PUBLIC INSURANCE AND MORTALITY: EVIDENCE FROM MEDICAID IMPLEMENTATION[*]

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December 3, 2014

Abstract:

This paper provides new evidence that Medicaid's introduction reduced mortality rates among nonwhite infants and children in the 1960s and 1970s. Medicaid required states to cover all cash welfare recipients, which induced substantial cross-state variation in the share of children immediately eligible for the program. Before Medicaid, higher- and lower eligibility states had similar levels and trends in infant and child mortality rates. After Medicaid, public insurance utilization increased and mortality fell more rapidly among nonwhite children and infants in high-Medicaid-eligibility states. The estimates suggest that Medicaid reduced mortality among nonwhite child recipients by 24 percent. The introduction of Medicaid can account for 8 percent of the decline in nonwhite child mortality and 15 percent of the reduction in the racial gap in child mortality between 1965 and 1979.

JEL Codes: I13, I14, I18, J10, J18, N32.

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Word Count: 13,115

[*] This project was generously supported by the NICHD (T32 HD0007339) UM Population Studies Center Traineeship and the Robert Wood Johnson Foundation's Health Policy Scholars program. The opinions and conclusions expressed herein are solely those of the author and should not be construed as representing the opinions or policy of any agency of the Federal government. I am grateful to Doug Almond, Hilary Hoynes, and Diane Schanzenbach for sharing the Regional Economic Information System (REIS) data for the 1959 to 1978 period and the Food Stamp roll-out data; Amy Finkelstein for sharing the American Hospital Association (AHA) Annual Survey data from 1948 to 1974; and Jean Roth for sharing the AHA data from 1976 to 1990. I am also grateful for helpful comments from Martha Bailey, John Bound, Tanya Byker, Jamein Cunningham, John DiNardo, Nic Duquette, Brian Kovak, Helen Levy, Sarah Miller, Lisa Neidert, Sayeh Nikpay, Holly Painter, Gary Solon, Laura Wherry and seminar participants at Northwestern University, the University of California Davis, and the University of California, Berkeley.

The establishment of means-tested public health insurance—Medicaid—in 1965 was among the largest public efforts in United States history to improve the health of the poor. The program's architects predicted "the beginning of a new era in medical care for low income families…the assurance of complete, continuous, family centered medical care of high quality to persons who are unable to pay for it themselves," (Department of Health, Education and Welfare 1967a). Today, Medicaid is the most common way that poor families pay for medical care, especially for children (Cohen and Martinez 2013). In 2011, it covered 60 million people, including 4 in 10 children, and cost federal and state governments 414 billion dollars—the third most expensive transfer program behind Medicare and Social Security (Center for Medicare and Medicaid Services 2012; Kaiser Family Foundation 2013).

While Medicaid's costs are large and controversial, its benefits in terms of health have been harder to quantify. Quasi-experimental research finds that legislative expansions of Medicaid eligibility led to large reductions in mortality for infants, children, teens and adults (Currie and Gruber 1996a; b; Sommers et al. 2012; Meyer and Wherry 2013). The corresponding increases in *any* insurance coverage are relatively small, however (see Card and Shore-Sheppard 2004), leaving considerable uncertainty about the mechanism for these effects.[1] Adding to this uncertainty is that the Oregon Health Insurance Experiment (OHIE)—the highest-quality study of Medicaid's effect on health—finds no evidence of improvements in clinical health measures (Baicker et al. 2013) or one-year mortality (Finkelstein et al. 2012) for adults. The absence of significant results in the OHIE, however, may reflect its short time horizon, the characteristics of its sample, or its statistical power. Consequently, for a variety of reasons, decades of research on Medicaid has provided limited evidence on the program's health effects.

This paper uses the introduction of Medicaid between 1966 and 1970 and the federal requirement that states cover all cash welfare recipients (the "categorically eligible") to provide new estimates of its

[1] Explanations for such large magnitudes include underreporting of Medicaid coverage (Card et al. 2004), additional health effects from increased disposable income (Leininger et al. 2012), investments due to increased provider revenue (Finkelstein 2007), increased take-up of other transfer programs (Bitler and Currie 2004), or omitted variables (Dave et al. 2008).

effects on the health of the poor. The statutory link between welfare *receipt* and Medicaid *eligibility* motivates two aspects of my analysis. First, it generated wide variation across states in welfare-based eligibility due to long-standing, institutional differences. Second, nonwhite children were six times as likely to be eligible for Medicaid under the categorical eligibility provision as white children (18 percent versus 3 percent), and four times as likely as nonwhite adults (4.5 percent). This suggests that Medicaid implementation should have had heterogeneous state-level health effects that were largest for nonwhite children in states with higher initial eligibility.

I estimate Medicaid's effects in a difference-in-differences framework that compares infant and child mortality rates before and after Medicaid implementation (first difference) between higher- and lower-eligibility states (second difference). State-level mortality rates by age, race, and cause of death from 1959 to 1979 facilitate an event-study analysis of Medicaid's longer-run effects up to nine years after implementation. This empirical strategy, based on "dose-response" type comparisons across states with different eligibility levels, obviates the need for comparisons between states that implemented Medicaid earlier and later, which differed in their pre-Medicaid mortality trends.

The results imply that Medicaid was very effective in achieving one of its primary goals: "prevent[ing]…premature death" (Department of Health, Education and Welfare [DHEW]1967a). After Medicaid's introduction, high-eligibility states experienced dramatic decreases in the mortality rates of younger nonwhite children (-14 percent) and nonwhite neonates (-8 percent) relative to low-eligibility states. The effects persist for nine years and are not present for white children, who qualified for and used Medicaid much less often than nonwhite children. The child mortality results are driven by reductions in causes for which there were effective treatments in the 1960s and 1970s, and the neonatal mortality results reflect improvements in care within the first few hours of life rather than improvements in measure of fetal health, such as birth weight. Newly-entered data on public health insurance

programs from 1963 to 1976 verify that high-eligibility states also had relative increases in children's public health insurance use, the primary mechanism for the mortality effects. The estimates imply that Medicaid reduces the mortality of children who use it by one quarter.

Several pieces of evidence support a causal interpretation of these estimates. First, levels and trends in state characteristics in the early 1960s including poverty, mortality, and medical resources are uncorrelated with welfare-based eligibility differences when Medicaid was implemented. Moreover, the results from an event-study specification (Jacobson et al. 1993) show directly that mortality rates in high- and low-welfare states did not trend differently in the seven years prior to Medicaid. Second, there is little evidence of differential changes after Medicaid in other programs that could affect mortality such as Food Stamps, Community Health Centers, or Head Start. There is also little evidence of sharp changes in welfare participation, which alleviates concerns that welfare receipt itself is driving the effects. Finally, the results are robust to controls for time-varying measures of state welfare participation, which suggests that they are not due other health-related public efforts for which categorical Medicaid eligibility might proxy.

The implied effects on treated infants and children are smaller than estimates from the eligibility expansions in the 1980s (Currie and Gruber 1996a; b), yet they still suggest that Medicaid played an important role in national mortality changes. I estimate that Medicaid implementation reduced aggregate nonwhite child mortality rates by 8 percent, and can account for 15 percent of the decline in the white-nonwhite mortality rate gap between 1966 and 1979.

These results are also the first to establish that the *introduction* of Medicaid reduced mortality. Some authors have argued that Medicaid implementation had limited health effects because poor families already received public or charity care (Roghmann et al. 1971; Klarman 1974; Matusow 1984) or because Medicaid provided low-quality services (Bernard and Feingold 1970). My results challenge

these claims and show that the expansion of public insurance for poor children over and above any pre-Medicaid charity/public arrangements had important health benefits immediately and in the longer-term. Because mortality is an extreme outcome, these results understate Medicaid's broader health benefits. These findings imply that proposals to eliminate Medicaid, allow states to opt out, or cap federal reimbursements (Grannemann and Pauly 1983; Smith and Haislmeier 2009) could hurt the health of poor children even if their care is taken up by private charity to the degree that it was in the 1960s.

I. WHAT DO WE KNOW ABOUT MEDICAID AND HEALTH?

A large literature in economics examines Medicaid's effects on insurance coverage and the use of medical care, but only a handful of quasi-experimental studies estimate its effect on health. This work consistently demonstrates that Medicaid eligibility expansions reduce mortality (the most common health outcome used in this literature), but they fail to find large effects on insurance coverage. Currie and Gruber (1996a, 1996b) find that a series of legislative increases in eligibility to pregnant women and to children during the 1980s reduced infant mortality rates by 8 percent and child mortality rates by 5 percent.[2] The estimated effect of these expansions on insurance coverage, however, range from 0 (Cutler and Gruber 1996) to about 3 percentage points (Dave et al. 2008) for pregnant women, and from a slight *reduction* (Yazici and Kaestner 2000) to an increase of between 2.4 and 4 percentage points for children (Cutler and Gruber 1996; Shore-Sheppard 2009). Assuming that Medicaid expansions only affect the health of those who change insurance status, dividing the mortality reductions by the increase in insurance implies that Medicaid reduces mortality of those who gain insurance coverage by more than 100 percent—an impossible result.[3]

[2] Studies that examine the same eligibility expansions in individual states find much smaller effects on infant and child mortality (Piper et al. 1990; Long and Marquis 1998), although most of them suffer from methodological limitations related to poorly defined control groups (Levy and Meltzer 2004).

[3] Different quasi-experimental research designs and populations produce similar conclusions. Meyer and Wherry (2013) use a regression discontinuity (RD) estimator based on a provision that granted eligibility to some children born after September 30, 1983. They find that mortality rates among black children born just after the cutoff fell by about 7 percent at ages 8 to 14, and annual internal-cause mortality rates fell by 11 percent at ages 15 to 18. Card and Shore-Sheppard (2004) use the same

Estimates of Medicaid's effect on health could be large relative to its effect on insurance for several reasons. The proportional treatment effects on mortality will be overstated because poorer families who actually take up Medicaid have high mortality rates and because survey data used to estimate take-up understate Medicaid coverage (Card et al. 2004; Davern et al. 2007).[4] Subsequent work also suggests that the 1980s expansions affected outcomes other than increased insurance coverage that may influence health. Families that dropped private coverage gained disposable income from savings on premiums, cost sharing, and wage offsets (Gruber and Yelowitz 1999; Leininger et al. 2012). Bitler and Currie (2004) show that increased income eligibility thresholds for Medicaid led to higher take up of in-kind food benefits, which also improve infant and child health (Hoynes et al. 2011). Expansions may also have spurred providers to invest in new technologies, which could prevent deaths among those not actually covered by Medicaid (Finkelstein 2007; Pauly and Pagán 2007; Freedman et al. 2014). Finally, Dave et al. (2008) argue that estimates based on the state-by-year variation in the expansions are biased by omitted variables, although this cannot explain the large RD estimates. Thus, quasi-experimental evidence suggests that Medicaid expansions have reduced mortality, but their magnitudes preclude an interpretation of these estimates as the effect of Medicaid coverage *per se*.

Additional uncertainty about Medicaid's effect on health comes from the Oregon Health Insurance Experiment (OHIE), a randomized expansion of adult eligibility in 2008. Results from the first year of post-randomization data show no effects on adult mortality rates (Finkelstein et al. 2012) or on a range of clinically-measured outcomes such as blood pressure or cholesterol (Baicker et al. 2013). More years of data may ultimately reconcile these results, but the disagreement across research designs in the short-

discontinuity and find that contemporaneous insurance coverage rose by only 10 percentage points. Sommers et al. (2012) show that recent expansions of adult eligibility reduced mortality by 6 percent, but increased insurance coverage by only 3 percentage points.

[4] The estimates in appendix 4 account for these factors, but the implied effects of Medicaid on mortality are still larger than on insurance in several cases. Appendices available at: http://www-personal.umich.edu/~ajgb/medicaid_appendix_ajgb.pdf.

run mortality estimates leaves open many important questions.[5] Whether and by how much Medicaid improves health in the shorter- or longer-term remains uncertain.

II. PUBLIC INSURANCE AND MORTALITY BEFORE AND AFTER MEDICAID

The potential for Medicaid *implementation* to improve health depends largely on health status and sources care for poor families before it began. In 1950, the federal and state governments began to share some of the costs of medical care that public assistance recipients obtained from private providers. Importantly, the federal contribution was capped, which made states reluctant to set generous eligibility or coverage criteria. By 1963 only one percent of children received subsidies for health care (figure I).[6]

The lack of publicly-financed care was not fully offset by non-profit or private sources, a fact reflected in income differentials in insurance, utilization, and health, especially for children. Figure I shows that in the early 1960s, over 30 percent of children were uninsured (compared to under 10 percent in 2012). Poor families especially lacked private insurance coverage: in 1959, only 8.9 percent of people with family incomes below $2,000 had doctor visit insurance, and less than a third had hospital or surgical insurance (Kovar 1960). Furthermore, only 8 percent of adults reported receiving charity care in 1960, but this may include worker's compensation benefits (Morgan et al. 1962).

These disparities in payment sources correspond to disparities in care. Data from the 1963 Survey of Health Services Utilization and Expenditure (SHSUE) show that only 45 percent of children in the bottom third of the income distribution had seen a physician in the previous year, compared to 77 percent of children in the top third (Center for Health Administration Studies and National Opinion Research Center 1984a).[7] Serious symptoms, such as 4-5 days of diarrhea, heart pain, or unexpected

[5] Oregon's Medicaid expansion under the Affordable Care Act, however, means that the OHIE will not be able to generate results for as many post-expansion years as in Sommers et al. (2012).
[6] 16 did not cover physician services, and 12 did not cover hospital services (Committee on Ways and Means of the House of Representatives 1961).
[7] In 2011, 77 percent of children in the bottom third of the income distribution had a checkup within the previous year compared to 83 percent in the top third (Minnesota Population Center and State Health Access Data Assistance Center 2012).

bleeding were more common for poorer children, and conditional on having less-serious symptoms such as a skin rash, a persistent cough or sore throat, or abdominal pain, children in the bottom third of the income distribution sought care much less often than those in the top third (28.5 percent versus 42 percent, standard error [s.e.] of the difference = 4.7).

A. A Brief History of Medicaid's Implementation

Medicaid (P.L. 89-97) was established by the 1965 amendments to the Social Security Act (SSA), and aimed to eliminate these income-based inequalities in health and health care.[8] States were required to implement Medicaid by 1970 or else lose federal reimbursements for existing medical programs. 26 states adopted Medicaid in 1966, 11 in 1967, and (most of) the rest between 1968 and 1970.[9]

While Medicaid was billed as an incremental change, it actually represented a major expansion in federal support for the medical care of poor families. The financial mechanism for this expansion was a move to an open-ended appropriation, which eliminated the caps on the federal contribution and increased the federal share of the cost of public medical payments from about 13 percent (Norman 1952) to between 50 and 83 percent. In return for increased federal funds, Medicaid required that states cover at least five types of care with *no patient cost sharing*—inpatient hospital, outpatient hospital, laboratory and x-ray, skilled nursing home, and physician services[10]—and mandated coverage for recipients of federally funded cash welfare programs (the "categorically eligible").[11]

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[8] Medicaid was was added to the SSA amendments as an "afterthought" (Ginzberg and Solow 1974; Grannemann and Pauly 1983) meant to undercut the American Medical Association's (AMA) opposition to the bill's most prominent component: Medicare. The final SSA amendments combined three proposals into Medicare Part A (compulsory hospital insurance for all elderly, the Democratic proposal), Medicare Part B (voluntary supplementary physician insurance for all elderly, the Republican proposal) and Medicaid (a federal/state funded public insurance program for the poor, the AMA's preferred program for the elderly). Assistant Secretary of the Department of Health, Education and Welfare Wilbur Cohen, remarked "It was the most brilliant legislative move I'd seen in thirty years…In effect, [Wilbur Mills (D-Arkansas)] had taken the A.M.A.'s ammunition, put it in the Republicans' gun and blown both of them off the map" (Harris 1966, pp. 40).

[9] Alaska did not adopt Medicaid until 1972 and Arizona did not adopt it until 1982.

[10] States could also choose to cover additional services, including home health care, clinic services, prescription drugs, eye care and dental care. The 1972 SSA amendments allowed states to charge co-payments for the optional services (Davis and Schoen 1978), but not for required services. Categorically eligible children were (and still are) exempt from cost sharing.

[11] Medicaid defined several other eligibility groups not discussed here. In particular, states could choose to cover the "medically needy"—families with incomes too high to qualify for cash public assistance, but with large medical bills that

B. Medicaid Eligibility by Age and Race

The cash welfare recipients who gained Medicaid eligibility included the poor elderly, blind, and disabled, but the Aid to Families with Dependent Children program (AFDC) program accounted for most categorical eligibility, especially among children. 89 percent of children on Medicaid in 1976 were categorically eligible, essentially all of them through AFDC (DHEW 1966; DHEW 1976a).

