Saving Teens

Using a Policy Discontinuity to Estimate the Effects of Medicaid Eligibility

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ABSTRACT

We examine the immediate and longer-term mortality effects of ~~childhood~~ public health insurance eligibility during childhood. Our identification exploits expansions in Medicaid eligibility that applied only to children born after September 30, 1983. This feature resulted in a large discontinuity in the cumulative years of eligibility of children at this birth date cutoff. Under the expansions, black children gained twice the years of Medicaid eligibility as white children. We find a later-life decline in the rate of disease-related mortality for black cohorts born after the cutoff. We find no evidence of a similar mortality improvement for white children.

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I. Introduction

Interest in public health insurance in the United States is in part motivated by the relatively poor health of children in families with low incomes. Not only are children from poor families more likely to be born in poor health, but the relative health of these children worsens with age (Case, Lubotsky, and Paxson 2002). To address this disparity in child health, U.S. policy has largely focused on increasing access to medical care for children through expansions in publicly provided health insurance.[1] These expansions aim to promote health among children by providing timely access to health services. A major focus is preventive care and the early diagnosis and treatment of physical and mental health needs with aims to limit pervasive health problems in adulthood (Rosenbaum et al. 2005).

There is strong evidence that public health insurance coverage for children increases their utilization of medical care, but the evidence that this leads to improved health outcomes is much more limited.[2] The strongest evidence of health improvements is seen in contemporaneous declines in child mortality associated with expanded public health insurance eligibility. In two seminal papers, Currie and Gruber (1996a, 1996b) find that Medicaid expansions in the mid-1980s and early 1990s reduced mortality among both infants and children aged one to 14, with declines in child deaths where the underlying cause was disease related (internal). The authors find no effect on child deaths due to other (external) causes, such as accidents or homicides. Howell et al. (2010) examine expansions in public insurance for children aged one to 17 occurring over a longer time period and find additional evidence of a decline in child mortality although concentrated among deaths due to external causes.

Until recently, there has been little attention to the long-term effects of public health insurance coverage for children. Yet many pediatric health services are designed to protect healthy children from future disease or risks, ~~such as immunizations and health education,~~ and the payoffs from these types of services might not be evident until later in life. Furthermore, for those children with mental and physical disabilities or chronic illness, early diagnosis and treatment may help the management of these conditions or limit their persistence into adulthood.[3] From a program evaluation standpoint, the effect of public coverage on the long-term health of children is an important component of program benefits.

1. A series of expansions of the Medicaid program beginning in 1984, and later expansions under the State Children's Health Insurance Program (SCHIP), are estimated to have more than doubled the share of children eligible for public health insurance from 17.8 percent in 1987 (Cutler and Gruber 1996) to 41.7 percent in 2000 (LoSasso and Buchmueller 2004).

2. Several papers have documented an increase in annual doctor visits (Currie and Gruber 1996a, Card and Shore-Sheppard 2004, Currie et al. 2008) and hospital use (Currie and Gruber 1996a, Dafny and Gruber 2005) under expansions in public health insurance for children. Yet, a number of studies using parental reports of child health measures, including child health status and activity limitations, find no evidence of improvement under public health insurance (for example, Currie and Gruber 1995; Racine et al. 2001; Currie, Decker, and Lin 2008). Howell and Kenney (2012) provide a comprehensive review.

3. Medical care may also have a positive impact through counseling regarding behavior risks and linking children to helpful social services. The standard economic argument is also applicable that providing free medical care may free up resources for making other investments with long-term impacts.

A small but growing literature considers the longer-term health effects of public health insurance for children. Two published papers examine later-life health using self-reported measures of health and with mixed findings. Using within-state variation in past eligibility for different birth cohorts, Currie, Decker, and Lin (2008) find evidence of better self-reported health status at ages nine to 17 among cohorts in states with greater eligibility for public health insurance at ages two, three, and four. De la Mata (2012) examines the longer-term effects of public eligibility by comparing health outcomes among children aged five to 18 in families with incomes just above and below the income eligibility thresholds for public health insurance. Examining outcomes five years after a given year of eligibility, she finds no evidence of significant effects on child health status, obesity, and the number of school days missed due to illness. More recently, a number of new ~~working~~ papers find evidence linking Medicaid eligibility during childhood with improved health (Boudreaux, Golberstein, and McAlpine ~~2014~~; Miller and Wherry ~~2014~~) or economic outcomes at later ages, including higher rates of high school and college completion (Cohodes et al. ~~2014~~) and improved labor market outcomes (Brown, Kowalski, and Lurie 2015). Each of these papers relies on variation within states over time in the generosity of Medicaid eligibility rules and assumes exogeneity in the timing of state eligibility changes.

This paper provides new evidence on the immediate and longer-term health effects of public health insurance during childhood using a more clearly exogenous source of variation than the existing literature. Our identification strategy exploits a unique feature of several early Medicaid expansions that extended eligibility only to children born after September 30, 1983. The change in eligibility occurred when these cohorts were almost eight years old, making it impossible that families could have altered their delivery dates in anticipation of the change. This policy discontinuity was first identified and used by Card and Shore-Sheppard (2004) to examine changes in insurance coverage and medical care utilization under the expansions.[4]

Building on their pathbreaking work, we show that this eligibility rule led to a large *cumulative* difference in public health insurance eligibility for children born before and after September 30, 1983. Children in families with incomes at or just below the poverty line gained close to five additional years of eligibility if they were born in October 1983 rather than just one month before. This gain in eligibility occurred between the ages of eight and 14. Using regression discontinuity methods, we examine mortality at these ages for cohorts of children born just before and after the birth date cutoff to identify the immediate impact of this gain in public health insurance eligibility.

Importantly, our research design also allows us to examine the impact of childhood Medicaid eligibility later in life by following cohorts born before and after the cutoff through age 23 to observe any *longer-term* impacts on mortality. We examine changes in mortality at the ages of 15–18 and 19–23 when cohorts on either side of the cutoff were equally likely to be eligible for public health insurance. This allows us

4. The authors found an increase in coverage among newly eligible children born after this date with little evidence of crowdout of private insurance. They also provide evidence that this new Medicaid coverage led to a large increase in the utilization of medical care.

to attribute any changes in mortality we observe at these ages to differences in Medicaid eligibility that occurred earlier in childhood.

As Lee and Lemieux (2010) have emphasized, the regression discontinuity methods that we employ can be just "as good as a randomized experiment." Although not an experiment, our research design approximates random assignment in public health insurance for children, a group for which there has been no randomized trials of private health insurance coverage or Medicaid.[5] And with this variation occurring nationwide, it offers advantages in terms of the generalizability of findings, larger sample sizes, and the ability to examine long-term impacts.[6] For these reasons, the results of this policy experiment merit special attention.

Following the literature, we examine rates of mortality by the underlying cause of death, distinguishing between deaths due to internal and external causes in our analysis. As mentioned earlier, both types of death have been linked to changes in public insurance coverage. We also examine changes in mortality separately by child race since black children were particularly likely to benefit from the Medicaid expansions due to their distribution of family income. Existing evidence also suggests that expansions in public insurance were more likely to affect black children.[7]

We find compelling evidence of a sizeable decline in the later-life mortality of black children under the Medicaid expansions. The regression estimates indicate a significant decrease in the internal mortality rate for black children born after September 30, 1983, at ages 15–18. The improvement in mortality for black children is not reversed during the early adult years at ages 19–23, although there are no clear additional gains at these ages resulting from childhood eligibility. We find no evidence of a similar decline in mortality for white children under the expansions.

This paper is structured as follows: Section II provides some background on the Medicaid expansions that resulted in discontinuous childhood eligibility at the September 30, 1983, birth date cutoff. Section III reviews our research design and data. Section IV presents our results, while Section V interprets them. Section VI concludes with a summary of our findings and their applicability to public policy.

5. The RAND Health Insurance Experiment randomized people into plans with different levels of cost sharing but provided all participants with health insurance. Researchers found no significant differences in health measures for children in families randomly assigned to health insurance plans providing medical care free of charge and those requiring some form of copayment despite reduced demand for ambulatory care for children in families enrolled in the cost-sharing plans (Valdez et al. 1985). Some gains in health were observed among poor adults with existing chronic health problems, while individuals between the ages 12 and 35 on the free-care plan showed some improvement in dental health (Manning et al. 1987).

6. For instance, while the Oregon Health Insurance Experiment found improvements in self-reported health and depression among adults gaining Medicaid coverage, it had less statistical power to detect changes in outcomes with lower prevalence including mortality and specific physical measures of health (Finkelstein et al. 2012, Baicker et al. 2013).

7. Currie and Gruber (1995) find a much larger mortality decline among black children than white children (more than 4× the size) in their analysis, although the changes are not statistically significant for either group. A recent paper by Goodman-Bacon (2014) finds large declines in nonwhite infant and child mortality following the introduction of Medicaid in the 1960s and 1970s. He finds no evidence of similar improvements in mortality for white infants and children. Howell et al. (2010) report no significant difference between black and white children in the effect of the eligibility expansions, which they test by including a black race interaction term in their model. The point estimate is not reported, however, making it difficult to gauge whether the estimate is small and precisely estimated or potentially large and imprecise.

II. The Discontinuity in Eligibility

Medicaid is a joint federal-state program that provides health services to certain low-income beneficiaries. Since 1967, the program has included a special benefit package for children designed to provide comprehensive health coverage including preventive care. This benefit package provides all Medicaid children with early and periodic screenings for physical, mental, and developmental health; vision, dental, and hearing examinations and followup care; and all health services they are found to need. Importantly, children with eligibility for Medicaid have "conditional coverage" in that, even if they do not actively enroll in the program, their expenses are covered in the event of hospitalization or high health care spending (Cutler and Gruber 1996).

Historically, not all children in poor families were eligible for Medicaid coverage. Eligibility for nondisabled children was previously linked to participation in the Aid to Families with Dependent Children (AFDC) program. Beginning in 1984, a series of Congressional acts expanded eligibility for Medicaid to children who were not traditionally eligible for AFDC, often with income levels above AFDC cutoffs. To phase in the Medicaid expansions, many of the legislative changes were applied only to children born after September 30, 1983 (see Table A1 in the online appendices for additional details).[8] This legislative provision meant that children born in October 1983 faced more generous eligibility criteria for the Medicaid program than children born just one month earlier in September 1983. The result of this provision was a large discontinuity in the number of childhood years of Medicaid eligibility for children born around this birth date cutoff.

