

**Three Essays in Health Policy Evaluation**

**by**

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“This is the work I really love because that’s what an excavator does.”  
-Twenty Trucks

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## INTRODUCTION

*“If an error was made, it was not in passing Medicare but in adding Medicaid as an afterthought.”*

*-Ginzberg and Solow (1974)*

*“Assessing ten years of experience with federal health programs is difficult...The existing evidence, however, gives ample reason for optimism.”*

*-Davis and Schoen (1978)*

The most expensive, ambitious and widespread health reforms in U.S. history came in the mid-1960s. Lyndon Johnson’s new administration mounted an “unconditional war on poverty” and viewed the elimination of health care disparities as a central part of this effort. The subsequent flurry of legislation passed by the 89th congress, established a large new public health insurance program for low-income families, Medicaid, as well as a range of programs to provide care to poor families directly, such as Community Health Centers (CHCs).

These programs still matter. Medicaid has over 60 million beneficiaries in 2011 and covered more than a third of children. It is the second most expensive item in nearly all state budgets and the third most expensive federal program (behind Social Security and Medicare). More than 1,100 Federally Qualified Health Centers, the outgrowth of CHCs, serve over 20 million people almost three quarters of whom are poor (**National Association of Community Health Centers 2013**). Both programs have also expanded under the Affordable Care Act. Since the insurance marketplaces opened in October 2013, Medicaid enrollment has increased by 4.8 million (**Wachino, Artiga, and Rudowitz 2014**), and many of these patients are expected to be seen in health centers.



Despite the size and importance of these long-standing federal health programs, there are gaps in our knowledge of whether and how they work to equalize health care use and improve health outcomes. For example, nearly all research on the effect of Medicaid on health relies on expansions of the program in the 1980s to slightly higher-income groups (cf. Currie and Gruber 1996b). This work finds that Medicaid's effects are much larger for poorer recipients, but since more recent eligibility expansions have primarily covered non-poor families, these effects may not reflect the benefits of Medicaid's initial expansion to poor children on welfare. Research on health centers tends to use proprietary data from a single center, city or state (cf. **Bellin, Geiger, and Gibson 1969**), which limits causal inference and the applicability of the results to the much broader, national program. This dissertation provides new evidence on the effects of these health safety net programs when they were first introduced under the War on Poverty.

The first chapter examines the effect of the introduction of Medicaid between 1966 and 1970 on infant and child mortality rates. I exploit the federal requirement that Medicaid cover all cash welfare recipients, which meant that Medicaid eligibility inherited large cross-state differences in welfare receipt that had emerged decades before. I use a difference-in-differences model that compares state-level infant and child mortality rates before and after Medicaid (first difference) in states with higher and lower initial welfare-based eligibility (second difference). The results show that mortality rates in higher- and lower-eligibility states were indistinguishable prior to Medicaid, but immediately after states adopted Medicaid programs, nonwhite mortality rates fell by eight percent in high-eligibility states relative to low-eligibility states. Using newly-entered administrative data from 1963-1976, I show that children's public insurance use increased by about six percentage points in the high-eligibility states relative to low-eligibility

states. Medicaid can account for at eight percent of the aggregate decline in nonwhite child mortality from 1965 to 1979.

The second chapter examines the effect of Medicaid implementation on income-based disparities in children's insurance coverage, health care use and medical spending. I document strong income disparities in health care use in the early 1960s and show that these disparities fell dramatically in the period after Medicaid implementation. I also use the 1963 and 1970 waves of the Survey of Health Services Utilization and Expenditure with specially obtained geographic identifiers to show that after Medicaid, income disparities in insurance coverage and primary care use fell disproportionately in areas with higher pre-existing rates of welfare-based Medicaid eligibility. These results suggest that Medicaid implementation made progress towards its goal of making "medical care of high quality readily available to those unable to pay for it," and they provide additional evidence on the mechanisms by which the mortality effects, documented in chapter 1, were achieved.

The third chapter, written with Martha Bailey, estimates the effect of the Community Health Center (CHC) program on older adult mortality rates. CHCs were initially established between 1965 and 1974 and provided (rather than financed) primary care. We use data from the National Archives to construct measures of this county-level roll out. Our estimates show that mortality for residents 50 and older fell sharply by two percent after CHC establishment, and that the effects persist for at least 15 years. This paper is among the first nationally representative evaluations of the CHC program.

This dissertation makes two types of contributions. First, evidence on health safety net programs specifically in the 1960s is relevant to the often virulent claims and firmly held opinions about Johnson's Great Society. Only recently have researchers compiled the necessary

data to evaluate the introduction of these programs rigorously. The three chapters outlined above suggest that Medicaid and CHCs generated significant and heretofore unknown benefits. Second, the introduction of these programs provides a unique opportunity to estimate their effects in a more general sense. More and more people have used of Medicaid and CHCs over time, but much of this growth was the result of individual choices or circumstances and, therefore, may not help identify these program's effects separately from other forces that determine their use. I argue that the introduction of Medicaid and CHCs (or aspects of their introduction) do provide unique quasi-experimental variation in poor families' exposure to health safety net programs and can, therefore, contribute new evidence on whether and how these programs work. Thus, the effects of Medicaid and CHCs in the 1960s are of interest in their own right, and they provide valuable new evidence on the extent to which these programs work in general.

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## **Chapter 1. PUBLIC INSURANCE AND MORTALITY: EVIDENCE FROM MEDICAID IMPLEMENTATION**

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

While Medicaid’s costs are large and controversial, its benefits in terms of health have been harder to quantify. Quasi-experimental research finds that legislative expansions of Medicaid eligibility led to large reductions in mortality for infants, children, teens and adults (**Currie and Gruber 1996a, b, Meyer and Wherry 2013, Sommers, Baicker, and Epstein 2012**). The corresponding increases in any insurance coverage are relatively small, however (see **Card and Shore-Sheppard 2004**), leaving considerable uncertainty about the mechanism for these effects.<sup>1</sup> Adding to this uncertainty is that the Oregon Health Insurance Experiment

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<sup>1</sup> Explanations for such large magnitudes include underreporting of Medicaid coverage (Card, Hildreth, and Shore-Sheppard 2004), additional health effects from increased disposable income (Leininger, Levy, and Schanzenbach 2012), investments due to increased provider revenue (Finkelstein 2007), or other omitted variables (Dave et al. 2008).

(OHIE)—the highest-quality study of Medicaid’s effect on health—finds no evidence of improvements in one-year mortality (**Finkelstein et al. 2012**) or clinical health measures (**Baicker et al. 2013**) for adults. The absence of significant results in the OHIE, however, may reflect its short time horizon, the characteristics of its sample, or its statistical power. Thus, for a variety of reasons, decades of research on Medicaid has provided limited evidence on its health effects.

This paper uses the introduction of Medicaid between 1966 and 1970 and the federal requirement that states cover all cash welfare recipients (the “categorically eligible”) to provide new estimates of Medicaid’s effects on the health of the poor. The statutory link between welfare receipt and Medicaid eligibility motivates two aspects of my analysis. First, it generated wide variation across states in welfare-based eligibility due to long-standing, institutional differences. Second, nonwhite children, for whom welfare receipt was relatively common, were six times as likely to be eligible for Medicaid under the categorical eligibility provision as white children (18 percent versus 3 percent), and four times as likely as nonwhite adults (4.5 percent). This suggests that Medicaid implementation should have had heterogeneous state-level health effects that were largest for nonwhite children in states with higher initial eligibility.

To estimate Medicaid’s effect, I use 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). I construct state-level mortality rates by age, race, and cause of death from 1959 to 1979, which facilitates an event-study analysis of Medicaid’s longer-run effects up to nine years after implementation. This empirical strategy, based on “dose-response” type comparisons across states with different eligibility

levels, obviates the need for comparisons between states that implemented Medicaid earlier and later, which differed in their pre-Medicaid mortality trends.

The results show that, after Medicaid's introduction, high-Medicaid-eligibility states experienced dramatic decreases in the mortality rates of nonwhite children (-12 percent) and nonwhite neonates (-8 percent) relative to low-eligibility states. The effects persist for nine years and are not present for white children, who were eligible for and used Medicaid much less often than nonwhite children. The child mortality results are driven by reductions in "internal" causes for which there were effective treatments in the 1960s and 1970s, and the neonatal mortality results reflect reductions in premature births and increases in hospital births rather than increases in birth weight. I use newly-entered data on public health insurance programs from 1963 to 1976 to verify that high-eligibility states also had relative increases in children's public health insurance use, the primary mechanism for the mortality effects. The estimates imply that Medicaid reduces the mortality of children who use it by up to 40 percent.

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

welfare programs, which suggests that they are not due to differences in other public efforts to improve the health of the poor.

The results imply that Medicaid was very effective in achieving one of its primary goals: “prevent[ing]...premature death” (**Department of Health, Education and Welfare 1967a**). The implied effects on treated infants and children are smaller than estimates from the eligibility expansions in the 1980s (**Currie and Gruber 1996a, b**), despite applying to more disadvantaged families, yet they still suggest that Medicaid played an important role in national mortality changes. I estimate that Medicaid implementation reduced aggregate nonwhite child mortality rates by 8 percent, and can account for 15 percent of the decline in the white-nonwhite mortality rate gap between 1966 and 1979.

These results are also the first to establish that the introduction of Medicaid reduced mortality. Some authors have argued that welfare families who gained eligibility in the 1960s did not benefit from Medicaid because they already received charity care (**Matusow 1984, Klarman 1963**) or existing public health benefits (**Olendzki 1974, Roghmann, Haggerty, and Lorenz 1971**). My results challenge these claims and show that the expansion of public insurance for poor children over and above any pre-Medicaid charity/public arrangements had important health benefits immediately and in the longer-term. Because mortality is an extreme outcome, the broader health benefits of Medicaid are likely much larger. These findings imply that proposals to eliminate Medicaid, allow states to opt out, or cap federal reimbursements (**Grannemann and Pauly 1983, Smith and Haislmeier 2009**) could hurt the health of poor children even if their care is taken up by private charity to the degree that it was in the 1960s. The estimates also inform the growing literature on health and the Great Society, which has not

considered the role of Medicaid, by far the largest contemporaneous program to target the health of the poor.<sup>2</sup>

### **1.1 What Do We Know About Medicaid and Health?**

The goal of the Medicaid program is to “promote a healthy population” through the “application of medical knowledge and the use of all health resources” (DHEW 1967a). The most obvious and plausible way that health insurance can have a causal effect on health is through the consumption of medical care (Levy and Meltzer 2004). This requires that households who qualify for Medicaid decide to take up coverage and use medical services, and that those services actually improve health relative to the care recipients would otherwise obtain (Gruber 1997).

A large literature in economics examines Medicaid’s effects on insurance coverage and the use of medical care, but only a handful of studies estimate its effect on health. The difficulty of evaluating its health effects arises because Medicaid provides health insurance mainly to lower-income families, so in terms of health its recipients are negatively selected both because their incomes are low and because of adverse selection in Medicaid take-up. Thus, comparing health outcomes between recipients and non-recipients confounds the program’s effect with underlying differences in factors that determine eligibility or insurance demand.

To overcome both challenges, quasi-experimental research relies on legislative expansions of Medicaid eligibility that are plausibly unrelated to other determinants of health. These studies consistently demonstrate that Medicaid eligibility expansions reduced mortality (the most common health outcome used in this literature), but they fail to find large effects on

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<sup>2</sup> Negative effects on mortality have been estimated for health programs such as hospital desegregation (Almond, Chay, and Greenstone 2006), Medicare (Chay, Kim, and Swaminathan 2012), and Community Health Centers (Bailey and Goodman-Bacon 2013), and non-health programs that serve poor families such as Head Start (Ludwig and Miller 2007) and Food Stamps (Almond, Hoynes, and Schanzenbach 2011).



insurance coverage. Currie and Gruber (1996a, 1996b) find that a series of legislative increases in eligibility to pregnant women and to children during the 1980s reduced infant mortality rates by eight percent and child mortality rates by five percent.<sup>3,4</sup> The estimated effect of these expansions on insurance coverage, however, range from zero (**Cutler and Gruber 1996**) to about three percentage points (**Dave et al. 2008**) for pregnant women, and from a slight reduction (**Yazici and Kaestner 2000**) to an increase of between 2.4 and four percentage points for children (**Cutler and Gruber 1996, Shore-Sheppard 2009**). Thus, assuming that Medicaid expansions only affect health through changes in insurance coverage, dividing the mortality reductions by the increase in insurance implies that Medicaid coverage reduces mortality by more than 100 percent—an impossible result.

Different quasi-experimental research designs and populations produce similar conclusions. **Meyer and Wherry (2013)** use a regression discontinuity (RD) estimator based on a provision in one of the 1980s reforms that granted eligibility to certain children born after September 30, 1983. They find that annual mortality rates among black children born just after the cutoff fell by about seven percent at ages 8 to 14, and annual internal-cause mortality rates fell by 11 percent at ages 15 to 18, suggesting that mortality effects vary over time.<sup>5</sup> **Card and Shore-Sheppard (2004)**, however, use the same discontinuity and find that contemporaneous insurance coverage increased by only 10 percentage points. For adults, **Sommers, Baicker, and**

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<sup>3</sup> The proportional effects are calculated by multiplying the estimated effect of eligibility on the mortality rate by the total change in eligibility in the sample (30 percentage points for pregnant women and 15 percentage points for children), and dividing by the pre-expansion mortality rates. See appendix 4 for details on the calculation, including a justification for mortality effects expressed as percentages and insurance effects reported in percentage *points*.

<sup>4</sup> In contrast, studies that examine the same eligibility expansions in individual states tend to find much smaller effects on infant and child mortality (Long and Marquis 1998, Piper, Ray, and Griffin 1990), although most of them suffer from methodological limitations related to poorly defined control groups (Levy and Meltzer 2004).

<sup>5</sup> De La Mata (2012) estimates RD models based on Medicaid's income cutoffs and finds no effect of Medicaid eligibility on self-reported health or school days missed among children up to five years after her initial observation of eligibility.

**Epstein (2012)** find that recent expansions of eligibility in three states reduced mortality by six percent, but increased insurance coverage by only three percentage points.

There are several mechanical reasons why estimates of Medicaid's effect on health could be large relative to its effect on insurance. First, the proportional mortality reductions will be overstated to the extent that poorer families who actually take up Medicaid have higher baseline mortality rates. Second, take-up estimates based on survey data understate Medicaid coverage (**Card, Hildreth, and Shore-Sheppard 2004, Davern, Klerman, and Ziegenfussi 2007**).<sup>6</sup> Appendix D uses auxiliary data on mortality rates by income and underreporting of Medicaid to account for these factors, but the implied effects of Medicaid on mortality are still larger than on insurance.<sup>7</sup>

The magnitudes of these estimated mortality effects relative to those on insurance coverage suggest that Medicaid expansions in the 1980s may have improved health through channels other than increased insurance coverage. For example, families that dropped private coverage may have gained disposable income from savings on premiums, out-of-pocket expenditures, and wage offsets, which could confer health benefits (**Leininger, Levy, and Schanzenbach 2012**). Alternatively, expansions may have lead providers to increase capacity or invest in new technologies, which could prevent deaths among those not actually covered by Medicaid (**Finkelstein 2007, Pauly and Pagán 2007**). Finally, **Dave et al. (2008)** argue that estimates based on the state-by-year variation in the expansions are biased by omitted variables, although this cannot explain the large RD estimates. Thus, quasi-experimental evidence suggests that Medicaid expansions have reduced mortality, but their magnitudes preclude an interpretation of these estimates as the effect of Medicaid coverage per se.

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<sup>6</sup> Dave et al. (2008) circumvent this problem by using administrative data on the payment source for hospital births. Consequently, they find slightly larger effects on total insurance coverage.

<sup>7</sup> Appendices 2 through 4 available at: [http://www-personal.umich.edu/~ajgb/medicaid\\_appendix\\_ajgb.pdf](http://www-personal.umich.edu/~ajgb/medicaid_appendix_ajgb.pdf).

Additional uncertainty about Medicaid’s effect on health comes from the Oregon Health Insurance Experiment (OHIE), a randomized expansion of adult eligibility in 2008. Results from the first year of post-randomization data show no effects on adult mortality rates (**Finkelstein et al. 2012**) or on a range of clinically-measured outcomes such as blood pressure or cholesterol (**Baicker et al. 2013**). While mortality is fairly low, even for poor adults, the OHIE has the power to detect the mortality estimates documented in **Sommers, Baicker, and Epstein (2012)**.<sup>8</sup> More years of data may ultimately reconcile these results, but the stark disagreement across research designs in the short-run mortality estimates casts some doubt on the interpretation of quasi-experimental results for all age groups.

The empirical evidence on the effect of Medicaid coverage on health leaves open many important questions. Growth of eligibility among groups on the “fringes of Medicaid” (**Levy and Meltzer 2004**) appears to have played a large role in reducing mortality for infants, children, and adults, but these changes are so large that they cannot be entirely attributed to Medicaid’s health insurance coverage. Furthermore, the RD evidence suggests that the effects can differ in important ways between the short- and longer-run, making it unclear how to interpret the short-run experimental evidence that shows no mortality benefits for adults. Therefore, whether and by how much Medicaid improves health in the shorter- or longer-term remains uncertain.

This paper contributes new quasi-experimental estimates of Medicaid’s effects on mortality, and the first estimates for its main recipients: infants and children on welfare. The program’s original introduction (1966-1970) was the largest ever change to means tested public

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<sup>8</sup> The implied effects of Medicaid on its recipients in Sommers, Baicker, and Epstein (2012) range from -80 percent to -177 percent (see appendix table A4.4). With 80 percent power, the smallest detectable one-year intention-to-treat effect in the Oregon data is a reduction of 160 deaths per 100,000, or -20 percent. The OHIE had 29 percent take-up, which implies that the smallest detectable effect of treatment on the treated is  $(-0.2/0.29 =)$  -69 percent—a large mortality effect, but one that is still smaller than quasi-experimental estimates for adults.

insurance in the US and provides tremendous variation in the insurance coverage of poor kids without the large crowd-out of private insurance that are present in more recent studies.

Furthermore, the time period allows me to generate flexible estimates of Medicaid's shorter- and longer-run effects.

## **1.2 Public Insurance and Mortality Before and After Medicaid**

The potential for Medicaid implementation to improve health depends largely on health status and alternative sources care for poor families before it began. In the 15 years prior to Medicaid, public health insurance for poor families was controlled by the states and consisted of direct reimbursements to medical providers financed jointly by state and federal governments. The federal contribution was capped, however, which made states reluctant to establish generous programs, which would leave them with the responsibility for medical costs exceeding the federal matching maximum. Consequently, less than one percent of children in 1963 received subsidies for health care, and many states restricted the services they covered: 16 states did not cover physician services, and 12 did not cover hospital services (**Committee on Ways and Means 1961**).

This lack of publicly-financed care was not offset by other non-profit or private sources, and this is reflected in income differentials in insurance, utilization, and health, especially for children. Private insurance coverage was relatively low in the 1960s: in 1959, only 8.9 percent of people with family incomes below \$2,000 had doctor visit insurance, and less than a third had either hospital or surgical insurance (**Kovar 1960**). Survey data from 1963 show that only 45 percent of children in the bottom third of the income distribution (family income less than \$4,000) had seen a physician within the previous year, compared to 77 percent of children in

families in the top third (income over \$7,000).<sup>9</sup> Serious symptoms, such as 4-5 days of diarrhea, heart pain, or unexpected bleeding were more common for poorer children, and conditional on having less-serious symptoms such as a skin rash, a persistent cough or sore throat, or abdominal pain, lower-income children were much less likely to receive care than higher-income children. Poor infants and adults died at twice the rate of the non-poor (**Kitagawa and Hauser 1973, MacMahon, Kovar, and Feldman 1972, Mathis 1969**). Thus, on the eve of Medicaid, with these statistics in mind, New York City's health commissioner, Dr. George James, cited poverty as the "the third leading cause of death" (**quoted in Humphrey 1968**).

### *1.2.1 A Brief History of Medicaid's Implementation*

Medicaid (P.L. 89-97) was established by the 1965 amendments to the Social Security Act (SSA), and aimed to eliminate these income-based inequalities in health and health care. Far from being the result of widespread demand for reform, though, the passage of Medicaid appears to have been an unexpected political move to undercut the American Medical Association's (AMA) opposition to Medicare (Harris 1966, Stevens and Stevens 1970, Goss 1995).<sup>10</sup> The AMA favored incremental changes to existing means-tested public insurance for the elderly poor. Medicaid applied this proposal to several non-elderly groups to incorporate the AMA's input while protecting Medicare's universal coverage. Thus, the conventional wisdom is that Medicaid was added to the SSA amendments as an "afterthought" (**Grannemann and Pauly 1983, Ginzberg and Solow 1974**). After the 1965 amendments to the SSA passed, states were

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<sup>9</sup> Tabulations from the 2011 National Health Interview Survey show that 77 percent of children in the bottom third of the income distribution (family income less than \$35,000) had a checkup within the previous year compared to 83 percent in the top third (family incomes above \$75,000; Minnesota Population Center and State Health Access Data Assistance Center (2012)).

<sup>10</sup> The final SSA amendments combined three proposals into Medicare Part A (compulsory hospital insurance for all elderly, the Democratic proposal), Medicare Part B (voluntary supplementary physician insurance for all elderly, the Republican proposal) and Medicaid (a federal/state funded public insurance program for the poor, the AMA's proposal). Assistant Secretary of the Department of Health, Education and Welfare Wilbur Cohen, remarked "It was the most brilliant legislative move I'd seen in thirty years...In effect, [Wilbur Mills (D-Arkansas)] had taken the A.M.A.'s ammunition, put it in the Republicans' gun and blown both of them off the map" (Harris 1966, pp. 40).

required to implement Medicaid by 1970 or else lose federal reimbursements for pre-existing medical programs. 26 states adopted Medicaid in 1966, 11 in 1967, and (most of) the rest between 1968 and 1970.<sup>11</sup>

Despite being billed as an incremental change, Medicaid represented a major expansion in federal support for the medical care of poor families. The financial mechanism for this expansion was a move to an open-ended appropriation, which eliminated the caps on reimbursement and increased the federal share of the cost of public medical payments from about 13 percent (Norman 1952) to between 50 and 83 percent. In return for increased federal funds, Medicaid required that states cover at least five types of care with no cost sharing—inpatient hospital, outpatient hospital, laboratory and x-ray, skilled nursing home, and physician services<sup>12</sup>—and mandated coverage for recipients of federally funded cash welfare programs (the “categorically eligible”).<sup>13</sup>

### *1.2.2 Medicaid Eligibility by Age and Race*

Cash welfare recipients included the poor elderly, blind, and disabled, but the categorical eligibility requirement had the biggest effect on public insurance eligibility of children through the program for single-parent families, Aid to Families with Dependent Children (AFDC). In January 1966, AFDC accounted for the largest share of categorical eligibility overall (62 percent), but virtually all categorical eligibility among children (**DHEW 1966**). The monthly

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<sup>11</sup> Alaska did not adopt Medicaid until 1972 and Arizona did not adopt it until 1982.

<sup>12</sup> States could also choose to cover a range of additional services, including home health care, clinic services, prescription drugs, eye care and dental care. The 1972 SSA amendments allowed states to charge co-payments for the optional Medicaid services (Davis and Schoen 1978), but not for the five required services. Categorically-eligible children were (and still are) exempt from cost-sharing.

<sup>13</sup> Medicaid defined several other eligibility groups not discussed here. In particular, states could choose to cover the “medically needy”—families with incomes too high to qualify for cash public assistance, but with large medical bills that pushed their net income below state-defined thresholds. The medically needy are an important group for understanding Medicaid costs (especially for nursing home care), but they account for only a small share of children on Medicaid and so I ignore this provision in the rest of the paper. For a detailed discussion of Medicaid eligibility see Gruber (2003), Advisory Commission on Intergovernmental Relations (1968), and Stevens and Stevens (1974).

AFDC rate among children under 18 was five times the rate for adults. Furthermore, children rarely qualified for Medicaid under other provisions. In 1976, 86 percent of children on Medicaid were eligible through AFDC (**DHEW 1976a**), indicating that AFDC receipt is an accurate proxy for their Medicaid eligibility.

Because of differences in family structure and income, however, a much higher share of nonwhite children received AFDC and gained Medicaid coverage than white children. I use two sources of data to measure racial differences in eligibility. For 1958 and 1961, I entered state-level data on the share of AFDC cases and children who were nonwhite (**Mugge 1960, DHEW1963**), and for 1967-1979 I calculate this share using microdata on AFDC recipients collected from the National Archives (**DHEW 2000, 2011**). Combining these shares with the administrative count of AFDC recipients and dividing by state population totals for children ages 0-19 gives an estimate of the AFDC rate for each race.<sup>14</sup> Figure 1-1 plots the age profiles of AFDC receipt using the 1967 data.<sup>15</sup> Children of both races received AFDC (and therefore gained Medicaid eligibility) at almost four times the rate of adults ( $0.184/0.049 = 3.75$  for nonwhites and  $0.03/.008 = 3.75$  for whites), but the differences by race are even larger. Nonwhite children received AFDC at more than six times the rate of white children ( $0.184/0.03 = 6.13$ ). The statutory connection between AFDC receipt and Medicaid eligibility, therefore, implies that nonwhite children had by far the highest eligibility rates for the new and relatively generous public insurance.

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<sup>14</sup> To construct an annual series I linearly interpolate the race shares between missing years, but multiply them by annual data on case or child counts from HHS. I cannot calculate age-specific AFDC rates for all years because the 1958 and 1961 reports only contain the race distribution of AFDC payees and children, without specifying their age, and the microdata from 1973-1979 only give age in bins. I use a binary measure of race to maintain consistency across AFDC data sources.

<sup>15</sup> I present the age profile for 1967 because it is the largest AFDC datasets with 265,707 observations (4,297 observations on average in each race/age cell). The age profile of welfare receipt in the 1970 Census is very similar (Appendix 2).

### 1.2.3 Medicaid Use By Age and Race

Not surprisingly, high rates of eligibility translated into relatively high rates of Medicaid use. The solid line in figure 1-2 shows the share of all children ages 0-19 who received medical services paid for by public insurance in the years before and after states began their Medicaid programs.<sup>16</sup> The public insurance rate increased from under one percent to 10 percent in the five years after the implementation of Medicaid.<sup>17</sup> Consistent with the eligibility differences by age, the increase for adults (not shown) was less than two percentage points. Annual data on public insurance use by race are not available, but several data sources show racial differences similar to those in eligibility. The ratio of public insurance use for nonwhite children ages 1-4 to the average child ages 0-19 is 2.7 in the 1976 Survey of Income and Education (**US Department of Commerce 2006**), and 3.7 in the 1976 National Health Interview Survey (see Appendix B).

### 1.2.4 The Expected Effects of Medicaid Implementation on Mortality

These changes may have affected a range of health outcomes, but the primary measure used in this paper (and in other work on Medicaid and health) is mortality. Death is an extreme health measure, but conceptually it is an unambiguous indicator of poor health, especially for children, and unlike other health measures, it is easily observed. Data on diagnoses, for example, would conflate potentially-offsetting changes in utilization with changes in underlying disease processes. Also, “preventing... premature death” (**DHEW 1967a**) was one of Medicaid’s explicit goals.

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<sup>16</sup> These data were entered from federal reports on means-tested public insurance from 1963 to 1976 (DHEW various years). See appendix 1 for data cleaning. The data measure *utilization* of benefits, referring to children who actually obtained medical care. More recent papers measure reported Medicaid *coverage*, referring to children who have signed up for but not necessarily used Medicaid. Utilization means more for health than coverage, and it incorporates the effects on the availability of providers who accept Medicaid patients, which would not be reflected by coverage data.

<sup>17</sup> For comparison, during the eligibility expansions in the 1980s the share of children under 15 enrolled in Medicaid rose by 0.074 (Currie and Gruber 1996a Table 1), and in the five years following enactment of the State Children’s Health Insurance Program in 1996, the share of children under 18 on public insurance increased by 0.027 (Cunningham 2003, Exhibit 1).



The expected effects of Medicaid on mortality hinge on the extent to which the medical care it provided actually prevented deaths. The share of deaths due to “internal” causes is a common measure of the sensitivity of mortality to medical interventions (**Currie and Gruber 1996a**).<sup>18</sup> Figure 1-3 shows that in 1965, internal causes account for nearly all infant deaths, more than 60 percent of deaths among 1 and 2 year olds, and about 50 percent of deaths among 3 to 12 year olds.<sup>19</sup> For older adults, cardiovascular-related conditions accounted for over three quarters of internal-cause deaths (**Bailey and Goodman-Bacon 2013**), but for children, many more internal-cause deaths were due to infectious disease.

The groups with the highest Medicaid eligibility and utilization rates, nonwhite children and infants, were especially at risk. Their mortality rates in 1965 were twice as high as for whites of the same age (**National Center for Health Statistics 1965, Table 1-9**), and they were much more likely to die of causes with “effective” treatments (**Beeson 1980**). Vital Statistics data from 1965 show that 35.4 percent of all nonwhite child deaths (ages 1-4) were due to infectious diseases, which primarily included pneumonia (18 percent), meningitis (4.6 percent), and gastroenteritis (3.2 percent). 26.4 percent of white child deaths were due to these causes (standard error [s.e.] of the difference = 0.8 percent). Perhaps due to such high death rates from infectious disease, nonwhite deaths were less likely than white child deaths (24 percent versus 36.5 percent, s.e. of the difference = 0.85 percent) to be attributed to non-infectious or chronic conditions such as congenital malformations or cancer. Nonwhite deaths were also more likely to

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<sup>18</sup> The International Classification of Disease (revision 7) defines a set of “external” causes that include mainly transportation-related accidents, drowning, falls, poisonings, choking, homicide and suicide. All other causes are “internal”.

<sup>19</sup> While internal-cause mortality is high for the elderly, they were already covered by two kinds of public insurance plans when Medicaid began. In July 1966, everyone over 65 received hospital insurance through Medicare part A, and 93 percent also received coverage for physician visits through Medicare part B (U.S. Social Security Administration 1969). Poor elderly households were covered by public insurance even before Medicare by the Kerr-Mills program. Thus, Medicaid represented a much smaller change in the availability of publicly-financed health care for people 65 and over.

be due to causes so general that they reflected inadequate medical care (5.36 percent versus 1.58 percent, s.e. of the difference = 0.3 percent), such as “ill-defined symptoms or conditions”.

Inexpensive, available treatments could often have prevented, managed, or cured these underlying causes of death. Pneumonia is the most common example: the vast majority of cases were bacterial, and when treated early with penicillin “approximately 95 per cent of patients...recover” (**Cecil et al. 1967**). Antibiotics were similarly effective for infections such as meningitis or gastroenteritis. Non-bacterial conditions could often be managed, if not prevented or cured. For example, nonwhite children (because of a genetic predisposition among African-Americans), were more than twice as likely to die from anemias (2.7 percent versus 1.2 percent), but a folate supplement “suppresses or controls the disease” (**Beeson 1980**). A small percentage of deaths in the mid-1960s could have been prevented with the recommended vaccines for smallpox, diphtheria, pertussis, tetanus, measles, and polio (and later, mumps and rubella). Compared to white children, nonwhite children were less likely to be fully vaccinated for these conditions (**National Center for Health Statistics 1976, Tables CD.I.47 and CD.I.48**), and more likely to die of them (1.6 percent versus 0.9 percent, s.e. of the difference = 0.2 percent).

Thus, I expect Medicaid implementation to have the strongest effect on the mortality of nonwhite children and infants for two reasons. First, Medicaid had the largest effect on public insurance eligibility among this group.<sup>20</sup> Second, they were much more likely to die of causes that could be prevented, managed, or cured by the available primary care.

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<sup>20</sup> In theory, Medicaid may reduce mortality through several other channels as well. If Medicaid paid for services that poor families had previously purchased out of pocket, then it may affect child health through an income effect without any change in the use of care. In this case, Medicaid may be effective, but as a cash rather than in-kind transfer program. However, the strong income-based disparities in actual medical care before Medicaid suggest that increases in purchasing power cannot be the primary mechanism for mortality changes. Alternatively, by increasing federal payments for medical care, Medicaid may also have led to responses among providers that could affect the mortality rates even of children not *on* Medicaid (Pauly and Pagán 2007). Finkelstein (2007) provides suggestive evidence that the introduction of Medicare increased hospital entry and investments in cardiac care technology. If

### 1.3 Research Design: Using Categorical Eligibility to Identify Heterogeneous Effects of Medicaid Implementation on Mortality

In addition to its demographic implications, the categorical eligibility requirement meant that the sudden increase in public insurance eligibility under new Medicaid programs varied widely across states. This cross-state eligibility variation is the basis of my research design. I identify Medicaid’s effect using a difference-in-differences model that compares state-level health outcomes before and after Medicaid implementation in states with higher and lower categorical eligibility. This section argues that pre-existing cross-state differences in welfare rates correspond to public insurance rates after Medicaid (relevance), and are unrelated to other time-varying determinants of health (excludability). I then outline an econometric specification that uses variation in the existence of Medicaid as well as variation in the size of initial eligibility to estimate effects on mortality.

#### *1.3.1 Cross-State Variation in AFDC-Based Categorical Eligibility and Public Insurance Use*

The most direct measure of the share of each state’s population that suddenly gained public insurance eligibility after Medicaid is the AFDC rate in the year of Medicaid implementation. Because most Medicaid programs began partway through the year and were subject to delays due to “shortages of welfare personnel to screen applications” (**Tax Foundation 1968, pp. 47**), the calendar year of implementation is just before states’ first full year with an operational Medicaid program. By this measure, about 4.7 percent of children were categorically eligible through AFDC, but state-level eligibility varied by a factor of seven, from 1.5 percent in New Hampshire to 11 percent in Mississippi.

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provider investments drive Medicaid’s health effects, then the health benefits should not be confined to groups with high Medicaid eligibility—a conclusion I reject in the results below.

As with the levels of welfare receipt (figure 1-1), cross-state variation in AFDC-based categorical eligibility was much higher for nonwhite than white children. The standard deviation of initial AFDC rates was more than five times higher for nonwhites than for whites (0.08 versus 0.015), and nonwhite child AFDC rates ranged from about 5 percent to 30 percent while white AFDC rates ranged from less than 0.5 percent to 10 percent. (Appendix table B-1 shows state welfare rates.)

Moreover, states with high nonwhite AFDC rates often had low white AFDC rates and vice versa. Table 1- 1 shows a cross-tabulation between states with white and nonwhite AFDC rates above or below the race-specific median in the year of Medicaid implementation. 20 out of 48 states (42 percent) are above the median for one race but below for the other, and the coefficient from a regression of nonwhite on white AFDC rates is positive (1.17) but insignificant (s.e. = 0.9), with an R<sup>2</sup> of 0.036. The racial differences in cross-state AFDC variation mean that my design will have more power to detect Medicaid's effect on nonwhite children than on white children, and the weak relationship between white and nonwhite AFDC rates suggests that this statistical power requires a race-specific measure of categorical eligibility. Therefore, whenever I compare race-specific outcomes such as mortality rates, I also use race-specific AFDC rates.

The cross-state differences in AFDC-based categorical eligibility also led to differences in actual public insurance use. The dashed lines in figure 1-3 plot children's public insurance utilization in high- and low-eligibility states (defined by the median overall AFDC rate). The difference between high- and low-AFDC states before Medicaid implementation was very small (0.007, s.e. = 0.003), but rose to 0.05 (s.e. = 0.006) after Medicaid was fully implemented. Thus, the initial AFDC rate strongly predicts post-Medicaid public insurance use. While the evidence

is more limited, cross-state differences in public insurance use also appear to be larger for nonwhite children than for white children. Data from the 1970 Survey of Health Services Utilization and Expenditures show that the share of nonwhite children under 5 who had medical care paid for by a public source (including Medicaid) is 17 percentage points higher in high-nonwhite-eligibility states (s.e. = 0.05) than in low-nonwhite-eligibility states. The difference for white children is 10 percentage points (s.e. = 0.03).

### *1.3.2 Determinants of State-Level Categorical Eligibility*

The research design compares health outcomes over time between states with higher- and lower-AFDC rates. For such an approach to uncover Medicaid's health effects, the AFDC distinction needs to be unrelated to changes in mortality except through its statutory connection to Medicaid eligibility. This is especially likely in the present case because AFDC differences across states emerged for institutional and economic reasons decades before Medicaid was implemented and were highly persistent over time.

AFDC rates vary both because of factors that affect eligibility—state policies, family structures and income—and factors that affect take-up—psychic costs and institutional barriers. Cliometric studies on welfare programs show that these variables differed across states at least as far back as the 1930s. **Moehling (2007)** demonstrates that cross-state differences in family structure and the generosity of transfer programs for one-parent families existed even before the implementation of the Aid to Dependent Children program (the original name of AFDC), and persisted through the 1990s. **Alston and Ferrie (1985)** argue that agricultural states restricted welfare programs in the 1930s in order to maintain a “loyal” workforce. In many states nonwhite families were kept off the rolls by discriminatory local application of vague eligibility provisions such as “suitable home” or “substitute parent” policies that were part of pre-AFDC

Mothers' Pension programs (**Bell 1965**). Finally, states that adopted AFDC latest had some of the lowest AFDC rates even 30 years later.

The ensuing cross-sectional variation in state AFDC rates was notably stable over time. For both races, AFDC rates up to two decades before strongly predict AFDC rates in the year of Medicaid implementation ( $AFDC_s^*$ ). The slopes from univariate regressions of  $AFDC_s^*$  on AFDC rates in 1961, 1958, and 1948 are positive, very precisely estimated, and most importantly they are not statistically distinguishable from each other. (p-values from a test of the null hypothesis that they are equal are 0.63 for nonwhite rates and 0.72 for white rates.) This suggests that variation across states in initial categorical eligibility did not emerge contemporaneously with Medicaid, but reflects long-standing differences apparent several decades beforehand.

The long-run nature of AFDC-based categorical eligibility does not necessarily imply that AFDC rates are uncorrelated with state-level characteristics, but in many cases this is true by the 1960s. For white children, rows (1) through (3) of table 1-2 show the child poverty rate in 1960, the probability that children lived in a single mother household in 1960, and the average AFDC benefit in 1967. White child poverty in low-AFDC states (column 1) and high-AFDC states (column 2) are indistinguishable (p-value of the difference is in column 3), but, consistent with **Moehling (2007)**, single motherhood and average benefit amounts are slightly significantly higher in the states with high white AFDC rates. Rows (7) through (9) show that for nonwhite children, none of these variables is significantly different between high- and low-AFDC states, perhaps because of the influence of institutional deterrence.

Additional evidence on the validity of the research design is found in rows (4) through (6) and (10) through (12) of table 1-2, which show that changes in observable variables do not differ between high- and low-AFDC groups. Changes in child poverty (between 1950 and 1960)

and infant and child mortality (in the five years before Medicaid) are indistinguishable for both whites and nonwhites. Panel C uses a binary measure of AFDC (not by race) to show that pre-Medicaid health resources (per-capita hospital beds and the share of children on public insurance) did not change differentially in the AFDC groups in the years prior to Medicaid. Common trends in observables are not implied by predetermined AFDC rates, but they do provide further evidence that AFDC differences did not emerge around the time of Medicaid implementation.

Table 2 supports the conclusion of cliometric research showing that the large cross-state differences in welfare-based Medicaid eligibility were inherited from long-run institutional variation in the welfare system. Comparing changes in health outcomes across states with different rates of categorical Medicaid eligibility is therefore unlikely to capture underlying differences across states due to differences in either the level or trend in state characteristics.

### *1.3.3 Data and Estimation Sample*

To measure health, I construct state-by-year infant and child mortality rates from the 1959 to 1979 Vital Statistics Multiple-Cause of Death Files (**US DHHS and NCHS 2009**), which contain the universe of civilian deaths that occurred in the U.S. by cause, age, race, and state of residence of the decedent.<sup>21</sup> For children, the age-specific mortality rate is the count of deaths in group a (ages 1-4 or 5-14) divided by the population in age a per 100,000. I also use two measures of infant mortality: neonatal mortality (deaths in the first 28 days of life per 1,000 live births) and post-neonatal mortality (deaths between 28 days and 1 year per 1,000 live

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<sup>21</sup> The exception is 1972, which contains a 50 percent sample, and 1981 and 1982 which contain a 50 percent sample for some states. In 1981 and 1982, I use Mortality Detail files, and in 1972, the mortality are based on the reduced sample.

births).<sup>22</sup> Finally, I end the sample in 1979 because the 1980s eligibility expansions largely eliminated the state differences that drive my results.<sup>23</sup>

### 1.3.4 Event-Study Specification with High- and Low-Eligibility Groups

The main estimating equation uses an event-study specification (**Jacobson, LaLonde, and Sullivan 1993**) for the log mortality rate of age group  $a$ . It includes state-by-year-level covariates and fixed effects in  $\mathbf{x}'_{st}$ , and interactions between a high-AFDC indicator,  $D_s$  defined in table 1-1 and dummy variables that measure the time relative to Medicaid implementation,  $1\{t - t_s^* = y\}$  (i.e., “event-time”):

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

Equation (1) is a difference-in-differences (DD) model where the high-AFDC states are the treatment group, the low-AFDC states are the control group, and pre/post treatment is defined by the year of Medicaid implementation. The most parsimonious set of covariates that preserves the DD interpretation is a high-AFDC dummy,  $D_s$ , and the baseline Medicaid-timing dummies,  $1\{t - t_s^* = y\}$ . My preferred specification includes per-capita income, per-capita hospital beds, and three sets of fixed effects: state fixed effects, region-by-year fixed effects, and a separate set of 21 year fixed effects for each Medicaid timing group from 1966 to 1970.

Conditional on region-by-year fixed effects, the estimates rely on mortality comparison between high- and low-AFDC states within each region. In particular, this controls for the strong convergence in mortality between the South and the rest of the U.S. due to hospital

<sup>22</sup> Denominators for the child rates were constructed by linearly interpolating population between the 1950 and 1960 censuses (Haines and ICPSR 2005) and the 1969 to 1988 Surveillance Epidemiology and End Results (SEER 2009) data. Denominators for the infant rates were calculated from Vital Statistics Natality Microdata from 1968-1979 (US DHHS and NCHS 2002) and entered state totals from Vital Statistics reports from 1959-1967.

<sup>23</sup> Appendix 2 presents results on a sample from 1959 to 1988 and, consistent with the convergence in Medicaid rates induced by the 1980s expansions, the results fade by 1988.



desegregation (**Almond, Chay, and Greenstone 2006**), region-level trends in school quality (**Stephens and Yang 2013**), or private insurance coverage (**Finkelstein and McKnight 2008**).<sup>24</sup>

The Medicaid-timing-by-year fixed effects eliminate comparisons between states that adopted Medicaid earlier or later. A DD model based only on the differential timing of Medicaid adoption is identified (**Decker and Gruber 1993, Strumpf 2011**), however mortality trends in earlier and later Medicaid states differ systematically and violate the identifying assumption of this “timing-only” estimator (see Appendix C). Policymakers at the time reported putting off Medicaid implementation because of fiscal concerns (**ACIR 1968**), and **Finkelstein (2007)** concludes that, with respect to hospital capacity, “the timing of state implementation of Medicaid was not random.” The Medicaid-timing-by-year fixed effects ensure that estimates of equation (1) rely only on comparisons between AFDC groups rather than between earlier and later Medicaid states.

The coefficients of interest are  $\pi_y^a$  and  $\gamma_y^a$ , which measure the covariate-adjusted difference in log mortality between high- and low-eligibility states in the seven years leading up to Medicaid’s introduction and the nine years after. I use a binary variable to measure eligibility because it yields this simple interpretation, but the results are unchanged by replacing  $D_s$  with the continuous AFDC rate in each state’s Medicaid implementation year. I define groups by the median AFDC rate so that they each have an equal number of states, but the results are not sensitive to defining  $D_s$  using an algorithm that maximizes the t-statistic on the difference in AFDC rates between the two groups (see Appendix B). I use AFDC rates for women because it is the appropriate measure of eligibility for the infant (especially neonatal) mortality regressions,

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<sup>24</sup> The main results use four census regions, but the results are not sensitive to different definitions, including moving the northern-most southern states (Delaware, Maryland and West Virginia) into the “Northeast” region, dropping the Deep South (Alabama, Georgia, Louisiana, Mississippi, and South Carolina) or dropping the South entirely.

and I use the same rates in the child regressions so that there is a common state grouping used in all of the results. The results for non-infant children are unchanged when I create state groups using the child AFDC rates (Appendix B). The dummy for the year before Medicaid  $1\{t - t_s^* = -1\}$  is omitted (to avoid collinearity with the state fixed effects), which normalizes the estimates of  $\pi_y^a$  and  $\gamma_y^a$  to zero in that event-year.<sup>25</sup>

The  $\pi_y^a$  are falsification tests that capture differences between the two AFDC groups in the pre-Medicaid period. If mortality rates in high-eligibility states were already falling prior to Medicaid, then these coefficients will be positive, declining, and statistically significant. The  $\gamma_y^a$  are intention-to-treat (ITT) effects of Medicaid on aggregate mortality in high-AFDC states relative to low-welfare states. Note that this specification identifies heterogeneity in Medicaid's effect. The estimates will equal zero if Medicaid affected mortality equally across states. Moreover, they will understate Medicaid's total effect on mortality because they "difference out" any portion of the effect that is common to low- and high-eligibility states. For example, if Medicaid led to investments in hospital technologies, as was the case for Medicare (Finkelstein 2007), then mortality effects arising from investment that is common to high- and low-AFDC states will not be captured by this empirical strategy.

The main results are plots of  $\pi_y^a$  and  $\gamma_y^a$  but I also present the coefficients from a "grouped" event-study specification that combines the event-time dummies into four bins ( $[-7, -2]$ ,  $[0]$ ,  $[1,4]$ ,  $[5,9]$ ) or a difference-in-difference (DD) specification that estimates one treatment effect (for event-years  $[1,9]$ ).

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<sup>25</sup> Event-time dummies that are more than seven years before or nine years after Medicaid implementation are grouped because not all states are observed at these event-years. (The data begin in 1959 and the earliest Medicaid year is 1966.)

#### 1.4 Intention-to-Treat Estimates of Medicaid's Effect on Mortality Rates

The primary mechanism through which Medicaid implementation should affect mortality is by increasing the utilization of (publicly-financed) health services. A test of this hypothesis is in figure 1-4, which shows first-stage estimates from both event-study and difference-in-difference specifications of equation (1) with child public insurance rates as the dependent variable. The public insurance data are only available from 1963-1976, so the figure shows coefficients for event-years -3 through 6. Public insurance use is indistinguishable between the high- and low-AFDC groups before Medicaid (the p-value from a joint significance test of the -3 and -2 coefficients is 0.15), but it rises in the high-AFDC states in the first year after Medicaid implementation and is 5.5 percentage points higher in the next six years (s.e. = 1.6). The estimates in figure 1-4 are weighted by the state population under age 19, but a Hausman test cannot reject the equality of the weighted and unweighted estimates. (The unweighted DD estimate is 0.037, s.e. = 0.016.) These results show that, even conditional on a rich set of covariates, AFDC-based eligibility is strongly associated with increases in public insurance after Medicaid.<sup>26</sup>

The wide differences in eligibility by race suggest that these first-stage results should be much larger for nonwhite children. The difference in AFDC rates between the high- and low-eligibility states used in figure 1-4 (based on the overall AFDC rate) is 1.1 percentage points. The difference for nonwhite AFDC rates between high- and low-nonwhite-eligibility states is 4.5 percentage points. This implies that the relevant first stage difference in child public insurance use for nonwhites is 22 percentage points, 4.1 times (4.5/1.1) larger than for the overall first-stage results.

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<sup>26</sup> Appendix 2 shows that per-recipient expenditures are not different after Medicaid in high- and low-AFDC states. This suggests that the size of the categorically-eligible population, while strongly related to Medicaid use, is not related to the generosity of state Medicaid programs, at least for children.

#### *1.4.1 Results for Age-Adjusted Child Mortality by Race*

Figure 1-5 plots the event-study estimates from my preferred specification of equation (1) for log age-adjusted mortality for white and nonwhite children ages 0-14. The results are weighted by state populations, but the unweighted point estimates are nearly identical (see Appendix B). The standard errors are clustered at the state-level to allow for arbitrary serial correlation within states.

The small pre-Medicaid estimates support the AFDC-based research design. In the seven years before Medicaid, high- and low-eligibility states had nearly identical mortality changes. The pre-Medicaid point estimates are between -0.023 and 0.028, and neither the white nor the nonwhite coefficients are jointly distinguishable from zero (p-values are 0.44 and 0.33, respectively). The distinct absence of differential mortality trends before Medicaid suggests that any potential bias must be due to omitted factors that change coincidentally with Medicaid implementation and not variables correlated with longer-run trends in mortality.

Consistent with the eligibility rates documented in Section II.B, the post-Medicaid estimates are strong and negative for nonwhite children. Nonwhite mortality fell slightly in the year of Medicaid implementation (time 0 on the x-axis), which matches the pattern in the first-stage estimates and reflects the fact that Medicaid programs were only partially implemented in the first calendar year. After the first year, however, nonwhite mortality in high-AFDC states fell significantly more than in low-AFDC states. The event-study estimates are highly jointly significant (the p-value on a joint F-test of the post-Medicaid coefficients is 0.0001), and the DD estimate shows that mortality was about eight percent lower (s.e. = 0.03). Aggregate nonwhite child mortality fell by about 40 percent in high-AFDC states in the ten years after Medicaid, so an effect of eight percent suggests that Medicaid was an important contributor to these declines.

The post-Medicaid estimates for white mortality, on the other hand, are essentially zero. This matches the differences in Medicaid eligibility and bolsters the claim that the effects are attributable to Medicaid. Alternative explanations for the results must not only correspond to the timing of Medicaid, but must also affect only nonwhite infants and children. Do these effects suggest that white children were unaffected or that the research design simply cannot detect their presumably smaller ITT effect? I use the relative magnitudes of AFDC receipt for whites and nonwhites to calculate the expected effect for white children if their underlying treatment effect was the same as for nonwhite children. The difference in average AFDC rates between the high- and low-eligibility groups was four times higher for nonwhites than for whites (0.045 versus 0.01), which suggests that, given a nonwhite effect of -0.08, the white ITT effect should be -0.02 (-0.08/4.1). The confidence interval of the white DD estimate (-0.011, 0.053) can rule out an effect of this size, which supports the claim that Medicaid had a smaller effect on white children than on nonwhite children. The rest of this section reports evidence for nonwhite mortality only. Results for whites are in Appendix B, and are small and insignificant for all age groups.

The results in figure 1-5 are from the full specification, but table 1-3 shows that these controls have only a small effect on the estimates. Panel A presents estimates from a “grouped” event-study specification, and panel B presents DD estimates. Columns 1 through 3 add covariates, beginning with the simplest specification and column 4 is an unweighted version of the full specification used in figure 1-5. The grouped event-study estimates are nearly identical in the first three columns and are slightly larger in the unweighted model. A Hausman test cannot reject the null hypothesis of equality between the estimates in columns four and five for either panel (**Deaton 1997**).<sup>27</sup> The DD estimates in panel B are also nearly identical, ranging

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<sup>27</sup> The motivation for a test comparing weighted and unweighted estimates is to detect unmodeled parameter heterogeneity or other forms of misspecification, in which case the two estimators may disagree (Solon, Haider, and

from -0.08 to -0.10.<sup>28</sup> Panel B also contains the p-value from a test of the DD restrictions from the grouped event-study model: that the pre-Medicaid coefficient equals zero and the post-Medicaid coefficients (except year 0) are equal. These restrictions are not rejected for any of the models in table 1-3.

Figures 4 and 5 provide evidence in line with the prediction that Medicaid's effect would be largest for nonwhite children in high-AFDC states. High- and low eligibility states were comparable in the years prior to Medicaid, but afterward, children in high-eligibility states used public insurance more and the mortality rates of nonwhite children fell more. This is consistent with a causal effect of Medicaid on mortality. Not all children were equally likely to benefit from Medicaid, though. If Medicaid-funded primary care is indeed the mechanism for the age-adjusted effects, then the results should be strongest for groups whose mortality was potentially affected most: infants and young children.

#### *1.4.2 Results for Nonwhite Neonatal and Post-Neonatal Mortality*

Panels A and B of figure 1-6 present event-study results for one of the groups that was most vulnerable and most likely to benefit: nonwhite infants. The results show that Medicaid's effect is concentrated among neonatal rather than post-neonatal mortality. As in figure 1-5, the pre-Medicaid coefficients in panel A are small (although the p-value from a joint F-test of the pre-Medicaid coefficients is 0.007), and after Medicaid the point estimates are negative and highly jointly significant (p-value less than 0.0001). The post-Medicaid point estimates fluctuate between 3 and 15 percent, but the DD estimate for this period is -0.08 and is precisely estimated

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Wooldridge 2013, DuMouchel and Duncan 1983). An example of this is in Appendix 3, which shows that, for a specification based only on timing (i.e., one that omits the heterogeneity exploited in my research design), the equality of weighted and unweighted estimates is rejected. My results are invariant to weighting, so I present weighted results because they are more precise.

<sup>28</sup> Appendix 2 contains event-study estimates from the simplest specification, which is equivalent to taking the difference in means between high- and low-AFDC states at each event-year. The results are unchanged from those in figure 5.

(s.e. = 0.022). Table 1-4 presents grouped event-study and DD results for nonwhite neonatal mortality, and shows that the estimates are similar across specifications as well as by whether or not they are weighted.<sup>29</sup> For the models with region-by-year fixed effects, the DD restrictions cannot be rejected, which suggests that -0.08 is a reasonable summary estimate of the effect on neonatal mortality during Medicaid's first ten years.

In contrast to the strong negative effects on neonatal mortality, panel B of figure 1-6 shows no differential reduction by AFDC group for post-neonatal mortality. This may seem strange, since, like neonates, post-neonatal infants died of causes that were easily addressed by primary care. Two factors can help explain this result. First, reductions in neonatal mortality may induce negative selection into the group of surviving post-neonatal infants, which would bias the post-neonatal results toward zero. Second, **Almond, Chay, and Greenstone (2006)** show that post-neonatal mortality among nonwhite infants, especially in the South, was already declining starting in 1965 because of federally-mandated hospital desegregation. Their results imply that, once hospitals in a state were desegregated, there would have been less room for Medicaid to reduce post-neonatal mortality because infants with acute, life-threatening conditions could already obtain effective care at hospitals. This was not true for prenatal care or labor and delivery, both of which may be more strongly connected to neonatal deaths and would have been affected by Medicaid coverage.

The pattern of results for infants suggest that Medicaid improved the health of babies at birth or, conditional on health, their probability of surviving the period immediately after birth. To examine the mechanisms for increased survival, I use the 1964-1969 and 1972 National Natality Surveys (or the National Natality Followback Survey, NNFBS). The NNFBS data

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<sup>29</sup> A Hausman test cannot reject the equality of the weighted and unweighted estimates in columns 3 and 4 for either the grouped event-study (p-value = 0.44) or the DD estimates (p-value = 0.54).

contain demographic and socioeconomic variables for the families of a sample of infants born in each survey year, and they include data collected from providers on the medical circumstances of the birth. The seven years of data contain 3,821 nonwhite births.<sup>30</sup> This allows me to examine birth outcomes other than mortality, such as low birth weight (less than 2500 grams, or 5.5 lbs.), prematurity (less than 36 weeks gestational age) and birth in a hospital, by family income, and birth order. In 1964 only ten states provided AFDC (and therefore Medicaid) to mothers pregnant with their first child. Therefore, comparing results for covered births (first or subsequent births in a state that provided AFDC to first-time pregnant mothers, or subsequent births in a state that did not) and non-covered births is an additional test of whether the effects are due to Medicaid.

Table 1-5 contains the results from linear probability models that contain the same fixed effects included in equation (1) as well as individual-level covariates: mother's age dummies, dummies for plural and first births, a dummy for the baby's sex, and separate sets of family income dummies for each year. The coefficients of interest are interactions between a post-Medicaid dummy and a high-AFDC dummy, but because the NNFBS contains individual-level data, I estimate separate effects for four groups defined by poverty status and the possibility of AFDC coverage during the perinatal period.

The evidence in table 1-5 suggests that Medicaid reduced nonwhite neonatal mortality not by increasing birth weight, but by reducing pre-term births and by increasing the share of births that occurred in hospitals. Column 1 provides no evidence that Medicaid implementation affected the probability of low birth weight. This differs from evidence based on the timing of

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<sup>30</sup> The NNFBS sampling frame is "legitimate births", meaning that the mothers were married at the time of the birth. While AFDC would have primarily covered *illegitimate* births, there is still some overlap between the NNFBS sample and categorically eligible mothers. Two thirds of AFDC mothers in the 1967 Characteristics Survey had been married at some point and a mother could have misreported her marital status to welfare authorities or on the child's birth certificate.



Medicaid implementation (**Decker and Gruber 1993**) but is consistent with evidence from Medicaid expansions (**Currie and Gruber 1996b**) and the introduction of national health insurance in Canada (**Hanratty 1996**).