AFDC-based Medicaid eligibility affected a much higher share of nonwhite children than white children. I use two sources of data to measure racial differences in eligibility. For 1958 and 1961, I entered state-level data on the race of AFDC payees and children (Mugge 1960; DHEW1963), and for 1967-1979 I calculate race shares using microdata on AFDC recipients collected from the National Archives (DHEW 2000; 2011). Multiplying these shares by AFDC participation counts and dividing by the relevant population gives an annual estimate of the AFDC rate by race.[12] Figure II plots age-specific AFDC rates using the 1967 data.[13] Children of both races received AFDC at almost four times the rate of adults, and nonwhite children received AFDC more than six times as often as white children. The statutory connection between AFDC receipt and Medicaid eligibility, therefore, implies that nonwhite children had by far the highest eligibility rates for the new, generous public insurance.

C. Medicaid Use By Age and Race

Not surprisingly, high rates of eligibility translated into high rates of Medicaid use. The solid line in figure III shows the share of all children ages 0-19 who received medical services paid for by public insurance in the years before and after states began their Medicaid programs.[14] The public insurance

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pushed their net income below state-defined thresholds. The medically needy account for a small share of children on Medicaid, and I ignore this provision in the rest of the paper. For details on Medicaid eligibility see Gruber (2003).

[12] I linearly interpolate the race shares between missing years. The 1958 and 1961 reports only contain the race distribution of the AFDC payee in each case and recipient children ages 0-19, so I only construct AFDC rates for women (using the payee data) and for children ages 0-19. I use a binary measure of race to maintain consistency across AFDC data sources.

[13] The 1967 AFDC dataset is the largest (265,707 observations; 4,297 observations on average in each race/age cell) and contains single-year ages. Age- and race-specific welfare rates in the 1970 Census are very similar (appendix figure 2.1).

[14] These data were entered from federal reports on means-tested public insurance from 1963 to 1976 (DHEW various years; see appendix 1). The data measure utilization of benefits, referring to children (ages 0-19, as defined by AFDC eligibility

rate for children increased by 10 percentage points in the five years after Medicaid implementation, but only 2 percentage points for adults (not shown). Annual data on public insurance use by race are not available, but several data sources corroborate these dramatic racial differences. The ratio of public insurance use for nonwhite children ages 1-4 to the average child ages 0-19 is 3.2 in the 1976 Survey of Income and Education (US Department of Commerce 2006), and 3.7 in the 1976 National Health Interview Survey (appendix figure 2.2).

D. The Expected Effects of Medicaid Implementation on Mortality

These changes may have affected a range of health outcomes, but the primary measure used in this paper (and in other work on Medicaid and health) is mortality. Death is an extreme health measure, but conceptually it is an unambiguous indicator of poor health, especially for children, and unlike other health outcomes, it is well-measured and consistently available. The expected effects of Medicaid on mortality hinge on the extent to which the medical care it covered actually prevented deaths. In this regard, the groups covered the most—nonwhite children and infants—had the greatest potential for medical-care-induced health improvements because they were relatively unhealthy in the early 1960s.

Figure IV shows that in 1965, internal causes—a common measure of the sensitivity of mortality to medical interventions—account for nearly all infant deaths, more than 60 percent of deaths among 1 and 2 year olds, and about 50 percent of deaths among 3 to 12 year olds.[15] Nonwhite children and infants were especially at risk. Their mortality rates in 1965 were twice as high as for whites of the same age (National Center for Health Statistics 1965, Table 1-9), and they were much more likely to die of causes with "effective" treatments (Beeson 1980). Among children ages 1 to 4 in 1965, 35.4 percent of all

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rules) who actually obtained medical care. More recent papers measure reported Medicaid coverage, referring to children who have signed up for but not necessarily used Medicaid. Utilization means more for health than coverage, and it incorporates the effects of provider participation in Medicaid, which would not be reflected by coverage data.

[15] The International Classification of Disease defines a set of "external" causes that include mainly transportation-related accidents, drowning, falls, poisonings, choking, homicide and suicide. All other causes are "internal". While internal-cause mortality is high for the elderly, Medicaid represented a smaller change in the availability of insurance because they were already covered by Medicare and the Kerr-Mills program, which targeted low-income seniors.

nonwhite child deaths were due to infectious diseases, compared to only 26.4 percent of white child deaths were due to these causes (s.e. of the difference = 0.8).[16]

Available treatments were usually effective and often inexpensive. For example, the vast majority of pneumonia cases were bacterial, and when treated early with penicillin "approximately 95 per cent of patients…recover" (Cecil et al. 1967). Many non-bacterial conditions could be managed, if not prevented or cured. Nonwhite children (partly because of a genetic predisposition among African-Americans), were more than twice as likely as white children to die from anemias, but a folate supplement "suppresses or controls the disease" (Beeson 1980). Nonwhite children were also less likely to be fully vaccinated (National Center for Health Statistics 1976, Tables CD.I.47 and CD.I.48), and more likely to die of conditions that could be vaccinated against (1.6 percent versus 0.9 percent, s.e. of the difference = 0.2). The resources to treat low-birth-weight babies also improved in the 1960s with the proliferation of neonatal intensive care units stocked with equipment such as temperature-controlled oxygenated incubators and ventilators (Budetti and McManus 1982). This held great potential for improving nonwhite neonatal infant mortality because nonwhite babies were significantly more likely to have low birth weight and low birth weight infants accounted for the majority of neonatal deaths (Armstrong 1972).[17]

Finally, Medicaid coverage almost certainly represented *new* insurance coverage at this time and, therefore, a meaningful increase in care. Figure I shows that the 1960s and 1970s are the only period in recent US history when changes in public coverage corresponded to similarly large reductions in the share of uninsured children (about 15 percentage points). Therefore, I expect Medicaid implementation

[16] This primarily includes pneumonia (18 percent), meningitis (4.6 percent), and gastroenteritis (3.2 percent). Nonwhite deaths were also more likely to be due to causes so general that they reflected inadequate medical care such as "ill-defined symptoms or conditions" (5.36 percent versus 1.58 percent, s.e. of the difference = 0.3).

[17] The technology to treat extremely low weight babies—artificial lung surfactant—had not yet been developed. Consequently, studies of matched birth and death records at this time show that birth weight specific mortality rates drove aggregate reductions in neonatal infant mortality, but that these reductions were proportionally smaller at the lowest weights (Williams and Chen 1982; David and Siegel 1983).

to have the strongest effect on nonwhite infant and child mortality both because they were especially exposed to Medicaid through its link with AFDC and because they died of conditions that were treatable with the kinds of medical care funded by Medicaid.

III. RESEARCH DESIGN: USING CATEGORICAL ELIGIBILITY TO IDENTIFY HETEROGENEOUS EFFECTS OF MEDICAID IMPLEMENTATION ON MORTALITY

The categorical eligibility requirement meant that the sudden increase in public insurance eligibility under Medicaid varied widely across states. This cross-state eligibility variation is the basis of my research design. I identify Medicaid's effect using a difference-in-differences model that compares state-level health outcomes before and after Medicaid implementation in states with higher and lower categorical eligibility. For this approach to uncover Medicaid's health effects, categorical eligibility must correspond to increases in public insurance rates after Medicaid (relevance), and be unrelated to changes in mortality except through its statutory connection to Medicaid eligibility (excludability). This section supports these assumptions using data on AFDC-based eligibility, public insurance participation and pre-Medicaid state characteristics.

A. Cross-State Variation in AFDC-Based Categorical Eligibility and Public Insurance Use

In addition to higher *levels* of categorical eligibility, nonwhite children also experienced much greater variation across states in their categorical eligibility rates. State-level eligibility for all children in the year *of* Medicaid implementation ranged from 1.5 to 11 percent, while eligibility for nonwhite children ranged from about 5 to 30 percent (appendix table 2.1).[18] The standard deviation of initial AFDC rates was more than five times higher for nonwhites than for whites (0.08 versus 0.015).[19]

[18] The calendar year of implementation is typically the year just before states' first full year with an operational Medicaid program because most programs began partway through the year and were subject to delays due to "shortages of welfare personnel to screen applications" (Tax Foundation 1968, pp. 47).

[19] Moreover, states with high nonwhite AFDC rates often had low white AFDC rates and vice versa. Table I shows that almost half of states (22 out of 48) states are above the (race-specific) median for one race but below for the other, and the coefficient from a regression of nonwhite on white AFDC rates is positive (1.4) but insignificant (s.e. = 1.0), with an R^2 of 0.04. The racial differences in cross-state AFDC variation mean that my design will have more power to detect Medicaid's effect on nonwhite children than on white children, and the weak relationship between white and nonwhite AFDC rates suggests that this statistical power requires a race-specific measure of categorical eligibility.

Cross-state differences in AFDC-based categorical eligibility correspond closely to differences in public insurance use. The dashed lines in figure III plot children's public insurance utilization in high- and low-eligibility states (defined by the median overall AFDC rate). The difference between high- and low-AFDC states before Medicaid implementation was very small (0.005, s.e. = 0.002), but rose to 0.05 (s.e. = 0.006) after Medicaid was fully implemented. Therefore, one identifying assumption holds: initial categorical eligibility strongly predicts post- Medicaid increases in public insurance use.

Cross-state variation in public insurance use also appears to be larger for nonwhite children than for white children. Data from the 1970 SHSUE show that the share of nonwhite children under 5 who had medical care paid for by a public source (including Medicaid) is 17 percentage points higher in high-nonwhite-eligibility states (s.e. = 0.05) than in low-nonwhite-eligibility states. The difference for white children is 10 percentage points (s.e. = 0.03).

B. Determinants of State-Level Categorical Eligibility

The plausibility of the remaining identifying assumption—that in the absence of Medicaid, mortality would have evolved similarly in higher- and lower-AFDC states—hinges on the claim that AFDC differed across states for reasons unrelated to *changes* in mortality in the mid-1960s. This is likely to hold for two related reasons.

First, AFDC rates were relatively long-run and stable features of states. AFDC rates vary because of factors that affect eligibility—state policies, family structures and income—and factors that affect take-up—psychic costs and institutional barriers. Cliometric studies of welfare programs show that these variables differed across states at least as far back as the 1930s, and data on AFDC rates by race in 1948, 1958 and 1961 provide direct evidence on this stability.[20] The slopes from univariate regressions of the

[20] Moehling (2007) demonstrates that cross-state differences in family structure and the generosity of transfer programs for one-parent families existed even before the implementation of the Aid to Dependent Children program (the original name of AFDC), and persisted through the 1990s. Alston and Ferrie (1985) argue that agricultural states restricted welfare programs in the 1930s in order to maintain a "loyal" workforce. Many states and localities kept nonwhite families off the rolls by the

state-level AFDC rate in the year of Medicaid implementation ($AFDC_s^*$) on AFDC rates in each prior year are positive, very precisely estimated, but most importantly they are not statistically distinguishable from each other. (p-values from a test of the null hypothesis that they are equal are 0.68 for nonwhite rates and 0.55 for white rates; see appendix figure 2.3.) That the variation across states in initial categorical eligibility did not emerge *contemporaneously* with Medicaid ameliorates concerns that states made policy choices (or recipients changed behavior) in anticipation of Medicaid's implementation. Instead, initial categorical eligibility reflects long-standing differences apparent decades beforehand.

Second, these longer-run institutional features impacted participation in ways that broke the association between policies, state characteristics an actual AFDC receipt, especially for nonwhite families. For example, since the 1940s, Texas' constitution required that increases in welfare spending, which were often necessary to comply with federal entitlement programs, be passed by popular referendum. This created major political barriers to expanding welfare programs and, not surprisingly, Texas had low white and nonwhite AFDC participation. Alternatively, Nebraska had long provided aid to unmarried mothers (Moehling 2007) so its nonwhite AFDC rate was close to the median even though white AFDC participation was among the lowest in the country. Finally, provisions related to cohabitation, relationships, and employment were disproportionately applied to nonwhite recipients so that even a high-benefit state like Illinois had low nonwhite participation relative to its statutory generosity.

The importance of idiosyncratic institutional differences means that AFDC participation in the year of Medicaid implementation is largely unrelated to state demographic or policy characteristics, especially for nonwhite families. Comparing observed AFDC rates to calculated eligibility levels in the

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disproportionate application of vague eligibility provisions such as "suitable home" or "substitute parent" policies that were part of pre-AFDC Mothers' Pension programs (Bell 1965).

1960 Census corroborates this.[21] For white women, state-level eligibility is positively and significantly related to receipt in 1960 (population-weighted OLS coefficient is 0.7, s.e. = 0.1), but for nonwhite families the relationship is small and insignificant (0.08, s.e. = 0.21; see appendix figure 2.4).[22]

Table II provides evidence on the specific correlates of initial categorical eligibility. Rows 1 through 3 of table II show the child poverty rate in 1960, the probability that children lived in a single mother household in 1960, and the average AFDC benefit in 1967 for white children. White child poverty in low-AFDC states (column 1) and high-AFDC states (column 2) are indistinguishable (*p*-value of the difference is in column 3). Consistent with Moehling (2007), single motherhood and average benefit amounts are slightly higher in the states with high white AFDC rates. Rows 7 through 9 show that for nonwhite children, none of these variables is significantly different between high- and low-AFDC states, perhaps due to the influence of institutional deterrence.

Evidence that these characteristics did not *change* differently in the AFDC groups provides additional support for the validity of the AFDC-based research design. Changes in child poverty (between 1950 and 1960) and infant and child mortality (in the five years before Medicaid) are indistinguishable for both whites (rows 4-6) and nonwhites (rows 10-12). Panel C shows that pre-

[21] I use the 1960 Census and a table of AFDC "needs standards" (one of several income eligibility thresholds) from 1961 (DHEW 1963; table 40) to calculate the share of women between 20 and 64 who are unmarried family heads, with at least one qualifying child (under 16 or under 18 and attending school), and monthly "countable income" (earnings minus "other" income and income of qualifying children) below the average family-size specific needs threshold in her state. Due to data limitations, this calculation ignores eligibility criteria such as coverage of unborn children, asset tests, the "payment test" (which compares adjusted income to a lower payment threshold). More importantly, I cannot account for more subjective eligibility criteria, such as requirements that heads accept work, "man-in-the-house" or "suitable home" provisions, or caseworker practices such as underbudgeting (requiring additional paperwork to increase recipients' grants after the birth of a child, deducting child support amounts regardless of whether or not the support order was paid; Piven and Cloward 1971). The importance of these criteria for actual categorical Medicaid eligibility strengthens the identification strategy based on observed AFDC rates because, as appendix figure 2.4 shows, they lead to nonwhite) AFDC rates that are orthogonal to the factors that determine eligibility.

[22] This clarifies why a common strategy used to study Medicaid—the use of a simulated eligibility variable based on posted rules—would not capture the relevant variation in categorical eligibility (which is based on AFDC receipt) in the 1960s. This approach would fail to assign low levels of nonwhite Medicaid eligibility, for example, in states with generous *de jure* regulations but restrictive *de facto* welfare systems.

Medicaid health resources (per-capita hospital beds and the share of children on public insurance) did not change differentially in the AFDC groups prior to Medicaid.

In summary, table II supports the conclusion of cliometric research showing that the large cross-state differences in welfare-based Medicaid eligibility were inherited from long-run institutional differences in the welfare system. Comparing changes in health outcomes across states with different rates of categorical eligibility is unlikely to confound Medicaid's effect with other factors that affect health.

C. Data and Estimation Sample

To measure health, I construct state-by-year infant and child mortality rates from the 1959 to 1979 Vital Statistics Multiple-Cause of Death Files (US DHHS and NCHS 2009), which contain the universe of civilian deaths that occurred in the U.S. by cause, age, race, and state of residence of the decedent.[23] For children, the age-specific mortality rate is the count of deaths in group *a* (ages 1-4 or 5-14) divided by the population in age *a* per 100,000. I also use two measures of infant mortality: neonatal mortality (deaths in the first 28 days of life per 1,000 live births) and post-neonatal mortality (deaths between 28 days and 1 year per 1,000 live births).[24]

D. Event-Study Specification with High- and Low-Eligibility Groups

Equation 1 describes a difference-in-differences (DD) model for demographic group k where the high-AFDC states are the treatment group, the low-AFDC states are the control group, and pre/post treatment is defined by the year of Medicaid implementation. The estimating equation is an event-study specification (Jacobson et al. 1993) that includes state-by-year-level covariates and fixed effects in $\boldsymbol{x}'_{st}$,

[23] The exception is 1972, which contains a 50 percent sample, and 1981 and 1982 which contain a 50 percent sample for some states. In 1981 and 1982, I use Mortality Detail files, and in 1972, the mortality data are based on the reduced sample.
[24] Denominators for the child rates were constructed by linearly interpolating population between the 1950 and 1960 censuses (Haines and ICPSR 2005) and the 1969 to 1988 Surveillance Epidemiology and End Results (SEER 2009) data. Denominators for the infant rates were calculated from Vital Statistics Natality Microdata from 1968-1979 (US DHHS and NCHS 2002) and entered state totals from Vital Statistics reports from 1959-1967. I end the sample in 1979 because the 1980s eligibility expansions largely eliminated the state differences that drive my results. Appendix figure 2.5 presents results on a sample from 1959 to 1988 and, consistent with the convergence in Medicaid rates induced by the 1980s expansions, the results fade by 1988.

and interactions between a high-AFDC indicator, D_s defined in table I and dummy variables that measure the time relative to Medicaid implementation, $1\{t - t_s^* = y\}$ (i.e., "event-time"):[25]

$$\ln\left(ASMR_{st}^{k}\right) = \boldsymbol{x}_{\boldsymbol{st}}'\boldsymbol{\beta}_{\boldsymbol{k}} + D_s\left[\sum_{y=-8}^{-2}\pi_y^k 1\{t - t_s^* = y\} + \sum_{y=0}^{10}\gamma_y^k 1\{t - t_s^* = y\}\right] + e_{st}^k \qquad (1)$$

My preferred specification includes per-capita income, per-capita hospital beds, state fixed effects and nonparametric controls for two kinds of time-varying unobservables: region-by-year fixed effects, and a separate set of 21 year fixed effects for each Medicaid timing group.

