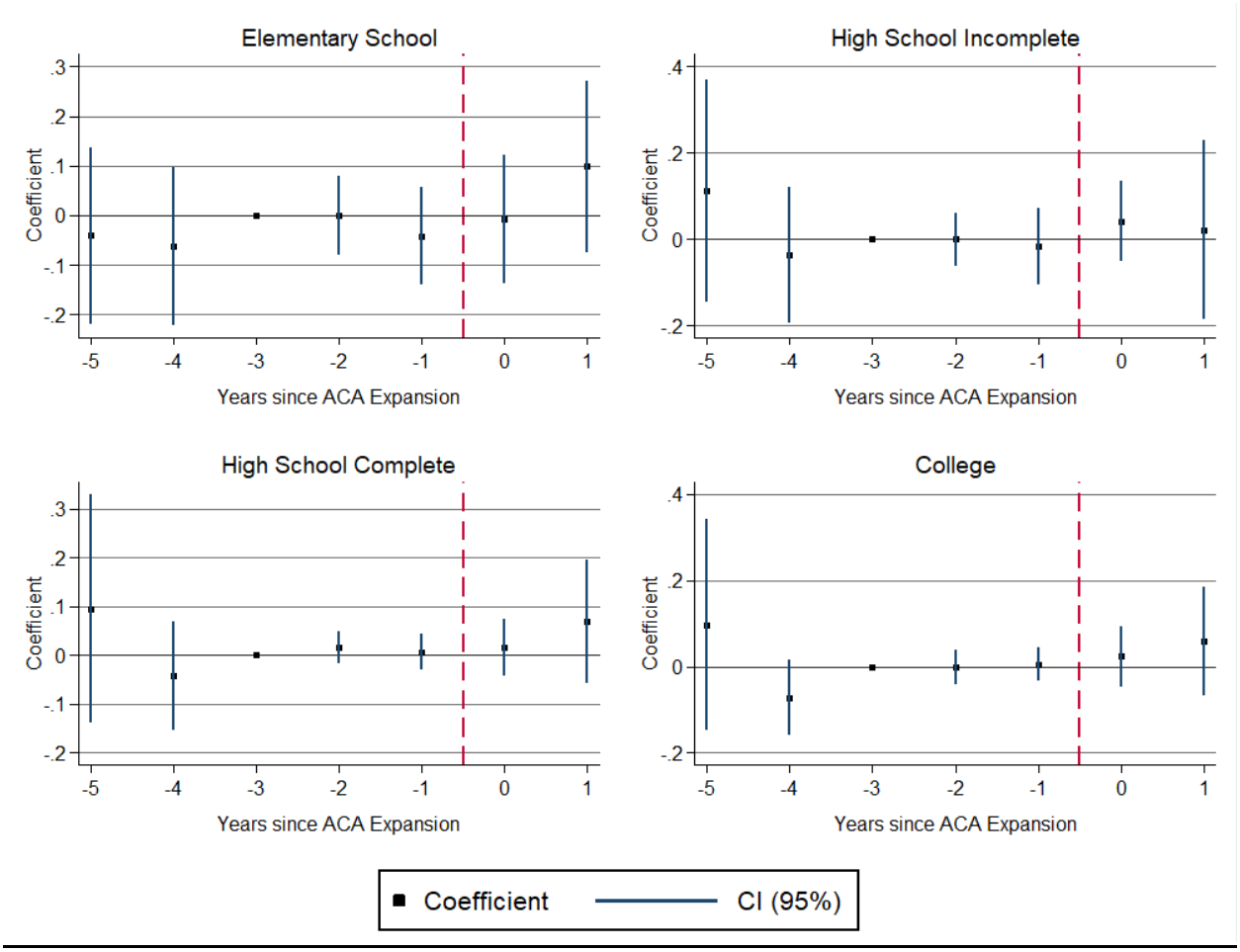
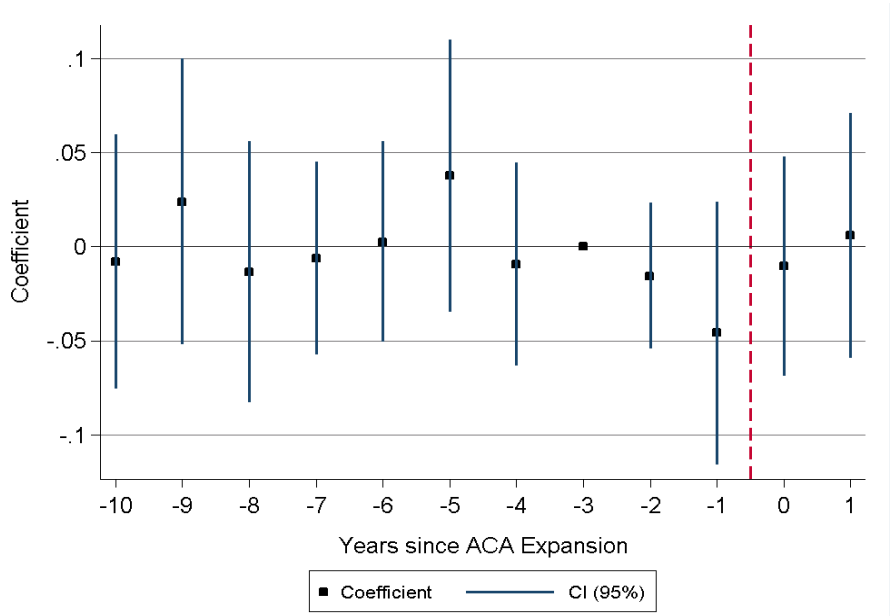


