Public Insurance and Mortality: Evidence from Medicaid Implementation

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This paper provides new evidence that Medicaid's introduction reduced infant and child mortality in the 1960s and 1970s. Mandated coverage of all cash welfare recipients induced substantial cross-state variation in the share of children immediately eligible for the program. Before Medicaid, higher- and lower-eligibility states had similar infant and child mortality trends. After Medicaid, public insurance utilization increased and mortality fell more rapidly among children and infants in high-Medicaid-eligibility states. Mortality among nonwhite children on Medicaid fell by 20 percent, leading to a reduction in aggregate nonwhite child mortality rates of 11 percent.

I. Introduction

The establishment of means-tested public health insurance—Medicaid—in 1965 was among the largest efforts in US history to improve the health of the poor. The program's architects predicted "the beginning of a new

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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" (DHEW 1967a, sec. D-5140). Fifty years later, Medicaid covers 40 percent of children and over 80 percent of poor children (Cohen and Martinez 2013).

While Medicaid's costs are large and controversial, its benefits in terms of health have been hard to quantify. Most research focuses on eligibility expansions in the 1980s and finds improvements in health but relatively small increases in insurance rates (Currie and Gruber 1996a, 1996b; Shore-Sheppard 2009; Bronchetti 2014; Wherry and Meyer 2016), suggesting that Medicaid's effects work through channels other than simply increasing coverage. In fact, the expansions affected savings and consumption (Gruber and Yelowitz 1999; Leininger, Levy, and Schanzenbach 2010), eligibility for other programs (Bitler and Currie 2004), and provider investments (Freedman, Lin, and Simon 2015), all of which may explain the large effects. Even recent experimental evidence from the Oregon Health Insurance Experiment finds improvements in self-reported health but no effects on clinical measures (Baicker et al. 2013) or 1-year mortality (Finkelstein et al. 2012). Thus, 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 ("categorical eligibility") to provide new estimates of its 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 because of long-standing, institutional differences in welfare programs. Second, children, especially nonwhite children, had the highest categorical eligibility rates, so the analysis has the most power to identify effects of Medicaid implementation for them.

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). This "dose-response" empirical strategy obviates the need for comparisons between states that implemented Medicaid earlier and later, which differed in their pre-Medicaid mortality trends. In contrast, welfare-based eligibility is uncorrelated with levels and trends in a

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range of state characteristics in the decades before Medicaid's introduction and with the contemporaneous growth in War on Poverty programs. The results from an event study specification (Jacobson, LaLonde, and Sullivan 1993) show directly that mortality rates in higher- and lower-welfare states did not trend differently for 16 years prior to Medicaid.

The results are the first to show that Medicaid's introduction achieved one of its primary goals: to "prevent . . . premature death" (DHEW 1967a, sec. D-1000). After Medicaid's introduction, higher-eligibility states experienced dramatic decreases in infant and child mortality rates relative to lower-eligibility states. The effects persist for 9 years and are strongest and most precise for nonwhite children. Infant mortality reductions come from improved acute care at birth: they are concentrated in the first few hours of life, there are no measurable improvements in fetal health (birth weight or sex ratios), and auxiliary evidence points to increases and improvements in hospital care. Child mortality reductions come largely from improved care for infectious disease: they are largest for the youngest, most susceptible children and for causes with effective treatments in the 1960s and 1970s. Newly entered data on public health insurance programs from 1963 to 1976 verify that welfare-based Medicaid eligibility led to meaningful 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 20–30 percent—smaller than comparable effects from the 1980s expansions (Currie and Gruber 1996a, 1996b) but large enough to matter demographically. Medicaid implementation reduced aggregate nonwhite child mortality rates by 11 percent and the difference in mortality rates between poor and nonpoor nonwhite children by one-third. Overall, the results show that the expansion of public insurance for poor children had important health benefits and suggest 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.

II. Public Insurance and Mortality Before and After Medicaid

In 1950, the federal government began to share with the states some of the costs of medical care that public assistance recipients obtained from private providers. The federal contribution was capped, however, which made states reluctant to establish generous programs with costs that might exceed the federal matching maximum. Consequently, few children received publicly financed health care, despite lacking other sources of payment. Figure 1 shows that, in the early 1960s, over 30 percent of all children were uninsured. Surveys show that over 90 percent of low-income

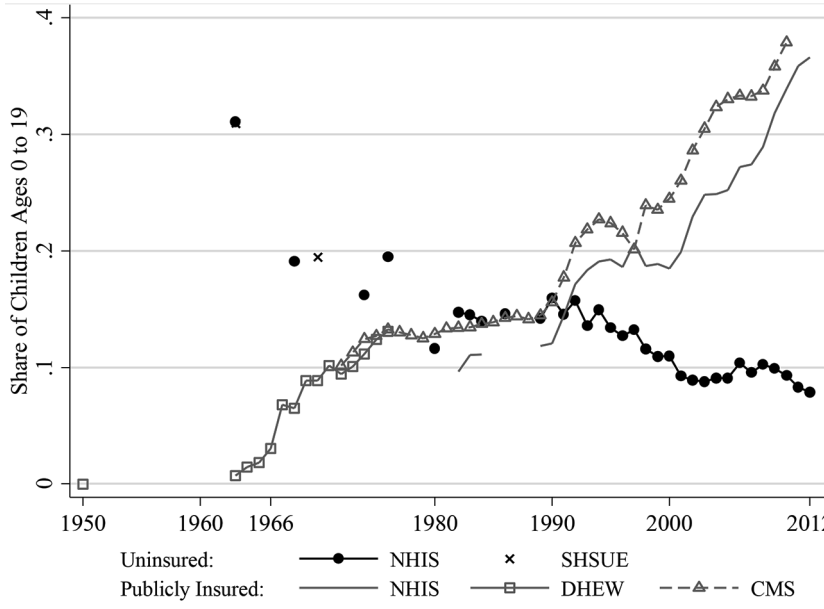

FIG. 1.—The share of uninsured and publicly insured children, 1950–2012. The figure plots the share of children aged 0–19 who received some form of means-tested public insurance or were uninsured between 1950 and 2012. The 1963, 1968, and 1974 data come from ICPSR National Health Interview Survey files and the 1976–2012 data come from the Integrated Health Interview Survey. NHIS/IHIS estimates of uninsurance are shown in closed circles, and estimates 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 aged 0–19. 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, 1963–76; 1965; 1967b; various years, 1971–76; 1974; 1975; 1976); CHS and NORC Survey of Health Services (1984); CDC and NCHS Health Interview Survey (2010); CMS (2012); Minnesota Population Center and State Health Access Data Assistance Center Integrated Health Interview Series (2012).

families reported having no doctor visit insurance (Kovar 1960). Furthermore, just 8 percent of adults reported receiving charity care in 1960 (Morgan et al. 1962).

The lack of payment sources for poor families corresponds to low utilization and poor health. Data from the 1963 Survey of Health Services

Utilization and Expenditure (SHSUE; conducted by the Center for Health Administration Studies and National Opinion Research Center) show that 45 percent of children in the bottom third of the income distribution had seen a physician in the previous year (32 percent among nonwhite children), compared to 77 percent of children in the top third.[1] Poor children suffered more from serious symptoms such as 4–5 days of diarrhea, heart pain, or unexpected bleeding, and conditional on having less serious symptoms such as a skin rash, a persistent cough or sore throat, or abdominal pain, they sought care much less often than high-income children.[2]

Medicaid (Public Law 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.[3] States were required to implement Medicaid by 1970 or else lose federal reimbursements for existing medical programs. Twenty-six states adopted Medicaid in 1966, 11 in 1967, and the rest between 1968 and 1970, except Alaska (1972) and Arizona (1982). Medicaid eliminated caps on federal financing, increased the federal reimbursement rate, 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—and mandated coverage for recipients of federally funded cash welfare programs ("categorical eligibility").[4] The solid line in figure 2 shows that the share of all children who received public medical services increased sharply after Medicaid began. The public insurance rate for children increased by 10 percentage points in the 5 years after Medicaid implementation but by only 2 percentage points for adults (not shown).

The categorical eligibility requirement led to large differences in eligibility between demographic groups and states. For children, Medicaid

[1] In 2011, the shares were 77 and 83 percent, according to an interview series by the Minnesota Population Center and State Health Access Data Assistance Center.

[2] The share seeking care conditional on symptoms was 28.5 percent for children in the bottom third of the income distribution in 1963 and 42 percent for children in the top third (standard error [s.e.] of the difference = 4.7).

[3] Medicaid 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's) opposition to Medicare. The SSA amendments combined Medicare Part A (compulsory hospital insurance, the Democratic proposal), Medicare Part B (voluntary supplementary physician insurance, the Republican proposal), and Medicaid (a federal/state public insurance program for the poor, the AMA's proposal). An administration official, Wilbur Cohen, remarked, "It was the most brilliant legislative move I'd seen in thirty years. . . . [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, 40).

[4] States could include additional services, including home health care, clinic services, prescription drugs, eye care, and dental care. States could also cover the "medically needy"—families that were ineligible for cash public assistance but had large medical bills. The medically needy account for a small share of children on Medicaid, so I ignore this provision in the rest of the paper. For details see Gruber (2003).

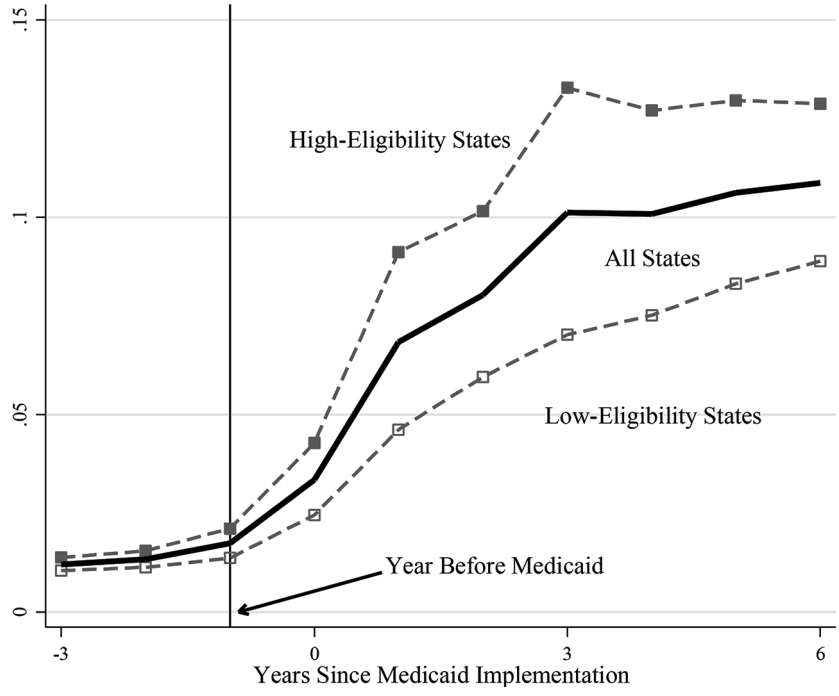

FIG. 2.—The share of children using public health insurance before and after Medicaid. The figure plots the share of children aged 0–19 who received medical services paid for by a means-tested public insurance program in the years before and after states implemented Medicaid. High- and low-eligibility states are defined by the median value of AFDC rates in the year states implemented Medicaid ($AFDC_s^*$). Sources: DHEW (1965; various years, 1963–76; 1967b; various years, 1971–76; 1974; 1975; 1976); Haines and ICPSR 2010 data; SEER (2013); DHHS Caseload Data 1960–99 (2012).

eligibility was nearly synonymous with participation in the Aid to Families with Dependent Children (AFDC) program: in 1976, 89 percent of children on Medicaid qualified through AFDC (DHEW 1966, 1976). Figure 3 shows that, in 1967, nonwhite children received AFDC at a much higher rate than other groups, and survey data show similar patterns in Medicaid utilization. AFDC-based categorical eligibility ranged across states from 0.11 to 4.4 percent for white families and from 0.4 to 25 percent for nonwhite families.