Conditional on region-by-year fixed effects, the estimates rely on mortality comparisons between high- and low-AFDC states *within* each region. In particular, this controls for the strong convergence in mortality between the South and the rest of the U.S. due to hospital desegregation (Almond et al. 2006), region-level trends in school quality (Stephens and Yang 2013), and regional differences in private insurance coverage (Finkelstein and McKnight 2008).[26]

The Medicaid-timing-by-year fixed effects eliminate comparisons between states that adopted Medicaid earlier or later. A DD model based *only* on the differential timing of Medicaid adoption is identified (Decker and Gruber 1993; Strumpf 2011), however in my context, differential mortality trends in earlier and later Medicaid states violate the identifying assumption of this "timing-only" estimator (see appendix 3). Policymakers at the time reported putting off Medicaid implementation because of fiscal concerns (ACIR 1968), and Finkelstein (2007) concludes that, with respect to hospital capacity,

[25] I use a binary variable to measure eligibility because it yields this simple interpretation, but the results are unchanged by replacing D_s with the continuous AFDC rate ($AFDC_s^*$) in each state's Medicaid implementation year (appendix figure 2.6 and table 2.3). I define groups by the median AFDC rate so that they each have an equal number of states, but the results are not sensitive to defining D_s using an algorithm that maximizes the t-statistic on the difference in AFDC rates between the two groups (appendix figure 2.7). I use AFDC rates for women because it is the appropriate measure of eligibility for the infant (especially neonatal) mortality regressions, and I use the same rates in the child regressions so that there is a common state grouping used in all of the results. The results for non-infant children are unchanged when I create state groups using the child AFDC rates (appendix figure 2.8).

[26] The main results use four census regions, but the results are not sensitive to different definitions, including moving the northern-most southern states (Delaware, Maryland and West Virginia) into the "Northeast" region, dropping the Deep South (Alabama, Georgia, Louisiana, Mississippi, and South Carolina) or dropping the South entirely (appendix table 2.2).

"the timing of state implementation of Medicaid was not random." The Medicaid-timing-by-year fixed effects ensure that estimates of equation 1 rely only on comparisons between AFDC groups rather than between earlier and later Medicaid states.

The coefficients of interest, π_y^k and γ_y^k, measure the covariate-adjusted difference in log mortality rates between high- and low-eligibility states in the seven years leading up to Medicaid's introduction and the nine years after. The dummy for the year before Medicaid is omitted (to avoid collinearity with the state fixed effects), which normalizes the estimates of π_y^k and γ_y^k to zero in that event-year.[27] The π_y^k are falsification tests that capture differences between the two AFDC groups in the pre-Medicaid period. Their pattern and statistical significance are a direct test of the common trends assumption. The γ_y^k are intention-to-treat (ITT) effects of Medicaid on aggregate mortality in high-AFDC states relative to low-AFDC states. This specification identifies *heterogeneity* in Medicaid's effect. The estimates will equal zero if Medicaid affected mortality equally across states. Moreover, they will understate Medicaid's total effect on mortality because they "difference out" any portion of the effect that is common to low- and high-eligibility states. For example, if Medicaid led to investments in hospital technologies, as was the case for Medicare (Finkelstein 2007), then mortality effects arising from investment that is *common* to high- and low-AFDC states will not be captured by this empirical strategy.

I also present the coefficients from a "grouped" event-study specification that combines the event-time dummies into four bins ([-7,-2], [0], [1,4], [5,9]) or a difference-in-difference (DD) specification that estimates one treatment effect for event-years [1,9]. The standard errors are clustered at the state-level to allow for arbitrary serial correlation within states.

[27] Event-time dummies that are more than seven years before or nine years after Medicaid implementation are grouped because not all states are observed at these event-years.

IV. ESTIMATES OF MEDICAID'S INTENTION-TO-TREAT EFFECT ON MORTALITY RATES

The primary mechanism through which Medicaid implementation should affect mortality is by increasing the utilization of (publicly-financed) health services. Figure V plots estimates of equation 1 using child public insurance rates from 1963-1976 as the dependent variable (so the figure shows coefficients for event-years -3 through 6). Before Medicaid, public insurance use is indistinguishable between the high- and low-AFDC groups (the *p*-value from a joint significance test of the -3 and -2 coefficients is 0.16). After Medicaid implementation, it rises in the high-AFDC states and is 4.7 percentage points higher on average in the next six years (s.e. = 1.6). These results show that, even conditional on a rich set of covariates, AFDC-based eligibility is strongly associated with increases in public insurance after Medicaid.[28] Although I cannot directly estimate this relationship by race, the differences in AFDC rates—1.1 percentage point difference between high- and low-AFDC groups for the overall rate and 4.5 percentage points for nonwhite rates—imply that the relevant effect on public insurance use for nonwhite children is 19 percentage points (.047*.045/.011).

A. Results for Age-Adjusted Child Mortality by Race

Figure VI presents event-study estimates of Medicaid's ITT effect on a summary mortality measure: log age-adjusted mortality for children ages 0-14.[29] The small pre-Medicaid estimates strongly support the AFDC-based research design in equation 1. In the seven years before Medicaid, high- and low-eligibility states had nearly identical mortality changes. The pre-Medicaid point estimates fluctuate between -0.018 and 0.029, and neither the white nor the nonwhite coefficients are jointly distinguishable from zero (*p*-values are 0.46 and 0.38, respectively). Concerns about different mortality trends between AFDC groups are not borne out in these results.

[28] Appendix figure 2.9 shows that per-recipient expenditures did not change differently after Medicaid in high- and low-AFDC states. High-eligibility states actually spent slightly less per recipient in my data ($702 versus $790 in 2012 dollars). This suggests that the size of the categorically-eligible population, while strongly related to Medicaid use, is not related to the value of services received by the average child on Medicaid.

[29] The estimates are weighted by state populations. Unweighted results are nearly identical (appendix figure 2.10).

Consistent with the high nonwhite eligibility rates, Medicaid's ITT effects on mortality are large and negative for nonwhite children. Nonwhite mortality fell slightly in the year of Medicaid implementation (time zero on the x-axis), which matches the pattern in the first-stage estimates and reflects the fact that Medicaid programs were only partially implemented in the first calendar year. After the first year, however, nonwhite mortality in high-AFDC states fell significantly more than in low-AFDC states. The event-study estimates are highly jointly significant (the *p*-value on a joint *F*-test of the post-Medicaid coefficients is <0.0001), and the DD estimate is a reduction of 9 percent (s.e. = 0.03).

The post-Medicaid estimates for white mortality, on the other hand, are not different from zero (DD estimate is 0.01, s.e. = 0.01). This is consistent with the much lower AFDC-based Medicaid eligibility for white children and bolsters the claim that the effects are attributable to Medicaid.[30]

Were white children were unaffected or does the research design fail to detect their presumably smaller ITT effect? Assuming that the individual-level treatment effect and take-up rate were the same for white and nonwhite children, the relative magnitudes of cross-state AFDC differences by race imply that the white ITT should be -0.02 (-0.09 nonwhite effect divided by the ratio of high/low eligibility differences, 4.1). The confidence interval of the white DD estimate (-0.01, 0.04) can rule out an effect of this size, which suggests that any effect Medicaid had on treated white children is smaller than its effect on nonwhite children, which is consistent with the large pre-Medicaid racial gaps in utilization and health. The rest of this section reports evidence for nonwhite mortality only. Results for white mortality are small and insignificant for all age groups (appendix figure 2.12).

Table III shows that covariates have only a small effect on the nonwhite treatment effects. Column 1 contains the simplest specification, which is equivalent to taking mean differences between eligibility groups in each event-year group. Columns 2 and 3 add fixed effects and continuous covariates. The last

[30] Appendix figure 2.11 plots estimates from a pooled model by race that interacts the treatment variables with a nonwhite dummy and includes state-by-year fixed effects. These covariates control for *any* factors that affect both white and nonwhite children. The results are similar to those in figure IV (DD estimate is -0.065, s.e. 0.021).

two columns show modified versions of the preferred specification without weights (column 4) or with state-specific linear trends (column 5). The estimates are very similar, with DD effects ranging from -0.07 to -0.10. A Hausman test cannot reject the null hypothesis of equality between the weighted and unweighted estimates for either panel (Deaton 1997).[31] Panel B also shows the *p*-value from a test of the DD restrictions in the grouped event-study model: the pre-Medicaid coefficient equals zero and the post-Medicaid coefficients (except year 0) are equal. These restrictions are not rejected for any model.

B. Results for Nonwhite Infant Mortality

One group with extraordinarily poor outcomes in the 1960s was nonwhite infants. Column 1 of table IV shows that Medicaid led to significant reductions in neonatal infant mortality. The pre-Medicaid coefficient (panel A, row 1) is very small, but the post-Medicaid point estimates are negative and highly jointly significant (*p*-value less than 0.0001).[32] The DD restrictions are not rejected, which suggests that the mortality reductions are persistent over Medicaid's first ten years and well-summarized by the DD estimate, -0.08 (s.e. = 0.02).

Because infant death certificates typically record the hour (within the first 24) or day (within the first 28) of death, I can also examine the timing of the neonatal mortality effects by constructing cumulative mortality by hour and day of death. Figure VII plots the estimated DD effects on the (log) probability of death before a given hour or day. The effects for the entire neonatal period manifest immediately and are actually strongest within the first four hours of life. High-AFDC states had about a 15.5 percent reduction (-0.17 log points, s.e. = 0.03) in neonatal mortality by the fourth hour relative to low-AFDC states. Throughout the first day, however, this effect shrinks, most likely due to negative selection into

[31] The motivation for a test comparing weighted and unweighted estimates is to detect unmodeled parameter heterogeneity or other forms of misspecification, in which case the two estimators may disagree (DuMouchel and Duncan 1983; Solon et al. 2013). An example of this is in appendix 3, which shows that, for a specification based only on timing (i.e., one that omits the heterogeity exploited in my research design), the equality of weighted and unweighted estimates is rejected. My results are invariant to weighting, so I present weighted results because they are more precise.

[32] Appendix table 2.4 shows that these estimates are similar across specifications.

the group of surviving infants: the weakest babies saved in the first hours do not survive the first day. The second panel shows that almost all of the neonatal mortality effect is present after the first day.

In contrast to the strong negative effects on very short-run infant mortality, column 2 of table IV shows no differential reduction by AFDC group in deaths after 28 days (post-neonatal mortality).[33] This may seem strange, since, like neonates, post-neonatal infants died of causes that were easily addressed by primary care. Two factors help explain this result. First, the dynamic selection effects apparent in figure VII likely bias the post-neonatal results toward zero. Second, Almond et al. (2006) show that black post-neonatal mortality in the South began declining in 1965 because of federally-mandated hospital desegregation. Once hospitals in a state were desegregated, Medicaid may have had little room to reduce post-neonatal mortality because infants with acute, life-threatening conditions could already obtain effective care at hospitals. Labor and delivery, which are much more connected to *neonatal* deaths and were commonly practiced outside the hospital, may have responded Medicaid's financial incentives over and above the effects of desegregation on hospital care for sick infants (see below).[34]

Several pieces of evidence point to increased survival *conditional on health* at birth as the primary channel for Medicaid's neonatal mortality effects rather than improvements in health at birth. Columns 3 and 4 of table IV show that Medicaid implementation had small, insignificant effects on the probability that nonwhite infants were classified as "very low birth weight" (< 1,500 grams or 3.3 pounds) or "low birth weight" (< 2,500 grams or 5.5 pounds).[35] Any effect that Medicaid did have on

[33] Results from a pooled model cannot reject the equality of the two DD estimates (*p*-value = 0.30), but the point estimates differ by a factor of four: -0.08 versus -0.02.

[34] Also note that desegregation itself is not a likely mechanism for my results because it implies relative increases in access to medical care for black families in the most segregated states, which are also primarily low-AFDC states. In other words, the effects of desegregation should have been strongest in my "control" group, which would bias the Medicaid estimates toward zero (and, as noted above, the results are robust to dropping the South).

[35] Birth weight is available in natality microdata from 1968 on, and was entered from printed volumes of Vital Statistics of the United States for 1959-1967. About one percent of births are missing birth weight information and the results in table IV assume they are missing at random. The share missing, however, is unrelated to the high-AFDC/post-Medicaid interaction (coefficient = 0.0005, s.e. = 0.005), which suggests that missing birth weight information is not driving the results. Appendix figure 2.13 presents event-study estimates for the birth weight variables.

birth weight cannot explain the mortality effects which are robust to controlling for birth weight variables (DD estimate = -0.064, s.e. = 0.020).[36] These results differ from evidence based on the timing of Medicaid implementation (Decker and Gruber 1993) and expanded Medicaid eligibility for poor mothers (Currie and Gruber 1996b). Nevertheless, they are consistent with the skepticism expressed by some perinatal epidemiologists about the effect of prenatal care on birth weight (Fiscella 1995; Alexander and Kotelchuck 2001), with evidence from the introduction of national health insurance in Canada (Hanratty 1996), and with Aizer et al. (2004) who find that improvements in hospital quality reduced neonatal mortality and prematurity among black Medicaid recipients, but had no effect on the probability of low birth weight.

Additional evidence consistent with Medicaid improving care at birth is in column 5 of table IV, which shows that Medicaid reduced nonwhite maternal mortality. Most causes of maternal death relate specifically to complications during or after delivery (hemorrhaging during birth or infections afterward) or to conditions treatable prior to labor (such as ectopic pregnancy), and should therefore reflect improvements in acute care at delivery. The maternal mortality models are estimated in levels rather than logs since many state-race-year cells have zero maternal deaths, especially after the mid-1960s.[37] The grouped event-study estimates are relatively noisy, but the DD estimate suggests that nonwhite maternal mortality (maternal deaths per 100,000 women) fell by about 1.57 deaths, or 20 percent relative to baseline rate of 8.17 deaths.[38] This result strongly suggests that the mechanisms for infant survival have to do with improvements in hospital care that also benefitted mothers (also see Kutinova and Conway 2008).

[36] This specification also addresses the potential bias from of differential selection induced by legalized abortion. Furthermore, the neonatal effects are also robust to dropping California, New York, and Washington, the states in the estimation sample that legalized abortion before the 1973 Roe v. Wade decision. See appendix table 2.2.

[37] Maternal mortality is age-adjusted using the national age distribution of women 15-54 in 1960. After 1970, about one third of cells have zero nonwhite maternal deaths, but this only represents about 5 percent of the nonwhite female population.

[38] Appendix figure 2.14 shows the maternal mortality event-study estimates. The effects appear to be concentrated outside the west region, where many states have very few maternal deaths. Dropping the western states yields event-study estimates with much smaller pre-Medicaid coefficients and a slightly larger treatment effect (-1.93, s.e. = 0.68).

One additional data source allows a direct test of the claim that hospital care is an important mechanism for the effects in table IV, as well as the ability to compare outcomes across different types of mothers. The 1964-1969 and 1972 National Natality Surveys (or the National Natality Followback Survey, NNFBS) contain demographic, socioeconomic, and medical variables for a sample of 3,821 nonwhite births over the seven survey years.[39] In 1967, 18 states provided AFDC (and therefore Medicaid) to mothers pregnant with their first child. Therefore, comparing results for covered births (higher-order births and first births in states that provided AFDC to first-time pregnant mothers) and non-covered births provides an additional test of whether the effects are due to Medicaid. Table V contains the results from linear probability models that contain the same fixed effects included in equation 1 as well as individual-level covariates: mother's age dummies, dummies for plural and first births, a dummy for the baby's sex, and separate sets of family income dummies for each year. The coefficients of interest are triple interactions between a post-Medicaid dummy, a high-AFDC dummy, and dummies for groups defined by poverty status and the possibility of perinatal Medicaid coverage.