To illustrate the discontinuity, we model childhood public eligibility for monthly cohorts born within four years of the birth date cutoff—between October 1979 and September 1987. Using detailed federal and state eligibility rules for the years 1979–2005, our simulation program uses information on state of residence, family structure and size, parent employment, and family income to calculate monthly public eligibility status through age 17.[9] To obtain a nationally representative sample of these characteristics of children and families, we employ data from the March Supplements to the Current Population Survey (CPS). We use a random sample of 500 children of ages zero to 17 from each year of the 1981–88 CPS and estimate the childhood eligibility for this pooled sample for each birth month.[10]

Figure 1 shows the results of our simulation for children in families with incomes below 150 percent of poverty, those most likely to be affected by the change in Medicaid eligibility rules. This graph depicts the average total number of years of public eligibility during childhood by birth month cohort. Each line on the graph represents an income

8. The appendices referred to in the text may be found online at http://jhr.uwpress.org/.

9. For the years prior to welfare reform, we estimate eligibility for Medicaid under AFDC, state Ribicoff rules, and federal and state Medicaid expansions. For 1997 forward, eligibility is estimated under the postwelfare reform eligibility rules for Medicaid, as well as under continuing state Medicaid expansions and new separate state programs funded by SCHIP. See Appendix A for additional details.

10. We use the 1981–88 CPS surveys because the information collected on income is for the previous calendar year. This simulation holds family characteristics constant over the child's lifetime. This convention may understate eligibility during early childhood and overstate eligibility during the later years of childhood if family income grows with child age and is not counterbalanced by changes in family size that alter the poverty thresholds to which income is compared.

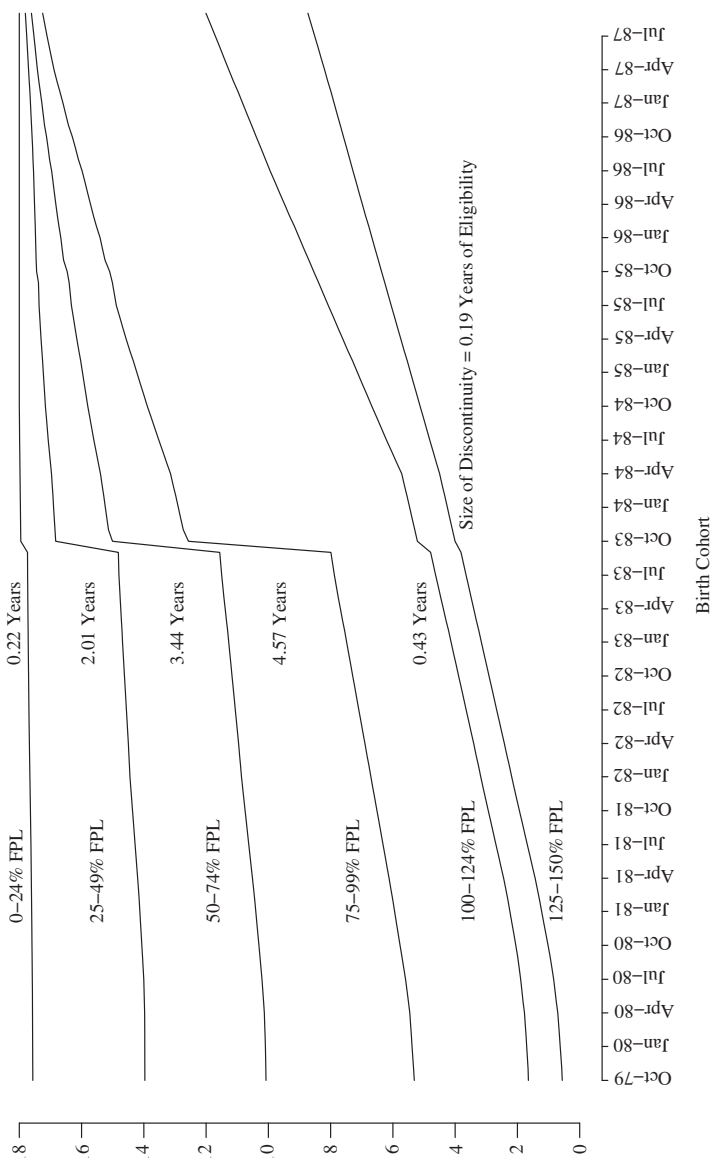

Figure 1

Average Years of Childhood Eligibility for Medicaid/CHIP by Birth Cohort and Family Income (Percent FPL)

Notes: Weighted average calculated using the characteristics and state of residence of a sample of children aged zero to 17 in the 1981–88 March CPS. See text for more information. Family income is indexed using the CPI-U and assumed to be constant over the child's lifetime.

Table 1
Childhood Medicaid Eligibility Gain for Children Born in October versus September 1983 by Child Race

	Average Gain (in Years) for Population	Percent Gaining Eligibility	Average Gain (in Years) for Children Gaining Eligibility
All children	0.42	9.24	4.53
Black children	0.82	17.13	4.81
White children	0.37	8.18	4.48

Note: Weighted average calculated using a sample of children aged zero to 17 in the 1981–88 March CPS and eligibility simulation methods detailed in the text.

bracket. For all income brackets, we see a gradual increase in childhood eligibility across birth cohorts prior to a jump in years of eligibility for children born in October 1983. Children born in October 1983 are the first cohort of children to be eligible for the Medicaid expansions with the September 30, 1983, birth date cutoff. Following this jump, we again see a gradual increase in childhood eligibility for the remaining birth cohorts.[11]

As depicted on Figure 1, the magnitude of the discontinuity in childhood eligibility at the September 30, 1983, cutoff varies by family income. The largest jump in years of eligibility occurs among children with family incomes above AFDC eligibility levels but below poverty. Children in families with incomes just below the poverty line see the largest gain with an additional 4.6 years of eligibility during childhood, while those with incomes between 50–74 percent of the poverty line see a gain of 3.4 years. We see a smaller discontinuity in childhood eligibility among children in families with incomes at or below AFDC levels, around 50 percent of the poverty line. Many of these children were already eligible for Medicaid through AFDC prior to the expansions.

We examine the effects of the expansions separately for black and white children. At the time of the Medicaid expansions, black children were nearly three times as likely to be in poverty as white children. Therefore, we may see a larger impact of the expansions among black children. Table 1 reports the estimated discontinuity in childhood eligibility at the September 30, 1983, cutoff by child race.[12] We estimate that ~~17.3~~ 1 percent of black children gained on average just under five years of eligibility compared to similar gains in eligibility for 8.2 percent of white children. As a result, black children gain, on

11. Much of this gradual increase is due to a larger share of each successive cohort's life occurring when younger children were Medicaid eligible under various age cutoffs rather than a gradual change in rules.

12. To compare the eligibility gains by child race, we use a sample that draws a maximum of 500 children aged zero to 17 from each race group and state cell from the pooled 1981–88 CPS years, giving us 50,874 child observations. Holding family characteristics constant, we estimate eligibility for each child for each year during childhood by calculating eligibility in July of the appropriate calendar year. We use these estimates to calculate the average gain in years of eligibility at the September 30, 1983, cutoff by child race. In calculating all averages that we report in the text and in tables, we multiply the CPS-provided survey weights by an adjustment factor specific to each cell in order to account for the manner in which we constructed our sample. This factor is calculated as the inverse of the ratio of the sum of the survey weights of individuals in the cell in our sample and the sum of the survey weights of individuals in the cell in the CPS.

average, 0.82 years of eligibility, which is more than twice the average years gained by white children (0.37 years).

To illustrate the timing of the gain in eligibility, Figure 2 compares the share of children eligible for public health insurance if born in October versus September 1983 at each year of age by race. The main legislative source behind the jump in eligibility is the implementation of the Omnibus Reconciliation Act of 1990 (OBRA90). Effective July 1, 1991, OBRA90 mandated state coverage to children younger than age 19 born after September 30, 1983, with family incomes below the poverty line. Children born in October 1983 were almost eight years-old when this change was implemented. For both black and white children, we see a sizeable jump in the share of October-born children with eligibility at age eight. However (as indicated in Table 1), a much larger share of black children is affected by the change in eligibility rules.

Faced with new and more generous eligibility criteria, the October 1983 cohort remains more likely to be eligible for public health insurance until the introduction of the State Children's Health Insurance Program (CHIP) in August 1997. Authorized by the Balanced Budget Act, CHIP allowed states to expand eligibility for public health insurance to children of higher incomes, regardless of the child's month of birth, under existing state Medicaid programs or new state programs. These expansions were quickly adopted by states and served to close any discontinuity in eligibility by age 15.

We distinguish between four different age ranges in our analysis. Ages four to seven capture a period prior to the implementation of OBRA90 where differences in eligibility at the birth date threshold are small. Our analysis of this period serves to investigate any differences in health for children at the cutoff, although this period also captures minor differences in eligibility. Later, we test for baseline differences in the health of these cohorts using measures of infant health and find no evidence of preexisting differences.

Next, we examine children at ages eight to 14. Children who benefitted from the expansions primarily gained eligibility during this age range, as shown in Figure 2. We interpret any discontinuity in health observed during this period as an immediate effect of public health insurance coverage, similar to the existing estimates using cross-sectional data in the literature (for example, Currie and Gruber 1996a). However, any difference in health observed in our estimates may also be due to a cumulative gain in exposure to health insurance coverage over the period. For instance, a child aged 14 born in October 1983 may have gained Medicaid coverage for the five years prior, beginning at age eight. There is little research that has examined the effect of multiyear coverage on health care access or outcomes for children[13] despite the potential complementarities in investments in health at different ages (Becker 2007).

Finally, we examine children at ages 15–18 and ages 19–23, which are periods following the gain in public eligibility. At these ages, cohorts born on either side of the birth date cutoff were equally likely to be eligible for public health insurance. By examining these older age groups, we are able to consider the longer-term effects of childhood coverage during late adolescence and early adulthood.

13. One exception, Cassedy, Fairbrother, and Newacheck (2008), shows that children with any gap in insurance coverage over a two-year period have higher odds of lacking a usual source of care and not having well-care visits than those with continuous insurance coverage, regardless of whether that continuous coverage was public or private. There is also a substantial literature finding that continuous coverage during a year is associated with better access to care (see Leininger 2009 for evidence and cites to the literature).