Column 2 shows that, for covered births to poor women, pre-term births (gestational age less than 36 weeks) fell by a precisely estimated 7.6 percentage points (s.e. = 2.6), but prematurity among births to non-poor mothers was unaffected. The point estimate for poor non-covered births is large and negative, however, but it is not distinguishable from zero. It is puzzling that a reduction in preterm births, which represent the smallest infants, was not paired with a reduction in the probability of low birth weight. This pattern of results is consistent with **Aizer, Lleras-Muney, and Stabile (2004)** who find that improvements in hospital quality reduced neonatal mortality and prematurity among black Medicaid recipients, but had no effect on the probability of low birth weight. Thus, Medicaid may have improved the quality of care in the weeks prior to birth, but did not increase the probability that the ultimately full-term infants weighed more than 2500 grams.

Improvements in acute care at birth are a more direct channel through which Medicaid could have specifically affected neonatal mortality. The estimates for the probability of giving birth in a hospital (column 3) imply that Medicaid had a large effect on the site of births. AFDC-covered births to poor mothers were almost seven percentage points more likely to occur in a hospital after Medicaid in high-eligibility states relative to low-eligibility states (s.e. = 0.027). The mean hospital birth probability for poor nonwhite women in high-AFDC states before Medicaid was 0.8, so these estimates imply that Medicaid accounts for more than a third of the gap across racial groups in the site of delivery. This result helps to rationalize the strong neonatal mortality results in figure 1-6 because it suggests that Medicaid improved the medical

circumstances at birth, which should naturally have the strongest effect on the earliest infant deaths.

The evidence in table 1-5 matches the conclusions from perinatal epidemiological research which finds that changes in the distribution of fitness at birth account for only a small share of neonatal mortality declines between 1950 and the late 1970s (**David and Siegel 1983, Lee et al. 1980, Williams and Chen 1982**). These results suggest that Medicaid implementation can help explain both the aggregate changes in neonatal mortality and the important contribution of survival conditional on health at birth.

#### *1.4.3 Results for Younger and Older Nonwhite Children*

The non-infant age groups that contribute to the summary results for children 0-14 in figure 1-6 provide an additional test of the proposition that Medicaid's effects arise through increased primary care. Younger nonwhite children had higher mortality rates than older nonwhite children and, as figure 1-3 shows, they were more likely to die of internal causes that were often easily treatable. If the age-adjusted results are due to Medicaid-funded primary care, then the mortality rates should respond more for younger children than for older children, and internal-cause deaths should drive the results.

Panels C and D of figure 1-6 present event-study results for all-cause nonwhite child mortality rates that bear out the first prediction. Panel C shows small and insignificant pre-Medicaid estimates for younger child mortality (p-value = 0.88) and a gradual reduction in mortality after Medicaid (p-value = 0.007) with a corresponding DD estimate of -0.126 (s.e. = 0.049). Panel D shows no evidence of an effect for children ages 5-14 (DD estimate = -0.012, s.e. = 0.03). Thus, in terms of all-cause age-group-specific mortality, the most vulnerable group was most affected.

Unlike the age-adjusted and infant results, the estimates for young nonwhite children are dependent on the inclusion of region-by-year fixed effects. This does not refute the conclusion that Medicaid had an effect on the mortality of young children, since there are strong reasons to expect differential mortality trends by region. The results across specifications in table 1-6 show that the DD estimate is about twice as large when these controls are included in columns three and four as it is in any of the models that exclude them (columns 1 and 2).

Evidence supporting the second hypothesis, that internal-cause mortality should respond more to Medicaid than does external cause mortality, is provided in table 1-7. The results show that nearly all of Medicaid's effect comes from reductions in internal cause deaths. The DD effect on internal-cause mortality is -0.14 and is very precisely estimated (s.e. = 0.03), while the effect on external-cause mortality is smaller (-0.09) and not distinguishable from zero (s.e. = 0.07). Appendix B also presents event-study results for internal-cause deaths showing that the region-by-year fixed effects account for a negative pre-Medicaid trend in internal-cause mortality for younger nonwhite children. Therefore, even though the results for nonwhite children are sensitive to the inclusion of these fixed effects, the event-study results support including them.

The causes of death highlighted in Section II were most often treated with antibiotics or other pharmaceutical treatments (**Beeson 1980**) when detected early enough. Table 1-8 provides additional first-stage DD estimates of Medicaid's effect on the utilization of four specific services: hospital admission, physician visits, prescription drugs, and dental services. Increases in utilization in high- versus low-eligibility states were largest for physician visits (0.027, s.e. = 0.008) and prescription drug use (0.033, s.e. = 0.008), which is consistent with the internal-cause mortality results in table 1-7. Hospital admissions and dental visits increase slightly, but the

effects are much smaller than for outpatient physician visits and prescription drugs. Thus, the increased patterns of public health care use correspond to the types of care that were effective in reducing the types of mortality that actually decreased for young children after Medicaid.

## **1.5 Evidence on Potential Threats to Identification**

The strength of the evidence presented above was partly based on the fact that the pre-Medicaid event-study coefficients were close to zero and statistically insignificant, while the post-Medicaid coefficients were negative and precisely estimated. This shows that high- and low-AFDC states were comparable before Medicaid, but diverged only after it was implemented and provides powerful evidence against the typical threat to difference-in-difference models: differential trends in the treatment and control groups. The pattern of estimates by age, race, cause of death, and, for infants, birth order, was also consistent with expectations about the causal effects of Medicaid implementation. The remaining plausible threats to identification, therefore, are variables that affect the mortality of the same groups that gained Medicaid coverage, that differ in high and low-AFDC states, and that change sharply at the same time as Medicaid implementation (but are not caused by it).

### *1.5.1 Direct Evidence on Other Federal Spending*

For example, the level of AFDC receipt in the year of Medicaid implementation may signal states' willingness to change their policies toward the health and mortality of the poor. In this case, the estimates of  $\gamma_y^a$  would capture the mortality-reducing effects of other policies enacted more in high-AFDC states than in low-AFDC states instead of Medicaid's effect. Note that time-invariant differences in such policies will be absorbed by the state fixed effects, and differences that are correlated with Medicaid timing but common within regions will be absorbed by the region-by-year fixed effects.

To test this threat to identification, I use recently collected data on expenditures or participation in federal programs that also expanded in the 1960s. I estimate versions of equation (1) with either per-capita program expenditures or participation rates for four major programs that could have also affected child mortality.<sup>31</sup> Panel A of figure 1-7 shows the results for per-capita federal expenditures (in thousands) for Community Health Centers (CHC), other health programs funded by the Community Action Program (CAP), and Head Start (per 1,000 children ages 1-9). For comparison, I also include public insurance expenditures on children per 1,000 children ages 1-19 (the expenditures version of the first-stage results in figure 1-5). Panel B shows the results of similar regressions for participation rates in the Food Stamp program, and for the white and non-white AFDC rates used to calculate  $D_s$ . For comparison, I reproduce the estimates for public insurance use from figure 1-5.

The estimates for public insurance spending, like the results for public insurance use, show a sharp break after Medicaid implementation and are, on average, about \$35,000 higher for each 1,000 children (DD estimate = 35,319, s.e. = 12,803). Changes in per-capita expenditures on the other programs are small and not strongly correlated with Medicaid implementation, making it unlikely that the expansion of related federal programs can explain the mortality results in section IV.

The DD estimate for community health center spending is statistically significant, but it is more than an order of magnitude smaller than the public insurance estimate. This also overstates the per-capita CHC spending for children because it is an average that includes much higher expenditures for older users. Furthermore, recent work finds no evidence that CHCs affect child

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<sup>31</sup> For the expenditures and participation rates that are not measured by race, the binary AFDC groups are created using the overall AFDC rate (as in the first-stage results) rather than race-specific AFDC rates.

or infant mortality (**Bailey and Goodman-Bacon 2013**), which means that even a large change in funding would not be a plausible explanation for the mortality reductions in section IV.

Panels A and B also show no correlation between the high-AFDC event-time dummies and two programs that have been shown to affect infant and child health: Head Start (**Ludwig and Miller 2007**) and Food Stamps (**Almond, Hoynes, and Schanzenbach 2011**). The results for Head Start in panel A (open circles) are small, indistinguishable from zero, and do not change sharply in the year of Medicaid implementation. The participation results in panel B provide similar evidence for the Food Stamp program (open circles): all of the event-study coefficients are small and the DD point estimate is less than 0.01 (s.e. = 0.01). Therefore, while Head Start and Food Stamps improve health for similar groups documented above, changes in those two programs are uncorrelated with Medicaid implementation and eligibility, and so they cannot account for the estimated Medicaid effects.

Panel B also suggests that changes in AFDC rates themselves cannot explain the mortality results. Neither white nor nonwhite AFDC rates change much on average in high-eligibility versus low-eligibility states (the white DD estimate is 0.001, s.e. = 0.002, and the nonwhite DD estimate is -0.01, s.e. = 0.01). Nonwhite AFDC rates, by virtue of being higher than white AFDC rates, vary more, especially several years after Medicaid nonwhite AFDC rates converge. Nevertheless, unlike public insurance use and mortality rates, the nonwhite AFDC rates do not change sharply at the time of Medicaid implementation, suggesting that they cannot account for the main Medicaid estimates. Furthermore, previous work finds no relationship between welfare receipt and health (**Currie and Cole 1993, Bitler, Gelbach, and Hoynes 2005**), so even if AFDC rates were correlated with Medicaid timing, it is unlikely that this could generate large mortality reductions.

### *1.5.2 Indirect Evidence Adding Controls for State-Level Welfare Programs*

The results in figure 1-7 use the estimator outlined in equation (1) to show that several observable federal programs cannot account for the Medicaid estimates. Another approach to rule out alternative explanations is to add related, time-varying measures of state welfare programs to equation (1). If the results are spurious, then alternative state-level transfer program measures should be highly correlated with any omitted variables that account for the post-Medicaid mortality reductions, and the main treatment effects should fall toward zero.

The first test along these lines exploits the low correlation between white and nonwhite AFDC rates and re-estimates the full DD models for nonwhite mortality rates, but adds interactions of the Medicaid event-time variables with a high-white-AFDC dummy. If high-AFDC states simply expanded their social safety net in ways that increased both white and nonwhite AFDC rates as well as the availability of other (omitted) services that were the true cause of mortality reductions, then the white and nonwhite treatment variables would contain essentially the same information about omitted variables that drive the nonwhite mortality rates. The coefficients on both the white and nonwhite Medicaid interactions would both be negative (although perhaps not significant due to collinearity), which would cast doubt on the main DD estimates. The results in table 1-9 show that the main treatment effects for age-adjusted mortality (ages 0-14), neonatal mortality, and younger child mortality (ages 1-4) are unchanged and the effects in high-white-AFDC states are small and insignificant (see Appendix B for event-study results). The robustness of the nonwhite effects and the insignificance of the white effects show that the main results are not driven by other welfare measures correlated with Medicaid timing, but instead come from specific measures of the relevant categorical eligibility rate.

The history of AFDC, however, suggests that white and nonwhite participation represent different omitted factors (see section III.B), so using white AFDC rates may not capture omitted determinants of mortality that are correlated specifically with nonwhite AFDC and state-level Medicaid adoption. Because the identification strategy is based on nonwhite AFDC rates at one point in (event) time, I can address this concern by including the actual state-by-year nonwhite AFDC rate as an additional covariate in equation (1). If a relevant omitted variable is correlated with nonwhite AFDC rates in the years surrounding Medicaid implementation, then including the nonwhite AFDC rate will eliminate the treatment effects that come from the interactions between Medicaid timing and  $D_s$ . Table 1-10 presents DD estimates that include the state-by-year nonwhite AFDC rate and its interaction with a post-1966 dummy, which explicitly allows the nonwhite AFDC controls to have different effects after the mid-1960s (**Moffitt 1987**). The treatment effects are only slightly reduced by these flexible controls for nonwhite state-by-year AFDC rates. The DD estimate for age-adjusted mortality falls only negligibly to -0.07 (s.e. = 0.04, p-value = 0.06) from -0.08 in table 1-3; the estimate for neonatal mortality is actually slightly larger (-0.09, s.e. = 0.03) than in column 4 of table 1-4; and the estimate for younger children falls from -0.13 in table 1-6 to -0.10 (s.e. = 0.05). Thus, even flexible controls for nonwhite AFDC rates do not eliminate the relationship between Medicaid implementation and nonwhite categorical eligibility.

## **1.6 Interpreting the Mortality Effects of Medicaid Implementation**

The preceding evidence suggests that Medicaid implementation was very successful at increasing public insurance coverage and reducing mortality among children. But given that previous studies have estimated effects for similar populations, what do these results mean for our understanding of how public insurance influences mortality generally? Here I argue that



these results make two main contributions. First, they provide the strongest evidence to date that Medicaid coverage itself reduces the mortality of its largest group: poor children and infants. Second, the results are the first to demonstrate that Medicaid implementation, the largest-ever change to public health insurance for low-income families, played an important role in aggregate changes in mortality rates and racial gaps in mortality.

### *1.6.1 The Average Treatment Effect of Medicaid on the Mortality of Treated Children*

Section I argued that previous estimates of Medicaid's effect on infant and child mortality were too large to be attributed to insurance coverage alone. This conclusion is based on the 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 and because a proportional effect is easier to compare across time periods with different underlying mortality rates. It is also a useful check on the plausibility of attributing a given result entirely to changes in insurance because the proportional ATET cannot be below -100%, as this implies that Medicaid reduces mortality by more than the 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.<sup>32</sup> This assumes that no categorically eligible Medicaid recipients dropped private insurance coverage, a common concern for later expansions. This is almost certainly true given low levels of private insurance in the 1960s, especially among AFDC recipients, who were employed less than 5 percent of the time (DHEW 1961). The limited scope for crowd-out, combined with the lack of care documented above,

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<sup>32</sup> In these calculations I use the estimates from the continuous AFDC specification shown in appendix 2. The reduced-form and first-stage effects from this specification are per percentage point of the AFDC rate, and so no rescaling of the first-stage (for all children) is required to make it comparable to the mortality effects for nonwhite children. Appendix 2 shows that the first stage estimate from this specification is 3.32 (s.e.=1.03), and the estimate for age-adjusted mortality is -1.45 (s.e.=0.42).

suggest that increases in income due to Medicaid coverage are less likely to account for the results than in more recent expansions.<sup>33</sup>

Because the mortality rates of the populations eligible for Medicaid are higher than the average, I make an additional adjustment based on the ratio of poor to overall mortality rates. Appendix D provides additional details on the calculation of the ATETs and the data sources for mortality rates by income.<sup>34</sup>

The point estimates and confidence intervals for the ATET from this paper and from the three most closely related Medicaid papers (**Currie and Gruber 1996a, b, Meyer and Wherry 2013**) are shown in figure 1-8. I construct confidence intervals using a parametric bootstrap procedure (**Efron and Tibshirani 1993**). I create 10,000 draws for the reduced-form and first-stage estimates using a normal distribution with means and standard errors taken from the relevant table, and the ATET estimate is calculated for each draw.<sup>35</sup> (Details on the bootstrap parameters and the resampling of the mortality adjustment are in appendix D.) Because the ATET is a ratio with a denominator that is close to zero for many draws, its empirical distribution is not symmetric. I calculate confidence intervals using a modified percentile method (**Johnston and DiNardo 1997**), and their resulting asymmetry is clear in figure 1-8.

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<sup>33</sup> Currie and Gruber (1996a) show that all children *and* Medicaid children in their sample went without a doctor visit in the previous year about 19 percent of the time. The corresponding figure for lower-income children in 1963 is 55 percent.

<sup>34</sup> Intuitively, imagine a policy that reaches half the population and reduces aggregate mortality (MR) by 10 percent. This can be expressed as the treated share times the change in mortality in the treated group  $\Delta MR^T$  divided by the aggregate mortality rate:  $\frac{\Delta MR}{MR} = \frac{\Delta MR^T}{MR} \cdot 0.5 + \frac{0}{MR} \cdot 0.5 = -0.1$ . This implies that the mortality change in the treatment group divided by the *overall* mortality rate is -20 percent. But if the underlying mortality rate in the treated population ( $MR^T$ ) differs from the average ( $MR$ ), this is not the proportional effect on their mortality. The proportional treatment effect equals  $\frac{\Delta MR^T}{MR^T} = -\frac{0.1}{0.5} \cdot \frac{MR}{MR^T}$ ; the reduced-form estimate, divided by the first-stage estimate, divided by the ratio of treated to overall mortality.

<sup>35</sup> When calculating the ATET for more recent papers, I use first-stage estimates for *any* health insurance rather than only Medicaid coverage and I adjust them by a factor of 0.85 to account for underreporting of Medicaid in most major surveys (Card, Hildreth, and Shore-Sheppard 2004, Davern, Klerman, and Ziegenfuss 2007).

Note that this method allows me to calculate confidence intervals for other papers without resampling from their data.

The ATET estimates reaffirm that Medicaid had a significant negative effect on nonwhite infant and child mortality rates, and the magnitudes suggest that the results could be due to Medicaid's insurance coverage alone. The ATET is -24 percent for nonwhite children under 14, -40 percent for younger nonwhite children (ages 1-4) and -32 percent for nonwhite neonates. The confidence intervals never cover zero, and for both the overall and neonatal estimates, the lower end of the confidence interval does not cross the maximum possible value, -100%. The comparable results for the effects of the 1980s expansions on children and infants, on the other hand, yield ATETs that are nearly five times as large. The implied ATET for infants is -182, and for children it is -190 percent. I can easily rule out values of this size for my ATET estimates. This is surprising since the AFDC children who gained insurance because of Medicaid implementation were much poorer and, therefore, less healthy than many of the children and infants who gained coverage in the 1980s. Nevertheless, magnitude of the ATET estimates means that these effects could be attributed to Medicaid coverage itself.

An additional point of comparison is the results in **Almond, Chay, and Greenstone (2006)** who find that desegregation of hospitals in the rural South reduced nonwhite post-neonatal mortality by about 50 percent. This effect is relatively close to my estimates for neonatal mortality (-32 percent), which is reassuring since desegregation and Medicaid coverage meant something similar for poor mothers: improved hospital access. Also note that desegregation itself is not a likely mechanism for my results because it implies relative increases in access to medical care for black families in the most segregated states, which are also

primarily low-AFDC states. In other words, the effects of desegregation should have been strongest in my “control” group, which would bias the Medicaid estimates toward zero.

The ATET estimates in figure 1-8 show that this paper’s results are among the smallest but most precisely estimated quasi-experimental estimates of Medicaid’s effect on mortality. The magnitudes also suggest that these are the only quasi-experimental estimates of Medicaid’s effect on infant and child mortality that can possibly be attributed to insurance coverage itself. Furthermore, the results are small enough that they could not have been detected even in the experimental design of the OHIE. Under the extreme assumption that the effect of insurance on mortality is the same for poor children and adults, my results are among the only ones that do not conflict with experimental evidence.

### *1.6.2 Medicaid’s Aggregate Costs and Benefits*

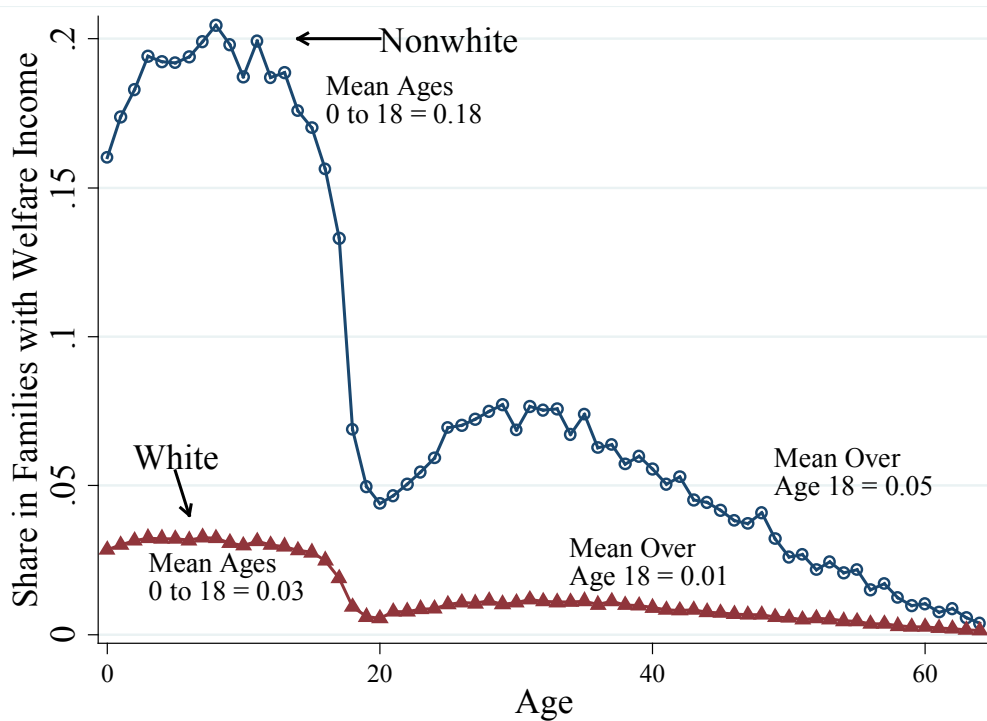
While these estimates are significantly smaller than those in the existing Medicaid literature, they still imply that Medicaid implementation played a major role in aggregate mortality changes in the 1960s and 1970s. A proportional effect per nonwhite Medicaid child of -24 percent, combined with a national share of nonwhite children on Medicaid of about 33 percent (the product of the national child Medicaid share, 12 percent, and the ratio of nonwhite to overall Medicaid use rates in the 1976 SIE, 2.7), suggests that Medicaid reduced aggregate annual nonwhite mortality rates by 8 percent in each of its first 10 years. I also use the ATET estimates to construct a counterfactual level of nonwhite child mortality and a counterfactual white/nonwhite mortality gap. This calculation suggests that, without Medicaid, the racial gap in child mortality in 1979 would have been 116 deaths per 100,000 children. The actual gap was 99 deaths per 100,000, and so by this calculation, Medicaid reduced the 1979 racial gap in child mortality by 15 percent (-18 deaths per 100,000 relative to a counterfactual gap of 116).

These calculations all refer to Medicaid's effect on period mortality rates, while the actual benefits accrued over time. Comparing the observed number of nonwhite child deaths to the counterfactual number in each year suggest that, between 1966 and 1979, 25,000 nonwhite deaths were averted due to Medicaid. Because most of these deaths would have occurred among neonatal infants and young children, the total number of life-years saved is much higher. The remaining life expectancy for a nonwhite child between ages 1-5 in 1966 was 65.5 years (NCHS 1967). Thus, a reduction in 25,000 deaths corresponds, roughly, to 1.64 million life-years saved. The public insurance data show that, through 1976, Medicaid spent about \$38 billion (in 2012 dollars) on all children. Dividing this expenditure by the number of nonwhite deaths averted by 1976 (20,189) shows that the cost per death is about \$1.9 million, and the cost per life-year saved is about \$29,000. The comparable estimate from **Currie and Gruber (1996b)** (adjusted to 2012 dollars) is about \$1.7 million per death averted. The expenditures used in this calculation, however, come from all children, while the mortality effects come only from younger nonwhite children, so the cost per life (and life-year) saved in terms of the actual spending on this group is much smaller.

This paper provides new evidence on the relationship between Medicaid and mortality using the original introduction of the program between 1966 and 1970. An especially vulnerable group, nonwhite infants and children, used Medicaid the most, and the results show that the majority of the mortality effects for children accrued to this population. While more recent policy changes have had similar qualitative effects on infant and child mortality, this paper's estimates are among the only quasi-experimental results that are small enough to be attributed to Medicaid coverage itself.

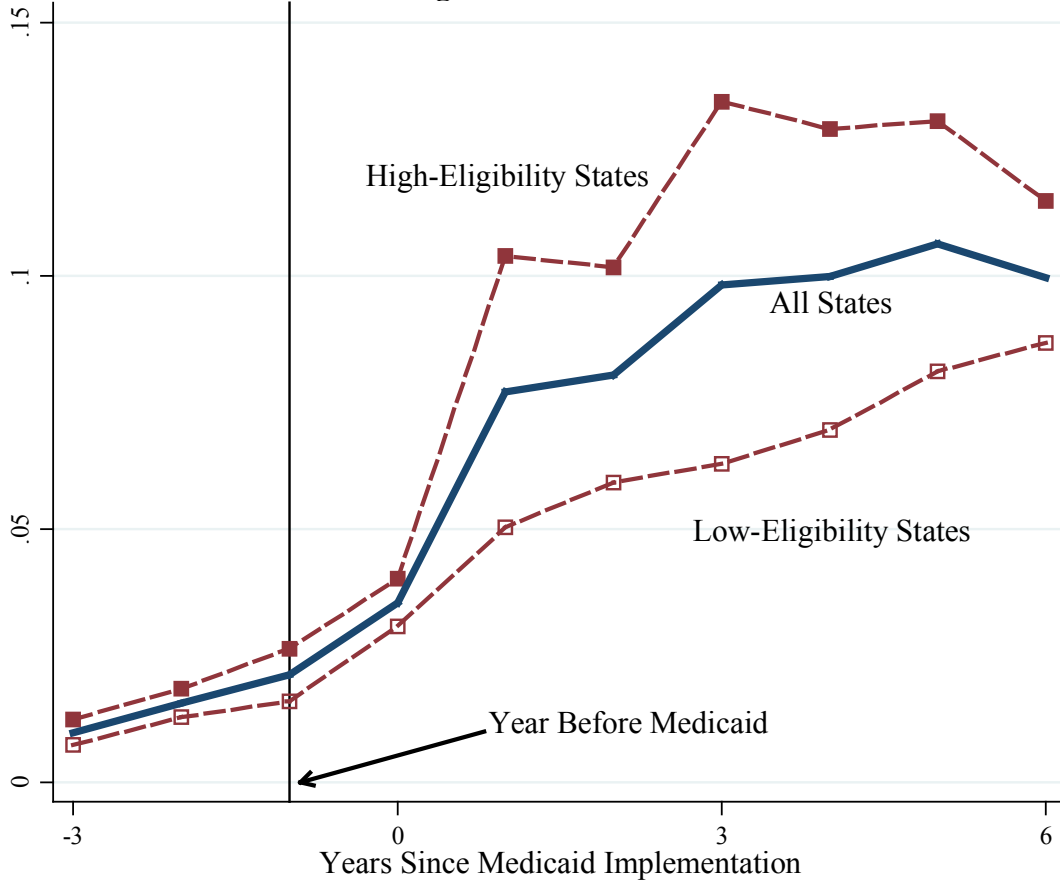
These estimates are also the first to demonstrate that Medicaid implementation was effective at improving infant and child health. These findings presumably understate Medicaid's broader effects because they only measure benefits in terms of mortality rather than reductions in morbidity. Therefore Medicaid, like several other federal health and anti-poverty programs established under the Great Society, played a central role in reducing racial and socioeconomic disparities in health and mortality in the 1960s and 1970s.

**Figure 1-1 Medicaid Categorical Eligibility: The Rate of AFDC Receipt by Age and Race, December 1967**



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

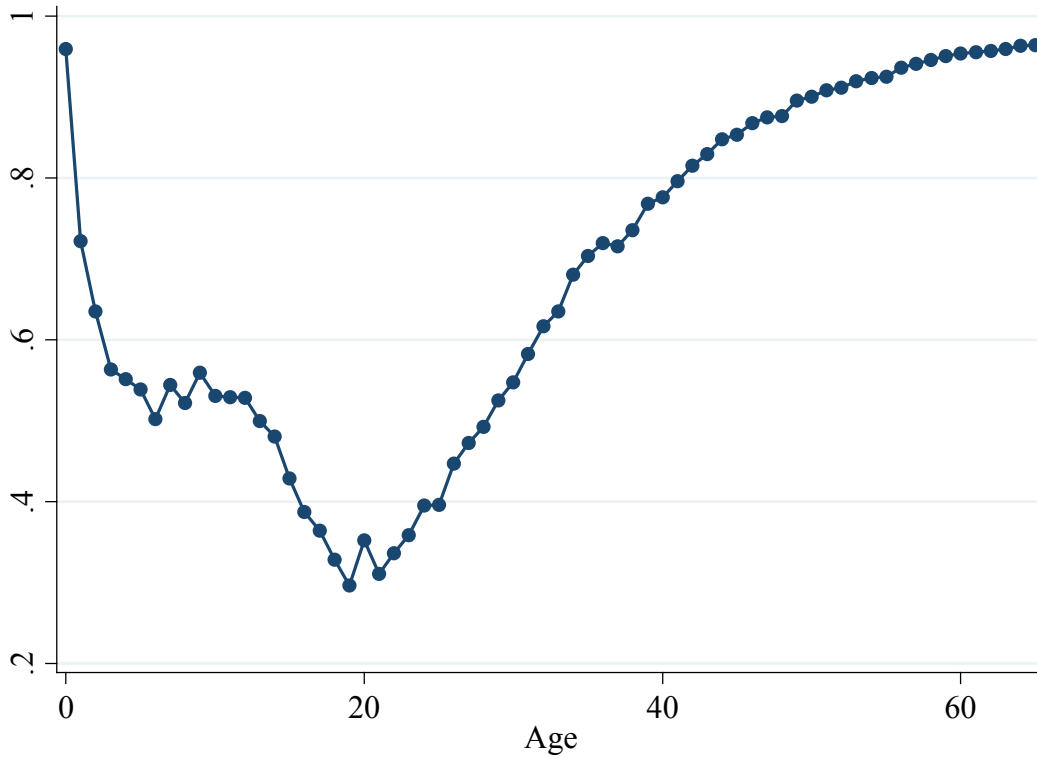
**Figure 1-2 The Share of Children Using Public Health Insurance before and after Medicaid**



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

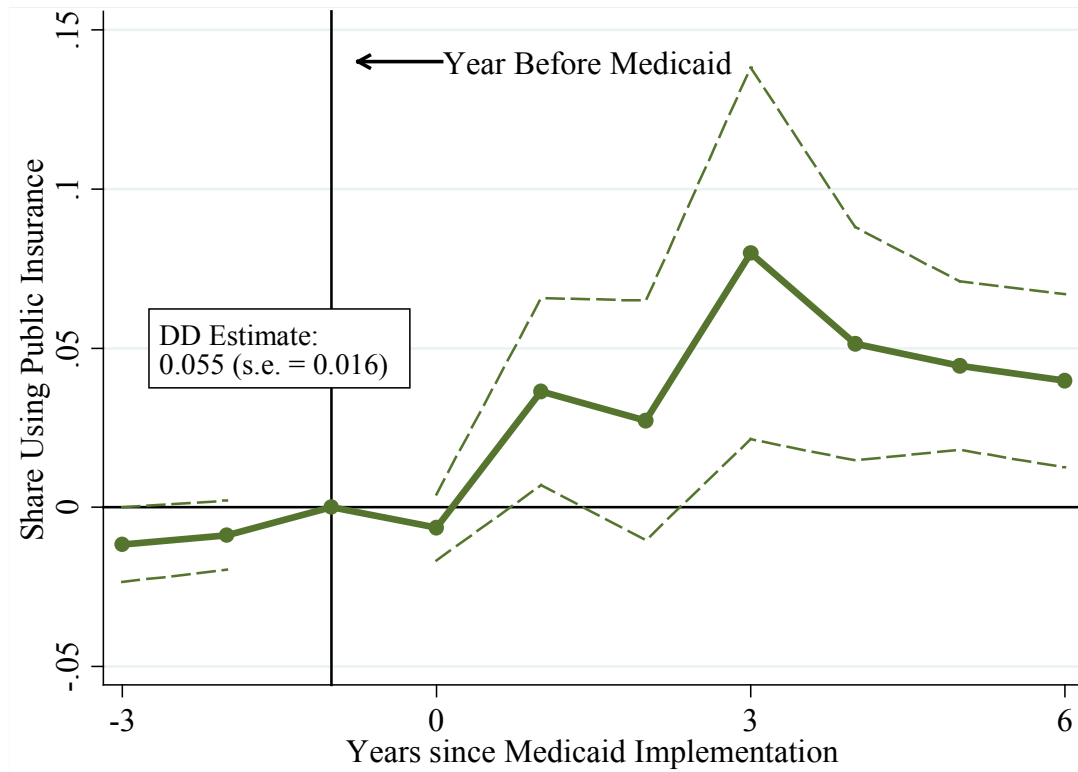


**Figure 1-3 Share of Deaths Due to Internal Causes by Age, 1965**



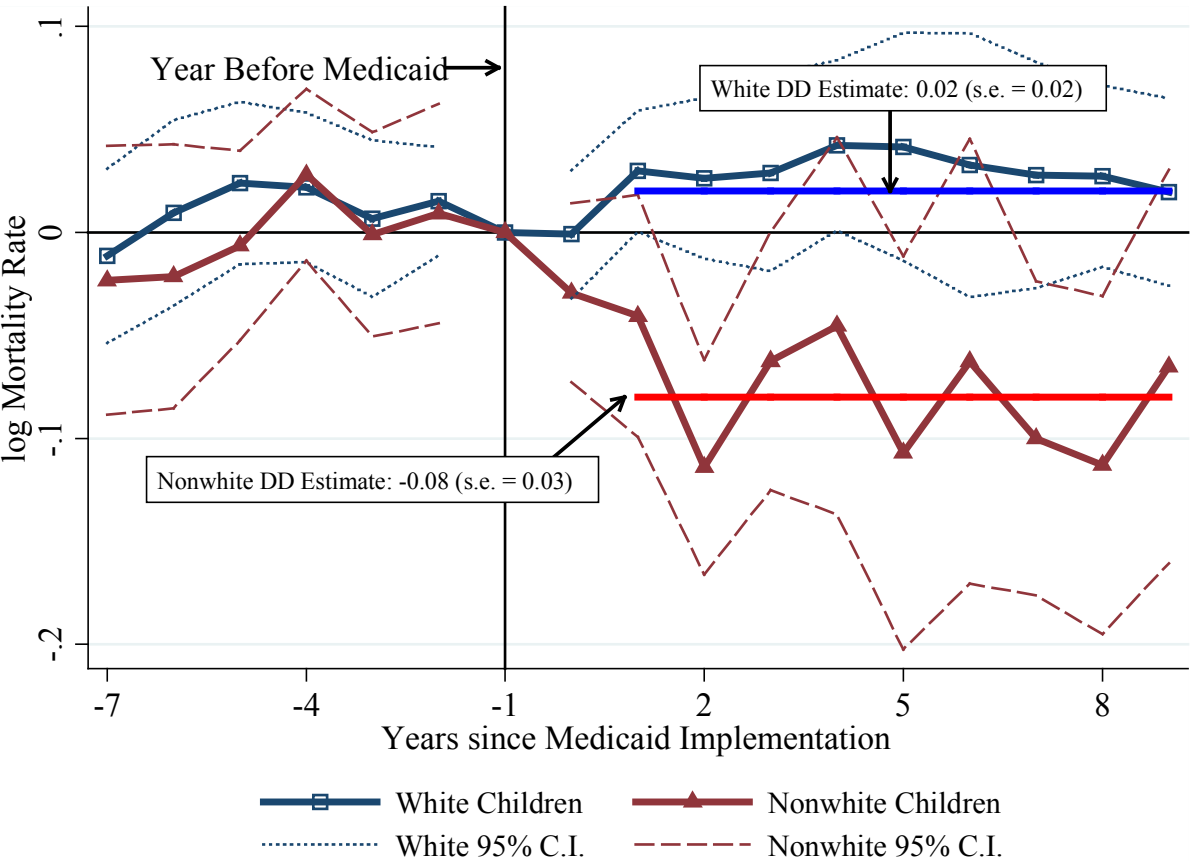
Notes: The figure plots the share of deaths at each single age due to internal causes of death. Internal causes include all deaths not due to “external” causes in the International Classification of Diseases Revision 7 (ICD7 codes E800-E999). Source: Vital Statistics Multiple-Cause of Death File, 1965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

**Figure 1-4 Regression-Adjusted Estimates of Medicaid’s Effect on Children’s Public Insurance Use**



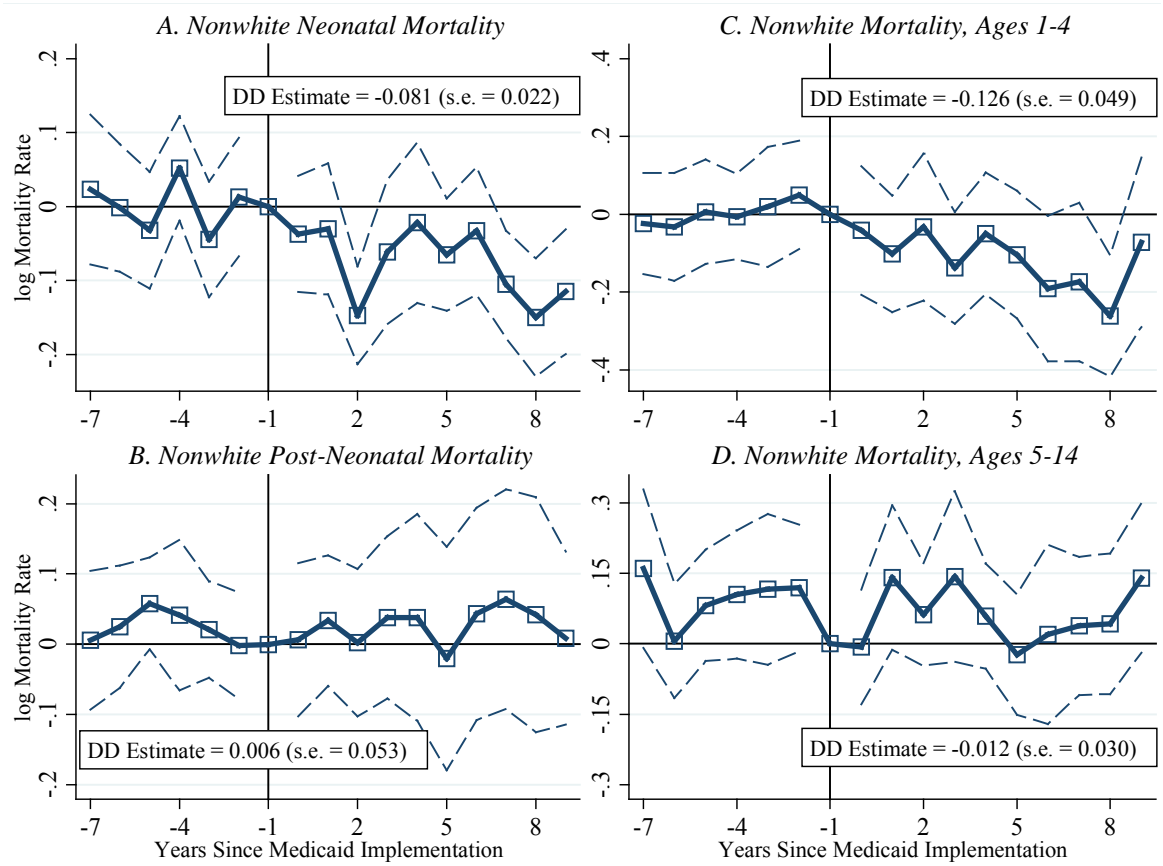
Notes: The dependent variable is the estimated share of children ages 0-21 that received services covered by a means-tested public insurance program. The figure plots the estimated coefficient on interactions between time-to-Medicaid dummies ( $1\{t - t_s^* = \gamma\}$ ) and a dummy variable for high-AFDC states ( $D_s$ ) in a regression model described in Section III. The year before Medicaid implementation is omitted so the estimates are normalized to zero in that year. The model also includes state fixed effects, per-capita income and hospital capacity variables, region-by-year fixed effects, and separate year fixed effects for each Medicaid timing group. The dashed lines are pointwise 95 percent confidence intervals based on standard errors clustered at the state level. The sample includes 618 state-year observations that have non-missing values for public insurance use between 1963 and 1976. The post-Medicaid coefficients are jointly significant at the 5% level (p-value = 0.03), and the pre-Medicaid coefficients are not (p-value = 0.15). The estimates are weighted by state populations aged 0-19, but a Hausman test cannot reject the null hypothesis that the weighted and unweighted estimates are equal (p-value = 0.78; Deaton 1997; Solon, Haider, and Wooldridge 2013). Source: See Appendix A.

**Figure 1-5 Regression-Adjusted Estimates of Medicaid’s Intention-to-Treat Effect on Child Mortality by Race**



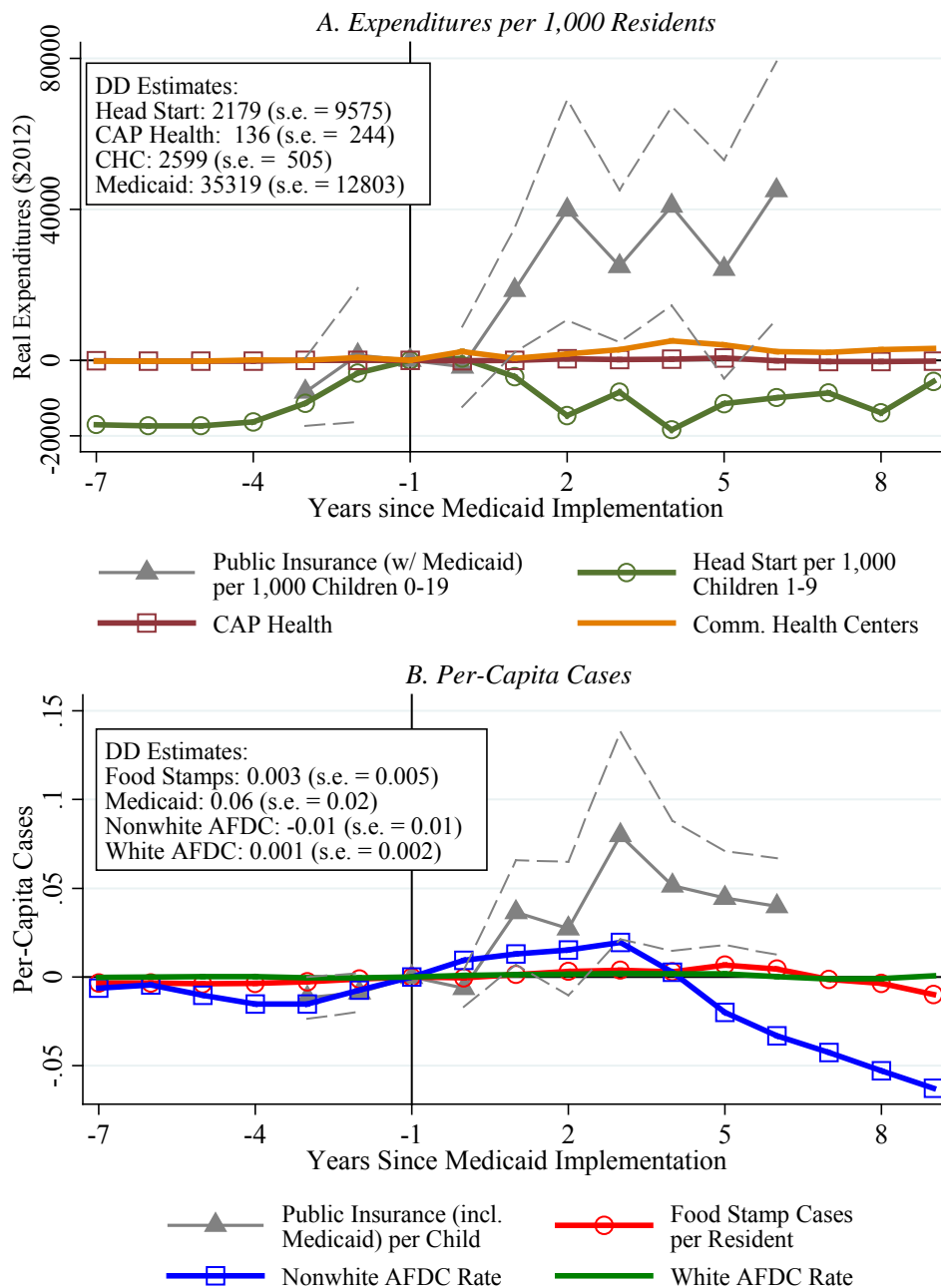
Notes: The dependent variable is the natural log of the age-adjusted mortality rate among children aged 0-14. The figure plots the estimated coefficients on interactions between time-to-Medicaid dummies ( $1\{t - t_s^* = y\}$ ) and a dummy variable for high-AFDC states ( $D_s$ ) in the regression model described in Section III. The year before Medicaid implementation is omitted so the estimates are normalized to zero in that year. States observed more than seven years before Medicaid (the latest-implementing states) or more than 10 years after (the earliest implementing states) are grouped into endpoint dummies and their coefficients are not shown (see McCrary 2007). High- and low-AFDC states are defined by the median race-specific AFDC rate as in table 1-1. In addition to the variables of interest—interactions between time-to-Medicaid dummies and a high-AFDC indicator ( $D_s 1\{t - t_s^* = y\}$ )—the model includes state fixed effects, separate year fixed effects for each Medicaid timing group, per-capita income and hospital variables, and region-by-year fixed effects. The broken lines are pointwise 95 percent confidence intervals based on standard errors clustered at the state level. The white sample includes 987 state-year observations between 1959 and 1979 for 47 states (Arizona, Alaska, Hawaii, and New Jersey are omitted). The nonwhite sample (924 observations) also excludes New Hampshire, Vermont and Maine because less than one percent of their children are nonwhite. The post-Medicaid coefficients for nonwhite children are jointly significant below the 1% level (p-value = 0.0001), and the pre-Medicaid coefficients are not jointly distinguishable from zero (p-value = 0.33). Neither the pre- nor the post-Medicaid coefficients for white children are distinguishable from zero (p-values = 0.44 and 0.57, respectively). A joint test of the difference-in-difference assumption ( $H_0$ : pre-Medicaid coefficients equal zero and the post-Medicaid coefficients are equal to each other), rejects the null hypothesis for the nonwhite estimates (p-value = 0.003), but not for the white estimates (p-value = 0.5). A Hausman test rejects the equality of weighted and unweighted nonwhite estimates, although the point estimates are quite similar, and the slightly more restrictive specifications presented in table 1-3 cannot detect differences between weighted and unweighted estimates. See Appendix B for unweighted event-study results. Source: Mortality data are from Vital Statistics Multiple-Cause of Death Files 1959-1979 (US DHHS and NCHS 2009). Population denominators are from the 1950 and 1960 Censuses (Haines and ICPSR 2005) and the 1969 to 1988 Surveillance Epidemiology and End Results (SEER 2009).

**Figure 1-6 Regression-Adjusted Estimates of Medicaid’s Effect on Nonwhite Mortality by Age Group for Infants and Children**



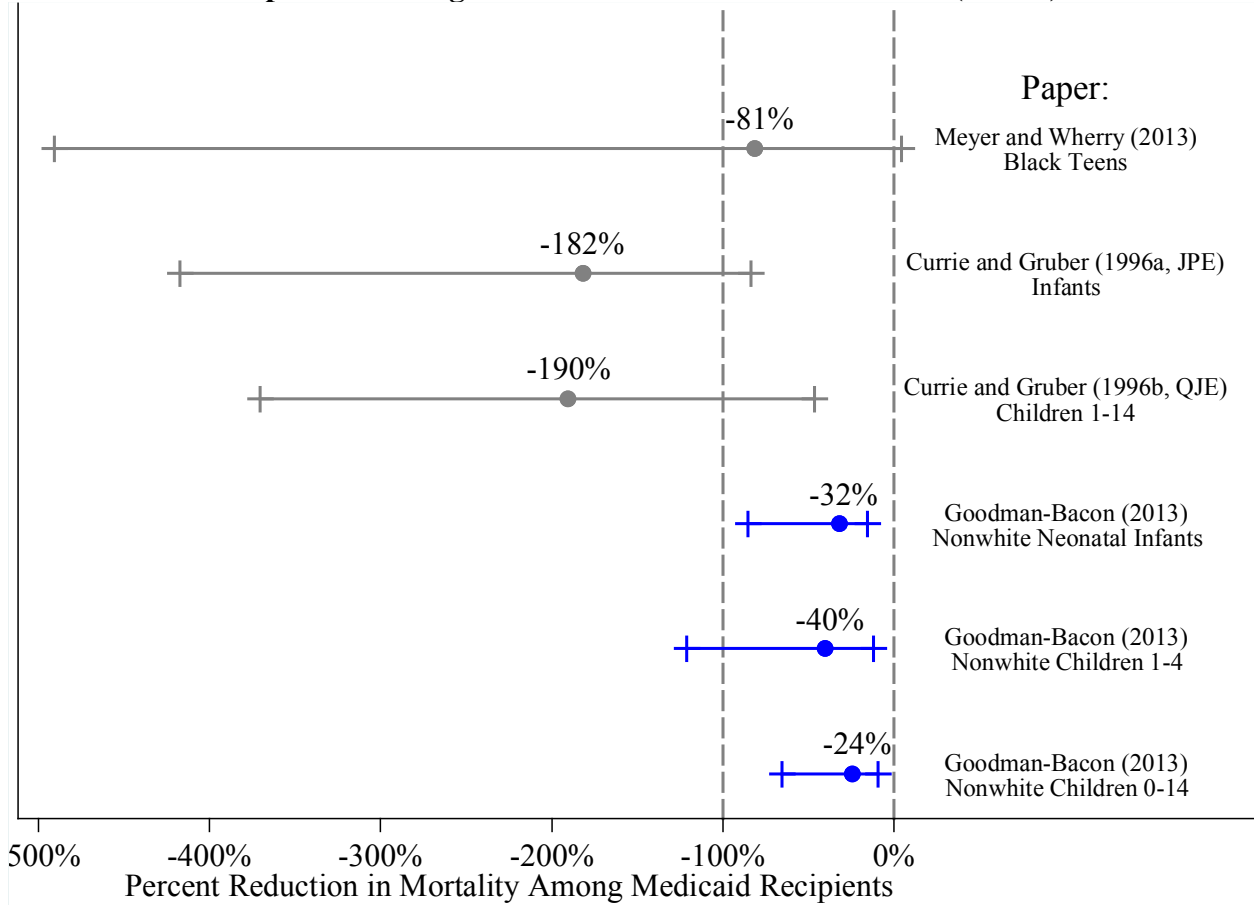
Notes: For details on the specification see notes to figure 1-5. The samples are the same as in figure 1-5 except that some cells have zero deaths and are missing for the log mortality rates. This eliminates 42 observations from panel A (N=882), 21 from panel B (N=903), 8 from panel C (N=916), and 16 from panel D (N=908). The dependent variables are the log nonwhite mortality rates for the indicated group. The neonatal mortality rate is defined as the number of deaths in the first 28 days of life per 1,000 births, the post-neonatal mortality rate is the number of deaths between 28 days and 1 year per 1,000 live births, and age-adjusted mortality rates for young children (1-4) and older children (5-14) are measured per 100,000 children and are calculated as described in the text. p-values from F-tests of the joint significance of the pre- and post-Medicaid coefficients are 0.007 and <0.01 for neonatal mortality; 0.45 and 0.71 for post-neonatal mortality; 0.88 and 0.007 for young child mortality; and 0.77 and 0.26 for older child mortality. A joint test of the difference-in-difference assumption (H0: pre-Medicaid coefficients equal zero and the post-Medicaid coefficients are equal to each other) rejects the null hypothesis for neonatal mortality (panel A, p-value = <0.01), younger child mortality (panel C, p-value = 0.015), and older child mortality (panel D, p-value <0.01), but not for the post-neonatal mortality (p-value = 0.345). Source: See notes to figure 1-5.

**Figure 1-7 The Relationship between Medicaid Implementation and Other Health-Related Programs**



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

**Figure 1-8 The Proportional Effects of Medicaid on the Mortality Rates of Newly Insured Recipients: Average Treatment Effects on the Treated (ATET)**



Notes: The figure plots the implied average treatment effects on the treated (ATET) for three comparable previous studies and for the DD results emphasized in this paper: all nonwhite children ages 0-14, nonwhite neonatal infants, and younger nonwhite children ages 1-4. To calculate the ATET, the reduced form ITTs are expressed as proportional changes in mortality rates, then divided the reduced form estimates by a first-stage estimate for any insurance coverage (adjusted for under-reporting when appropriate), then adjusted for differential baseline mortality among poorer Medicaid recipients. The vertical dashed lines represent zero, indicating that Medicaid does not reduce mortality, and -100%, which is the largest possible value of the ATET because a group's mortality rate cannot be reduced by more than its baseline level. For my ATET estimates I used the continuous specification of the first-stage and reduced form equations shown in appendix figure B-5 and appendix table B-3. They imply a similar ATET as the binary specification but are more precisely estimated as noted in the text. This is the point estimate of the ATET labeled on the figure. See appendix D for details on the scaling and the data sources. The confidence intervals are generated using a modified percentile method from 10,000 replications of a parametric bootstrap procedure (Johnston and DiNardo 1997, Efron and Tibshirani 1993, Valetta 1993). I generate bootstrap draws of the reduced-form and first-stage coefficients from normal distributions with means and standard errors equal to the point estimates 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.

**Table 1-1 High-Eligibility and Low-Eligibility Groups by Race, Year of Medicaid Implementation**

		White AFDC Rate	
		Low	High
Nonwhite AFDC Rate	Low	13 States: District of Columbia, Georgia, Illinois, Louisiana, Maryland, Michigan, New Hampshire*, North Carolina, Ohio, South Carolina, Texas, Virginia, Wisconsin	11 States: Idaho, Kentucky, Maine*, New Mexico, Oregon, Pennsylvania, Tennessee, Vermont*, Washington, West Virginia, Wyoming
	High	11 States: Arkansas, Connecticut, Delaware, Florida, Indiana, Kansas, Mississippi, Montana, Nebraska, Nevada, North Dakota	15 States: California, Alabama, Colorado, Iowa, Massachusetts, Minnesota, Missouri, New Jersey*, New York, Oklahoma, Rhode Island, South Dakota, Utah

Notes: \*Maine, New Hampshire, and Vermont are excluded from the nonwhite estimation sample because less than 1 percent of their populations were nonwhite. New Jersey is excluded from all models because it lacks race codes in its mortality files for 1962 and 1963. Alaska, Hawaii, and Arizona are also excluded from all models.

**Table 1-2 Balancing Test: The Relationship between Pre-Medicaid State Characteristics and AFDC-Based Categorical Eligibility**

	(1)	(2)	(3)
	Low-AFDC	High-AFDC	H0: (1)=(2) (p-value, t-test)
<i>A. By White AFDC Rate, t*</i>			
1) White Child Poverty Rate, 1960	0.22 (0.10)	0.24 (0.09)	0.41
2) White Children in Single Mother Households, 1960	0.026 (0.01)	0.033 (0.01)	0.01
3) Average White AFDC Benefit, 1967	133 (47.4)	161 (41.5)	0.04
4) Change in White Child Poverty Rate, 1950 to 1960	-0.14 (0.07)	-0.14 (0.05)	0.98
5) Change in White Child Mortality Rate, t*-5 to 5	-0.13 (0.08)	-0.13 (0.07)	0.77
6) Change in White Infant Mortality Rate, t*-5 to 5	-0.09 (0.10)	-0.08 (0.07)	0.61
<i>B. By Nonwhite AFDC Rate, t*</i>			
7) Nonwhite Child Poverty Rate, 1960	0.65 (0.20)	0.62 (0.17)	0.53
8) Nonwhite Children in Single Mother HH, 1960	0.16 (0.05)	0.15 (0.05)	0.44
9) Average Nonwhite AFDC Benefit, 1967	146 (38.44)	159 (58.75)	0.38
10) Change in Nonwhite Child Poverty Rate, 1950 to 1960	-0.08 (0.15)	-0.15 (0.16)	0.12
11) Change in Nonwhite Child Mortality Rate, t*-5 to 5	-0.12 (0.19)	-0.07 (0.19)	0.40
12) Change in Nonwhite Infant Mortality Rate, t*-5 to 5	-0.14 (0.15)	-0.10 (0.17)	0.38
<i>C. Overall AFDC Rate, t*</i>			
13) Change in Hospital Beds per Capita, 1960-1965	38.15 (17.01)	43.78 (19.12)	0.29
14) Change in Child Public Insurance Rate, 1963-1965	0.01 (0.01)	0.01 (0.02)	0.68

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



**Table 1-3 Medicaid's Effect on Log Nonwhite Age-Adjusted Child Mortality Across Specifications**

	(1)	(2)	(3)	(4)
<i>A. Grouped Event-Study Estimates</i>				
<i>Pre-Medicaid</i>				
(Years -7 to -2)*High-AFDC	0.02 [0.03]	0.01 [0.03]	-0.003 [0.022]	-0.04 [0.04]
<i>Post-Medicaid</i>				
(Year 0)*High-AFDC	-0.02 [0.02]	-0.03 [0.02]	-0.03 [0.02]	-0.02 [0.11]
(Years 1 to 4)*High-AFDC	-0.06 [0.02]	-0.05 [0.03]	-0.07 [0.03]	-0.11 [0.05]
(Years 5 to 9)*High-AFDC	-0.11 [0.04]	-0.09 [0.06]	-0.09 [0.04]	-0.14 [0.05]
R2	0.74	0.94	0.96	0.84
<i>B. Difference-in-Differences Estimates</i>				
Post-Medicaid*High-AFDC	-0.10 [0.04]	-0.08 [0.05]	-0.08 [0.03]	-0.09 [0.03]
R2	0.67	0.94	0.96	0.84
DD Test (p-value)	0.18	0.58	0.60	0.55
Covariates	High-AFDC FE, Time-to- Medicaid Dummies	State FE, Medicaid- Timing-by- Year FE + Xst	(2) + region- by-year FE	(3), unweighted
Population Weighted?	Y	Y	Y	N

Notes: Panel A contains the estimated coefficient on interactions between groups of time-to-Medicaid dummies ( $1\{t - t_s^* \in [a, b]\}$ ) and a dummy variable for high-AFDC states ( $D_s$ ) from five specifications of the regression model described in Section III. Standard errors clustered at the state-level are in brackets. The results correspond to the event-study results in figure 1-5. I group the pre-Medicaid period (from seven to two years before Medicaid), the first year during which Medicaid programs were only partially in place, years 1-4 and years 5-9. The year before Medicaid implementation is omitted so the estimates are normalized to zero in that year. States observed more than seven years before Medicaid (the latest-implementing states) or more than 9 years after (the earliest implementing states) are grouped into endpoint dummies and their coefficients are not shown (see McCrary 2007). The sample contains 924 observations on 43 states (excluding Alaska, Hawaii, Arizona, New Jersey, New Hampshire, Vermont and Maine) between 1959 and 1979. The row labeled “DD Test” contains the p-value from an F-test of the difference-in-differences restrictions: the pre-Medicaid coefficient is zero and post-Medicaid coefficients (not including year zero) are equal to each other. Failure to reject these restrictions suggests that estimating a constant treatment effect (for time 1-9) and forcing the pre-Medicaid period (-7 to -1) to equal zero are valid restrictions. The estimates of this specification are presented in panel B. The covariates are the same as in panel A except that the (Years -7 to -2)\*High-AFDC variable is omitted, a dummy for the year of Medicaid implementation is included (but not shown), and Post-Medicaid\*High-AFDC refers to all post-Medicaid years between 1 (the year after implementation) and 9. Although the unweighted estimates in column (4) show larger reductions in mortality than the weighted estimates in column (3), a Hausman test does not reject the null hypothesis that they are equal for either the grouped event-study model (p-value = 0.34) or the DD model (p-value = 0.81) (Deaton 1997; Solon, Haider and Wooldridge 2013). Sources: See notes to figure 1-5.