Column 1 of table V shows that poor nonwhite mothers whose births were covered by Medicaid were more likely to give birth in an institution (almost always a hospital). AFDC-covered births to poor mothers were almost 7 percentage points more likely to occur in a hospital after Medicaid in high-eligibility states relative to low-eligibility states (s.e. = 0.027, baseline hospital probability is 0.8), but births not covered by Medicaid were unaffected. This effect also appears in aggregate data on hospital births (appendix figure 2.15). The estimates are strongest in the South where most of the racial convergence in hospital births occurred, but there is a positive trend break in the estimates using all

[39] The NNFBS sampling frame is "legitimate births", meaning that the mothers were married at the time of the birth. While AFDC would have primarily covered illegitimate births, there is still some overlap between the NNFBS sample and categorically eligible mothers. Two thirds of AFDC mothers in the 1967 Characteristics Survey had been married at some point, and about 13 percent of nonwhite respondents in the 1967-1969 NNFBS report receiving welfare income (compared to 11 percent of nonwhite women overall). Furthermore, mothers could have misreported marital status to welfare authorities or on the child's birth certificate.

states as well. Note that the movement of births into hospitals cannot explain the entire neonatal morality effect, which is robust to dropping the South. No data exist to quantify two other hospital-based channels through which Medicaid may affect infant survival: improved care at a given hospital (Currie and Gruber 2001) and sorting of newly insured mothers into better hospitals (Aizer et al. 2004).[40]

Medicaid-induced improvements in the medical care at birth result rationalize all of the infant results presented so far: strong reductions in immediate mortality rates, no significant changes in post-neonatal mortality or birth weight, and reductions in maternal mortality. These results also match the conclusions from perinatal epidemiological research which finds that changes in the distribution of fitness at birth account for only a small share of neonatal mortality declines since 1950 (Lee et al. 1980; Williams and Chen 1982; David and Siegel 1983; Cutler and Meara 1999). Medicaid implementation can help explain both the aggregate changes in race-specific neonatal mortality and the important contribution of survival conditional on health at birth.

C. Results for Younger and Older Nonwhite Children

Section II.D also documented high mortality rates for healthcare-amenable conditions among nonwhite children. Columns 1 and 2 of table VI show that Medicaid's effects on this group are concentrated among younger children ages 1 to 4. Grouped event-study estimates for younger child mortality (column 1 of panel A) show a small and insignificant pre-Medicaid coefficient (0.01, s.e. = 0.05) and sharp reduction in mortality after Medicaid (p-value = 0.007) with a corresponding DD

[40] Consistent with evidence in table IV, column 2 provides no evidence that Medicaid implementation affected the probability of low birth weight. Column 3 shows that pre-term births to poor women fell, but, that this effect was present for both covered *and* non-covered births (although the latter coefficient is not distinguishable from zero). Gestational age is also missing for about 5 percent of the nonwhite sample and is subject to potentially time-varying measurement error that does not apply to directly measured outcomes such as birth weight.

estimate of -0.14 (s.e. = 0.05). Column 2, however, shows no evidence of an effect for children ages 5-14 (DD estimate = -0.01, s.e. = 0.03.[41]

Columns 3 through 6 bear out another prediction from section II.D: that internal-cause mortality responds more to Medicaid than external cause mortality. Nearly all of Medicaid's effect on younger nonwhite children comes from reductions in internal-cause deaths (column 3). The DD effect on internal-cause mortality is a 15.6 percent reduction and is very precisely estimated (-0.17 log points, s.e. = 0.04), while the effect on external-cause mortality is smaller (-0.09; column 4) and not distinguishable from zero (s.e. = 0.06).

The final two columns suggest that the internal-cause effects arise mainly through Medicaid's effect on infectious disease mortality. Columns 5 and 6 split internal-cause deaths into those that are treatable and untreatable according to Beeson (1980).[42] Almost 90 percent of internal causes are treatable by this measure, two-thirds of which are infectious diseases. The treatment effects on younger nonwhite child mortality are driven by a 14-percent reduction in treatable cause mortality (-0.15 log points, s.e. = 0.04). The reductions in the grouped-event-study specification (panel A, column 5), however, are about 20 percent relative to the year before Medicaid implementation (the smaller DD estimate arises because of a negative coefficient in the 6 years before Medicaid). Estimates for untreatable causes, on the other hand are very noisy (column 6). The DD estimate is large and negative, but all of the grouped-event-study point estimates are positive and the DD restrictions are strongly rejected.[43]

[41] See appendix figure 2.12 for event-study estimates and table 2.5 for alternative specifications for younger child mortality.
[42] Beeson categorizes a range of conditions according to a ten-point scale of treatability in 1975. I assume all internal-cause deaths are treatable unless their 1975 treatment falls in the bottom 6 categories of treatability. Treatable conditions generally meet one of the following criteria: treatment suppresses or controls disease, but must be maintained indefinitely; treatment of manifestations substantially improved and/or diversified; effective treatment in most circumstances; effect preventive treatment. Untreatable conditions include all cancers except leukemia; degenerative conditions of the central nervous system such as multiple sclerosis; certain chronic conditions such as asthma or rheumatic fever; infectious conditions such as coccidiosis ("valley fever"); and some rare parasitic diseases.
[43] Appendix figures 2.16 and 2.17 present event-study estimates associated with columns 3-6 of table VI.

Fatal infectious diseases at this time were most often treated with antibiotics or other drugs when detected early enough, so in order to achieve the mortality reductions in table VI, Medicaid would have had to provide such care. To test this, table VII provides additional first-stage DD estimates of Medicaid's effect on the utilization of four specific services: hospital admission, physician visits, prescription drugs, and dental services. Consistent with the reductions in internal-cause and infectious-disease mortality in table VII, increases in utilization in high- versus low-eligibility states were largest for physician visits (0.025, s.e. = 0.008) and prescription drug use (0.030, s.e. = 0.008). Hospital admissions and dental visits increase slightly, but the effects are much smaller than for outpatient physician visits and prescription drugs. Thus, the increased patterns of public health care use correspond to the types of care that were effective in reducing the types of mortality that actually decreased for young children after Medicaid.

V. Evidence on Potential Threats to Identification

The results presented above show that high- and low-AFDC states were comparable before Medicaid, but diverged only after it was implemented. The variety of sensitivity checks and consistency across specifications provide strong evidence against the typical threat to difference-in-difference models: differential *trends* in the treatment and control groups. The pattern of estimates by age, race, cause of death, and, for infants, birth order, was also consistent with expectations about the causal effects of Medicaid implementation. The remaining threats to identification, therefore, are variables that affect the mortality of the same groups covered by Medicaid, that differ in high and low-AFDC states, and that change sharply at the same time as Medicaid implementation (but are not caused by it).

A. Direct Evidence on Other Federal Spending

The level of AFDC receipt in the year of Medicaid implementation may signal states' willingness to *change* their policies toward the health and mortality of the poor. In this case, the estimates of γ_y^k would

capture the mortality-reducing effects of other policies enacted more in high-AFDC states than in low-AFDC states instead of Medicaid's effect.

To test this hypothesis, I estimate versions of equation 1 using recently collected measures of per-capita expenditures or participation rates for four major programs that could have also affected child mortality.[44] Panel A of figure VIII shows the results for per-capita federal expenditures for Community Health Centers (CHC), other health programs funded by the Community Action Program (CAP), and Head Start (per 1,000 children ages 1-9). For comparison, I also include estimates for public insurance expenditures on children per 1,000 children ages 1-19 (the expenditures version of the first-stage results in figure V). Panel B shows the results of similar regressions for participation rates in the Food Stamp Program, and for the white and non-white AFDC rates used to calculate D_s (alongside the public insurance estimates from figure V).

Changes in per-capita expenditures on other health-related programs are both uncorrelated with Medicaid implementation and small, especially relative to the large effects on public insurance spending and utilization. This is especially reassuring for two programs that have been shown to affect infant and child health: Head Start (Ludwig and Miller 2007) and Food Stamps (Almond et al. 2011).[45] The event-study results for both Head Start spending (panel A) and Food Stamp participation (panel B) are small, indistinguishable from zero, and do not change sharply in the year of Medicaid implementation. It is unlikely that the expansion of related federal programs explains the mortality results in section IV.

Panel B also shows that changes in AFDC rates themselves cannot explain the mortality results. Neither white nor nonwhite AFDC rates change much on average in high-eligibility versus low-

[44] For the expenditures and participation rates that are not measured by race, the binary AFDC groups are created using the overall AFDC rate (as in the first-stage results) rather than race-specific AFDC rates.

[45] The DD estimate for community health center spending is statistically significant, but it is more than an order of magnitude smaller than the public insurance estimate. This also overstates the per-capita CHC spending for children because is an average that includes much higher expenditures for older users. Furthermore, Bailey and Goodman-Bacon (forthcoming) find no evidence that CHCs affect child or infant mortality, which means that even a large change in funding would not be a plausible explanation for the mortality reductions in section IV.

eligibility states (the white DD estimate is 0.001, s.e. = 0.002, and the nonwhite DD estimate is -0.01, s.e. = 0.01). Several years after Medicaid, nonwhite AFDC rates converge to some extent, but unlike public insurance use and mortality rates, they do not change sharply at the time of Medicaid implementation. Furthermore, evidence on the relationship between welfare receipt and health is mixed (Currie and Cole 1993; Bitler et al. 2005; Leonard and Mas 2008), so even if AFDC rates were correlated with Medicaid timing, it is not clear that this could generate large mortality reductions.[46]

B. Indirect Evidence Adding Controls for State-Level Welfare Programs

The results in figure VIII show that several observable federal programs cannot account for the Medicaid estimates. Another approach to rule out alternative explanations is to add other measures of state welfare programs to equation 1. If the results are spurious, then other transfer program measures should be highly correlated with any omitted variables that account for the post-Medicaid mortality reductions, and the main treatment effects should fall toward zero.

The first approach is a falsification test that asks whether another measure of state welfare rates can account for the post-Medicaid mortality changes for nonwhite children. I add interactions of the event-time variables with a high-*white*-AFDC dummy to the regressions for nonwhite mortality rates. If high-AFDC states simply expanded their social safety net in ways that increased the availability of other (omitted) services that were the true cause of mortality reductions, then the white and nonwhite treatment variables would contain essentially the same information about omitted variables that drive the nonwhite mortality rates. The results in panel A of table VIII show that the main treatment effects are

[46] Appendix table 2.7 uses data from the 1970 Census to show that cross-state migration for nonwhite families between 1965 and 1970 was not related to the high- and low-AFDC distinction. The share of families who move from low- to high-AFDC states is very small (less than 2 percent) and almost identical to the share of families who move from high- to low-AFDC states. This is true for all parents, parents of young children, single parents of young children and single parents on welfare with young children. This is consistent with the evidence in appendix figure 2.9 that per-recipient benefits did not increase in higher-categorical eligibility states, and suggests that selective migration cannot explain the mortality results.

unchanged when white-AFDC interactions are included, and that the effects in high-white-AFDC states are small and insignificant.

The history of AFDC, however, suggests that white and nonwhite participation may represent different omitted factors (section III.B), and that omitted determinants of mortality may be specifically correlated with nonwhite AFDC and state-level Medicaid adoption. Because the identification strategy is based on nonwhite AFDC rates at one point in (event) time, I can address this concern by including the actual state-by-year nonwhite AFDC rate as an additional covariate in equation 1. If changes in nonwhite welfare rates stand in for underlying shifts in factors such as discrimination, industrial structure or safety-net policy that also affect mortality, then including the observed nonwhite AFDC rate will capture all the relevant variation in mortality during this time period, and eliminate the estimated Medicaid effects. Panel B of table VIII presents DD estimates that include the state-by-year nonwhite AFDC rate *and* its interaction with a post-1966 dummy, which further allows the nonwhite AFDC controls to have different effects during the Medicaid period (Moffitt 1987). The treatment effects are only slightly reduced by these flexible controls and remain statistically significant, suggesting that the changes in nonwhite AFDC during the 1960s cannot account for the strong correlation between initial categorical eligibility and the timing of Medicaid implementation.

VI. CONCLUSION: INTERPRETING THE MORTALITY EFFECTS OF MEDICAID IMPLEMENTATION

The preceding evidence suggests that Medicaid implementation succeeded in increasing public insurance coverage and reducing mortality among children. But given that previous studies have estimated effects for similar populations, how do these results affect our understanding of how public insurance influences mortality generally?

A. The Average Treatment Effect of Medicaid on the Mortality of Treated Children

Section I argued that existing estimates of Medicaid's effect on infant and child mortality are too large to be attributed to new insurance coverage alone. This conclusion is based on the proportional average treatment effects on the treated (ATET) of Medicaid coverage. This parameter is comparable across studies because it is not tied to the scale of a particular policy change or to the baseline mortality rate of different target populations. It is also a useful check on the plausibility of attributing a given result *entirely* to changes in insurance because the proportional ATET cannot be below -100 percent, as this implies that Medicaid reduces mortality by more than its baseline level.

To calculate the ATET, I first divide the DD mortality estimate for nonwhite children by the appropriate first-stage estimate for insurance coverage.[47] This assumes that no categorically eligible Medicaid recipients dropped private insurance coverage. This type of crowd-out is not a concern in the 1960s when private insurance coverage among AFDC recipients, whose full-time employment rate was below 5 percent (DHEW 1963), was certainly close to zero.[48] I also adjust for the higher mortality rates of Medicaid recipients using survey data on mortality by income (see appendix 4).

Figure IX plots estimates of the ATET from this paper and from the three most closely related Medicaid papers (Currie and Gruber 1996a; b; Meyer and Wherry 2013).[49] I construct confidence intervals using a parametric bootstrap procedure (Efron and Tibshirani 1993) that uses 10,000 draws of the reduced-form and first-stage estimates from normal distributions with means and standard deviations equal to the point estimates and standard errors, and calculates the ATET for each draw (see appendix

[47] Here I use the estimates from the continuous AFDC specification shown in column 3 of appendix table 2.3. The reduced-form effect (-1.52, s.e. = 0.38) and first-stage effect (3.52, s.e. = 0.95) from this specification are per percentage point of the AFDC rate, and so no rescaling of the first-stage (for all children) is required to make it comparable to the mortality effects for nonwhite children. As discussed above, these estimates are more precise than the simpler binary specification because they leverage more AFDC variation.

[48] This is also borne out in figure I, which shows that the magnitude of public coverage gains and reductions in uninsurance correspond closely in the 1960s and 1970s (but not since), reflecting the limited scope for crowd-out.

[49] For more recent papers, I use first-stage estimates for any health insurance rather than Medicaid coverage and adjust them by a factor of 0.85 to account for underreporting of Medicaid in survey data (Card et al. 2004; Davern et al. 2007).

4).[50] This method allows me to calculate confidence intervals for other papers without resampling from their data. I calculate confidence intervals using a modified percentile method (Johnston and DiNardo 1997), because the distribution of the ATET is not symmetric. This highlights an advantage of the parametric bootstrap over an approach based on a linear approximation (delta method) which would yield a symmetric and misleading confidence interval.

The ATET estimates reaffirm that Medicaid had a significant negative effect on nonwhite infant and child mortality rates, and the magnitudes suggest that the results could be due to Medicaid's insurance coverage alone. The ATETs imply a 24 percent mortality reduction for nonwhite children under 14, a 38 percent reduction for younger nonwhite children (ages 1-4) and a 25 percent reduction for nonwhite neonates. The confidence intervals never include zero, and for both the overall and neonatal estimates, the lower end of the confidence interval does not cross the maximum possible value (-100 percent).

The ATETs from the 1980s expansions on the other hand are five times as large. The estimates imply a 99 percent mortality reduction for infants, a 188 percent reduction for children, and an 84 percent reduction for black teens.[51] This is surprising since the AFDC children who gained insurance because of Medicaid implementation were poorer and less healthy than many of the groups who gained coverage in the 1980s. Improved technology, particularly artificial lung-surfactant for premature infants may explain some of the bigger effects in the 1980s (Bharadwaj et al. 2013). Another interpretation is that some of the other health-related consequences of the 1980s expansions—increased consumption for crowd-out families or take-up of other programs, for example—were not at work in the 1960s, when

[50] The confidence intervals in figure IX assume zero correlation between the components of the ATET. Appendix 4 presents confidence interval estimates under a series of assumptions about the correlation between the reduced form and first-stage effects. The confidence intervals never include zero for any value of the correlation, although they are wider when the two parameters are positively correlated. Notably, the confidence interval for age-adjusted mortality also never crosses -100 percent. I thank Alejandro Molnar for this suggestion.