Figure 6: Leads and Lags Graphs for High-vs-Low Uninsurance and Poverty

Graphs show leads and lags regressions of triple differences for high versus low uninsurance and high vs. low poverty counties, of *ln*((amenable mortality/100,000 persons)+1+ for persons aged 55-64, in Full-Expansion States versus No-Expansion States, over 2004-2015. High (low) uninsurance counties are those with highest (lowest) uninsurance rates in 2013 containing 20% of U.S. population, and similarly for high (low) poverty counties. Covariates are same as in Figure 2. Regressions include county and year FE, and county-population weights. y-axis shows coefficients on lead and lag dummies; vertical bars show 95% CIs around coefficients, using standard errors clustered on state. Coefficient for year -3 is set to zero.

Panel A. High-Uninsurance vs. Low-Uninsurance Counties



Panel B. High-Poverty vs. Low-Poverty Counties

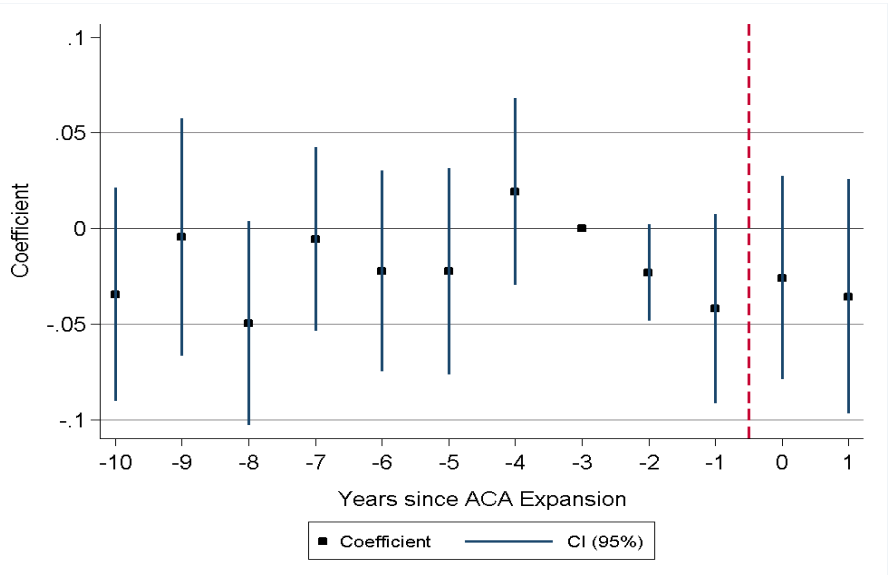
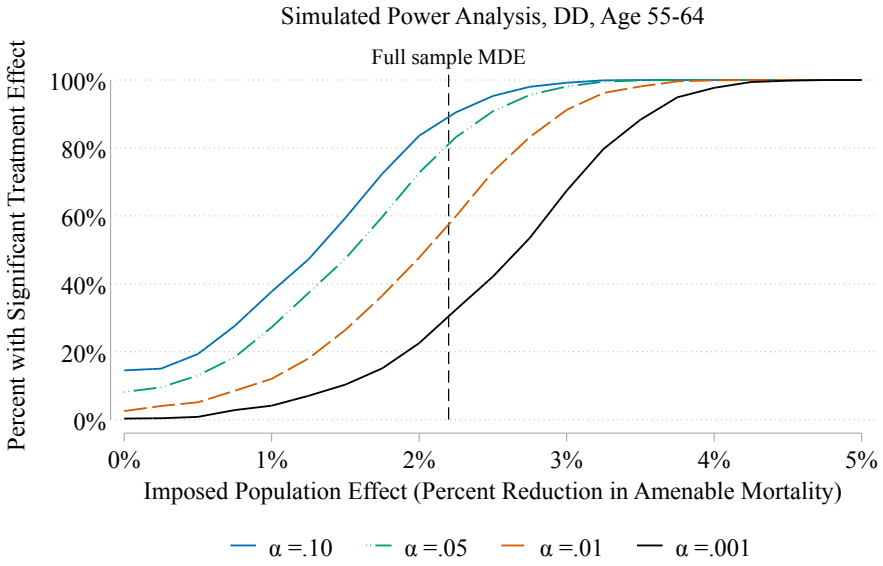