**Table 1-4 Medicaid’s Effect on Log Nonwhite Neonatal Mortality Across Specifications**

	(1)	(2)	(3)	(4)
<i>A. Grouped Event-Study Estimates</i>				
<i>Pre-Medicaid</i>				
(Years -7 to -2)*High-AFDC	0.00 [0.03]	0.01 [0.04]	-0.002 [0.035]	-0.001 [0.068]
<i>Post-Medicaid</i>				
(Year 0)*High-AFDC	-0.03 [0.02]	-0.04 [0.03]	-0.04 [0.04]	-0.11 [0.08]
(Years 1 to 4)*High-AFDC	-0.06 [0.02]	-0.03 [0.03]	-0.07 [0.04]	-0.08 [0.09]
(Years 5 to 9)*High-AFDC	-0.13 [0.03]	-0.08 [0.02]	-0.10 [0.03]	-0.07 [0.06]
R2	0.67	0.92	0.93	0.79
<i>B. Difference-in-Differences Estimates</i>				
Post-Medicaid*High-AFDC	-0.09 [0.03]	-0.06 [0.03]	-0.08 [0.02]	-0.07 [0.04]
R2	0.60	0.92	0.93	0.79
DD Test	0.00	0.07	0.29	0.99
Covariates	High-AFDC FE, Time-to-Medicaid Dummies	State FE, Medicaid-Timing-by-Year FE + Xst	(2) + region-by-year FE	(3), unweighted
Population Weighted?	Y	Y	Y	N

Notes: For details on dependent variables see notes to figure 1-6, for details on specification and sources see notes to table 1-3. A Hausman test cannot reject the null hypothesis that the weighted and unweighted estimates in columns 3 and 4 are equal for either the grouped event-study model (p-value = 0.44) or the DD model (p-value = 0.54) (Deaton 1997; Solon, Haider, and Wooldridge 2013).

**Table 1-5 Medicaid's Effect on Nonwhite Birth Outcomes by AFDC Coverage, 1964-1972**

	(1)	(2)	(3)
Coefficient on High-AFDC*Post-Medicaid	Low Birth Weight P(Birthweight < 2500 grams)	Prematurity P(Gestational Age < 36 Weeks)	Birth in an Institution
Covered Births:			
Poor	-0.004 [0.052]	-0.076 [0.026]	0.065 [0.027]
Non-Poor	0.007 [0.06]	-0.006 [0.059]	-0.048 [0.034]
Non-Covered Births:			
Poor	-0.003 [0.085]	-0.090 [0.118]	0.012 [0.044]
Non-Poor	-0.013 [0.088]	-0.010 [0.088]	0.041 [0.029]
Observations	3,821	3,630	3,821
R2	0.11	0.08	0.20

Notes: The table contains estimated coefficients from a linear probability model that contains interactions between (1) a dummy that equals one for all years after (but not including) the year of Medicaid implementation, (2) an indicator for high-AFDC states and (3) indicators for whether mothers were poor/non-poor and covered/not covered by AFDC. Most states did not provide AFDC and, therefore, Medicaid coverage to first-time pregnant women. The definition of AFDC coverage in these results is a subsequent birth or a first birth in a state that provided AFDC to first-time pregnant mothers. The coefficients represent separate DD estimates for each of the four groups defined by poverty and AFDC coverage. The model also includes state fixed effects, separate year fixed effects for each Medicaid timing group, region-by-year fixed effects and dummies for 10 bins of family income interacted with year dummies, dummies for each year of the mother's age, an indicator for the sex of the child, and an indicator for plural births. Standard errors clustered at the state level are in brackets. The regressions are weighted by the sampling weights. Source: National Natality Followback Surveys 1964-1966 and 1972, National Natality Surveys 1967-1969.

**Table 1-6 Medicaid's Effect on Log Nonwhite Child Mortality Ages 1-4 Across Specifications**

	(1)	(2)	(3)	(4)
<i>A. Grouped Event-Study Estimates</i>				
<i>Pre-Medicaid</i>				
(Years -7 to -2)*High-AFDC	0.09 [0.05]	0.02 [0.04]	0.003 [0.053]	-0.06 [0.16]
<i>Post-Medicaid</i>				
(Year 0)*High-AFDC	-0.001 [0.057]	-0.004 [0.059]	-0.04 [0.08]	-0.06 [0.18]
(Years 1 to 4)*High-AFDC	0.002 [0.06]	-0.01 [0.07]	-0.08 [0.06]	-0.13 [0.15]
(Years 5 to 9)*High-AFDC	-0.002 [0.075]	-0.06 [0.1]	-0.16 [0.07]	-0.27 [0.14]
R2	0.55	0.83	0.86	0.65
<i>B. Difference-in-Differences Estimates</i>				
Post-Medicaid*High-AFDC	-0.07 [0.08]	-0.06 [0.07]	-0.13 [0.05]	-0.16 [0.07]
R2	0.51	0.83	0.86	0.65
DD Test	0.18	0.73	0.52	0.28
Covariates	High-AFDC FE, Time- to-Medicaid Dummies	State FE, Medicaid- Timing-by- Year FE + Xst	(2) + region-by- year FE	(3), unweighted
Population Weighted?	Y	Y	Y	N

Notes: For details on dependent variables and sample see notes to figure 1-6, for details on specification and sources see notes to table 1-3. A Hausman test rejects the null hypothesis that the weighted and unweighted estimates in columns (4) and (5) are equal for either the grouped event-study model (p-value = <0.01) and the DD model (p-value = <0.01) (Deaton 1997; Solon, Haider and Wooldridge 2013).

**Table 1-7 Medicaid’s Effect on Log Nonwhite Child Mortality Ages 1-4 by Cause of Death**

	(1)	(2)
<i>DV Cause:</i>	<i>Internal Causes</i>	<i>External Causes</i>
<i>Mean Nonwhite Mortality Rate (per 100,000 children ages 1-4) by Cause in t*:</i>	97.4	61.3
<i>A. Grouped Event-Study Estimates</i>		
<i>Pre-Medicaid</i>		
(Years -7 to -2)*High-AFDC	0.00	-0.05
	[0.07]	[0.08]
<i>Post-Medicaid</i>		
(Year 0)*High-AFDC	-0.10	0.02
	[0.12]	[0.12]
(Years 1 to 4)*High-AFDC	-0.14	-0.02
	[0.07]	[0.10]
(Years 5 to 9)*High-AFDC	-0.15	-0.22
	[0.06]	[0.12]
R2	0.88	0.57
<i>B. Difference-in-Differences Estimates</i>		
Post-Medicaid*High-AFDC	-0.14	-0.09
	[0.03]	[0.07]
R2	0.93	0.94
DD Test	1.00	0.22

Notes: External causes are defined by the “E” codes in the International Classification of Diseases (revision 7 and 8). Internal causes of death are all non-external causes, as in figure 1-3. The specification is the same as column (3) of table 1-6. It 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. Sources: See notes to figure 1-6.

**Table 1-8 Medicaid’s Effect on Children’s Public Insurance Utilization by Type of Service**

	(1)	(2)	(3)	(4)
	Inpatient Hospital	MD Services	Prescription Drugs	Dental
Post-Medicaid*High-AFDC	0.004 [0.001]	0.027 [0.008]	0.033 [0.008]	0.012 [0.005]
R2	0.85	0.92	0.90	0.87
DD Test	0.04	0.14	0.64	0.92

Notes: The dependent variable is the share of children who had public insurance payments made for the services described in columns 1-4, calculated from administrative reports described in Appendix A. The model includes state fixed effects, separate year fixed effects for each Medicaid timing group, per-capita income and hospital variables, and region-by-year fixed effects. The row labeled “DD Test” contains the p-value from an F-test of the difference-in-differences restrictions: the pre-Medicaid coefficient is zero and post-Medicaid coefficients (not including year zero) are equal to each other. The test is conducted using the coefficients from a grouped event-study model (not shown) as in tables 3, 4 and 6. Post-Medicaid\*High-AFDC refers to all post-Medicaid years between 1 (the year after implementation) and 6. Source: See notes to figure 1-4.

**Table 1-9 Falsification Test: Medicaid’s Effect on log Nonwhite Mortality Including Medicaid Timing Variables Interacted with High-White-AFDC States**

	(1)	(2)	(3)
Dependent Variable:	log Nonwhite Mortality, Ages 0-14	log Nonwhite Neonatal mortality	log Nonwhite Mortality, Ages 1-4
<i>Treatment Effects</i>			
Post-Medicaid*High-Nonwhite-AFDC	-0.09 [0.03]	-0.09 [0.02]	-0.14 [0.05]
<i>False Treatment Effects</i>			
Post-Medicaid*High-White-AFDC	0.05 [0.03]	0.02 [0.03]	0.05 [0.04]
R2	0.96	0.93	0.86

Notes: The table contains estimated coefficients on interactions between a post-Medicaid dummy and an indicator for high-nonwhite-AFDC states in row 1 and high-white-AFDC states in row 2. The estimated treatment effects for nonwhite mortality are robust to the inclusion controls for white AFDC rates before and after Medicaid implementation. Source: see notes to table 1-3.

**Table 1-10 Robustness Check: Medicaid’s Effect on log Nonwhite Mortality Controlling for Nonwhite Welfare Rates**

	(1)	(2)	(3)
Dependent Variable:	log Nonwhite Mortality, Ages 0-14	log Nonwhite Neonatal mortality	log Nonwhite Mortality, Ages 1-4
Post-Medicaid*High-Nonwhite-AFDC	-0.07 [0.04]	-0.09 [0.03]	-0.10 [0.05]
R2	0.96	0.93	0.86

Notes: The table contains estimated coefficients on interactions between post-Medicaid dummy and an indicator for high-nonwhite-AFDC states as in equation (1). The regressions also includes state-by-year nonwhite welfare rates ( $AFDC_{st}$ ) and their interaction with a post-1966 dummy ( $AFDC_{st} \cdot 1\{y \geq 1966\}$ ). These controls account for omitted factors that are correlated with levels and changes in specifically nonwhite AFDC rates, and any change in the influence of these factors on mortality in the mid-1960s. The results show that the estimated treatment effects of Medicaid in high-nonwhite-AFDC states are robust to controls for AFDC rates themselves. Source: see notes to table 1-3.

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## **Appendix A DATA SOURCES**

### **A.1 Data on Recipients of Aid to Families with Dependent Children**

The data on the characteristics of AFDC recipients come from two sources. The race share of adult and child recipients were entered from two printed reports: “Characteristics and Financial Circumstances of Families Receiving Aid to Dependent Children, Late 1958” (**Mugge 1960**), and “Characteristics of Families Receiving Aid to Families with Dependent Children, November-December 1961” (**DHEW 1963**). Biennial microdata on recipients comes from the National Archives Surveys of Recipients of Aid to Families with Dependent Children 1967-1979 (**DHEW 2000, 2011**). Except for the 1967 file, the data are at the AFDC unit level.

The race shares for women are the sample-weighted means among household heads. The race shares for children are the means weighted by product of the sample weight and the number of recipient children in the household (this assumes that the race of the children is the same as the race of the AFDC payee). In some years, the race code for Latina recipients is missing or varies strongly between years (from “other” to “white”). In these cases I assign Latina recipients the average value of the binary race code observed among all other Latina recipients. I linearly interpolate the race shares for missing years between 1958 and 1979. To construct race-specific recipient counts I multiply the estimated race shares by state-level counts of AFDC cases or children (available from HHS). Note that, for women, the count of cases is a more accurate number than the count of adults because nearly all AFDC cases contained one mother, but some contained more than one adult. To calculate race-specific recipient rates, I divide by the state population of women ages 15-54 or the population of children ages 0-19 (**Haines and ICPSR 2010, SEER 2013**).

## A.2 Data on State Public Insurance Use

Data on public insurance were collected from a series of reports on the numbers of recipients and amount of payments to medical “vendors” on behalf of public assistance recipients (**DHEW Various Years**). The reports contain separate tables for recipients and expenditures and for each reason for eligibility (blind, disabled, elderly, membership in a single parent family, or other). The rows of each table are states and the columns are the type of medical service received and a total (unduplicated for recipients). This paper uses the tables that refer to child recipients eligible by virtue of their status as a “dependent child” in a qualifying family (i.e., one parent).

Table A1.1 contains the time periods of the primary source documents, the numbers of states reporting under pre-Medicaid “Medical Vendor Payment” programs or under Medicaid (Title XIX) programs, and variables describing whether states reported the number of families who had a public insurance payment, separate data on the number of adults and children, or whether they made no vendor payments or did not report. When states report the number of families receiving a public insurance payment I assume that 1.5 children and 0.5 adults used care, which are approximately half the average numbers of children and adults per AFDC case in the mid-1960s.

To construct a state-by-year dataset of public insurance rates from the source data that are at the fiscal year, calendar year, or half-calendar year level, I first convert the dataset into half-year probabilities of receiving public insurance. For annual data, I divide the child recipient count by the child population to obtain an annual probability  $p^A$  and I infer the corresponding half-year probability ( $p^H$ ) using:  $p^A = 1 - (1 - p^H)^2$ . This assumes that  $p^H$  is constant over the

year. For half-year data, I calculate  $p^H$  directly. This leads to a dataset of public insurance probabilities at the half-year level. (Before 1976, the US fiscal year ran from July 1 to June 30, so the half year probabilities calculated from fiscal year  $y$  data are for the second half of calendar year  $y-1$  and the first half of calendar year  $y$ .) I linearly interpolate  $p^H$  for missing half years (for example, the second half of CY 1963 and the first half of CY 1964) and use these probabilities to reconstruct a dataset of public insurance probabilities at the calendar year level. These are used in figures 1-2 and 1-4 and in table 1-8.

**Table A-1 Structure of Public Insurance Data for Families with Dependent Children**

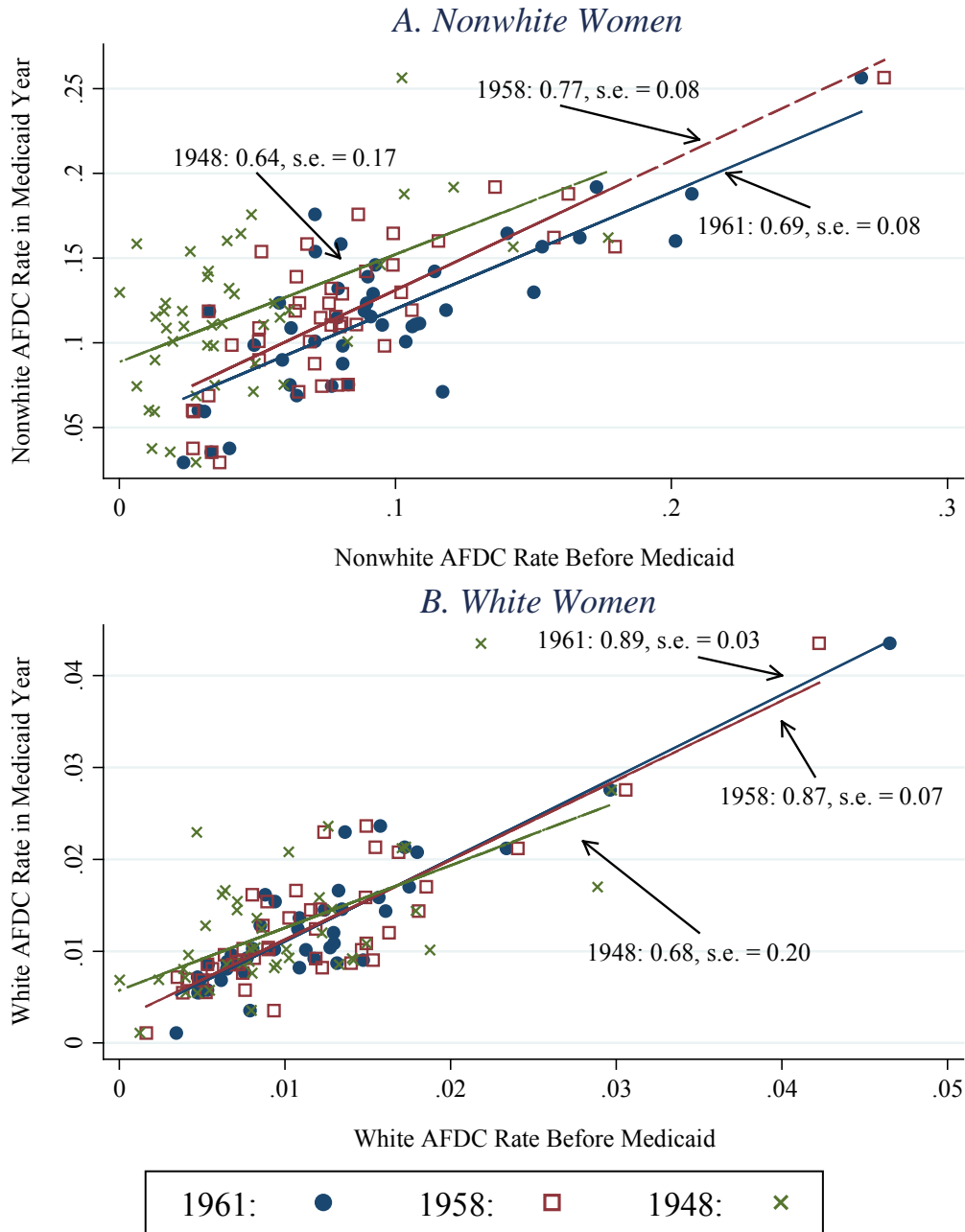
Time Period	Program	States Reporting for Families	States Reporting Children/Adults	States without Vendor Payments	States with No Data
FY 1963	Medical Vendor Program	27	14	13	0
FY 1965	Medical Vendor Program	25	17	11	1
2nd Half CY 1965	Medical Vendor Program	26	16	9	3
1st Half CY 1966	Medical Vendor Program	19	15	8	3
	Title XIX (Medicaid)	6	3	.	0
2nd Half CY 1966	Medical Vendor Program	13	8	3	3
	Title XIX (Medicaid)	9	15	.	3
2nd Half CY 1967	Medical Vendor Program	0	9	0	5
	Title XIX (Medicaid)	0	33	.	7
CY 1968	Medical Vendor Program	0	8	3	1
	Title XIX (Medicaid)	0	41	.	1
1st Half CY 1969	Medical Vendor Program	0	7	2	2
	Title XIX (Medicaid)	0	36	.	7
CY 1969	Medical Vendor Program	0	7	1	1
	Title XIX (Medicaid)	0	43	.	2
CY 1970	Medical Vendor Program	0	0	0	2
	Title XIX (Medicaid)	0	50	.	2
CY 1972	Medical Vendor Program	0	0	2	0
	Title XIX (Medicaid)	0	49	.	3
FY 1973	Medical Vendor Program	0	0	0	1
	Title XIX (Medicaid)	0	48	.	5
FY 1974	Medical Vendor Program	0	0	0	1
	Title XIX (Medicaid)	0	48	.	5
FY 1975	Medical Vendor Program	0	0	0	1
	Title XIX (Medicaid)	0	48	.	5
FY 1976	Medical Vendor Program	0	0	0	1
	Title XIX (Medicaid)	0	46	.	7

Notes: The source data include Guam, Puerto Rico and the Virgin Islands, so the total number of states is 54.



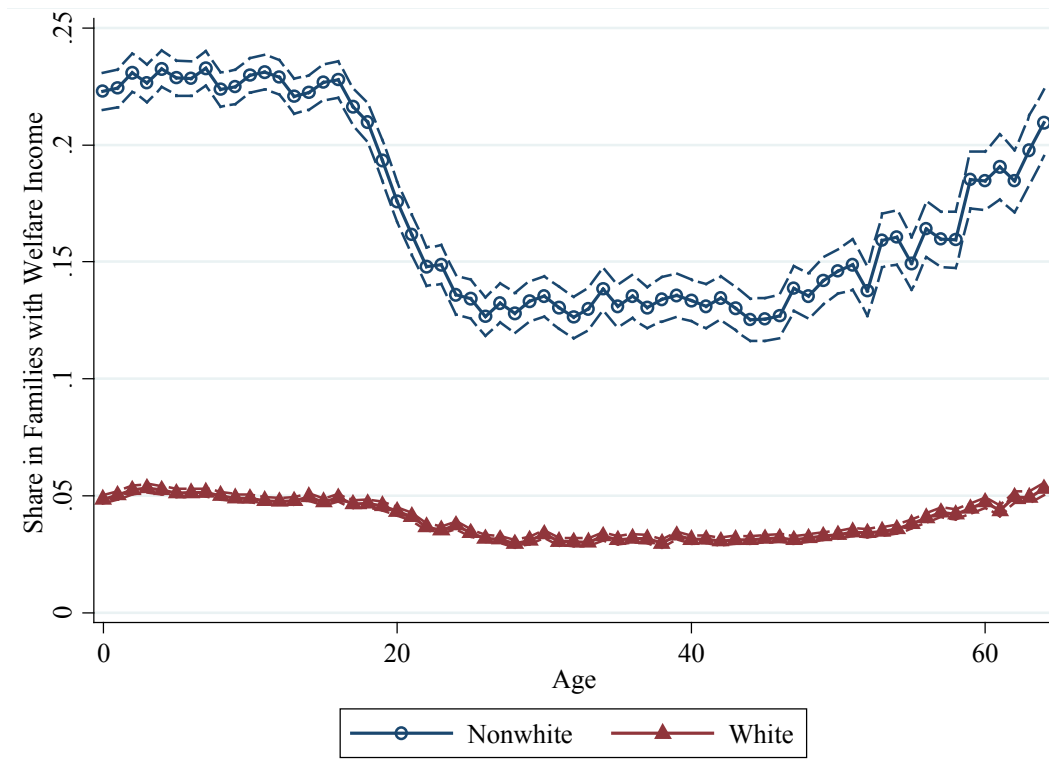
## **Appendix B ADDITIONAL RESULTS**

**Figure B-1 Stability in Cross-State AFDC Variation, 1948 through Medicaid Implementation**



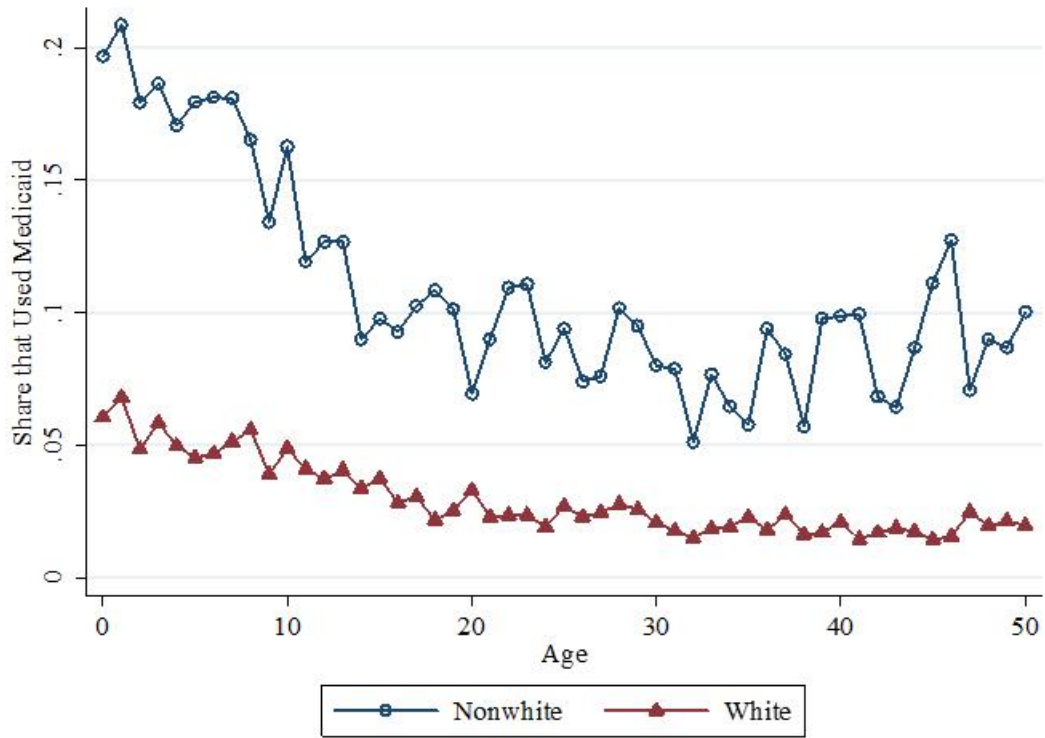
Notes: The figure presents scatter plots and fitted values of the relationship between the paper’s primary measure of categorical eligibility—the AFDC rate in the year of Medicaid implementation (y-axis)—and three measures of AFDC rates in years prior to each state’s Medicaid year. The results show that the cross-state variation in AFDC rates was very stable over time. For both white and nonwhite women, pre-Medicaid AFDC rates strongly predict AFDC rates in the year of Medicaid and the relationship itself does not change over time. P-values from a test of the null hypothesis that all the slopes are equal (estimated from a joint regression) are 0.63 for nonwhite women and 0.72 for white women.

**Figure B-2 The Age Distribution of Welfare Receipt in the 1970 Census**



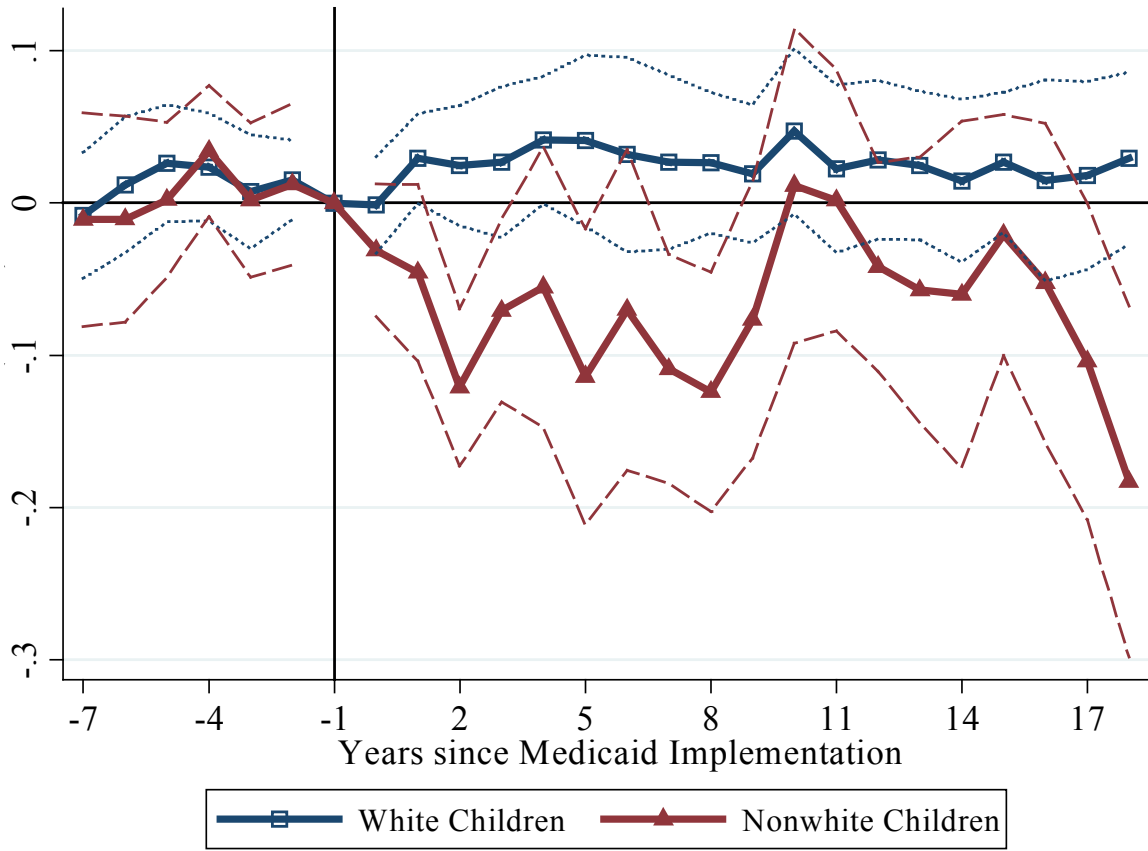
Notes: Data from the 1970 Census of Population State Sample Forms 1 and 2. The figures plots the mean value of a dummy variable equal to one if a respondent lived in a household where at least one person reported positive welfare income. The dashed lines are 95% confidence intervals. The average welfare receipt is higher than in figure 1-2 because the Census question is not restricted to AFDC. This increases the adult welfare rate by the most, but it does not necessarily mean that their Medicaid eligibility rates were higher because this includes General Assistance, a state program not included in the definition of categorical (Medicaid) eligibility. Source: Ruggles et al. 2010.

**Figure B-3 The Age Distribution of Medicaid Receipt, 1976 Survey of Income and Education**



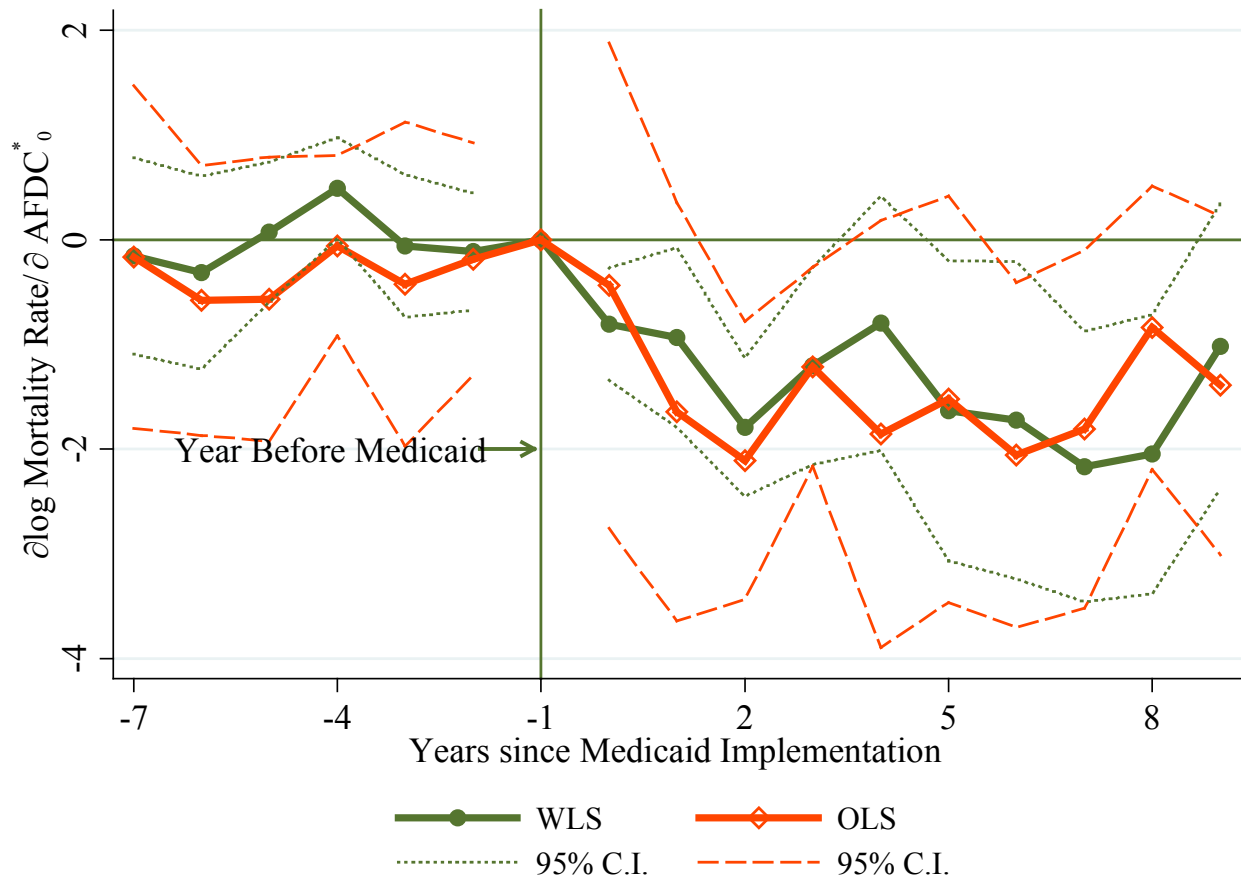
Notes: The figures plots the mean value of a dummy variable equal to one if a respondent report using Medicaid in the previous year. 3,547 observations (out of 440,815) are missing and are dropped from the calculation. Source: 1976 Survey of Income and Education (Census Bureau and ICPSR)

**Figure B-4 Regression-Adjusted Estimates of Medicaid’s Intention-to-Treat Effect on Child Mortality by Race, 1959-1988**



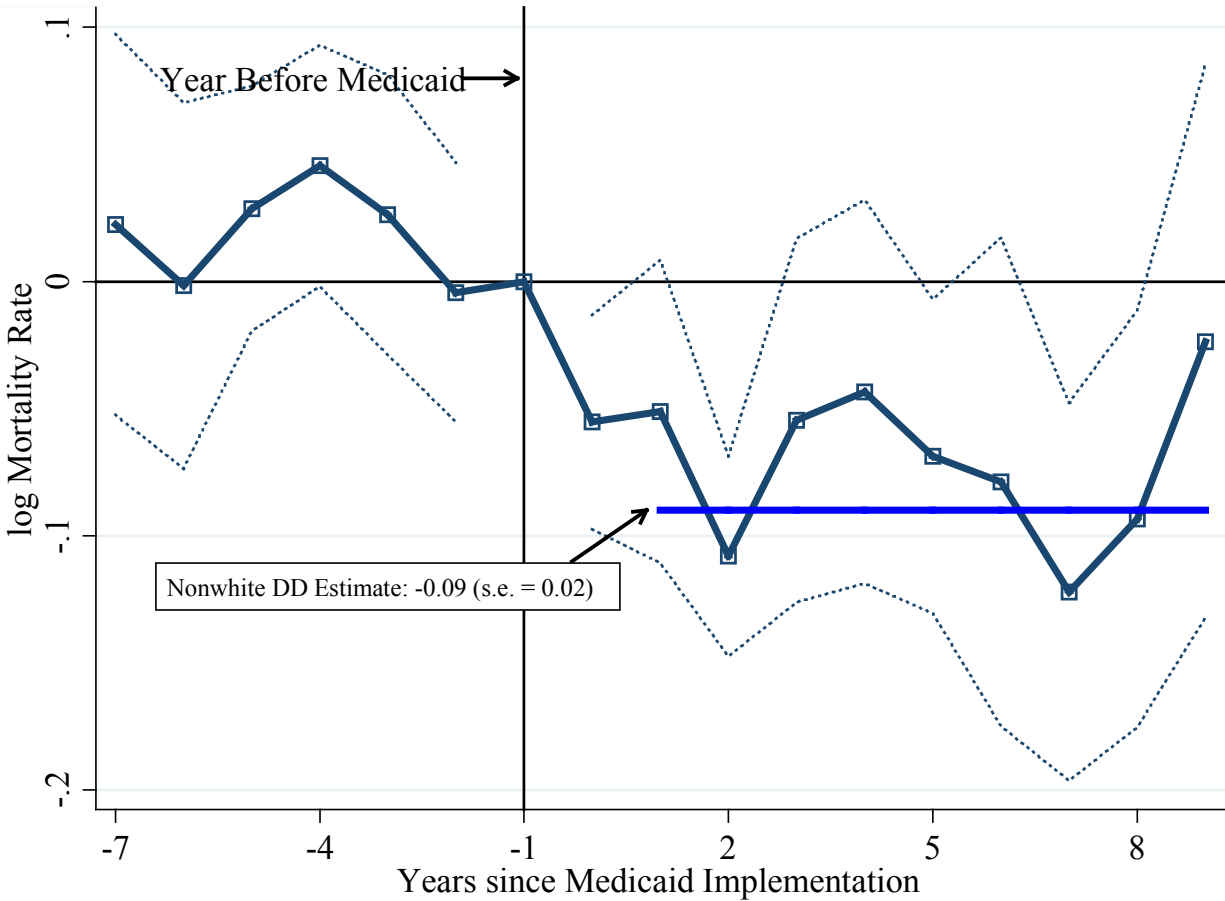
Notes: see notes to figure 1-6. The sample extends through 1988 and so the estimated treatment effects cover event years -7 through 18.

**Figure B-5 Medicaid's Intention-to-Treat Effect on Age-Adjusted Nonwhite Child Mortality by Race, Continuous AFDC Specification**



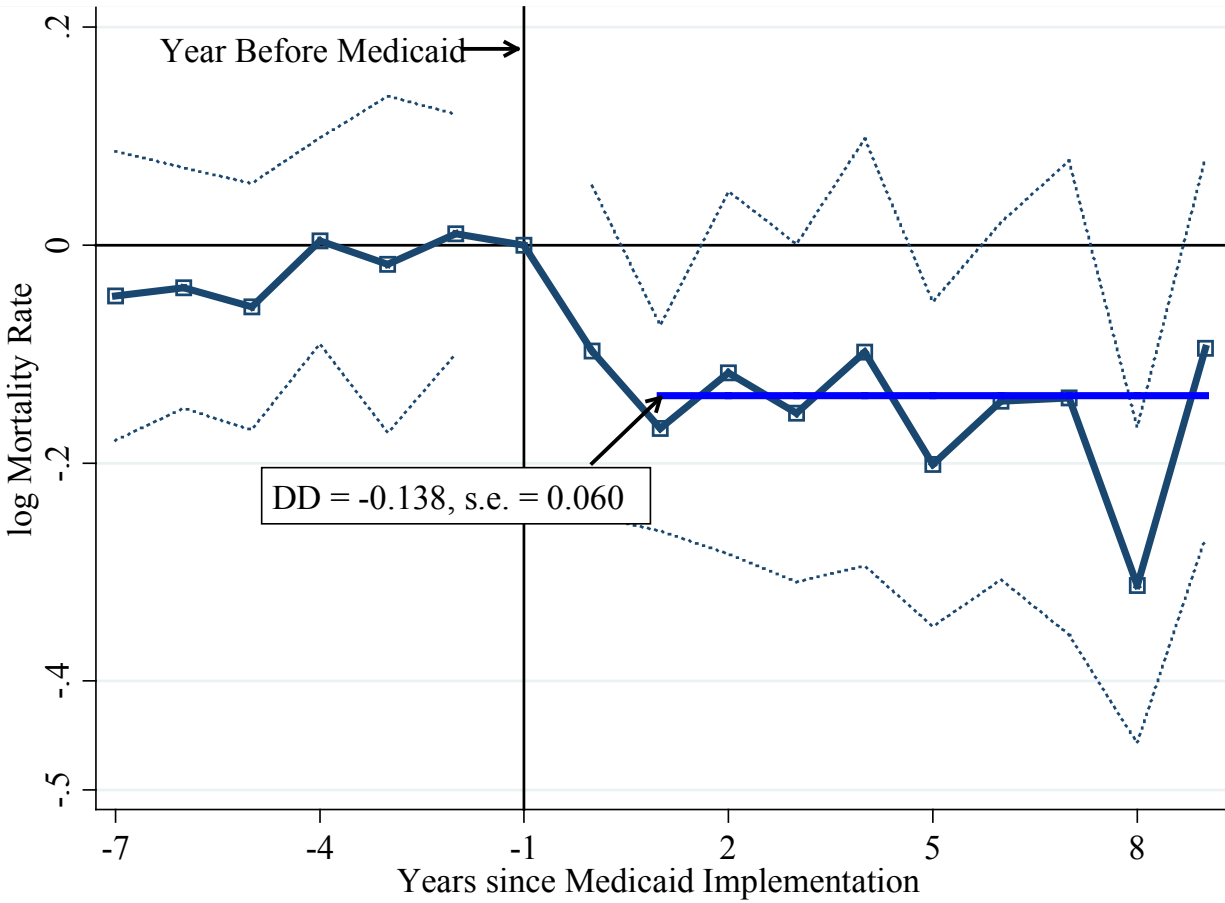
Notes: The figure plots the estimated coefficients on interactions between Medicaid event-time dummies and the value of states' AFDC rates (for women) in the year of Medicaid implementation (see appendix table 2.1). Results are presented with and without population weights.

**Figure B-6 Medicaid's Intention-to-Treat Effect on Age-Adjusted Nonwhite Child Mortality, Alternative Algorithm to Define High- and Low-AFDC Groups**



Notes: The specification and sample are the same as in figure 1-6. The high-AFDC dummy is defined according to the following algorithm. (1) Rank states from low to high according to their AFDC rates in the year of Medicaid implementation (AFDC\*). (2) For ranks  $j = 1, \dots, 44$  (the number of states in the nonwhite estimation sample) create a dummy equal to one for states with rank higher than  $j$  ( $D_j$ ) and regress AFDC\* on  $D_j$ . (3) Store the  $t$ -statistic on  $D_j$  ( $t_j$ ). (4) Define a high-AFDC dummy equal to one for states with rank less than  $j^*$ , where  $j^* = \max\{t_j\}$ . This creates two groups that maximize the precision of the measured difference in initial Medicaid eligibility rates. For nonwhites, the 12 highest AFDC states are the high-AFDC (treatment) group created by this algorithm and 31 states are in the low-AFDC (control) group.

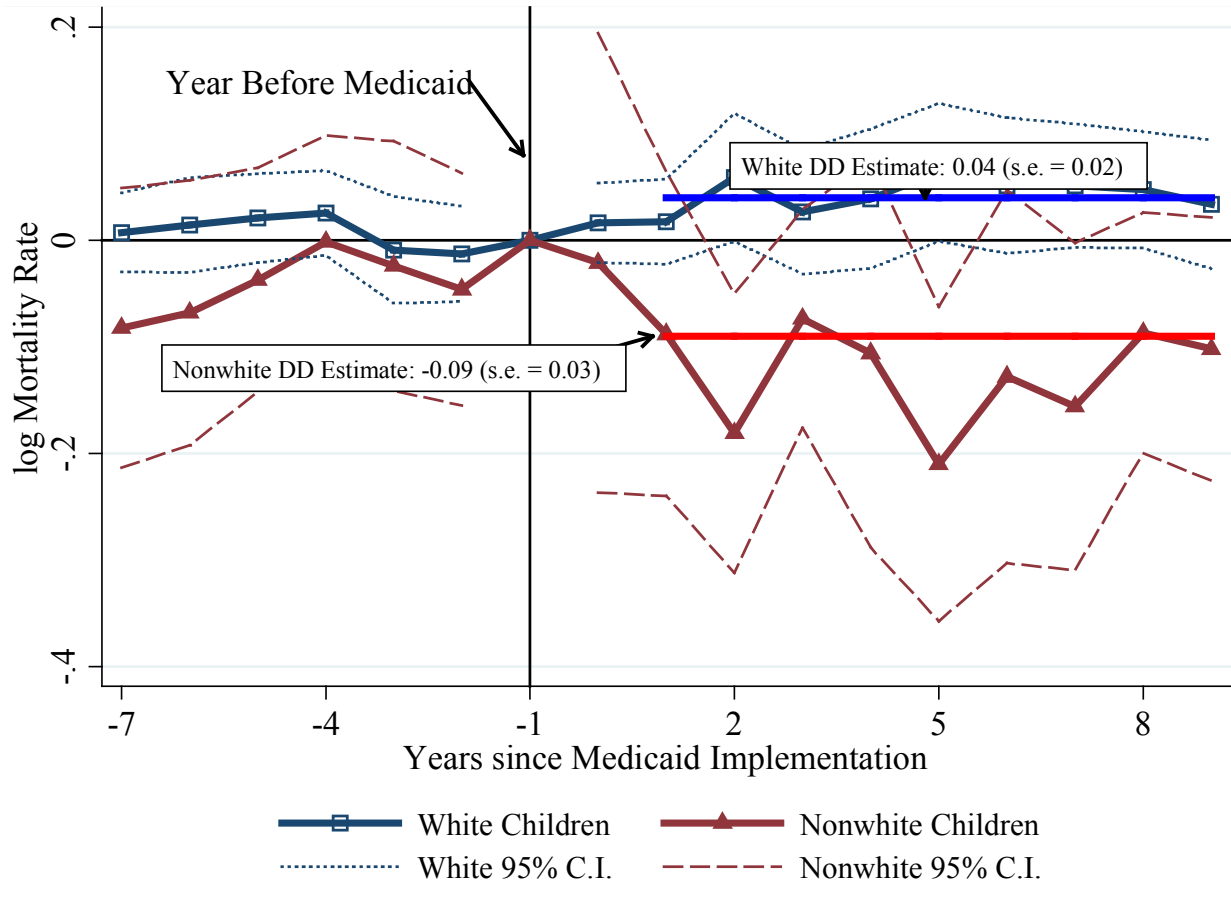
**Figure B-7 Medicaid's Intention-to-Treat Effect on Nonwhite Child Mortality Ages 1-4, High- versus Low-Child-AFDC Groups**



Notes: See notes to figures 5 and 6. The high- and low-AFDC dummies are created using child AFDC rates (see appendix table 2.1) rather than rates for women.

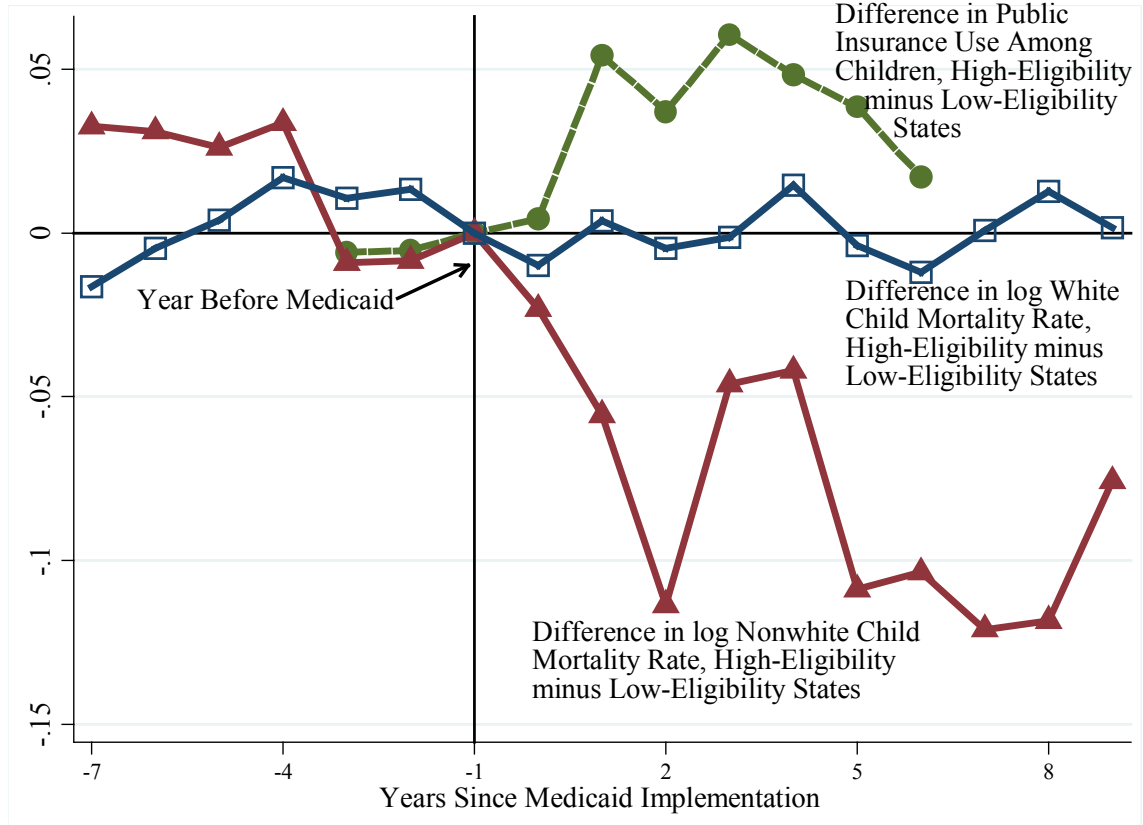


**Figure B-8 Unweighted Estimates of Medicaid’s Intention-to-Treat Effect on Age-Adjusted Nonwhite Child Mortality by Race**



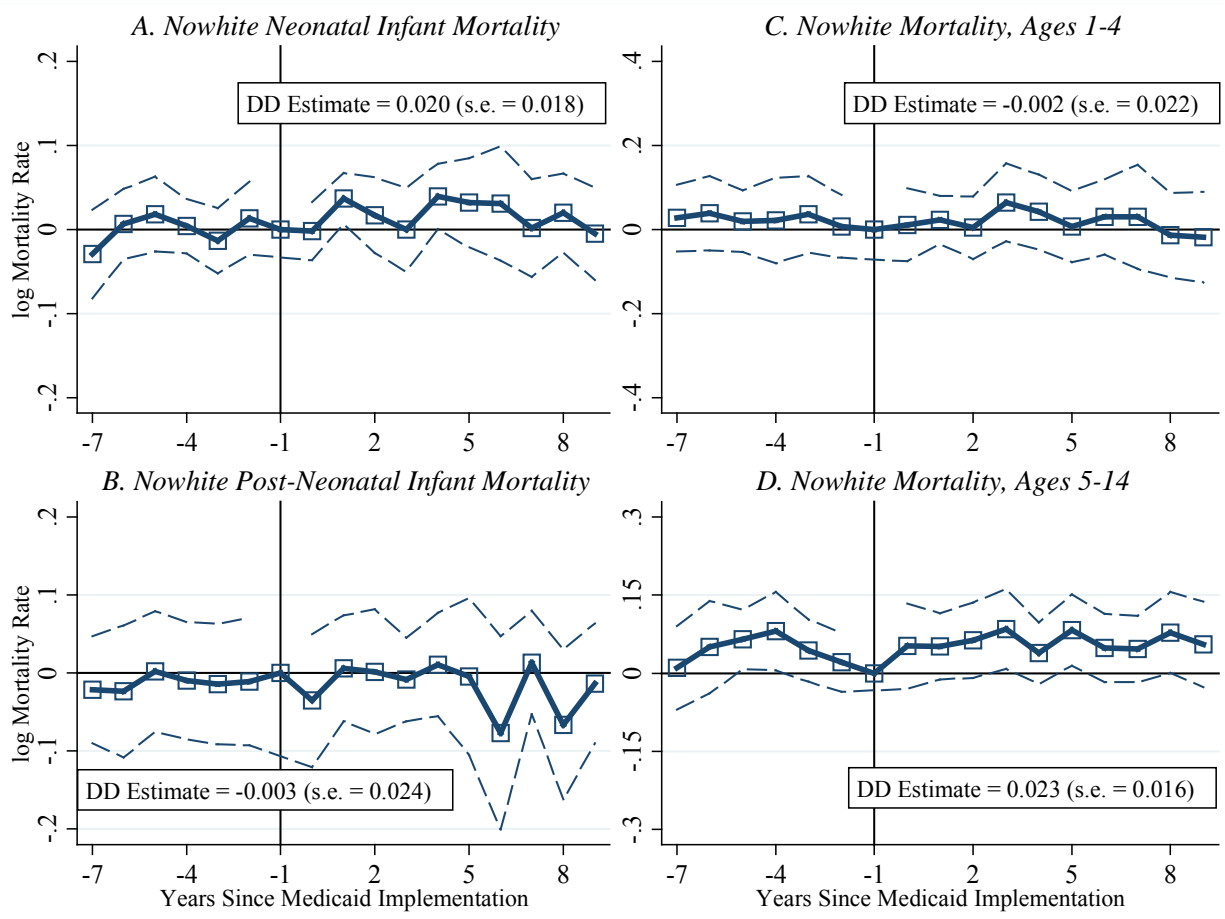
Notes: The specification and sample are the same as in figure 1-5 except that the results are estimated without population weights.

**Figure B-9 Unadjusted Estimates of Medicaid’s Effect on Children’s Public Insurance Use and Mortality**



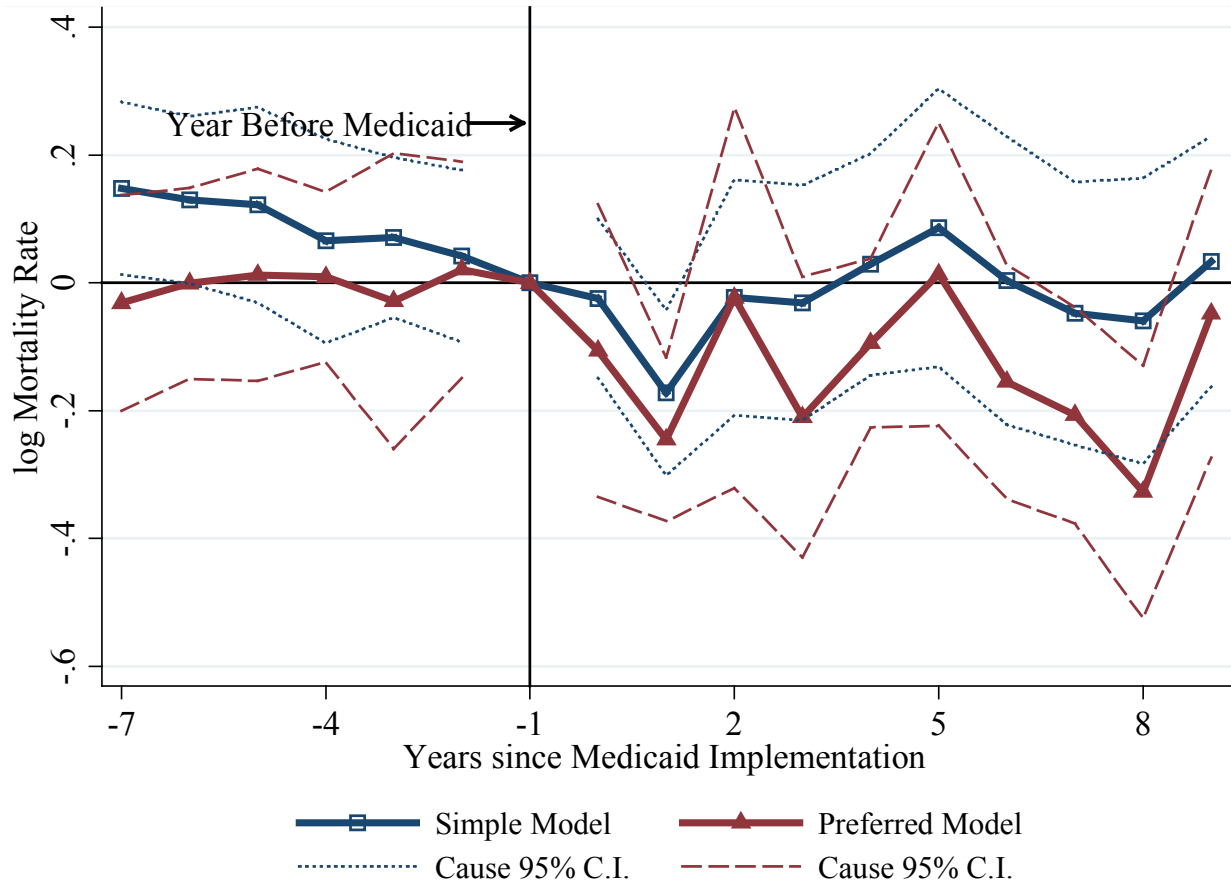
Notes: The figure plots mean differences between high-AFDC and low-AFDC states in the share of children who used services covered by public health insurance programs and the log mortality rate of children ages 0-14 by race. States are split into high- and low-AFDC groups based on the median AFDC rate in the year that states implemented Medicaid. The solid lines show the reduced form relationship between Medicaid implementation and age-adjusted mortality rates by race without adjusting for covariates. The dashed line shows the first stage relationship between Medicaid implementation and public insurance utilization without adjusting for covariates. Source: AFDC cases are from Health and Human Services Caseload Data 1960-1999 (HHS 2012), population data are from 1960 population estimates (Haines and ICPSR 2005), and the Survey of Epidemiological End Results (SEER 2009), data on public insurance use are collected from various editions of “Recipients of Medical Vendor Payments Under Public Assistance Programs” and “Medicaid State Tables” (DHEW 1963-1976). See Appendix A for details on the public insurance data.