Poor nonwhite children not only received Medicaid at the highest rate but also had the most to gain in terms of health. Figure 4 shows that the share of deaths due to internal causes—a common measure of the sensitivity of mortality to medical interventions—accounts for nearly all in-

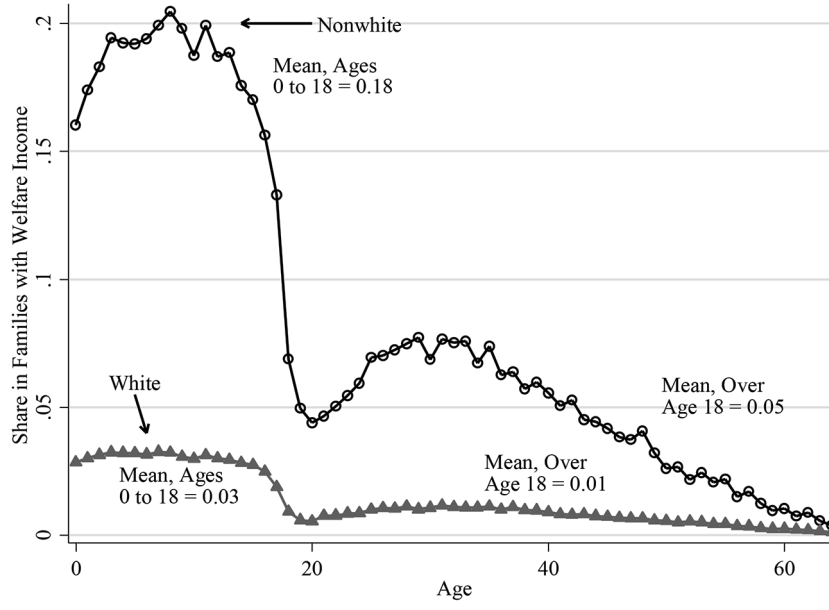

FIG. 3.—Medicaid categorical eligibility: the rate of AFDC receipt by age and race, December 1967. The figure plots the estimated shares of white and nonwhite people of each age who received a payment from the AFDC program in December 1967. The series are constructed by calculating the joint age and 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 intercensal population estimates (US Census Bureau). 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 for white children. Sources: DHEW (2011); US Census Bureau (2004).

fant and most child deaths.[5] Mortality rates for nonwhites, however, were twice as high as those for whites of the same age (1965 Vital and Health Statistics, series 22, table 1-9), and these deaths were more likely to be due to causes with "effective" treatments (Beeson 1980, 80).[6] 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). Nonwhite children died from anemias twice as often as white children, even though a folate supplement "suppresses or controls the disease" (Beeson 1980, 80). They also received recom-

[5] 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."

[6] For example, 35.4 percent of nonwhite child deaths (ages 1–4) in 1965 were due to infectious diseases, compared to 26.4 percent of white child deaths.

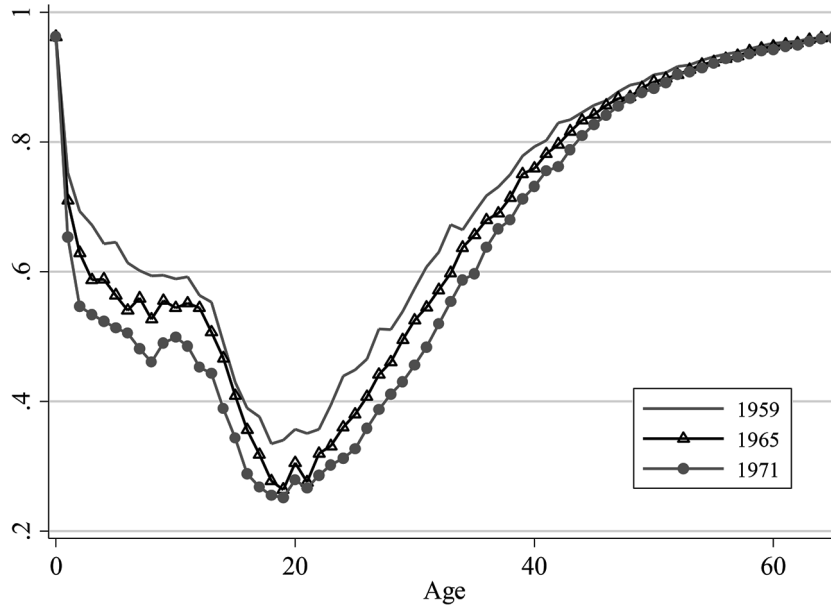

FIG. 4.—Share of deaths due to internal causes by age: 1959, 1965, and 1971. The figure plots the share of deaths at each single age due to internal causes of death. Internal causes include all deaths not due to "external" causes in the International Classification of Diseases Revision 7 (ICD7 codes E800–E999). Source: Vital Statistics Multiple-Cause of Death Files.

mended vaccinations less often (NCHS 1976, tables CD.I.47, CD.I.48) and suffered higher mortality from causes that could be vaccinated against (1.6 of deaths vs. 0.9 percent of deaths; s.e. of the difference = 0.2). Poor infants could have benefited from simple improvements in labor and delivery care. Existing monitoring technology could detect distress "in time for the fetus to be rescued by rapid delivery" (Desmond 1998, 196), and neonatal interventions improved throughout the 1960s and 1970s (Budetti and McManus 1982; Cutler and Meara 1999). Moderately low–birth weight babies responded well to postnatal treatments such as oxygenated, temperature-controlled incubators and negative pressure ventilators ("iron lungs").

Finally, Medicaid coverage almost certainly represented new insurance coverage at this time and, therefore, a meaningful increase in care. Figure 1 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). This suggests that Medicaid's introduction had considerable scope to improve the health of poor children.

III. Data

To estimate the effects of Medicaid's introduction, I combine several data sources that contain information on public insurance eligibility and utilization, health outcomes, and potential confounders.

A. *Categorical Eligibility*

To measure racial differences in AFDC-based categorical eligibility, I calculate the nonwhite share of AFDC payees and children using printed tables for 1958 and 1961 (Mugge 1960; DHEW 1963) and microdata on AFDC recipients collected from the National Archives (DHEW 2000, 2011) for 1967–79. I interpolate the race shares between missing years, multiply by AFDC participant counts (US DHHS 2012), and divide by population counts to get an estimate of the state-by-year-by-race AFDC rate. The AFDC data allow me to construct rates for adult women and for children aged 0–19, which are similar to rates in the 1970 census.[7]

B. *Public Insurance Use*

To measure the share of children who used public insurance, I entered data from Department of Health, Education, and Welfare (DHEW) reports from 1963 to 1976. The data measure utilization of benefits, referring to children (aged 0–19, as defined by AFDC eligibility rules) who actually obtained medical care. Utilization means more for health than the coverage information available in most surveys because it incorporates the effects of provider participation in Medicaid. Even so, coverage and utilization were very close for categorically eligible children: the 1976 Survey of Income and Education and the Office of Economic Opportunity 11-City Survey (Bailey and Goodman-Bacon 2015) show that 90 percent of families on welfare who report Medicaid coverage in a given year also report using Medicaid.[8]

C. *Health Outcomes*

This paper, like other work on Medicaid and health, focuses on mortality. Death is an extreme health outcome, but conceptually it is an unam-

[7] Data on population counts are taken from Historical, Demographic, Economic, and Social Data for 1790–2002, from Michael Haines and the Inter-University Consortium for Political and Social Research (ICPSR) and SEER (2013). The online appendices describe the data sources, provide additional support for the design, and present alternative specifications and additional results.

[8] The new public insurance data also show that other categories of adult Medicaid were correlated with AFDC rates (the coefficient from a regression of logs of non-AFDC on AFDC adults in 1975 is 1.05, s.e. = 0.04). Adults are not a falsification test because they had nontrivial Medicaid exposure driven by different policies correlated with child eligibility.

biguous indicator of poor health, and it is well measured and consistently available. I construct state-by-year infant and child mortality rates (ASMR) from 1950 to 1979 using Multiple-Cause of Death Files (Department of Health and Human Services [DHHS] and National Center for Health Statistics [NCHS]) and printed volumes from various years of *Vital Statistics of the United States* (DHEW).[9] For children, I also calculate mortality rates for internal and external causes of death and for treatable and untreatable internal causes following Beeson (1980).[10] I summarize all-cause mortality using the age-adjusted child mortality rate, which weights together age-specific rates using the 1960 national age distribution.

For infants, Vital Statistics data contain a range of other health and utilization measures, including mortality rates by hour and day of death, low and very low birth weight rates, the male/female sex ratio, maternal mortality, and the share of births that took place in a hospital.[11] I also use the 1964–69 and 1972 National Natality Surveys (or the National Natality Followback Survey [NNFBS]), which contain socioeconomic, infant health, and birth site information for a sample of birth certificates.[12]

IV. Research Design: Difference-in-Differences Using Initial Categorical Eligibility

The research design exploits the categorical eligibility requirement in a difference-in-differences model that compares state-level health outcomes before and after Medicaid began between states with higher and lower categorical eligibility, measured by the AFDC rate in the year

[9] Data for 1972 are a 50 percent sample. Infant mortality is measured as deaths per 1,000 live births, and child mortality is measured as deaths per 100,000 children. Denominators for the child rates were constructed by linearly interpolating population between the 1950 and 1960 censuses (Haines and ICPSR data; see n. 7) and the 1969–79 SEER data. Denominators for the infant rates were calculated from Vital Statistics Natality Microdata from 1968–79 and entered state totals from Vital Statistics reports from 1950–67. I end the sample in 1979 because the 1980s eligibility expansions largely eliminated the state differences that drive my results and, as expected, attenuate the results.

[10] Beeson categorizes a range of conditions according to a 10-point scale of treatability in 1975. I assume all internal-cause deaths are treatable unless their 1975 treatment falls in the bottom six categories of treatability.

[11] The data on hospital births were collected by Amy Finkelstein and Heidi Williams with support from National Institute on Aging grant P30-AG012810 and are publicly available through the National Bureau of Economic Research.

[12] The NNFBS sampling frame is "legitimate births," 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, 13 percent of nonwhite respondents in the 1967–69 NNFBS received welfare income (compared to 11 percent of all nonwhite women), and more than a third of black Medicaid births in California in 1967–68 were legitimate (California Public Health Service 1973). Mothers could also have misreported marital status to welfare authorities or on their children's birth certificates.

of Medicaid implementation ($AFDC_s^*$).[13] The primary identifying assumption of this design—that in the absence of Medicaid, mortality would have evolved similarly in higher- and lower-AFDC states—is likely to hold for two related reasons.

First, AFDC rates were relatively long-run, stable, institutional features of states and did not emerge contemporaneously with Medicaid. I estimate univariate regressions of $AFDC_s^*$ on AFDC rates in 1948, 1958, and 1961. The slopes are positive, precisely estimated, and not statistically distinguishable across years, which ameliorates concerns that states made policy choices or recipients changed behavior in anticipation of Medicaid's implementation. Cliometric research shows that the factors that determine both welfare eligibility (policies, family structures, and income) and take-up (psychic costs and institutional barriers) differed across states at least as far back as the 1930s.[14]

Second, the long-run institutional variation is largely uncorrelated with state policies and characteristics in the 1960s. For example, since 1945, Texas's constitution has required that any welfare spending over 1 percent of the state budget (even if necessary to comply with federal cost-sharing arrangements) be passed by popular referendum, creating political barriers to expanding welfare programs. Meanwhile, Nebraska had long provided aid to unmarried mothers (Moehling 2007), and its nonwhite AFDC rate was close to the median even though its white AFDC rate was among the lowest in the United States. Rules on 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.[15]

Table 1 tests this claim directly. For a range of state characteristics (y_{st}), each row shows pre-Medicaid means and estimates of β_0 and β_1 from the following equation:

[13] The calendar year of implementation is typically the year just before a state's first full Medicaid year. Most began midyear and were delayed because of "shortages of welfare personnel to screen applications" (Tax Foundation 1968, 47).