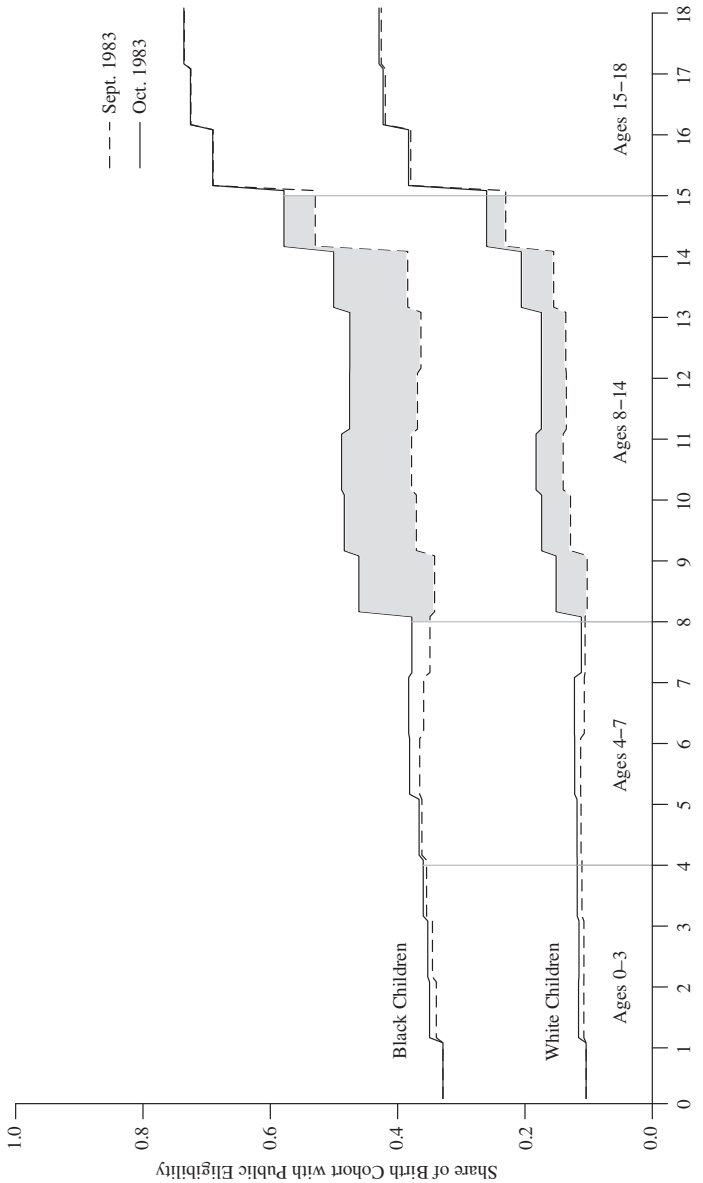

Figure 2

Average Public Eligibility at Each Age of Childhood by Birth Month Cohort and Child Race

Notes: Weighted average calculated using the characteristics and state of residence of a sample of black or white children aged zero to 17 in the 1981–88 March CPS. See text for more information. Family income is indexed using the CPI-U and assumed to be constant over the child's lifetime.

III. The Research Design

Our research design uses comparisons across the September 30, 1983, birth date cutoff to measure the impact of Medicaid eligibility on mortality. Because factors other than date of birth determine childhood eligibility for public health insurance, such as childhood family income, this is an example of a "fuzzy" RD design. Birth cohort is defined in our analysis using the month and year of birth since this was the finest information on date of birth that we were able to acquire in the mortality data. Our sample includes all cohorts born between October 1979 and September 1987. We denote birth cohorts using the integer values $c \in [-48, 47]$, where $c = 0$ for the birth cohort October 1983, the first cohort born after the cutoff date September 30, 1983.

For each outcome variable, we first present graphical evidence to visually check whether there is evidence of a discontinuity at the September 30, 1983, birth date cutoff. We then present regression estimates to confirm the statistical significance of any observed discontinuity and its robustness to the inclusion of covariates. Our main analysis is based on a regression discontinuity (RD) model of the form

$$(1) \quad y_{ca} = \alpha + f(\gamma;\, c) + \beta 1(c \geq 0) + \delta_m M_c + \varepsilon_{ca}$$

where y_{ca} represents the mortality rate for cohort c at age a, $f(\cdot)$ is a smooth function of birth month cohort with parameter vector γ, and $1(c \geq 0)$ is an indicator for whether the cohort was born after September 30, 1983. We also include calendar month dummies M_c given the evidence on seasonal variation in maternal characteristics for births and its relation to later outcomes (Buckles and Hungerman 2013).[14] We interpret β as the effect of the eligibility expansions averaged across the full sample of children at the cutoff.[15]

The central question for implementation is how to model the function of birth month cohort $f(\cdot)$. We present estimates of the treatment effect using both linear and quadratic specifications of the regression function. We also report estimates where we allow the elements of the parameter vector of these specifications to differ on the two sides of the birth date cutoff (specifically linear and quadratic splines). In addition, we examine the sensitivity of our estimates to the use of narrower windows of observations around the birth date cutoff and present results for four-year ($c \in [-48, 47]$), three-year ($c \in [-36, 35]$), and two-year ($c \in [-24, 23]$) windows of birth month cohorts on either side of the cutoff. All regressions are estimated using OLS.

As described above, we estimate Equation 1 separately for each of the following age groups: four to seven, eight to 14, 15–18, and 19–23. In accompanying graphs, we plot the means of the outcome variable by birth cohort in the four-year observation window separately for each of the four age groups. The lines are fitted values from a regression that includes a second-order polynomial in birth cohort and a dummy variable for children born after September 30, 1983. In our regression results, heteroskedasticity-robust standard errors are clustered by birth month cohort to account for our grouping of narrow age ranges into broader ones, as we describe below.[16]

14. The inclusion of calendar month dummies will also account for the influence of other policies that may have differential effects based on month of birth. One example might be birth date cutoffs for school entry.
15. In other words, we estimate the proportion eligible times an intent-to-treat parameter.
16. Clustering on each value of the discrete assignment variable can also account for uncertainty in the choice of the functional form, as shown by Lee and Card (2008).

A. Outcomes and Data

To measure the effects of childhood Medicaid eligibility on immediate and long-term health, we use mortality records for all deaths in the United States. In contrast to parental reported measures, mortality is a clear and objective measure of health. However, examination of this measure is not without its limitations. Given that mortality is a relatively infrequent event among children, any changes in population-level mortality rates are likely to be small and may be difficult to assess. In addition, a focus on mortality may miss other important but difficult to measure effects of public health insurance on the health or quality of life of children. Acknowledging these limitations, reduced mortality remains an unambiguous and important outcome of expanded health insurance coverage.

Mortality is also an outcome for which potentially we might also see longer-term impacts of health insurance coverage for children. Children face lower mortality rates during the middle childhood years and higher rates of death in early and late childhood.[17] Given this relationship, the impact of childhood coverage on underlying health might be more apparent during the adolescent years when mortality is higher.

We ~~construct aggregate mortality using~~ information from the National Vital Statistics System (NVSS) Multiple Cause of Death files for the years 1979–2011. We construct cohort-specific death rates, where cohort is defined as the month and year of birth. While the date of birth of the decedent is excluded from the publicly available mortality files, we are able to access birth month and year as restricted information at Census Research Data Centers. Necessarily, we drop all observations from the mortality data that ~~is~~ missing information on child birth month or year. The average share of child observations with missing birth date information for each year of mortality data is about 2 percent. We also exclude those observations where the birth year information contradicts the reported age of the child. In addition, we use only observations for children born in the United States with residence in the 50 U.S. states and the District of Columbia. None of these restrictions are discontinuous at the September 30, 1983, cutoff and our results do not change when using an unrestricted sample (see sensitivity analyses in online Appendix B).

For our outcome measure, we construct the rate of death for each of the 96 birth-month cohorts in our sample for the following age subgroups: four to five, six to seven, eight to ten, 11–12, 13–14, 15–16, 17–18, 19–20, and 21–23. The observations for each of our four age group regressions include the 96 cohorts times the two or three age subgroups that make up the four age ranges described earlier. The definition of these four wider age groups was motivated by the ages affected by the expansions as shown in Figure 2. To construct rates of death, we tally the total number of deaths for each birth cohort c at specified years of age a. We then divide this number by the total population at risk for each birth cohort and age, defined as the total population of births for each birth cohort minus the total number of deaths for all ages prior to age a.[18] We construct

17. Figure A1 shows the age profile of rates of mortality due to internal and external underlying causes for children in their first year of life through age 19 in the year preceding the Medicaid expansions. For both types of death, the relationship with child age takes on a U-shape, though it is less pronounced for internal causes.

18. We obtain birth counts from the NVSS Birth Data files for the years 1979–87. The 1979–84 files offer only a 50 percent random sample of births for a handful of states, stratified by month of birth and county of occurrence. For each of these states and years, we double the number of births for each birth cohort to approximate the universe of births. We make this adjustment for the following states and years: Ariz., Calif., Del., Ga., and D.C. in 1979–84, N.D. in 1979–82, N.M. in 1979–81, and Ark. and S.D. in 1979.

separate outcomes for black and white children and express all final rates as the number of deaths per 10,000 children in each birth cohort. In sensitivity analyses, we examine regressions using the numerator only (number of deaths for each birth cohort) as an alternative dependent variable, with no meaningful impact on our findings.[19]

In our results, we distinguish between internal and external causes as the underlying cause of death, which we classify according to the medical code reported on the death certificate. For the years 1999–2011, the cause of death is reported according to the Tenth Revision of the International Classification of Diseases (ICD-10), while the Ninth Revision (ICD-9) is used for the ~~1980~~ 98 years. Because the two classifications differ slightly, we include an ICD-10 dummy in our regression specifications to account for this change.[20]

We also examine more narrow classifications of the underlying causes of death. For internal causes, we code subcategories following the chapter structure of the ICD-10.[21] We code subcategories of external causes following the groupings in a tabulation list developed by the National Center for Health Statistics for use with mortality data classified by ICD-10.[22] To aid in constructing comparable measures over time, we draw on the assignment of ICD-9 codes to the ICD-10 tabulation list presented in Anderson et al. (2001).[23]

Table 2 presents the average rates of death for cohorts born prior to September 30, 1983, in our sample by race and age group. Mortality is much higher among older children across all groups, with mortality rates for 19-23-year-olds almost four times as high as those of children ages four to seven. Although deaths due to internal causes are more common among older children, it is the frequency of deaths due to external causes that drives most of the increase in total mortality for the older age groups. Black children have higher rates of mortality than white children across all age groups.