**Figure B-10 Regression-Adjusted Estimates of Medicaid’s Effect on White Mortality by Age Group for Infants and Children**



Notes: See notes to figure 1-6. The sample excludes Alaska, Hawaii and New Jersey but, unlike the nonwhite sample, includes Maine, New Hampshire and Vermont. Results are weighted by state populations.

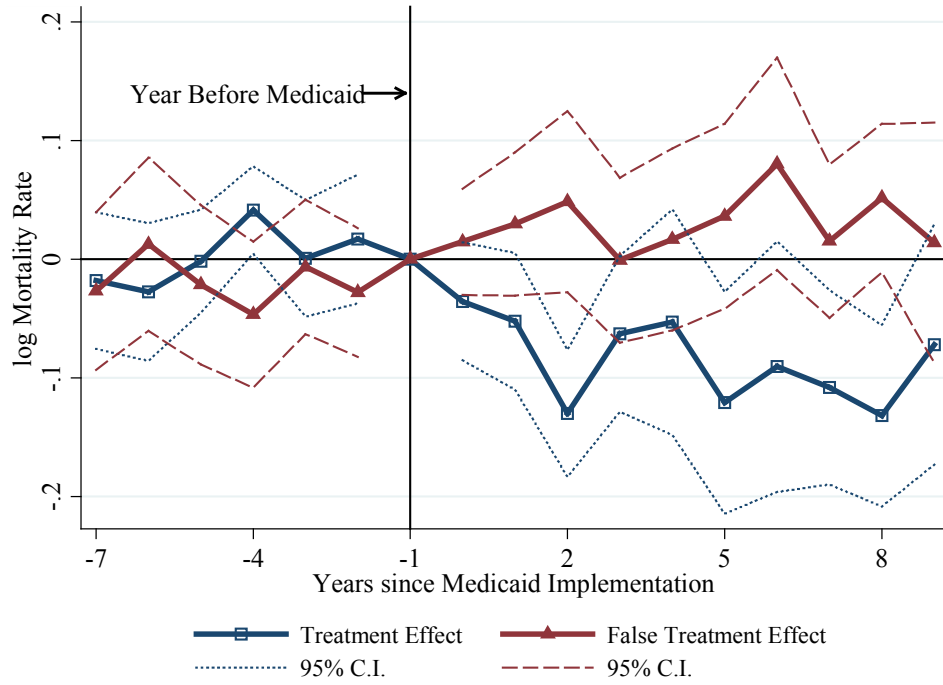
**Figure B-11 Medicaid's Effect on Nonwhite Child (1-4) Internal-Cause Mortality Across Specifications**



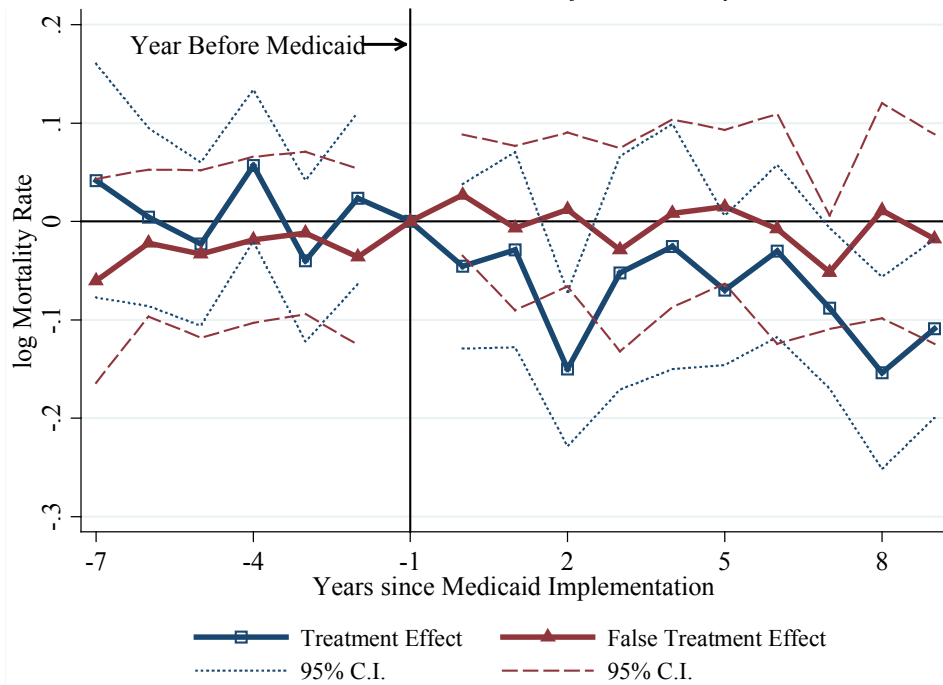
Notes: See notes to figure 1-6 and table 7. The figure plots event study estimates from the simplest DD model (column 1 of table 7) and the preferred model (column 3 of table 7). It shows that region-by-year fixed effects control for a pre-Medicaid trend in internal-cause mortality among younger nonwhite children.

**Figure B-12 Falsification Test: Controlling for Medicaid-Timing Interacted with High-White-AFDC States, Event-Study Specification**

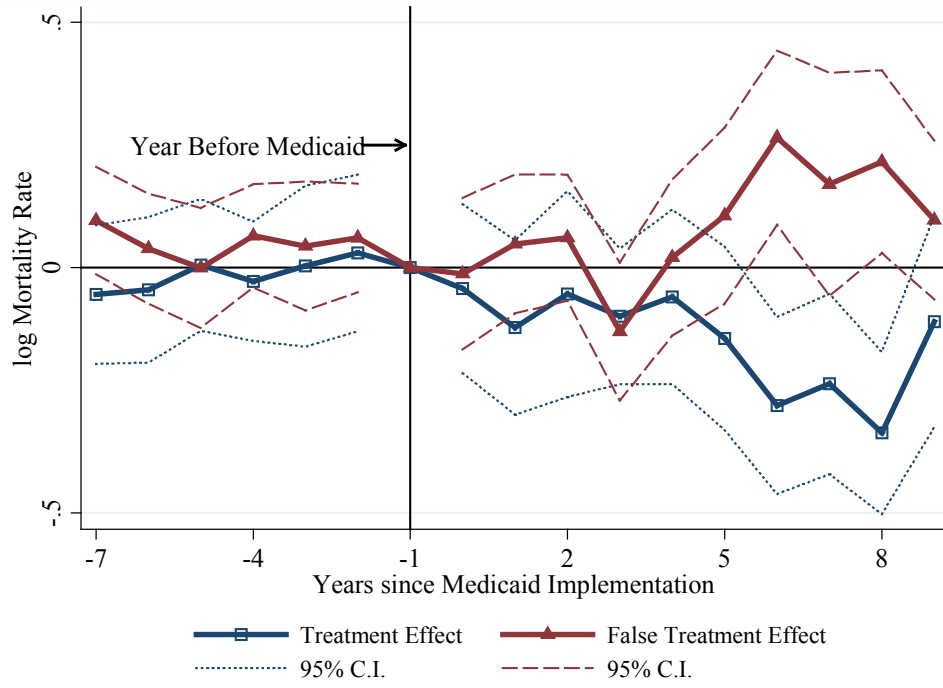
*A. Age-Adjusted Nonwhite Child Mortality (0-14)*



*B. Nonwhite Neonatal Infant Mortality*

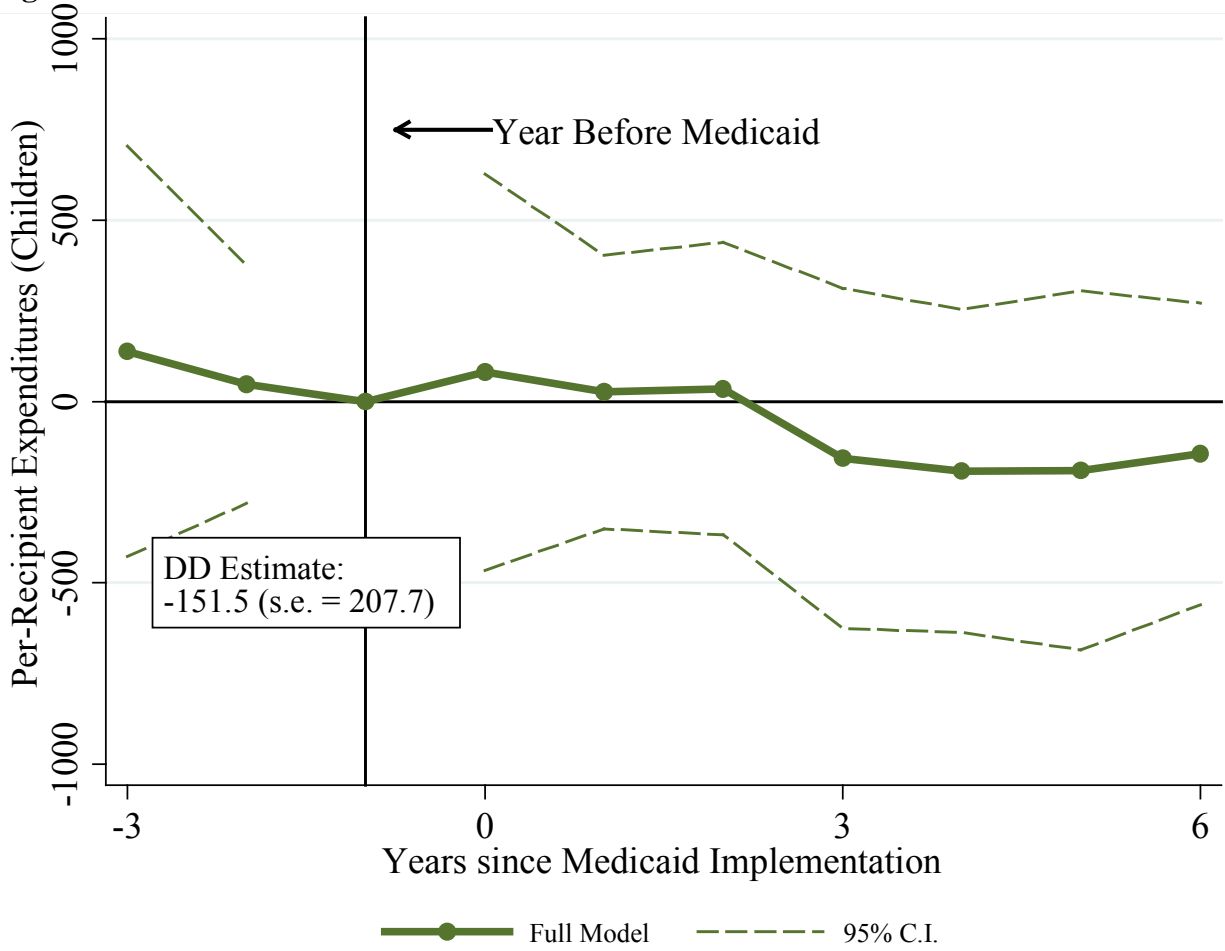


C. Younger Nonwhite Child Mortality (1-4)



Notes: See notes to figures 5 and 6 and table 9.

**Figure B-13 Medicaid's Effect on Public Insurance Expenditures Per Child Recipient, High- versus Low-AFDC States**



Notes: The figure plots event-study coefficients on the interaction between a high-AFDC dummy and Medicaid event-time dummies. The dependent variable is the ratio of total public insurance expenditures on children divided by the number of child recipients, expressed in 2012 dollars. The figure shows that the generosity (or intensity of utilization per recipient) did not vary with Medicaid eligibility, either before or after Medicaid implementation.

**Table B-1 State Welfare Rates in the Year of Medicaid Implementation**

State	White Women	Nonwhite Women	White Children	Nonwhite Children
Alabama	0.012	0.124	0.023	0.202
Arkansas	0.010	0.119	0.018	0.169
California	0.023	0.119	0.051	0.230
Colorado	0.024	0.154	0.041	0.305
Connecticut	0.010	0.142	0.021	0.255
Delaware	0.007	0.115	0.014	0.213
District of Columbia	0.001	0.038	0.004	0.099
Florida	0.009	0.176	0.019	0.284
Georgia	0.008	0.060	0.016	0.100
Idaho	0.015	0.071	0.024	0.100
Illinois	0.005	0.111	0.013	0.249
Indiana	0.009	0.132	0.016	0.221
Iowa	0.015	0.160	0.027	0.282
Kansas	0.010	0.123	0.022	0.233
Kentucky	0.021	0.098	0.038	0.184
Louisiana	0.009	0.075	0.017	0.127
Maine*	0.020	0.061	0.037	0.096
Maryland	0.007	0.099	0.015	0.185
Massachusetts	0.015	0.129	0.032	0.246
Michigan	0.009	0.088	0.017	0.160
Minnesota	0.014	0.165	0.024	0.235
Mississippi	0.009	0.158	0.019	0.217
Missouri	0.011	0.119	0.024	0.231
Montana	0.009	0.157	0.017	0.170
Nebraska	0.009	0.146	0.017	0.261
Nevada	0.007	0.130	0.013	0.225
New Hampshire*	0.007	0.036	0.015	0.057
New Jersey**	0.021	0.226	0.048	0.416
New Mexico	0.028	0.074	0.050	0.097
New York	0.016	0.115	0.040	0.263
North Carolina	0.009	0.090	0.015	0.144
North Dakota	0.010	0.188	0.017	0.217
Ohio	0.008	0.101	0.017	0.199
Oklahoma	0.017	0.162	0.035	0.264
Oregon	0.017	0.111	0.032	0.210
Pennsylvania	0.010	0.101	0.025	0.216
Rhode Island	0.021	0.192	0.045	0.337
South Carolina	0.004	0.035	0.006	0.052
South Dakota	0.012	0.256	0.021	0.257
Tennessee	0.014	0.109	0.033	0.171



Texas	0.006	0.029	0.013	0.058
Utah	0.021	0.139	0.033	0.202
Vermont*	0.014	0.004	0.026	0.012
Virginia	0.006	0.059	0.011	0.103
Washington	0.016	0.075	0.030	0.133
West Virginia	0.044	0.110	0.101	0.198
Wisconsin	0.008	0.111	0.014	0.168
Wyoming	0.013	0.069	0.023	0.088

Notes: Race-specific AFDC rates are calculated as described in text. AFDC rates for women are per woman ages 15-54. AFDC rates for children are per child 0-18. \*Excluded from nonwhite sample because less than one percent of population is nonwhite. \*\*New Jersey is excluded from all race-specific outcomes because its mortality files lack race codes in 1962 and 1963.

**Table B-2 Medicaid's Effect on Log Nonwhite Age-Adjusted Child Mortality with Alternative Region Definitions and Samples**

Sample/Region Definition:	Full Sample	No Mississippi	No Deep South	No South	Move Delaware, Maryland and West Virginia to Northeast
Post-Medicaid*High-AFDC	-0.08 [0.03]	-0.07 [0.03]	-0.08 [0.03]	-0.15 [0.04]	-0.05 [0.04]
R2	0.96	0.96	0.95	0.95	0.96

Note: Deep South includes Alabama, Georgia, Louisiana, Mississippi, and South Carolina.

**Table B-3 Medicaid's Effect on Log Nonwhite Mortality and Public Insurance Use, Continuous AFDC Rate Specification**

	(1)	(2)	(3)	(4)
<i>A. Age-Adjusted Nonwhite Child Mortality (0-14)</i>				
Post-Medicaid*Nonwhite-AFDC0	-1.63 [0.31]	-0.74 [0.52]	-1.45 [0.42]	-1.35 [0.36]
<i>B. Nonwhite Neonatal Infant Mortality</i>				
Post-Medicaid*Nonwhite-AFDC0	-1.86 [0.2]	-0.85 [0.37]	-1.32 [0.28]	-1.04 [0.41]
<i>C. Nonwhite Younger Child Mortality (1-14)</i>				
Post-Medicaid*Nonwhite-AFDC0	-1.19 [0.69]	-1.17 [0.87]	-2.49 [0.84]	-2.39 [0.99]
<i>D. Children's Public Insurance Use (0-19)</i>				
Post-Medicaid*AFDC0	2.24 [1.03]	3.13 [1.15]	3.32 [1.03]	2.13 [1.02]
Covariates	AFDC0, Time-to-Medicaid Dummies	State FE, Medicaid-Timing-by-Year FE + Xst	(2) + region-by-year FE	(3), unweighted
Population Weighted?	Y	Y	Y	N

Notes: The table contains the estimated coefficients on an interacted between a post-Medicaid dummy and the value of states' AFDC rates (for women) in the year of Medicaid implementation (see appendix table 2.1). Mortality results from this continuous specification for nonwhite child mortality (0-14), neonatal infant mortality, and young child mortality (1-4) are in panels A through C. First-stage results from this continuous specification are in panel D. The t-statistics for these continuous mortality estimates (coefficient divided by standard error from column 3) are larger than the corresponding binary estimates in tables 3, 4 and 6. For age-adjusted mortality they are 3.45 versus 2.35; for neonatal infant mortality they are 4.71 versus 3.68; for young child mortality they are 2.96 versus 2.59. The first-stage t-statistic from the binary first stage specification in figure 1-5 is slightly higher than that shown here (3.43 versus 3.22)

## **Appendix C ESTIMATES USING THE STAGGERED TIMING OF MEDICAID IMPLEMENTATION**

One strategy to identify the effect of Medicaid implementation is to estimate difference-in-difference models that use variation in when states implemented Medicaid (see Strumpf 2011, Decker and Gruber 1993; for Canada see Hanratty 1996). I do not use this source of variation because there is strong evidence that earlier and later Medicaid states are not comparable. Finkelstein (2007, fn. 4) concludes that “the timing of state implementation of Medicaid was not random with respect to hospital outcomes” and I argue that the same holds with respect to mortality rates.

26 states implemented Medicaid in 1966, 16 more from 1967 to 1969 and 7 states established programs in 1970 at the latest date stipulated in the original legislation.<sup>36</sup> Because Medicaid increased federal reimbursement for public assistance costs, “the order in which states moved in establishing Medicaid programs was dictated by concerns about maximizing the federal share of vendor programs” (Stevens and Stevens 1974, pp 80). This incentive led “more affluent industrial states” to adopt Medicaid earlier than poorer states with smaller welfare programs (Fein 1986 pp 115). Strumpf (2011, table 2) shows that local government expenditures on public welfare and health programs are half as large in later Medicaid states than earlier ones. Relative to earlier states, later Medicaid states had significantly higher 1960 child poverty rates and lower 1965 AFDC rates for both whites and nonwhites.

There are also limitations inherent in difference-in-differences estimates based only on variation in treatment timing. **Bitler, Gelbach, and Hoynes (2003)** show that in a model in

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<sup>36</sup> Alaska (1972) and Arizona (1982) missed this deadline, although the threat to withhold reimbursements were “not only not made but never considered seriously” (Stevens and Stevens 1974, pp 137).

which all units are treated but at different times, the difference-in-difference estimate (with year fixed effects but not unit fixed effects) only uses variation from the periods in which some units are treated and others are not. This means that estimates based on Medicaid timing would only use variation in mortality rates in the five years from 1966 to 1970, only uncovering treatment effects that occur exactly at the time of treatment.

A final argument against the timing-only estimator is that in the presence of strong cross-state heterogeneity of the kind exploited in this paper, difference-in-differences estimates based on Medicaid's roll-out need not identify a meaningful average of the state-level effects (Deaton 1997, Dumouchel and Duncan 1983). This appears to be a problem for the first-stage estimates using timing. Medicaid is clearly responsible for the large increases in public insurance utilization but the DD estimates of the effect based on timing are not significant.

The results below show event-study and difference-in-difference estimates from a version of (1) without the high-AFDC interactions or Medicaid-timing-group-by-year fixed effects. Instead it includes state fixed effects, region-by-year fixed effects, continuous covariates and the Medicaid event-time dummies:

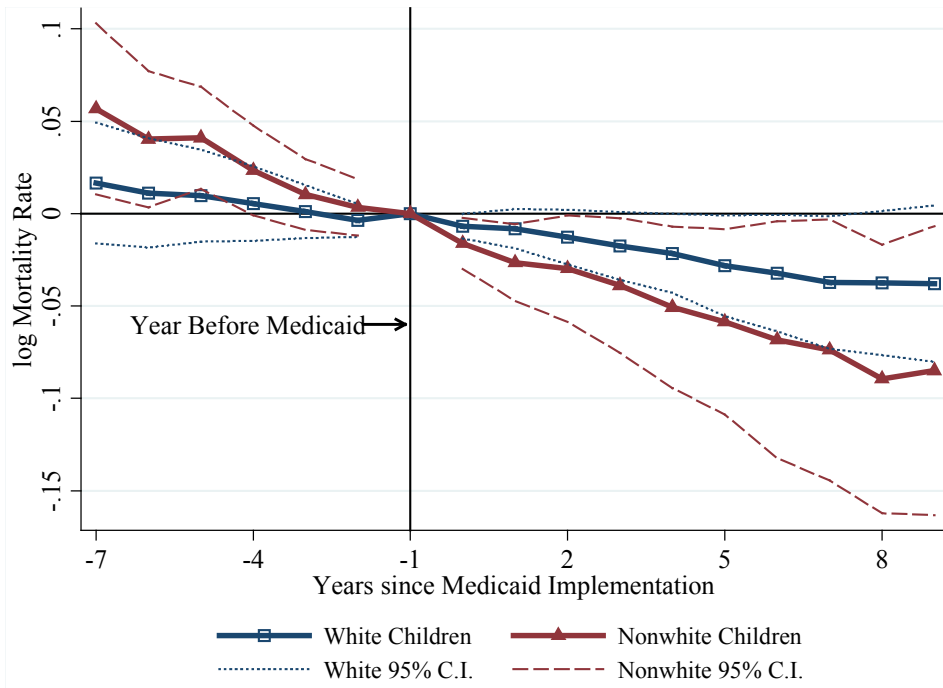
$$\ln(ASMR_{st}^a) = \mathbf{x}'_{st}\tilde{\boldsymbol{\beta}}_a + \sum_{y=-8}^{-2} \tilde{\pi}_y^a \mathbf{1}\{t - t_s^* = y\} + \sum_{y=0}^{10} \tilde{\gamma}_y^a \mathbf{1}\{t - t_s^* = y\} + u_{st}^a \quad (C1)$$

The tildes are meant to distinguish the coefficients from those in equation (1). The dependent variable is the log of age-adjusted child mortality.

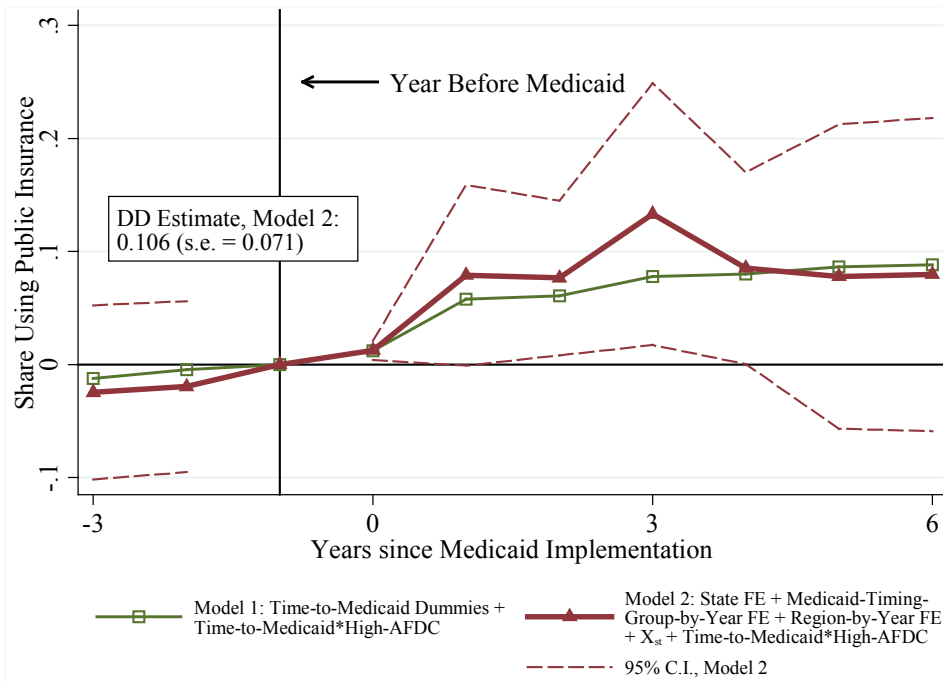
The event-study results (figure A3.1) are clearly driven by strong negative trends in mortality that are correlated with Medicaid timing. Perhaps surprisingly, the associated DD results are very close to zero, suggesting that Medicaid implementation did not affect mortality. Figure A3.3 plots the year fixed effects (for the Northeast region) from the event-study and DD

models. The year fixed effects for the restricted DD specification capture a large part of the strong negative trend that is apparent in the event-study results. This follows from the small amount of variation in Medicaid timing, and it is why the event-study results appear strongly negative (they absorb part of the time trend) but the DD results are small (the year effects account for most of the time trend).

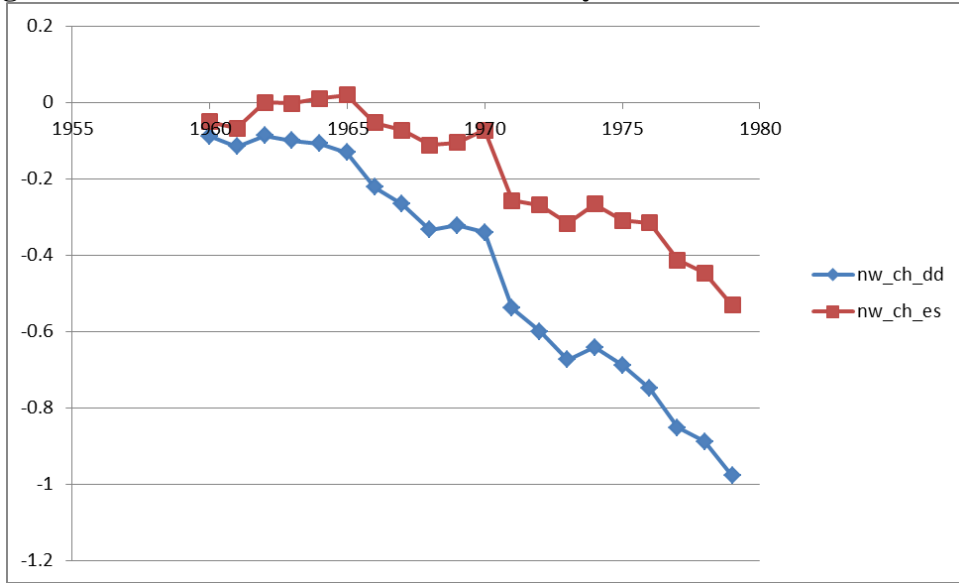
**Figure C-1 Event-Study Estimates for log Child Mortality Using Medicaid Timing**



**Figure C-2 First-stage Estimates Using Medicaid Timing**



**Figure C-3 Year Fixed Effects in Event-Study and Difference-in-Difference**



**Figure C-4 Difference-in-Difference Estimates for log Child Mortality Using Medicaid Timing**

	(1)	(2)	(3)	(4)
<i>A. Log White Child Mortality (0-14)</i>				
Post-Medicaid	0.02	0.02	0.00	0.01
	[0.01]	[0.01]	[0.01]	[0.02]
R2	0.96	0.96	0.97	0.95
<i>B. Log Nonwhite Child Mortality (0-14)</i>				
Post-Medicaid	0.00	0.00	0.01	0.03
	[0.01]	[0.01]	[0.02]	[0.03]
R2	0.93	0.94	0.95	0.83
Covariates	State FE, Year FE	(2) + per- capita income, hospitals and hospital beds	(3) + region-by- year FE	(4), unweighted
Population Weighted?	Y	Y	Y	N

Notes: The p-value from a Hausman test of the difference between the weighted and unweighted estimates (columns 3 and 4) for nonwhite mortality easily reject the null hypothesis that they are equal with a p-value of 0.02. For white mortality the p-value is 0.215.

### Appendix D RE-SCALING QUASI-EXPERIMENTAL ESTIMATES

Quasi-experimental studies first estimate the reduced-form intention-to-treat effect (ITT) of a given policy change on an aggregate mortality rate. The estimating equation (excluding the other covariates for simplicity) relates mortality rates to the policy variable,  $Z_{st}$ :

$$\ln(ASMR_{st}^a) = \beta_0 + \gamma Z_{st} + v_{st}^a \quad (D1)$$

The policy instrument in Currie and Gruber (1996a, b) is the share of a national sample of children or women in the March CPS who are eligible for Medicaid in each state and year. This measure parsimoniously summarizes the cross-sectional and intertemporal variation in eligibility that came from many federal and state policy changes throughout the 1980s. In Meyer and Wherry (2012) the instrument is the discontinuous jump in eligibility that occurs for children born just after September 30, 1983. In Sommers, Baicker and Epstein (2012) the instrument is a dummy for being in a treatment state (NY, ME, AZ) after an expansion of Medicaid eligibility. In the OHIE (2012) the instrument is a dummy for winning the eligibility lottery. In this paper the instrument is the interaction of the high-eligibility dummy with a post-Medicaid dummy.

The proportional reduced-form estimate of  $\gamma$  and the relevant population are shown in table A1. Assuming that the policy only affects mortality through changes in the public

insurance rate, the probability limit of the OLS estimate of  $\gamma$  (the ITT) is  $\frac{\partial \ln(ASMR_{st}^a)}{\partial Z_{st}} = \delta_a \tau$ ,

where  $\delta_a$  is the structural effect of Medicaid coverage on log mortality rates and  $\tau = \frac{cov(m_{st}^a, Z_{st})}{var(Z_{st})}$

is the coefficient from a univariate first-stage regression of  $m_{st}^a$  on the policy variable  $Z_{st}$ . This estimate is not comparable across studies because it is driven both by differences in the implied treatment effect on the treated and by the take-up rate associated with a given policy change. In



order to compare the results of different studies, I calculate the proportional effect of Medicaid on the mortality of the population who became newly insured because of a given policy change.

Medicaid primarily affects poor children and non-elderly adults, whose take-up and mortality rates exceed the average. Writing the public insurance rate and the mortality rate as weighted averages by poverty status allows the reduced form and the take-up estimate to be expressed in terms of the response of mortality and Medicaid use among the poor under the assumption that non-poor households are unaffected. Letting  $p$  be the poverty rate, define  $m_{st}^a = pm_{st,poor}^a + (1-p)m_{st,nonpoor}^a = pm_{st,poor}^a$ , and  $ASMR_{st}^a = pASMR_{st,poor}^a + (1-p)ASMR_{st,nonpoor}^a$ . This implies that the first stage estimate  $\tau$  equals  $p\tau_{poor}$ , and that  $\delta_a$  equals  $p \left[ \frac{\partial \ln(ASMR_{st,poor}^a)}{\partial Z_{st}} \right] \frac{ASMR_{st,poor}^a}{ASMR_{st}^a}$  (because if nonpoor households do not receive public insurance then  $\frac{\partial ASMR_{st,nonpoor}^a}{\partial Z_{st}} = 0$ ). A large body of research suggests that for almost all age groups, races and time periods the mortality rates of the poor exceed the average. Therefore, the proportional effect of Medicaid coverage on the mortality rate of its recipients is the reduced-form effect,  $\gamma$  (a percent change), divided by the first stage coefficient,  $\tau$  (a percentage point change), and the

ratio of poor to overall mortality rates:  $\frac{\gamma}{\tau} \frac{ASMR_{st}^a}{ASMR_{st,poor}^a} = \left[ \frac{\partial \ln(ASMR_{st,poor}^a)}{\partial Z_{st}} \cdot \frac{1}{\tau_{poor}} \right] =$

$$\delta_a \frac{ASMR_{st}^a}{ASMR_{st,poor}^a} .^{37}$$

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<sup>37</sup> The ratio of poor to overall mortality rates equals the ratio of the share of poor decedents to the poverty rate. The mortality rate among the poor,  $\frac{D_p}{N_p}$ , equals  $\frac{S \cdot D}{P \cdot N}$  where  $S$  is the share of deaths to the poor,  $P$  is the poverty rate,  $D$  is the total number of deaths and  $N$  is the population. Dividing by the overall mortality rate,  $\frac{D}{N}$  shows that ,

$$\left( \frac{D_p}{N_p} \right) \left( \frac{D}{N} \right)^{-1} = \frac{S}{P} .$$

**Figure D-1 Proportional Intention-to-Treat Effects of Medicaid on Mortality and Effect on the Level of Medicaid Coverage, Quasi-Experimental Studies**

Paper	Population	Notes	Proportional Reduced Form Estimate	
			Source	Proportional Effect of Policy on Mortality (%)
Currie and Gruber (1996a)	Infants	Smallest overall mortality estimate	Table 3, Column 6, Row 1	$-2.82/9.95 = -0.280$
Currie and Gruber (1996b)	Children		Table VI, Column 1, Row 1	$-1.27/3.76 = -0.340$
Meyer and Wherry (2012)	Black Teens	Smallest mortality estimate, internal causes	Table 7, Column 8, Row 6	$-0.34/0.8/2.55 = -0.167$ **
Sommers, Baicker and Epstein (2012)	Adults		Table 2, Column 2, Row 1	$-19.6/320 = -0.061$
Sommers, Baicker and Epstein (2012)	White Adults		Table 2, Column 2, Row 2	$-14/309 = -0.045$
Sommers, Baicker and Epstein (2012)	Nonwhite Adults		Table 2, Column 2, Row 3	$-41/361 = -0.114$

\*\* Meyer and Wherry (2013) is unique in examining cumulative eligibility over ten years during childhood. They find that by age 18, nonwhite children at the birthdate cutoff who were of the correct age to gain eligibility in the 1980 expansions had accumulated 0.8 more years of Medicaid eligibility on average. Thus, the proportional ITT estimate per year of eligibility equals the discontinuity in period mortality rates for teens (-0.34), divided by the cumulative gain in eligibility for teens (0.8) divided by the baseline mortality rate (2.55). This number, -0.167 is the proportional reduction in teen mortality rates for each year of additional cumulative Medicaid eligibility.

## Figure D-2 Effect of Medicaid on Any Insurance Coverage, Quasi-Experimental Studies

Estimated Effect on Any Insurance Coverage

Paper	Population	Notes	Source	Implied change in any insurance (percentage points)
Dave et al. (2008)	Infants/Mothers	Administrative Data, NHDS	Table 1, Column 7, Row 1	0.13
Cutler and Gruber (1996)	Children		Table IV, Row 3	0.12
Card and Shore-Sheppard (2004)	~7 year old children, family income between 60% and 140% of FPL		Table 3	0.10
Sommers, Baicker and Epstein (2012)	Adults		Table 3, Column 2, Row 1	0.03
Sommers, Baicker and Epstein (2012)	White Adults	Larger than change in Medicaid	Table 3, Column 2, Row 2	0.03
Sommers, Baicker and Epstein (2012)	Nonwhite Adults		Table 3, Column 2, Row 3	0.03

## Figure D-3 Ratio of Poor to Overall Mortality Rates

Paper/Dataset	Population	Notes	Implied poor/average mortality ratio
National Natality Followback Survey (1980)	Infants	26% of infant deaths were to low-income infants (<\$9,000), 23% of live births were to low-income families	$0.26/0.23 = 1.13$
National Longitudinal Mortality Study	Children	26% of child deaths under age 14 were to poor children, and 20% of children were poor (sample is representative of the 1983 population)	$0.26/0.20 = 1.27$
Meyer and Wherry (2012), National Mortality Followback Survey (1993), March CPS (1993)	Black Teens	64% of deaths are to poor black teens, and 36.8% of black teens were poor in 1993.	$0.64/0.368 = 1.74$
National Mortality Followback Survey (1986), March CPS (1986)	All Adults	26% of deaths are to poor adults, 11% of adults were poor in 1986	$0.26/0.11 = 2.39$
National Mortality Followback Survey (1986), March CPS (1986)	White Adults	23% of white deaths are to poor adults, 9.5% of white adults were poor in 1986	$0.23/0.095 = 2.41$
National Mortality Followback Survey (1986), March CPS (1986)	Nonwhite Adults	50% of nonwhite deaths are to poor adults, 22% of nonwhite adults were poor in 1986	$0.50/0.22 = 2.29$

**Figure D-4 Proportional Treatment Effects of Medicaid Coverage on the Treated:  
Adjusting Existing ITT Estimates for Net Insurance Gain and Differential Baseline  
Mortality Levels**

Paper	Population	Lower-bound Proportional ATET estimate*
Currie and Gruber (1996a)	Infants	-182%
Currie and Gruber (1996b)	Children	-190%
Meyer and Wherry (2012)	Black Teens	-81%**
Sommers, Baicker and Epstein (2012)	Adults	-80%
Sommers, Baicker and Epstein (2012)	White Adults	-57%
Sommers, Baicker and Epstein (2012)	Nonwhite Adults	-177%

First stage estimates that are based on survey data (all except for the infant estimates) are adjusted for underreporting by a factor of 0.85 (**Card, Hildreth, and Shore-Sheppard 2004**).

\*Lower-bound in the sense that these are the smallest magnitudes of a negative treatment effect.

\*\*Card and Shore-Sheppard (2004) show that contemporaneous increase in insurance coverage at the birthdate cutoff is at most 10 percent. This calculation assumes that for every year of cumulative eligibility (estimated in Meyer and Wherry [2013]), insurance coverage increases by 0.1 years, which may understate the longer-run Medicaid take-up rate.

## **Chapter 2 THE EFFECT OF MEDICAID IMPLEMENTATION ON CHILDREN'S HEALTH CARE USE AND EXPENDITURES**

In 2012, means-tested public health insurance payments accounted for more than 17 percent of all personal health care outlays in the U.S. and for 3 percent of gross domestic product.<sup>38</sup> The goal of these programs, mainly Medicaid, is to “make medical care of high quality readily available to those unable to pay for it” (**Department of Health, Education and Welfare 1967a**). Despite the large expenditures meant to equalize health inputs, however, income remains a significant predictor of the quantity and quality of health care received (**Currie, Decker, and Lin 2008, Fiscella et al. 2000**).

Medicaid's critics argue that it is unaffordable and ineffective. The 2013 House budget resolution describes Medicaid's costs as “nearly impossible to check” (**House Budget Committee 2013, pg. 38**) and more than a third of governors who oppose the Affordable Care Act's Medicaid expansions feel that it is a “broken program” that “harms its beneficiaries” (**Sommers, Baicker, and Epstein 2012**). Some proponents, on the other hand, argue that health care disparities would be worse without Medicaid and that they persist in areas where public efforts are inadequate (cf. **Coughlin, Long, and Shen 2005, Kellermann and Weinick 2012**).

Empirical research in economics uses program expansions since the 1980s and finds that public insurance increases the use of medical care for poorer recipients, but can have zero or even negative effects for higher-income recipients (**Card and Shore-Sheppard 2004, Currie and Gruber 1996a, b, 2001, Dave et al. 2008, Finkelstein et al. 2012, Sommers, Baicker, and Epstein 2012**). Most Medicaid recipients have historically been poor children, and more recent

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<sup>38</sup> Author's calculations from the National Health Expenditure Accounts.

eligibility expansions have generally affected higher-income families. Thus, while existing estimates are relevant to marginal expansions of Medicaid coverage, they are less well-suited to judge Medicaid's cumulative benefits, the bulk of which may have accrued to the types of disadvantaged children who gained coverage in the 1960s.

This paper provides new evidence on Medicaid's effect on children's health care use and expenditures using the program's original implementation between 1966 and 1970. I bring together several data sources to argue that a large part of Medicaid's effects on health care use probably were associated with its original introduction. First, poor children had much lower insurance rates and used much less medical care in the 1960s than at any time since, and so Medicaid's introduction arguably represented a larger change than later expansions in the availability and cost of medical care. Second, after Medicaid, public coverage increased, uninsurance fell, and the gap in health care use between richer and poorer children closed at rates not seen at any time since.

To provide more formal evidence that Medicaid was responsible for these, I exploit the administrative requirement that state Medicaid programs cover welfare recipients. This meant that many more children were eligible for the new public insurance program in areas with higher rates of welfare receipt. Using data from the 1963 and 1970 Surveys of Health Services Utilization and Expenditure, I estimate triple-difference models that compare changes in health care use and spending before and after Medicaid implementation (first difference), in areas with higher and lower pre-existing welfare rates (second difference), between poor and non-poor children (third difference).

Consistent with an effect of Medicaid, I find that increases in insurance coverage and primary care use for poor children relative to non-poor children were larger in local areas with

higher welfare participation. There is no evidence that hospital admissions increased—in fact, hospital admission rates of poor children fell slightly compared to non-poor children in higher-eligibility areas. The results appear to be driven by nonwhite children, a group with high eligibility for and participation in Medicaid.

## 2.1 Research on Medicaid and Children’s Health Care Use

In 2012, public insurance covered over a third of all children (31 million), but as recently as 1950, the US had no system of public insurance.<sup>39</sup> Figure 2-1 documents this transition in several administrative and survey datasets. Three eras of public coverage growth are clear: Medicaid implementation (1966-1975), the 1980s eligibility expansions (1989-1994) and the roll out of the State Children’s Health Insurance Program (SCHIP) and the Great Recession (2000-2012).

Research on Medicaid implementation relies on cross-sectional comparisons by Medicaid eligibility or participation or on time-series variation in outcomes among eligible families. **Davis and Reynolds (1976)** use data from 1969 to show that public assistance recipients (eligible for Medicaid) have higher predicted utilization than non-public assistance recipients (ineligible for Medicaid). **Loewenstein (1970)** fielded a survey fielded during Medicaid’s roll-out (1968 and 1969) that was specifically designed to assess the effect of Medicaid on poor households. She compares outcomes by eligibility status in states with Medicaid programs, and between respondents in states with and without Medicaid programs. Children who qualified for Medicaid through public assistance had higher primary and acute care utilization than other groups and were less likely to report out-of-pocket expenditures for this care.

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<sup>39</sup> A federally-funded public insurance program for recipients of aid under the Federal Emergency Relief Administration existed during the Depression, but was discontinued upon passage of the Social Security Act. Similarly, some farmers in the late 1930s were covered by pre-payment medical plans organized by agricultural income maintenance programs and local medical societies (Starr 1982, Stevens 1971).

Not all of this work finds that targeted groups used more or better care. **Rogmann, Haggerty, and Lorenz (1971)** compare utilization patterns by Medicaid participation status on a sample of children from Rochester, New York. They show that children on Medicaid are less likely to have a regular source of care, more likely to use the emergency room and had fewer medical contacts. **Klarman (1974)** argues that poor families ineligible for Medicaid would be worse off to the extent that pre-Medicaid charity arrangements were curtailed. **Olendzki (1974)** provides time-series evidence suggesting that this was the case for older welfare recipients in New York City.

In light of these results, researchers have drawn a wide range of conclusions about Medicaid implementation. **Davis and Schoen (1978)** argue that “most of the recent gains of the poor—greater access to adequate health care services, reduced mortality rates and other improvements in health—must be credited to Medicaid” (pg. 50), while **Ginzberg and Solow (1974)** describe Medicaid as “a mistake” (pg. 219). The reliance on cross-sectional or time series variation, however, means that most studies of the implementation period cannot control for underlying differences between eligible and ineligible families, Medicaid and non-Medicaid families, early versus late Medicaid states or for trends in utilization. Thus, the lack of strong research designs makes it difficult to interpret the existing research on Medicaid’s initial roll-out.

Consequently, recent empirical research on public insurance and children exploits variation in eligibility during the second two expansion periods. This work tends to follow the empirical approach of Currie and Gruber (1996a, 1996b), who summarize the effect of several legislative eligibility expansions for children and pregnant women between 1984 and 1992 by calculating Medicaid eligibility on a fixed sample of CPS respondents (either children or women) according to the changing rules in each state. They use this “simulated eligibility”



measure, which captures the policy-induced variation in Medicaid eligibility, as an instrument for children's actual eligibility status in two-stage least squares models of health care use. Their estimates suggest that Medicaid expansions increased primary and acute care use among children as well as the probability that mothers obtained timely prenatal care.<sup>40</sup>

Not all groups experienced increases in utilization in response to the 1980s reforms, though. In particular, expansions that targeted poor pregnant women increased insurance coverage, prenatal care and the use of certain birth technologies (c-sections, fetal monitors, for instance), while expansions that applied to higher-income women had no detectable effects on coverage and actually reduced the use of birth technologies (**Currie and Gruber 2001**). **Card and Shore-Sheppard (2004)** find similar heterogeneity for newly eligible children: those above the federal poverty line very rarely took up Medicaid and were no more likely to have a recent doctor visit, while overall coverage and utilization increased for poor children.<sup>41</sup>

Non-poor infants and children account for the majority of the newly Medicaid-eligible population since the 1980s (Card and Shore-Sheppard 2004, table 2; Currie and Gruber 1996a pg. 436, Currie and Gruber 1996b), and, consistent with the quasi-experimental evidence, figure 2-1 shows that the associated change in uninsurance has been small. The increase in public coverage since 1990 was about 20 percentage points, but the share of uninsured children fell by only about 7 percentage points. This points to the limits of what can be learned about Medicaid's effects from expansions up the income distribution. When higher-income families

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<sup>40</sup> A number of studies examine the same eligibility expansions in individual states and find much smaller effects on utilization (Long and Marquis 1998, Piper, Ray, and Griffin 1990).

<sup>41</sup> A large literature on the crowd-out of private insurance focuses on specification issues related to age-specific trends in insurance coverage during the 1980s and late 1990s. Cutler and Gruber (1996) find that half of new Medicaid children dropped private coverage, which accounts for the small effects on net insurance coverage. Shore-Sheppard (2009) and Card and Shore-Sheppard (2004) argue that the small insurance effects are the results of low take-up and low crowd-out. This interpretation matters from a public finance perspective (high-crowd-out implies a large shift in costs from the private to the public sector), but both scenarios are consistent with the notion that public insurance expansions to higher income groups have small effects on health care utilization.

gain eligibility, they may drop private insurance, health care utilization may change little, and the main effect may be to shift costs from the private to the public sector. Yet for its first 20 years, Medicaid primarily served a group in which none of these responses were likely: very poor children with no other source of health insurance. This suggests that the effects of different types of Medicaid expansions, specifically its introduction, can contribute to an understanding of the program's heterogeneous effects across time periods and recipients with different characteristics.

## **2.2 Children's Health Insurance Status and Health Care Use Before Medicaid**

One reason to expect Medicaid implementation to have had especially strong effects on children's health care use is that poor children had very low levels of insurance coverage and health care in the early 1960s. In other words, Medicaid was very well targeted to children with few sources of payment for medical bills and consequently, low consumption of health services.

### *2.2.1 Sources of Payment for Health Care before Medicaid*

Health insurance was much less common in the mid-20th century than today (**Thomasson 2002**), and it was especially rare for low-income families. Figure 2-1 shows that about 30 percent of all children were uninsured in 1963, but the share is 77 percent for children in the bottom decile of family income (NHIS). Health insurance also primarily covered hospital and surgical care rather than outpatient doctor visits. 15 percent of all children in the 1963 NHIS had doctor visit coverage, compared to less than 5 percent of children in the lowest income decile. The children that Medicaid targeted, those on welfare, would have had even lower coverage rates because most families got coverage through their employer and only 5 percent of mothers on welfare worked in a given year (**Mugge 1960**).

To a limited extent, poor families could rely on charity medical care. **Morgan et al. (1962)** report that 8 percent of families in 1959 received some form of charity care (table 13-1, pg 143). Not only is the level of charity care low, it is not clear charity patients received high quality care. By the early 1960s, hospital outpatient departments, the most common supplier of free care to the poor, had become “crowded, uncomfortable, lacking in concern for human dignity, and to make it worse, no longer free” (**Yerby quoted in Sardell 1988, pg. 46**). Also, charity care was also not always provided altruistically. One hospital administrator in 1966 argued that Medicaid would hamper medical research on the grounds that patients would come to hospitals “only for medical care and [would not] be interested in taking part in new and as yet unaccepted methods of treatment” (**Stevens and Stevens 1974, pg. 99**). Thus, the quality and appropriateness of the available charity care may not have been comparable to paid care.

Publicly financed care was also uncommon. The 1950 amendments to the Social Security Act first authorized welfare offices to receive federal cost sharing for direct payments to medical providers (“vendor payments”) on behalf of welfare recipients. (Previously an allowance for medical care was included in the cash grant.) Most often under this system, as under Medicaid, providers who chose to see welfare patients agreed to accept a pre-arranged fee.<sup>42</sup> Federal matching for vendor payments was subject to per-family caps on the total benefit (cash plus medical), though, so reimbursements that exceeded the difference between the family’s cash grant and the federal cap were the responsibility of the state. This difference was often small, and actually zero for the poorest recipients whose benefits equaled the cap.<sup>43</sup>

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<sup>42</sup> This allowed state welfare agencies to negotiate with providers or, in some states, make monthly contributions to a pooled fund out of which recipients’ medical expenditures were paid (American Public Welfare Association 1953).

<sup>43</sup> From 1950 to 1958 federal matching was limited on a per-case basis. Federal matching was not available for the portion of any recipient’s payments (cash plus vendor payments) that exceeded a federal cap,  $c$ . Federal matching payments equaled  $\sum_i \max(c, s \cdot (g_i + m_i))$  where  $c$  is the per-case limit,  $s$  is the federal share of reimbursable costs,  $g_i$  is recipient  $i$ ’s cash payment and  $m_i$  is recipient  $i$ ’s medical vendor payment. In 1958 federal matching was changed from a per-case to a state-wide averaging basis. Under averaging, federal matching payments then equaled

These limits meant that medical vendor payments were rare, especially for families with children. Federal and state governments spent \$3.8 billion (in 2012 dollars) on vendor payments in 1960, only 12 percent of which went to families with children (**DHEW 1971a**).<sup>44</sup> Furthermore, figure 2-1 shows that in 1963 less than 1 percent of children received such payments. Thus, just before Medicaid's passage, many children, and the vast majority of poor children, were faced with either paying the full cost of their care or relying on charity or public sources, or foregoing care. The strong income gradients in utilization suggest that most chose the latter.

### 2.2.2 *Income and Children's Health Care Use, 1963 and 2006*

The result was both low utilization and a relatively high financial burden for poor families. The solid circles in panel A of figure 2-2 show the share of children (ages 0-19) in the 1963 National Health Interview Survey (**NHIS; Minnesota Population Center and State Health Access Data Assistance Center 2012**) at each income bin who report having seen a doctor within one year. The poorest children in 1963 saw a doctor about half as often as the richest children (43.2 percent versus 79.7 percent). The fitted line comes from a univariate regression on the binned data, and the resulting gradient implies that moving from the poverty threshold for a family of four (\$3,000 in 1960 dollars or \$22,509 in 2012 dollars) to the median income (\$5,600 in 1960 dollars or \$42,017 in 2012 dollars) was associated with an increase in the probability of an MD visit of about 5 percentage points. About half of the gap between the richest and the poorest children, though, appears in the lowest three income bins, suggesting that

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$max\left(c, s\frac{1}{N}\sum_i(g_i + m_i)\right) \cdot N$ , where  $N$  is the public assistance caseload. Under general averaging, the total payments to some individuals were allowed to exceed the per-case limit,  $c$  and still be eligible for reimbursement. No data are available on the extent to which recipients obtained larger vendor payments under averaging than under per-case limits.

<sup>44</sup> Well over half went to the elderly poor.

deep poverty was associated with especially low medical care use.<sup>45</sup> Panel B shows a positive relationship between income and the probability that children were admitted to the hospital in 1963 (in solid squares). The slope estimate for the binned data is not significant, but the poorest children in 1963 were much less likely to use hospital care than the richest children (3.4 percent versus 6.2 percent).<sup>46</sup>

To gauge the magnitude of these differentials, the open symbols in figure 2-2 show the income profile for doctor visits and hospital admissions in 2006. The poorest children (family income less than \$5,000) were almost as likely to see a physician as children in the highest income bin (89 versus 93 percent). The estimated gradient is statistically significant (0.0005, s.e. = 0.0001), but it is one-fifth as large as in 1963 (p-value on the difference in slopes is 0.003). In 2006, lower-income children were actually more likely to have a hospital admission than higher income children. The estimated gradient is not distinguishable from zero, although it is distinguishable from the 1963 slope (p-value = 0.007). Regressing the hospital admissions shares on a dummy for income less than \$15,000 does produce a statistically significant difference, however (0.029, s.e. = 0.007). Note that these crude measures of health care

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<sup>45</sup> Data from the 1963 Survey of Health Services Utilization and Expenditure (SHSUE), described below, show that the steep income gradient in children's use of care holds even after conditioning on the presence of medical symptoms (see appendix figure A1). Respondents are asked whether they had each of 19 symptoms and if they did, whether they sought care for that symptom in 1963. The most common was "sore throat or running nose with a fever as high as 100f for at least two days" (17%) and "getting up some mornings tired and exhausted even with a usual amount of rest" (15%). The least common were "unexplained loss of over ten pounds in weight" (1%) and "unexpected bleeding from any part of the body not caused by accident or injury" (3%). A full list of symptoms is in appendix table A1. 44% of children (1,219) reported any symptom. Poorer children actually report a similar number of symptoms as richer children (about 0.78), although they are more likely to report serious symptoms such as heart pain (7.6 percent of poor children versus 3.2 percent of non-poor children) or sudden bleeding (4.8 percent of poor children versus 3 percent of non-poor children). I calculate the probability of care conditional on having symptoms as the share of reported symptoms for which they sought care. The income gradient (per \$10,000) in that measure is 0.017 (s.e. = .007).

<sup>46</sup> The NHIS data do provide support for a non-linear relationship between MD visits or hospital admissions and income. Adding a quadratic term in income to a regression on the binned data fits the concave relationship well, increasing the  $R^2$  from 0.73 to 0.95 for doctor visits and from 0.19 to 0.40 for hospital admissions. Regressing the probability of hospital admission on a dummy for the two lowest income bins produces an even better fit ( $R^2 = 0.53$ ) and a significant coefficient estimate (-0.011, s.e. = 0.004).

disparities do not reflect the differentials that have been shown to remain in other aspects of health care use such as having a usual source of care or waiting times (**Newacheck, Hughes, and Stoddard 1996**). The main point to draw from figure 2-2 is that even these crude measures of health care use varied much more strongly by income in 1963 than today.

Figure 2-3 provides some evidence that these income gradients were due to families foregoing formal medical care whenever possible because of concerns over costs. The solid symbols in both panels plot the income profile of the share of parents in the 1963 SHSUE (described below) who “strongly agree” with two statements: “I’ll avoid seeing a doctor whenever possible” (panel A) and “The costs of medical care, in general, are much too high” (panel B). These questions were only fielded among adults, and I interpret the parents’ responses as representative of health care use for all family members. The most striking feature of both profiles is the difference between the lowest two income deciles and the rest of the distribution. Almost 40 percent of parents in the lowest decile (and 30 percent in the second decile) avoided doctors, compare to less than 20 percent for higher-income households (difference = 0.192, s.e. = 0.025). The pattern is similar for the cost question, except for the level is shifted up by more than 20 percentage points (difference between bottom 20 and top 80 percent is 0.117, s.e. = 0.034).

The open symbols in figure 2-3 provide evidence on comparable questions from the 2010 Household Tracking Health Survey (**HTHS; Center for Studying Health System Change 2012**). For the top 6 income groups, the share of parents who avoid doctors in 2010 is unchanged from 1963 at about 13 percent. The main difference is that about half as many of the poorest respondents report avoiding doctors, while more respondents near the median income (42,000) report avoiding doctors than in 1963. Panel B shows the share of HTHS respondents

who report putting off or foregoing medical care because of costs. It is difficult to compare the levels of this question the 1963 question on costs, but changes in the pattern of responses by income is similar to panel A, with middle rather than low-income respondents the most likely to put off care because of cost.