[51] The RD mortality estimate in Meyer and Wherry (2013) is based on differences in cumulative eligibility and mortality rates observed years after the discontinuity arose. The RD estimates for insurance coverage, however, refer to contemporaneous coverage (Card and Shore-Sheppard 2004). The dynamics of Medicaid participation, therefore, mean that the true longer-run first stage (cumulative participation) could be larger or smaller than assumed here.

categorically eligible Medicaid families spent little on medical care prior to gaining insurance and already received welfare by definition. This would suggest that the effects documented here reflect the effect of Medicaid coverage itself, and that Medicaid's effect per *eligible person* in the 1980s reflects additional causal channels.[52]

Even though the ATET estimates are smaller than previous studies, they imply large individual health effects compared to other interventions. A 24 percent reduction in neonatal mortality is equivalent to the effect of gaining a full pound in birth weight (Almond et al. 2005); approximately ten times the effect of Food Stamp implementation on the birth weight of treated black infants (Almond et al. 2011). Chay and Greenstone (2003) find that improvements in air quality that followed the 1970 Clean Air Act reduced neonatal infant mortality by about 18 percent. The desegregation of Southern hospitals led to a larger reduction—about 50 percent—in black post-neonatal mortality (Almond et al. 2006), which may follow from the clear course of treatment for babies with gastrointestinal disease. The infant mortality effects are also comparable to those of many public health programs/phenomena. For example, the installation of lead pipes at the turn of the century increased infant mortality by "between 25 and 50 percent" (Troesken 2003), and investments in water sanitation facilities on Indian reservations could have reduced infant mortality by as much as 25 percent (Watson 2006).

B. Medicaid's Aggregate Costs and Benefits

The estimates not only imply an important reduction in individual-level mortality risk, but also a major role for Medicaid implementation in aggregate mortality changes in the 1960s and 1970s. A proportional mortality reduction per nonwhite Medicaid child of 24 percent, combined with a national share of nonwhite children on Medicaid of about 32 percent (the product of the national child Medicaid share, 12 percent, and the ratio of Medicaid use rates for nonwhite children under 14 to all children

[52] Scaling the ITT by eligibility instead of new coverage yields a proportional reduction in mortality of 28 percent per eligible infant (Currie and Gruber 1996b, pp. 1276) and 34 percent per eligible child (Currie and Gruber 1996a, pp. 454).

under 19 in the 1976 SIE, 2.7), suggests that Medicaid reduced national nonwhite child mortality rates by 8 percent in each of its first 10 years. Using the ATET estimates to construct a counterfactual nonwhite child mortality rate suggests that, without Medicaid, the racial child mortality gap in 1979 would have been 116 deaths per 100,000 children. The actual gap was 99 deaths per 100,000, which implies that Medicaid reduced the 1979 racial gap in child mortality by 16 percent (appendix table 2.7)

These calculations all refer to Medicaid's effect on period mortality rates, while the actual benefits accrued over time. Comparing the observed number of nonwhite child deaths to the counterfactual number in each year suggest that, between 1966 and 1979, 22,000 nonwhite deaths were averted due to Medicaid (1,600 deaths per year). Most of these deaths would have occurred among neonatal infants and young children, for whom the remaining life expectancy in 1966 was about 65.5 (NCHS 1966), which implies a gain of 1.43 million life-years saved.

The public insurance data show that, through 1976, Medicaid spent about $4 billion (in 2012 dollars) per year on *all* children ages 0 to 19. Assuming that expenditures on children ages 0 to 14 were proportional to their share of child Medicaid recipients (78 percent in 1976), and that *no* white children benefited, this implies a cost per death averted of about $1.94 million and a discounted cost per life-year saved of about $68,000.[53] Similar calculations by age group show that infant deaths were significantly cheaper to avoid than deaths among young children: the cost per death averted is $270,000 for nonwhite neonates and $760,000 for young nonwhite children. The comparable estimates from Currie and Gruber (1996b; 1996a) are about $1.7 million and $2.6 million (2012 dollars) per infant and child death averted, which suggests that Medicaid implementation achieved its mortality reductions at costs that were either significantly lower than or comparable to those of more recent expansions.

[53] A discount rate of 3 percent and the assumption that the private value of additional life years is constant across ages implies that the present discounted dollar value of 65 additional life years is equivalent to an immediate payout of 28.7 times the value of an additional year: $(1-0.97^{65})/(1-0.97) = 28.7$. Note that the costs reflect age-group-specific spending on all children, not just nonwhite children. Expenditure data by race and age would imply much lower costs for nonwhite children.

These costs refer to the contemporaneous expenditures relative to life years gained, but exclude the potential for Medicaid to have later-life benefits. Medicaid's benefits may extend into later life health (Miller and Wherry 2014), educational attainment (Cohodes et al. 2014), and increased tax receipts (Brown et al. 2014). To the extent that early health investments of Medicaid implementation complement later-life health production, human capital investments and labor supply, the life-cycle benefits of Medicaid may add significantly to the contemporaneous benefits documented here.

This paper provides new evidence on the relationship between Medicaid and mortality using the original introduction of the program between 1966 and 1970. The results are the first to examine Medicaid *implementation* and suggest that the program was quite well targeted during this period: nonwhite infants and children suffered very high mortality rates in the 1960s, used Medicaid the most, and experienced the largest mortality reductions. While more recent policy changes have had similar qualitative effects on infant and child mortality, this paper's estimates are among the only quasi-experimental results that are small enough to be attributed to Medicaid coverage itself. These findings presumably understate Medicaid's broader effects because they only measure benefits in terms of mortality rather than reductions in morbidity. Therefore Medicaid, like several other federal health and anti-poverty programs established under the Great Society, played an even larger role in reducing racial and socioeconomic disparities in health and mortality in the 1960s and 1970s.

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Table I. High-Eligibility and Low-Eligibility Groups by Race, Year of Medicaid Implementation

		White AFDC Rate	
		Low	**High**
Nonwhite AFDC Rate	**Low**	*13 States:* District of Columbia, Georgia, Illinois, Louisiana, Maryland, Michigan, New Hampshire*, North Carolina, Ohio, South Carolina, Texas, Virginia, Wisconsin	*11 States:* Idaho, Kentucky, Maine*, New Mexico, Oregon, Pennsylvania, Tennessee, Vermont*, Washington, West Virginia, Wyoming
	High	*11 States:* Arkansas, Connecticut, Delaware, Florida, Indiana, Kansas, Mississippi, Montana, Nebraska, Nevada, North Dakota	*13 States:* Alabama, California, Colorado, Iowa, Massachusetts, Minnesota, Missouri, New Jersey*, New York, Oklahoma, Rhode Island, South Dakota, Utah

Notes: *Maine, New Hampshire, and Vermont are excluded from the nonwhite estimation sample because less than 1 percent of their populations were nonwhite. New Jersey is excluded from all models because it lacks race codes in its mortality files for 1962 and 1963. Alaska, Hawaii, and Arizona are also excluded from all models. The high- and low-AFDC distinction comes from taking the set of race-specific of state AFDC rates in the year of Medicaid implementation and splitting states at the median.

Table II. Balancing Test: The Relationship between Pre-Medicaid State Characteristics and AFDC-Based Categorical Eligibility

		Low-AFDC	High-AFDC	H_0: (1)=(2) (*p*-value from *t*-test)
		(1)	(2)	(3)
		A. By White AFDC Rate, t^*		
1)	White Child Poverty Rate, 1960	0.22	0.25	0.29
		(0.10)	(0.09)	
2)	White Children in Single Mother Households, 1960	0.03	0.03	0.01
		(0.01)	(0.01)	
3)	Average White AFDC Benefit, 1967	133.44	158.74	0.04
		(47.36)	(40.81)	
4)	Change in White Child Poverty Rate, 1950 to 1960	-0.14	-0.14	0.94
		(0.07)	(0.05)	
5)	Change in White Child Mortality Rate, t^*-6 to t^*-1	-0.13	-0.13	0.77
		(0.08)	(0.07)	
6)	Change in White Infant Mortality Rate, t^*-6 to t^*-1	-0.09	-0.08	0.61
		(0.10)	(0.07)	
		*B. By Nonwhite AFDC Rate, t**		
7)	Nonwhite Child Poverty Rate, 1960	0.65	0.62	0.53
		(0.20)	(0.17)	
8)	Nonwhite Children in Single Mother Households, 1960	0.16	0.15	0.44
		(0.05)	(0.05)	
9)	Average Nonwhite AFDC Benefit, 1967	145.86	159.29	0.38
		(38.44)	(58.75)	
10)	Change in Nonwhite Child Poverty Rate, 1950 to 1960	-0.08	-0.15	0.12
		(0.15)	(0.16)	
11)	Change in Nonwhite Child Mortality Rate, t^*-6 to t^*-1	-0.12	-0.07	0.40
		(0.19)	(0.19)	
12)	Change in Nonwhite Infant Mortality Rate, t^*-6 to t^*-1	-0.14	-0.10	0.38
		(0.15)	(0.17)	
		*C. By Overall AFDC Rate, t**		
13)	Change in Hospital Beds per Capita, 1960-1965	0.12	0.21	0.39
		(0.33)	(0.41)	
14)	Change in Child Public Insurance Rate, 1963-1965	0.01	0.01	0.89
		(0.01)	(0.01)	

Notes: Columns 1 and 2 contain unweighted means in high- and low-AFDC states. Standard deviations are in parentheses. Panel A groups states by the median value of the white AFDC rate in the year of Medicaid implementation calculated as the number of AFDC cases with a white payee divided by the white female population 20-54. Panel B groups states are grouped by the nonwhite AFDC rate calculated the same way. Panel C uses the overall AFDC rate because the medical variables are not available by race. Column 3 contains the *p*-value from a *t*-test of the equality of the means in columns 1 and 2. Average AFDC benefits are family-size-adjusted averages of benefits reported in the 1967 AFDC Recipient Characteristics Study. Sources: 1950 and 1960 Census Integrated Public Use Microsample (Ruggles et al. 2010), American Hospital Association (various years), DHEW (1963-1976).

Table III. Medicaid's Effect on Log Nonwhite Age-Adjusted Child Mortality Across Specifications

	(1)	(2)	(3)	(4)	(5)
	A. Grouped Event-Study Estimates				
Pre-Medicaid					
(Years -7 to -2)*High-AFDC	0.02	0.01	0.000	-0.04	0.01
	[0.03]	[0.03]	[0.022]	[0.04]	[0.02]
Post-Medicaid					
(Year 0)*High-AFDC	-0.02	-0.03	-0.03	-0.02	-0.03
	[0.02]	[0.02]	[0.02]	[0.11]	[0.02]
(Years 1 to 4)*High-AFDC	-0.06	-0.06	-0.07	-0.11	-0.08
	[0.02]	[0.03]	[0.03]	[0.05]	[0.03]
(Years 5 to 9)*High-AFDC	-0.11	-0.10	-0.10	-0.14	-0.12
	[0.04]	[0.05]	[0.04]	[0.05]	[0.05]
R^2	0.74	0.94	0.96	0.84	0.97
	B. Difference-in-Differences Estimates				
Post-Medicaid*High-AFDC	-0.10	-0.09	-0.09	-0.10	-0.07
	[0.04]	[0.05]	[0.03]	[0.04]	[0.03]
R^2	0.67	0.94	0.96	0.84	0.97
DD Test (*p*-value)	0.18	0.50	0.45	0.62	0.41
Covariates	High-AFDC FE, Time-to-Medicaid Dummies	State FE, Medicaid-Timing-by-Year FE + X_{st}	(2) + region-by-year FE	(3), unweighted	(3) + state-specific linear trends
Population Weighted?	Y	Y	Y	N	Y
Mortality Rate in High-AFDC States in *t*-1*	387 deaths per 100,000				

Notes: Panel A contains the estimated coefficient on interactions between groups of time-to-Medicaid dummies ($1\{t - t_s^* \in [a, b]\}$) and a dummy variable for high-AFDC states (D_s) from five specifications of the regression model described in Section III. Standard errors clustered by state are in brackets. The results correspond to the event-study results in figure VI. Effects are shown for the pre-Medicaid period (from seven to two years before Medicaid), the implementation year ("Year 0", when many Medicaid programs were only partially in place), years 1-4 and years 5-9. The estimates are normalized to zero in the year before Medicaid implementation (the omitted category). Periods more than seven years before Medicaid (in the latest-implementing states) or more than 9 years after (in the earliest implementing states) are grouped into endpoint dummies and their coefficients are not shown (see McCrary 2007). The sample contains 924 observations on 43 states (excluding Alaska, Hawaii, Arizona, New Jersey, New Hampshire, Vermont and Maine) between 1959 and 1979. The row labeled "DD Test" contains the *p*-value from an *F*-test of the constant-coefficient difference-in-differences restrictions: the pre-Medicaid coefficient is zero and post-Medicaid coefficients (*not* including year zero) are equal to each other. Failure to reject these restrictions suggests that estimating a constant treatment effect (for time 1-9) and forcing the pre-Medicaid period (-7 to -1) to equal zero are valid restrictions. The estimates of this specification are presented in panel B. The covariates are the same as in panel A except that the (Years -7 to -2)*High-AFDC variable is also omitted, a dummy for the year of Medicaid implementation is included (but not shown), and Post-Medicaid*High-AFDC refers to all event-years between 1 (the year *after* implementation) and 9. Although the unweighted estimates in column 4 are larger than the weighted estimates in column 3, a Hausman test does not reject the null hypothesis that they are equal for either the grouped event-study model (*p*-value = 0.24) or the DD model (*p*-value = 0.97) (Deaton 1997; Solon, Haider and Wooldridge 2013). Sources: See notes to figure VI.

Table IV. Medicaid's Effect on Nonwhite Infant Health

Dependent Variable:	(1) log Neonatal Infant Mortality Rate (deaths before 28 days)	(2) log Post-Neonatal Infant Mortality Rate (deaths 28 days to 1 year)	(3) log Very Low Birth Weight Rate (under 1,500 grams)	(4) log Low Birth Weight Rate (under 2,500 grams)	(5) Age-Adjusted Maternal Mortality Rate per 100,000 Women
		A. Grouped Event-Study Estimates			
Pre-Medicaid					
(Years -7 to -2)*High-AFDC	-0.003	0.04	-0.03	0.02	0.63
	[0.036]	[0.03]	[0.04]	[0.01]	[1.2]
Post-Medicaid					
(Year 0)*High-AFDC	-0.04	0.004	-0.10	-0.01	-0.34
	[0.04]	[0.053]	[0.06]	[0.02]	[1.26]
(Years 1 to 4)*High-AFDC	-0.06	0.02	-0.07	0.01	-1.04
	[0.04]	[0.04]	[0.05]	[0.02]	[1.19]
(Years 5 to 9)*High-AFDC	-0.09	0.01	-0.07	0.03	-1.05
	[0.03]	[0.07]	[0.05]	[0.02]	[1.59]
R^2	0.93	0.94	0.80	0.92	0.86
		B. Difference-in-Differences Estimates			
Post-Medicaid*High-AFDC	-0.08	-0.02	-0.04	0.004	-1.57
	[0.02]	[0.05]	[0.03]	[0.016]	[0.71]
R^2	0.93	0.94	0.80	0.92	0.86
DD Test (*p*-value)	0.30	0.50	0.71	0.12	0.85
Rate in High-AFDC States in *t*-1*	24.63	11.27	23.20	132.57	8.17

Notes: The table presents grouped event-study (panel A) and constant-coefficient DD estimates (panel B) of Medicaid's intention-to-treat effect on nonwhite infant outcomes based on equation (1). The specification includes state fixed effects, region-by-year fixed effects, Medicaid-timing-by-year fixed effects, per-capita income and per-capita hospital beds. Columns 1 and 2 contain estimates for the log of neonatal and post-neonatal infant mortality rates for nonwhite infants. Columns 3 and 4 contain estimates for the log of the share of nonwhite infants that weighted less than 1,500 grams (very low birth weight) and 2,500 grams (low birth weight). Column 5 contains estimates for the *level* of age-adjusted maternal mortality (per 100,000) among nonwhite women. In the average year (between 1959 and 1979) about one third of states have no nonwhite maternal deaths, which motivates the use of levels rather than logs. The final row shows the average level (not log) of the dependent variable among high-AFDC states in the year prior to Medicaid implementation. Sources: See notes to figure VI. Birth weight data from 1959-1967 are entered from printed volumes of Vital Statistics of the United States.

Table V. Medicaid's Effect on Nonwhite Birth Outcomes by Mother's AFDC Eligibility, 1964-1972

	(1)	(2)	(3)
Dependent Variable:	Born in an Institution	Low Birth Weight (Birth Weight < 2500 grams)	Premature (Gestation < 36 Weeks)
Post-Medicaid*High-AFDC:			
	Potentially Medicaid-Eligible Mothers		
Poor	0.065	-0.004	-0.076
	[0.027]	[0.052]	[0.026]
Non-Poor	-0.048	0.007	-0.006
	[0.034]	[0.06]	[0.059]
	Medicaid Ineligible Mothers		
Poor	0.012	-0.003	-0.090
	[0.044]	[0.085]	[0.118]
Non-Poor	0.041	-0.013	-0.010
	[0.029]	[0.088]	[0.088]
Observations	3,821	3,821	3,630
R^2	0.20	0.11	0.08

Notes: The table contains estimated coefficients from a linear probability model that includes triple-interactions between a dummy that equals one for all years after (but not including) the year of Medicaid implementation, an indicator for high-AFDC states and indicators for whether mothers were poor/non-poor and covered/not covered by AFDC. Most states (34) excluded first-time pregnant women from AFDC and, therefore, Medicaid. The definition of "Medicaid Eligible" in these results is a subsequent birth or a first birth in a state that provided AFDC to first-time pregnant mothers. The model also includes state fixed effects, separate year fixed effects for each Medicaid timing group, region-by-year fixed effects and dummies for 10 bins of family income interacted with year dummies, dummies for each year of the mother's age, an indicator for the sex of the child, and an indicator for plural births. Standard errors clustered at the state level are in brackets. The regressions are weighted by the sampling weights. Source: National Natality Followback Surveys 1964-1966 and 1972, National Natality Surveys 1967-1969.