Among all children, the most common causes of internal death are neoplasms (tumors), diseases of the nervous system (which include meningitis, epilepsy, and cerebral palsy), cardiovascular diseases, respiratory diseases, and congenital malformations and chromosomal abnormalities. With the exception of neoplasms, deaths from these causes are more frequent among black children than white children. Additionally, infectious and parasitic diseases are also a major cause of death among black children.

19. The results of this analysis may be found in Figures A3 and A4 and Tables A6 and A7.

20. Earlier versions of this paper included additional covariates including age and calendar year dummies. We no longer include age dummies in our regression specification since these covariates are balanced on either side of the birth date threshold. In addition, we have replaced the calendar year dummies with the ICD-10 dummy in order to more directly control for changes in the classification of deaths over time.

21. There are 18 chapters of internal causes in the ICD-10. Because deaths falling under a handful of chapters are extremely rare among children, we chose the 11 chapters with the highest number of deaths in our sample and grouped the remaining seven chapters into an "other" category for a total of 12 categories.

22. We follow the List of 113 Selected Causes of Death (113-cause list) to create our subcategories for external causes. We have omitted deaths due to "Operations of war and their sequelae" since there were no deaths in our sample falling under this subcategory.

23. Anderson et al. (2001) provide detailed information on the classification and rule changes between the ICD-9 and ICD-10 revisions and construct comparability ratios by case of death. They find substantial discontinuities in mortality trends resulting from implementing the ICD-10. Again, the inclusion of the dummy for the ICD-10 period in our regression specification should serve to control for this change.

Table 2
Average Rate of Death for Children Born Before September 30, 1983, by Underlying Cause of Death, Race, and Age Group

	Black Children				White Children			
	4–7	8–14	15–18	19–23	4–7	8–14	15–18	19–23
Deaths from all causes	3.62	3.33	8.94	14.43	2.40	2.22	6.35	8.86
Deaths due to internal causes	1.53	1.46	2.32	3.71	1.20	0.99	1.39	2.02
Infectious and parasitic diseases	0.15	0.15	0.17	0.37	0.08	0.06	0.07	0.08
Neoplasms (malignant and nonmalignant)	0.32	0.29	0.42	0.53	0.40	0.29	0.38	0.49
Diseases of blood, blood-forming organs, immune mechanism	0.07	0.06	0.07	0.19	0.02	0.02	0.02	0.02
Endocrine, nutritional, and metabolic diseases	0.05	0.05	0.10	0.22	0.07	0.07	0.10	0.15
Mental and behavioral disorders	0.02	0.01	0.03	0.06	0.02	0.01	0.03	0.09
Diseases of the nervous system	0.21	0.21	0.26	0.31	0.15	0.14	0.19	0.23
Cardiovascular and circulatory system diseases	0.14	0.20	0.47	0.80	0.09	0.11	0.21	0.32
Respiratory system diseases	0.15	0.21	0.25	0.28	0.09	0.08	0.11	0.13
Digestive system diseases	0.06	0.04	0.08	0.14	0.04	0.03	0.03	0.06
Congenital and chromosomal abnormalities	0.22	0.12	0.16	0.16	0.19	0.11	0.10	0.12
Symptoms and signs not elsewhere classified	0.09	0.08	0.18	0.30	0.04	0.04	0.11	0.24
All other diseases	0.03	0.05	0.14	0.35	0.03	0.02	0.04	0.09

Deaths due to external causes	2.09	1.86	6.61	10.72	1.20	1.23	4.95	6.84
Accidents (unintentional injuries)	1.81	1.24	2.49	3.30	1.11	0.94	3.52	4.62
Transport accidents	0.83	0.63	1.79	2.55	0.62	0.63	2.94	3.35
Nontransport accidents	0.98	0.61	0.69	0.74	0.48	0.31	0.57	1.24
Falls	0.02	0.01	0.02	0.03	0.02	0.01	0.05	0.08
Discharge of firearms	0.05	0.08	0.15	0.11	0.02	0.06	0.07	0.05
Drowning and submersion	0.26	0.29	0.31	0.18	0.15	0.08	0.15	0.12
Exposure to smoke, fire, and flames	0.53	0.14	0.07	0.07	0.15	0.05	0.04	0.05
Poisoning/exposure to noxious substances	0.02	0.01	0.05	0.23	0.01	0.01	0.13	0.78
Other and unspecified	0.11	0.07	0.10	0.13	0.13	0.10	0.13	0.16
Intentional self-harm (suicide)	0.00	0.09	0.52	0.92	0.00	0.15	0.88	1.32
Assault (homicide)	0.23	0.49	3.47	6.33	0.07	0.11	0.48	0.69
Legal intervention	0.00	0.00	0.03	0.07	0.00	0.00	0.01	0.02
Events of undetermined intent	0.04	0.03	0.10	0.09	0.01	0.02	0.06	0.18
~~Operations of war and their sequelae~~	~~0.00~~	~~0.00~~	~~0.00~~	~~0.00~~	~~0.00~~	~~0.00~~	~~0.00~~	~~0.00~~
Complications of medical and surgical care	0.01	0.01	0.00	0.00	0.01	0.01	0.01	0.00

Notes: Average of annual rates of death per 10,000 children calculated for each birth month cohort between October 1979 and September 1983. Mortality rates constructed using National Vital Statistics System Multiple Cause of Death files for the years ~~1983~~ 2011 and NVSS Birth Data files for the years 1979–83.

A common cause of deaths due to external causes is transport-related accidents. Accidental drowning and submersion is also a frequent cause of death at younger ages. Among older children, deaths due to suicide and homicide are prevalent, with suicide more likely among white children and homicide more likely among black children.

IV. Results

Figure 3 graphs internal-cause mortality rates by birth month cohort for each race at ages four to seven, eight to 14, 15–18, and 19–23. In the panels on the left, we see very little evidence of a change in internal mortality for white children born after the September 30, 1983, cutoff. For black children, we see some evidence of a decline in internal mortality at the cutoff at ages eight to 14, although the plot is relatively noisy. There is particularly strong evidence, however, of a decline in internal mortality for black cohorts born after the cutoff at ages 15–18. This suggests a longer-term mortality improvement for black children under the expansions. There is no visual evidence, however, of a similar discontinuity at ages 19–23.

Table 3 provides the corresponding regression estimates. Each cell reports the estimated discontinuity for children at the September 30, 1983, birth date cutoff, or the coefficient β in Equation 1, from a regression for the specified age group and window size. Again, there is some evidence of a decline in internal mortality at ages eight to 14 for black children born after the cutoff—that is, during the period of extended Medicaid coverage. However, the coefficient estimates are only significant under the quadratic spline specification.

When we examine the longer-term impact of the expansions at ages 15–18, we find strong evidence supporting a decline in internal-cause deaths for black children. The coefficient estimates using the four-year observation window are statistically significant under all four functional form specifications. With a baseline rate of 2.32 deaths per 10,000, the point estimates indicate just over a 19 percent decrease (19.1–19.6) in the internal mortality rate for black children at these ages. For the most part, the coefficient estimates are robust to alternative window sizes, indicating similarly sized and significant declines. The estimates under the quadratic spline specification for the smaller windows are imprecise and in the case of the two-year window no longer negative. However, the confidence intervals on these estimates are much larger than under the other functional form specifications and do not allow us to rule out declines similar to those in the other specifications. Combined with the compelling visual evidence in Figure 3, these findings strongly indicate a mortality improvement for black children under the expansion in Medicaid eligibility. We do not, however, see evidence of similar declines at ages 19–23. Furthermore, neither the regression results nor the visual evidence indicate any improvement in the internal mortality of white children.

Figure 4 presents external-cause mortality rates by birth month cohort for each race and age group. The majority of panels in this figure reveal a strong downward trend in external mortality. Separate from this trend, we see little compelling evidence of changes in mortality for cohorts born after the September 30, 1983, threshold. If anything, the plots for white children suggest an increase in external mortality at the cutoff at ages eight to 14 and 15–18, as does the plot for black children at ages 15–18. We also see some evidence of a decline in external mortality at ages four to seven for black children, although this particular plot is fairly noisy.

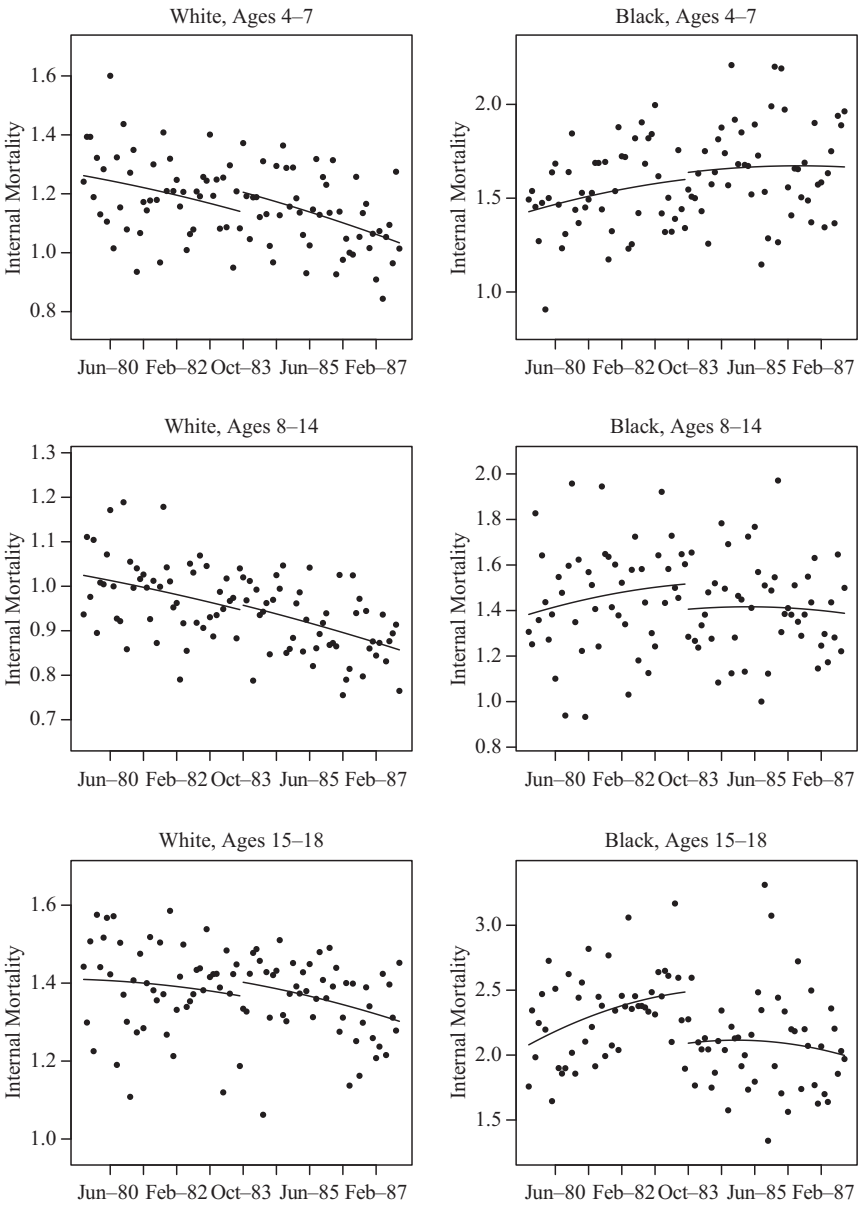

Figure 3

Child Mortality from Internal Causes by Child Race

Notes: Mortality rates by cohort (per 10,000 children) were constructed using Multiple Cause of Death Data files for the years 1979–2011 and Birth Data files for 1979–87. Points represent means of the age-specific mortality rates for each birth cohort, as described in the text. The lines are fitted values from a regression that includes a quadratic in birth cohort and a dummy variable for children born after September 30, 1983.