[14] Moehling (2007) demonstrates that cross-state differences in family structure and the generosity of transfer programs for one-parent families existed even before the 1935 SSA 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 using vague eligibility provisions such as "suitable home" or "substitute parent" policies that were part of pre-AFDC mothers' pension programs (Bell 1965).

[15] State-level eligibility estimated in the 1960 census is positively and significantly related to AFDC receipt for white women (ordinary least squares coefficient is 0.8, s.e. = 0.1), but not for nonwhite women (0.3, s.e. = 0.3). 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 variation in categorical eligibility based on AFDC receipt. This approach would fail to assign low nonwhite Medicaid eligibility, e.g., in states with generous de jure regulations but restrictive de facto welfare systems.

$$y_{st} = \alpha + \beta_0 \text{AFDC}_s^* + \beta_1 \text{AFDC}_s^* \times (y - y^{\text{PRE}}) + u_{st}.$$

This embeds a test for balance in levels in the year y^{PRE}, the last available year before Medicaid's passage ($H_0 : \beta_0 = 0$), and in linear pre-Medicaid trends ($H_0 : \beta_1 = 0$).

The results provide little evidence that AFDC_s^* is correlated with levels or trends in state characteristics. Panel A shows no strong correlations between initial AFDC-based Medicaid eligibility and levels or trends in mortality or birth weight rates.[16] Panel B shows no evidence for the hypothesis that higher-AFDC states have more or increasingly disadvantaged populations, measured by levels or trends in child poverty, family structure, adult earnings, or education.[17] The final row of panel B and the first row of panel C show that higher-AFDC states did not have more generous welfare benefits or different levels or trends in government expenditures (Sylla et al. 2006). Panel C tests for balance in other health care variables that could affect child health: hospital capacity and private insurance rates.[18] Despite strong growth in these variables in the mid-twentieth century, the trends are uncorrelated with "initial" AFDC rates just prior to Medicaid. The null relationship in levels also suggests that AFDC_s^* will not capture heterogeneity in Medicaid's effect due to preexisting insurance rates (Finkelstein and McKnight 2008) or providers' ability to serve new public insurance recipients.

While AFDC_s^* fails to predict a wide range of state characteristics, figure 2 shows that it strongly predicts public insurance use. The dashed lines 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 small (0.005, s.e. = 0.002) but rose to 0.05 (s.e. = 0.006) after Medicaid was fully implemented.

Event study specification using Medicaid implementation and AFDC_s^*.— Equation (1) describes an event study specification (Jacobson et al. 1993) for demographic group k in which pre/post treatment is defined

[16] The marginally significant downward trend in low birth weight for nonwhites is small compared to the rate of low birth weight. A one standard deviation increase in nonwhite AFDC (5 percentage points) is associated with only a 4 percent larger reduction in low birth weight over 16 years ($-0.07 \times 5 \times 16/138.7 = 0.04$). Note that the levels of low and very low birth weight rates do not differ significantly by AFDC_s^*, suggesting that changes in the technology to treat premature infants during this time would not have disproportionately benefited higher- vs. lower-AFDC states.

[17] The one exception is that AFDC rates are positively related to the share of white children living without a father. These socioeconomic variables did not change differentially in higher-AFDC states between 1960 and 1970, when civil rights legislation had its biggest effects on nonwhite families.

[18] Amy Finkelstein shared the hospital data. I entered private insurance data from printed reports (Health Insurance Council 1953–59; Health Insurance Institute 1959–82).

TABLE 1
BALANCING TEST: THE RELATIONSHIP BETWEEN INITIAL AFDC RATES AND PRE-MEDICAID STATE CHARACTERISTICS IN LEVELS AND TRENDS

	NONWHITE			WHITE		
DEPENDENT VARIABLE	Pre-Medicaid Mean (1)	Level ($AFDC_s^*$) (2)	Trend ($AFDC_s^* \times$ Year) (3)	Pre-Medicaid Mean (4)	Level ($AFDC_s^*$) (5)	Trend ($AFDC_s^* \times$ Year) (6)
	A. Demographic Outcomes 1950–65 (Measured by Race)					
Child mortality	425.5	1.75	.13	206.8	2.22	.06
		[2.61]	[.15]		[2.83]	[.71]
Infant mortality	40.6	.08	.01	21.5	.05	−.03
		[.22]	[.01]		[.22]	[.07]
Very low birth weight	23.2	.12	.0004	10.1	−.09	−.01
		[.17]	[.0066]		[.15]	[.01]
Low birth weight	138.6	−.38	−.07	71.6	2.23	−.10
		[.44]	[.04]		[1.87]	[.08]
	B. Socioeconomic Outcomes 1950 and 1960 (Measured by Race)					
Poverty (0–14)	56.7	−.80	−.02	20.2	−.53	−.07
		[.75]	[.02]		[3.0]	[.10]
Living without father (0–14)	29.2	.11	−.009	7.4	1.00	.03
		[.13]	[.017]		[.43]	[.03]
Median earnings (25–44)	2,999	30.64	2.51	4,675	132.10	12.37
		[37.67]	[1.69]		[217.80]	[12.45]
Grade 12+ (25–44)	34.2	.25	.001	58.3	1.46	−.03
		[.45]	[.01]		[2.41]	[.07]
AFDC benefit (1967)	153.9	2.91		147.4	10.85	
		[1.33]			[10.41]	

	C. Other Outcomes (Not Measured by Race)					
Log public expenditure per 1,000 (1932, 42, 62)	−1.8	.021	−.001	−1.8	.12	−.005
		[.03]	[.001]		[.25]	[.006]
Hospital beds per 1,000 (1950–65)	4.9	.03	−.001	4.9	.09	<.0001
		[.03]	[.001]		[.20]	[.0119]
Hospital insurance per 1,000 (1952–65)	817.9	.68	−.32	817.9	−12.78	.74
		[4.70]	[.26]		[29.56]	[1.74]
Medical insurance per 1,000 (1952–65)	591.2	7.62	−.04	591.2	51.56	−.19
		[4.65]	[.38]		[29.36]	[2.43]

SOURCE.—DHHS/NCHS Multiple Cause of Death Files (1959–65); Vital Statistics of the United States (1950–65); Haines and ICPSR (2005); SEER (2013); 1950 and 1960 Census Integrated Public Use Microsample; DHEW (2011); American Hospital Association (1950–65); Health Insurance Institute (1952, 1954–65); Sylla, Legler, and Wallis (2006).

NOTE.—The table presents (weighted) estimates from the following model: $y_{st} = \alpha + \beta_0 \text{AFDC}_s^* + \beta_1 \text{AFDC}_s^* \times (y - y^{\text{PRE}}) + u_{st}$. The year 1965 is the latest pre-Medicaid year (y^{PRE}) except in panel B (1960) and the last row of panel C (1962); β_0 is the relationship between AFDC_s^* and levels of each variable in y^{PRE} (cols. 2 and 5); β_1 is the relationship between AFDC_s^* and linear trends in each variable (cols. 3 and 6).

by dummy variables that measure the time relative to Medicaid implementation, $1\{t - t_s^* = y\}$ (i.e., "event time"), and treatment/control groups are defined by the continuous value of initial AFDC rates, AFDC_s^*:[19]

$$\ln(\text{ASMR}_{st}^k) = \boldsymbol{x}_{st}'\boldsymbol{\beta}_k + \text{AFDC}_s^* \left[\sum_{y=-17}^{-2} \pi_y^k 1\{t - t_s^* = y\} + \sum_{y=0}^{10} \gamma_y^k 1\{t - t_s^* = y\} \right] + e_{st}^k. \quad (1)$$

My preferred specification of x_{st}' 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 year fixed effects for each Medicaid timing group. I modify the four census regions to match the definition of the South in Chay, Guryan, and Mazumder (2009).[20] This controls for the strong convergence in mortality between the South and the rest of the United States due to hospital desegregation (Almond, Chay, and Greenstone 2006), trends in school quality (Stephens and Yang 2013), and differences in private insurance coverage (Finkelstein and McKnight 2008).

Medicaid-timing-by-year fixed effects eliminate comparisons between states that adopted Medicaid earlier or later and force identification to come only from comparisons across AFDC_s^*. A difference-in-differences (DD) model based only on the differential timing of Medicaid adoption is identified (Decker and Gruber 1993; Strumpf 2011), but differential mortality trends in earlier and later Medicaid states violate the identifying assumption of this "timing-only" estimator. Policy makers at the time reported putting off Medicaid implementation because of fiscal concerns (Advisory Commission on Intergovernmental Relations 1968), and Finkelstein (2007, 10) concludes that, with respect to hospital capacity, "the timing of state implementation of Medicaid was not random."

The coefficients of interest, π_y^k and γ_y^k, measure the (covariate-adjusted) relationship between log mortality and initial AFDC rates in the 16 years leading up to Medicaid's introduction and the 9 years after. The dummy for the year before Medicaid is omitted, which normalizes the estimates

[19] I use AFDC rates for women because this is the appropriate measure of eligibility for the infant (especially neonatal) mortality regressions. The results for noninfant children are unchanged when I use child AFDC rates.

[20] The South includes Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, and Virginia; "border" states include Delaware, Kentucky, Maryland, Texas, and West Virginia, plus Oklahoma and Washington, DC, which are not categorized in Chay et al. (2009).

of π_y^k and γ_y^k to zero in that event year.[21] The π_y^k are falsification tests that capture the relationship between initial categorical eligibility and outcomes before Medicaid existed. 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 an additional percentage point of initial Medicaid eligibility on aggregate mortality. This specification identifies heterogeneity in Medicaid's effect. The estimates will equal zero if Medicaid affected mortality equally across states, and they will understate Medicaid's total effect because they "difference out" common aspects of Medicaid's effect.

V. Estimates of Medicaid's ITT 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 5 plots estimates of equation (1) using child public insurance rates from 1963–76 as the dependent variable. Before Medicaid, public insurance use is uncorrelated with $AFDC_s^*$ (the p-value from a joint significance test of the -3 and -2 coefficients is .70). After Medicaid, public insurance use is 3.8 percentage points higher (s.e. = 0.94; see table 2) for each percentage point of $AFDC_s^*$. These results show that, even conditional on covariates, AFDC-based eligibility is strongly associated with increases in public insurance after Medicaid.[22]

A. *Results for Age-Adjusted Child Mortality by Race*

Figure 6 presents event study estimates of Medicaid's ITT effect on log age-adjusted mortality for children aged 0–14. The small estimates in the 16 years before Medicaid for nonwhite children (panel A) strongly support the AFDC-based research design by ruling out differential trends. The estimates fall sharply immediately after Medicaid implementation, though, matching the pattern in the first-stage estimates (see fig. 5), and remain negative for 9 years. After Medicaid, nonwhite child

[21] Event time dummies that are more than 16 years before or 9 years after Medicaid implementation are grouped because not all states are observed at these event years. I also present the coefficients from a "grouped" event study specification that combines the event time dummies into six bins ([−16, −12], [−11, −8], [−7, −2], [0], [1, 4], [5, 9]) and a DD specification that estimates one treatment effect for event years [1, 9]. I cluster standard errors at the state level to allow for arbitrary serial correlation within states.

[22] Per-recipient expenditures did not change differently after Medicaid in higher- and lower-AFDC states. There is also no cross-sectional relationship between $AFDC_s^*$ and per-recipient expenditures after Medicaid (coefficient = −2.7, s.e. = 14). 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.