Figures 2, figure 2-3, and the statistics on sources of payment show that poor children had few ways to obtain health care in the early 1960s. It is not surprising then that they often went without it. Medicaid extended generous coverage to a large share of this group, and the subsequent changes in coverage and utilization were among the largest in US history.

### *2.2.3 Changes in Children's Insurance Status and Health Care Use After Medicaid*

Medicaid was established by Title XIX of the 1965 Social Security Act (SSA) amendments (P.L. 89-97), and represented one of the biggest ever expansions in children's public insurance use. Figure 2-1 highlights the magnitude of the growth in public coverage (about 13 percentage points), and the historic decline in children's uninsurance that accompanied it (a fall of at least 15 percentage points). That poor children made unprecedented gains in health care use during this period provides another reason why Medicaid's introduction may have had especially strong effects on utilization.

#### Medicaid Implementation, 1966-1970

Medicaid is jointly financed by federal and state governments and so, as in subsequent expansions, states had some discretion over when to begin their Medicaid programs. 26 states, including the largest programs in California and New York, implemented Medicaid in 1966. 11 more states began Medicaid programs in 1967 and the rest of the states, except Arizona and Alaska, implemented Medicaid between 1968 and 1970.<sup>47</sup>

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<sup>47</sup> Half of the 14 states that implemented Medicaid after 1967 did so in January, 1970, the latest possible date. The SSA amendments had required that states run Medicaid programs by that date or else risk the loss of all federal

Medicaid's break with vendor payment programs resulted from three main incentives and mandates. First, Medicaid removed the cap on reimbursements that had hampered vendor payments and offered more generous federal match rates. An open-ended federal commitment made it easier for states to remove limits on care and expand coverage.

Second, Medicaid required coverage for recipients of the categorical cash transfer programs for the blind, disabled, elderly and single-parent families, many of whom had been ineligible for vendor payments.<sup>48</sup> This provision, known as "categorical eligibility", had the largest effect on children through the Aid to Families with Dependent Children (AFDC) program. About 5 percent of all children (but 20 percent of nonwhite children) were eligible this way and more than 86 percent of children who were actually on Medicaid in 1976 qualified through their participation in AFDC.

Third, Medicaid required that states cover at least five services: inpatient hospital, outpatient hospital, laboratory and x-ray, skilled nursing home, and physician services. This requirement was a major change from earlier programs and ensured that Medicaid provided meaningful coverage.<sup>49</sup>

Together these factors led to a nearly ten-fold increase in the share of children with public medical payments. Figure 2-1 shows that in 1965, just prior to Medicaid's passage, about

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vendor payment matching. Alaska did not begin its Medicaid program until 1972 because it claimed that the coverage requirements would make nearly all Native Alaskans eligible and would make the cost "unbearable" (Stevens and Stevens 1974, pg. 61). Arizona did not implement a Medicaid program until 1982 and is dropped from the current analysis.

<sup>48</sup> For the purpose of Medicaid eligibility, state-level restrictions on public assistance receipt such as residency or age requirements were superseded by Title XIX and so poor families who did not receive cash assistance because of these limitations were also a required group. States could also choose to cover and receive federal matching for people who fit one of the public assistance categories (blind, disabled, elderly, or a member of a single-parent family) and did not receive cash assistance but had high medical costs. This was an important source of eligibility for the elderly, but not for children. Title XIX defined several other complicated eligibility groups not discussed here. For a detailed discussion of Medicaid eligibility see Gruber (2003), Advisory Commission on Intergovernmental Relations (1968) and Stevens and Stevens (1974).

<sup>49</sup> States could also choose to cover a range of additional services, including home health care, clinic services, prescription drugs, eye care and dental care.



1.5 percent of children under age 19 received vendor payments. By 1976, almost 15 percent of children received Medicaid benefits. Furthermore, these children were very poor and disproportionately nonwhite. The AFDC eligibility thresholds that determined children's categorical eligibility were almost always below the poverty line at this time—administrative data on recipients from 1967 shows that this was true for 96 percent of families—and benefits were even lower than the eligibility threshold. Categorical eligibility rates (through AFDC) were higher among nonwhite than among white children by a factor of 6.

These gains in public coverage coincided with similarly large reductions in the uninsurance rate among children, which fell from over 30 percent in the early 1960s to under 20 percent by the mid-1970s after states implemented Medicaid. Even though the total growth in children's public insurance rates has been greater in the last 30 years than in the period after Medicaid implementation, the pace was more gradual and the corresponding declines in the share of children without insurance were much smaller.

#### Changes in Income-Based Health Care Disparities, 1963-2010

The timing of changes in children's income-based health care disparities also point to Medicaid as a potential cause. Figure 2-4 plots the value of the income gradient in doctor visit and hospital admission probabilities in each year between 1963 and 2006. For both measures of health care use almost all the convergence between poorer and richer children occurred between the 1960s and the mid-1970s. For doctor visits, the gap between richer and poorer children grew slightly during the 1980s and fell again in the mid-1990s, although this may be an artifact of a change in the relevant NHIS question. Appendix figure A2 shows that the relative gains of poorer respondents, particularly in the probability of doctor visits, are not evident on a sample of nonelderly, childless adults, a group unaffected by Medicaid. Comparing these changes to the

periods of increasing public insurance enrollment shows that the years after Medicaid implementation were some of the only ones when public insurance increased and poor children increased their relative health care consumption.

There was also strong convergence between white and nonwhite children conditional on income (as well as falling income gradients within race). Appendix figures A3 and A4 plot coefficients from regressions on of the following form, estimated separately by year on data collapsed by income bins ( $j$ ) and race ( $r$ ):  $y_{jr} = \alpha_0 + \alpha_1 WHITE_r + \beta_0 INC_j \times (1 - WHITE_r) + \beta_1 INC_j \times WHITE_r + \epsilon_{jr}$ .  $\beta_0$  and  $\beta_1$  are comparable to the gradients in figure 2-4, and measure within-race convergence.  $\alpha_1$  measures between the white/nonwhite difference at the median income (income is measured relative to the median bin). Income gradients fell for both races, but especially for nonwhite children, and nonwhite children gained on average relative to white children. At the median income, nonwhite children were 17 percentage points less likely to see a doctor in 1963, but equally likely in 2006. 12 percentage points of this decline occurred between 1963 and the mid-1970s. Low-income nonwhite children, therefore, gained by far the most in terms of health care use in the 1960s and 1970s both because their consumption increased relative to higher income nonwhite children and because the consumption of all nonwhite children moved closer to that of white children since the 1960s.

### **2.3 Research Design: Categorical Eligibility and Changes in Children's Health Care Use and Expenditures, 1963-1970**

The previous two sections provided evidence consistent with a strong effect of Medicaid implementation on levels and changes in children's health care use. Yet national comparisons over time and by income cannot account for other factors that changed in the 1960s and may have also affected poor children's health care use. For instance, child poverty fell from 27 to 15

percent (**DeNavas-Walt, Proctor, and Smith 2013**), and private insurance coverage rose, both of which could increase consumption through either an income or a price effect. Rapid advances in medical technology (**Clemens 2013**) and its diffusion across providers (**Finkelstein 2007**) increased the health returns to medical care. (These advances, however, mainly addressed conditions common among the elderly but rare among children such as acute care for heart attacks.) Health knowledge and behaviors changed quickly in the 1960s, although this need not have benefitted poor children disproportionately (**Goldman and Lakdawalla 2001**). **Aizer and Stroud (2010)**, for example, show that higher-income mothers reduced their smoking rates more than lower income mothers in the years after the 1964 Surgeon General's report outlining its perceived health risks. Finally, a range of other federal health programs rolled out in the 1960s, such as Community Health Centers or Head Start, although the reach of these programs means that they cannot account for the changes in disparities documented above (the health center program was relatively small and Head Start targeted pre-school age children).

To provide additional evidence on Medicaid's effect on children's health care use and expenditures, I compare utilization before and after Medicaid in areas with higher and lower pre-existing welfare rates. The categorical eligibility requirement meant that after Medicaid implementation, cross-sectional variation in AFDC participation immediately translated into differences in public insurance eligibility. If Medicaid increases health care use, then this suggests that observed changes in utilization should be largest in areas with high categorical eligibility. Furthermore, since the categorical eligibility requirement extended Medicaid to children on welfare, these changes should be concentrated among poor children. My basic approach is to compare changes over time in insurance coverage, health care use and expenditures, across areas with different levels of pre-existing welfare rates, between poor and

non-poor children. A correlation between changes in poor/non-poor gaps in health care use and AFDC rates is consistent with an effect of Medicaid

### *2.3.1 Data on Health Care Use and Categorical Eligibility*

This empirical strategy requires data on health care use and expenditures before and after Medicaid with geographic identifiers that allow me to merge on AFDC-based Medicaid eligibility. Geographic information is not available in the public use NHIS, by far the largest health survey of the time, so I use the 1963 and 1970 waves of the Survey of Health Services Utilization and Expenditure (SHSUE; **Center for Health Administration Studies and National Opinion Research Center 1984a, b**) with specially obtained primary sampling unit (PSU) codes. Because Medicaid eligibility was much higher and more closely tied to AFDC among young children than adults, I begin with a sample of children or grandchildren of household heads who are under age 10. 67 PSUs are in both waves of the survey, which represent at least one, but potentially up to five counties.<sup>50</sup>

The SHSUE sample limits my analysis in several ways. First, I cannot generate evidence on pre-Medicaid trends in health care use or time-varying effects, both of which are important in the results of **Goodman-Bacon (2014)** because there are only two years of data. Second, the sample size presents a challenge to a strategy that relies on comparisons across areas (PSUs), over time, and between groups (by race or poverty, for example). The median number of observations in each PSU-poverty cell is 20. For comparison, the median PSU-poverty-year cell size in the 1963-1970 waves of the NHIS is 43 (and there are 298 PSUs). Because the identifying variation is at the PSU-poverty-year level, the estimator described below will be relatively noisy.

I use two auxiliary data sources to construct race-specific AFDC rates in each PSU. The number of AFDC cases by county are available in a series of federal reports on county-level

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<sup>50</sup> The 1970 wave added extra rural PSUs, which I drop because they have no corresponding 1963 observations.

public assistance. Because nonwhite children were covered at about six times the rate of white children, and race-specific AFDC rates are not strongly correlated, an AFDC measure calculated without respect to race will misrepresent the cross-sectional heterogeneity in nonwhite eligibility. To split the county-level AFDC totals into white and nonwhite cases, I use a large administrative microdataset of AFDC recipients from December 1967 obtained from the National Archives (**Goodman-Bacon 2014; see appendix 1**). This is the only AFDC data with county codes and sufficient sample size to calculate race shares. I use the microdata to calculate the share of cases that are white/nonwhite, multiply it by the county-level caseload total in February 1968 (**DHEW 1968a**) and divide by race-specific county populations of women ages 15-54 to obtain race-specific AFDC rates (among women) by county.<sup>51</sup> This measure can only be constructed in one year and so the PSU-level AFDC rate here differs from that used in **Goodman-Bacon (2014)**, which is measured in each state's year of Medicaid implementation.

The final estimation sample contains 3,504 observations (1,599 in 1963 and 1,806 in 1970). Table 2-1 shows summary statistics by poverty status and year, using the survey weights in 1970 (none were provided in 1963).<sup>52</sup> Average age, family size, income, racial composition and urban shares are similar to those in Census data (one exception is that a lower share poor children in 1970 are white than in the Census).

Panel B of table 2- 1 shows the means of insurance status and three binary measures of health care use. Consistent with figure 2-1, poor children's insurance coverage rises by 14 percentage points, from 33 percent to 57 percent, between 1963 and 1970 while non-poor

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<sup>51</sup> I measure county populations by linearly interpolating between the 1960 Census and the 1969 Surveillance, Epidemiology, and End Results (SEER) population data (Haines and ICPSR 2010, SEER 2013).

<sup>52</sup> The 1963 survey was a flat sample and so no weighting is necessary. The 1970 survey oversampled nonwhites, the elderly and the urban poor and provided post-stratification weights to match the race, SMSA status, family size and income distribution in the 1970 March Current Population Survey. The documentation provided by ICPSR lists an incorrect starting column for the final weight. Finkelstein and McKnight (2008) note that the problem and choose not to weight. Here I read in the correct weight and use it throughout the analysis.

children's coverage increases only 3 percentage points (82 to 85 percent).<sup>53</sup> The utilization variables refer to the previous 12 months and equal one if children had seen a doctor, had a physical/checkup (not necessarily with a doctor), or had a hospital admission. Some convergence between poor and non-poor children is evident for both measures of primary care, but the rates of hospital admissions do not change between the two years.

Panel C shows four measures of expenditures (in 2012 dollars): total health spending, out-of-pocket health spending, the probability of out-of-pocket spending and out-of-pocket share of total expenditures. I follow **Finkelstein and McKnight (2008)** and calculate out-of-pocket expenditures in 1963 as total spending minus insurance payments, which implicitly records any public or charity care funds to out-of-pocket. Poor children spend less than non-poor children and are less likely to have any out-of-pocket spending in both years. The out-of-pocket share, however, falls by much more for poor children than for non-poor children.

### *2.3.2 Empirical Strategy: Comparisons by Categorical Eligibility, Year and Poverty Status*

Figure 2-5 illustrates how I use the AFDC rates and the two waves of the SHSUE to generate evidence on Medicaid's effects. In panel A, I split the sample into 5 bins of the nonwhite AFDC rate and plot nonwhite children's insurance rate for four groups: poor children and non-poor children in 1963 and 1970. The series with circles show that insurance rates were similar in higher- and lower-AFDC areas in both year for non-poor children. The series with

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<sup>53</sup> The SHSUE asked respondents about their insurance status in both waves, but in 1970 Medicaid is specifically *excluded* from the definition of insurance (this is also true in the NHIS until 1982). Respondents are not asked if they are covered by Medicaid, but whether or not they had any "free" care, including Medicaid and care at public clinics (which often would have sought Medicaid reimbursement). I construct a measure of insurance coverage that equals one if children were covered by private insurance or if they reported "free" care in 1970. This is similar to the way the Integrated Health Interview Survey calculates public coverage in 1976.

triangles, however, show that insurance rates were much lower in high-AFDC PSUs for poor children in 1963, but no different in 1970.

Panel B plots the differences between 1963 and 1970s for poor children (dashed line) and non-poor children (solid line) against AFDC rates. The growth in insurance rates for poor children was about 40 percentage points in the highest-AFDC areas and zero in the lowest. The change in insurance rates for non-poor children was about -20 percentage points in the highest-AFDC areas and 20 percentage points in the lowest. This difference in these two series, shown in solid squares, is strongly positively related to the AFDC rate. The fitted line is from an OLS regression on the 5 observations in solid squares and its slope is a triple-difference estimate of Medicaid's effect on nonwhite insurance rates. The first difference is over time, the second difference is between poor and non-poor children and the third difference is between higher- and lower-AFDC PSUs. Consistent with an effect of Medicaid implementation, the result suggests that the relative growth by poverty status in nonwhite children's insurance coverage was 5.44 percentage points greater in areas with one percentage point more women on AFDC in 1967.

I formalize this three-way comparison in a triple-difference specification (DDD):

$$y_{ijt} = \alpha_j + \gamma_0 D_{1970} + \gamma_1 D_{poor} + \phi_0 AFDC_{r(i)j}^* D_{1970} + \phi_1 D_{1970} D_{poor} + \phi_2 AFDC_{r(i)j}^* D_{poor} + \delta AFDC_{r(i)j}^* \times D_{1970} \times D_{poor} + \varepsilon_{ijt} \quad (1)$$

$y_{ijt}$  is one of the outcomes listed in table 2- 1 for person  $i$ , in PSU  $j$ , in year  $t$ . In models that pool both races, observations are always assigned their race-specific PSU-level AFDC rate ( $AFDC_{r(i)j}^*$ ) for the reasons discussed above. I also present models estimated separately on the white and nonwhite samples, which is equivalent to a more demanding specification in which every variable in (1) is interacted with a race dummy. As expected, these estimates are generally less precise.

$\alpha_j$  are PSU fixed effects that control for time- and poverty-invariant differences across PSUs, including any relationship between outcomes and the level of AFDC.  $D_{1970}$  controls for common trends in  $y_{ijt}$ , and  $D_{poor}$  controls for time-invariant differences by poverty status. The two-way interactions ( $AFDC_j * D_{1970}$ ,  $D_{1970} D_{poor}$ , and  $AFDC_j * D_{poor}$ ) allow for differential trends in higher- and lower-AFDC areas, differential trends by poverty status and different correlations between AFDC rates and outcomes for poor and non-poor children. Note that  $\phi_0$ , the coefficient on the interaction of the AFDC rate and the 1970 dummy is a difference-in-differences (DD) estimate of Medicaid's effect on the outcomes of nonpoor children.

The coefficient of interest is  $\delta$ , the linear relationship between AFDC rates and the difference in trends between poor and non-poor children.<sup>54</sup> It bears a direct relationship to the fitted line in panel B of figure 2-5. Each solid square in panel B is the change in insurance for poor children minus the change in insurance for non-poor children. The fitted line is the relationship between those double-differences and the AFDC rate—the triple-difference estimate of  $\delta$ . Because  $\delta$  measures the linear relationship between changes in probabilities and the continuous AFDC rate (measured between 0 and 1), its units implicitly refer to the change in percentage points associated with a one unit difference (ie. 100 percentage points) in AFDC rates.<sup>55</sup>

For simplicity, I present estimates of equation (1) without any additional covariates. Appendix tables A3-A6 shows estimates that also include individual-level covariates (sex, race, and a quadratic function in age included separately for the two survey years), a dummy for the

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<sup>54</sup> In pooled models, variation between white and nonwhite children in insurance status and AFDC rates also help identify  $\delta$ . I present models estimated separately by race as well in which this source of variation is not used.

<sup>55</sup> Although the three utilization outcomes are binary, the main results are coefficients from linear probability models. Appendix tables A7-A9 shows that average marginal treatment effects from logit specifications, calculated as described in Puhani (2012) and Karaca-Mandic, Norton, and Dowd (2012), are very similar.



presence of a Community Health Center in 1970 (**Bailey and Goodman-Bacon 2013**) as well as PSU fixed effects and region-by-race-by-year fixed effects. The 16 region-by-race-by-year fixed effects (4 Census regions, two races and two years) control, among other things, for any effects of the desegregation of specifically Southern hospitals (**Almond, Chay, and Greenstone 2006**) or public investments in school quality (**Stephens and Yang 2013**). Adding these covariates does not change the conclusions, but it usually reduces the t-statistics on the DDD estimates.

#### **2.4 Estimated Effects of Medicaid Implementation on Health Care Use and Expenditures by Poverty Status**

The basis of the research design is the fact that abruptly after Medicaid, higher-AFDC areas had many more children eligible for public insurance than lower-AFDC areas because of the categorical eligibility requirement. Table 2-2 presents estimates from equation (1) suggesting that these eligibility differences corresponded to increases in insurance coverage, especially for poor children. Column (1) shows both the DD estimate (the coefficient on the AFDC times 1970 interaction,  $\phi_0$ ) and the DDD estimate for the full sample.

The relationship between AFDC rates and trends in insurance for non-poor children is near zero (-0.05, s.e. = .093), but the DDD effect for poor children is large, positive and significant at the 10 percent level (2.3, s.e. = 1.36, p-value = 0.097). This means that the implied DD effect for poor children, the sum of the two coefficients in table 2-2, is almost the same as the DDD effect (2.24 versus 2.3), and it is actually even more precisely estimated ( $F_{1,66} = 4.12$ ; p-value = 0.046). The DDD effect suggests that in cells that differed in their AFDC rates by 10 percentage points (about two standard deviations), poor children's insurance coverage increased after Medicaid by 23 percentage points more than non-poor children, or about half of the poor/non-poor gap in 1963. The average AFDC rate across all PSU-race cells is 0.042, which

suggests that Medicaid can account for half ( $.042 \times 2.3 = 0.097$ ) of the 19 percentage point reduction in the poor/non-poor gap in insurance coverage shown in table 2-1.

Columns (2) and (3) present separate estimates by race. The DDD estimate for nonwhite children is more than twice as large as and more precisely estimated than the pooled effect (5.34, s.e. = 2.45, p-value = 0.053) despite having only about one third as many observations. This is mainly because nonwhite AFDC rates vary across PSUs much more than white AFDC rates (standard deviations are 0.08 versus 0.01). Note that this effect is nearly identical to the result in figure 2-5. Compared to the pooled model, the first row of column (2) shows a larger negative relationship between categorical eligibility and changes in insurance for non-poor nonwhite children, but it is not significantly different from zero (-1.97, s.e. = 2.19). The implied DD estimate for poor nonwhite children (3.38) is significantly different from zero ( $F_{1,66} = 4.22$ ; p-value = 0.047).

The DDD estimate for white children in column (3) is even larger and the DD estimate for non-poor children is very close to zero, but the standard errors is almost twice as large as in the nonwhite sample and so it is not distinguishable from zero (6.74, s.e. = 4.47). This highlights the difficulty in using the AFDC-based identification strategy to estimate Medicaid's effect on white children—their overall AFDC rate were so low that there is very little geographic variation to take advantage of in a model like (1).<sup>56</sup> Controlling for the additional fixed effects described above actually induces a large negative DD effect for non-poor white children (-5.29, s.e. = 2.62), while leaving the other estimates largely unchanged (see appendix table E-3).

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<sup>56</sup> Appendix table A7 shows that the marginal DDD effects from a logit specification are nearly identical for the full sample and for nonwhite children, but much smaller for white sample (3.49, s.e. = 3.26).

### 2.4.1 Medicaid's Effect on Primary Care Use by Poverty Status

Table 2-2 suggested that Medicaid increased insurance among poor children, and table 2-3 suggests that it also increased their use of primary care relative to non-poor children. Panel A contains estimates for the probability that children saw a doctor and panel B contains estimates for the probability that they had a physical or checkup in the previous year. The DDD effect on the relative doctor visit probability of poor children is 1.88 (s.e. = 1.1, p-value = 0.094), while the DD point estimate for non-poor children is small (0.25, s.e. = 0.63). Using the average AFDC rate to gauge Medicaid's contribution to poor children's relative gains in terms of doctor visits (14 percentage points, see table 2-1), suggests that Medicaid can account for over half of this convergence ( $0.042 * 1.88 = 0.079$ ).

The DDD estimate for poor children's probability of a checkup, though, is more than twice as large and very precise (3.92, s.e. = 1.26). The relationship between AFDC and changes in non-poor children's checkups is negative (-1.18, s.e. = 0.67), but the implied DD estimate for poor children ( $3.92 - 1.18 = 2.74$ ) remains large and significant ( $F_{1,66} = 4.49$ ; p-value = 0.038).

The estimates for nonwhite children are similar to the pooled estimates, but the standard errors are larger. The DDD estimate for the probability of a doctor visit is 2.41 (s.e. = 2.15), and the magnitude comes at least partly from a negative relationship between increases in the doctor visit probability and AFDC rates for non-poor nonwhite children (DD estimate is -1.38, s.e. = 1.67). The DDD estimate for nonwhite children's physical/checkup probability is larger (3.69) and significant at the 10-percent level (s.e. = 2.02; p-value = 0.075). The large DDD effect in column 2 fits with the fact that the probability of a physical actually did converge strongly by poverty status between 1963 and 1970 for nonwhite children (the change in the gap is about -19 percentage points). This was not true for white children (the change in the gap actually rose by

4.4 percentage points), and their DDD estimate for the probability of a physical is smaller than for nonwhite children and very imprecise (2.36, s.e. = 5.70). The white DDD point estimate for the probability of a doctor visit is slightly larger than for nonwhite children, but it is not distinguishable from zero.

Larger estimates for the probability of a physical than for the probability of a doctor's visit is consistent with other findings that Medicaid recipients do not always see traditional fee-for-service providers. **Rogmann, Haggerty, and Lorenz (1971)** find that many Medicaid recipients used a Neighborhood Health Center, which were staffed with nurses as well as doctors. **Baker and Royalty (2000)** develop a model of public and private physician Medicaid participation and provide evidence that eligibility expansions should increase the share of Medicaid patients in public facilities, but not necessarily private offices. If patients in these settings were more likely to see nurses or physician's assistants than doctors, the increase in checkups could exceed the increase in "doctor" visits.

One specific reason to expect this pattern of effects in the context of Medicaid implementation is that by 1970 Medicaid was developing a special program for "Early and Periodic Screening, Diagnosis and Treatment" (EPSDT). Authorized in 1967, although not fully implemented until the mid-1970s, EPSDT was in place in almost 60 percent of states in January 1970 (**DHEW 1970**). This program placed the burden on states to ensure that young children on or eligible for Medicaid received a comprehensive health screening—something very similar to the outcome variable in panel B of table 2-3.<sup>57</sup> Some state Medicaid programs, such as in Connecticut, provided EPSDT through "neighborhood health centers through Head Start

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<sup>57</sup> Stevens and Stevens (1974, pg. 257 fn. 50) note that this included "a full health history, an analysis of physical growth, developmental assessment, unclothed physical inspection, ear, nose, mouth, and throat inspection, vision testing, hearing testing, anemia testing, sickle cell, TB, urine and lead-poisoning testing, as well as nutritional and immunization status reports."

programs and city health department programs” (DHEW 1970, pg. 34), many of which employed non-MD health professionals and Currier (1977) notes that the screenings can be done “by a nurse or a paramedic, rather than by a physician.” Thus, the findings from the pooled and nonwhite samples that the DDD estimates are larger for physicals or checkups than for doctor visits in general is consistent with this feature of Medicaid’s care for children.

#### 2.4.2 Medicaid’s Effect on Acute Care Use by Poverty Status

Table 2-4 contains the estimated effects on the probability that children had a hospital admission. Both the pooled DDD estimates and those for nonwhite children suggest small negative effects on poor children’s (relative) use of inpatient care, although neither is precisely estimated. The relationship for non-poor children is small in both samples and the DDD effect is larger and negative (-0.78, s.e. = 0.55 for all children; -0.69, s.e. = 0.8 for nonwhite children). Relative to the baseline hospital admission rates, these effects are large. The DDD point estimate in column 1, for example, suggests that at the average AFDC rate, by about 3 percentage points ( $-0.78 \times 0.042 = -0.03$ ), while the actual gap in 1963 was only 1 point. This result is broadly consistent with table 8 in Goodman-Bacon (2014), though, which shows that the relationship between AFDC rates and changes in the share of children using publicly-financed hospital services was small, especially relative to their use of MD services.

The DDD estimate for white children, however, is very large (-4.73) and significant (s.e. = 1.93). Relative to the standard deviation of white AFDC rates (0.012), this estimate suggests that the gap in hospital admissions fell by 5.6 percentage points more in an area with a one standard deviation higher AFDC rate. This magnitude is not consistent with either the levels or average changes in white hospital admissions.

Two factors may contribute to the weaker evidence on nonwhite children's hospital use. First, a higher share of hospital admissions than doctor visits may be unavoidable and, therefore, unaffected by Medicaid. Second, changes in primary care for children may offset hospital admission either because children sought inappropriate inpatient care before Medicaid or because improvements in health obviate the need for hospital admissions. It is also possible that the effects vary by age in a way that I cannot detect in the SHSUE. **Goodman-Bacon (2014)** shows that Medicaid increased the probability that births to poor nonwhite mothers occurred in a hospital. Infants are a only about 10 percent of the estimation sample, though, and it is not feasible to estimate separate DDD effects on this subsample.

#### *2.4.3 Medicaid's Effect on Out-of-Pocket Spending*

For expenditures, table 2-5 presents estimates the share of all spending paid out-of-pocket. This outcome has two main advantages relative to spending levels. First, it eliminates the skewness in the level of total and out-of-pocket spending, which has traditionally presented a challenge to empirical modeling (**cf. Manning et al. 1987**). Second, it implicitly divides out any unobserved, local-level differences in health care prices, which could bias estimates of Medicaid's effect on spending levels.

Medicaid did not charge copays or premiums to the categorically eligible and so to the extent that Medicaid children only received covered services, their out-of-pocket spending would have been zero. However, not all states covered common services such as prescription drugs, and so additional primary care could have increased out-of-pocket expenditures on uncovered services. The results in table 2-5, however, suggest that the out-of-pocket share fell more in higher-AFDC areas among non-poor children, and that for the full sample and for nonwhite children these reductions were actually weaker for the poor. The DD estimates in the first row

are strongly negative and significant for all samples. For columns (1) and (2), the DDD effect is about half this magnitude, positive and relatively precisely estimated (for the white sample the DDD estimate is smaller, negative and very imprecise). The implied DD effects for poor children are themselves significant (-1.5 for all children,  $F_{1,66} = 12$ ,  $p\text{-value} < 0.01$ ; -1.67 for nonwhite children,  $F_{1,37} = 6.23$ ,  $p\text{-value} = 0.017$ ; -4.6 for white children,  $F_{1,66} = 3.54$ ,  $p\text{-value} = 0.06$ ), but the DDD estimates show clearly that the effects on the out-of-pocket share for all and nonwhite children are smaller.

One drawback of the out-of-pocket share is that it is only defined for children with non-zero health expenditures—about 87 and 93 percent of the samples in 1963 and 1970. Appendix table E-10 presents estimates of equation (1) with a dummy for having health expenditures on the left-hand side. Poor children are more likely to report any health expenditure in higher-AFDC areas (consistent with the primary care results in table 2-2), which suggests that the composition of the sample may have changed by poverty status in way that confounds the estimates in table 2-5. It also may be the case the out-of-pocket share masks important effects on the upper tail of spending. **Finkelstein and McKnight (2008)** use the SHSUE to show that Medicare's introduction reduced the upper end of out-of-pocket spending among the elderly. That type of analysis is not possible using this identification strategy in the SHSUE because the sample sizes are too small to calculate points in the distribution of spending within PSU-poverty cells.<sup>58</sup>

Finally, it is possible that Medicaid's most important effects on children's expenditures occurred among non-poor respondents. Medicaid covered the "medically needy", which included children whose income was not itself low enough to qualify them for categorical

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<sup>58</sup> Because Medicare was implemented nationally, Finkelstein and McKnight (2008) are able to use between 500 and 1,000 observations on either side of the age 65 cutoff to calculate spending distributions before and after Medicare in age-based treatment and control groups.

welfare programs, but whose medical expenses were very high. For this group, guaranteed to have high expenditures by definition, Medicaid paid all costs after a family's income minus medical bills fell below a specified threshold. To the extent that the number of medically needy children is correlated with AFDC rates in this sample, then Medicaid could have a large negative effect on measures of out-of-pocket spending for specifically non-poor children. While it is not possible to check this in the SHSUE, it is the case that the share of states with medically needy programs is positively related to AFDC rates.

## **2.5 Conclusion: Medicaid's Early Role in Reducing Income-Based Disparities in Health Care**

This paper provides new evidence that Medicaid implementation increased children's insurance coverage and primary care use among poor children and reduced out-of-pocket spending more broadly. This is among the first quasi-experimental evidence on the effect of Medicaid implementation on health care use. My estimates suggest that Medicaid played a strong role the unprecedented national convergence in health care use and insurance between poor and non-poor children during the 1960s and 1970s. For all children, it can account for about half of the convergence between 1963 and 1970 in insurance rates and the probability that children had a doctor's visit. Medicaid's effect on the probability that children had a checkup (not necessarily with a doctor) is even larger, which is consistent with the new screening effort in place in about half the states by 1970.

These findings provide additional support for research design in **Goodman-Bacon (2014)** based on pre-Medicaid welfare participation and its statutory connection to Medicaid eligibility. They are also consistent with the finding that Medicaid implementation reduced child mortality mainly through increases in children's use of primary care (**Goodman-Bacon 2014**). The results



fit with more recent evidence on the heterogeneity of Medicaid's effect on health care use in higher- and lower-income families (**Card and Shore-Sheppard 2004, Currie and Gruber 1996b, 2001**): coverage and utilization increase the most for poor children, but out-of-pocket spending appears to fall the most for non-poor children. The limitations of the available health expenditure data, however, prevent a more detailed analysis of Medicaid and health spending.

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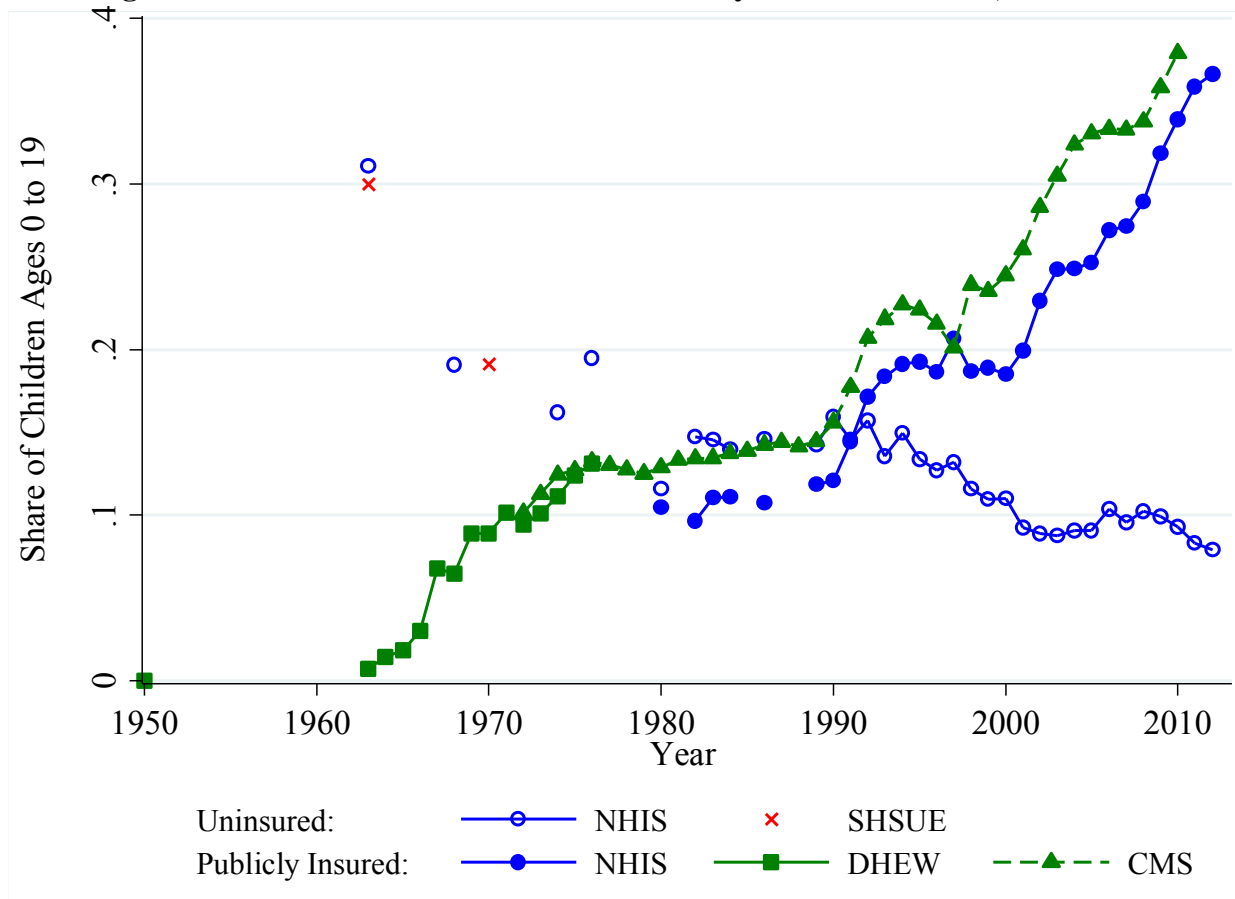
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**Figure 2-1 The Share of Uninsured and Publicly Insured Children, 1950-2012**



Notes: The figure plots the share children ages 0 to 19 that received some form of means-tested public insurance or were uninsured from 1950 to 2012. The 1963, 1968 and 1974 data are obtained from ICPSR National Health Interview Survey (NHIS) files. Children are classified as having no insurance if they report not having hospital insurance, surgical insurance or doctor insurance or (in 1968 and 1974) if they do not list coverage through “Medicare, Medicaid or welfare” as a reason for not having insurance (children with no valid response or who do not know whether they have any type of insurance are excluded). The data from 1976-2012 are obtained from the Integrated Health Interview Survey Files. Uninsurance is based on the variable HASNOCOVR (which is calculated similarly) and public insurance is based on the variable HIPUBLICR (which includes Medicaid and CHIP receipt).

The share of uninsured children in the SHSUE is calculated in a similar way. In 1963, I use a direct question on the number of health insurance policies. In 1970, I use responses to a direct question on insurance coverage and also count children as insured 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.”

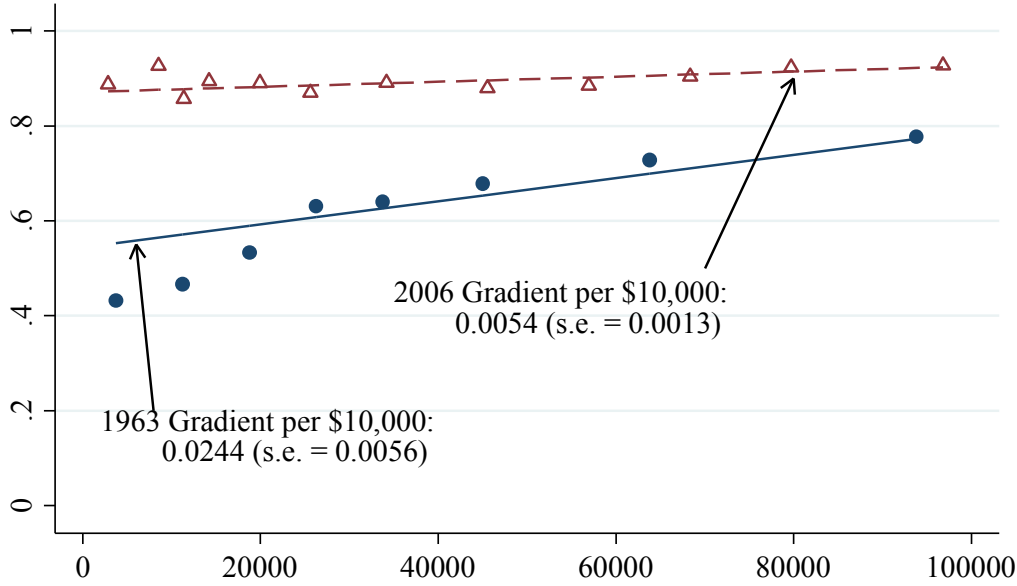
The solid squares and triangles are based on administrative data, and show the ratio of unduplicated annual counts of Medicaid recipients (rather than enrollees) to the population age 0 to 19. For a description of the Department of Health, Education and Welfare data see Goodman-Bacon (2014), appendix 1. The Center for Medicare and Medicaid Services (CMS) data are from the 2012 Medicare and Medicaid Statistical Supplement Table 13.4, which gives the unduplicated annual number of Medicaid beneficiaries (not enrollees). Population denominators are from the Survey, Epidemiology, and End Results (SEER) data and the 2000-2010 intercensal population estimates.

Sources: DHEW (various years); **Center for Medicare and Medicaid Services (2012)**; **Center for Health Administration Studies and National Opinion Research Center (1984a, b)**; **United States Department of Health and Human Services, Centers for Disease Control and Prevention, and National Center for Health Statistics (2010a, b, c)**; **Minnesota Population Center and State Health Access Data Assistance Center (2012)**.

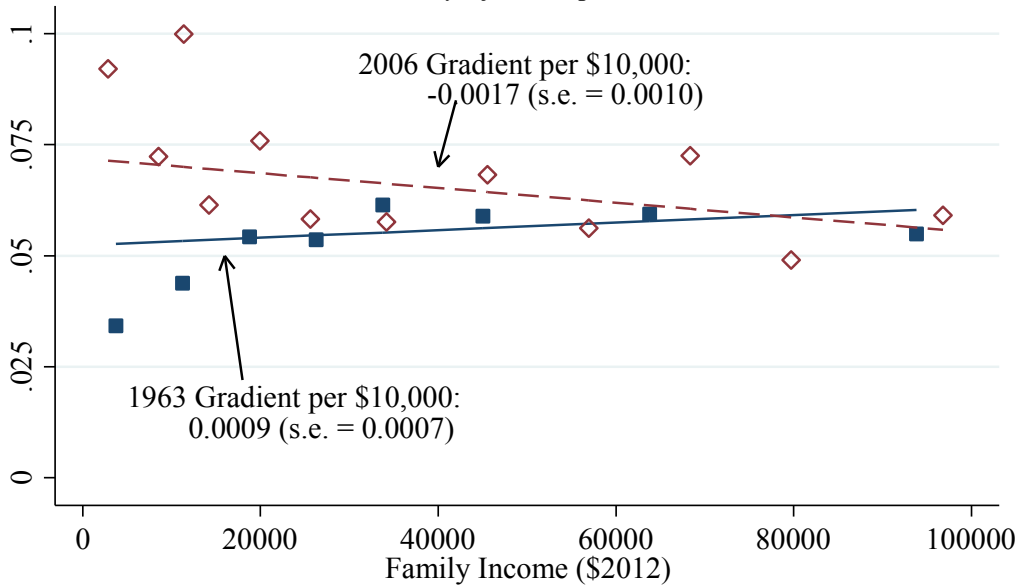


**Figure 2-2 Income Profiles of Children’s Health Care Use, 1963 and 2006**

*A. Probability of a Doctor Visit*

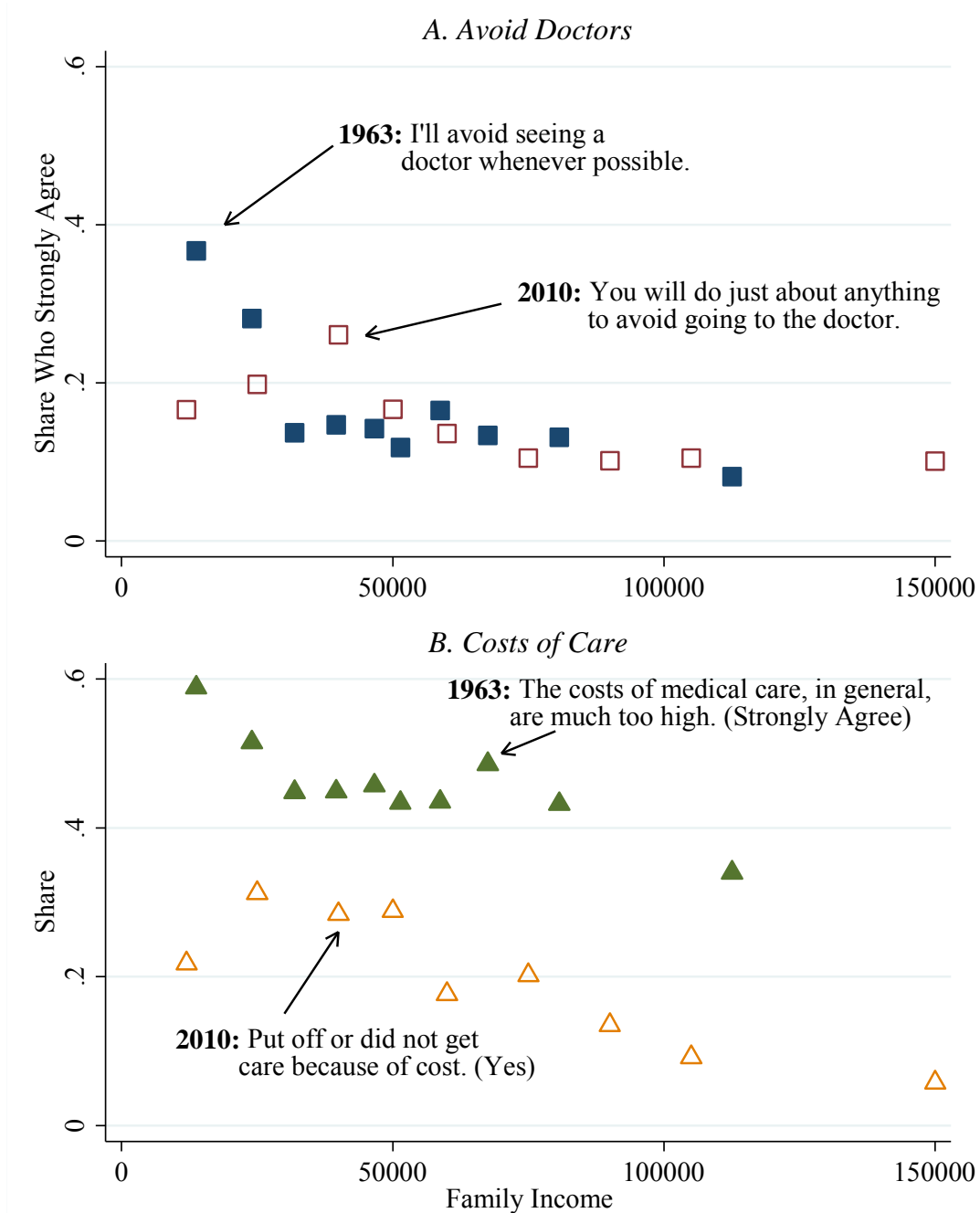


*B. Probability of a Hospital Admission*



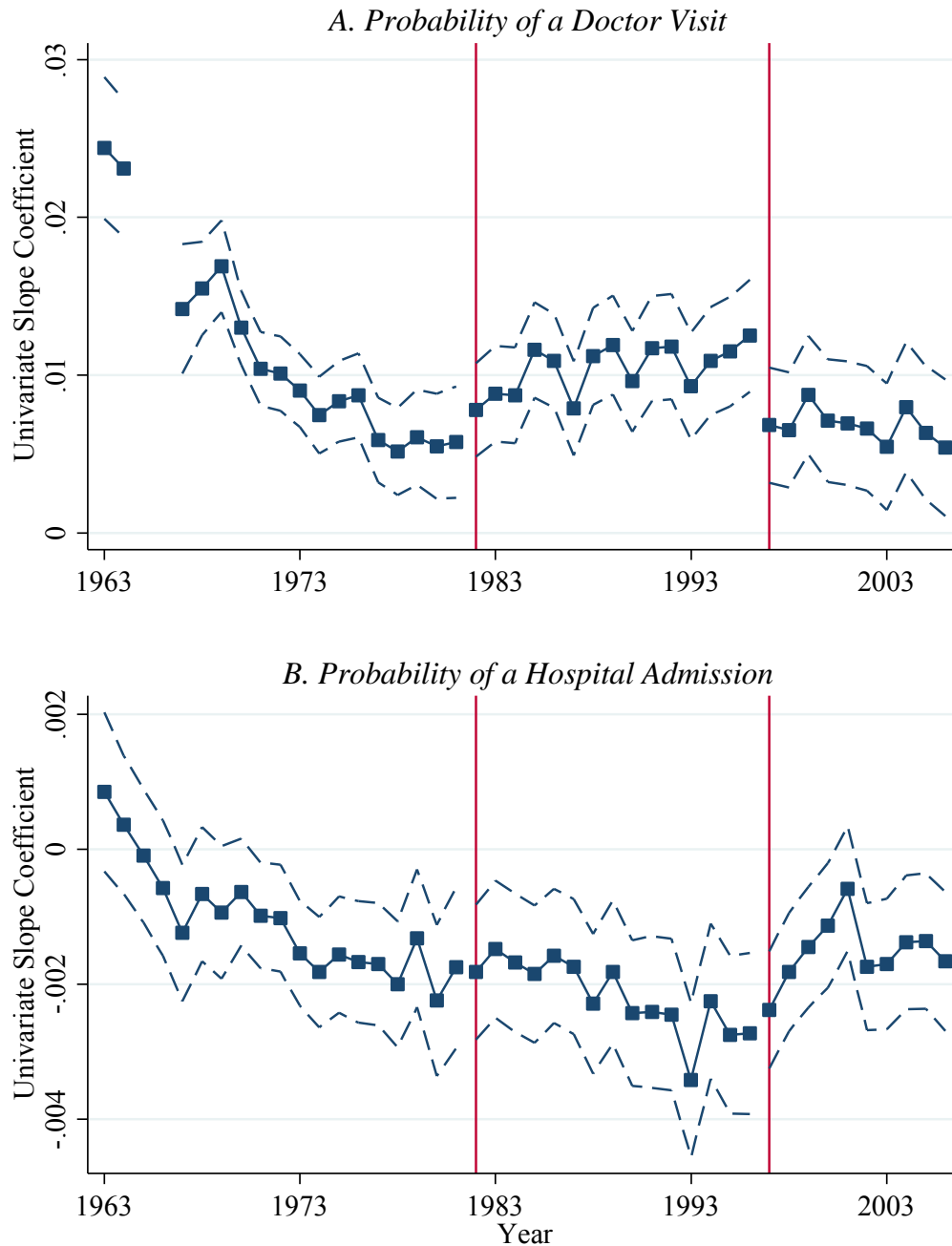
Notes: Panel A shows the relationship between children’s health care use and family income in 1963 and 2006 using data from the National Health Interview Survey. Panel A plots the share of children (and grandchildren) under age 18 who report having seen a “medical doctor” (in 1963) or a “doctor or other health care professional” (in 2006) within one year, by bins of family income. The income profile for 1963 is in solid circles and for 2006 it is in open triangles. I assign respondents the midpoint of each income bin and adjust the values using the CPI-U to be in 2012 dollars. The shares are calculated using the person-level survey weights. The lines represent predicted values from a univariate OLS regression on the binned data weighted by the sum of the survey weights in each bin. Gradient refers to the estimated slope coefficient. Panel B is constructed in the same way but the outcome variable is the probability that children had a hospital admission in the last year. The 1963 (2006) profile is in solid squares (open diamonds). Source: **Minnesota Population Center and State Health Access Data Assistance Center (2012)**

**Figure 2-3 Income Profiles of Parent’s Opinions About Health Care Use and Costs, 1963 and 2010**



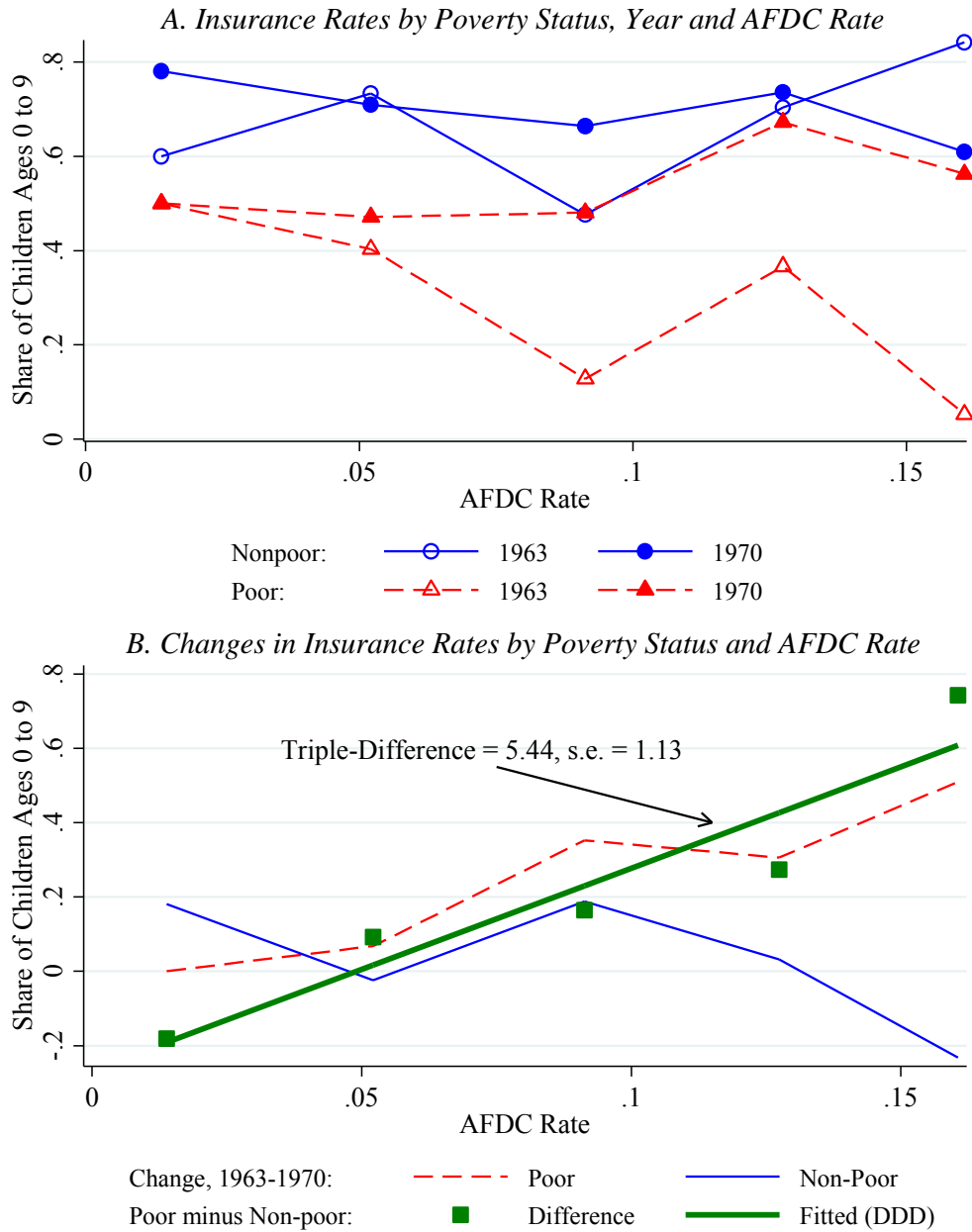
Notes: The figure plots the responses of parents in the 1963 Survey of Health Services Utilization and Expenditure and the 2010 Household Tracking Health Survey (HTHS). Panel A plots the share of parents in each income bin who “strongly agree” with the noted statements about “avoiding” doctors. Panel B plots the share of parents in 1963 who “strongly agree” with the noted statement about medical costs and the share of parents in 2010 who report having put off or foregone care because of “worry about the cost”, “the doctor or hospital wouldn’t accept your health insurance” or “your health plan wouldn’t pay for the treatment.” I assign respondents the median value of income within each decile and adjust these values using the CPI-U to be in 2012 dollars. I assign top-coded values in the HTHS \$150,000. Source: **Center for Health Administration Studies and National Opinion Research Center (1984a, b), Center for Studying Health System Change (2012)**

**Figure 2-4 The Income Gradient of Children’s Health Care Use by Year, 1963-2006**



Notes: The figure plots the income gradient in doctor visit and hospital admission probabilities in the NHIS between 1963 and 2006. (Figure 2 shows the 1963 and 2006 gradients.) Each dot represents the estimated slope coefficient from a regression on each year of data collapsed by income category ( $j$ ) of the form:  $y_j = \alpha + \beta \frac{Income_j}{10,000} + \epsilon_j$ . The dashed lines are unadjusted 95-percent confidence intervals. The text of the doctor visit question changed twice during this period (denoted by vertical lines). The text from 1963-1981 is “About how long has it been since [person] saw or talked to a medical doctor?”; from 1982-1996 is “About how long has it been since [person] last saw or talked to a medical doctor or assistant?”; and from 1997-2012 is “About how long has it been since [you/anyone in the family] last saw or talked to a doctor or other health care professional about [your own/sample child's] health?” The main change to the hospital data came in 1997, when self-reports were used as opposed to responses checked against hospital records (1963-1996). Source: See notes to figure 2-2.

**Figure 2-5 The Relationship Between Categorical Eligibility and Nonwhite Children’s Insurance Rates by Poverty Status**



Notes: Panel shows the share of nonwhite children who had some form of insurance (either private or, in 1970, reported a payment from a “public” source) by poverty status (non-poor children in blue circles with solid lines, poor children in red triangles with dashed lines), year (1963 in open symbols, 1970 in closed symbols) for 5 bins of the nonwhite AFDC rate. Non-poor children’s insurance rates were unrelated to AFDC in both years. The relationship is negative for poor children in 1963 but flat in 1970. Panel B shows the change over time in insurance status for poor children (dashed red line) and non-poor children (solid blue line) by bins of the AFDC rate, and the solid green squares show the difference between the two. The thick solid line is a linear fit through these differences, and its slope is a triple-difference estimate of Medicaid’s effect on nonwhite children’s insurance coverage. The first difference is pre/post Medicaid, the second difference is between the bins of AFDC (ie. categorical eligibility) and the third difference is between poor and non-poor children. Source: see notes to table 2-1.