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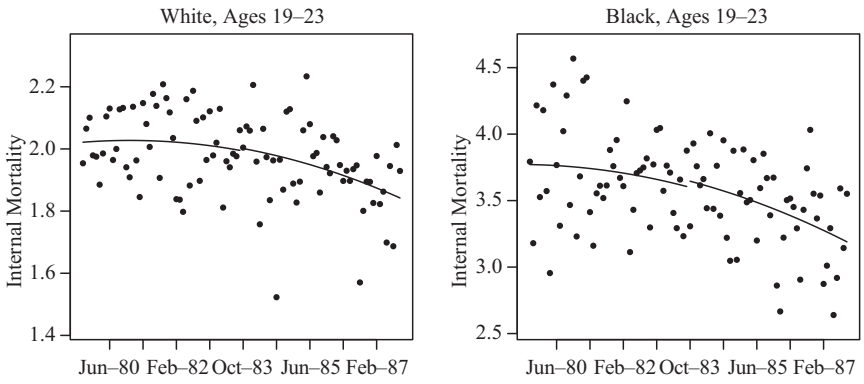

Figure 3 (*continued*)

Table 4 presents the regression estimates for external mortality. Again, we find some evidence of a baseline difference at ages four to seven for black children at the cutoff. However, the estimates are not consistently significant across functional forms and are not statistically significant when using smaller windows of observations. Without strong visual support as well, we do not interpret these estimates as convincing evidence of differences in external mortality for black children at these ages.

The evidence of an increase in external mortality for white children at the cutoff at ages eight to 14 is surprisingly more robust. Our estimates using the four-year observation window indicate a 7–9 percent increase in external mortality over the baseline of 1.23 deaths per 10,000 children. With the exception of the quadratic spline specification, the estimates using smaller windows around the cutoff are of similar size and statistically significant. Visually, however, Figure 4 does not present evidence of a change in external mortality of the size seen in the regression estimates.[24] The lack of compelling visual evidence in the raw data gives us pause in the interpretation of this finding as an effect of the Medicaid expansions. Later, we investigate the likelihood of encountering an estimate of this size by chance.

We find no evidence of a change in external mortality for either black or white children aged 15–18 and mixed evidence of a change for black children aged 19–23. The regression estimates under the two-year window and under the more flexible quadratic spline specification indicate a statistically significant decline in external mortality. However, the estimates using larger window sizes suggest much smaller declines and even reverse in sign. The plot of the raw data in Figure 4 does not indicate a discontinuity at the cutoff for this group. Taken together, we do not interpret this to be a robust finding.

24. We should note that the inclusion of calendar month dummies in the regression specification appears to lead to slightly larger and more significant estimates. This suggests that seasonality might be making it difficult to discern a discontinuity in the graph. We should emphasize, however, that both the size and significance of the decline in internal mortality that we find for black cohorts born after September 30, 1983, are robust to the inclusion of calendar month dummies. Results for specifications without calendar month dummies may be found in Tables A8 and A9.

Table 3
Change in Annual Internal-Cause Mortality Rate for Children Born After September 30, 1983, by Race and Age Group

	Black Children			White Children		
	Four-Year Window	Three-Year Window	Two-Year Window	Four-Year Window	Three-Year Window	Two-Year Window
Ages 4–7						
Linear	0.024	0.042	–0.009	0.036	–0.013	–0.031
	(0.089)	(0.104)	(0.132)	(0.041)	(0.049)	(0.056)
Linear spline	0.023	0.043	–0.006	0.036	–0.014	–0.030
	(0.090)	(0.103)	(0.129)	(0.041)	(0.049)	(0.056)
Quadratic	0.024	0.042	–0.009	0.036	–0.013	–0.031
	(0.089)	(0.104)	(0.134)	(0.041)	(0.048)	(0.056)
Quadratic spline	0.095	0.007	0.060	–0.005	–0.022	–0.037
	(0.114)	(0.129)	(0.155)	(0.061)	(0.070)	(0.074)
Baseline mean	1.525	1.562	1.560	1.205	1.186	1.192
N	192	144	96	192	144	96
Ages 8–14						
Linear	–0.098	–0.081	–0.144	0.014	0.018	0.029
	(0.090)	(0.099)	(0.125)	(0.029)	(0.033)	(0.042)
Linear spline	–0.099	–0.083	–0.146	0.013	0.017	0.028
	(0.086)	(0.096)	(0.122)	(0.028)	(0.032)	(0.041)
Quadratic	–0.094	–0.079	–0.142	0.014	0.018	0.029
	(0.087)	(0.097)	(0.123)	(0.028)	(0.032)	(0.041)
Quadratic spline	–0.206*	–0.268**	–0.321**	0.012	–0.001	–0.045
	(0.109)	(0.117)	(0.153)	(0.038)	(0.041)	(0.050)
Baseline mean	1.462	1.489	1.484	0.988	0.980	0.971
N	288	216	144	288	216	144
Ages 15–18						
Linear	–0.443***	–0.465***	–0.460**	0.028	–0.000	–0.009
	(0.126)	(0.151)	(0.195)	(0.048)	(0.052)	(0.068)
Linear spline	–0.447***	–0.465***	–0.453**	0.025	–0.000	–0.006
	(0.124)	(0.148)	(0.195)	(0.045)	(0.050)	(0.065)
Quadratic	–0.448***	–0.471***	–0.467**	0.022	–0.001	–0.010
	(0.125)	(0.148)	(0.194)	(0.046)	(0.051)	(0.066)

(*continued*)

Table 3 (*continued*)

	Black Children			White Children		
	Four-Year Window	Three-Year Window	Two-Year Window	Four-Year Window	Three-Year Window	Two-Year Window
Quadratic spline	–0.456**	–0.349	0.053	0.008	0.011	0.077
	(0.197)	(0.258)	(0.320)	(0.071)	(0.072)	(0.085)
Baseline mean	2.322	2.387	2.440	1.393	1.378	1.384
N	192	144	96	192	144	96
Ages 19–23						
Linear	0.052	0.098	0.067	–0.007	–0.024	–0.071
	(0.127)	(0.143)	(0.162)	(0.054)	(0.065)	(0.084)
Linear spline	0.049	0.096	0.070	–0.009	–0.024	–0.070
	(0.126)	(0.143)	(0.164)	(0.054)	(0.065)	(0.085)
Quadratic	0.052	0.098	0.067	–0.007	–0.024	–0.071
	(0.125)	(0.143)	(0.163)	(0.054)	(0.065)	(0.085)
Quadratic spline	0.120	0.091	0.175	0.005	–0.041	–0.045
	(0.174)	(0.189)	(0.234)	(0.070)	(0.086)	(0.097)
Baseline mean	3.712	3.694	3.680	2.019	2.018	2.011
N	192	144	96	192	144	96

Notes: Coefficients from OLS regressions with the specified function in birth cohort, a dummy for deaths classified under ICD-10 and calendar month dummies. Standard errors reported in parentheses are robust and clustered by birth month cohort, *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

A. Specific Causes of Death

Our results provide compelling evidence of a decline in mortality under the expansions for black children born after September 30, 1983, aged 15–18. We also find evidence suggesting a smaller increase in external mortality for white children born after the cutoff at ages eight to 14. To better understand the potential role of expanded Medicaid eligibility behind these changes in mortality, we study more specific underlying causes of death at these ages using the classification described earlier. The existing literature examining Medicaid mortality effects has not examined changes in specific cause mortality.

Since total mortality rates for children are fortunately low, deaths due to specific causes are particularly low frequency events. To better model the determinants of specific causes of death, we focus on Poisson regression estimates. Nevertheless, in our regression discontinuity framework that works best with a very large sample, our estimates still rely heavily on sometimes very noisy observations for the months immediately surrounding the September 1983 cutoff. For this reason, although we report the estimated