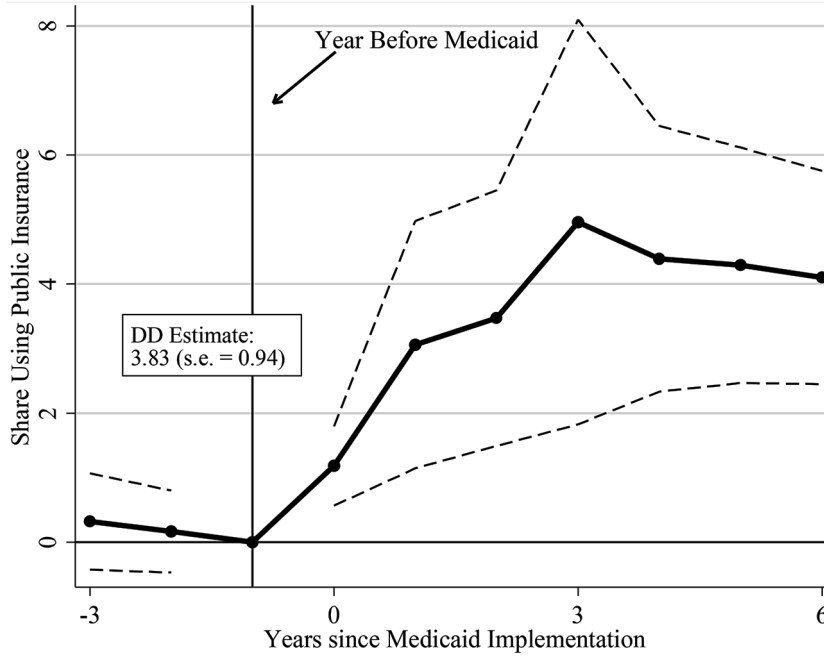

FIG. 5.—Regression-adjusted estimates of Medicaid's effect on children's public insurance use. The dependent variable is the estimated share of children aged 0–19 who received services covered by a means-tested public insurance program. The figure plots the estimated coefficient on interactions between time-to-Medicaid dummies ($1\{t - t_s^* = y\}$) and initial AFDC rates ($AFDC_s^*$) 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 645 state-year observations that have nonmissing 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 = .54; Deaton 1997; Solon et al. 2015).

mortality fell by 1.4 percent (s.e. = 0.34; table 3) for each percentage point difference in initial AFDC rates.

Consistent with the large differences in AFDC variation by race, panel B shows noisy but suggestive estimates for white child mortality rates. The pretrend is relatively flat starting about 10 years before Medicaid, and white mortality fell after Medicaid in higher-AFDC states (the event study point estimates are positive mainly because there is a jump down in the omitted period, −1). The DD coefficient is imprecise but similar to the

TABLE 2
First-Stage Estimates: The Relationship between Initial AFDC Rates and Children's Public Insurance Use

	Dependent Variable: Share of Children Who Used Public Insurance by Type of Service				
	Any (1)	Hospital (2)	Doctor (3)	Drugs (4)	Dental (5)
	A. Grouped Event Study Estimates				
Pre-Medicaid:					
(Years −3 to −2) × $AFDC_s^*$	.26	−.009	.08	.21	.002
	[.36]	[.04]	[.17]	[.23]	[.095]
Post-Medicaid:					
(Year 0) × $AFDC_s^*$	3.80	.42	2.04	2.11	.67
	[1.18]	[.11]	[.47]	[.51]	[.38]
(Years 1 to 4) × $AFDC_s^*$	4.26	.29	2.38	2.75	.87
	[.93]	[.15]	[.44]	[.52]	[.29]
(Years 5 to 6) × $AFDC_s^*$	4.28	.14	1.66	2.22	.75
	[1.03]	[.24]	[.73]	[.72]	[.29]
DD Test (p-value)	.44	.77	.52	.01	.55
	B. Difference-in-Differences Estimates				
Post-Medicaid × $AFDC_s^*$	3.83	.37	2.14	2.26	.76
	[.94]	[.09]	[.44]	[.46]	[.28]
Bootstrap p-value	(.003)	(.001)	(.001)	(.002)	(.008)
Post-Medicaid utilization	10.80	1.10	7.29	5.88	2.29

Source.—DHEW (various years), Haines and ICPSR (2005), and SEER (2013).

Note.—Panel A presents estimated coefficients on the interaction between groups of time-to-Medicaid dummies ($1\{t - t_s^* \in [a, b]\}$) and the initial AFDC rate ($AFDC_s^*$). The model includes per capita hospitals and hospital beds; per capita income; and state, region-by-year, and Medicaid-timing-by-year fixed effects. Estimates are weighted by state populations. The public insurance data measure the share of all children 0–19 (not measured by race) who used medical services paid for by a means-tested public insurance program from 1963 to 1976. The earliest balanced event year is 3 years before Medicaid began, but the estimates are not sensitive to imposing a value of zero before 1950, the first year federal cost sharing in medical vendor payments was authorized. The estimates are normalized to zero in the year before Medicaid implementation. Both $AFDC_s^*$ and public insurance utilization rates range from 0 to 100, so the coefficients give the effect in percentage points of a 1 percentage point difference in initial AFDC-based Medicaid eligibility. Standard errors, clustered by state, are in brackets. 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 0) are equal to each other. The estimates of this specification are presented in panel B. The (years −3 to −2) × $AFDC_s^*$ variable is omitted, (year 0) × $AFDC_s^*$ is included (but not shown), and post-Medicaid × $AFDC_s^*$ refers to all event years between years 1 and 6. The p-values from 1,000 draws of a wild cluster bootstrap percentile t procedure are in parentheses in panel B (Cameron et al. 2008). The sample includes 645 state-year observations that have nonmissing 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).

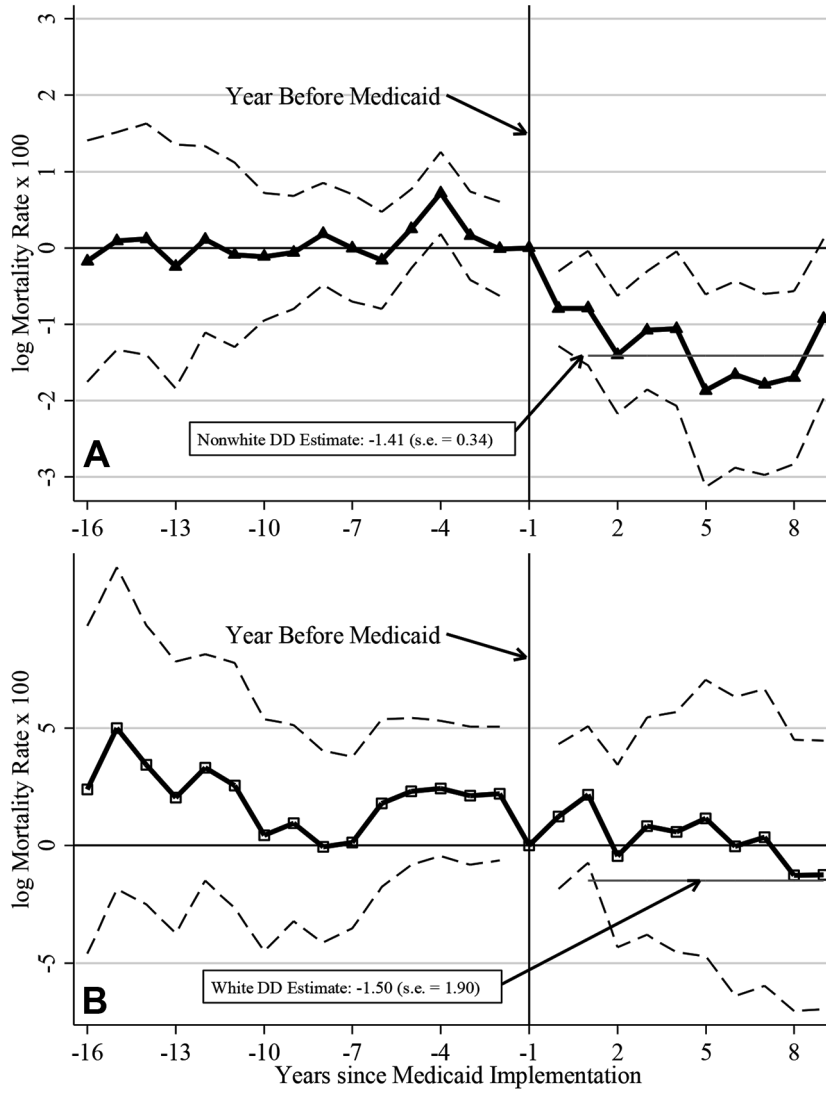

FIG. 6.—Regression-adjusted estimates of Medicaid's intention-to-treat effect on child mortality by race. The dependent variable is the natural log of the age-adjusted mortality rate among children aged 0–14. The figure plots the estimated coefficients on interactions between time-to-Medicaid dummies ($1\{t - t_s^* = y\}$) and initial AFDC rates ($AFDC_s^*$) from 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 16 years before Medicaid (the latest implementing states) or more than 9 years after (the earliest implementing states) are grouped into endpoint dummies, and their coefficients are not shown. Alaska, Hawaii, and Arizona are omitted, and West Virginia is omitted from the white sample. 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. Sources: Vital Statistics Multiple-Cause of Death Files, 1959–79 (DHHS and NCHS); Haines and ICPSR (2005); SEER (2013).

nonwhite estimate (−1.50, s.e. = 1.90) and not statistically distinguishable in a pooled model (difference = −0.09, s.e. = 1.90). The results suggest that Medicaid affected mortality similarly for white and nonwhite children, but the AFDC-based design has sufficient power to detect effects only on nonwhite children. The rest of the results focus on nonwhite child outcomes.

Table 3 shows that the treatment effects on nonwhite children are robust across a range of specifications.[23] Estimates from the simplest possible specification (col. 1), which is equivalent to comparing the slope between mortality and $AFDC_s^*$ across event times, show a significant effect of /Medicaid about half as large as in the full specification from figure 6 (col. 2). Neither omitting the population weights (col. 3) nor including state-specific linear time trends (col. 4) changes the results.[24] Panel B also shows the *p*-value from a test of the DD restrictions in the grouped event study model: the pre-Medicaid coefficients equal zero and the post-Medicaid coefficients (except year 0) are equal. These restrictions are not rejected for any model.

The final two columns provide further support for the AFDC-based research design. Column 5 stacks child mortality rates by race, includes state-by-year fixed effects, and presents coefficients on the triple interaction between the Medicaid dummy, $AFDC_s^*$, and a nonwhite dummy. Column 6 presents two-stage least squares estimates that instrument for the $AFDC_s^*$ interactions using similar variables constructed from 1958 AFDC rates. Neither specification alters the main conclusion that Medicaid reduced nonwhite child mortality.

B. Results for Nonwhite Infant Mortality by Age

The age-adjusted mortality rates discussed above are largely driven by infants, a group for which we have information on deaths by precise age, birth weight, and site of birth. Panel A of figure 7 plots event study estimates for infant mortality by age at death and shows that Medicaid's effects are strongest just after birth. The pre-Medicaid point estimates for first-day mortal-

[23] The analysis includes 45 states, which is typically enough to avoid the bias that arises from standard error estimation using a small number of clusters (Bertrand, Duflo, and Mullainathan 2004; Cameron, Gelbach, and Miller 2008). Panel B of table 3 also shows two-sided *p*-values from 1,000 draws of a wild cluster bootstrap percentile *t* procedure, as suggested by Cameron et al.

[24] A Hausman test cannot reject the null hypothesis of equality between the weighted and unweighted estimates for either panel A (*p*-value = .50) or panel B (*p*-value = .90; Deaton 1997). The motivation for this test is to detect unmodeled heterogeneity or other forms of misspecification, which lead the two estimators to disagree (DuMouchel and Duncan 1983; Solon, Haider, and Wooldridge 2015). I fail to reject the null of equality, so I present the more precise weighted results.