**Table 2-1 Survey of Health Services Utilization and Expenditure Child Sample Characteristics**

	(1)	(2)	(3)	(4)
	1963		1970	
	Poor	Non-Poor	Poor	Non-Poor
<i>A. Demographics</i>				
Age	4.70 (2.89)	4.67 (2.81)	4.72 (3.05)	4.92 (2.85)
Family Size	5.92 (1.26)	5.08 (1.27)	5.37 (1.56)	5.06 (1.38)
Family Income (\$1970)	3,487 (1,557)	10,112 (6,503)	3,242 (1,342)	12,459 (8,112)
Share White	0.55 (0.5)	0.89 (0.32)	0.52 (0.5)	0.89 (0.32)
Share Urban	0.59 (0.49)	0.69 (0.46)	0.80 (0.4)	0.76 (0.43)
<i>B. Utilization</i>				
Share with Insurance	0.33 (0.47)	0.82 (0.38)	0.57 (0.5)	0.85 (0.36)
Share with a Doctor Visit	0.47 (0.5)	0.78 (0.42)	0.60 (0.49)	0.77 (0.42)
Share with a Physical/Checkup	0.40 (0.49)	0.65 (0.48)	0.45 (0.5)	0.67 (0.47)
Share with a Hospital Admission	0.06 (0.24)	0.07 (0.25)	0.06 (0.25)	0.07 (0.26)
<i>C. Expenditures</i>				
Total Expenditures	35.0 (104.2)	63.2 (105.9)	68.1 (204.4)	106.2 (208.2)
Out-of-Pocket Expenditures	27.3 (85.8)	48.3 (70.9)	17.3 (37.8)	57.3 (90.6)
Share with Out-of-Pocket Expenditure	0.72 (0.45)	0.91 (0.29)	0.69 (0.46)	0.93 (0.26)
Share of Expenditures Out-of-Pocket	0.97 (0.16)	0.91 (0.24)	0.51 (0.47)	0.80 (0.33)
Observations	371	1,228	621	1,185

Notes: Sample includes kids under 10 who are children or grandchildren of the household head. This is the same sample used in the regression estimates. 1970 means are calculated using sample weights. Source: **Center for Health Administration Studies and National Opinion Research Center (1984a, b)**

**Table 2-2 The Relationship between Categorical Eligibility and Changes in Children’s Insurance Rates by Poverty Status**

Dependent Variable:	(1)	(2)	(3)
	<i>Has Private Insurance or Public Medical Payments (including Medicaid)</i>		
AFDCj × 1 {1970}	-0.05	-1.97	-0.11
	[0.93]	[2.19]	[2.96]
AFDCj × 1 {1970} × Poor	2.30	5.34	6.74
	[1.36]	[2.45]	[4.47]
P(Insurance), Non-poor, 1963	0.82	0.68	0.84
P(Insurance), Poor, 1963	0.33	0.30	0.36
R2	0.26	0.30	0.24
Observations	3,405	1,202	2,203
PSUs	67	40	67
Sample	All Children	Nonwhite Children	White Children

Notes: The table presents estimated coefficients from equation (1) on the interaction of AFDC rates and a 1970 dummy and the triple interaction of those two variables with a dummy for poor children. The outcome variable equals one for children who are covered by private insurance or, in 1970, report a payment from a “public” source. Standard errors are clustered at the PSU-level. Source: see notes to table 2-1.

**Table 2-3 The Relationship between Categorical Eligibility and Changes in Children's Primary Care Use by Poverty Status**

	(1)	(2)	(3)
Dependent Variable:	<i>A. Saw a Doctor in the Last Year</i>		
AFDCj × 1 {1970}	0.25	-1.38	3.55
	[0.63]	[1.67]	[2.69]
AFDCj × 1 {1970} × Poor	1.88	2.41	3.32
	[1.1]	[2.15]	[4.44]
P(Doctor Visit), Non-poor, 1963	0.78	0.65	0.79
P(Doctor Visit), Poor, 1963	0.47	0.30	0.62
R2	0.15	0.28	0.09
Observations	3,405	1,202	2,203
PSUs	67	40	67
Dependent Variable:	<i>B. Had a Physical or Checkup in the Last Year</i>		
AFDCj × 1 {1970}	-1.18	-1.84	2.37
	[0.67]	[1.42]	[2.28]
AFDCj × 1 {1970} × Poor	3.92	3.69	2.36
	[1.26]	[2.02]	[5.7]
P(Physical), Non-poor, 1963	0.65	0.66	0.65
P(Physical), Poor, 1963	0.40	0.43	0.38
R2	0.13	0.22	0.15
Observations	2,974	1,036	1,938
PSUs	67	39	67
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-2. The dependent variables equal one for children who saw a physician during the survey year (panel A) and who had a physical or a checkup during the survey year (panel B). Source: see notes to table 2-1.

**Table 2-4 The Relationship between Categorical Eligibility and Changes in Children's Hospital Admissions by Poverty Status**

Dependent Variable:	(1)	(2)	(3)
	<i>Had a Hospital Admission in the Last Year</i>		
AFDCj × 1 {1970}	0.34	0.15	1.83
	[0.28]	[0.36]	[1.01]
AFDCj × 1 {1970} × Poor	-0.78	-0.69	-4.72
	[0.55]	[0.8]	[1.93]
P(Hospital Admission), Non-poor, 1963	0.07	0.03	0.07
P(Hospital Admission), Poor, 1963	0.06	0.06	0.06
R2	0.04	0.09	0.05
Observations	3,405	1,202	2,203
PSUs	67	40	67
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-2. The dependent variable equals one for children who were admitted to the hospital overnight during the survey year. Source: see notes to table 2-1.



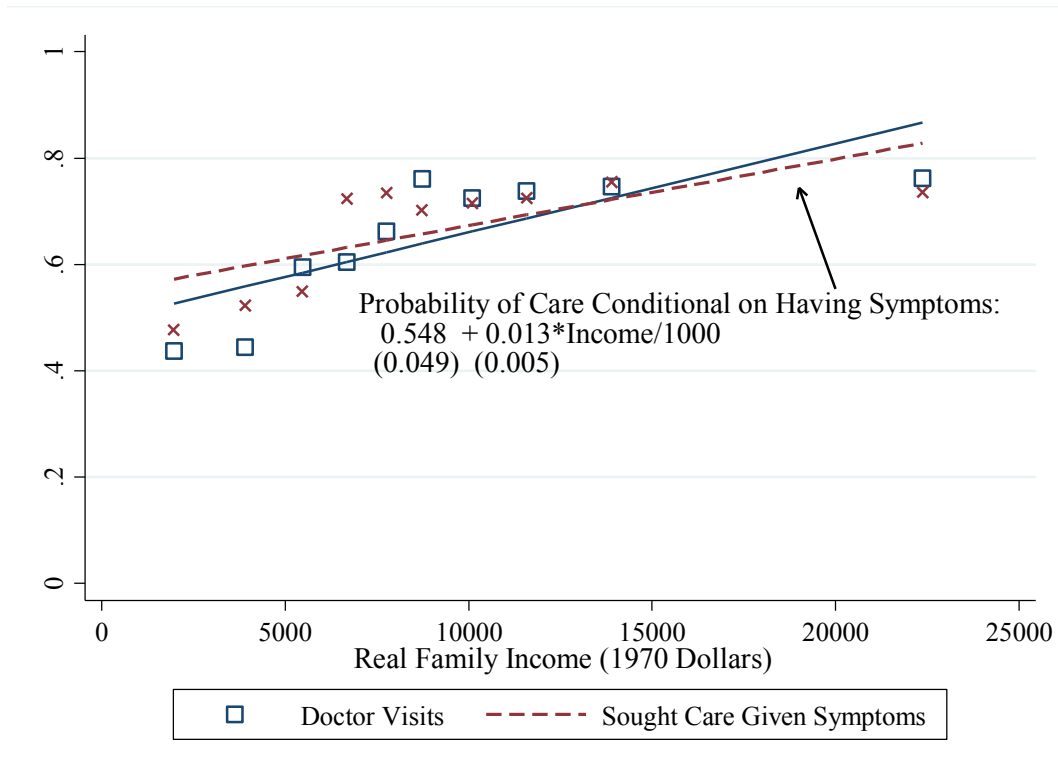
**Table 2-5 The Relationship between Categorical Eligibility and Changes in Children's Out-of-Pocket Expenditures by Poverty Status**

Dependent Variable:	(1)	(2)	(3)
	<i>Out-of-Pocket Share of Health Spending</i>		
AFDCj × 1{1970}	-2.83	-3.29	-3.70
	[0.51]	[0.73]	[1.32]
AFDCj × 1{1970} × Poor	1.34	1.62	-0.91
	[0.68]	[0.85]	[2.21]
Out-of-Pocket Share, Non-poor, 1963	0.92	0.96	0.91
Out-of-Pocket Share, Poor, 1963	0.97	0.99	0.96
R2	0.20	0.46	0.15
Observations	2,935	913	2,022
PSUs	67	38	67
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-2. The dependent variable equals one for children who were admitted to the hospital overnight during the survey year. Source: see notes to table 2-1.

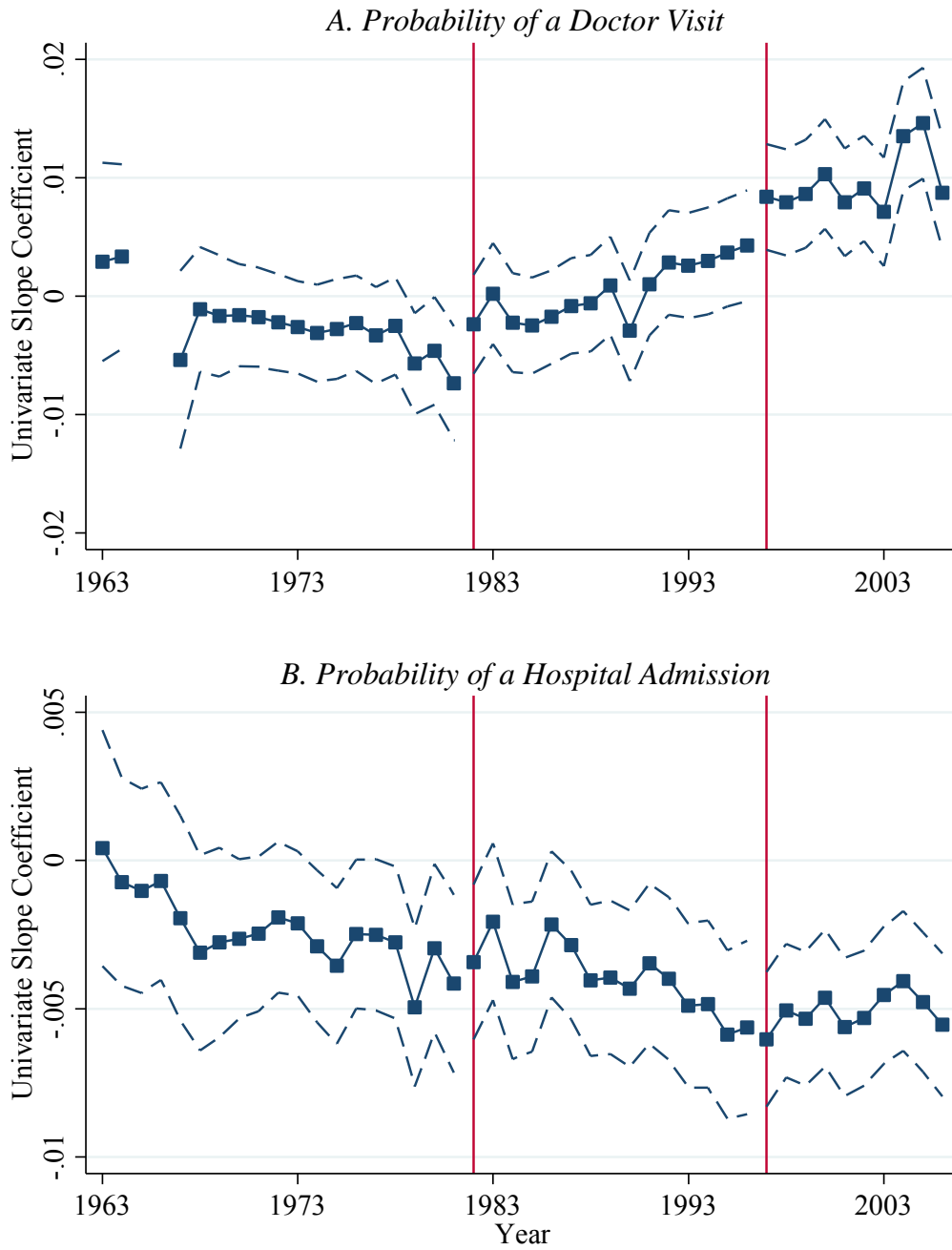
### Appendix E ADDITIONAL RESULTS

**Figure E-1 Income Profile of Children's Doctor Visits with and without Conditioning on Symptoms**



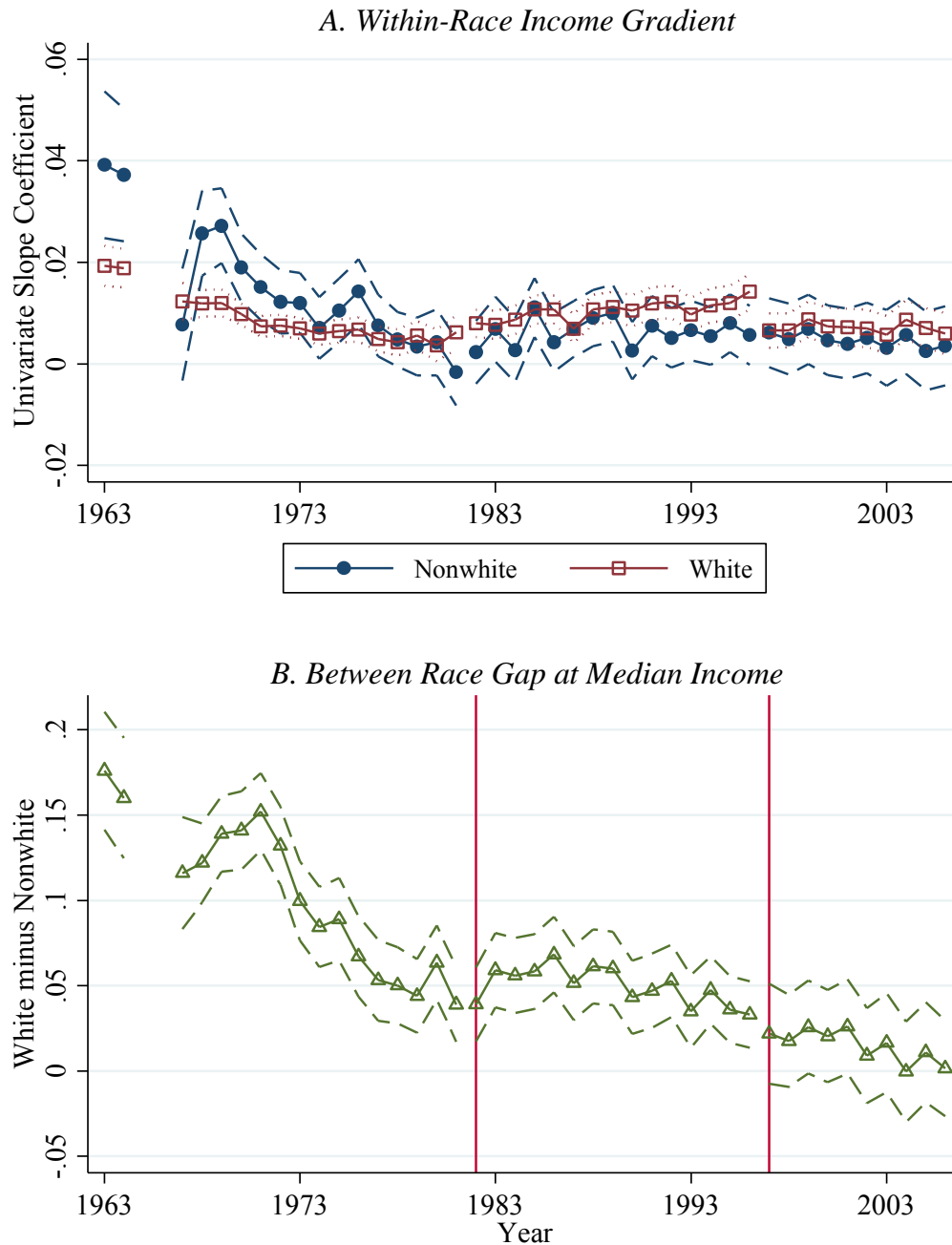
Notes: The figure is constructed the same way as figure 2-2 except using the SHSUE data. The x's incorporate information on children's symptoms (see text and table E-1).

**Figure E-2 Income Gradients for Nonelderly Household Heads without Children**



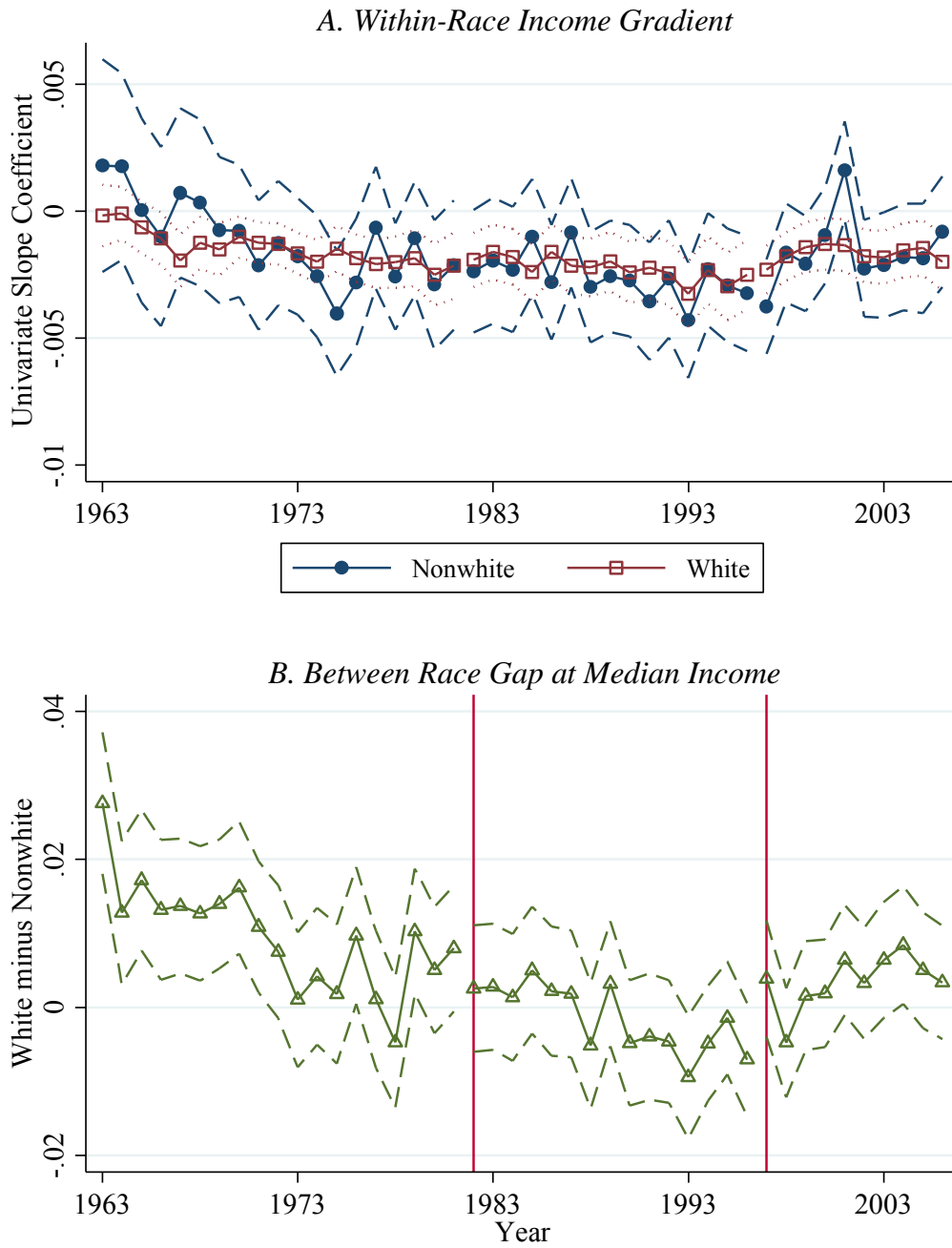
Notes: See notes to figure 2-4.

**Figure E-3 Within and Between Race Convergence in Doctor Visits**



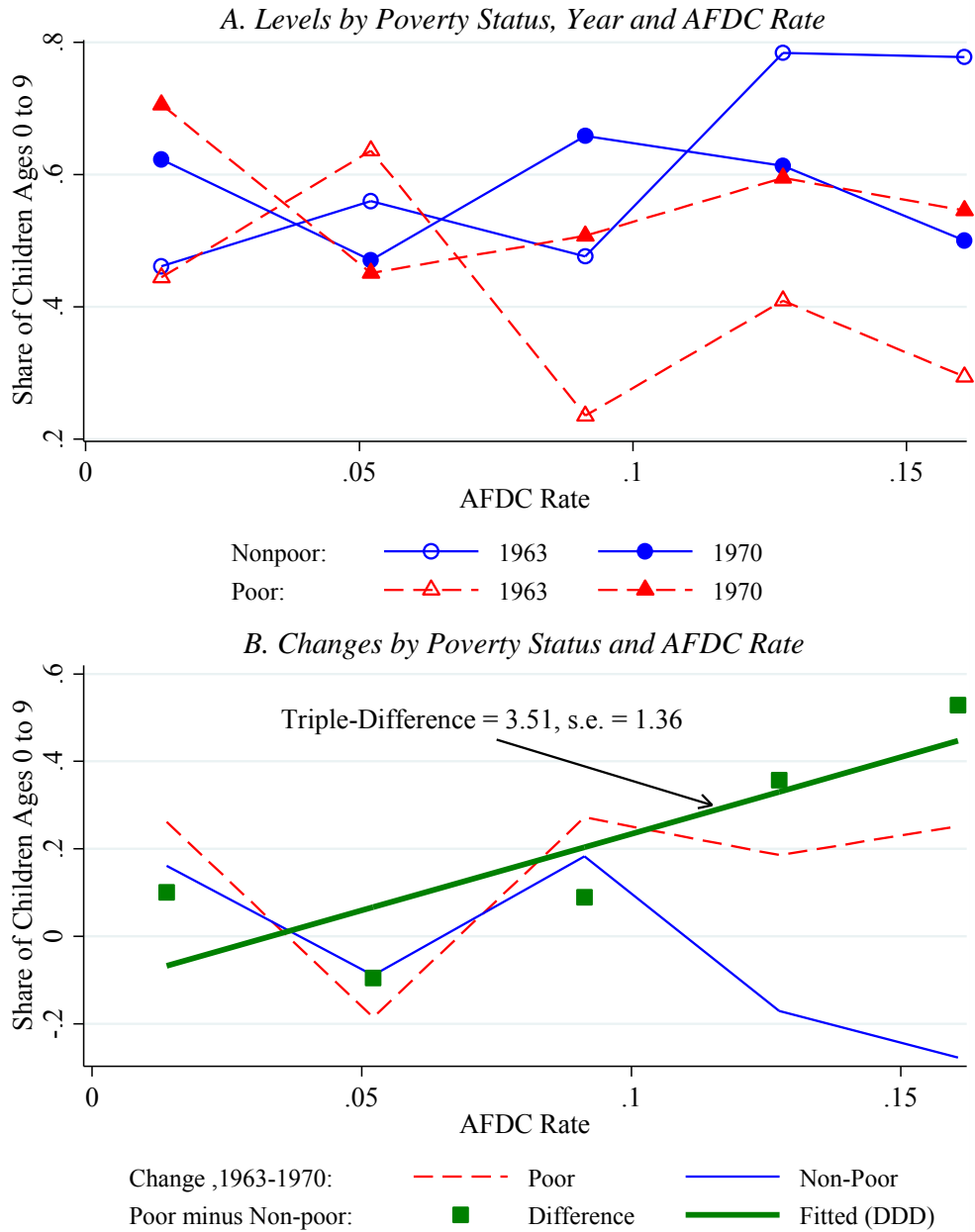
Notes: I collapse the NHIS data to the income-bin ( $j$ ), race ( $r$ ) and year ( $t$ ) level. The figure plots coefficients from the following regression estimated by year:  $y_{jr} = \alpha_0 + \alpha_1 WHITE_r + \beta_0 INC_j \times (1 - WHITE_r) + \beta_1 INC_j \times WHITE_r + \epsilon_{jr}$ . Panel A plots estimates of  $\beta_0$ , the income gradient for nonwhite children and  $\beta_1$ , the income gradient for white children. Panel B plots estimates of  $\alpha_1$ , the racial gap in utilization at the median income ( $INC_j$  represents median income within each bin relative to the level of income in the median bin). The dashed lines are unadjusted confidence intervals. The mean values of  $y_{jr}$  are calculated using the person-level survey weights and the regression are weighted by the sum of the survey weights for respondents with non-missing outcome data in each bin.

**Figure E-4 Within and Between Race Convergence in Hospital Admissions**



Notes: See notes to figure E-3.

**Figure E-5 The Unadjusted Relationship Between Categorical Eligibility and Nonwhite Children's Probability of a Recent Checkup by Poverty Status**



Notes: See notes to figure 2-5. The dependent variable equals one for children who report having had a physical or checkup in during the previous year. Source: see notes to table 2-1.

**Table E-1 SHSUE Symptom Questions**

Did anyone have (symptom) during 1963?	Respondents	Had Symptom in 1963		Did not Seek Care in 1963	
		Number	Share	Number	Share Given Symptom
cough any time during the day or night which lasted for three weeks?	2,611	162	(0.06)	39	(0.24)
sudden feelings of weakness or faintness?	2,611	52	(0.02)	18	(0.35)
getting up some mornings tired and exhausted even with a usual amount of rest?	2,609	66	(0.03)	46	(0.7)
frequent headaches?	2,613	73	(0.03)	28	(0.38)
skin rash or breaking out on any part of the body?	2,614	278	(0.11)	66	(0.24)
diarrhea (loose bowel movements) for four or five days?	2,610	86	(0.03)	25	(0.29)
shortness of breath even after light work?	2,613	22	(0.01)	6	(0.27)
waking up with stiff or aching joints or muscles?	2,611	16	(0.01)	8	(0.5)
sudden feelings of weakness or faintness? **	2,606	14	(0.01)	5	(0.36)
frequent backaches?	2,612	13	(0.0)	7	(0.54)
unexplained loss of over ten pounds in weight?	2,614	2	(0.0)	0	-
repeated pains in or near the heart?	2,610	15	(0.01)	5	(0.33)
repeated indigestion or upset stomach?	2,610	42	(0.02)	19	(0.45)
repeated vomiting for a day or more?	2,614	120	(0.05)	57	(0.48)
sore throat or running nose with a fever as high as 100f for at least two days?	2,607	673	(0.26)	201	(0.3)
nose stopped up, or sneezing, for two weeks or more?	2,610	179	(0.07)	49	(0.27)
unexpected bleeding from any part of the body not caused by accident or injury?	2,612	65	(0.02)	32	(0.49)
abdominal pains (pains in the belly or gut) for at least a couple of days?	2,610	54	(0.02)	23	(0.43)
any infections, irritations, or pains in the eyes or ears?	2,611	245	(0.09)	37	(0.15)
Share with Any Symptom	2,778	1,219	(0.44)		

Notes: the table shows responses for all children under 18.

**Table E-2 SHSUE Opinion Questions**

	Share that "Strongly Agree"	
	1963	1970
If you wait long enough, you can get over most any disease without getting medical aid.	0.03	0.03
Good personal health depends more on an individual's strong will power than on vaccinations, shots, and vitamins.	0.10	0.05
Some home remedies are still better than prescribed drugs for curing illness.	0.07	0.04
No matter how well a person follows his doctor's orders, he has to expect a good deal of illness in his lifetime.	0.15	0.08
A person understands his own health better than most doctors do.	0.11	0.06
Modern medicine can cure most any illness.	0.20	0.09
The medical profession is about the highest calling a man can have in this country.	0.29	0.19
Most doctors are more interested in their incomes than in making sure everyone receives adequate medical care.	0.10	0.11
Choosing your own doctor is about the most important thing in getting good medical care.	0.46	0.33
The care I have generally received from doctors in the last few years was excellent.	0.51	0.29
Even if a person is feeling good, he should get a general physical examination every year.	0.51	
I'll avoid seeing a doctor whenever possible.	0.17	
I wouldn't go to a hospital unless there was just no other way to take care of me.	0.24	
If a doctor told me I needed a major operation, I would have it done immediately.	0.36	
I do the best I can to take care of my own health.	0.51	
Thinking back to my own childhood, say up to the time I was 16, I remember a great deal of illness and death in my family.	0.09	
The costs of medical care, in general, are much too high.	0.46	
Some kind of health insurance which covers all the medical expenses I (and my family) might have, is a good idea.	0.60	
Health insurance which covers all medical costs, but is good only with hospitals and doctors who sign up with it is a good idea.	0.10	
Respondents	2036	2632

Notes: The table shows responses for all parents of minor children.



**Table E-3 The Regression-Adjusted Relationship Between Categorical Eligibility and Changes in Children’s Insurance Rates by Poverty Status**

	(1)	(2)	(3)
Dependent Variable:	<i>Has Private Insurance or Public Medical Payments (including Medicaid)</i>		
AFDCj × 1 {1970}	-0.75 [1.74]	-2.98 [2.55]	-5.29 [2.62]
AFDCj × 1 {1970} × Poor	2.48 [1.46]	5.53 [2.77]	7.59 [4.4]
P(Insurance), Non-poor, 1963	0.82	0.68	0.84
P(Insurance), Poor, 1963	0.33	0.30	0.36
R2	0.28	0.33	0.26
Observations	3,405	1,202	2,203
PSUs	67	40	67
Sample	All Children	Nonwhite Children	White Children

Notes: The table presents estimated coefficients from equation (1) on the interaction of AFDC rates and a 1970 dummy and the triple interaction of those two variables with a dummy for poor children. The outcome variable equals one for children who are covered by private insurance or, in 1970, report a payment from a “public” source. Standard errors are clustered at the PSU-level.

**Table E-4 The Regression-Adjusted Relationship Between Categorical Eligibility and Changes in Children’s Primary Care Use by Poverty Status**

	(1)	(2)	(3)
Dependent Variable:	<i>A. Saw a Doctor in the Last Year</i>		
AFDCj × 1 {1970}	1.15 [1.6]	-1.19 [2.01]	4.65 [2.92]
AFDCj × 1 {1970} × Poor	1.35 [1.13]	3.53 [2.66]	2.61 [4.12]
P(Doctor Visit), Non-poor, 1963	0.78	0.65	0.79
P(Doctor Visit), Poor, 1963	0.47	0.30	0.62
R2	0.20	0.32	0.14
Observations	3,405	1,202	2,203
PSUs	67	40	67
Dependent Variable:	<i>B. Had a Physical or Checkup in the Last Year</i>		
AFDCj × 1 {1970}	-0.37 [1.43]	-2.57 [1.46]	6.02 [2.49]
AFDCj × 1 {1970} × Poor	3.28 [1.22]	4.44 [2.3]	1.22 [5.51]
P(Physical), Non-poor, 1963	0.65	0.66	0.65
P(Physical), Poor, 1963	0.40	0.43	0.38
R2	0.16	0.25	0.18
Observations	2,974	1,036	1,938
PSUs	67	39	67
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-2. The dependent variables equal one for children who saw a physician during the survey year (panel A) and who had a physical or a checkup during the survey year (panel B). Source: see notes to table 2-1.

**Table E-5 The Regression-Adjusted Relationship Between Categorical Eligibility and Changes in Children’s Hospital Admissions by Poverty Status**

	(1)	(2)	(3)
Dependent Variable:	<i>Had a Hospital Admission in the Last Year</i>		
AFDCj × 1 {1970}	1.02 [0.48]	0.24 [0.66]	2.41 [1.28]
AFDCj × 1 {1970} × Poor	-0.84 [0.55]	-0.64 [0.8]	-4.73 [2.04]
P(Hospital Admission), Non-poor, 1963	0.07	0.03	0.07
P(Hospital Admission), Poor, 1963	0.06	0.06	0.06
R2	0.05	0.14	0.06
Observations	3,405	1,202	2,203
PSUs	67	40	67
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-2. The dependent variable equals one for children who were admitted to the hospital overnight during the survey year. Source: see notes to table 2-1.

**Table E-6 The Regression-Adjusted Relationship Between Categorical Eligibility and Changes in Children’s Out-of-Pocket Expenditures by Poverty Status**

	(1)	(2)	(3)
Dependent Variable:	<i>A. Had Out-of-Pocket Expenditure</i>		
AFDCj × 1 {1970}	-3.78 [1.55]	-3.78 [2.16]	-0.88 [1.38]
AFDCj × 1 {1970} × Poor	1.65 [1.36]	1.47 [2.54]	2.29 [3.36]
P(Out-of-Pocket), Non-poor, 1963	0.91	0.76	0.93
P(Out-of-Pocket), Poor, 1963	0.72	0.60	0.83
R2	0.16	0.29	0.09
Observations	3,405	1,202	2,203
PSUs	67	40	67
Dependent Variable:	<i>B. Out-of-Pocket Share of Total Expenditure</i>		
AFDCj × 1 {1970}	-1.80 [0.65]	-1.83 [0.77]	-0.77 [1.48]
AFDCj × 1 {1970} × Poor	1.00 [0.63]	1.19 [1.08]	-1.31 [2.09]
Out-of-Pocket Share, Non-poor, 1963	0.92	0.96	0.91
Out-of-Pocket Share, Poor, 1963	0.97	0.99	0.96
R2	0.23	0.49	0.17
Observations	2,935	913	2,022
PSUs	67	38	67
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-2. The dependent variable equals one for children who were admitted to the hospital overnight during the survey year. Source: see notes to table 2-1.

**Table E-7 The Relationship Between Categorical Eligibility and Changes in Children's Insurance Rates by Poverty Status, Logit Average Marginal Treatment Effects**

Dependent Variable:	(1)	(2)	(3)
	<i>Has Private Insurance or Public Medical Payments (including Medicaid)</i>		
AFDCj×1{1970}	-0.08 [0.7]	-1.85 [2.11]	-0.22 [2.41]
AFDCj×1{1970} Poor	2.03 [1.07]	5.65 [2.6]	3.49 [3.26]
P(Insurance), Non-poor, 1963	0.82	0.65	0.83
P(Insurance), Poor, 1963	0.33	0.27	0.36
log likelihood	-0.15	-0.03	-0.11
Observations	3,323	1,178	2,101
Sample	All Children	Nonwhite Children	White Children

Notes: The estimates are average marginal effects (over the full sample) of the variables listed in the left column. See Puhani (2008) for details on this method of calculating marginal effects, especially as relates to the argument in Ai and Norton (2003).

**Table E-8 The Regression-Adjusted Relationship Between Categorical Eligibility and Changes in Children’s Primary Care Use by Poverty Status, Logit Average Marginal Treatment Effects**

	(1)	(2)	(3)
Dependent Variable:	<i>A. Saw a Doctor in the Last Year</i>		
AFDCj×1 {1970}	0.26 [0.56]	-1.39 [1.71]	4.10 [3.05]
AFDCj×1 {1970} Poor	1.21 [0.92]	2.21 [1.87]	1.61 [4.34]
P(Doctor Visit), Non-poor, 1963	0.77	0.65	0.79
P(Doctor Visit), Poor, 1963	0.47	0.31	0.62
log likelihood	-0.19	-0.03	-0.14
Observations	3,383	1,182	2,154
Dependent Variable:	<i>B. Had a Physical or Checkup in the Last Year</i>		
AFDCj×1 {1970}	-1.17 [0.66]	-1.96 [1.43]	2.51 [2.4]
AFDCj×1 {1970} Poor	3.66 [1.16]	3.63 [1.94]	2.13 [5.92]
P(Physical), Non-poor, 1963	0.65	0.68	0.64
P(Physical), Poor, 1963	0.40	0.44	0.38
log likelihood	-0.19	-0.03	-0.15
Observations	2,974	1,023	1,917
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table 2-A7.

**Table E-9 The Regression-Adjusted Relationship Between Categorical Eligibility and Changes in Children’s Hospital Admissions by Poverty Status, Logit Average Marginal Treatment Effects**

Dependent Variable:	(1)	(2)	(3)
	<i>Had a Hospital Admission in the Last Year</i>		
AFDCj×1{1970}	0.59	-0.13	1.96
	[0.48]	[1.2]	[0.99]
AFDCj×1{1970} Poor	-1.09	-0.73	-6.52
	[0.83]	[1.8]	[2.61]
P(Hospital Admission), Non-poor, 1963	0.08	0.04	0.09
P(Hospital Admission), Poor, 1963	0.07	0.08	0.08
log likelihood	-0.08	-0.01	-0.07
Observations	3,054	935	1,900
Sample	All Children	Nonwhite Children	White Children

Notes: See notes to table E-7.

**Table E-10 The Relationship Between Categorical Eligibility and Changes in Children's Probability of Health Spending by Poverty Status**

Dependent Variable:	<i>Had Any Health Expenditure</i>		
AFDCj×1{1970}	0.07	-2.13	-0.18
	[0.69]	[1.91]	[0.79]
AFDCj×1{1970} Poor	1.54	1.15	2.63
	[0.83]	[1.84]	[2.54]
Out-of-Pocket Share, Non-poor, 1963	0.92	0.76	0.94
Out-of-Pocket Share, Poor, 1963	0.73	0.60	0.84
R2	0.13	0.28	0.07
Observations	3,405	1,202	2,203
PSUs	67	40	67
Sample	All Children	Nonwhite Children	White Children

See notes to table 2-5. The dependent variable equals one for children who have positive health expenditures.



### **Chapter 3 THE WAR ON POVERTY'S EXPERIMENT IN PUBLIC MEDICINE: COMMUNITY HEALTH CENTERS AND THE MORTALITY OF OLDER AMERICANS**

**with Martha J. Bailey**

In 1965 U.S. policymakers began a bold experiment in the provision of health care to the poor. Unlike the era's large public insurance expansions that subsidize the purchase of health care from private providers (Medicare and Medicaid), Community Health Centers (CHCs) used federal funds to deliver primary care to underserved populations.<sup>59</sup> From the outset CHCs sought to increase the availability and convenience of care while reducing the cost to patients. CHCs charged on a "pay as you can" sliding-scale for services and medications, were located in disadvantaged neighborhoods, and offered home visits and transportation to appointments.

The CHC model of primary care has survived for 50 years and enjoyed a significant expansion in funding since 1995 (figure 3- 1).<sup>60</sup> By 2008, over 8,000 CHC sites operated in every state and served over 20 million Americans, 40 percent of whom were uninsured and 70 percent of whom were poor (Adashi et al. 2010). In 2010 the Affordable Care Act (ACA) appropriated an additional \$11 billion over five years to establish CHCs as one of the pillars of health care reform—infrastructure intended to help serve the millions of Americans projected to gain health insurance under its provisions. Part of the rationale for the expansion of CHCs relies on a widely-held belief that they improve access to primary care and curb health care cost increases (Cunningham 2006, Falik et al. 2006, Rust et al. 2009, Hawkins and Schwartz 2003).

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<sup>59</sup> The earliest health centers were called "neighborhood health centers." Today CHCs include "Federally Qualified Health Centers" (FQHC) and so-called "Look-Alike FQHCs." We use "community health centers" in the paper to refer to them all.

<sup>60</sup> Between 2001 and 2007, the Federal Health Center Growth Initiative doubled health center funding from \$1 to \$2 billion (US DHHS 2008). The 2009 American Reinvestment and Recovery Act allocated funds to build 126 new facilities and expand 1,100 existing CHCs (Iglehart 2010).

Yet the limitations of existing studies caution against strong causal claims. Not only have studies had difficulty identifying CHCs' effects on health care utilization, but few have measured their effects on health. Even if CHCs increase service use, they need not improve health or reduce health care costs if—as articulated by the program's early critics—CHC services are lower quality than those available from private and non-profit providers (covered by Medicare and Medicaid for many CHC patients).<sup>61</sup> If lower quality services gradually erode health, CHCs could ultimately worsen health and raise health care costs over the longer term.

This paper uses the rollout of CHCs from 1965 to 1974 to provide the first evidence on their long-term health effects and, more generally, the impact of increasing the availability of primary care to the poor. This historical vantage point allows us to evaluate the cumulative impact of providing primary care up to 15 years after CHCs began, while using the “great administrative confusion” at the Office of Economic Opportunity (OEO) as our source of identifying variation (Levine 1970). Our main results show that the establishment of CHCs predicts sharp and persistent reductions in age-adjusted mortality rates. Within one decade of CHCs operating, the reduction in age-adjusted mortality rates averaged 2 percent in treated counties. We find little evidence that CHCs affected infant and child mortality rates. Their effect on aggregate mortality was driven by reductions in cardiovascular-related causes among adults 50 and older.

Our rough translation of these intention-to-treat effects into effects on the treated implies a 7 to 9 percent reduction in age-adjusted mortality among the poor ages 50 and older—an effect

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<sup>61</sup> Early critics referred to the program as “a step toward socialism” (Sardell 1988: 61). Doctors feared competition from CHCs and expressed concerns about the quality of their care owing to their greater staffing with nurses and social workers and the development of practice guidelines by “lay persons” (62). In 1966, the president of the American Medical Association (AMA) spoke against the program, arguing that the government's role in health care should be limited to “the overall stimulation and support of private enterprise, rather than undertaking specific operational or directional capacities” (Hudson 1966: 99).

equal to 20 to 28 percent of the 1966 poor/non-poor mortality gap for this age group. These results are robust to and often strengthened by standard sensitivity tests such as controlling for state-by-year fixed effects, county-level federal per-capita medical spending, and linear county-level trends, as well as by reweighting areas without CHCs so that observed characteristics resemble the distribution of those characteristics in areas with CHCs. Our placebo tests show that CHCs had no measurable impact on accident-related mortality in any specification, which is consistent with their provision of primary (but not emergency) care. We also find little evidence that the expansion of hospital capacity or other War on Poverty programs drive the results. Interestingly, large effects of CHCs on mortality rates occurred among those eligible for Medicare, but we find no accompanying increase in Medicare enrollment or spending.

The body of evidence suggests that CHCs improved older-adult health in a variety of ways. Our analysis of the Survey of Health Services Utilization and Expenditures (SHSUE) highlights two important reasons for this. For one, as CHCs reduced the cost of primary care, the share of older, poor adults reporting a “regular source of care” increased by 25 percent. Reporting a regular source of care is a stronger predictor of positive health outcomes than is having health insurance (Sox et al. 1998) and is also highly correlated with compliance with treatment for hypertension (Shea et al. 1992). In addition, the SHSUE show that the share of the older poor with out-of-pocket prescription drug expenditures fell by almost 40 percent in locations with CHCs (medications were not covered by Medicaid or Medicare but highly subsidized or free at CHCs’ in-house pharmacies). These findings—and the likelihood that reductions in mortality understate the broader health benefits of CHCs—highlight the value of interventions that increase access to primary care, even for populations with near universal health

insurance coverage. Our analysis concludes with estimates of CHCs' costs per life-year which are one third to one eighth the size that of that achieved by Medicare during the same period.

### **3.1 History and Expected Effects of Community Health Centers**

In his first State of the Union Address in January 1964, President Lyndon B. Johnson declared an “unconditional war on poverty.” Central to his war was the 1964 Economic Opportunity Act (EOA), which aimed to “eliminate the paradox of poverty in the midst of plenty.” The Office of Economic Opportunity (OEO) was created to administer the EOA initiatives and make direct grants to local organizations.<sup>62</sup>

Initially, the OEO focused on programs to promote human capital and community development like Head Start and Job Corps, but OEO administrators soon discovered that health problems and little access to care limited participation in their programs. Few charity or reduced-cost providers existed in many parts of the U.S. during the 1950s and 1960s (Sardell 1988: 45). Doctor Raymond Wheeler, a member of the Citizen’s Board of Inquiry into Hunger in the U.S. from 1967 to 1971, vividly described the limited access to medical care among the poor:

We saw hundreds of people whose only hope of obtaining medical care was to become an emergency which could not be turned away. We heard countless stories of driving 50 or 100 miles to a city general hospital after refusal of care at a local hospital (1971: 2)

Even if an outpatient department existed locally, the “four-hour wait, multiple referrals, incredible discontinuity of care and various other indignities” (Knowles 1964: 733) certainly would have deterred many of the poor from seeking care for their non-emergency conditions.

Harrington’s iconic description of the era links poor health to the persistence of poverty:

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<sup>62</sup> OEO funding was intended to ensure “the maximum feasible participation” of the poor and empower those who had been excluded from local politics to create positive changes in their communities. This direct-to-local-organization granting made the War on Poverty programs controversial because they challenged traditional political structures and entrenched local interests.

The poor get sick more than anyone else in the society. That is because they live in slums, jammed together under unhygienic conditions; they have inadequate diets, and cannot get decent medical care. When they become sick, they are sick longer than any other group in the society. Because they are sick more often and longer than anyone else, they lose wages and work, and find it difficult to hold a steady job. And because of this, they cannot pay for good housing, for a nutritious diet, for doctors. At any given point in the circle, particularly when there is a major illness, their prospect is to move to an even lower level and to begin the cycle, round and round, toward even more suffering (1962).

To address the health needs of the poor, the OEO initiated the CHC program in 1965. The program aimed to deliver affordable, comprehensive care to disadvantaged populations. The initial wave of CHC grants established eight demonstration projects administered through medical schools, hospitals, and boards of health. The 1966 amendments to the EOA expanded funding through the OEO, and the 1967 Partnership for Health Amendments allocated additional funds for the Department of Health Education and Welfare (DHEW) to initiate CHCs (Davis and Schoen 1978: 163). By 1974 CHCs existed in 117 counties nationwide with each site annually serving an average of 13,330 registered patients (Ibid: 178). Figure 3-1 shows that annual federal expenditures on CHCs reached around \$863 million (2012 dollars) in 1974 and fluctuated around this mean through 1990.

The OEO model allowed any organization to receive funding. Administrators reported receiving applications from “various and sundry groups” often having little to do with the legislation (Gillette 1996: 196, quoting Theodore M. Berry, assistant director of the OEO). Awardees tended to be “one leading-edge, creative person who managed to get enough resources together...pulling them [others at the organization] kicking and screaming into something that

they really didn't want to be in, but that had lots of dollars attached to it" (May et al. 1980: 587). The first wave of CHC grants (1965-1974) reflects the often arbitrary funding process typical of the War on Poverty (Ludwig and Miller 2007, Bailey 2012, Bailey and Duquette 2013)—a claim we support quantitatively later in the paper.

The focus of the CHC program and the allocation of funding changed with the enactment of the 1975 Special Health Revenue Sharing Act. This Act mandated that funding for CHCs follow the Index of Medical Underservice (IMU), which also qualified underserved areas for HMOs, federal investments in health care professionals, and other federal health funding.<sup>63</sup> In practice, the 1975 Act also meant that grants were disproportionately awarded to rural and underdeveloped areas (Sardell 1982). By 1980, CHCs had started in another 497 counties—two thirds of which were located in areas with fewer than 50,000 residents.

### *3.1.1 What Did Community Health Centers Do and Whom Did They Serve?*

At their most basic level, CHCs provided low-cost primary medical care in poor communities. Data on their services in the first decade of the program are sparse, but one 1973 financial audit provides a snapshot (Davis and Schoen 1978: 187; reprinted in appendix table G-1). Per registered patient (registrant, not all of whom sought care in a given year), CHCs provided medical care an average of 2.6 times per year and filled medical prescriptions an average of 2.5 times per year at in-house pharmacies. Laboratory tests were performed an average of 1.8 times, dental care was provided 0.6 times, and X-rays were made 0.3 times per year. These services were either free or highly subsidized.

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<sup>63</sup> Initially constructed as part of the Health Maintenance Organization Act in 1973, the IMU was developed by DHEW and the University of Wisconsin to identify areas of medical need. The IMU aggregates information on per-capita primary medical care physicians, infant mortality rates, poverty rates, and the percentage of an area's population age 65 or older.

The CHC model distinguished itself by offering a range of auxiliary services as well. The OEO targeted “alternative” delivery strategies because administrators believed that existing providers (private physicians and crowded hospital out-patient departments) had discouraged the poor from seeking care. To improve outreach and communication, CHCs hired health center employees from the communities they served (Rudd et al. 1976). To reduce wait and travel times, CHCs located in underserved neighborhoods and a single center often had multiple clinics or mobile units. More than 90 percent provided home health care and transportation to appointments (Health Services Administration 1974: 60).<sup>64</sup>

If CHCs meaningfully reduced the costs of receiving primary care, they should (weakly) increase the use of primary care on both the extensive and intensive margins. Moreover, this effect should be largest among those least able to afford care (those without insurance, for instance) or travel to receive it. We have found only one nationally representative data source that allows us to test this claim. The SHSUE covers the period before and after the CHC program began (1963 and 1970) and asks respondents to describe their use of “clinics” (including CHCs) (Center for Health Administration Studies/National Opinion Research Center 1981). Because the SHSUE sampled the same primary sampling units (PSUs) in both periods, we use a differences-in-differences (DD) estimator to compare changes in medical care in areas in 17 of the observed PSUs with CHCs while adjusting for changes in outcomes in the 56 PSUs without them.

We find that the use of a “clinic” as the regular source of care (no regular source of care is coded as a zero) increased by 17 percentage points more (s.e. 0.04) in areas that received a CHC before 1970, relative to a baseline of 9 percentage points. Consistent with CHCs

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<sup>64</sup> The 1972 Directory of CHCs (DHEW 1972), for instance, reports that the CHC program in Denver, Colorado, had two health centers, 11 satellite health “stations,” a staff of over 800 members, and was supposed to serve a population of 287,869 (almost 106,000 patients were enrolled in 1971). The CHC program in Oakland, California, provided comprehensive ambulatory care 24 hours per day, employed 52 physicians, and aimed to serve a population of 40,000 (17,289 were enrolled in 1971).

disproportionately serving the poor, the DD estimate for poor households is 25 percentage points larger (s.e. 0.06) than for household with incomes above 300 percent of the poverty line (0.03, s.e. = 0.02).<sup>65</sup> This suggests a 93-percent increase in the likelihood poor households used a clinic as their regular source of care. Whereas the SHSUE show no differential change in clinic use in nonurban areas receiving CHCs, they show a substantial increase in urban areas (0.23, s.e. 0.09).

### 3.1.2 Trends in Mortality Rates

Our analysis follows the literature on health in the 1960s and uses mortality rates to proxy for health status. Although mortality is a limited measure of health, it is reliably and consistently measured for our period of interest. We compute mortality rates using the 1959 to 1988 Vital Statistics Multiple-Cause of Death Files (US DHHS and NCHS 2007), which contain the universe of civilian deaths reported by cause, age, and the decedent's county of residence. We combine the mortality data with county population and birth data to create infant mortality rates as well as age-specific and age-adjusted (AMR) mortality rates, which we also disaggregate into six leading causes of death: (1) diseases of the heart, (2) other cardiovascular diseases (mainly cerebrovascular causes like strokes), (3) cancer, (4) infectious disease, (5) diabetes, and (6) accidents (see appendix A for details on coding and appendix C for trends by cause).

Figure 3-2 describes our data and also shows that the CHC program rolled out during a period of dramatic declines in U.S. mortality rates. The U.S. AMR fell by around 29 percent from 1960 to 1988, which reflected improvements in all age groups. For infants, children (ages 1

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<sup>65</sup> See appendix A for a description of the SHSUE. This estimate comes from a three-way fixed effects model. Income categories are defined relative to the poverty line categories and include households below 100 percent, households between 100 and 299 percent, and households above 300 percent of the federal poverty line. Households 300 percent and above are omitted. Dummy variables for each category are included directly and interacted with the implementation of CHCs before 1970 and the 1970 dummy. Covariates in all specifications include indicators for sex, 5-year age-groups, race, education, PSU fixed effects, a dummy variable for observation in 1970, and an interaction of whether the PSU had a CHC by 1970 and the 1970 dummy. Reported DD estimates refer to the coefficient on the interaction.



to 19), adults (ages 20 to 49), and older adults (ages 50+), mortality rates fell by 62, 49, 22, and 28 percent, respectively. The causes of these declines reflect different factors for different groups, and some of these improvements may be related to CHCs.

Large declines in infant mortality were driven by improvements in infant survival conditional on fitness at birth (Lee et al. 1980).<sup>66</sup> Improved access to medical care resulting from the desegregation of hospitals and the initiation and expansion of Medicaid significantly reduced infant mortality (Almond, Chay and Greenstone forthcoming; Goodman-Bacon 2013; Currie and Gruber 1996b). During the 1980s, acute neonatal care (neonatal intensive care units) helped less healthy infants survive. Improvements in access to medical care also reduced the mortality of older children, especially mortality due to common childhood diseases such as pneumonia and meningitis (11 percent of all childhood deaths). Medicaid coverage played an important role for poor children as it increased care for conditions easily treatable with antibiotics (Goodman-Bacon 2013, Currie and Gruber 1996a). Finally, reductions in accident-related deaths among children (40 percent of childhood deaths in 1965) played an important role in reducing child mortality in the 1960s and 1970s, reflecting greater seat belt use and lower speed limits.

The reduction in older adult mortality (ages 50+) accounted for 75 percent of the aggregate decline in mortality rates from 1960 to 1988. The most important proximate factor was the reduction in deaths due to major cardiovascular causes (CVD, causes 1 and 2 above). Over the period of interest, Goldman and Cook (1984) find that roughly half of the decline in ischemic heart disease was due to changes in lifestyle (cholesterol reduction and smoking) and half was due to medical treatment. Innovations in medical treatment included the inpatient treatment of

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<sup>66</sup> Although fitness at birth improved over the period with the growth in nutritional support for young children and mothers through the Food Stamps and the Women, Infants and Children programs, these programs contributed to improvements in birth weight more than infant survival (Almond, Hoynes and Schanzenbach 2011; Hoynes, Page, and Stevens 2011).

acute cardiovascular incidents (including drugs to dissolve blood clots, bypass surgery and angioplasty, Cutler and Meara 2004) and the development of medications to manage hypertension on an outpatient basis (Long et al. 2006).

Anti-hypertensive drugs, including diuretics and vasodilators, are believed to be among the most important contributors to the reduced mortality rates for adults 50 and older because they prevented potentially fatal incidents for a relatively low cost (Crimmins 1981: 244, Freis 1995, Cutler and Kadiyala 2003). After randomized trials by the Veterans Administration Cooperative Group (VACG) demonstrated these drugs' effectiveness, the National Institutes of Health launched a campaign to promote awareness of hypertension. People were encouraged to "know your [blood pressure] number," and physicians were encouraged to screen and treat the disease.<sup>67</sup>

### *3.1.3 The Expected Effects of Community Health Centers on Mortality Rates*

The expected effects of CHCs on mortality rates depends both on the incidence of causes CHCs might prevent and the effectiveness of CHCs' care (relative to the alternatives). For infants and children, for instance, deaths are relatively rare (less than 9 percent of all deaths over the 1959-1988 period were to individuals under age 15), and fewer could have been prevented with primary care. Approximately 41 percent of child deaths due to "external causes" were

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<sup>67</sup> The first VACG study randomly assigned 73 middle-aged men with moderate hypertension (diastolic blood pressure between 115 and 129 mm Hg) to a treatment group that received three hypertension medications. 73 men were also assigned to a control group that received placebo medications. The control group experienced 27 morbid events and 4 deaths while the treatment group experienced only two morbid events and zero deaths (VACG 1967). Researchers terminated the study for moderate hypertensives after six months due to the large observed treatment effects. The second VACG study used the same methodology but focused on 380 men with low hypertension (diastolic blood pressure between 90 and 114 mm Hg). The study followed patients for an average of 3.3 years during which time 21 control patients and 10 treatment patients died. In addition, more than twice as many control patients had assessable morbid events (VACG 1970). During the 1970s and 1980s, the share of hypertensives who learned their blood pressure number increased from 50 to over 70 percent and the share taking anti-hypertensive medication increased from 35 to over 55 percent (Cutler and Kadiyala 2003: figures 12 and 13).

unlikely to be affected by CHCs.<sup>68</sup> Even though infant mortality rates (mostly due to internal causes) were high, CHCs primary and preventative care may not have increased survival. CHCs could have helped parents get an earlier diagnosis of potentially lethal diseases and afford medications for treatment, but they were not substitutes for hospitals' acute care for sick infants. In short, we do not expect CHCs to have large effects on infant and child mortality.

The overwhelming number of deaths that CHCs could have prevented occurred among adults ages 50 and older. This group not only comprised a large share of U.S. deaths (80 to 88 percent from 1960 to 1988), but many of these deaths would have been responsive to the provision of primary care. CHCs could reduce mortality rates by increasing early detection of health problems, providing free or highly subsidized medications to treat conditions (not covered by Medicare or Medicaid in most cases, Finkelstein and McKnight 2008; Davis and Schoen 1978: 55), and increasing awareness about Medicare (Chay et al. 2011) and Medicaid, both of which could increase access to life-saving hospital treatments. Similarly, CHCs' outreach and follow-up efforts could have had important effects on the management of common, fatal, chronic conditions like hypertension that required consistent and long-term use of medication. Finally, CHCs may have reduced the mortality of community members who did not use their medical services by reducing emergency room crowding, lowering the incidence of infectious disease, and spreading health information. For all of these reasons, we do expect CHCs to have large effects on mortality rates of older adults.