Table VI. Medicaid's Effect on Nonwhite Child Mortality

	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable is the log Mortality Rate for:	Ages 1-4	Ages 5-14	Ages 1-4, Internal Causes	Ages 1-4, External Causes	Ages 1-4, Treatable Causes	Ages 1-4, Untreatable Causes
			A. Grouped Event-Study Estimates			
Pre-Medicaid						
(Years -7 to -2)*High-AFDC	0.01	0.10	0.012	-0.05	-0.06	0.40
	[0.05]	[0.05]	[0.065]	[0.09]	[0.07]	[0.13]
Post-Medicaid						
(Year 0)*High-AFDC	-0.04	-0.009	-0.10	0.02	-0.21	0.49
	[0.08]	[0.06]	[0.11]	[0.12]	[0.10]	[0.15]
(Years 1 to 4)*High-AFDC	-0.09	0.10	-0.15	-0.02	-0.18	0.14
	[0.05]	[0.04]	[0.07]	[0.09]	[0.09]	[0.11]
(Years 5 to 9)*High-AFDC	-0.17	0.04	-0.16	-0.22	-0.22	0.15
	[0.07]	[0.05]	[0.06]	[0.12]	[0.07]	[0.16]
R^2	0.86	0.76	0.88	0.57	0.88	0.64
			B. Difference-in-Differences Estimates			
Post-Medicaid*High-AFDC	-0.14	-0.01	-0.17	-0.09	-0.15	-0.20
	[0.05]	[0.03]	[0.04]	[0.06]	[0.04]	[0.08]
R^2	0.86	0.76	0.88	0.56	0.88	0.64
DD Test (*p*-value)	0.52	0.10	0.98	0.22	0.49	0.00
Mortality Rate in High-AFDC States in *t*-1*	155.43	57.14	94.06	61.37	76.15	17.91

Notes: The table presents grouped event-study (panel A) and constant-coefficient DD estimates (panel B) of Medicaid's intention-to-treat effect on nonwhite infant outcomes based on equation (1). For details on the specification and sources see notes to table III. Columns 1 and 2 contain estimates for the log of younger (ages 1-4) and older (ages 5-14) child mortality rates for nonwhite children. Mortality rates for older children are age-adjusted across the 5-9 and 10-14 age groups using the national shares in these bins in 1960. Columns 3 and 4 contain estimates for the log of internal- and external-cause mortality rates for younger nonwhite children. For the definition of internal and external causes see notes to figure IV. Columns 5 and 6 contain estimates for infectious- and non-infectious-disease mortality rates for younger nonwhite children, which together account for the vast majority of internal-cause deaths. For the ICD codes included in these groups see appendix 1. The final row shows the average level (not log) of the dependent variable among high-AFDC states in the year prior to Medicaid implementation. Sources: See notes to figure VI.

Table VII. Medicaid's Effect on Children's Public Insurance Utilization by Type of Service

	(1)	(2)	(3)	(4)
Dependent Variable is Share of Children with Public Payments for:	Hospital	MD Services	Prescription Drugs	Dental
Post-Medicaid*High-AFDC	0.004	0.025	0.030	0.012
	[0.002]	[0.008]	[0.008]	[0.006]
R^2	0.84	0.92	0.90	0.86
DD Test (*p*-value)	0.01	0.13	0.72	0.71

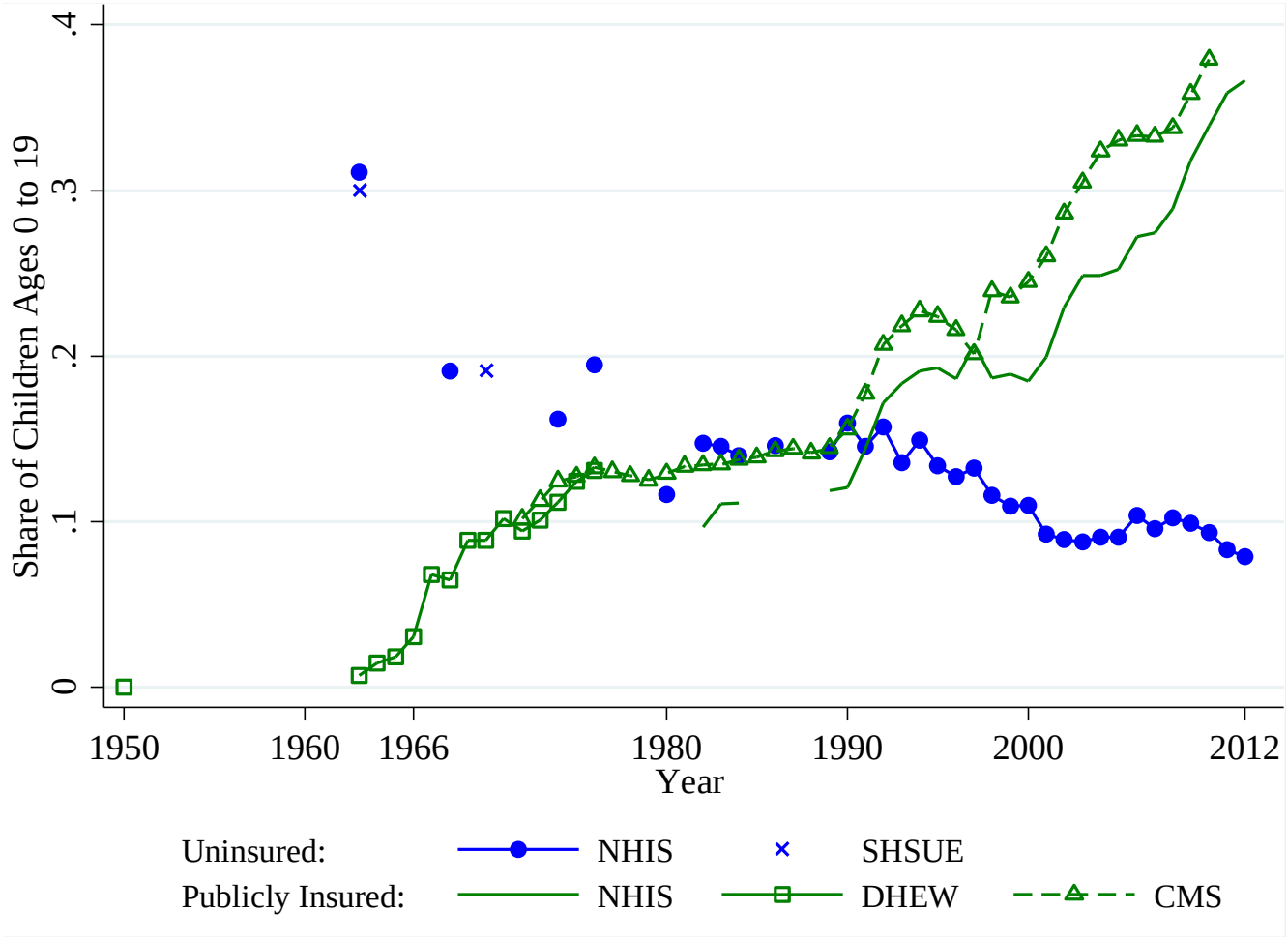
Notes: The dependent variable is the share of children who had public insurance payments made for the services described in columns 1-4, calculated from administrative reports described in appendix 1. The model includes state fixed effects, separate year fixed effects for each Medicaid timing group, per-capita income and hospital variables, and region-by-year fixed effects. The row labeled "DD Test" contains the *p*-value from an *F*-test of the difference-in-differences restrictions: the pre-Medicaid coefficient is zero and post-Medicaid coefficients (*not* including year zero) are equal to each other. The test is conducted using the coefficients from a grouped event-study model (not shown) as in tables III, IV and VI. Source: See notes to figure V.

Table VIII. Robustness of Medicaid's Mortality Effects to Time-Varying AFDC Controls

	(1)	(2)	(3)
Dependent Variable:	log Nonwhite Mortality, Ages 0-14	log Nonwhite Neonatal Infant Mortality	log Nonwhite Mortality, Ages 1-4
	A. Controlling for White-AFDC Medicaid Timing Interactions		
Treatment Effects:			
Post-Medicaid*High-Nonwhite-AFDC	-0.10	-0.16	-0.08
	[0.03]	[0.04]	[0.03]
Falsification Test:			
Post-Medicaid*High-White-AFDC	0.05	0.06	0.02
	[0.03]	[0.04]	[0.03]
R^2	0.96	0.86	0.93
	B. Controlling for State-by-Year Nonwhite AFDC Rate		
Post-Medicaid*High-Nonwhite-AFDC	-0.08	-0.09	-0.12
	[0.04]	[0.02]	[0.05]
R^2	0.96	0.93	0.86

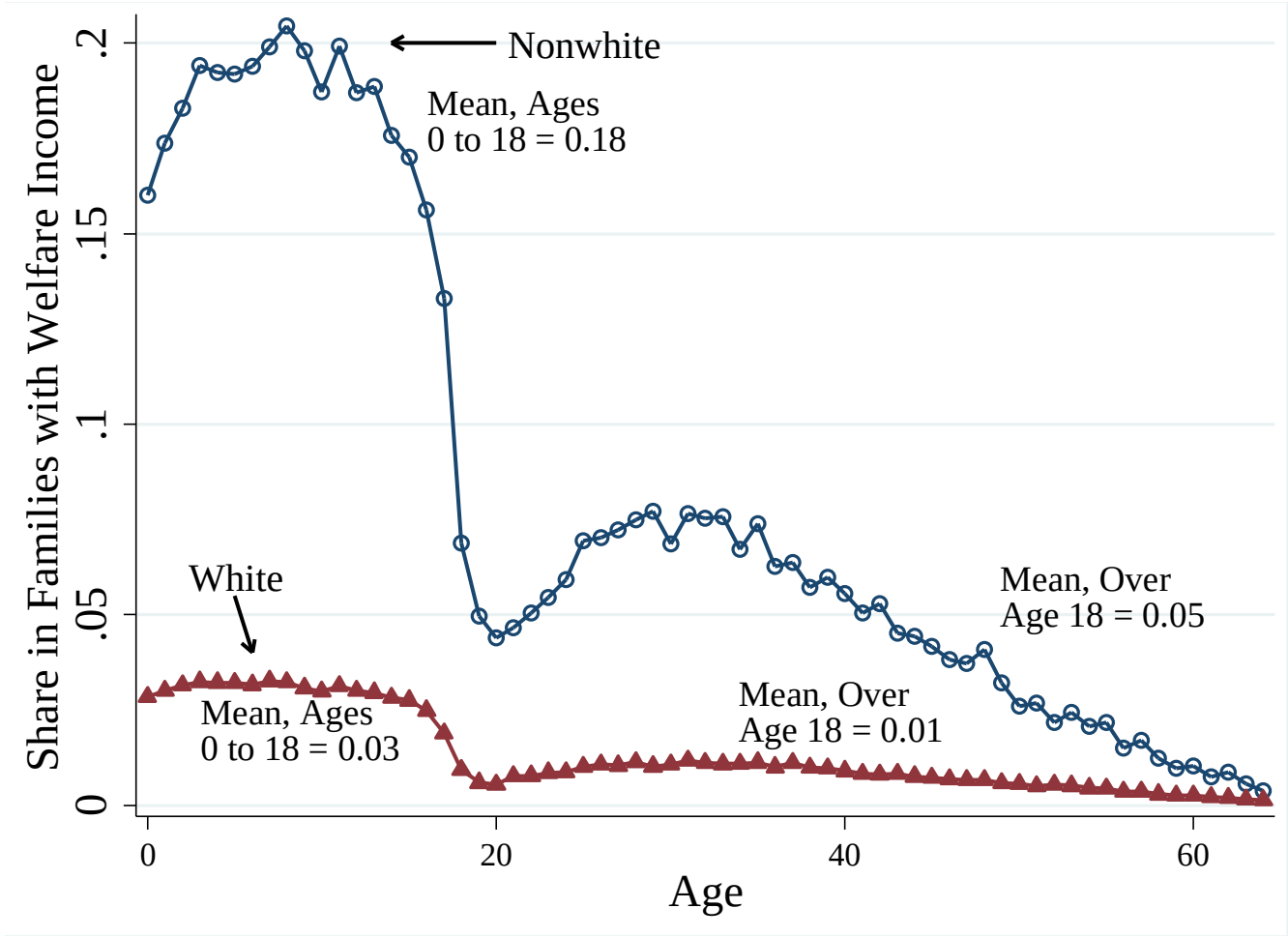
Notes: The table contains estimates from two specification tests. Panel A presents coefficients on interactions between a post-Medicaid dummy and an indicator for high-nonwhite-AFDC states (treatment effects) and high-white-AFDC states (falsification test). The estimated treatment effects for nonwhite mortality are robust to the inclusion controls for white AFDC rates before and after Medicaid implementation. Panel B presents estimated coefficients on interactions between post-Medicaid dummy and an indicator for high-nonwhite-AFDC states as in equation (1). The regressions also includes state-by-year nonwhite welfare rates ($AFDC_{st}$) and their interaction with a post-1966 dummy ($AFDC_{st} \cdot 1\{y \geq 1966\}$). These controls account for omitted factors that are correlated with levels and changes in specifically nonwhite AFDC rates, and any change in the relationship between these factors on mortality in the mid-1960s. The results show that the estimated treatment effects of Medicaid in high-nonwhite-AFDC states are robust to controls for AFDC rates themselves. Source: see notes to table III.

Figure I. The Share of Uninsured and Publicly Insured Children, 1950-2012



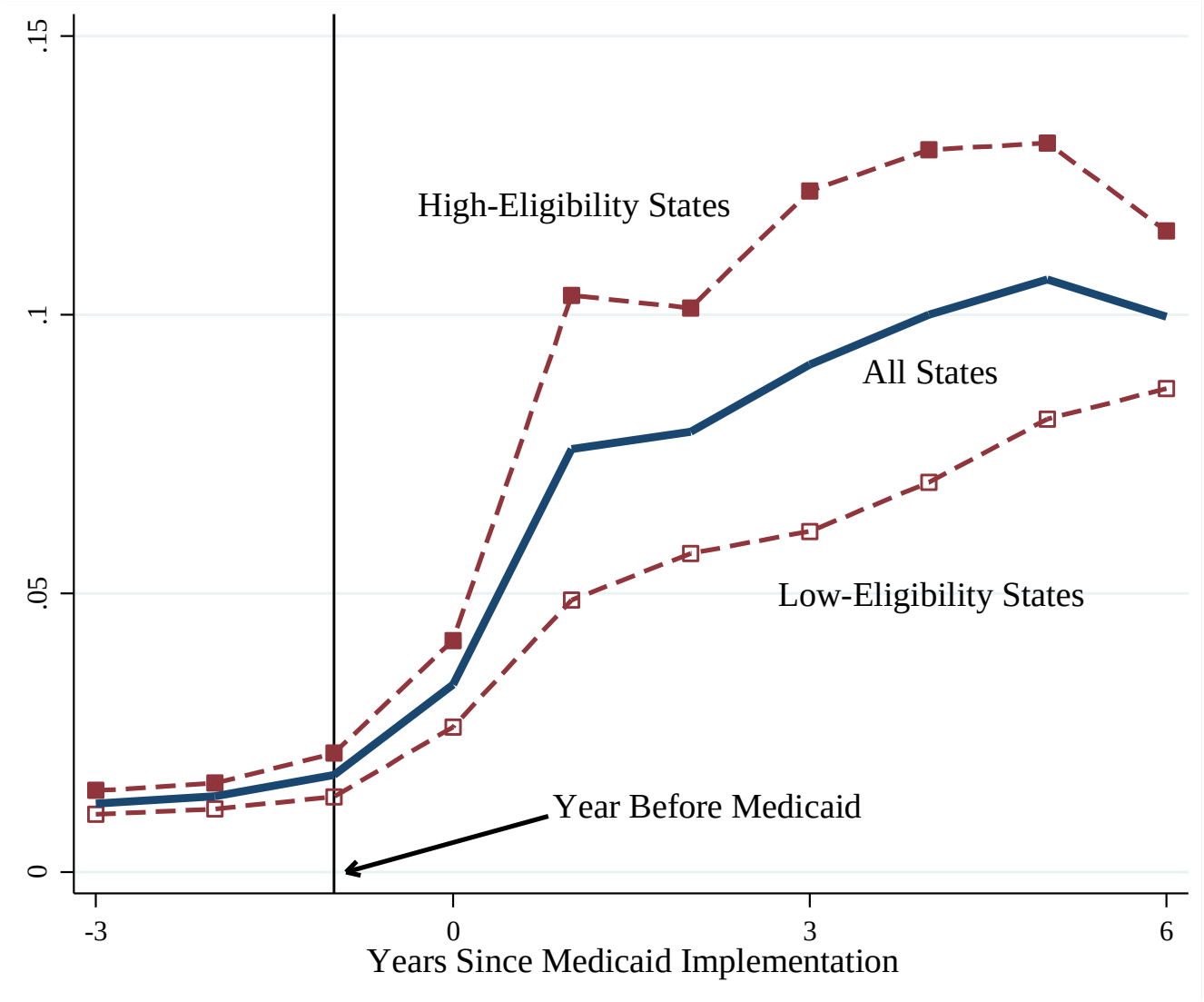
Notes: The figure plots the share children ages 0 to 19 that received some form of means-tested public insurance or were uninsured from 1950 to 2012. The 1963, 1968 and 1974 data come from ICPSR National Health Interview Survey files and the 1976-2012 data come from the Integrated Health Interview Survey. NHIS/IHIS estimates of uninsurance are shown in closed blue circles, and of public insurance (including Medicaid and the State Children's Health Insurance Program) are shown in the solid line. Children are classified as having no insurance if they report no hospital insurance, surgical insurance or doctor insurance and (in 1968 and 1974) if they do not list coverage through "Medicare, Medicaid or welfare" as a reason for not having insurance (children with missing or unknown insurance status are excluded). The share of uninsured children in the SHSUE is calculated using direct questions on the number of health insurance policies. In 1970, children who report expenditures paid by "public aid (receiving welfare payments), Medicaid (receiving no welfare payments), and/or free or part pay clinic or public hospital services" are counted as insured. The open squares and triangles are based on administrative data, and show the ratio of unduplicated annual counts of Medicaid child *recipients* (rather than enrollees) to the population age 0 to 19. For a description of the Department of Health, Education and Welfare (DHEW) data see appendix 1. The Center for Medicare and Medicaid Services (CMS) data are from the 2012 Medicare and Medicaid Statistical Supplement Table 13.4. Population denominators are from the SEER and the 2000-2010 intercensal population estimates. This share is set to zero in 1950, when federal participation in medical costs of welfare recipients was first authorized. Sources: DHEW (various years) ; Center for Medicare and Medicaid Services (2012); Center for Health Administration Studies and National Opinion Research Center (1984a; 1984b); United States Department of Health and Human Services et al. (2010a; 2010b; 2010c); Minnesota Population Center and State Health Access Data Assistance Center (2012).