TABLE 3
REDUCED-FORM ESTIMATES: THE RELATIONSHIP BETWEEN INITIAL AFDC RATES AND LOG NONWHITE AGE-ADJUSTED CHILD MORTALITY BY SPECIFICATION, COEFFICIENTS × 100

	(1)	(2)	(3)	(4)	(5)	(6)
	A. Grouped Event Study Estimates					
Pre-Medicaid:						
(Years −16 to −12) × $AFDC_s^*$	−.11	−.006	−.65	.83	.28	1.12
	[.37]	[.69]	[.67]	[.72]	[.77]	[.99]
(Years −11 to −8) × $AFDC_s^*$	−.02	−.008	−.28	.47	.11	−.15
	[.28]	[.4]	[.52]	[.46]	[.44]	[.85]
(Years −7 to −2) × $AFDC_s^*$	.07	.17	−.21	.34	.04	−.60
	[.25]	[.24]	[.39]	[.26]	[.28]	[.69]
Post-Medicaid:						
(Year 0) × $AFDC_s^*$	−.07	−.82	−.53	−.84	−1.06	−1.13
	[.2]	[.25]	[1.1]	[.26]	[.43]	[.42]
(Years 1 to 4) × $AFDC_s^*$	−.67	−1.07	−1.64	−1.21	−1.14	−1.50
	[.22]	[.34]	[.56]	[.4]	[.4]	[.48]
(Years 5 to 9) × $AFDC_s^*$	−.82	−1.59	−1.58	−1.88	−1.78	−1.45
	[.35]	[.51]	[.51]	[.69]	[.49]	[.81]
R^2	.78	.96	.86	.97	1.00	.95
DD test (p-value)	.80	.20	.90	.28	.98	.05

	B. Difference-in-Differences Estimates					
Post-Medicaid × AFDC$_s^*$	−.75	−1.41	−1.27	−1.26	−1.57	−1.46
	[.24]	[.34]	[.43]	[.51]	[.47]	[.4]
Bootstrap p-value	(.06)	(.003)	(.001)	(.03)	(.002)	(.015)
R^2	.78	.96	.86	.97	1.00	.96
Observations	1,418	1,418	1,350	1,418	2,828	1,407
Covariates	High-AFDC FE, time-to-Medicaid dummies	(1) + state FE, Medicaid-timing-by-year FE, region-by-year FE, X_{st}	(2), unweighted	(2) + state-specific linear trends	Pooled races, (2) × nonwhite + state-by-year FE	(2), IV using 1958 AFDC rates
Mortality rate in $t^* - 1$	391.5 deaths per 100,000					

NOTE.—Panel A presents estimated coefficients on the interaction between groups of time-to-Medicaid dummies ($1\{t - t_s^* \in [a, b]\}$) and the continuous value of initial AFDC rates. The estimates are multiplied by 100 to represent the effect in log points of a 1 percentage point difference in initial AFDC rates. Standard errors, clustered by state, are in brackets. The estimates are normalized to zero in the year before Medicaid implementation. Arizona is excluded because it implemented Medicaid in 1982, and Alaska and Hawaii are excluded because they are not measured in Vital Statistics data before 1959. New Jersey did not report race codes in 1962 or 1963, so I allocate total deaths by race using interpolated race share of deaths. Column 3 omits the smallest nonwhite states: Maine, New Hampshire, and Vermont. The row labeled DD test contains the p-value from an F-test of the constant-coefficient DD restrictions: the pre-Medicaid coefficients are jointly zero, and post-Medicaid coefficients (not including year 0) are equal to each other. DD estimates are shown in panel B. A Hausman test does not reject the null hypothesis that weighted and unweighted estimates are equal for either the grouped event study model (p-value .50) or the DD model (p-value .90; Deaton 1997; Solon et al. 2015). The p-values from 1,000 draws of a wild-cluster bootstrap percentile t procedure are in parentheses in panel B (Cameron et al. 2008). Sources: See the notes to fig. 6.

ity are small and insignificant (a linear pretrend is −0.027, s.e. = 0.042) and fall sharply after Medicaid. The DD estimate in table 4 shows that, for each percentage point of initial eligibility, first-day mortality is 1.5 percent lower after Medicaid (s.e. = 0.47, col. 1). The effects in the rest of the neonatal period (days 2–28) are similar but noisier (−1.61, s.e. = 0.86) because only 30 percent of neonatal deaths occur after the first day.[25] The row labeled "contribution to IMR effect" combines the proportional reductions by age group with baseline mortality rates and shows that the first day accounts for half of the overall infant mortality coefficient (very close to its share of deaths, 0.47).

Figure 8 uses data on the hour and day of death (available beginning in 1959) to decompose the neonatal effects further. Each point is a DD estimate of the cumulative mortality rate through each hour or day, so the right-most point is comparable to the neonatal effect from table 4. The results show that nearly all of Medicaid's effect manifests immediately after birth: it is biggest after 3 hours (−1.90 percent for each percentage point of initial eligibility, s.e. = 0.51) and rebounds slightly during the first day. The attenuation most likely reflects selective survival: the weakest babies saved in the first hours do not survive the first day.

Several features of the infant results by age help rule out a closely related explanation: hospital desegregation. First, the desegregation of southern hospitals after 1965 mainly affected post-neonatal infectious disease deaths (Almond et al. 2006), which no infants could have contracted within one day. Second, most southern states implemented Medicaid in 1970, while desegregation began in 1965. Figure 7 does not show a pretrend in first-day or neonatal mortality that would point toward desegregation as an explanation. Third, the model includes year fixed effects for southern and border states, where desegregation was concentrated, and the estimates are unchanged by dropping these states. Fourth, the effects on first-day and neonatal mortality are robust to including post-neonatal mortality rates as a proxy control for the timing, incidence, and health effects of hospital desegregation (as well as other determinants of infant survival). The DD effects on first-day mortality (−1.42, s.e. = 0.46) and neonatal mortality (−1.25, s.e. = 0.35) change little after conditioning on PNMR directly.

[25] The estimates for post-neonatal mortality (PNMR), on the other hand, show a downward pretrend (−0.13, s.e. = 0.08) and are not consistent with an effect of Medicaid. While the DD estimate in col. 4 of table 4 is very similar to the neonatal estimate, the DD restrictions are rejected, suggesting that this is a not a reliable specification for PNMR. Furthermore, this result is sensitive to small sample changes (it falls to −0.89 [s.e. = 0.72] if Texas is dropped) and different AFDC measurements (the effect is −2.6 [s.e. = 4.2] using a binary measure of AFDC that cuts states at the median). The neonatal results are robust to dropping Texas (−1.0, s.e. = 0.37) and to using a binary AFDC measure (−7.1, s.e. = 3.2).

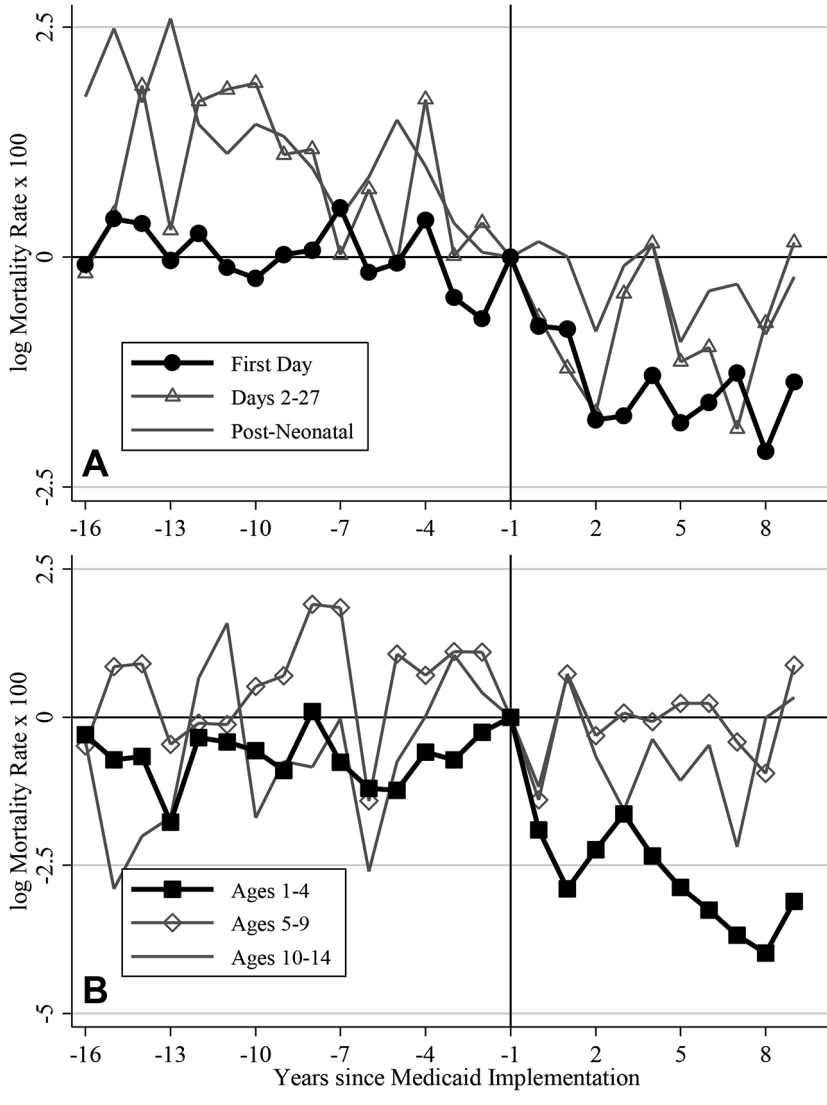

FIG. 7.—Regression-adjusted estimates of Medicaid's intention-to-treat effect on nonwhite child mortality by age. See the notes to figure 6.

C. *Results for Nonwhite Health at Birth versus Conditional Mortality*

Reductions in immediate infant deaths could be due to improvements in health at birth or reductions in mortality rates conditional on fitness at birth. The first three columns of table 5 show small and, in most cases,

TABLE 4
THE RELATIONSHIP BETWEEN INITIAL AFDC RATES AND LOG NONWHITE INFANT MORTALITY BY AGE AT DEATH, COEFFICIENTS × 100

	DEPENDENT VARIABLE: LOG MORTALITY RATE DURING:				
	Day 1 (1)	Days 2–27 (2)	Neonatal Period (before 28 Days) (3)	Post-Neonatal Period (28 Days to 1 Year) (4)	First Year (5)
	A. Grouped Event Study Estimates				
Pre-Medicaid:					
(Years −16 to −12) × $AFDC_s^*$	.16	.85	.31	2.02	1.00
	[.67]	[1.29]	[.73]	[1.03]	[.73]
(Years −11 to −8) × $AFDC_s^*$	−.10	1.47	.37	1.22	.72
	[.51]	[1.18]	[.55]	[.85]	[.50]
(Years −7 to −2) × $AFDC_s^*$	−.09	.47	.08	.75	.35
	[.44]	[.92]	[.45]	[.52]	[.26]
Post-Medicaid:					
(Year 0) × $AFDC_s^*$	−.79	−.76	−.71	.18	−.26
	[.48]	[1.19]	[.52]	[.59]	[.30]
(Years 1 to 4) × $AFDC_s^*$	−1.39	−.77	−1.15	−.14	−.73
	[.57]	[.77]	[.47]	[.46]	[.30]
(Years 5 to 9) × $AFDC_s^*$	−1.63	−.92	−1.37	−.49	−.99
	[.61]	[1.10]	[.51]	[.83]	[.40]
DD test (p-value)	.93	.57	.53	.05	.31
	B. Difference-in-Differences Estimates				
Post-Medicaid × $AFDC_s^*$	−1.50	−1.61	−1.47	−1.44	−1.44
	[.47]	[.86]	[.40]	[.63]	[.44]
Bootstrap p-value	(.02)	(.11)	(.01)	(.06)	(.01)
Contribution to IMR effect	49%	21%	68%	34%	100%
	C. Difference-in-Differences Estimates with Birth Weight Controls				
Post-Medicaid × $AFDC_s^*$	−1.27	−1.57	−1.31	−1.44	−1.33
	[.43]	[.87]	[.40]	[.59]	[.42]
Bootstrap p-value	(.03)	(.14)	(.01)	(.04)	(.01)
Contribution to IMR effect	45%	22%	65%	37%	100%
Observations	1,395	1,361	1,405	1,387	1,417
Rate in $t^* - 1$	17.8	7.1	24.9	12.7	37.6

NOTE.—Panels A and B are structured in the same way as in table 3. The results in panel C control for low and very low birth weight rates (cf. Currie and Gruber 1996a). The rows labeled contribution to IMR effect show the share of the total effect on infant mortality accounted for by each age group. They are calculated by multiplying each coefficient (divided by 100) by the level in $t^* - 1$ (which is approximately equal to the effect on the level of mortality) and dividing by the same ratio using estimates from col. 5. Sources: See notes to fig. 6.