Offsetting these potential gains, however, CHCs may have reduced the quality of care. Although many accounts suggest that CHCs provided higher quality care than did hospital outpatient departments, physicians raised concerns over the use of nurses and social workers in CHCs and the development of practice guidelines by "lay persons" (Sardell 1988: 62). This is

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<sup>68</sup> External causes are things like accidents, homicides, suicides, et cetera.

especially relevant in our period of study as alternative sources of care were private providers who may have been covered under Medicaid or Medicare. If CHCs provided lower quality care than these private alternatives, then CHCs' diversion of care from higher quality sources could increase mortality rates. This possibility makes the overall effects of CHCs on mortality rates theoretically ambiguous and motivates our empirical investigation.

#### Previous Studies of the Effects of Community Health Centers

A large body of research has examined CHCs, but its reliance on empirical methodologies with limited internal and external validity cautions against strong causal inferences regarding the health effects of CHCs.<sup>69</sup> The literature's use of cross-sectional variation is tenuous because communities with CHCs differ in a number of observable ways. Table 3-1 shows that counties receiving CHCs (we call these "treated" counties) tended to be more urban, more affluent (greater share of households with incomes above \$10,000), have lower poverty rates (smaller share of households with incomes below \$3,000, the 1960 poverty line for a family of four), have more nonwhites per population, and have more active physicians per capita. They also tended to have a medical school. This may seem contrary to the program's mission of reaching the underserved but is consistent with local advocates submitting grant proposals on behalf of needier residents. More affluent, urban areas were best situated to do this. These cross-sectional differences motivate the inclusion of county fixed effects in all of our specifications.

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<sup>69</sup> Much of the literature examines a single CHC or a narrow geographic area and therefore cannot separate the effects of CHCs from changes in local policies or circumstances. For examples of case studies, see Bellin et al. (1969) and Moore et al. (1972) on Boston, Chabot (1971) on Denver, Hochheiser et al (1971) on Rochester, Gordis (1973) on Baltimore, and O'Conner et al. (1990) on an unspecified area in rural North Carolina. Two studies compare outcomes in five cities (Okada and Wan 1980, Freeman et al. 1982). Focusing on later periods, Deprez et al. (1987) use data from 36 CHCs in Maine in 1980, Ulmer et al. (2000) study medical records from 20 clinics across 10 states, and Epstein (2001) focuses on clinics in Virginia only. In studies of CHCs' effects on diabetes, Chin et al. (2000) examine 55 Midwestern cities and Bell et al. (2001) study 14 clinics in North Carolina.

More recent studies of CHCs use panel data to account for these cross-sectional differences using fixed effects. In one of the earliest studies of this type, Goldman and Grossman (1988) examine infant mortality rates in the 678 largest counties between 1970 and 1978 and show that an increase in the number of CHCs is associated with an overall reduction in infant mortality rates, particularly among blacks (see also Shi et al. 2003 and O'Malley et al. 2005). Using a 1996 to 2006 county panel and a specification with clinic and year fixed effects, Lo Sasso and Byck (2010) provide the best evidence to date that increases in CHC funding raise service availability and staffing while reducing uncompensated care. It is possible, however, that their results reflect omitted variables that affect both CHC performance and administrator decisions. For instance, Lo Sasso and Byck's results could capture administrator decisions to defund poorly performing CHCs or increase CHC funding as part of a community development effort, both of which could lead their empirical strategy to overstate the effects of CHCs. On the other hand, if administrators increased funding to help failing CHCs or in response to declining local investments in community health, their estimates could understate the effects of CHC funding. To avoid both sources of endogeneity, we rely on variation in when and where CHCs were established rather than changes in funding—a decision that we discuss under research design.

Another limitation of the literature is that few studies consider the health effects of CHCs. The handful that do focus on infants (Chabot 1971, Goldman and Grossman 1988, Shi et al. 2004a, and Shi et al. 2004b) or specific health conditions (Gordis 1973 for rheumatic fever; Dignan et al. 1979 for cardiovascular disease; Hicks et al. 2006 for asthma; Chin et al. 2000 and Hicks et al. 2006 for diabetes; O'Connor et al. 1990 and Hicks et al. 2006 for hypertension; Hedberg et al. 1996). Our analysis is the first to consider the longer-term impacts of CHCs on

mortality rates as well as characterize heterogeneity in these effects by age group, race and population density.

### **3.2 Data and Research Design: Using The Rollout of Community Health Centers To Identify Their Mortality Effects**

Newly compiled data on when and where CHCs were established facilitate this paper's research design. The following sections describe our data on CHCs, present empirical evidence motivating our research design, and discuss our empirical specifications.

#### *3.2.1 Data on Health Centers and County-Level Mortality Rates*

We use two data sources to document the rollout of CHCs from 1965 to 1974: the National Archives Community Action Program (NACAP) electronic files and hand-entered annual Public Health Service Reports (OEO 1966, 1967, 1968; DHEW 1972a, 1972b; Zwick 1972, GAO 1973; Health Services Administration 1974; Rudd et al. 1976). Both are verified using primary sources. Our final database contains information on (1) the county where CHCs delivered services, which allows each federal grant to be linked to county-level mortality rates, and (2) the date when each county received its first CHC services grant (this excludes planning grants), which provides a consistent proxy for the year each CHC became operational. Our robustness checks use supplemental data on CHC grants between 1975 and 1980 from the National Archives Federal Outlays files (NAFO) and primary sources. These data allow us to identify 497 largely rural CHCs begun in this later period. However, the lack of exact start dates for many of these CHCs, the dependency of qualification for CHC funding on the IMU (leading to the packaging of new CHCs with other health services), and the limited data on the implicit first stage lead us to present estimates that rely solely on CHCs established between 1965 and

1974. (See appendix A for details about the data and source material and appendix H for results using all CHCs).

Figure 3-3 shows the rollout of the CHC program between 1965 and 1980 by the year and the county of establishment, the smallest area consistently identified over our period of interest in the mortality files. Counties with CHCs established in fiscal years 1965 to 1974 are shaded. The first CHCs in Columbia Point (Boston), Massachusetts, and Mound Bayou, Mississippi, were established in fiscal year 1965 in collaboration with Tufts Medical School. As the CHC program expanded, it achieved broad geographic coverage. All U.S. regions had CHCs: between 1965 and 1974, 48 were established in the South, 21 in the Northeast, 22 in the Midwest, and 26 in the West. There is also considerable within-state variation in CHC establishment dates.

### 3.2.2 *Event-Study Specification*

Our empirical strategy uses variation in when and where CHC programs were established to evaluate their effects on mortality. Two empirical tests support a key identifying assumption—that the timing of establishment is uncorrelated with other determinants of changes in mortality. First, most 1960 socio-demographic characteristics of counties receiving a CHC in our period of interest in table 3-1 fail to predict when a CHC was established (appendix table I-1).<sup>70</sup> Significant exceptions are urban share and share of physicians (MDs) per county population. This is not surprising because larger, denser places had more resources and organizations that could apply for funding and CHCs were set up in locations with physicians to staff them. To account for these potential threats to internal validity, we include urban-group-by-year fixed

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<sup>70</sup> We choose these characteristics because they have been shown to predict the timing of the implementation of other War on Poverty programs. Almond et al. (forthcoming) and Hoynes and Schanzenbach (2009) report statistically significant relationships between these characteristics and both the timing of Medicare certification and the initiation of the food stamps program. The power of our study relative to Hoynes and Schanzenbach is limited in that we do not have information on the month of program initiation and that not all of the counties in the U.S. received CHCs.

effects and linear trends interacted with the number of physicians in 1960 in our primary specifications.

Second, we examine whether CHC establishment timing is correlated with levels or trends in pre-program AMR. This could be the case if, for instance, proposals originated sooner in locations with higher mortality rates or the OEO prioritized locations using mortality rates or their correlates. Figure 3-4 plots the AMR in 1965 and changes in the AMR from 1960 to 1965 against the year of CHC establishment in treated communities, and shows no evidence of either scenario (see appendix figure I-1 for results by age group). The establishment of CHCs is uncorrelated with pre-existing levels and changes in AMR in either univariate or multivariate regressions. In summary, the lack of a systematic correlation between CHC establishment, most socio-demographic characteristics, or mortality rates, is consistent with the oral history’s characterization of a “wild” funding process. After presenting the empirical strategy and results, the last section of the paper returns to these concerns and tests for specific threats to the internal validity of the research design.

Our empirical strategy exploits variation in the location of CHCs and the timing of their establishment within a flexible event-study framework (Jacobson et al. 1993),

$$(1) \quad Y_{jt} = \theta_j + \gamma_{u(j)t} + \delta_{s(j)t} + \mathbf{X}'_{jt}\boldsymbol{\beta} + \sum_{y=-7}^{-2} \pi_y D_j 1(t - T_j^* = y) + \sum_{y=0}^{15} \tau_y D_j 1(t - T_j^* = y) + \varepsilon_{jt}.$$

Here  $Y_{jt}$  is a mortality outcome in county  $j$  in year  $t = 1959, \dots, 1988$ .<sup>71</sup>  $\theta_j$  is a set of county fixed effects, which absorbs time-invariant differences in observable (table 3-1) and

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<sup>71</sup> Because models are weighted by the relevant 1960 population, we exclude New York, Los Angeles and Chicago from our sample. These places had two million more residents in 1960 than the next largest counties and would receive disproportionate weight in our regressions. We also eliminate 18 counties with missing data. These sample restrictions leave us 3,044 counties in our primary sample.



unobservable characteristics and allows consistent estimation of  $\pi$  and  $\tau$  even in the presence of differences between treated and untreated locations.  $\gamma_{u(j)t}$  is a set of urban-group-by-year fixed effects.<sup>72</sup>  $\delta_{s(j)t}$  is a set of either year fixed effects or state-by-year fixed effects, which captures time-varying national changes such as Medicare or state-level implementation of Medicaid and the Civil Rights Act (Almond et al. forthcoming).  $X_{jt}$  includes a constant, the interaction of 1960 characteristics with linear time trends (share of population in urban area, in rural area, under 5 years of age, 65 or older, nonwhite, with 12 or more years of education, with less than 4 years of education, in households with income less than \$3,000, in households with incomes greater than \$10,000, total active MDs), and annual county-level per capita measures of government transfers from the Bureau of Economic Analysis Regional Information System (REIS) (cash public assistance benefits such as Aid to Families with Dependent Children, Supplemental Security Income, and General Assistance; medical spending such as Medicare, Medicaid, and military health care; and cash retirement and disability payments).<sup>73</sup>

We use a binary indicator of treatment,  $D_j$ , equal to one if the county ever received a CHC grant. This captures “treatment” with a CHC. A practical reason for the choice of a binary treatment variable is that the NACAP data are missing grant amounts for 1969, which we have not been able to recover from other sources. The substantive reason is that larger per-capita grants tended to support greater infrastructure development rather than a larger “dose” of services.<sup>74</sup> The estimates characterizing the effects of CHCs are the coefficients on the

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<sup>72</sup> These are defined as year dummies interacted with five categories of a county’s population share in urban areas,  $u$ : 0,  $0 < u < 25$ ,  $25 \leq u < 50$ ,  $50 \leq u < 75$ ,  $75 \leq u \leq 100$ . This captures the differential diffusion of medical technologies and changes in health in areas with varying levels of urbanization.

<sup>73</sup> For the purposes of this analysis these covariates are fairly comprehensive because OEO administrators would have had limited information beyond them. The county characteristics in  $X$  are comparable to specifications in Almond et al. (2011) except that we also add information on the number of physicians per capita.

<sup>74</sup> This claim is consistent with historical reports and our empirical findings. Our analysis of heterogeneity in effects by above- or below-median per-capita first CHC grants (for CHCs not funded in 1969) shows that substantial

interaction of  $D_j$  with event-year dummies,  $1(t-T_j^* = y)$ , which are equal to one when the year of observation is  $y = -7, \dots, 0, \dots, 15$ , years from  $T_j^*$ , the date when a CHC was received in county  $j$  ( $y = -1$  omitted). Observations more than 6 years before or more than 14 years after CHC program establishment are captured by dummies,  $1(t-T_j^* \leq -7)$  and  $1(t-T_j^* \geq 15)$ . The point estimates,  $\pi_y$ , describe the evolution of mortality in eventually treated counties before CHCs began net of changes in untreated counties after adjusting for model covariates. They allow a direct evaluation of the assumption that the location and timing of CHCs is unrelated to pre-program changes in mortality.  $\tau_y$  describe the divergence in outcomes  $y$  years after the CHC was established net of changes in untreated counties after adjusting for model covariates. These estimates are the intention-to-treat effects of CHCs on mortality relative to the year before CHCs began ( $y = -1$ ).

We summarize the magnitudes and joint statistical significance of the event-study estimates in a DD specification that replaces the individual event-year dummies,  $1(t-T_j^* = y)$ , with year groups,  $D_j^g$ , where  $D_j^g$  is a binary variable equal to 1 if county  $j$  is observed in event-year group  $g$ , where  $g$  is a category for  $y \leq -7$ ,  $-6 \leq y \leq -2$ ,  $0 \leq y \leq 4$ ,  $5 \leq y \leq 9$ ,  $10 \leq y \leq 14$ , and  $y \geq 15$  ( $y = 0$  is omitted). For both the event-study and DD specifications, tables and figures present only coefficients estimated using a balanced set of counties (only event-years  $-6$  to  $14$ ). To explore the sensitivity of our results, we add covariates sequentially, estimate models with county-specific, linear time trends ( $\theta_j t$ ) (rather than parameterizing county trends using table 3-1 characteristics), and reweight the untreated counties using a function of the estimated propensity of receiving a CHC to balance the characteristics of treated and untreated counties in table 3-1

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differences in funding (\$7 versus \$45 dollars per-capita, 2012 dollars) reduced mortality rates by statistically indistinguishable amounts. Results using cumulative dollars are reported in appendix table G4.

(DiNardo et al. 1996, Heckman et al. 1998).<sup>75</sup> Standard errors are corrected for an arbitrary within-county covariance structure (Arellano 1987).

### **3.3 Estimates of the Relationship between Community Health Centers and Mortality**

The results presented in this section suggest that CHCs dramatically reduced mortality rates. The analysis begins by examining the effects of CHCs on age-adjusted mortality rates aggregated over all age groups and causes. To shed light on the possible mechanisms for these effects, we examine the relationship of CHCs with mortality rates in different age groups and mortality attributable to different causes.

#### *3.3.1 Results for All-Cause, Age-Adjusted Mortality Rates*

We summarize the effect of CHCs in treated locations using the age-adjusted mortality rates for all ages and causes. Figure 3-5 plots weighted, event-study estimates from our baseline specification that includes state-by-year fixed effects,  $\delta_{s(j)t}$ , and county-level covariates,  $\mathbf{X}_{jt}$ . The series plotted with circles presents results for 1959-1988 and defines treatment using all CHCs begun between 1965 and 1980. The thick-line series presents results from the same sample and defines treatment using only the early CHCs (1965-1974, dashed lines show 95-percent confidence intervals for this series). The open-triangle series presents results for the early CHCs on a sample that uses the 388 counties with geographic identifiers through 1998.

The results are similar in the three cases and provide no evidence of a differential trend in mortality in treated locations before the CHC program began, which implies that the econometric model captures the wide-spread declines in the AMR in the years prior to CHC establishment (urban-by-year effects are plotted in appendix figure I-2). The estimates of the pre-CHC effects

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<sup>75</sup> See appendix figures D3 and D4 and appendix table D4 for complete details on how we construct the propensity scores and descriptive statistics on the distribution of propensity scores in treated and untreated counties.

( $\pi$ ) are small in magnitude and statistically indistinguishable from zero. Following the establishment of CHCs, mortality rates fall sharply. In the first five years after the early CHCs began, the AMR was 10 deaths lower per 100,000 in the baseline specification for treated locations—a reduction of 1.1 percent over a baseline AMR of 946 deaths per 100,000 (table 3-2, column 2). In years 5 to 9 after these CHCs began, the AMR was 2 percent lower per 100,000 in treated locations (table 3-2, column 2).

Post-CHC declines in mortality rates are not as sharp for the later centers (those funded between 1975 and 1980) as for the earlier centers (1965-1974), although these differences are not statistically significant.<sup>76</sup> A potential reason for this is that legislation in 1975 substantially changed the selection of new CHC sites to favor more rural areas (table 3-1). More urban CHCs could provide greater convenience to their potential patients and their effects could be amplified by knowledge-based spillovers and externalities. In contrast, rural CHCs struggled to reach their highly dispersed target populations (section I.A shows that more rural areas did not experience an increase in clinic use after CHCs began), and spill-overs and externalities may have been limited. Consistent with this hypothesis, figure 3-6 shows that the mortality effects of the early CHCs appear exclusively in more urban areas (see appendix H for results for all CHCs). Table 3-4 presents DD summary estimates showing that the effects of CHCs were almost six times larger in more urban counties 5 to 9 years after the CHC began and more than seven times as large in years 10 to 14—estimates statistically different at the 5 and 11 percent levels, respectively.

The longer 1959 to 1998 sample sheds light on the persistence of CHCs' effects for the 388 counties (of the 3,044 counties in our primary analysis) that can be identified through 1998.

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<sup>76</sup> The trend break in mortality rates at  $t=0$  is apparent for both groups, and the mortality reductions are economically and statistically significant. However, estimates for all CHCs are around 30 percent smaller at year 9. Misclassification error in establishment dates of later CHCs (due to data limitations described previously) may also induce attenuation.

Figure 3-5 shows that this sample restriction has little effect on the estimates, which are nearly identical in the years where they overlap with the full sample. During the additional ten years (event years 15 to 24) for which we can estimate treatment effects, mortality rates remain significantly lower than those before CHCs began. In short, the treatment effect outlasts the initial enthusiasm of War on Poverty advocates and survives multiple administrative changes in the program. This persistence suggests CHCs provided long-run, cumulative benefits to communities evident 25 years later.

Table 3-2A examines the robustness of these estimates by sequentially adding covariates: county fixed effects,  $\theta_j$ , and urban-group-by-year fixed effects,  $\gamma_{u(j)t}$  (column 1); then state-by-year fixed effects,  $\delta_{s(j)t}$ , and county-level covariates,  $\mathbf{X}_{jt}$  (column 2, our baseline specification); and finally county-specific linear trends rather than covariates interacted with linear trends (column 3). A final specification reweights the comparison group using the inverse propensity scores of receiving a CHC (column 4, see table I-5 for trimmed estimates). Across specifications, the magnitudes and standard errors are similar. The addition of over 2,600 county-specific linear trends in column 3 alters the estimates by 1 death per 100,000 in years 0 to 4, by fewer than 5 deaths per 100,000 in years 5 to 9, and increases the effect size by 0.3 deaths in years 10 to 14. While inverse propensity score reweighting reduces the estimates' magnitudes, neither set of estimates (columns 3 or 4) are statistically distinguishable from our baseline specification. The robustness of the estimates in the reweighted sample is particularly helpful in narrowing the scope of omitted variables bias. Since the reweighted sample has a slightly larger number of physicians per capita and is slightly more likely to have a medical school (though neither difference is statistically different, table 3-1, columns 7 and 8), it is hard to argue that the diffusion of hospital treatments (such as the development of bypass surgery in 1968)

disproportionately affected CHC locations. Overall, the results imply that within 10 years CHCs reduced age-adjusted mortality rates by around 2 percent in treated counties.

### *3.3.2 The Relationship between CHCs and Mortality by Age Group*

CHCs' population-level effects reflect large changes in the mortality of individuals 50 or older, because they comprise the majority of deaths in the population. Figure 3-7 disaggregates CHCs' effects by four age groups. As expected, the pattern for older adults using our baseline model (figure 3-7D) is similar to the population-level estimates: CHCs are associated with a 2 percent reduction in age-adjusted mortality rates of older adults (see table 3-2B for alternative specifications).<sup>77</sup> The relationship between CHCs and mortality rates of other age-groups, however, is less evident. Although previous work has found a relationship between infant mortality and CHCs (Goldman and Grossman 1988), we do not find evidence of this relationship (figure 3-7A). However, 95-percent confidence intervals include sizable changes of  $\pm 2$  percent during the first five years CHCs operated.<sup>78</sup> This reflects both relatively small sample sizes (infants deaths comprise a maximum of 2 percent of all deaths in this period) and the fact that CHCs' primary care probably did not save many sick infants (who went to hospitals). Similarly, figure 3-7B provides no evidence that CHCs reduced child deaths, though these estimates are imprecise. 95-percent confidence intervals include changes in child mortality rates as large as 4 percent in either direction. Finally, figure 3-7C shows little evidence of a short-run relationship between adult deaths and CHCs but some evidence of a long-run relationship. This result partly reflects increases in sample size (adult mortality comprised around 10 percent of all deaths in this period) but is also consistent with CHCs' expected effects. Sustained access to primary care

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<sup>77</sup> Heterogeneity in CHCs' effects for ages 50 to 64, 65 to 79, and 80+ are in appendix table G5.

helps adults manage chronic and potentially lethal conditions, which would result in longer-term (but not immediate) mortality reductions. The lag between CHC establishment and reductions in mortality for this group, however, suggest caution in interpreting this effect.

In summary, all-cause, age-adjusted mortality rates fell rapidly after CHCs began. A large share of this fall is attributable to reductions in mortality risk among individuals 50 and older. CHCs' effects on other age groups may have been less immediate and, owing to the relative rarity of preventable deaths in these groups, more difficult to detect. The data provide some evidence that CHCs benefitted younger adults over the longer-term. However, we find no evidence of a relationship between CHCs and infant or child deaths.

### *3.3.3 The Relationship between CHCs and Mortality by Cause of Death*

Heterogeneity in the relationship between CHCs and mortality rates by cause of death provide more information on the possible mechanisms and, in particular, the hypothesis that CHCs reduced the costs of detecting and managing chronic conditions. Because CHCs' effects on mortality are concentrated among adults 50 and older, table 3-3A presents DD estimates by cause for this group. Using our baseline specification, column 1 presents estimates for all causes, and columns 2 to 7 present estimates for six leading causes of death. The results show that the greatest mortality reductions occurred for CVD-related causes: five to nine years after CHCs began, heart-disease-related mortality fell by 1.8 percent (27 deaths per 100,000, column 2) and other CVD-related mortality fell by 4 percent (17 deaths per 100,000, column 3). These findings are consistent with CHCs making blood pressure testing and treatment less expensive (due to more convenient testing and cheaper medications), both of which would improve treatment

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<sup>78</sup> Goldman and Grossman (1988: 69) find that CHCs reduced infant mortality by 0.1 and 0.3 deaths per 1,000 live births, or by 0.5 and 1.5 percent of the 1970 baseline rate. Our design cannot rule out effects of this size.

compliance. Reductions in CVD-related deaths may also reflect counseling about the warning signs of a heart attack or stroke, or simple advice to improve diet or quit smoking.

The effects of CHCs, however, appear to have extended beyond controlling hypertension. Mortality due to cancer and diabetes also fell just after CHCs were established. In years 5 to 9, deaths due to cancer were 1.8 percent lower (–11 deaths per 100,000, column 4), which may reflect increased longevity due to earlier detection. Deaths due to infectious diseases (column 5) and diabetes (column 6) were also lower but not statistically significant at conventional levels. In a falsification test, deaths due to accidents fail to register economically or statistically significant declines in any of the event-year groups (column 7).

One explanation for these effects is the interaction of CHCs' auxiliary services with Medicare. For instance, CHCs could affect older adult mortality by increasing knowledge of and enrollment in Medicare (passed in 1966) and by providing transportation to hospitals for the Medicare eligible. If an interaction with Medicare were driving our effects, then we would not expect mortality rates to fall after CHC establishment for the Medicare ineligible. Table 3-3B presents evidence rejecting this claim. For those 50 to 64, mortality due to diseases of the heart (column 2) and other CVD-related causes (column 3) fell by 1.4 and 5.3 percent, respectively, in years 5 to 9. Similarly, deaths due to cancer (column 4), infectious diseases (column 5) and diabetes (column 6) fell by 1.7, 4.5 and 7 percent, respectively, over the same time frame. Deaths by accident (column 7) are the only category not to improve after CHCs began, even though deaths due to this cause for 50 to 64 year olds occurred more often than those due to infectious disease and diabetes. This pattern of results is consistent with CHCs reducing mortality risk through their direct provision of primary care and low cost medications.



Given these results, another explanation for CHCs' effects is that CHCs effectively provided health insurance for the under- or uninsured poor. If this were the main mechanism, then we would expect mortality rates not to fall after CHC establishment for the Medicare eligible—a group with generous and near universal health insurance. Yet, table 3-3C shows that estimates for individuals ages 65 and older were also large. Five to nine years after CHCs began, deaths due to heart disease (column 2) and other CVD-related causes (column 3) were 1.7 and 3.2 percent lower. Mortality risk attributable to cancer was 2.3 percent lower (column 4). Reassuringly, deaths due to accidents were not significantly lower.

In summary, the effects of CHCs on the Medicare ineligible (50 to 64 year olds) show that the overall effects for older adults were not completely explained by an interaction with Medicare-funded services. Post-establishment reductions in deaths due to cancer, infectious diseases, and diabetes are consistent with CHCs promoting prevention, earlier diagnosis, and treatment compliance for a broad set of chronic conditions, including but not limited to hypertension. Moreover, CHCs' effects on the Medicare eligible suggest their potential for improving health outcomes for those with generous health insurance, suggesting that CHCs' primary care and auxiliary services complemented medical services paid for by health insurance.

#### *3.3.4 Translation of Intention to Treat Effects into Average Treatment Effects on the Treated*

These intention-to-treat estimates (ITT) average the effect of CHCs over all county residents ages 50 and older regardless of whether they benefited from CHC services. We use two approaches to approximate the implied average treatment effect on the “treated” (ATET)—those who, as a consequence of CHC establishment, obtained direct or indirect benefits they would not have otherwise received. The first approach assumes that all poor, older adults in a county with a

CHC program were “treated” even if they did not use the CHC facility. That is, CHCs affected both patients and their communities (through the eradication of infectious disease, knowledge spillovers, and reduced crowding in emergency rooms), but health effects were limited to the poor. Dividing the reduction in AMR (61: table 3-2B, average over columns 1 to 4 for years 5 to 9) by the older adult poverty rate in 1965 (22 percent) yields an ATET of 278 deaths per 100,000.

The second approach more narrowly assumes that CHCs only benefited their patients. We use the 1970 SHSUE to approximate the share of respondents ages 50 and older who lived in treated counties that had used CHCs over a five-year period. This approach suggests that 16 percent of all older adults in a county benefitted.<sup>79</sup> Dividing the AMR reduction by this figure yields an estimated ATET of 381 deaths per 100,000 ( $-61/0.16$ ). Of course, the ATETs could be even larger if some CHC users would have obtained the same services otherwise.

These two approaches imply a range of ATETs between 278 to 381 deaths per 100,000. ATETs in this range are roughly one half to two thirds the size of Chay et al.’s (2011) estimate of Medicare on one-year mortality rates. Using the 1966-1968 Mortality Followback Survey (MFS) to construct mortality rates by poverty status for those ages 50 and older, the ATETs suggest that

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<sup>79</sup> As a starting point, the SHSUE shows that 1.4 percent (1,358/100,000) of those 50 and older had used “a clinic not connected with a hospital—such as a Board of Health clinic or neighborhood health center.” 28 percent of older adults lived in CHC counties, which implies that 5 percent (1.4/0.28) of residents 50 and older in counties with CHCs had used them by 1970. We adjust this figure in two additional steps. First, we inflate the estimate to reflect the high degree of retrospective underreporting of clinic use. Bound et al.’s (2001) survey of measurement error in the reporting of public program use shows that survey responses agree with administrative records as little as 50 percent of the time; survey responses agree with provider records of “clinic visits” as little as 39 percent of the time (p. 3813). This implies that the SHSUE one-year utilization rate may have been as high as 12.8 percent ( $5,000/.39 = 12,820$  per 100,000). Second, we use nine surveys conducted by the OEO in CHC catchment areas between 1968 and 1972 to adjust the estimates for cumulative use over five years (see appendix A for details on data). For respondents 50 and older, 76 percent of physician visits that occurred in the previous five years took place one year prior to the survey, which inflates our estimates of 5-year CHC use to 16 percent (similar to the PSU-level increase in “clinic use” reported in section I)—very close to the direct evidence of 17 percent at the PSU level (section I.A). See appendix F for more details.

CHCs reduced the annual AMR by 7 to 9 percent for the poor within a decade.<sup>80</sup> Consistent with CHCs reducing health disparities, the effects are also equivalent to 20 to 28 percent of the mortality gap between the poor and the non-poor for this age group. The implied magnitudes are, therefore, plausible given the poor’s higher mortality rates and the greater potential need for medical services among those using CHCs.

### **3.4 Mechanisms for the Effects of Community Health Centers on Mortality**

Thus far we document a large, negative relationship between CHCs and older adult mortality for a broad set of causes of death. To understand the mechanisms for CHCs’ mortality effects, this section, first, examines effect heterogeneity by pre-treatment community characteristics and, then, examines directly the role of increases in the use of primary care, anti-hypertensive drugs, and Medicare utilization.

#### *3.4.1 Heterogeneity in the Relationship of CHCs with Older Adult Mortality*

##### *Rates*

Our heterogeneity analysis provides descriptive evidence relating to hypotheses in the historical literature. For ease of interpretation, we implement these tests by replacing the event-year dummies in equation 1 with  $\sum_k (\sum_{g=-2}^{-1} \tilde{\pi}_g^k D_j^k D_j^g + \sum_{g=0}^3 \tilde{\tau}_g^k D_j^k D_j^g)$ , where  $D_j^k$  is equal to 1 if a county received a CHC between 1965 and 1974 and belongs to group k (defined subsequently).

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<sup>80</sup> Vital Statistics mortality data do not contain information on income or education. The 1966-1968 National Mortality Followback Survey (US DHHS and ICPSR 1986) sampled death certificates from 1966 to 1968 and surveyed death informants about the decedent’s household income in 1965. Together with the 1965 CPS population estimates, these data allow us to compute mortality rates by income (King et al. 2010). This survey shows that the AMR for those 50 and older was 50 percent higher among those living in a household with a total income of less than the 1965 poverty line for a family of four (<\$3,000) than among those above this threshold (4,127 versus 2,769).

One hypothesis in the literature is that CHCs reduced excess mortality—deaths that would not have occurred if individuals had access to primary care and treatment for chronic conditions. Although the CHC locations do not appear related to their pre-program mortality rates (table 3-1, figure 3-4), this hypothesis suggests that CHCs could have larger effects in areas with higher mortality. The results in table 3-4 bear this out. Areas with above-median mortality rates (column 2) experienced more than 50 percent larger absolute and relative reductions in mortality rates than did areas with below-median mortality rates (column 1) 5 to 9 years after the CHC began; the reduction was twice as high in the above-median group in years 10 to 14.

A second hypothesis is that CHCs would have larger effects in areas that were underserved by physicians (MDs). Table 3-4, however, is inconsistent with this hypothesis. Rather, areas with more physicians per capita in 1960 saw a 50 percent greater absolute reduction in mortality rates 5 to 9 years after CHCs began. The proportional reductions were 2.4 percent reduction in the high-MD counties ( $-79/3,276$ , column 4) and 1.9 percent in the low-MD counties ( $-58/3,007$ , column 3), but we fail to reject the hypothesis that reductions in mortality rates were equal across these groups ( $p$ -value = 0.36).<sup>81</sup> This evidence suggests that the convenience or affordability of care may be more important than CHCs' direct provision of physicians.

A third hypothesis is that CHCs benefitted nonwhite families more, both because nonwhite families tended to have lower incomes and also because War on Poverty programs actively sought to ameliorate racial disparities. In contrast, table 3-4 shows that the results for white (columns 6) mortality are larger and more precise than those for nonwhites (column 5), although the estimates are not statistically different ( $p$ -value = 0.28). One explanation is the

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<sup>81</sup> This may reflect the positive correlation between per-capita MDs and a county's urban share, which figure 6 shows to be associated with the effects of CHCs. Three quarters of treated counties with urban shares above (below) the median have per-capita MD values above (below) the median as well.

higher noncompliance of nonwhites with prescribed treatments (Simeonova 2013). A second explanation is that fewer nonwhites had heard of CHCs as shown by surveys in the 1960s in 11 cities of CHC catchment areas (appendix table K3). A third explanation is that “frailty” differed between older white and nonwhite adults (Manton and Stallard 1984). If nonwhites had higher mortality rates at younger ages, then this would lead to a healthier surviving population of nonwhite adults after age 50—adults who had less to gain from CHC care.

A final hypothesis is that CHCs’ effects may have been largest in the South: the census region with the most poverty and unmet medical need. Table 3-4’s estimates also fail to support this hypothesis. The relationship between CHCs and mortality rates is statistically indistinguishable in specifications which omit the South (column 9) and those that omit other regions (columns 7,8 and 10). The effects of CHCs appear in all census regions.

These results suggest that primary care and the management of chronic conditions were important mechanisms for CHCs’ mortality effects. CHCs’ effects were largest in areas with the highest pre-program mortality rates, urban areas, and areas with more (not fewer) physicians per capita. Thus, their effects do not appear to be driven by the provision of health care professionals to underserved areas (i.e., those with few MDs, those with higher poverty rates, or those in the South) or concentrated among nonwhites.

### *3.4.2 Did CHCs Increase the Use of Primary Care and Prescription Drugs?*

The 1963 and 1970 SHSUE provide direct evidence on CHCs’ role in increasing the use of primary care and prescription medications. For ease of interpretation, we estimate the simple DD model described in section I.A separately for three household income groups (below 100 percent of the poverty line, 101 to 299 percent, and 300 percent or above). The results presented in table 3-5 provide evidence that CHCs increased the likelihood of older adults in poverty

reporting a “regular source of care” by around 25 percent (0.19/0.77, column 1). Reporting a regular source of care is a stronger predictor of positive health outcomes than is having health insurance (Sox et al. 1998); it is also highly correlated with compliance with anti-hypertension treatment (Shea et al. 1992). In addition, CHCs are associated with a 37 percent reduction (−0.22/0.58, column 3) in out-of-pocket prescription drug expenditures among older adults in poverty. CHCs, however, are not associated with an increase in the likelihood of seeing a physician (column 5) or an increase in medical visits (column 4). (The latter estimate is economically significant but imprecise.)

These changes are not evident among higher income individuals in areas that received CHCs (table 3-5, panels B and C). The same outcomes have the opposite signs, are smaller in magnitude, and are statistically insignificant with one exception: out-of-pocket prescription drug expenditures were 20 percent lower among older individuals with incomes between 100 and 299 percent of the poverty line. This suggests that the near poor used CHCs’ in-house pharmacies, which likely increased their compliance with medication regimens.

### *3.4.3 How Important Were Changes in the Use of Anti-Hypertensive Medications?*

The findings that CHCs reduced prescription drug expenditures for the poor or near poor and that they reduced CVD-related deaths suggest that CHCs increased access to anti-hypertensive medications for these individuals. A simple back-of-the-envelope calculation allows us to evaluate anti-hypertensive medications as a mechanism. To this end, we use the 1973 Hypertension Detection and Follow-up Program (HDFP)—a large, community-based,

randomized trial of the mortality effects of hypertension drug treatment over five years.<sup>82</sup> HDFP participants ages 50 to 70 who were prescribed anti-hypertensive drugs and provided with stepped up care (similar to the outreach and follow-up provided by CHCs) experienced a reduction in the five-year, all-cause mortality rates of -2,160 deaths per 100,000 (HDFP 1979; table 3-9). To translate this result into a population-level effect (not just for hypertensives as in the HDFP), we multiply this treatment effect by the share of people with hypertension (26.2 percent, National Health Examination Survey) and also by the share in treated communities who used CHCs (16 percent, section III.D). If CHCs provided anti-hypertensive medication but no screening (as in the HDFP study), the aggregate, age-adjusted five-year mortality rate would have fallen by 91 deaths ( $2,160 \times 0.262 \times 0.16$ ) per 100,000. This estimate understates CHCs' effects on hypertension-related mortality through screening, education, and other channels.

To facilitate a comparison, we translate our ITT estimates of one-year mortality reductions for years 5 to 9 (-61, table 3-2B) into a five-year mortality reduction using the mean age-adjusted mortality rate in year -1 (3,213 deaths per 100,000, table 3-2) as the counterfactual mortality rate. Our estimates imply a reduction of 264 deaths [ $= (1 - .03213)^5 - (1 - (.03213 - .0006))^5$ ] per 100,000 in five-year mortality rates. Comparing this estimate to the HDFP results implies that treatment for hypertension could account for 34 percent (91/264) of our ITT effects. In short, anti-hypertensive medication could be an important part of the story, but it does not

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<sup>82</sup> The 1973 HDFP trial obtained a sample of over 10,000 participants with hypertension (diastolic blood pressure over 90 mm Hg). A randomly selected treatment group saw HDFP staff on a flexible schedule, received frequent follow-up, and, importantly, was prescribed anti-hypertensive medication. The control group learned about their hypertension and was referred to their usual source of care (and may have taken anti-hypertensive drugs). The HDFP did not assess the role of screening and diagnosis (all participants were screened and informed of their hypertension), but the treatment group received anti-hypertensive drugs and auxiliary services similar to the services provided by CHCs.

appear to be the entire story.<sup>83</sup> This is consistent with CHCs' medical and auxiliary services improving the prevention, detection, and management of other chronic conditions among older adults.

#### 3.4.4 *Did CHCs Increase the Use of Medicare?*

For the elderly, CHCs may also reduce mortality by complementing services provided by Medicare. For instance, CHCs may increase knowledge about and use of Medicare-covered services (e.g., via counseling and transportation to hospitals). To examine this channel, we estimate our baseline model using as dependent variables real, county-level per-capita military and Medicare expenditures (available from 1959 to 1988, figure 3-8A) and Medicare per enrollee expenditures (available for 1966 forward, figure 3-8B).<sup>84</sup> If the effects of CHCs on elderly mortality arose primarily through increased use of Medicare-funded hospital treatments such as bypass surgery, then we should see per-enrollee Medicare spending increase after CHCs began. Figure 3-8, however, provides little evidence of such a relationship. Total per-capita medical spending evolved smoothly before and after CHCs began, and per-enrollee Medicare expenditures did not increase differentially after CHCs began (panel A). The absence of large aggregate changes, however, could imply no effect or could result from the aggregation of offsetting effects. On the one hand, CHCs could increase awareness of Medicare while, on the other, reducing the need for Medicare-covered services. If greater awareness led some patients to use Medicare-covered services while diverting others from hospitals, CHCs could have no effect

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<sup>83</sup> As a comparison, the RAND Health Insurance Experiment found that nearly half of the reduction in blood pressure difference between those receiving free care and those with co-payment plans was attributable to the one-time initial blood pressure screening (Newhouse et al. 1993: 229, 243).

<sup>84</sup> Because information on all per-capita medical expenditures is available from 1959 to 1988, figure 8A presents estimates for this outcome from -6 forward. In the specifications using Medicare expenditures as the dependent variable (figures 8A and 8B), we omit the pre-treatment coefficients from the figure because they are not based upon a balanced set of counties. In figure 8B, we exclude counties with CHCs established before 1966 for the same reason.



on Medicare expenditure per capita or per enrollee (although they would increase efficiency). Panel B provides suggestive evidence of patient diversion. In fact, CHCs appear to have reduced Medicare enrollment (p-value = 0.013 on a joint test that post-coefficients are not zero), suggesting that some elderly could have used CHCs rather than hospitals.<sup>85</sup> In summary, the available evidence points to longer-term benefits of primary care and lower-cost medication provided by CHCs as mechanisms. Interactions of CHC care with public insurance provided by Medicare do not seem to be important factors driving their mortality effects.

### **3.5 Alternative Explanations? Potential Threats to Internal Validity**

A final section examines potential threats to the internal validity of the study, which include local shocks that both reduce mortality and occur concurrently with or just after CHCs began. For instance, coincident changes in other federal spending (e.g., other OEO programs), other local medical resources, or Medicaid coverage could drive our estimates. We first investigate whether CHC establishment coincided with other federal OEO grants using newly compiled data on grants for other federal programs. This could have happened inadvertently or because certain communities were more effective in obtaining funding (e.g., more affluent urban areas, table 3-1). Large coincident increases in other federal funding (that also reduced older adult mortality) could threaten the internal validity of our estimates.<sup>86</sup> To investigate this, we estimate regressions similar to equation (1) and replace the dependent variable with a binary measure equal to one if county  $j$  received a grant for a program in year  $t$ . Figure 3-9A shows little evidence that CHC establishment coincided with increases in other local funding. To fix ideas, we use a binary variable equal to 1 if a county received a CHC grant (thick line, no markers). By

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<sup>85</sup> CHCs did not collect Medicare reimbursements in this period.

<sup>86</sup> Evidence that other OEO programs reduced mortality makes this a reasonable concern. Ludwig and Miller (2007) document the relationship between Head Start and child mortality and Almond et al. (2011) document the relationship between Food Stamps and infant mortality. These programs could benefit older adults indirectly by freeing up or increasing family resources.

construction, the estimates reach one in the year CHCs began (100 percent of treated counties received a CHC grant in event-year 0). The share tapers to around 50 percent five years later as some CHCs received multi-year grants.<sup>87</sup> For our estimates to confound changes in other federal funding with CHCs, grants for other programs would need to show a similar level shift or trend-break around year 0. No such patterns emerge, however, for other Community Action Program (CAP) health projects, CAP administration (including local development projects), programs serving the elderly, Head Start, legal services, or Food Stamps. We cannot rule out funding changes in programs we do not measure, but these patterns are reassuring.<sup>88</sup>

We next investigate whether the establishment of CHCs coincided with local changes in other health resources as proxied by local hospital capacity. This could be the case if, for instance, the leading edge individual getting the CHC grant also received a grant to increase local hospital size. Using data from 1948 to 1990 from the American Hospital Association's (AHA) Annual Survey, figure 3-9B provides little evidence that this was the case. Using the number of hospitals and the number of hospital beds (both measured per 1,000 residents) as dependent variables, the estimates show that both outcomes evolved smoothly before and after CHCs began. The absence of a trend-break in hospital beds per capita (0.008, s.e. 0.017) provides little evidence that we misattribute changes in local hospital capacity to CHCs. Moreover, the trend break in the number of hospitals shows that medical resources in untreated areas worked against the mortality reductions we find. Specifically, the marginally significant slowdown in the growth of the number of hospitals after CHCs began (0.0002, s.e. 0.0001) is consistent with areas

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<sup>87</sup> For instance, 92 percent of locations treated before 1975 also received a federal grant between 1978 and 1980. Only a handful of CHCs closed over the entire period we consider.

<sup>88</sup> Note that a coincident expansion of, say, local health programs *in response to* a CHC would not confound our estimates, but would represent another causal channel ("crowd-in") through which CHCs reduced mortality.

without CHCs catching up to areas receiving CHCs. If growing hospital resources reduced mortality rates in areas without CHCs, this should work against the mortality effects we find.

A final explanation for the mortality effects of CHCs is that counties with CHCs may have benefited disproportionately from Medicaid. Whereas our baseline model accounts for Medicaid's state-level roll-out by using state-by-year fixed effects, Medicaid's effects may have been larger in the poorer, urban areas also served by CHCs (for reasons unrelated to CHCs).<sup>89</sup> To evaluate this, we include dummies for event time relative to state Medicaid implementation interacted with county-level characteristics such as high 1960 poverty rates, high numbers of active physicians, and the presence of a medical school. Our estimates of CHCs' effects, however, remain similar in magnitude and statistically indistinguishable from our baseline estimates (appendix figure 3-E3). In summary, we find little evidence that correlated local shocks in federal spending, medical resources, or state Medicaid programs compromise the internal validity of our research design.

### **3.6 The Longer-Term Returns to Primary Care**

Since 1965, the CHC experiment has been an important yet understudied part of the U.S. health care safety net—not least because the CHC program costs so much less than Medicare and Medicaid. Even recent political support for CHCs relates, in part, to their role as an alternative to expanding public health insurance (Mickey 2011). An important lesson from our analysis is that public investments in the delivery of primary care may yield large returns for the underserved and underinsured population, and even to individuals eligible for Medicare.

The CHC program's rollout from 1965 to 1974 presents a rare opportunity to quantify the effects of changes in access to primary care among the underserved. Within an event-study

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<sup>89</sup> Regressing the year in which a CHC was established on the year in which Medicaid was passed results in a correlation of 0.071 (s.e. 0.081). Excluding Arizona, which is an outlier as a late Medicaid adopter, the correlation is 0.216 (s.e. 0.150). State-wide effects of Medicaid are captured in the specifications with state-by-year fixed effects.

framework, our empirical strategy exploits the disorganized grant-making process during the early years of the War on Poverty. Consistent with accounts of “great administrative confusion” at the OEO (Levine 1970), our data show no association of CHC program establishment with a variety of pre-treatment county characteristics: 1965 mortality rates, changes in mortality rates from 1960 to 1965, funding for other OEO programs, or local expansions in hospital capacity. The establishment of a CHC, however, predicts sharp reductions in older-adult mortality.

Our results imply that CHC-induced increases in primary care led to sustained health improvements over at least 15 years. One decade after CHCs were established, age-adjusted all-cause mortality rates remained almost 2 percent lower than pre-program rates, owing primarily to large reductions in cardiovascular-related deaths among adults over age 50. The implied treatment effects on the treated are a 7 to 9 percent reduction in age-adjusted mortality rates among residents likely to have benefited from CHCs, which amounts to a 20 to 28 percent reduction in the 1966 poor/non-poor mortality gap for the same age group. Some of CHCs’ longer-term benefits accrued to individuals ineligible for Medicare (ages 50 to 64), but the program achieved large mortality reductions among the Medicare eligible without an accompanying increase in Medicare spending. Important reasons for this are that CHCs reduced the cost of prevention, diagnosis, and management of chronic conditions and provided free or substantially discounted prescription medications.

What do these findings imply about the cost-effectiveness of CHCs relative to other public health interventions? Assuming that mortality is the only outcome CHCs affected allows us to estimate CHCs’ cost per year-of-life gained. Multiplying our baseline estimates (figure 3-7D) by the older adult population in treated counties during CHCs’ first ten years implies 81,644 years of life gained after CHCs began. We obtain the total cost of the CHC program over its first

ten years by estimating the average annual federal cost of CHCs and multiplying by the 114 CHCs in our sample. This yields a total of \$4.4 billion in 2012 dollars and a cost-per-year-of-life ratio of approximately \$54,000. The cost-effectiveness of Medicare at implementation provides a natural point of comparison. Using Chay et al.'s (2011) regression-discontinuity estimates of Medicare's effects on elderly mortality at the time of implementation and our data on total Medicare expenditures suggests a cost-per-year-of-life ratio ranging from approximately \$2.5 to \$7.1 million in 2012 dollars.<sup>90</sup> Scaling these estimates by remaining life expectancy (Chay et al. estimate 14.5 years) reduces the cost per one year of life to between \$161,373 to \$459,000 in 2012 dollars—3 to 8 times the ratio for CHCs begun in the same period. Adjusting the CHC cost-ratios for the program's impact on life expectancy could further shift these calculations in CHCs' favor. Both ratios are considerably less than the value of a statistical life in Ashenfelter and Greenstone (2004), who cite a preferred estimate of \$2.19 million in 2012 dollars (\$1.54 million in 1997 dollars), and also much lower than earlier surveys (Viscusi 1992, Manning et al. 1989).

These cost ratios likely understate the broader effects of increasing access to primary care by expanding CHCs, because mortality fails to capture changes in morbidity, disability and other gains in health and well-being. These cost ratios, however, suggest that CHCs achieved their primary objective of improving health at much lower cost than larger public insurance programs—especially for the elderly. Whether CHCs' health benefits remain this large today and whether CHCs benefited the non-elderly remain important areas for future research.

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<sup>90</sup> Chay et al. (2011) present much smaller cost-effectiveness estimates based on total gains in life expectancy and the *additional* spending on inpatient hospital care induced by Medicare. Here we use the average annual total cost of Medicare.

### 3.7 References

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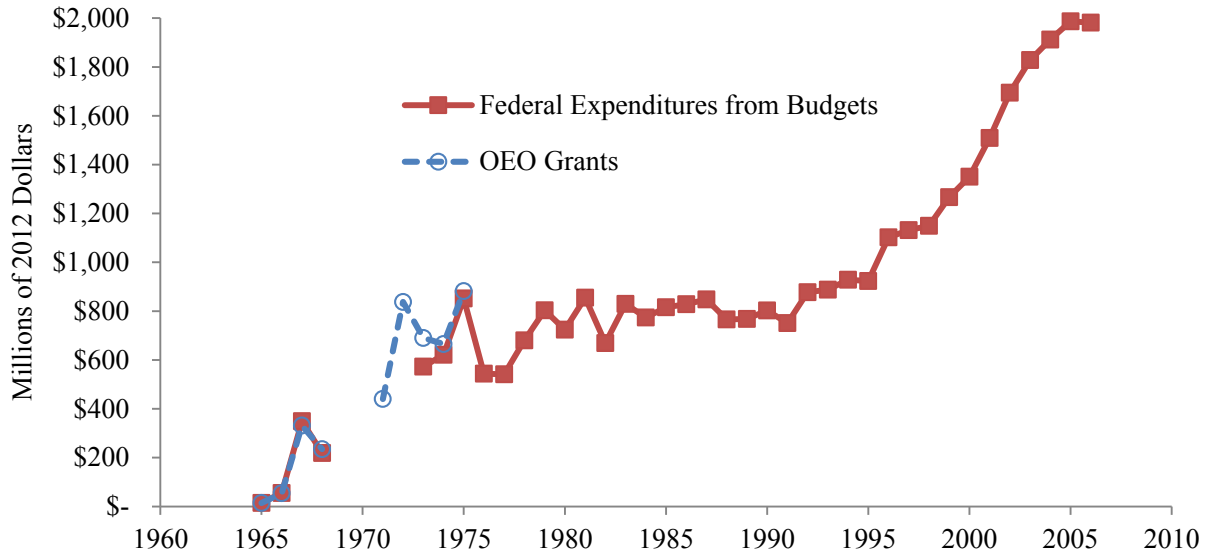


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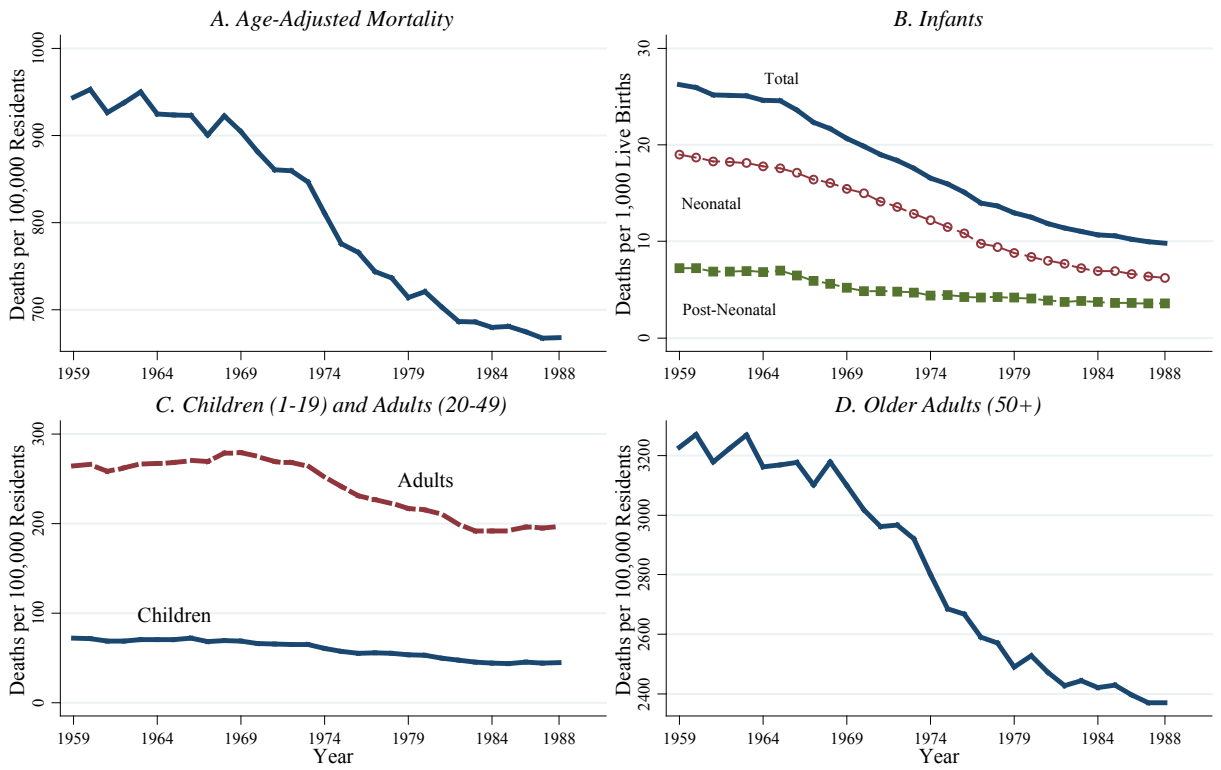


**Figure 3-1 Community Health Center Funding, 1965 to 2000**



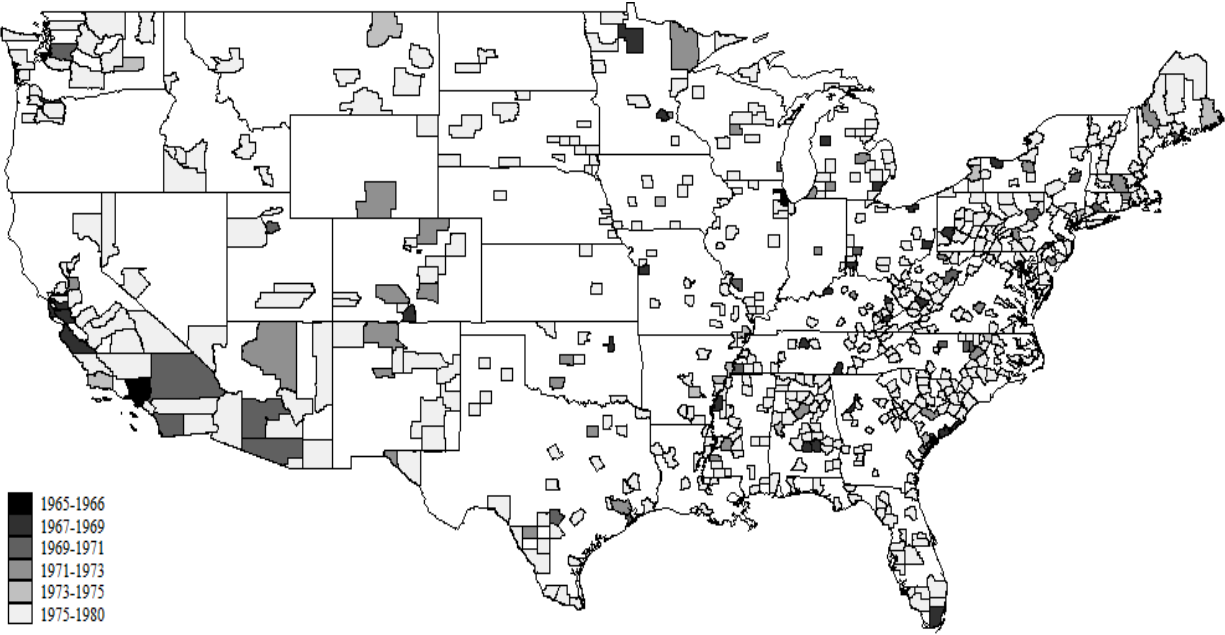
Federal expenditure data are expenditures for the Neighborhood or Community Health Center program. Differences between federal expenditures and state grants received may be due to double-counting of centers or for funding spread over multiple years that is reported in one year only. Source: Information on OEO grants comes from the NACAP and NAFO files. Federal expenditures data are taken from line-items in the Budget of the United States Government and U.S. Dept. of Health, Education and Welfare.