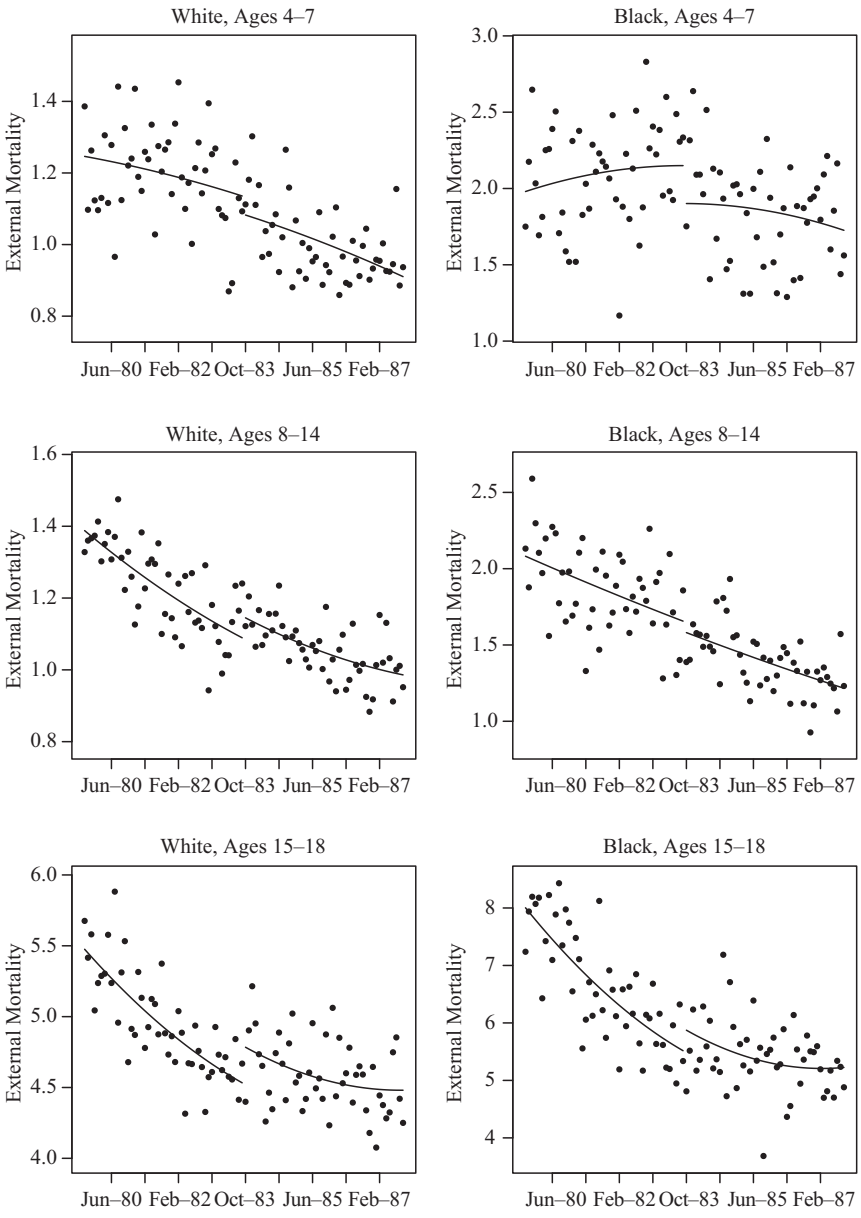

Figure 4

Child Mortality from External Causes by Child Race

Notes: Mortality rates by cohort (per 10,000 children) were constructed using Multiple Cause of Death Data files for the years 1979–2011 and Birth Data files for 1979–87. Points represent means of the age-specific mortality rates for each birth cohort, as described in the text. The lines are fitted values from a regression that includes a quadratic in birth cohort and a dummy variable for children born after September 30, 1983.

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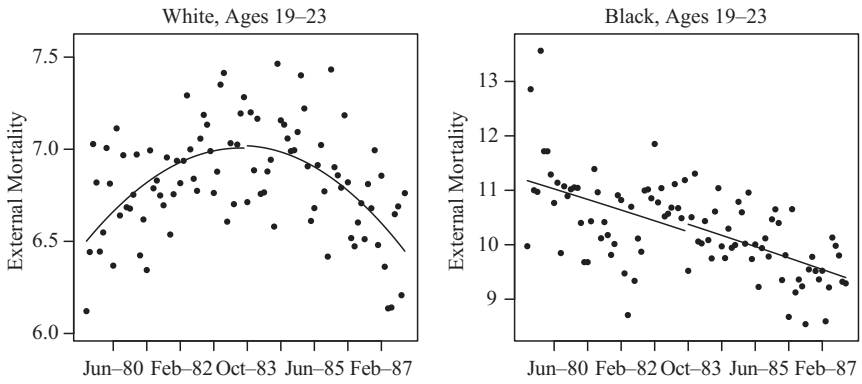

Figure 4 (*continued*)

marginal effects from this analysis in the Appendix, we do not emphasize the point estimates themselves in the discussion that follows.

We begin by examining changes in specific causes of internal mortality for black children aged 15–18. The results suggest that the largest contributors to the decline in internal mortality are decreases in deaths due to diseases of the nervous system and to neoplasms, although the estimates vary in size and significance across specifications and observation windows (estimates are reported in Table A10). We also see declines for black children spread across other causes, including infectious and parasitic diseases, mental and behavioral disorders, cardiovascular and circulatory system diseases, and respiratory system diseases. Although individually insignificant, these estimates in the aggregate support a large decline in the total number of internal-cause deaths occurring among older black children.

Next, we examine changes in specific causes of external mortality for white children at ages eight to 14 (estimates reported in Table A11). Again, the estimates in this analysis vary in size and significance across specifications and observation windows. But the evidence largely suggests an increase in deaths resulting from transportation-related accidents, accidental poisoning and exposure to noxious substances, and "other" non-transportation accidents. The top causes for this latter category at these ages include accidental mechanical suffocation, accidents caused by machinery, and accidents caused by electric currents. There is also evidence of an increase in deaths resulting from intentional self-harm or suicide.

B. Specification Checks

In the results reported above, the finding of a decrease in internal mortality among black cohorts gaining Medicaid at ages 15–18 holds up to a range of functional form specifications and window sizes. It also passes visual inspection. We also find persistent evidence, albeit to a lesser extent, of an increase in external mortality among white cohorts gaining Medicaid eligibility during the period of coverage gain.

To gauge how likely it would be to arrive at similar effect sizes by chance, we estimate jumps at nondiscontinuity points for these outcomes and age ranges. We run a series of

Table 4
Change in Annual External-Cause Mortality Rate for Children Born After September 30, 1983, by Race and Age Group

	Black Children			White Children		
	Four-Year Window	Three-Year Window	Two-Year Window	Four-Year Window	Three-Year Window	Two-Year Window
Ages 4–7						
Linear	–0.269*	–0.254	–0.257	–0.074	–0.010	0.022
	(0.136)	(0.169)	(0.193)	(0.045)	(0.047)	(0.058)
Linear spline	–0.275**	–0.267*	–0.276	–0.075	–0.010	0.022
	(0.122)	(0.137)	(0.167)	(0.045)	(0.048)	(0.058)
Quadratic	–0.269**	–0.254*	–0.257	–0.074	–0.010	0.022
	(0.128)	(0.141)	(0.165)	(0.045)	(0.047)	(0.058)
Quadratic spline	–0.226	–0.108	–0.191	0.086	0.088	0.075
	(0.160)	(0.192)	(0.244)	(0.065)	(0.072)	(0.084)
Baseline mean	2.093	2.094	2.147	1.196	1.197	1.174
N	192	144	96	192	144	96
Ages 8–14						
Linear	–0.048	–0.127	–0.098	0.107**	0.105**	0.123*
	(0.096)	(0.112)	(0.134)	(0.041)	(0.050)	(0.063)
Linear spline	–0.049	–0.130	–0.100	0.106***	0.103***	0.118**
	(0.094)	(0.111)	(0.136)	(0.031)	(0.038)	(0.046)
Quadratic	–0.046	–0.123	–0.095	0.110***	0.108***	0.128***
	(0.094)	(0.109)	(0.135)	(0.033)	(0.039)	(0.047)
Quadratic spline	–0.119	–0.054	–0.120	0.089*	0.071	0.032
	(0.148)	(0.157)	(0.164)	(0.046)	(0.054)	(0.056)
Baseline mean	1.864	1.792	1.785	1.227	1.183	1.146
N	288	216	144	288	216	144
Ages 15–18						
Linear	0.185	0.025	–0.072	0.162	0.035	0.006
	(0.423)	(0.453)	(0.488)	(0.203)	(0.206)	(0.170)
Linear spline	0.101	0.023	–0.039	0.118	0.033	0.022
	(0.247)	(0.294)	(0.390)	(0.095)	(0.110)	(0.121)
Quadratic	0.041	–0.042	–0.094	0.085	–0.002	–0.006
	(0.278)	(0.320)	(0.421)	(0.101)	(0.107)	(0.117)

(*continued*)

Table 4 (*continued*)

	Black Children			White Children		
	Four-Year Window	Three-Year Window	Two-Year Window	Four-Year Window	Three-Year Window	Two-Year Window
Quadratic spline	–0.062	–0.251	–0.312	0.055	0.053	0.030
	(0.307)	(0.349)	(0.449)	(0.135)	(0.160)	(0.145)
Baseline mean	6.613	6.249	5.951	4.953	4.812	4.693
N	192	144	96	192	144	96
Ages 19–23						
Linear	0.071	–0.294	–0.993***	0.018	–0.074	–0.115
	(0.260)	(0.275)	(0.331)	(0.120)	(0.121)	(0.139)
Linear spline	0.067	–0.312	–1.024***	0.006	–0.085	–0.121
	(0.256)	(0.239)	(0.302)	(0.098)	(0.111)	(0.139)
Quadratic	0.071	–0.294	–0.993***	0.018	–0.074	–0.115
	(0.260)	(0.242)	(0.296)	(0.094)	(0.107)	(0.139)
Quadratic spline	–0.854***	–1.305***	–1.134***	–0.163	–0.246*	–0.211
	(0.304)	(0.304)	(0.404)	(0.131)	(0.136)	(0.175)
Baseline mean	10.723	10.522	10.488	6.840	6.889	6.978
N	192	144	96	192	144	96

Notes: Coefficients from OLS regressions with the specified function in birth cohort, a dummy for deaths classified under ICD-10 and calendar month dummies. Standard errors reported in parentheses are robust and clustered by birth month cohort, *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

placebo tests for breaks occurring either prior to or following the September 30, 1983, cutoff. To examine jumps in external mortality for whites aged eight to 14, we estimate breaks at the 120 available cutoff points using a rolling four-year window of data among cohorts born either entirely before or after the cutoff.[25] Each regression includes a quadratic function in birth cohort, a dummy for deaths classified under ICD-10, and calendar month dummies. The top panel of Figure A5 presents the resulting RD estimates for all 120 simulations. Ten, or 8 percent, of the simulations lead to an estimate larger in absolute value than our estimate at the September 30, 1983, cutoff.

We run a similar series of simulations to estimate breaks in internal mortality at nondiscontinuity points for blacks at ages 15–18.[26] The bottom panel of Figure A5

25. We estimate breaks at each month of birth between January 1975–September 1979 and October 1987–December 1988. For this exercise, we are using mortality rates that are constructed by dividing the number of deaths by the unadjusted birth cohort size. We are unable to subtract out deaths prior to the age in question for cohorts born prior to 1979 since birth date information is only available in the mortality data files starting in this year.

26. The data available allows us to estimate breaks at each month of birth between January 1972–September 1979 and October 1987–December 1992.

presents the distribution of coefficient estimates. In a total of 108 simulations, zero (0 percent) of the simulations lead to an estimate larger in absolute value than the estimated decline in the internal mortality rate at September 30, 1983. This analysis strongly suggests that our estimated decline in black internal mortality is larger than estimates that we might observe due to chance. In contrast, it appears more likely that we might be picking up an estimate for whites that may not necessarily be linked to the change in Medicaid policy.