Figure II. Medicaid Categorical Eligibility: The Rate of AFDC Receipt by Age and Race, December 1967



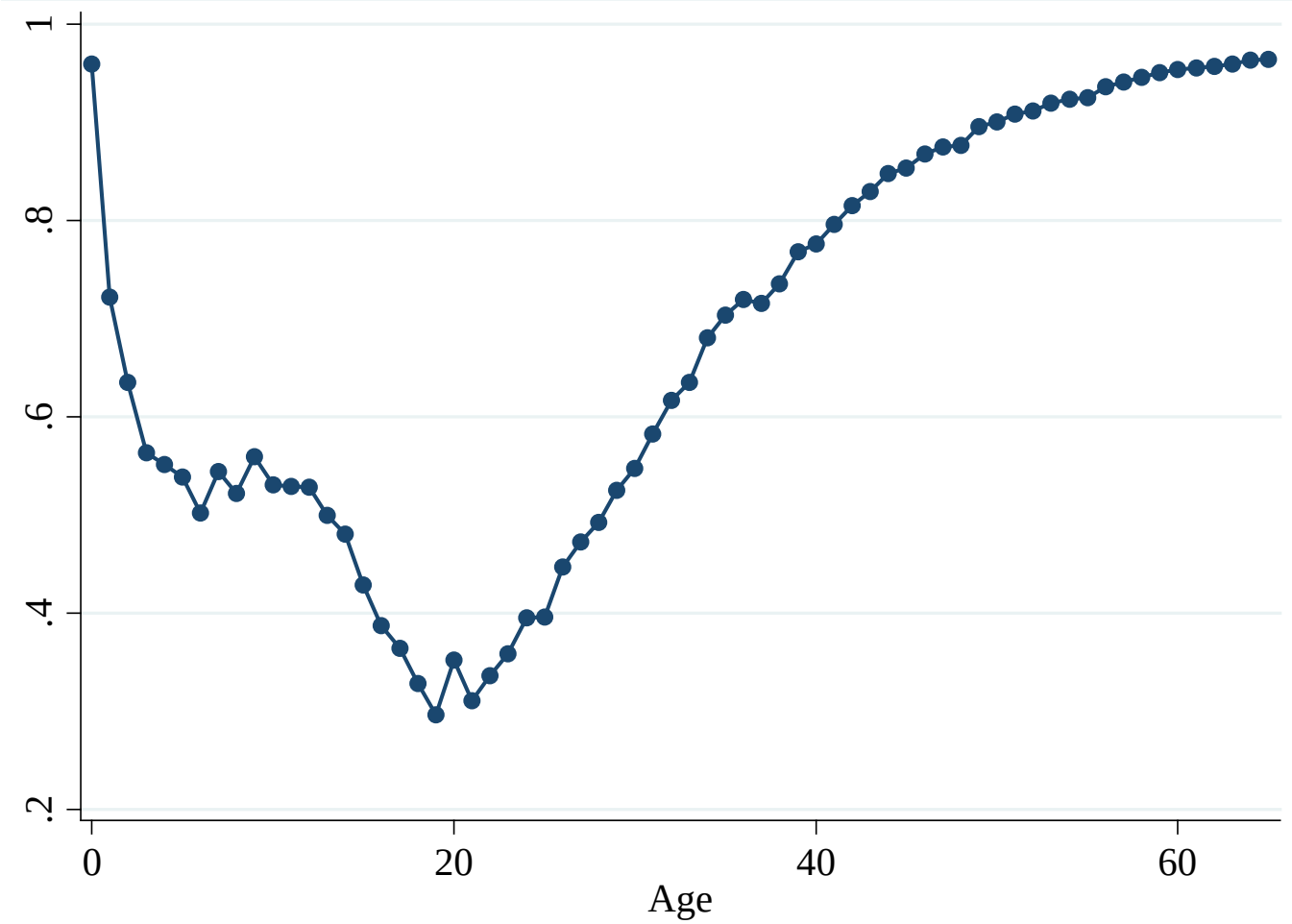
Notes: The figure plots the estimated shares of white and nonwhite people of each age who received a payment from the Aid to Families with Dependent Children (AFDC) program in December 1967. The series are constructed by calculating the joint age and race distribution of AFDC recipients using the 1967 AFDC Study, multiplying it by the total number of AFDC cases in December 1967, and dividing by the inter-censal population estimates (U.S. Census Bureau 2001). AFDC receipt was the most common way that families qualified for Medicaid because of the requirement that welfare recipients be covered ("categorical eligibility"). The figure shows that categorical eligibility for Medicaid was about four times higher for children than for adults, and six times higher for nonwhite children than white children.

Figure III. The Share of Children Using Public Health Insurance before and after Medicaid



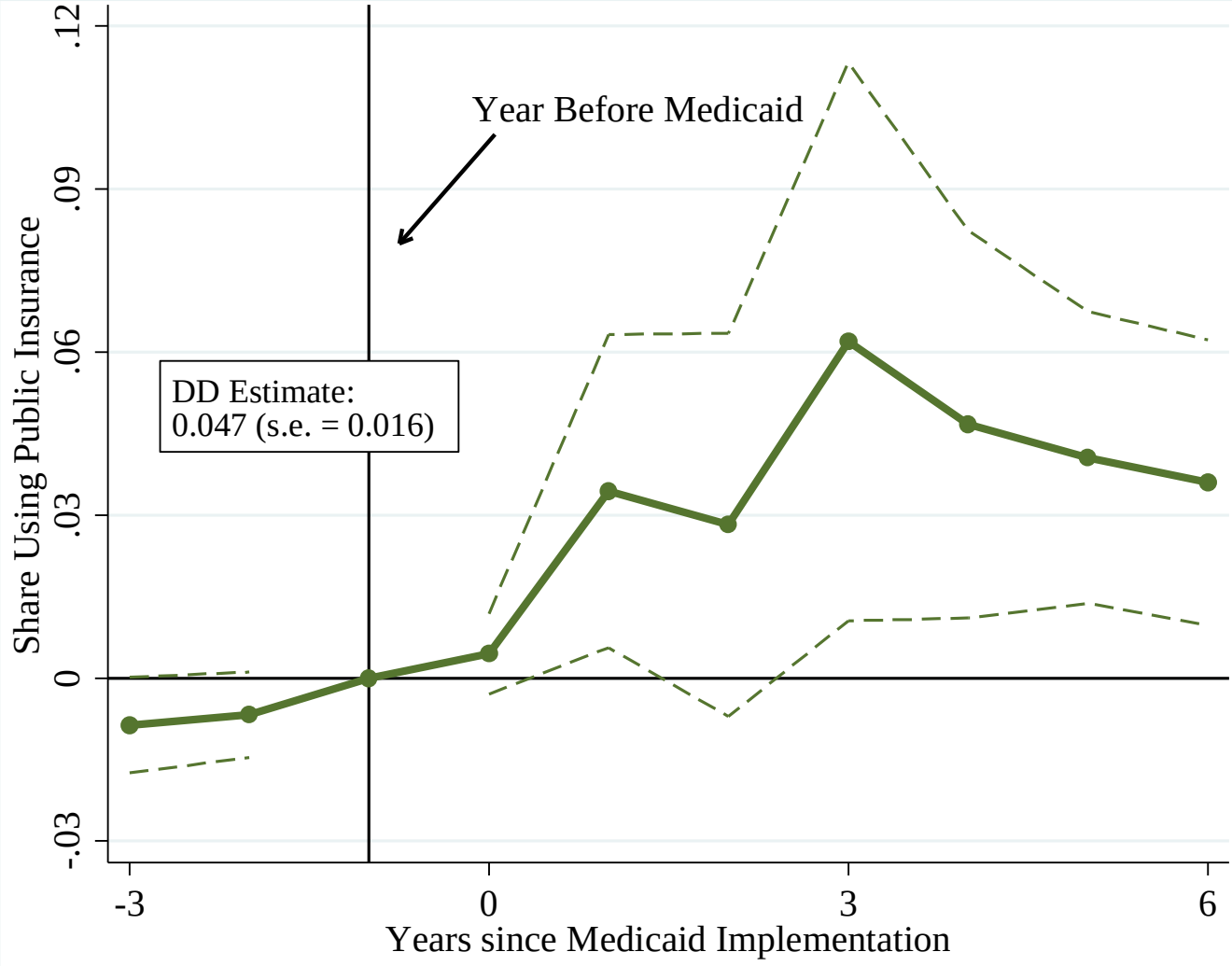
Notes: The figure plots the share of children ages 0-19 who received medical services paid for by a means-tested public insurance program in the years before and after states implemented Medicaid. Source: AFDC cases are from Health and Human Services Caseload Data 1960-1999 (HHS 2012); population data are from 1960 population estimates (Haines and ICPSR 2005), and the Survey of Epidemiological End Results (SEER 2009); data on public insurance use are collected from various editions of "Recipients of Medical Vendor Payments Under Public Assistance Programs" and "Medicaid State Tables" (DHEW 1963-1976). See appendix 1 for a description of the public insurance data.

Figure IV. Share of Deaths Due to Internal Causes by Age, 1965



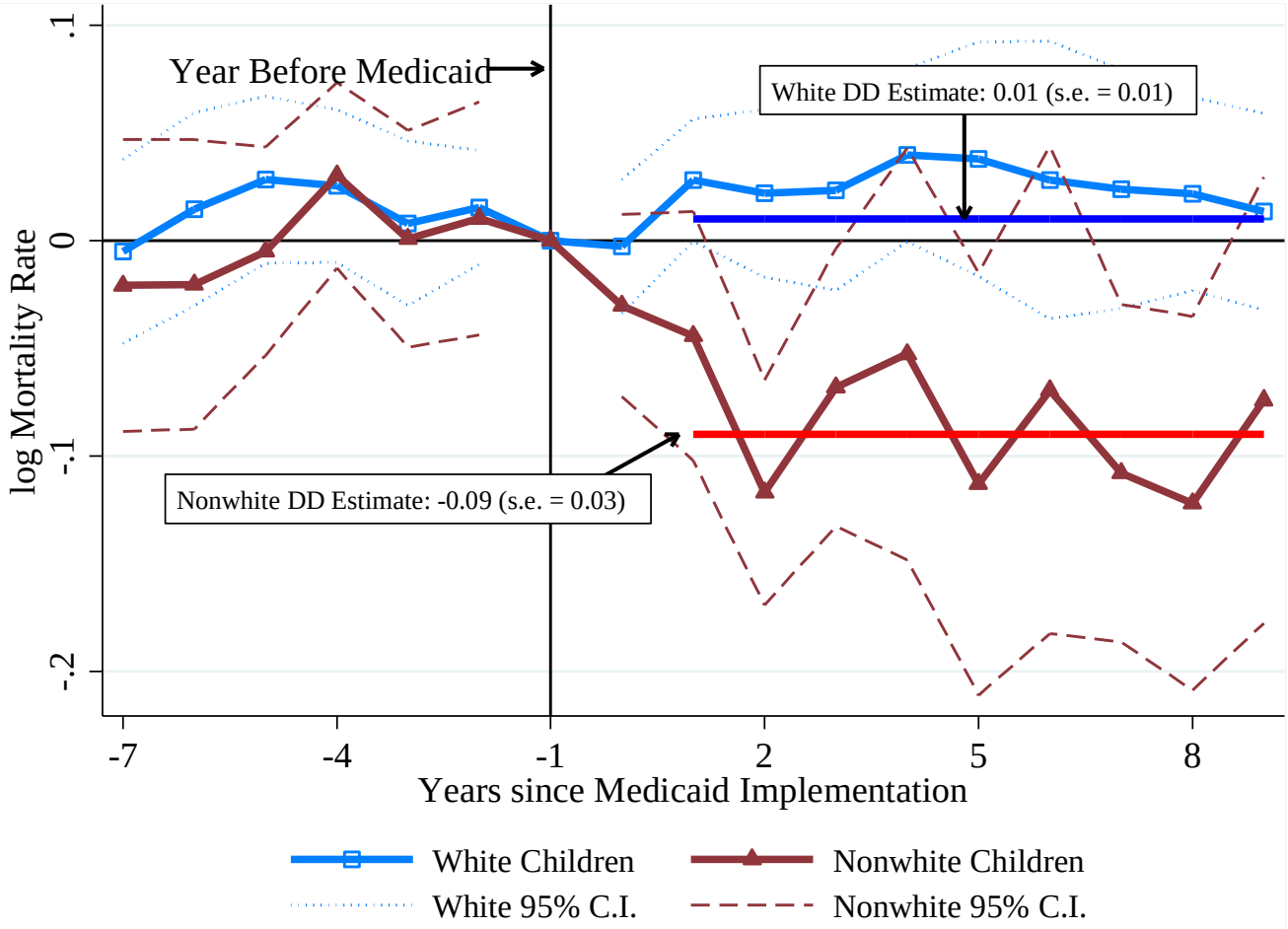
Notes: The figure plots the share of deaths at each single age due to internal causes of death. Internal causes include all deaths not due to "external" causes in the International Classification of Diseases Revision 7 (ICD7 codes E800-E999).
Source: Vital Statistics Multiple-Cause of Death File, 1965 (US DHHS 2007).