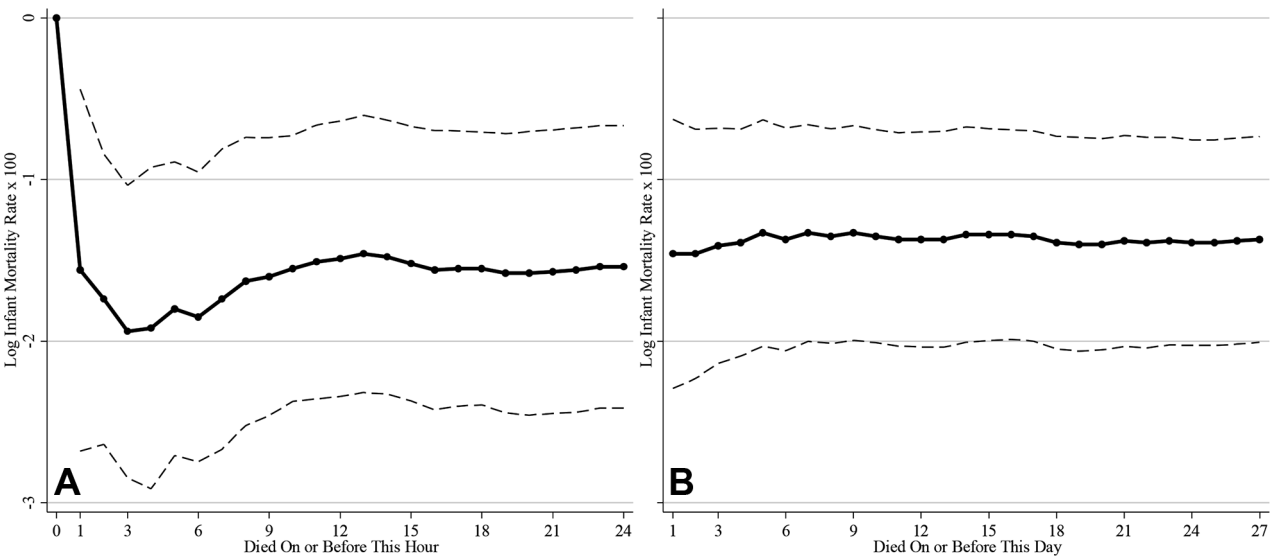

FIG. 8.—Regression-adjusted estimates of Medicaid's intention-to-treat effect on cumulative nonwhite infant mortality by hour and day of death. The figure plots DD estimates of Medicaid's effect on nonwhite infant mortality rates that include the same covariates used in figure 6. The dependent variable is the log of the infant mortality rate before each hour (panel A) or day (panel B) during the first 27 days of life. Moving from left to right on the *x*-axis is equivalent to expanding the window in which mortality is measured. The rightmost estimate is therefore comparable to the effect on neonatal infant mortality rates from column 3 of table 4. Because mortality rates are calculated conditional on being born alive, the effect at "hour 0" is normalized to zero. Data on deaths by detailed hour and day are available only beginning in 1959. Sources: See the notes to figure 6 and table 4.

TABLE 5
THE RELATIONSHIP BETWEEN INITIAL AFDC RATES AND NONWHITE HEALTH AT BIRTH, COEFFICIENTS × 100

	DEPENDENT VARIABLE			
	Log Very Low Birth Weight (≤1,500 Grams) (1)	Log Low Birth Weight (≤2,500 Grams) (2)	Male-to-Female Sex Ratio at Birth (3)	Maternal Mortality (4)
	A. Grouped Event Study Estimates			
Pre-Medicaid:				
(Years −16 to −12) × $AFDC_s^*$	−.21	.55	.07	
	[.76]	[.33]	[.13]	
(Years −11 to −8) × $AFDC_s^*$	−.22	.61	−.13	
	[.61]	[.26]	[.11]	
(Years −7 to −2) × $AFDC_s^*$	−.90	.20	.07	.13
	[.53]	[.20]	[.13]	[.18]
Post-Medicaid:				
(Year 0) × $AFDC_s^*$	−1.02	−.07	.08	.15
	[.76]	[.25]	[.19]	[.19]
(Years 1 to 4) × $AFDC_s^*$	−1.02	.15	−.05	−.07
	[.61]	[.19]	[.15]	[.18]
(Years 5 to 9) × $AFDC_s^*$	−1.45	.25	.03	−.02
	[.67]	[.25]	[.14]	[.21]
DD test (p-value)	.03	.01	.03	.67
	B. Difference-in-Differences Estimates			
Post-Medicaid × $AFDC_s^*$	−.75	−.17	−.02	−.16
	[.44]	[.3]	[.07]	[.10]
Bootstrap p-value	(.13)	(.63)	(.74)	(.12)
Observations	1,327	1,369	1,438	1,008
Rate in $t^* - 1$	23.6	138.5	1.03	7.35

NOTE.—For details on the specification and sources, see the notes to fig. 6. Columns 1 and 2 present coefficients from regressions of the log birth weight variables. Connecticut and Massachusetts are dropped because they did not report birth weight before 1958. Column 3 presents coefficients from a regression of the ratio of male to female births (higher values indicate better infant health since male fetuses are weaker than female fetuses). Column 4 presents coefficients from a regression of the level of age-adjusted maternal mortality using data starting in 1959. Coefficients in columns 1–3 are multiplied by 100.

imprecise effects of Medicaid on three measures of health at birth: the log of very low and low birth weight rates and the male/female sex ratio at birth.[26] The DD estimate for very low birth weight is negative and rel-

[26] Male fetuses are more vulnerable to in utero conditions, so the share of boys carried to term can be interpreted as a measure of average fetal health (Sanders and Stoecker 2015). Birth weight is taken from natality microdata for 1968–79 and from printed volumes from 1950–67. About 0.5 percent of births are missing weight data, and table 4 assumes they are missing at random. Changes in the share missing (per 1,000 births) are unrelated to $AFDC_s^*$ (coefficient = 0.23, s.e. = 0.39).

atively precise (p-value = .12), but the DD restrictions are strongly rejected, the point estimates are slightly negative in the years prior to Medicaid (panel A), and the estimates are sensitive to including the oldest data points.[27] Both the grouped event study and DD point estimates for low birth weight and the sex ratio are small and insignificant. These results differ from evidence based on the timing of Medicaid implementation (Decker and Gruber 1993) and, to some extent, expanded Medicaid eligibility for poor mothers (Currie and Gruber 1996b); however, they are consistent with the skepticism about the effect of prenatal care on birth weight (Fiscella 1995; Alexander and Kotelchuck 2001), with evidence from the introduction of Canadian national health insurance (Hanratty 1996), and with evidence from Medicaid hospital payment reforms in California (Aizer, Lleras-Muney, and Stabile 2004).[28]

Strong mortality reductions without improvements in fitness at birth imply that Medicaid improved survival conditional on health at birth. Panel C of table 4 tests this by adding the birth weight variables to the infant mortality regressions. (Birth weight–specific mortality rates require linked birth and death records, which are not available for this period.) The mortality effects change very little, which lends further support to the claim that Medicaid increased survival conditional on health at birth.

D. Results for Nonwhite Labor and Delivery Care

Medicaid could have reduced such short-run infant mortality rates without improving fetal health mainly through improvements in acute care at birth. Consistent with this explanation, column 5 of table 4 shows that Medicaid reduced an outcome that is highly correlated with hospital care: maternal mortality.[29] Each percentage point of initial eligibility reduces maternal mortality by 0.16 deaths per 100,000 women (s.e. = 0.10), or about 2 percent of the baseline level, which suggests that the

[27] The very low birth weight estimate falls by half in a sample starting in 1959 (−0.42, s.e. = 0.37), but infant mortality results are unaffected (−1.4, s.e. = 0.32).

[28] In economics, Evans and Lien (2005) use a 1992 bus strike in Pittsburgh as an instrument for prenatal care visits and do not detect effects on birth weight or infant health. They find that prenatal care reduces maternal smoking, but advice to avoid smoking would have been less common in the 1960s, only a few years after the Surgeon General's report on smoking in pregnancy was released, and also possibly less common among providers serving poor women (Aizer and Stroud 2010).

[29] At least two-thirds of all maternal deaths in 1965 related to acute events such as abortion (spontaneous or induced; 19 percent of maternal deaths) or complications during and after delivery (33 and 16 percent). Many causes of earlier death could have been prevented in hospitals, such as severe preeclampsia. I use the level rather than the log of age-adjusted maternal mortality (using the 1960 national age distribution of women 15–54) since many cells have no maternal deaths.

mechanisms for infant survival have to do with hospital care that also benefited mothers (cf. Kutinova and Conway 2008).[30]

Table 6 uses the NNFBS to provide direct evidence on labor and delivery care for detailed subgroups. In 1967, 18 states provided AFDC (and Medicaid) to first-time pregnant mothers. Comparing results by potential Medicaid coverage (higher-order births and first births in states that covered them) and poverty status provides an additional test of whether the effects are due to Medicaid. I estimate linear probability models that include the same fixed effects included in equation (1) as well as dummies for mother's age, plurality, order, sex, and family income bins interacted with years. The coefficients of interest are triple interactions between a post-Medicaid dummy, $AFDC_s^*$, and dummies for groups defined by poverty status and the possibility of perinatal Medicaid coverage.

Column 1 shows that each percentage point of initial eligibility is associated with a 0.62 percentage point increase in the hospital birth probability for poor nonwhite mothers whose births were covered by Medicaid (s.e. = 0.32, baseline hospital probability is 88 percent), but births not covered by Medicaid were unaffected.[31] Columns 2 and 3 reestablish that Medicaid did not affect health at birth: even for poor nonwhite women whose births could have been covered by Medicaid, initial eligibility is uncorrelated with changes in the probability of low birth weight or prematurity.

No nationally representative 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). Data from California, however, support an important role for hospital switching. Between 1965 and 1970, the share of black births in public county hospitals fell from 51 to 18 percent (their table 9), and perinatal mortality rates—primarily those in noncounty facilities—fell by 16 percent (their table 2), while the birth weight rate distribution remained constant (California

[30] Maternal mortality is quite noisy in the early 1960s, and the results are much more precise in alternative specifications: controlling for event time −7 (−0.22, s.e. = 0.10), using a binary AFDC measure (−1.74, s.e. = 0.66), or estimating a linear probability model for any maternal deaths (−0.008, s.e. = 0.004). These changes address a spike in maternal mortality in Alabama (a relatively high-AFDC state) in 1963 (event time −7).

[31] This effect also appears to some extent in aggregate data on hospital births in the South. Online app. fig. 2.E13 plots event study estimates of Medicaid's effect on the racial gap in hospital births. The estimates show a clear upward trend break in the South, where most of the racial convergence in hospital births occurred. The DD estimate using $AFDC_s^*$ is quite noisy (0.25, s.e. = 0.39), but one that uses a binary specification is more precise (0.046, s.e. = 0.029, bootstrap p-value = .14). The effect using all states is much smaller, suggesting that the movement of births into hospitals cannot explain the entire neonatal mortality effect, which is robust to dropping the South.