Figure 7: Simulation-Based Power Analysis

Power curves for simulated Medicaid expansion, as of January 1, 2012, applied to persons aged 55-64 during pre-treatment period (2007-2013). Graphs show power (likelihood of detecting a statistically significant effect on amenable mortality, at the indicated confidence levels, for two-tailed test), given imposed "true" population average effect. Curves are based on 1,000 replications of the DD and triple difference regressions models used in Table 2. In each draw, we select 20 pseudo-treated states at random from the combined set of 41 treated and control states, and remove a fraction of the observed deaths at random from the treated states, where the fraction removed corresponds to an assumed true treatment effect, and vary the imposed treatment effect from 0-5% in increments of 0.1%. Curves for α = .10/.05/.01/.001 correspond to 90%/95%/99%/99.9% confidence levels, respectively. Dashed vertical lines show minimum detectable effect (MDE) at 95% confidence level with 80% power.

Panel A. DD Analysis



Panel B. Triple Difference Analysis

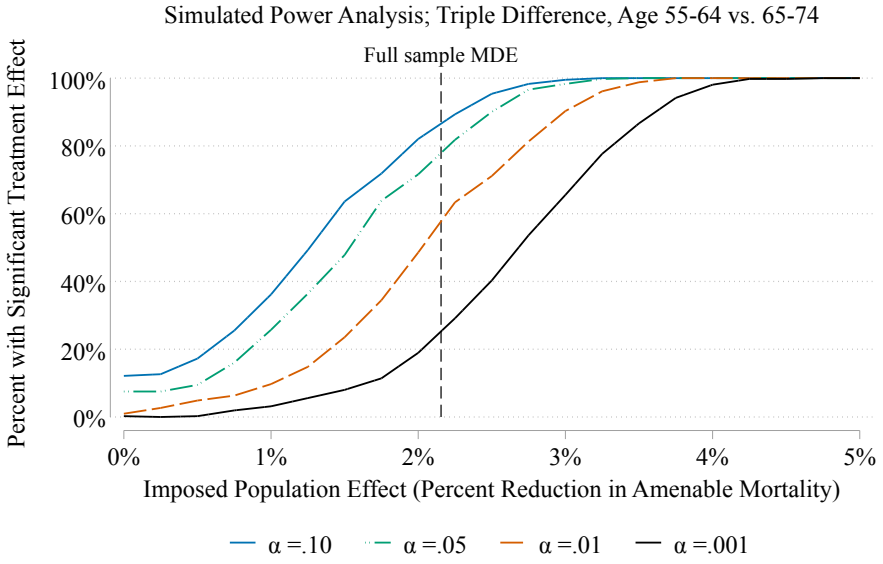
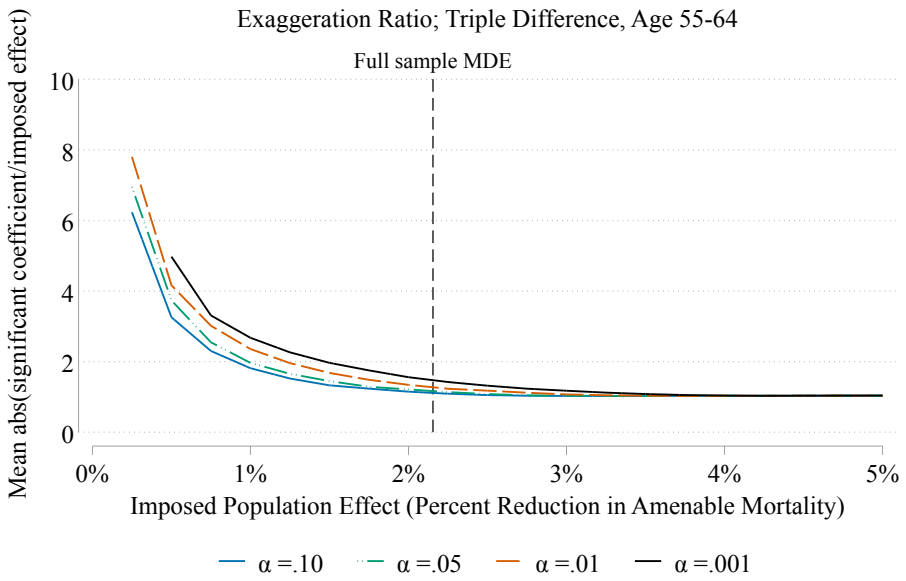