Figure V. Regression-Adjusted Estimates of Medicaid's Effect on Children's Public Insurance Use



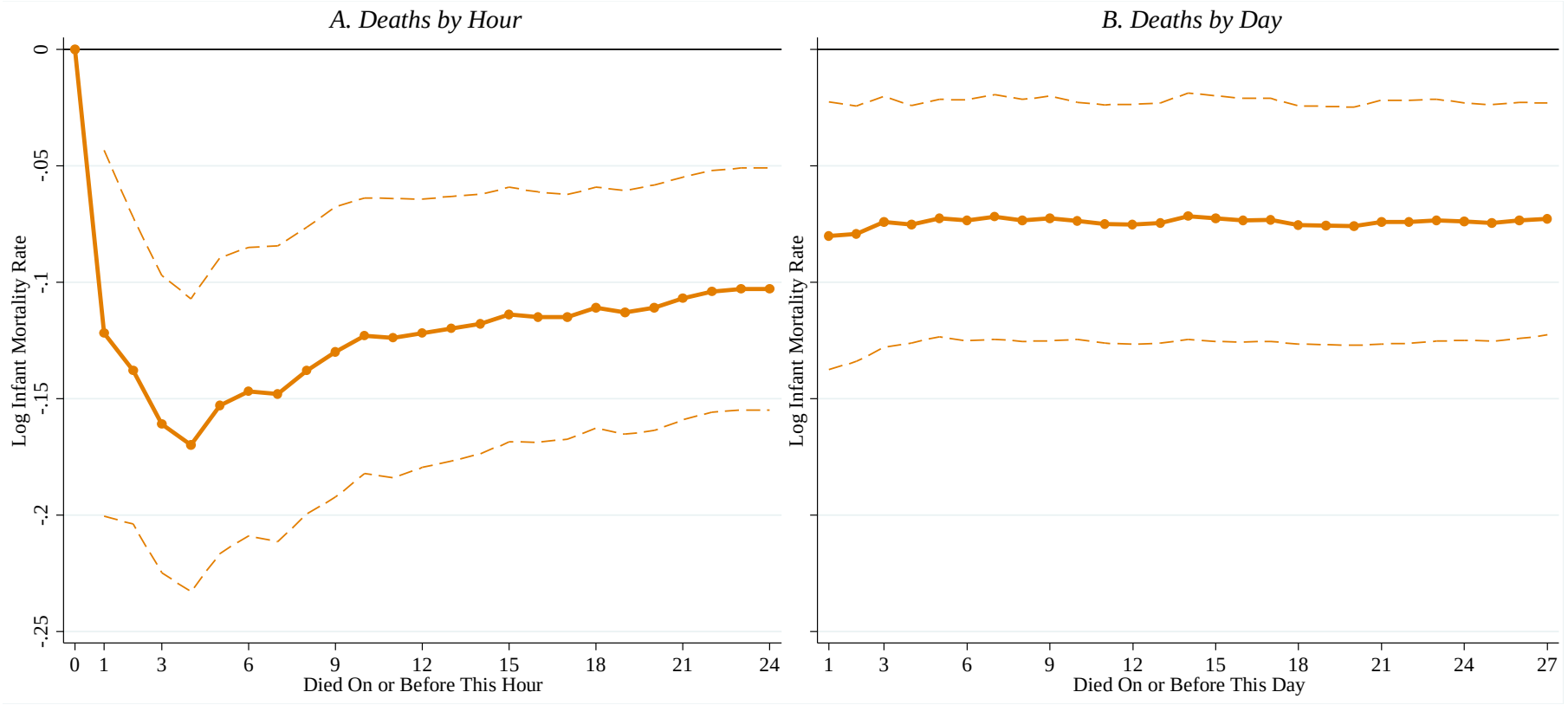
Notes: The dependent variable is the estimated share of children ages 0-21 that received services covered by a means-tested public insurance program. The figure plots the estimated coefficient on interactions between time-to-Medicaid dummies ($1\{t - t_s^* = y\}$) and a dummy variable for high-AFDC states (D_s) in a regression model described in Section III. The year before Medicaid implementation is omitted so the estimates are normalized to zero in that year. The model also includes state fixed effects, per-capita income and hospital capacity variables, region-by-year fixed effects, and separate year fixed effects for each Medicaid timing group. The dashed lines are pointwise 95 percent confidence intervals based on standard errors clustered at the state level. The sample includes 605 state-year observations that have non-missing values for public insurance use between 1963 and 1976, except West Virginia (which, prior to Medicaid, reports numbers of recipients for whom premiums into a pooled medical fund were paid as opposed to actual utilization). The estimates are weighted by state populations aged 0-19, but a Hausman test cannot reject the null hypothesis that the weighted and unweighted estimates are equal (p-value = 0.54; Deaton 1997; Solon, Haider, and Wooldridge 2013). Source: See appendix 1.

Figure VI. Regression-Adjusted Estimates of Medicaid's Intention-to-Treat Effect on Child Mortality by Race



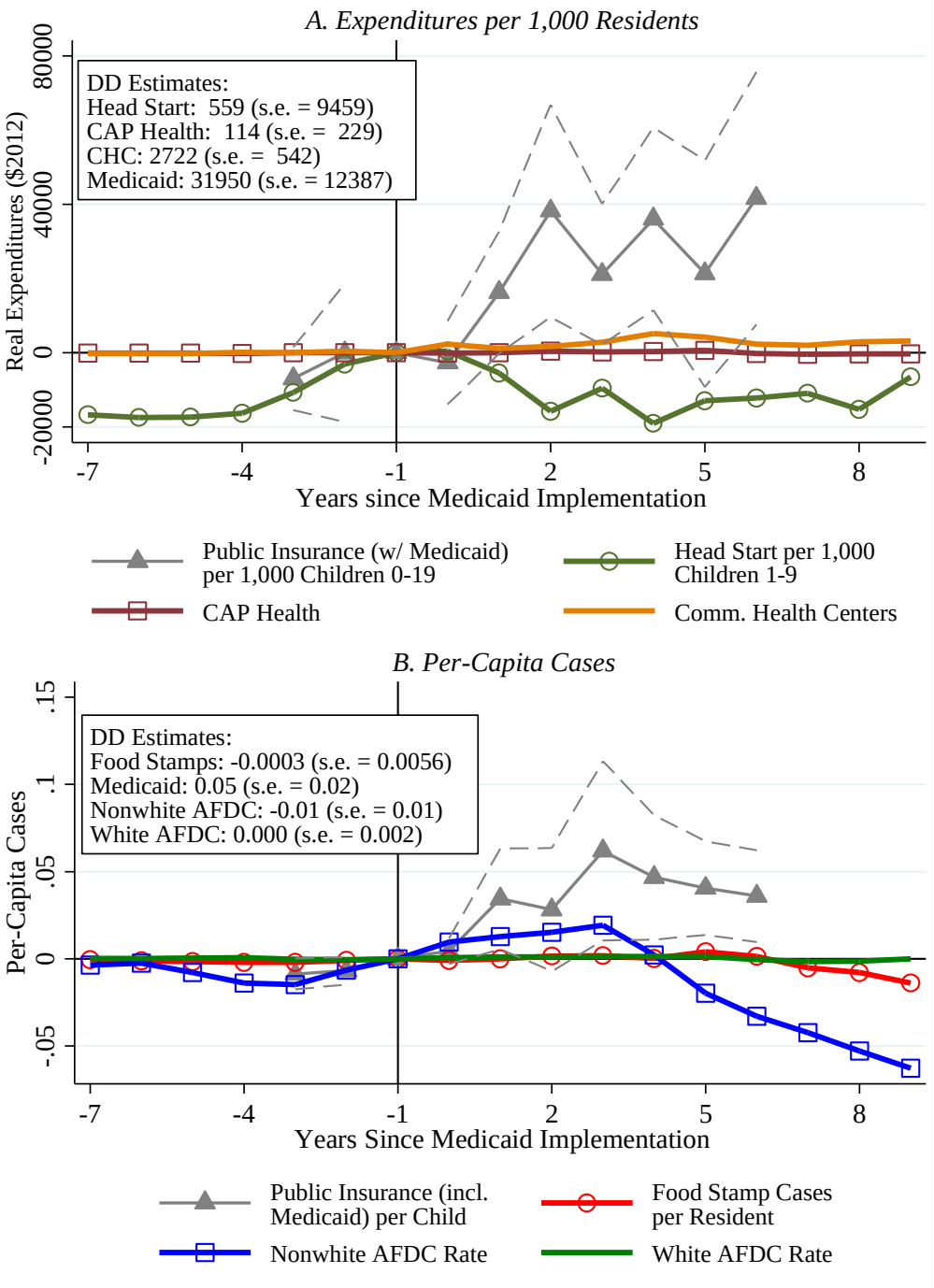
Notes: The dependent variable is the natural log of the age-adjusted mortality rate among children aged 0-14. The figure plots the estimated coefficients on interactions between time-to-Medicaid dummies ($1\{t - t_s^* = y\}$) and a dummy variable for high-AFDC states (D_s) in the regression model described in Section III. The year before Medicaid implementation is omitted so the estimates are normalized to zero in that year. States observed more than seven years before Medicaid (the latest-implementing states) or more than 10 years after (the earliest implementing states) are grouped into endpoint dummies and their coefficients are not shown (see McCrary 2007). High- and low-AFDC states are defined by the median race-specific AFDC rate as in table I. In addition to the variables of interest—interactions between time-to-Medicaid dummies and a high-AFDC indicator ($D_s 1\{t - t_s^* = y\}$)—the model includes state fixed effects, separate year fixed effects for each Medicaid timing group, per-capita income and hospital variables, and region-by-year fixed effects. The broken lines are pointwise 95 percent confidence intervals based on standard errors clustered at the state level. The white sample includes 987 state-year observations between 1959 and 1979 for 47 states (Arizona, Alaska, Hawaii, and New Jersey are omitted). The nonwhite sample (924 observations) also excludes New Hampshire, Vermont and Maine because less than one percent of their children are nonwhite. The post-Medicaid coefficients for nonwhite children are jointly significant below the 1% level (p-value = 0.0001), and the pre-Medicaid coefficients are not jointly distinguishable from zero (p-value = 0.38). Neither the pre- nor the post-Medicaid coefficients for white children are distinguishable from zero (p-values = 0.46 and 0.59, respectively). A joint test of the difference-in-difference assumption (H_0: pre-Medicaid coefficients equal zero and the post-Medicaid coefficients are equal to each other), rejects the null hypothesis for the nonwhite estimates (p-value = 0.001), but not for the white estimates (p-value = 0.59). A Hausman test rejects the equality of weighted and unweighted nonwhite estimates, although the point estimates are quite similar, and the slightly more restrictive specifications presented in table III cannot detect differences between weighted and unweighted estimates. See appendix 2 for unweighted event-study results. Source: Mortality data are from Vital Statistics Multiple-Cause of Death Files 1959-1979 (US DHHS and NCHS 2009). Population denominators are from the 1950 and 1960 Censuses (Haines and ICPSR 2005) and the 1969 to 1988 Surveillance Epidemiology and End Results (SEER 2009).

Figure VII. Regression-Adjusted Estimates of Medicaid's Intention-to-Treat Effect on Nonwhite Infant Mortality by Hour and Day of Death



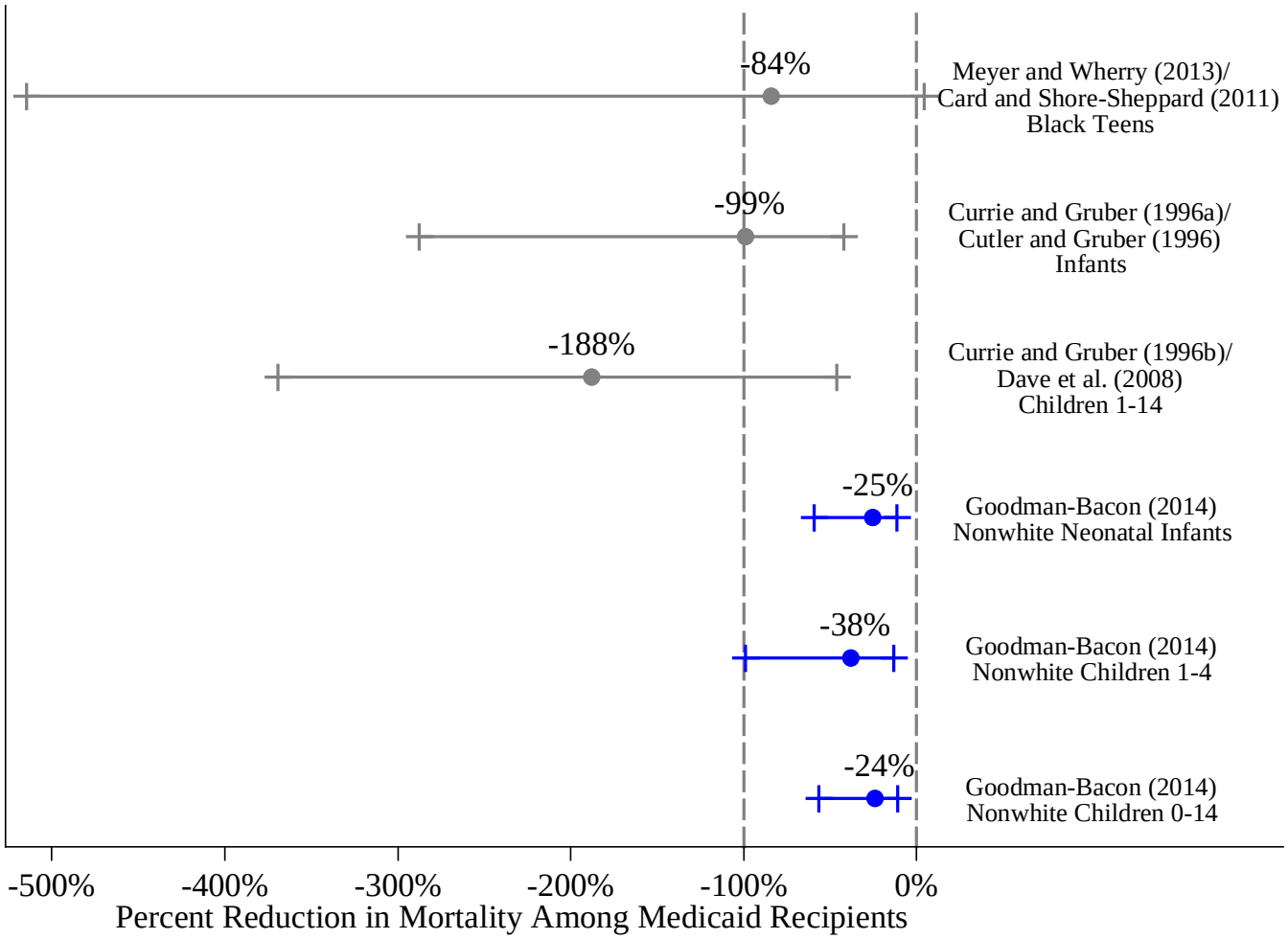
Notes: The figure plots DD estimates of Medicaid's effect on nonwhite infant mortality rates based on the same specification as in figure VI. The dependent variable is the log of the infant mortality rate before each hour (panel A) or day (panel B) during the first 27 days of life. The right-most estimate is therefore equivalent to the effect on neonatal infant mortality rates from column 1 of table IV. Because mortality rates are calculated conditional on being born alive, the effect at "hour zero" is normalized to zero. The estimates at 24 hours and 1 day exhibit a shift because of differences in the age units in which infant deaths are reported. This is unlikely to affect the pattern of estimates within the first day and has no effect on the estimates within the first 28 days. Source: see notes to figure VI and table IV.

Figure VIII. The Relationship between Medicaid Implementation and Health-Related Programs



Notes: The figure plots estimated coefficients on interactions between Medicaid timing dummies and a dummy variable for high-AFDC states in a regression model described in Section III. The dependent variable in panel A is funding per capita (in 2012 dollars) and the dependent variable in panel B is the number of cases per resident or the number of children who used public insurance per child 0-19. The sample for other program funds contains 1,008 observations on 48 states from 1959 to 1979. The results show that other federal health programs or programs that have been shown to affect health outcomes (Head Start: Ludwig and Miller 2007; Community Health Centers: Bailey and Goodman-Bacon 2013; Food Stamps: Almond, Hoynes, and Schanzenbach 2012) did not grow coincidentally with Medicaid in high-AFDC states relative to low-AFDC states. Sources: National Archives Community Action Program and Federal Outlays Files, Public Health Service Reports, DHEW (1963-1976) and (HHS 2012). I thank Hilary Hoynes for sharing the Food Stamp caseload data.

Figure IX. The Proportional Effects of Medicaid on the Mortality Rates of Newly Insured Recipients: Average Treatment Effects on the Treated (ATET)



Notes: The figure plots the implied average treatment effects on the treated (ATET) and 95-percent confidence intervals for three comparable previous studies and for the DD results emphasized in this paper: all nonwhite children ages 0-14, nonwhite neonatal infants, and younger nonwhite children ages 1-4. To calculate the ATET, the reduced-form ITTs are expressed as proportional changes in mortality rates, divided by a first-stage estimate for *any* insurance coverage (adjusted for under-reporting when appropriate), then adjusted for differential baseline mortality among poorer Medicaid recipients. The rightmost column lists the source of the mortality/first-stage estimates (see appendix tables 4.1-4.5 for the parameters). The vertical dashed line at zero indicates that Medicaid does not reduce mortality, and at -100% indicates the largest possible value of the ATET (because a group's mortality rate cannot be reduced by more than its baseline level). For my ATET estimates I used the continuous specification of the first-stage and reduced form equations shown in appendix figure 2.6 and appendix table 2.3. They imply a similar ATET as the binary specification but are more precisely estimated as noted in the text. The 95-percent confidence intervals are generated using a modified percentile method from 10,000 replications of a parametric bootstrap procedure (Johnston and DiNardo 1997, Efron and Tibshirani 1993, Valetta 1993). I generate bootstrap draws of the reduced-form and first-stage coefficients from normal distributions with means and standard deviations equal to the point estimates and standard errors reported in each paper. The confidence intervals are generated by taking the 5th percentile of the empirical distribution of the ATET for draws below the point estimate and the 95th percentile of the empirical distribution above the point estimate. See appendix 4 for details on the bootstrap procedure and alternative estimators for the confidence intervals.