TABLE 6
THE RELATIONSHIP BETWEEN INITIAL AFDC RATES AND NONWHITE BIRTH OUTCOMES BY MOTHER'S AFDC ELIGIBILITY, 1965–72, COEFFICIENTS × 100

	DEPENDENT VARIABLE		
	Born in an Institution (1)	Low Birth Weight (Birth Weight <2,500 Grams) (2)	Premature (Gestation <36 Weeks) (3)
Post-Medicaid × $AFDC_s^*$:			
Potentially Medicaid-eligible mothers:			
Poor	.64	−.24	−.03
	[.33]	[.54]	[.47]
Nonpoor	.20	.20	.54
	[.27]	[.86]	[.61]
Medicaid-ineligible mothers:			
Poor	.20	−.03	.37
	[.31]	[1.03]	[.9]
Nonpoor	.12	−.12	.13
	[.21]	[.7]	[.51]
Observations	3,946	3,946	3,748
Mean dependent variable for poor mothers before Medicaid	88.2	12.2	7.8

SOURCE.—National Natality Followback Surveys, 1964–66 and 1972; National Natality Surveys, 1967–69.

NOTE.—The table contains estimated coefficients (multiplied by 100) 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, $AFDC_s^*$, and indicators for whether the mother's income was below 150 percent of the poverty line and whether the birth was potentially eligible for Medicaid. 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.

Public Health Service 1973). The results are similar to those of Aizer et al. (2004), who find that moving Medicaid mothers into better hospitals reduced neonatal mortality and prematurity among black Medicaid recipients but had no effect on birth weight.

Medicaid's effects on infants match perinatal epidemiological research, which ascribes only a small share of neonatal mortality declines since 1950 to changes in fitness at birth (Lee et al. 1980; Williams and Chen 1982; David and Siegel 1983; Collins and Thomasson 2004; Cutler and Meara 2004). Therefore, Medicaid can help explain the aggregate changes in race-specific neonatal mortality and the important contribution of acute care and survival conditional on health at birth.

E. Results for Nonwhite Child Mortality by Age and Cause

While infants accounted for most deaths under age 14, the vast majority of Medicaid children were older than 1. Panel B of figure 7 shows that Medicaid's effects on this group are concentrated among the youngest children. Each percentage point of initial eligibility is associated with a −2.23 percent reduction in younger child mortality (s.e. = 0.55; col. 1, table 7). Columns 2 and 3 suggest that the effects decline with age. The DD estimate for ages 5–9 is negative but less than a quarter of the effect for younger children (−0.51, s.e. = 0.40), and the result for ages 10–14 is nearly zero (−0.13, s.e. = 0.56).

Columns 4 and 5 bear out another prediction from Section II: internal-cause mortality responds more to Medicaid than does external-cause mortality. The DD effect on internal-cause mortality is a 1.88 percent reduction and is very precisely estimated (s.e. = 0.41), while the proportional effect on external-cause mortality is smaller (−0.96, s.e. = 0.55) and statistically distinguishable (p-value = .12). About 70 percent of the combined effect comes from internal causes ($-1.88 \times 48/(-1.88 \times 48 - 0.96 \times 39.2) = 0.70$). Columns 5 and 6 split internal-cause deaths into untreatable and treatable causes (most of which are infectious diseases). The effect on treatable cause mortality is −2.16 (s.e. = 0.51), compared to only −1.09 (s.e. = 0.80) for untreatable causes.

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 6, Medicaid would have had to provide such care. Columns 2–5 of table 2 provide additional first-stage DD estimates of Medicaid's effect on hospital admission, physician visits, drug prescriptions, and dental services. The pattern of increases in public health care use corresponds to the types of care that were effective in reducing the types of mortality that actually decreased for young children after Medicaid. One percentage point of initial eligibility is associated with more than a 2 percent increase in children's use of (publicly financed) doctor visits and prescription drugs but smaller increases in hospital admissions and dental visits (largely because baseline utilization is low).

VI. Evidence on Potential Threats to Identification

The event study results rule out differential trends as a source of bias but not variables that differ by $AFDC_s^*$ and change sharply at the same time as Medicaid implementation (but are not caused by it). If initial AFDC-based Medicaid eligibility signals states' willingness to adopt other social policy reforms of the 1960s, then the estimates of γ_y^k could capture the effects of related War on Poverty programs. To test this hypothesis, I estimate versions of equation (1) using per capita expenditures or

participation rates for five programs that could have also affected child mortality: Community Health Centers (CHC), other health programs funded by the Community Action Program, Head Start (per 1,000 children aged 1–9), the Food Stamp Program, and AFDC itself.[32]

Panel A of figure 9 shows that $AFDC_s^*$ is uncorrelated with post-Medicaid changes in per capita expenditures on other health-related programs. This is especially reassuring for Head Start and Food Stamps, which also affect infant and child health (Ludwig and Miller 2007; Almond, Hoynes, and Schanzenbach 2011).[33] Panel B also shows that AFDC participation itself does not change differentially after Medicaid in higher-AFDC states. Furthermore, evidence on the relationship between welfare receipt and health is mixed (Currie and Cole 1993; Bitler, Gelbach, and Hoynes 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.[34]

Another approach to rule out alternative explanations is to add other measures of state welfare programs that should be highly correlated with omitted determinants of mortality. Panel A of table 8 presents the results of a placebo test that adds interactions of the event time variables with initial white AFDC rates to the regressions for nonwhite mortality rates. The treatment effects are unchanged, and the relationship between changes in nonwhite mortality and white eligibility is small.

The history of AFDC, however, suggests that omitted determinants of mortality such as discrimination, industrial structure, or safety net policy may be specifically correlated with nonwhite AFDC participation. Because the identification strategy uses nonwhite AFDC rates at one point in time, I address this concern by controlling for the actual state-by-year nonwhite AFDC rate and its interaction with a post-1966 dummy to allow for possible structural changes in AFDC participation during the 1960s (Moffitt 1987). Panel B of table 8 shows that the treatment effects are only slightly reduced by these flexible controls, suggesting that the changes in nonwhite AFDC rates during the 1960s cannot account for

[32] For the expenditures and participation rates that are not measured by race, $AFDC_s^*$ is based on overall AFDC rate (as in the first-stage results) rather than race-specific AFDC rates.

[33] The DD estimate for CHC spending is statistically significant, but it is more than an order of magnitude smaller than the public insurance estimate. This includes much higher expenditures for older users, so it overstates the per capita CHC spending for children. Furthermore, Bailey and Goodman-Bacon (2015) find no evidence that CHCs affect child or infant mortality, which means that even a large change in funding is not a plausible explanation for the mortality results.

[34] Net cross-state population flows between 1965 and 1970 are uncorrelated with $AFDC_s^*$. This is consistent with the fact that per-recipient benefits did not increase in higher-AFDC states and suggests that selective migration cannot explain the mortality results.

TABLE 7
THE RELATIONSHIP BETWEEN INITIAL AFDC RATES AND LOG NONWHITE CHILD MORTALITY BY AGE AND CAUSE, COEFFICIENTS × 100

	Dependent Variable: Log Mortality Rate For:						
	Ages 1–4 (1)	Ages 5–9 (2)	Ages 10–14 (3)	Internal Causes, Ages 1–14 (4)	External Causes, Ages 1–14 (5)	Treatable Causes, Ages 1–14 (6)	Untreatable Causes, Ages 1–14 (7)
	A. Grouped Event Study Estimates						
Pre-Medicaid:							
(Years −16 to −12) × $AFDC_s^*$	−.75	.17	−1.31	−.34	−.96		
	[.92]	[.86]	[1.1]	[.88]	[.79]		
(Years −11 to −8) × $AFDC_s^*$	−.41	.80	−.47	.11	−.25		
	[.89]	[.73]	[1.05]	[.66]	[.88]		
(Years −7 to −2) × $AFDC_s^*$	−.77	.75	−.34	−.15	−.31	−.35	.73
	[.72]	[.71]	[.99]	[.77]	[.87]	[.89]	[1.37]
Post-Medicaid:							
(Year 0) × $AFDC_s^*$	−1.90	−1.38	−1.27	−1.05	−2.04	−1.22	−.61
	[1.06]	[.96]	[1.29]	[.85]	[.97]	[.93]	[1.58]
(Years 1 to 4) × $AFDC_s^*$	−2.27	.11	−.57	−1.67	−.90	−1.85	−.63
	[.72]	[.82]	[1.05]	[.68]	[.71]	[.78]	[1.08]
(Years 5 to 9) × $AFDC_s^*$	−3.38	−.02	−.77	−2.33	−1.83	−3.11	−.30
	[.93]	[.91]	[1.12]	[.72]	[.98]	[.78]	[1.11]
DD test (p-value)	.25	.53	.56	.68	.53	.06	.85

	B. Difference-in-Differences Estimates						
Post-Medicaid × AFDC$_s^*$	−2.23	−.51	−.13	−1.88	−.96	−2.16	−1.09
	[.55]	[.40]	[.56]	[.41]	[.55]	[.51]	[.80]
Bootstrap p-value	(.001)	(.18)	(.81)	(.001)	(.12)	(.002)	(.28)
Observations	1,362	1,305	1,279	1,349	1,357	929	849
Rate in $t^* - 1$	153.5	58.5	55.1	48.0	39.2	35.7	12.3

NOTE.—For details on the specification and sources, see the notes to fig. 6. Columns 1–3 contain estimates for the log of age group–specific mortality rates for nonwhite children. Columns 4 and 5 contain estimates for the log of age-adjusted internal- and external-cause mortality rates (see the notes to fig. 4). Columns 6 and 7 contain estimates for the log of age-adjusted infectious- and noninfectious-disease mortality rates, which together account for the vast majority of internal-cause deaths. For the ICD codes included in these groups, see online app. 1. The final row shows the average level (not log) of the dependent variable in the year prior to Medicaid implementation.

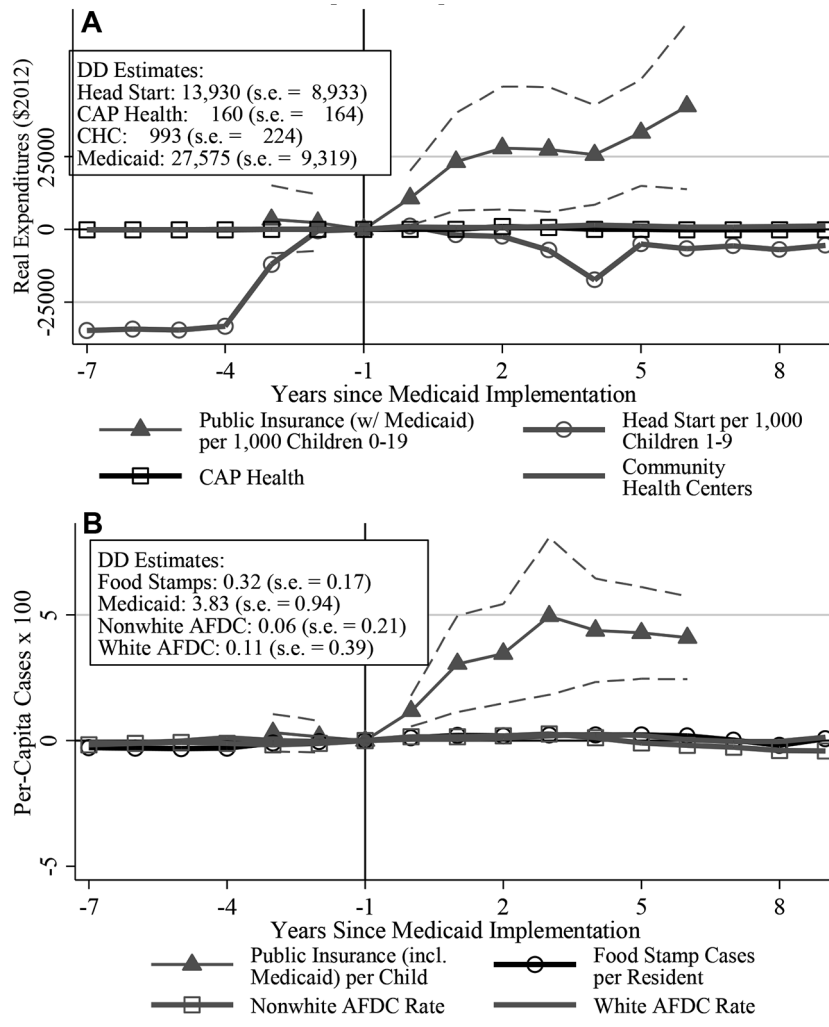

FIG. 9.—The relationship between Medicaid implementation and health-related programs. The figure plots estimated coefficients on interactions between Medicaid timing dummies and $AFDC_s^*$ in a regression model described in Section III. The dependent variable in panel A is funding per capita (in 2012 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 aged 0–19 (measured from 0 to 100). 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 2015; Food Stamps: Almond et al. 2011) did not grow coincidentally with Medicaid in higher-AFDC states. Sources: National Archives Community Action Program and Federal Outlays Files, Public Health Service Reports, DHEW (1963–76), and USHHS (2012). I thank Hilary Hoynes for sharing the Food Stamp caseload data.