Figure 8. Power Analysis Extensions: Exaggeration Ratio and Likelihood of Wrong Sign

We conduct the same power analyses as in Figure 7 and then plot, for the instances in which a statistically significant effect is found at the indicated confidence levels, the ratio of |estimated effect|/imposed true effect ("exaggeration ratio") (Panel A), and the likelihood that the sign of the estimated effect is opposite from the imposed true effect. Curves for α = .10/.05/.01/.001 correspond to 90%/95%/99%/99.9% confidence levels, respectively. Dashed vertical lines show the minimum detectable effect size (MDE).

Panel A. Exaggeration Ratio



Panel B. Probability that Estimated Effect Has Wrong Sign

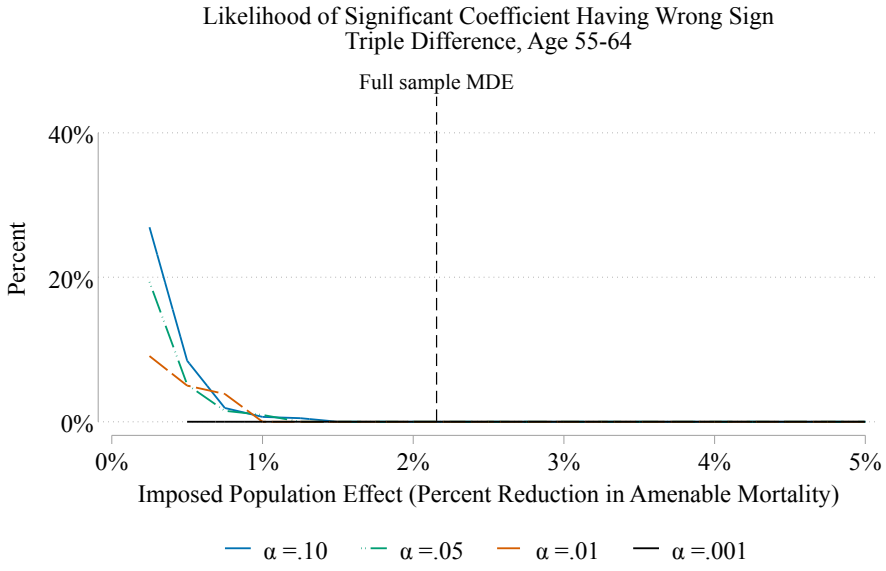


Figure 9. Pseudo-Shocks to Individual States in 2012-2013

Scatter plot of pseudo-treatment effects for individual Full-Expansion and No-Expansion states, using a sample period of 2007-2013 and a pseudo-shock to that state at Jan. 1, 2012, using the remaining Full- and No-Expansion states as a control group. Treatment effects are estimated using the DiD model as in Table 2. Downward sloping line is regression line for regression of pseudo-treatment effect on ln(state population in 2012) and constant term.