This agrees with findings from another check on our results indicating the absence of a robust relationship between Medicaid eligibility and white external mortality (included in the working paper Meyer and Wherry 2012). When calculating simple differences that compare the mortality of children born during the 12 months before and after the September 30, 1983, cutoff, we find no significant difference in the external mortality rates of white children born before and after the cutoff. In contrast, we find significant evidence of a decline in internal mortality for black children at ages 15–18. A difference-in-differences estimate that compares this change to that experienced by white children indicates a significant decline of 0.37 deaths per 10,000 black children.[27]

One potential concern with the RD design is the possibility of other changes affecting birth cohorts at the September 30, 1983, cutoff. If these changes affect the health of these cohorts, we may erroneously attribute differences in later-life mortality to the gain in Medicaid eligibility. To investigate this possibility, we test for breaks in cohort characteristics that existed prior to the change in Medicaid policy. Using natality data available for the birth cohorts in our sample, we construct and test measures of cohort size, the mean age of the mother, the shares of each cohort with a high school-educated mother and married mother, receipt of prenatal care at any time during the pregnancy, and receipt of prenatal care in the first trimester. We also test for differences in early life health by examining measures of the prevalence of preterm birth, low birth weight, and very low birth weight births for each cohort.

Figures A6 and A7 present plots for each of these cohort characteristics by child race. Regression analyses (reported in Tables A12 and A13) indicate some evidence of a decrease in cohort size, a decline in marriage among mothers, and a small increase in receipt of any prenatal care (one-fifth of a percentage point over a baseline of 97 percent) among black cohorts born after the September 30, 1983, cutoff. The size and statistical significance of the covariates, however, vary across specifications and window sizes. Among white cohorts, we find stronger evidence of an increase in the receipt of any prenatal care for cohorts born after the cutoff, although again the change is small in size (one-tenth of a percentage point over a base of 99 percent). Importantly, for both race groups, we find no evidence of differences in infant health at the September 30, 1983, cutoff.

Given the sensitivity of the estimates that are significant, we do not view the results of these tests as a concern for the interpretation of our findings. The evidence of a change in birth cohort size at the cutoff for blacks is not particularly robust and is accounted for in

27. Results may be found in Table 8 in Meyer and Wherry (2012). While providing a simple plausibility check on our RD results, this analysis relies on several strong assumptions. The differences estimates assume that there are no trends in mortality rates, while the difference-in-differences assume that any underlying trends in mortality are similar across race groups. In addition, the difference-in-differences estimate should be interpreted as the relative difference in the effect of the expansions for black children compared to white children, acknowledging that white children born after the cutoff also benefitted from the Medicaid expansions.

our construction of mortality rates. And without the concern of the potential manipulation of birth dates, continuity of the density of births at the cutoff is not necessary for the RD design (Imbens and Lemeiux 2008). The observed changes in mother's marital status and prenatal care receipt are small and, if they were to represent actual discontinuities, are unlikely to account for changes in later-life health of the magnitude we find in the mortality data. When we include these variables in our RD specification, the estimates of the decline in internal mortality for blacks are robust in their magnitude, although we do lose some precision (Table A14). Finally, placebo simulations that test for breaks in these characteristics at nondiscontinuity points indicate that it is relatively common to observe jumps of a similar magnitude at other cutoffs.[28]

C. Additional Analyses

In addition to the analyses already presented, we also explored whether changes in mortality differed among children residing in states with more or less generous expansions in Medicaid eligibility. Across states, there is variation in the average gain in years of public health insurance eligibility at the September 30, 1983, cutoff due to differences in Medicaid policies before and after the expansions.[29] We should emphasize that this source of variation in Medicaid eligibility across states is not as clearly exogenous as birth date. States with improving child mortality may be less likely to expand coverage, for example. However, this type of variation is the main source of identifying variation that has been used in the literature on Medicaid expansions.

To estimate the eligibility gain for each state, we follow the literature and use a simulated eligibility measure constructed using the national sample of children. This captures variation in average eligibility gains by state resulting from legislative rather than sociodemographic differences. Ranking the state estimates, we divide the states into two groups. The "high-eligibility" group includes the states with the largest average gain in years of eligibility, while the "low-eligibility" group includes the states with the smallest average gain in years of eligibility.[30] We repeat the analysis for each state group using state of residence information available in the mortality records.[31]

28. See Appendix C for additional details on this analysis and the placebo simulations.

29. States' eligibility criteria in place prior to the expansions depended on state-determined AFDC program rules and optional eligibility programs. In addition, state decisions regarding optional Medicaid and CHIP expansions varied in terms of timing and child age and family incomes covered.

30. We use this method to identify the high- and low-eligibility gain states for children of each race in addition to the identification for all children. Table A15 reports the state groups for all children and for each race group. Table A16 presents summary characteristics for children in each of the state groups in 1983 prior to the first Medicaid eligibility expansions. The table suggests that more children stood to gain from the Medicaid eligibility expansions in the high eligibility gain states due to lower preexpansion eligibility levels for Medicaid.

31. To construct denominators in this analysis, we rely on birth cohort size by state. The mobility of children and their families between state groups before, during, or after the period of eligibility gain may influence our estimates. When we conduct the analysis using the logged number of deaths rather than rates of death, we have similar findings. Mobility is still a concern, however, if children move across state groups from the period of coverage to later ages. Encouragingly, during this time period children were less likely to move to a different state at ages ten to 17 than at earlier ages (U.S. Census Bureau 2001). The estimates indicate that less than 3 percent of children of these ages move to a different state per year. In addition, a sizeable share of these movers would presumably relocate to states that are members of the same state group.

We do not find major differences in the mortality effects across state groups.[32] In particular, we find no evidence that children residing in the high-eligibility gain states saw greater improvements in health. When we examine the results for each state group by child race, we find evidence of a decline in internal mortality for older black children in both groups. However, if anything, our results are indicative of a slightly larger improvement in black mortality in the low-eligibility gain states.

We find a similarly counterintuitive finding in a subsequent analysis that divides deaths into counties with high and low child poverty rates during the period of expansion.[33] We find evidence of a significant decrease in internal mortality at ages 15–18 among all children in counties with lower poverty rates and no similar improvement in counties with high poverty rates. When we examine changes in mortality in each group by child race, we find evidence of declines in internal mortality among blacks in both county groups. However, our coefficient estimates suggest a larger improvement in black mortality in the low-poverty counties.

One potential explanation for not finding stronger improvements in health in the expected locations in these analyses is that larger expansions in eligibility may not directly translate into higher levels of enrollment or improvements in access to medical care for children. Increases in enrollment require active outreach by states and localities and the dissemination of eligibility information to parents. In addition, larger numbers of new beneficiaries may strain limited supply-side resources, particularly if physicians opt not to accept Medicaid clients due to low reimbursement rates or slow payment.

V. Interpreting the Estimates: Plausibility, Comparisons to Past Work and Cost Per Life Saved

In this paper, we find evidence of a sizeable decrease in the internal mortality rate of older black teens as a result of increased childhood eligibility for Medicaid. Our point estimate indicates a 19 percent decrease at ages 15–18. We find no evidence of a similar effect among white children, although the smallest of our implied 95 percent confidence intervals allows us only to rule out a decline greater than 4.5 percent for white teens. This finding is consistent with previous work examining the Medicaid expansions of this period. In their working paper, Currie and Gruber (1995) estimate that an increase in the fraction of children eligible for Medicaid at the state level had a much larger impact (more than four times as big) on mortality for blacks than for whites, although neither estimate is significantly different from zero. The authors also

32. Results for all races may be found in Figures A9 and A10 and Tables A17 and A18. Results for each state group by child race are available from the authors.

33. We classify counties into two groups using the population-weighted median of 1989 poverty rates by county for children aged zero to 17 from the Census Small Area Income and Poverty Estimates as a cutoff. Using information on county of residence in the mortality data, we then total deaths in the "high-poverty" and "low-poverty" counties and rerun the analysis for each group using the log number of deaths as the dependent variable. The results for all races are reported in Tables A19 and A20 and Figures A11 and A12. Results for each county group by child race are available from the authors.

provide suggestive evidence that newly covered black children under the Medicaid expansions used more healthcare than their white counterparts. As noted below, our overall effect of the eligibility expansions on mortality is smaller than found in Currie and Gruber (1996a).

The size of our point estimate for black children is large but more reasonable when we consider that deaths among black children are highly concentrated among those who would gain coverage under the expansions. Using information on deaths for children aged 15–18 prior to the expansions in the 1993 National Mortality Followback Study, we estimate that 64 percent of the deaths occurring among black children at these ages were to decedents with household incomes below the poverty line.[34] Combining this information with our measured 19 percent decline in internal mortality among blacks under the expansions, this implies a 30 percent (19 divided by 0.64) decline in the internal mortality rate for poor black children.[35] The tightest of the confidence intervals on our estimates for black teens indicate that the eligibility effect is at least a 9 percent decline in internal mortality with 95 percent probability (or a decline of 0.20 deaths per 10,000). This lower-bound estimate would imply a 12.5 percent decline for poor black children.

This estimate is large but not implausible given the large increases in eligibility for poor black children. Our first-stage estimates indicate that 32 percent of poor black children at the birth date cutoff gained an average of 5.2 years of Medicaid eligibility under the expansions (not shown in Table 1). In reality, it is more likely that a larger share of children gained eligibility but for shorter intervals of time given fluctuations in income and known "churn" in Medicaid enrollment for children (see Fairbrother et al. 2007). And, while existing evidence indicates low takeup rates of Medicaid coverage (for instance, 8 percent as estimated by Card and Shore-Sheppard 2004), a new study of Medicaid enrollment under these expansions by race indicates that takeup for black children was higher than average at approximately 30 percent (Wherry et al. 2015). The authors find no evidence of a change in Medicaid coverage for nonblack children. For the children who did enroll in Medicaid, sustained increases in insurance coverage could result in cumulative effects that are larger in magnitude than previously measured contemporaneous effects.

Finally, it is reasonable to expect that children who do take up coverage are those most in need of medical care (specifically, the conditional-coverage phenomenon; see Marton and Yelowitz 2014).[36] Our analysis of specific causes indicates that the deaths that were avoided were most likely related to serious and chronic childhood disease, which would support this hypothesis. It is worth noting also that even if the

34. Approximately 40 percent of black children aged 15–18 lived in poverty in 1993 (authors' calculation from the March supplement to the 1994 Current Population Survey), indicating that the baseline death rate for poor black children was 0.64/0.4 = 1.6 times higher than for the general black population.