TABLE 8
THE ROBUSTNESS OF MEDICAID'S MORTALITY EFFECTS TO TIME-VARYING AFDC CONTROLS, COEFFICIENTS × 100

	Dependent Variable		
	Log Nonwhite Mortality, Ages 0–14 (1)	Log Nonwhite Neonatal Infant Mortality (2)	Log Nonwhite Mortality, Ages 1–4 (3)
	A. Controlling for White-AFDC Medicaid Timing Interactions		
Treatment effects:			
Post-Medicaid × nonwhite $AFDC_s^*$	−1.43	−1.40	−2.13
	[.55]	[.62]	[.78]
Bootstrap *p*-value	(.05)	(.12)	(.04)
Falsification test:			
Post-Medicaid × white $AFDC_s^*$	.09	.06	.19
	[.25]	[.26]	[.30]
	B. Controlling for State-by-Year Nonwhite AFDC Rate		
Post-Medicaid × $AFDC_s^*$	−1.23	−1.37	−1.59
	[.37]	[.30]	[.78]
Bootstrap *p*-value	(.005)	(.001)	(.16)

NOTE.—The table contains estimates from two specification tests. Panel A presents coefficients on interactions between a post-Medicaid dummy and nonwhite $AFDC_s^*$ (treatment effects) and white $AFDC_s^*$ (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 a post-Medicaid dummy and an indicator for high-nonwhite-AFDC states as in eq. (1). The regressions also include 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. Sources: See the notes to fig. 6.

the strong correlation between initial categorical eligibility and the timing of Medicaid implementation.

VII. Discussion: 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 effects on infant and child mortality are too large to come from 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 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. 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.[35] I also adjust for the higher mortality rates of Medicaid recipients using survey data on mortality by income.

Figure 10 plots estimates of the ATET from this paper and from the three most closely related Medicaid papers (Currie and Gruber 1996a, 1996b; Wherry and Meyer 2013).[36] 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.[37] 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.

[35] This is also borne out in fig. 1, 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. Loewenstein (1971) finds that 8 percent of categorically eligible Medicaid families in 1968 had any health insurance, and only 3 percent had anything other than hospital or surgical insurance. Among all poor families in states that had not yet adopted Medicaid (mostly in the South plus New Jersey and Indiana), only 6 percent had doctor visit insurance.

[36] For more recent papers, I use first-stage estimates for any health insurance other than Medicaid coverage and adjust them by a factor of 0.85 to account for underreporting of Medicaid in survey data (Card, Hildreth, and Shore-Sheppard 2004; Davern, Klerman, and Ziegenfussi 2007).

[37] The confidence intervals in fig. 10 assume zero correlation between the components of the ATET. Across the full range of values for this correlation, the confidence intervals never include zero or −100 percent. I thank Alejandro Molnar for this suggestion.

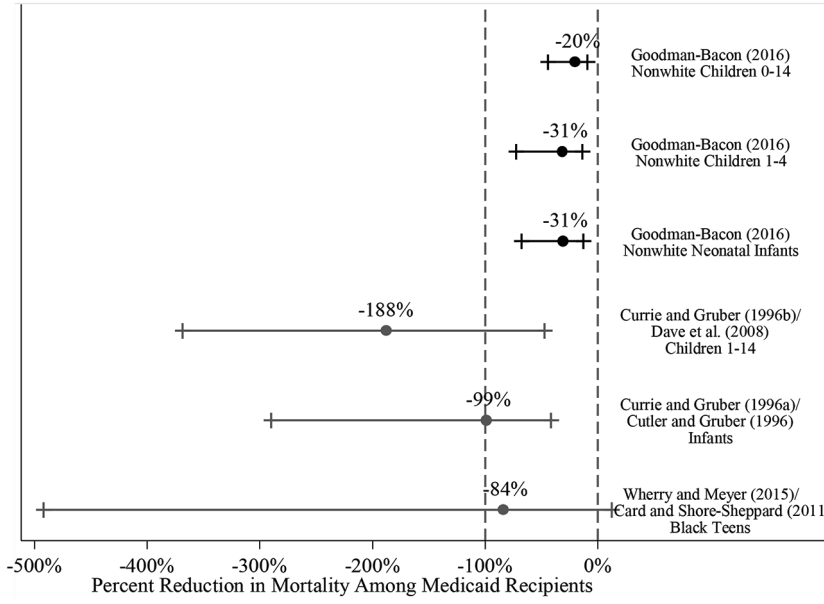

FIG. 10.—The proportional effects of Medicaid on the mortality rates of newly insured recipients: average treatment effects on the treated (ATET). The figure plots the implied ATET and 95 percent confidence intervals for three comparable previous studies and for the DD results emphasized in this paper: all nonwhite children aged 0–14, nonwhite neonatal infants, and younger nonwhite children aged 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 underreporting when appropriate), then adjusted for differential baseline mortality among poorer Medicaid recipients. The right-most column lists the source of the mortality/first-stage estimates. The vertical dashed line at zero indicates that Medicaid does not reduce mortality, and the dashed line at −100 percent indicates the largest possible value of the ATET (because a group's mortality rate cannot be reduced by more than its baseline level). The 95 percent confidence intervals are generated using a modified percentile method from 10,000 replications of a parametric bootstrap procedure (Efron and Tibshirani 1993; Valetta 1993; Johnston and DiNardo 1997). 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 online appendix 4 for details on the bootstrap procedure and alternative estimators for the confidence intervals.

The ATET estimates reaffirm that Medicaid significantly reduced nonwhite infant and child mortality rates, and the magnitudes are smaller than −100 percent, satisfying a necessary condition for attributing effects to Medicaid's insurance coverage alone. The ATETs imply a 20 percent mortality reduction for nonwhite children under 14 and a 31 percent re-

duction for younger nonwhite children (aged 1–4) and nonwhite neonates.[38]

These estimates imply large individual health effects compared to other interventions. A 30 percent reduction in neonatal mortality is comparable to the effect of gaining a full pound in birth weight (Almond, Chay, and Lee 2005)—about 10 times the effect of Food Stamp implementation on birth weight among treated black infants (Almond et al. 2011). Chay and Greenstone (2003) find that the 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 gastroenteritis or pneumonia. The infant mortality effects are also comparable to those of many public health programs. 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 reduced infant mortality by as much as 25 percent (Watson 2006).

The ATETs from the 1980s expansions, on the other hand, are much larger than the effects in this paper. The estimates imply a 99 percent mortality reduction for infants, a 188 percent reduction for children, and an 84 percent reduction for black teens.[39] This is surprising since the AFDC children who gained insurance because of Medicaid implementation were poorer and less healthy than many of the groups that 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, Løken, and Neilson 2013). Another interpretation is that some of the other consequences of the 1980s expansions—increased consumption for crowd-out families or take-up of other programs—were not at work in the 1960s, when categorically eligible families spent little on medical care and received welfare by definition. This suggests that the

[38] Another measure of the size of the treatment effects is the "number needed to treat" (NNT) to avert one death, which equals the inverse of the absolute reduction in mortality risk. One advantage of this parameter over a proportional ATET is that it reflects secular changes in mortality rates. Between 1966 and 1979, the average NNT based on the age-adjusted effects is 915 children, but this grows from 548 in 1966 (when counterfactual mortality among treated children was 840.2 deaths per 100,000) to 1,403 in 1979 (when counterfactual mortality among treated children had fallen to 356.6 deaths per 100,000). Average NNT for neonates is 97 (ranging from 75 to 130) and 1,204 for young children (ranging from 703 to 1,695).

[39] The regression discontinuity (RD) mortality estimate in Wherry and Meyer (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.

mortality reductions documented here reflect the effect of new Medicaid coverage itself and that Medicaid's effect per eligible person in the 1980s reflects additional causal channels.[40]

What do these effects imply for current Medicaid policy? Some channels documented above, such as increases in hospital births, are not relevant for low-income families today, while others, such as hospital switching, have continued to be important mechanisms for improving infant survival (Aizer et al. 2004). Technological improvements also mean that losing Medicaid coverage today may imply a larger cost in terms of health than in the past. The extent to which these estimates apply to children who would be affected by current Medicaid reform proposals is an open question, but given the lack of policy variation for children, this paper provides clear new support for the notion that Medicaid—in any era—improves child health.

B. Medicaid's Aggregate Costs and Benefits

The results imply not only an important reduction in individual-level mortality risk but also a role for Medicaid implementation in aggregate mortality changes in the 1960s and 1970s. By 1976, about 35 percent of nonwhite children used Medicaid. Auxiliary survey data suggest that counterfactual mortality among treated children was 447.3 deaths per 100,000.[41] A 20 percent ATET, therefore, implies that Medicaid implementation reduced aggregate mortality by 11 percent and the poverty gap in nonwhite child mortality by a third.

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 suggests that, between 1966 and 1979, 35,087 nonwhite deaths were averted as a result of Medicaid (2,506 deaths per year). Most of these deaths would have occurred among neonates and young children, for whom the remaining life expectancy in 1966 was about 65.5 (Vital Statistics life tables), which implies a gain of 2.3 million life-years saved.

Through 1976, Medicaid spent about $5.8 billion (in 2012 dollars) per year on all children aged 0–19. Assuming that expenditures on children aged 0–14 were proportional to their share of child Medicaid recipients (78 percent in 1976) and that no white children benefited, this implies a

[40] 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, 1276) and 34 percent per eligible child (Currie and Gruber 1996a, 454).

[41] An observed aggregate mortality rate of 252.7 and an estimated actual mortality rate among the treated of 357.7 imply an aggregate mortality rate among those not treated of $(252.7 - 0.35 \times 357.7)/0.65 = 196.5$, a counterfactual poverty gap in mortality of 250.7, and a counterfactual aggregate mortality rate of $0.65 \times 196.1 + 0.35 \times 447.3 = 283.9$ deaths per 100,000.

cost per death averted of about $1.83 million and a discounted cost per life-year saved of about $64,000.[42] Infant deaths were significantly cheaper to avoid than deaths among young children: the cost per death averted is $160,000 for nonwhite neonates and $2.1 million for young nonwhite children. Comparable estimates from the 1980s expansions (Currie and Gruber 1996b, 1996a) are about $1.7 million and $2.6 million (2012 dollars) per infant and child death averted, which shows that Medicaid achieved mortality reductions at this time for a fraction of the cost of more recent expansions.

These costs refer only to the contemporaneous expenditures relative to life-years gained, but Medicaid's benefits may extend into later life in terms of health (Miller and Wherry 2014; Boudreaux, Golberstein, and McAlpine 2016; Goodman-Bacon 2016), educational attainment (Cohodes et al. 2014), and productivity (Brown, Kowalski, and Lurie 2014; Goodman-Bacon 2016). 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 to the contemporaneous benefits documented here.

VIII. Conclusion

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. These findings presumably understate Medicaid's broader effects because they measure benefits only in terms of mortality rather than reductions in morbidity or delayed health and productivity effects. Therefore, Medicaid, like several other federal health and antipoverty programs established under the Great Society, played a major role in improving health and reducing mortality in the 1960s and 1970s.

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[42] 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: $(1 - 0.97^{65})/(1 - 0.97) = 28.7$.

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