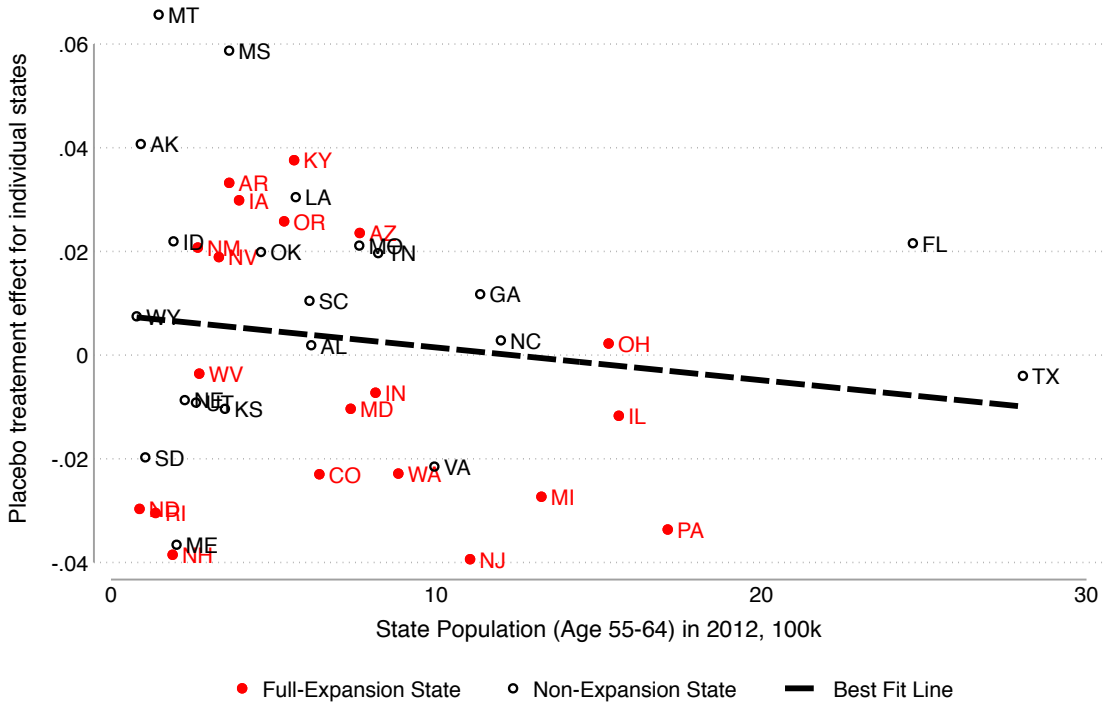


Figure 10. Simulation Based Power Analysis with Known First Stage

Power curves for simulated Medicaid expansion, as of January 1, 2012, applied to persons aged 55-64 during pre-treatment period (2007-2013). Graphs show power (likelihood of detecting a statistically significant effect on amenable mortality, at the indicated confidence levels, for two-tailed test), given imposed "true" population average effect. Curves are based on 1,000 replications of a triple difference specification. In each draw, we select 20 pseudo-treated states at random from the combined set of 41 treated and control states. We further break each county into a treated and untreated population. We consider an increasing share of those treated in each treated county (the first-stage), namely 1%, 3%, 5%, 10%, 15%, and 20%. We remove a fraction of the observed deaths at random from the treated states and treated portions of each county, where the fraction removed corresponds to an assumed true treatment effect, varying this imposed treatment effect from 0-10%. All reported curves have $\alpha = .05$, corresponding to a 95% confidence level. All control variables and standard errors are as in Table 2.