35. This calculation assumes that the change in deaths estimated under the policy occurred among poor children only. It also assumes that the share of internal-cause deaths for black children aged 15–18 with incomes under poverty is similar to the share calculated for all deaths in the National Mortality Followback Study.

36. In their study of takeup and the utilization of health care under OBRA90, Card and Shore-Sheppard (2004) find evidence that supports this hypothesis. They estimate that children with new Medicaid coverage are 60 percent more likely to have an annual doctor visit than those without coverage.

typical newly eligible child is not enrolled in Medicaid, upon commencing any costly hospital or physician care, it is likely that an eligible child would be enrolled or receive retroactive benefits.[37]

The decrease in later-life mortality for black children is supported by recent work documenting a sizeable decline in adult hospitalizations and emergency department (ED) visits among the cohorts gaining childhood Medicaid eligibility. Using a similar regression discontinuity model, the point estimates from Wherry et al. (2015) indicate that black children born after the September 30, 1983, cutoff experienced between 8 and 13 percent fewer hospitalizations and 3–4 percent fewer ED visits at age 25 relative to those born just before the cutoff. The effects of child Medicaid eligibility are even more pronounced for hospital and ED visits related to chronic health conditions, which are plausibly influenced by access to care in childhood.

Surprisingly, we also find some evidence of an increase in external mortality for white children at the cutoff at ages eight to 14. The point estimates from the regressions suggest between a 7 and 9 percent increase in external mortality. As discussed earlier, the evidence for this finding is less convincing than the decrease we observe in internal mortality for blacks. Sensitivity analyses indicate that there is a reasonable probability that an estimate of this magnitude may be found due to chance. Combined with less-than-persuasive graphical evidence, we are wary of interpreting it as an effect of the Medicaid expansions. That said, the change occurs during the period of eligibility gain and possible explanations could include moral hazard or income effects that lead to increases in risky behaviors (see the discussion in Cutler and Zeckhauser 2000 and Cawley and Ruhm 2011, respectively), reductions in healthcare quality under the crowdout of private coverage, or the consequence of strained supply-side resources under expansions in overall levels of coverage.

A. Comparisons to Previous Work

The results presented here diverge from the literature in two important ways. First, we are unable to detect significant evidence of a contemporaneous decline in child mortality associated with expanded childhood eligibility for public health insurance. We did, however, find persuasive evidence of longer-term mortality effects among black children during the period just following the gain in public coverage. This is the first study documenting the potential longer-term mortality effects of expansions in public health insurance among children.[38]

Second, our estimate for the mortality effect of public eligibility is smaller than the estimate found by Currie and Gruber (1996a). We use some rough calculations to demonstrate this point. In our four-year window analysis, we estimate a decline in the annual internal mortality rate of older black children aged 15–18 of about 0.44 deaths per 10,000 children. With black children representing approximately 15.6 percent of all

37. Eligibility could be granted retroactively for up to three months prior to the date of application. In addition, many states were giving children the opportunity to apply for Medicaid at the sites where they received health care (Congressional Research Service 1993).

38. A new working paper by Brown, Kowalski, and Lurie (2015) examines the impact of Medicaid eligibility during childhood on adult mortality and economic outcomes, using IRS data. The authors find evidence of mortality decreases for adults in their late twenties; they do not study mortality during the adolescent years.

children and our calculation of 0.42 years of eligibility gain on average for all children, our estimates imply an overall decline of $(0.44 \times 4 \times 0.156) \div 0.42 = 0.654$ deaths per 10,000 children of all races gaining one year of eligibility.[39] This mortality derivative with respect to eligibility ignores any potential adverse effect for white children and yet remains about half the size of that found by Currie and Gruber. They found that 1.277 deaths in a year were avoided for every 10,000 children aged one to 14 of all races made eligible for that year.[40]

B. Cost Per Life Saved

To further interpret our results, we estimate the approximate cost to the Medicaid program of each child life saved.[41] By its nature, this calculation ignores any effects of the expansions on morbidity or quality of life. To calculate the cost of provision of Medicaid services for every 10,000 children, we again use the estimate of an 8 percent takeup rate under the expansions. With the average gain in eligibility of 0.42 years among all children, this rate implies an increase in Medicaid coverage of $0.42 \times 0.08 = 0.034$ years per child. Using an estimate of the cost of Medicaid coverage per child during this time period, this implies the Medicaid expansion cost $\$1{,}430 \times 0.034 \times 10{,}000 = \$486{,}200$ (in 2011 dollars) for every 10,000 children.[42] With our estimate that $0.44 \times 4 \times 0.156 = 0.275$ deaths per 10,000 children were prevented by the expansions, we calculate that it cost $\$486{,}200 \div 0.275 = \$1.77$ million (in 2011 dollars) to save one life.[43]

Although these calculations are similar to those used in the existing literature, this estimate may understate costs since it relies on the average cost of Medicaid coverage rather than the marginal cost for newly enrolled children. Using data from the Medicaid Statistical Information System, Brown, Kowalski, and Lurie (2015) estimate that each year of additional Medicaid eligibility under the expansions increased Medicaid spending by $575 in 2011 dollars. This would imply an increase in Medicaid spending of $2.42 million per 10,000 children made eligible and therefore a cost per life saved of $8.8 million. Although this indicates a smaller return, when combined with other potential long-term benefits of public health insurance including improvements in economic outcomes (Brown, Kowalski, and Lurie 2015; Cohodes et al. ~~2014~~) and reductions in healthcare utilization (Miller and Wherry ~~2014~~, Wherry et al. 2015), there may be significant offsets to the cost of these expansions.

39. In 2000, 15.6 percent of all children in the United States were black. We consider only the reduction to black mortality observed at ages 15–18 in our calculation and assume that the expansions had no effect on mortality at other ages or for other race groups. We multiply 0.44 by four for the four years between ages 15–18.
40. While the literature does not provide a comparable estimate, when we consider a takeup rate among newly eligible children of 8 percent, as estimated by Card and Shore-Sheppard (2004), our estimates imply a decrease of 8.175 deaths per year for every 10,000 children newly enrolled in Medicaid under the expansions.
41. The back-of-the-envelope calculations in this section implicitly assume a social discount rate of zero. A higher rate of social discount would imply a somewhat higher cost per life saved.
42. The per capita Medicaid payment for categorically needy AFDC children in FY 1992 was $891 (U.S. House of Representatives 1996). This is $1,430 in 2011 dollars.
43. Despite the differences in our estimated mortality effects, this estimate is lower than the $1.61 million (1992 dollars) cost per child life saved ($2.68 in 2011 dollars) calculated by Currie and Gruber (1996a). This difference is due mostly to the lower estimate of the takeup of public health insurance in our context.

VI. Conclusions

The effectiveness of public health insurance in improving the health of children is an important policy question. Despite drastic expansions in public health insurance over the last 30 years, the evidence connecting this expanded coverage to improved child health remains hard to come by. Identifying the impact of health insurance on health is a well-known problem since individuals with insurance differ from those without and since health itself may affect coverage (Levy and Meltzer 2008). While randomly assigning health insurance overcomes this problem, there have been few opportunities for experimental study. Only one experimental study has compared individuals with and without health insurance coverage, and its focus was on low-income adults not children.

This paper uses a novel quasi-experimental design to provide new evidence on the health effects of public health insurance for children. While not an experimental study, our research design approximates random assignment in public health insurance for children by exploiting changes in Medicaid eligibility rules that differentially affected those born before and after September 30, 1983. Early expansions under Medicaid in the 1980s and 1990s extended eligibility only to children born after this date. We show this led to a large, cumulative difference in public health insurance eligibility for children born on either side of the September 30, 1983, cutoff. Children in families with incomes at or just below the poverty line gained close to five additional years of eligibility if they were born in October 1983 rather than just one month before.

An additional advantage of the research design is that variation in childhood eligibility is tied to birth date, which provides an easy way to track individuals over time to study the longer-term effects of insurance coverage. In general, very little is known about the long-term effects of health insurance coverage on health (Cutler and Zeckhauser 2000). It is possible that the payoffs from public health insurance coverage, particularly for children, might not be fully evident until later in life. Without considering the long-term effects of insurance coverage, we may be missing an important component of program benefits.

Using a regression discontinuity design, we examine the impact of public health insurance eligibility during childhood on immediate and longer-term mortality by comparing outcomes for cohorts of children born just before and after the September 30, 1983, cutoff. The discontinuity in public health insurance eligibility under the expansions occurred between the ages of eight and 14. We measure mortality outcomes for this period, as well as through age 23, to gauge any longer-term impacts. We examine differential effects of the expansions by the race of the child because black children were more likely to be affected by the Medicaid expansions. They gained more than twice the years of eligibility on average as white children at the September 30, 1983, birth date cutoff.

The results provide compelling evidence of an improvement in mortality for black children under the Medicaid expansions. Although there is some suggestion of a decline in mortality rates during the period of coverage (ages eight to 14), we find strong support for a sizeable decline in the later-life internal-cause mortality of black children aged 15–18. With deaths due to internal causes likely to be influenced by access to medical care, this result supports an improvement in the underlying health of black children under the

eligibility expansions. Furthermore, we find evidence that this gain in health is not reversed during the early adult years.

We find no evidence of a similar decline in the mortality of white children under the expansions. Surprisingly, we find some evidence of an increase in external mortality for white children during the period of coverage gain. We mention several potential explanations for this result but also provide evidence that keeps us from drawing any definitive conclusion. Future work may be able to resolve whether there are adverse effects of public expansions for certain population groups.

This paper provides important new evidence of longer-term mortality improvements among black children under public health insurance. There are several limitations to our analysis and to the generalizability of our findings. First, as is the case with all quasi-experimental designs, the identification of program effects is susceptible to confounders that, in this case, would differentially affect cohorts born on either side of the September 30, 1983, birth date cutoff. However, we know of no other social policy or economic influences during this time period that would have differentially affected cohorts born before and after this date. Second, our estimates rely on variation in Medicaid coverage for children in the 1990s, which may be limited in generalizability to contemporary changes in Medicaid coverage or for different populations gaining coverage. Third, although we investigate changes in the underlying causes of death, we are limited in our ability to shed more light on the specific mechanisms behind the observed changes in mortality. Finally, our analysis is limited by the amount of time that has passed following the policy change. With time (and further study), we will be able to look at longer-term adult outcomes for the cohorts of children affected by the change in Medicaid policy.

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