**Figure 3-2 All-Cause Mortality Rates by Age Group, 1959 to 1988**



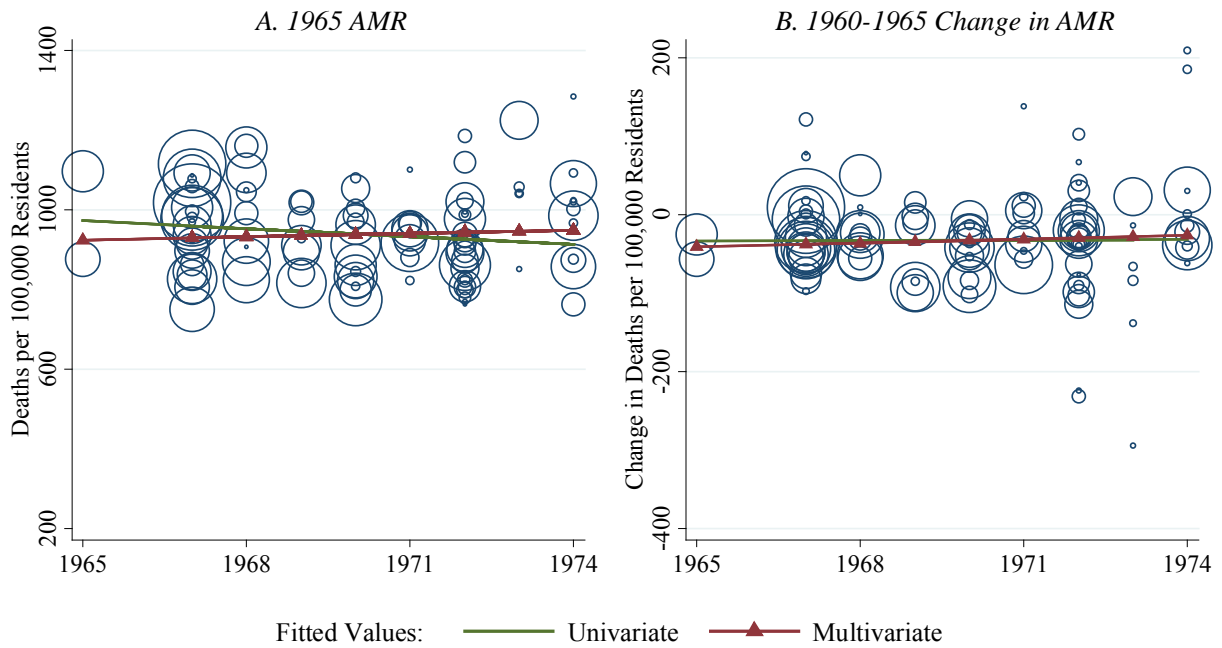
Sources: Vital Statistics Multiple-Cause of Death Files (US DHHS 2007), 1950 and 1960 population estimates (Haines and ICPSR 2005), and 1969 to 1988 population statistics (SEER 2009).

**Figure 3-3 Establishment of Community Health Centers by County of Service Delivery, 1965 to 1980**



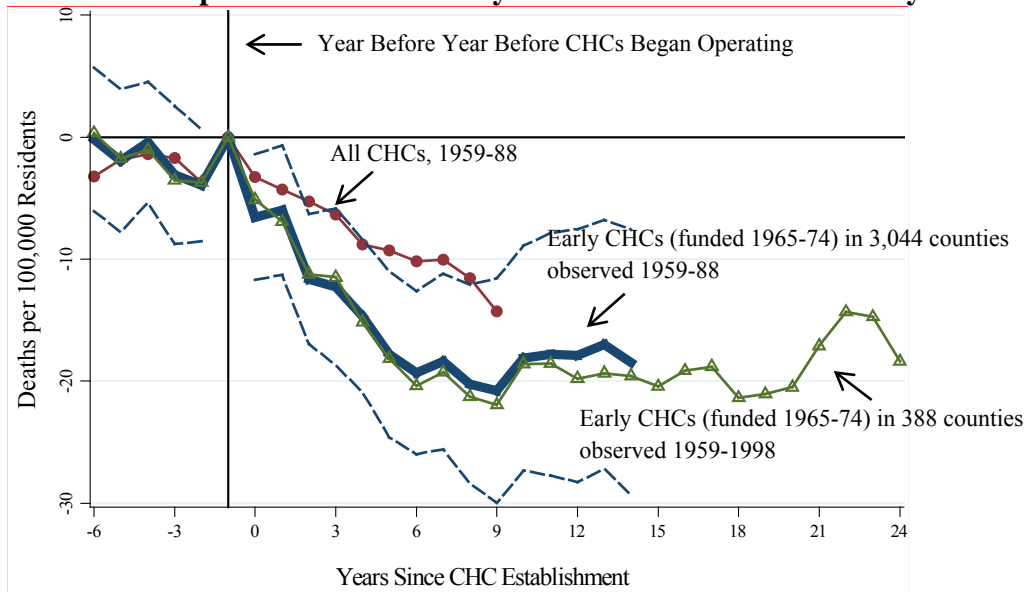
Note: Dates are the first year that a CHC was established in the county. Source: Information on CHCs drawn from NACAP and PHS Reports.

**Figure 3-4 Relationship between Community Health Centers Initiation and Mortality Rates**



Notes: AMR=Age adjusted mortality rate. The dependent variable refers to (levels of or changes in) age-adjusted mortality over all ages. Univariate fitted values are from regressions of the dependent variable on the year CHCs were established for the 114 treated counties in the estimation sample. The estimated univariate slopes are -6.6 (s.e. = 6.1) for panel A, and 0.2 (s.e. = 1.4) for panel B. Multivariate regressions follow Almond et al. (2012) and include the 1960 share of the county population that is urban, rural, between ages 0 and 4, older than 64, nonwhite, has more than 12 years of education, has less than 4 years of education, has family income less than \$3,000, has family income more than \$10,000; and the per-capita number of physicians (see table 1). The estimated multivariate slopes are 2.8 (s.e. = 2.5) for panel A and 1.6 (s.e. = 1.7) for panel B. Source: See figures 1 and 2.

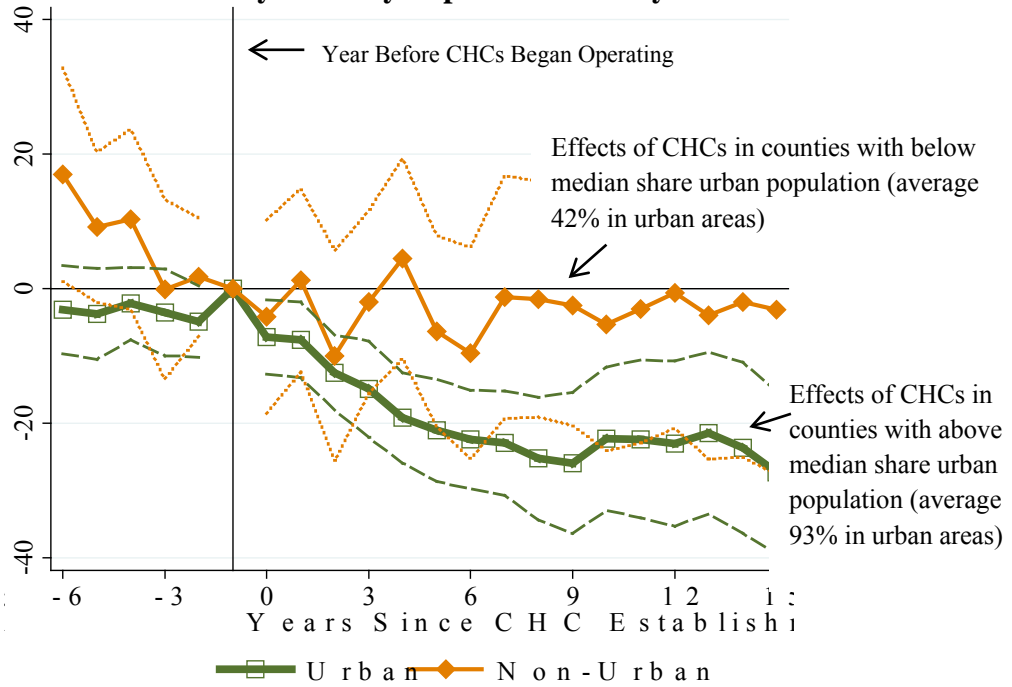
**Figure 3-5 The Relationship between Community Health Centers and Mortality Rates**



Notes: The dependent variable is the age-adjusted mortality rate (AMR) per 100,000 residents. The coefficients are weighted, least-squares estimates of  $\pi$  and  $\tau$  from our baseline specification of equation 1. Dashed lines are 95-percent confidence intervals using standard errors corrected for an arbitrary covariance structure at the county level. Weights are the total county populations in 1960. See text for further model details. The year prior to the establishment of a CHC is omitted because CHCs were funded for the entirety of years 1 to 14 but only for part of year 0. Samples: 1959-1988: 3,044 U.S. counties with valid data on 1960 characteristics (91,320 county-year observations); 1959-1998: 388 U.S. counties that are identified in each year of Vital Statistics data (15,520 county-year observations). Source: Mortality rates constructed from the 1959 to 1988 Vital Statistics Multiple-Cause of Death Files (US DHHS 2007), 1950 and 1960 population estimates (Haines and ICPSR 2005), and 1969 to 1988 population statistics (SEER 2009). Information on CHCs is drawn from NACAP and PHS Reports.

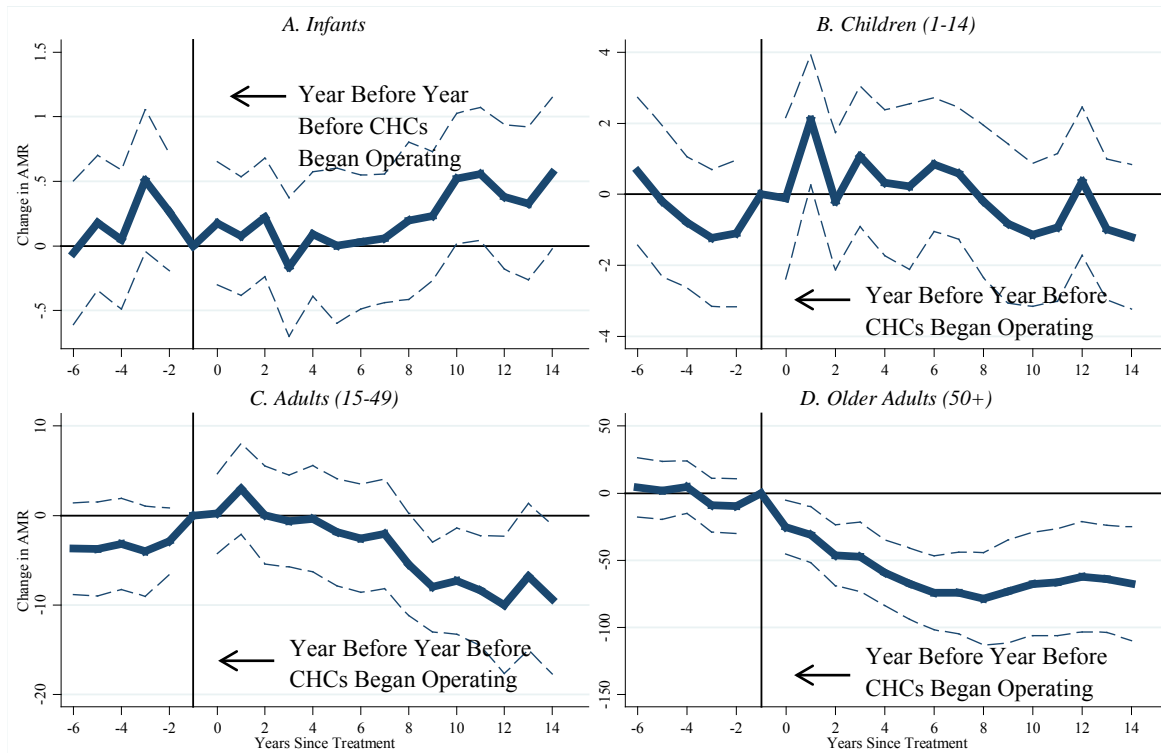


**Figure 3-6 Heterogeneity in the Relationship between Community Health Centers and Mortality Rates by Population Density**



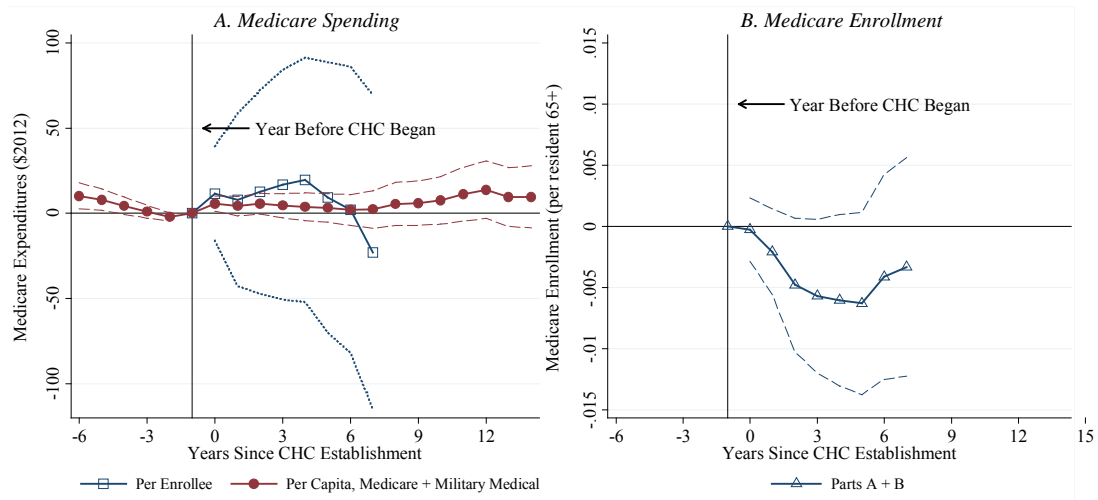
Notes: The coefficients are weighted, least-squares estimates of  $\pi$  and  $\tau$  from our baseline specification of equation 1 where the event-study dummies are estimated separately for areas with above (labeled “urban”) and below (labeled “non-urban”) the median urban share of the population among treated counties in 1960 (81%). See figure 3-5 notes for details on the specification and sources.

**Figure 3-7 The Relationship between Community Health Centers and Age-Group Mortality Rates**



Notes: The dependent variable is the all-cause, age-adjusted mortality rate for the indicated age group. Infant mortality is measured per 1,000 live births and mortality rates for other groups are measured per 100,000 residents. Weights are the appropriate county populations in 1960. Infant sample: 2,963 counties with valid data on 1960 characteristics identified in both mortality and natality files (88,890 county-year observations). Mean of infant mortality rate in treated counties in t-1: 22.1. Non-infant sample: 3,044 U.S. counties with valid data on 1960 characteristics (91,320 county-year observations). Mean of AMR in treated counties in t-1 for children is 63.8; for adults is 287.6; and for older adults is 3225.9. See notes to figure 3-5 for details.

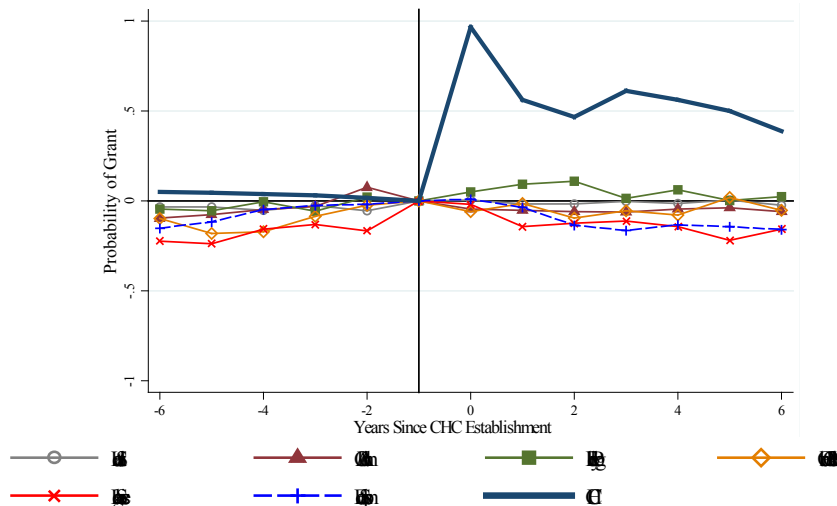
**Figure 3-8 The Relationship between Community Health Centers and Medicare Utilization**



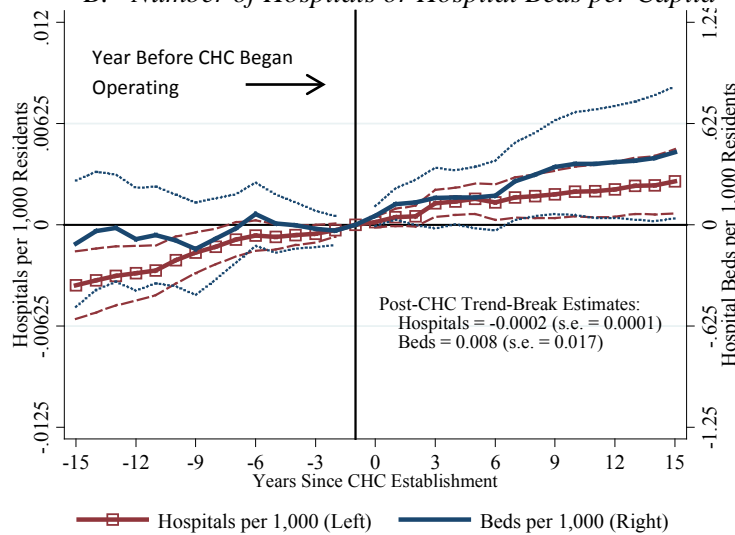
Notes: The figure plots weighted least-squares estimates of  $\pi$  and  $\tau$  from our baseline specification of equation 1. The dependent variable in panel A is real (\$2012) Medicare spending per enrollee by (parts A and B) and in panel B it is the Medicare enrollment rates (enrollees divided by county population 65 and older). In treated counties in the year before CHC establishment, the sample means are 0.97 and 0.93 for enrollment in parts A and B, \$1,089.83 for per-enrollee spending on part A, and \$429.22 for B. 1973 is missing and is linearly interpolated. Data from July 1966 through December 1967 is allocated to calendar years 1966 and 1967 in proportion to the number of months (1/3 and 2/3). Sources: County-level Medicare (US SSA 1969-1977; US HFA 1978-1980) and the Area Resource File (US DHHS 1994). Data on Medicare and military medical expenditures (panel A) were shared by Almond, Hoynes and Schanzenbach (2011).

### Figure 3-9 Relationship between Community Health Centers Establishment, Other Federal Program Grants, and Hospital Capacity

#### A. Share of Counties Receiving a Federal Grant, by Program



#### B. Number of Hospitals or Hospital Beds per Capita



Notes: The figure plots weighted least-squares estimates of  $\pi$  and  $\tau$  from our baseline specification of equation 1. In panel A, the dependent variable is equal to 1 if the county received any federal grant for the indicated program in a given year. In the case of Food Stamps, the dependent variable is equal to 1 at the date of implementation. In panel B, the dependent variables are hospitals per thousand residents (left vertical axis) and beds per thousand residents (right vertical axis). The sample excludes mental institutions, tuberculosis sanatoriums, military hospitals, and correctional hospitals. The sample means are 0.025 for hospitals per capita and 6.18 for beds per capita in treated counties in the year before CHC establishment. We omit REIS variables from panel B specifications (because they are not measured before 1959) and the AHA variables (because they are the key left hand side variables). Data for 1954, 1977 and 1979 are missing and are linearly interpolated. Trend break estimates come from a model which contains an event-time variable, an interaction between event-time and post-treatment, and a dummy for post-treatment. See figure 3-5 notes for details on the specification and sample. Sources: NACAP, NAFO, Public Health Services Reports (see appendix A); Almond, Hoynes and Schanzenbach (2011) for the Food Stamp data, 1948 to 1975 AHA Surveys (provided by Amy Finkelstein), and the 1972 to 1990 AHA Surveys (provided by the NBER).

**Table 3-1 1960 Characteristics of Counties Receiving Community Health Centers, 1965 to 1980**

	CHC Established in				(5) CHC Before 1975 (N=114)	Full Sample		Reweighted Sample	
	(1) 1965- 1967 (N=26)	(2) 1968- 1970 (N=32)	(3) 1971- 1974 (N=56)	(4) 1975- 1980 (N=497)		(6) Other Counties (N=2930)	(7) P-value on t-test of difference (5)-(6)	(8) Other Counties (N=2930)	(9) P-value on t-test of difference (5)-(8)
Mean Total 1960 Population	675,466	432,075	249,452	75,548	397,876	38,784	<0.01	128,379	0.51
Percent of Total 1960 Population:									
in urban area	74.6	74.8	57.8	36.2	66.4	30.5	<0.01	80.5	0.39
in rural area	4.5	4.0	10.3	18.9	7.2	23.4	<0.01	3.5	0.49
in Northeast	15.4	15.6	19.6	13.5	17.5	6.6	<0.01	5.9	0.70
in Midwest	23.1	18.8	16.1	19.3	18.4	35.1	<0.01	14.8	0.85
in South	38.5	40.6	44.6	53.3	42.1	45.6	0.47	35.1	0.91
in West	23.1	25.0	19.6	13.9	21.9	12.8	<0.01	44.1	0.46
under 5 years of age	11.0	11.8	12.3	11.5	11.8	11.1	<0.01	12.3	0.63
65 or older	9.7	8.5	8.4	9.6	8.7	10.7	<0.01	6.6	0.44
Nonwhite	14.7	17.7	18.3	16.1	17.3	10.4	<0.01	15.1	0.86
with <4 years of education	9.9	10.3	13.2	13.6	11.6	11.1	0.55	7.9	0.57
with >12 years of education	39.6	41.4	38.1	33.2	39.4	36.4	0.77	53.0	0.59
in households with income <\$3k	24.2	23.8	29.7	37.1	26.8	36.2	<0.01	18.2	0.47
in households with income > \$10k	14.8	13.8	12.1	8.0	13.2	7.7	<0.01	22.1	0.37
Medical Resources:									
Total Active MDs (per 1k)	1.6	1.6	0.9	0.6	1.3	0.6	<0.01	1.4	0.69
Any Medical Students, 1969	0.6	0.5	0.2	0.0	0.4	0.0	<0.01	0.3	0.87
Age-Adjusted Mortality in 1965	1,027.9	928.3	918.1	978.7	946.0	979.3	0.1	798.2	0.49

Notes: County characteristics are not weighted by 1960 county populations so that they can be interpreted as the shares for the average county in the relevant category. Column 8 applies propensity-score weights as described in the text and appendix figure G-4. Column (8) p-values are based on a parametric percentile-t bootstrap procedure with 1,000 replications (Jeong and Maddala 1993, Horowitz 2001). The table sample (our estimation sample) contains 3,044 counties. Sources: 1960 County and City Databooks (Haines 2005) and 1990 Area Resource Files (US DHHS 1994). Information on CHCs described in figure 3-1 notes.

**Table 3-2 Robustness Checks on the Relationship between Community Health Centers and All-Cause Mortality Rates**

	(1)	(2)	(3)	(4)
<i>A. Age-Adjusted Mortality, All Ages</i>				
Mean at t*=-1	929.3			
Years -6 to -2	0.0 [2.9]	-2.0 [2.1]	1.0 [2.8]	-2.7 [2.1]
Years 0 to 4	-5.6 [3.5]	-10.1 [2.3]	-9.1 [2.6]	-9.0 [2.4]
Years 5 to 9	-12.1 [4.6]	-18.9 [3.5]	-14.2 [3.7]	-15.7 [3.5]
Years 10 to 14	-9.4 [5.6]	-17.5 [4.8]	-17.8 [4.9]	-11.8 [4.6]
R2	0.82	0.85	0.96	0.87
<i>B. Age-Adjusted Mortality, 50 Years and Older</i>				
Mean at t*=-1	3,213			
Years -6 to -2	10.6 [10.2]	-2.0 [8.0]	4.6 [10.4]	-3.3 [8.1]
Years 0 to 4	-29.5 [13.7]	-41.1 [9.6]	-33.5 [11.2]	-38.2 [8.9]
Years 5 to 9	-58.4 [17.3]	-72.0 [14.8]	-52.1 [15.6]	-62.3 [11.7]
Years 10 to 14	-48.7 [21.1]	-64.1 [19.3]	-61.4 [19.2]	-46.9 [15.3]
R2	0.78	0.80	0.95	0.84
Covariates	C, U-Y	C, U-Y, S-Y, R, D·Year	C, U-Y, S-Y, R, C·Year	C, U-Y, S-Y, R, P-weights

Notes: Models presented are weighted least-squares estimates of equation 1 using event-year categories. C: county fixed effects; U-Y: urban by year fixed effects; S-Y: state-by-year fixed effects; R: annual, county-level covariates; D·Year: 1960 characteristics interacted with linear time trends; C·Year: county-specific linear time trends; P-weights: uses an estimate of the propensity of receiving a CHC to reweight untreated counties. See text for more details. Weights are the appropriate county populations in 1960. See notes to figure 3-5 and 6 for details on sample and sources.

**Table 3-3 The Relationship between Community Health Centers and Cause-Specific Mortality Rates for Older Adults**

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
DV Cause:	All-Cause	Heart Disease	Cerebrovascular Disease	Cancer	Infectious Disease	Diabetes	Accident
<i>A. Age-Adjusted Mortality, Older Adults (50+)</i>							
Mean at t*=-1	3,213	1461	424.4	607.4	127.2	72.3	92.6
Years -6 to -2	-2	3.6	2.6	-6.1	3.3	-0.91	-1.7
	[8.0]	[6.3]	[3.0]	[2.9]	[2.2]	[1.0]	[1.6]
Years 0 to 4	-41.1	-16.1	-10.1	-7.7	-1.9	-1.2	-0.81
	[9.6]	[5.8]	[3.2]	[3.3]	[1.7]	[1.0]	[1.3]
Years 5 to 9	-72	-26.5	-16.8	-11.2	-0.5	-1.7	-0.58
	[14.8]	[9.1]	[4.5]	[4.7]	[2.4]	[1.1]	[1.7]
Years 10 to 14	-64.1	-19.9	-12.1	-11.4	1.6	-3.2	-0.33
	[19.3]	[11.8]	[4.7]	[5.4]	[3.2]	[1.5]	[2.0]
R2	0.8	0.8	0.77	0.25	0.31	0.2	0.33
<i>B. Age-Adjusted Mortality, Ages 50-64</i>							
Mean at t*=-1	1,465	564	121	370	50	32	60
Years -6 to -2	-2.7	-1.3	1.6	-0.35	0.54	-0.86	-1.1
	[6.4]	[5.1]	[1.6]	[2.8]	[1.1]	[0.8]	[1.4]
Years 0 to 4	-14	-7.5	-3.3	-1.4	-1.8	-1.3	0.34
	[6.5]	[3.8]	[1.6]	[3.0]	[1.03]	[0.8]	[1.4]
Years 5 to 9	-32.7	-8.1	-6.3	-6.2	-2.2	-2.2	0.43
	[9.8]	[5.4]	[2.1]	[3.7]	[1.1]	[0.92]	[1.4]
Years 10 to 14	-35.5	-2.3	-5.7	-8.5	-1.8	-2.5	-0.2
	[12.9]	[6.2]	[2.3]	[4.4]	[1.7]	[1.0]	[1.4]
R2	0.58	0.71	0.71	0.18	0.17	0.11	0.25
<i>C. Age-Adjusted Mortality, Ages 65+</i>							
Mean at t*=-1	5,898	2,821	885	967	244	134	142
Years -6 to -2	0.47	12.2	4.27	-10.4	11.6	-1.9	-0.1
	[17.5]	[14.4]	[6.6]	[6.4]	[5.2]	[1.9]	[3.14]
Years 0 to 4	-80.3	-21.6	-19.2	-17.5	-3.5	-2.1	-0.92
	[20.7]	[19.2]	[6.8]	[7.0]	[4.5]	[2.6]	[2.2]
Years 5 to 9	-132.7	-47.9	-29.1	-22.3	-5.7	-3.4	-2.3
	[29.1]	[29.4]	[8.8]	[9.2]	[5.9]	[3.5]	[3.1]
Years 10 to 14	-108.6	-46	-19.4	-15.8	-1.2	-7.8	-1.1
	[36.1]	[40.7]	[10.4]	[10.4]	[6.2]	[4.9]	[4.1]
R2	0.76	0.71	0.71	0.18	0.17	0.11	0.25

Notes: The dependent variable is the age-adjusted, age-group specific mortality rate by cause for our baseline specification. See notes to figure 3-5 for details on the sample and sources.

**Table 3-4 Heterogeneity in the Relationship between Community Health Centers and Mortality Rates**

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	<i>Age-Adjusted Mortality, Older Adults (50+)</i>									
Mean at t*=-1	3,000	3,349	3,007	3,276	3,710	3,153	3,207	3,165	3,221	3,316
Years -6 to -2	-13.3	7.3	-13.0	2.7	43.5	-5.7	43.5	-5.7	43.5	-5.7
	[12.4]	[9.7]	[18.1]	[8.8]	[26.9]	[8.7]	[26.9]	[8.7]	[26.9]	[8.7]
Years 0 to 4	-48.4	-38.9	-50.4	-39.8	-21.5	-43.6	-21.5	-43.6	-21.5	-43.6
	[11.6]	[13.3]	[16.9]	[11.1]	[30.2]	[9.8]	[30.2]	[9.8]	[30.2]	[9.8]
Years 5 to 9	-54.2	-89.4	-58.0	-79.5	-57.9	-69.0	-57.9	-69.0	-57.9	-69.0
	[18.8]	[19.6]	[24.5]	[17.4]	[39.8]	[15.0]	[39.8]	[15.0]	[39.8]	[15.0]
Years 10 to 14	-38.7	-88.4	-49.6	-72.8	-34.5	-61.1	-34.5	-61.1	-34.5	-61.1
	[25.8]	[24.6]	[34.1]	[22.6]	[42.3]	[19.4]	[42.3]	[19.4]	[42.3]	[19.4]
R2	0.82		0.82		0.80		0.83		0.87	0.82
Characteristic defining stratification Group	1960 AMR		1960 MDs per 1k pop.		Race		Dropping One Region at a Time			
	Below Median	Above Median	Below Median	Above Median	Nonwhite	White	NE	MW	S	W
Mean characteristic in group	3,193	3,579	0.4	1.3	100	100				

The dependent variable is the AMR. This table reports model 2 estimates of the effects of  $\tilde{\pi}_g^k$  and  $\tilde{\tau}_y^k$  obtained by replacing equation 1's event-study dummies with  $\sum_k (\sum_{g=-2}^{-1} \tilde{\pi}_g^k D_j^k D_j^g + \sum_{y=0}^3 \tilde{\tau}_y^k D_j^k D_j^g)$ , where  $D_j^k$  is equal to 1 if the county received a CHC between 1965 and 1974 and belongs to group k. k is defined as the group of treated counties with the indicated characteristic. Columns (7)-(10) are from separate regressions, each dropping one region from the analysis at a time as indicated in the column header, and are for 2,423, 1,691, 1,418, and 2,367 counties, respectively.



**Table 3-5 The Relationship between Community Health Centers and Primary Care Use among Older Adults by Poverty Status**

	(1)	(2)	(3)	(4)	(5)
	Regular Source of Care	Any Prescription Drug Exp.	Any Out-of-Pocket Prescription Drug Exp.	Scheduled Visits + Hosp. Admits	Saw Physician Last Year
	<i>A. Household Income Less than 100 Percent of Poverty Line</i>				
Mean Dependent Variable in 1963 in Treated PSUs	0.77	0.58	0.58	7.35	0.67
CHC × 1970	0.19 [0.08]	-0.15 [0.12]	-0.22 [0.11]	3.60 [4.35]	0.04 [0.1]
Observations	1055	1055	1055	1055	1055
R2	0.19	0.20	0.24	0.22	0.15
	<i>B. Household Income between 100 and 299 Percent of the Poverty Line</i>				
Mean Dependent Variable in 1963 in Treated PSUs	0.86	0.52	0.52	8.73	0.69
CHC × 1970	-0.03 [0.05]	-0.04 [0.06]	-0.10 [0.06]	-1.35 [1.46]	0.00 [0.06]
Observations	2314	2314	2314	2314	2314
R2	0.08	0.08	0.09	0.08	0.08
	<i>C. Household Income over 300 Percent of the Poverty Line</i>				
Mean Dependent Variable in 1963 in Treated PSUs	0.89	0.56	0.55	7.56	0.71
CHC × 1970	-0.04 [0.04]	0.03 [0.07]	0.01 [0.06]	-0.12 [2.39]	0.02 [0.06]
Observations	1374	1374	1374	1374	1374
R2	0.12	0.16	0.15	0.12	0.15
H0: Coef. in Panel C = Coef. in Panel A (p-value)	0.01	0.17	0.07	0.45	0.90

Notes: See text for details on the dependent variables and equation 3 for details on specification. “1970” corresponds to the coefficient on  $D_t$ , and “CHC\*1970” corresponds to the coefficient on  $D_t D_j^*$ , the interaction of the dummy variable for 1970 and the dummy variable for receiving a CHC before 1970. Mean DV gives the mean of the dependent variable in treated PSUs in 1963. The sample includes respondents ages 50 and older. Source: Survey of Health Services Utilization and Expenditure 1963 and 1970 (Center for Health Administration Studies/National Opinion Research Center 1981).

## **Appendix F DATA APPENDIX**

### **F.1 Community Health Center Data**

Data on CHC grants are taken from the NACAP files and PHS reports and are validated using primary source materials (OEO 1966, OEO 1967, OEO 1968, DHEW 1972a, DHEW 1972b, Zwick 1972, GAO 1973, Health Services Administration 1974, Rudd et al. 1976). We first use the published information on CHCs in the primary source documents to identify grants in the NACAP and PHS data that fund CHCs. Second, we drop grant observations which are listed as “planning grants” either in the datasets or in the primary source materials. The remaining grants are used to construct the year in which a county first received a CHC program.

### **F.2 Mortality Data**

We construct mortality rates using Multiple Cause of Death (MCD) files (US DHHS 2007) for all years except 1981 and 1982, because the MCD files contain a 50% sample of deaths for some states in these years. For 1981 and 1982, we instead use the Mortality Detail files. The 1972 MCD file (and Mortality Detail file) contains a 50% sample of deaths for all states, so we multiply death counts by two in this year. All mortality rates are based on county of residence of the decedent. We do not include information on decedents who live outside the continental United States, and the publicly available mortality files exclude foreign military deaths. For 1964, records for approximately 6,000 deaths in Massachusetts are not recorded in the Vital Statistics data. This affects all counties in Massachusetts.

The age-specific mortality rate,  $ASMR_{ta}$ , in year  $t$  is the count of deaths for age group  $a$  (50–54, 55–59, ..., 75–79, 80–84, and 85+) divided by the population in age group  $a$  in year  $t$  per

100,000. The age-adjusted mortality rate in year  $t$  is a weighted sum of age-specific mortality rates,  $AMR_t = \sum_{a=1}^8 s_a ASMR_{ta}$ , where  $s_a$  is the 1960 national population share of age group  $a$  (among those 50 and older). Denominators for these rates were constructed by linearly interpolating population between the 1950 and 1960 censuses (Haines and ICPSR 2005) and the 1969 to 1988 Surveillance Epidemiology and End Results (SEER 2009) data. The age-group-specific mortality rates used in this analysis are age-adjusted by 5-year groups. “Age adjusting” (holding  $s_a$  fixed) means that changes in mortality rates reflect changes in the likelihood of dying rather than changes in population age structure. Diseases of the heart and other cardiovascular disease constitute “major cardiovascular disease” (CVD). We include general arteriosclerosis in “diseases of the heart.”

The causes of death used in table 3-5 and figure G-1 are based on the 33/34 cause recodes generated by NCHS. This recode as well as 3-digit International Cause of Death (ICD) codes used to define the causes examined in this paper are shown in table A1. There are two ICD revisions between 1959 and 1988, and they are incorporated into the mortality data in 1968 (7th Revision to 8th Revision) and 1979 (8th Revision to 9th Revision). Age-adjusted rates for these causes trend smoothly through the 1968 and 1979 ICD revisions. Note that the causes of death we consider are not comprehensive.

### **F.3 Surveys of Health Services Utilization and Expenditure 1963 and 1970**

These data are part of a series of nationally representative health surveys conducted by the National Opinion Research Center (NORC), and are made available by ICPSR. The 1963 data (7,782 respondents) are meant to be representative of the non-institutionalized population of the continental United States (no weights are provided), and the 1970 data (11,619 respondents) oversampled the urban poor, the aged and rural families (sample weights are provided).

Information on utilization and payments are verified with the providers whenever possible. The sample sizes of older adults are 1,684 in 1963 and 3,059 in 1970.

The publicly available versions do not contain geographic identifiers (see Finkelstein and McKnight 2008), but we obtained restricted identifiers for the primary sampling units (PSUs) and segments (sub-PSU-level sampling areas). Segments (defined in the data in 1970 only) generally correspond to towns, several of which make up a PSU (defined in both survey years). We use a PSU-level CHC treatment variable. In 1970, we match each segment to a county, merge the county to our CHC treatment dates, and define a PSU as treated if any portion of it in 1970 was in a county that had a CHC by 1970.

The variable numbers and questions used to construct the outcome variables in table 5 are shown below in table A1. Respondents were interviewed in 1964 and 1971 about their health care use and expenditures in calendar years 1963 and 1970. The questionnaire for ‘other’ clinic visits in 1970 specifically prompts respondents to answer if they visited a “neighborhood health center”, although this detail is not included in the computerized documentation for that question.

#### **F.4 Information on Medicare Utilization**

Figure 8 relies on newly entered county-level information from Medicare reports (US SSA 1969-1977; US HFA 1978-1980) and the Area Resource File (US DHHS 1994). The data on Medicare enrollment and use is from the following sources:

- United States Social Security Administration (US SSA), Office of Research and Statistics. (1969). Health insurance for the Aged and Disabled, 1966 and 1967. Section 1.1: Reimbursement by State and County, Washington DC.
- (1970). Health insurance for the Aged and Disabled, 1968. Section 1.1: Reimbursement by State and County, Washington DC.
- (1971). Health insurance for the Aged and Disabled, 1969. Section 1.1: Reimbursement by State and County, Washington DC.
- . (1973). Health insurance for the Aged and Disabled, 1970. Section 1.1: Reimbursement by State and County, Washington DC.

- (1973). Health insurance for the Aged and Disabled, 1971. Section 1.1: Reimbursement by State and County, Washington DC.
- (1975). Health insurance for the Aged and Disabled, 1972. Section 1.1: Reimbursement by State and County, Washington DC.
- (1977). Health insurance for the Aged and Disabled, 1974 and 1975. Section 1.1: Reimbursement by State and County, Washington DC.
- United States Health Care Financing Administration (US HFA), Office of Policy Planning, and Research. (1978). Medicare: Health Insurance for the Aged and Disabled, 1976. Section 1.1: Reimbursement by State and County, Washington DC.
- (1978). Medicare: Health Insurance for the Aged and Disabled, 1977. Section 1.1: Reimbursement by State and County, Washington DC.
- (1980). Medicare: Health Insurance for the Aged and Disabled, 1978 and 1979. Section 1.1: Reimbursement by State and County, Washington DC.

**Table F-1 SHSUE Questions Used in Table 3-5 and in Text**

Variable	1963	1970
Regular Source of Care	IS THERE A PARTICULAR MEDICAL PERSON OR CLINIC YOU (PERSON) USUALLY GO(ES) TO WHEN SICK OR OR ADVICE ABOUT HEALTH? (Q129)	SOURCE OF REGULAR MEDICAL CARE (Q 130)
Prescription Drug Expenditures	EXPENDITURES - PRESCRIBED DRUG (Q123)	TOTAL PAYMENTS FOR PRESCRIPTION DRUGS. (Best Estimate Data, Q406)
Out-of-Pocket Prescription Drug Expenditures	Total Expenditures (Q123) - Insurance Expenditures (Q108)	OUT-OF-POCKET PAYMENTS FOR PRESCRIPTION DRUGS (Q 405)
Total Visits	Sum of OB and Non-OB Doctor Office, Nurse Office, Home Visits, Hospital Visits and Hospital Admissions (Q5 - Q17)	OB and Non-OB MD Visits + OB and Non-OB Hospital Admissions (Q308, Q316, Q318 and Q319)
Saw a Physician Last Year	SAW PHYSICIAN OR NOT (Q 132)	DID (PERSON) SEE PHYSICIAN? (Q 301)
'Other' Clinic Use		TOTAL NUMBER OF VISITS TO OTHER CLINIC (E.G., PUBLIC HEALTH CLINIC) (Social Service Data, Q171)

**Table F-2 ICD Code Groups**

34 Cause Recode	1959-1967 (ICD 7)	1968-1978 (ICD 8)	1979-1988 (ICD 9)	Recode
10	1-19	10-19	10-18	Infectious Disease
20	20-29	90-97	90-97	Infectious Disease
30	30-138	Remainder of 0-136	1-9, 20-88, 98-139	Infectious Disease
50	150-159	150-159	150-159	Cancer
60	160-164	160-163	160-165	Cancer
70	170	174	174-175	Cancer
80	171-179	180-187	179-187	Cancer
90	180-181	188-189	188-189	Cancer
100	204	204-207	204-208	Cancer
110	140-148 190-203 165 205	140-149, 170-173, 190-203, 208, 209	140-149, 170-173, 190-203	Cancer
120	260	250	250	Diabetes
150	400-402 410-416	390-398	390-398	Diseases of the Heart
160	440-443	402, 404	402-404	Diseases of the Heart
170	420	410-413	410-414	Diseases of the Heart
180	421-434	420-429	415-429	Diseases of the Heart
190	444-447	400, 401, 403	401, 403	Diseases of the Heart
200	330-334	430-438	430-438	Other CVD
210	450	440	440	Diseases of the Heart
220	451-468	441-448	441-448	Other CVD
230	480-493	470-474, 480-486	480-487	Infectious Disease
330	810-835	810-825	810-825	Accidents
340	800-802 840-962	800-807, 825-949	800-807, 826-949	Accidents
370	990-999 965	980-999	980-999	Accidents

**F.5 County Codes**

We re-combine all counties that split or merge after 1959. Using Forstall (1995), we make the changes noted below (not all county changes are assigned a year, and these instances contain a “-“ below).

**Table F-3 Non-Virginia County Code Changes**

stfips	new_cofips	old_cofips	year	note
4	12	27	1983	La Paz County, AZ split off from Yuma county in 1983.
13	510	215	1971	The city of Columbus, GA became a consolidated city-county in

				1971. Previously part of Muscogee (stfips==215).
29	186	193	-	Ste. Genevieve county, MO changed codes. Always changed to 186.
32	510	25	1969	Ormsby County (25) became Carson City (510) in 1969.
35	6	61	1981	Cibola County, NM split off from Valencia County in 1981.
46	71	131	1979	Washabaugh County was annexed to Jackson County in 1979.
55	78	83, 115	1961	Menominee split off from Shawano and Oconto Counties.

**Table F-4 Virginia County Code Changes**

stfip s	new_cofip s	old_cofip s	year	note
51	83	780	1995	South Boston City rejoins Halifax County.
51	510	13	-	Alexandria City//Arlington County
51	515	19	1968	Bedford City splits from Bedford County.
51	520	191	-	Bristol City//Washington County
51	530	163	-	Buena Vista City//Rockbridge County
51	540	3	-	Charlottesville City//Albemarle County.
51	550	129	1963	Norfolk County merges (w/ South Norfolk City) to form Chesapeake City.
51	550	785	1963	South Norfolk City merges (w/ Norfolk County) to form Chesapeake City.
51	560	75	-	Clifton Forge City//Alleghany County.
51	590	143	-	Danville City//Pittsylvania County.
51	595	81	1967	Emporia City splits from Greenville County.
51	600	59	1961	Fairfax City splits from Fairfax County.
51	620	175	1961	Franklin City splits from Southampton County.
51	630	177	-	Fredericksburg City//Spotsylvania County.
51	660	165	-	Harrisonburg City//Rockingham County.
51	670	149	-	Hopewell City//Prince George County.
51	678	163	1966	Lexington City splits from Rockbridge County.
51	680	31	-	Lynchburg City//Campbell County.
51	683	153	1975	Manassas City splits from Prince William County.
51	685	153	1975	Manassas Park City splits from Prince William County.
51	690	89	-	Martinsville City//Henry County.
51	710		-	Norfolk City came from Norfolk County, which was ultimately combined into Chesapeake City. Census notes that Norfolk, Portsmouth, and Chesapeake cities (and including Norfolk and South Norfolk Counties before 1963) are often combined into one group.



51	730	53	-	Petersburg City//Dinwiddie County.
51	735	199	197 5	Poquoson City splits from York County.
51	740		-	Portsmouth City came from Norfolk County before it was Chesapeake City.
51	750	121	-	Radford City//Montgomery County.
51	770	161	-	Roanoke City//Roanoke County.
51	775	161	196 8	Salem City splits from Roanoke County.
51	780	83	196 0	South Boston City splits from Halifax County.
51	790	15	-	Staunton City//Augusta County.
51	800	123	197 4	Nansemond County merges into Suffolk City.
51	810	151	196 3	The rest of Princess Anne County merges into Virginia Beach City.
51	840	69	-	Winchester City//Frederick County.

We further make county changes necessary to use the SEER population data. These changes can be found here: <http://seer.cancer.gov/popdata/methods.html>.

**Appendix G HEALTH CENTER ACTIVITIES AND SERVICES**

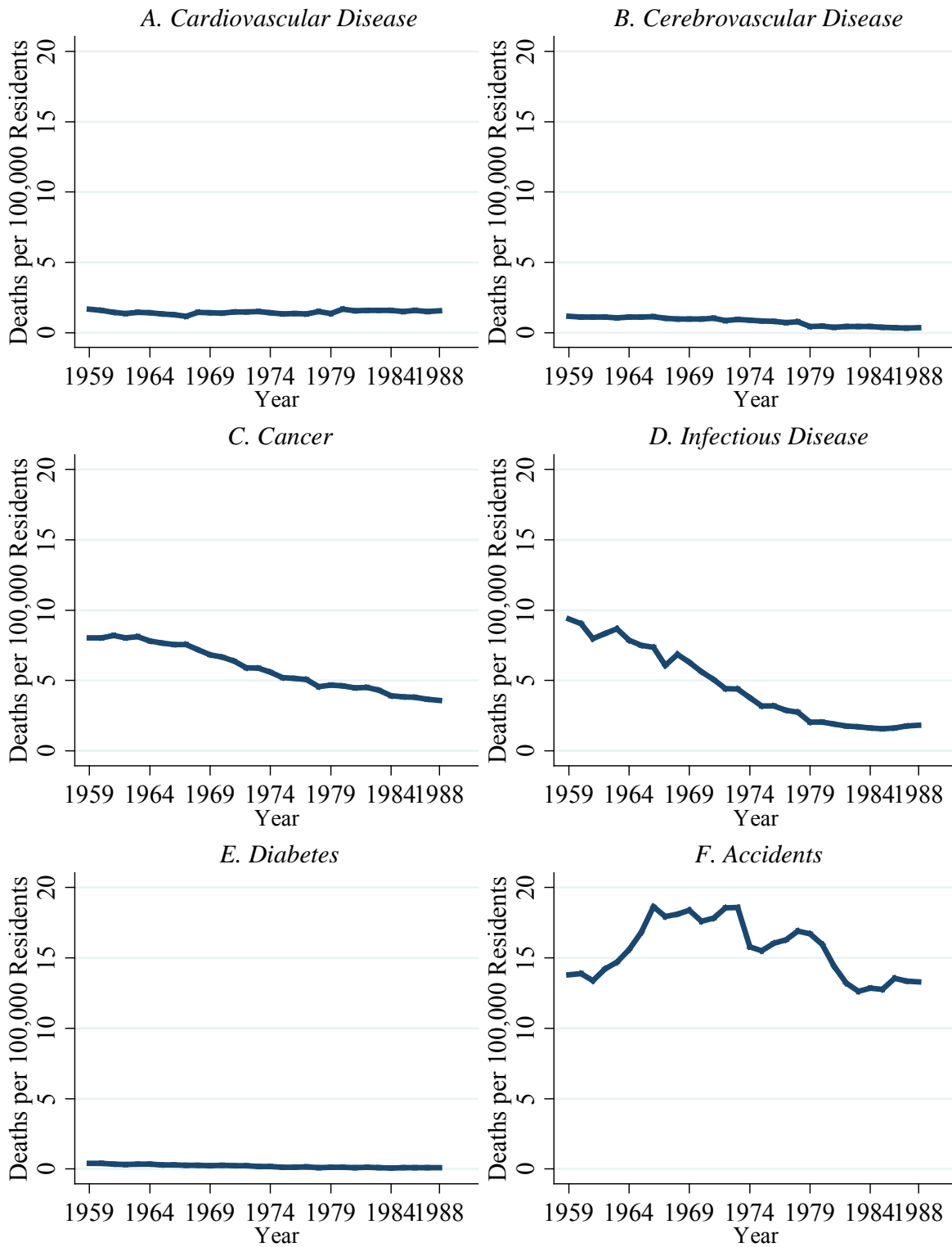
**Table G-1 Services Provided by Neighborhood Health Centers as of September 1973**

	Services per Person per Year Delivered by NHC				
	Medical Care	Prescriptions	Laboratory Tests	Dental Care	X-Rays
<b>All</b>	2.6	2.5	1.8	0.59	0.3
<b>Predominant ethnic group<sup>1</sup> served</b>					
<b>White</b>	3.2	1.9	1.5	0.63	0.26
<b>Black</b>	2.7	2.8	1.9	0.64	0.3
<b>Ratio, white to black</b>	1.19	0.68	0.79	0.98	0.87
<b>Location</b>					
<b>Urban</b>	2.6	2.5	1.9	0.59	0.32
<b>Rural</b>	2.4	2.2	1.5	0.57	0.24
<b>Ratio, urban to rural</b>	1.08	1.14	1.27	1.04	1.33
<b>Region</b>					
<b>Northeast</b>	3.1	1.8	1.7	0.68	0.25
<b>Midwest (North Central)</b>	2.3	2.4	1.9	0.44	0.28
<b>South</b>	2.8	3.3	2	0.7	0.32
<b>West</b>	2.2	2.4	1.7	0.51	0.36

Source: Davis and Schoen (1978), table 6-2. <sup>1</sup>According to Davis and Schoen, this designates the ethnic group of the "majority of registrants." Centers with no dominant group are excluded from calculations by race.

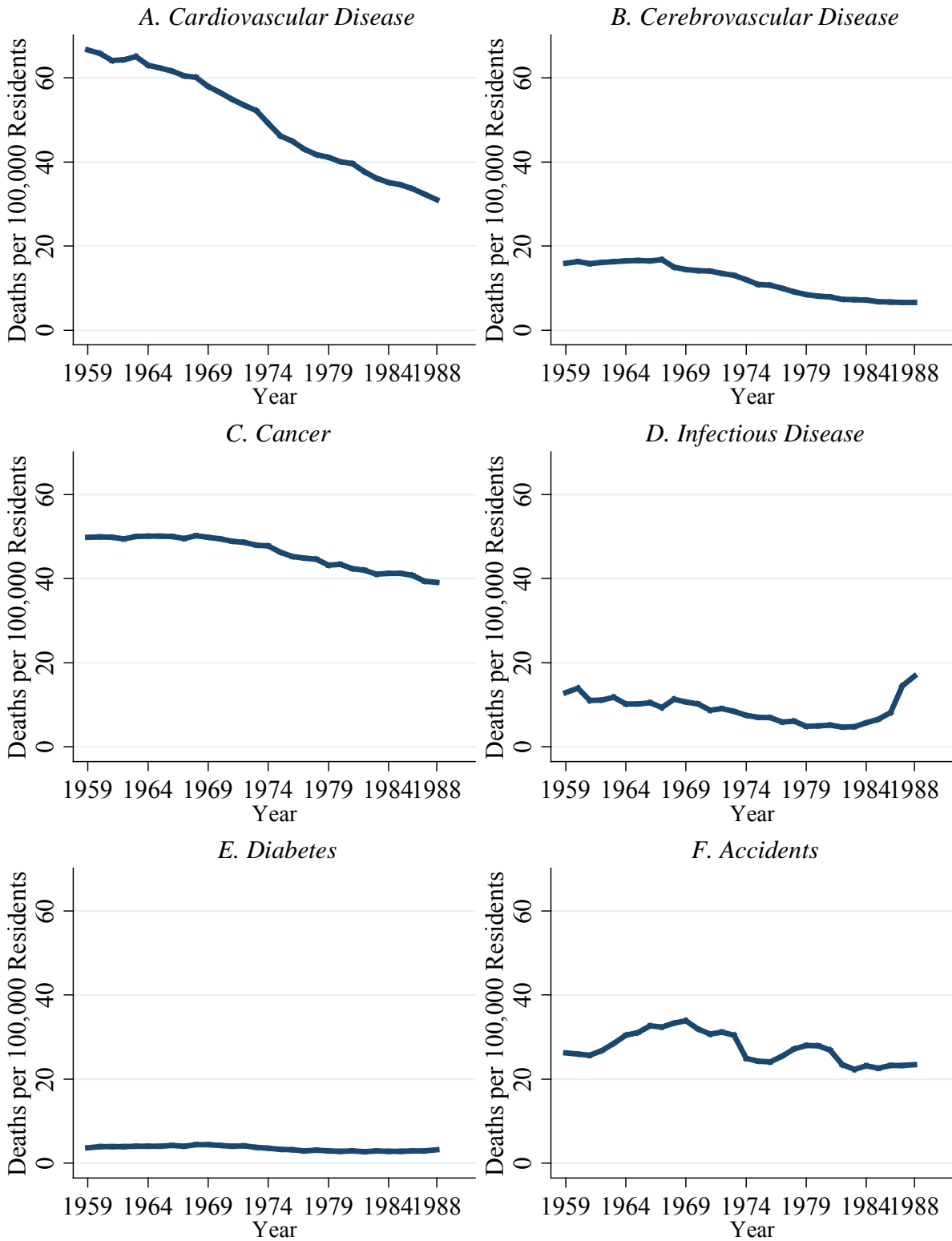
## **Appendix H MORTALITY SUMMARY STATISTICS**

**Figure H-1 Age-Adjusted Child Mortality by Cause (Ages 1-19), 1959 to 1988**



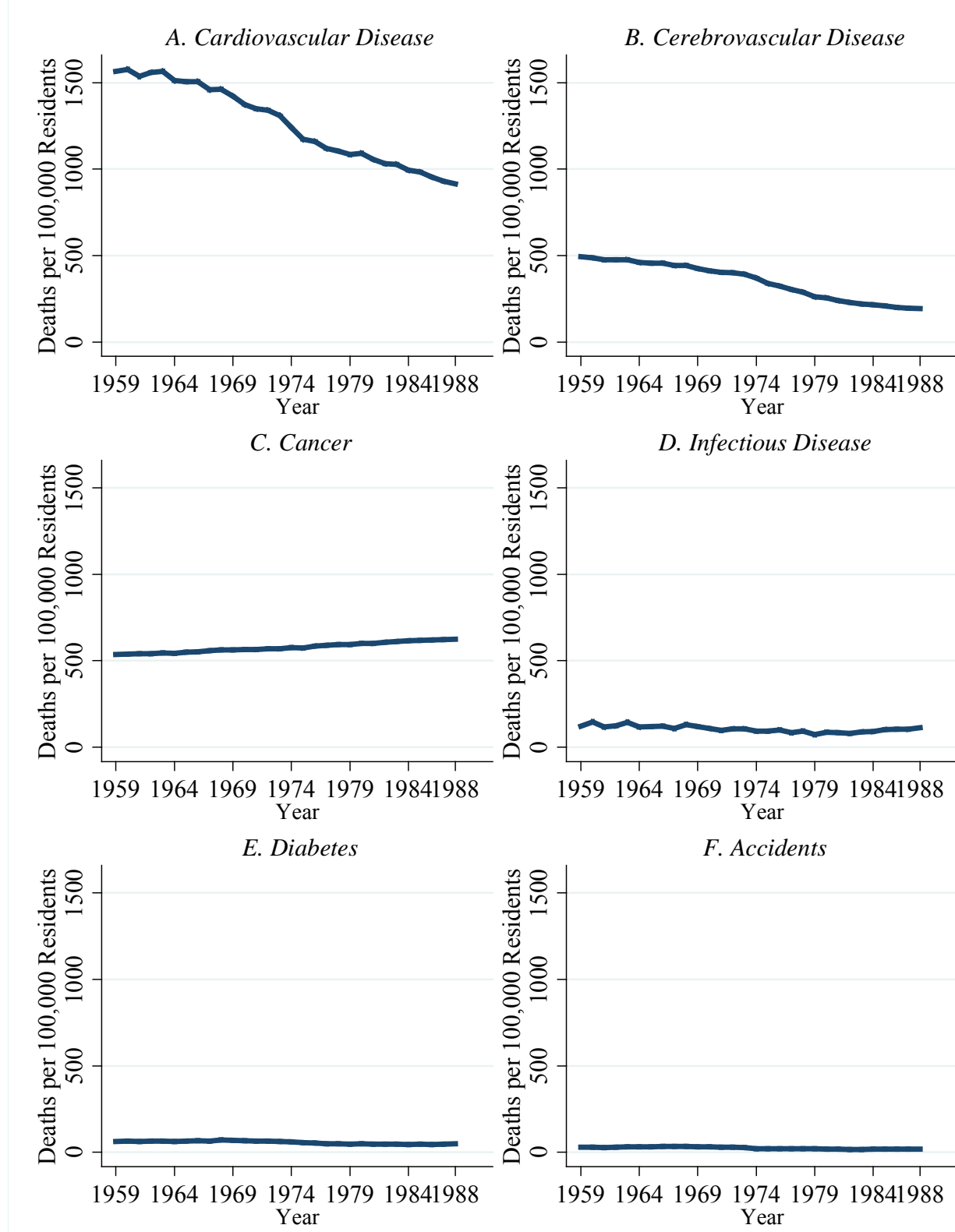
Notes: See notes to figure 3-1 and appendix A.

**Figure H-2 Age-Adjusted Adult Mortality by Cause (Ages 20-49), 1959 to 1988**



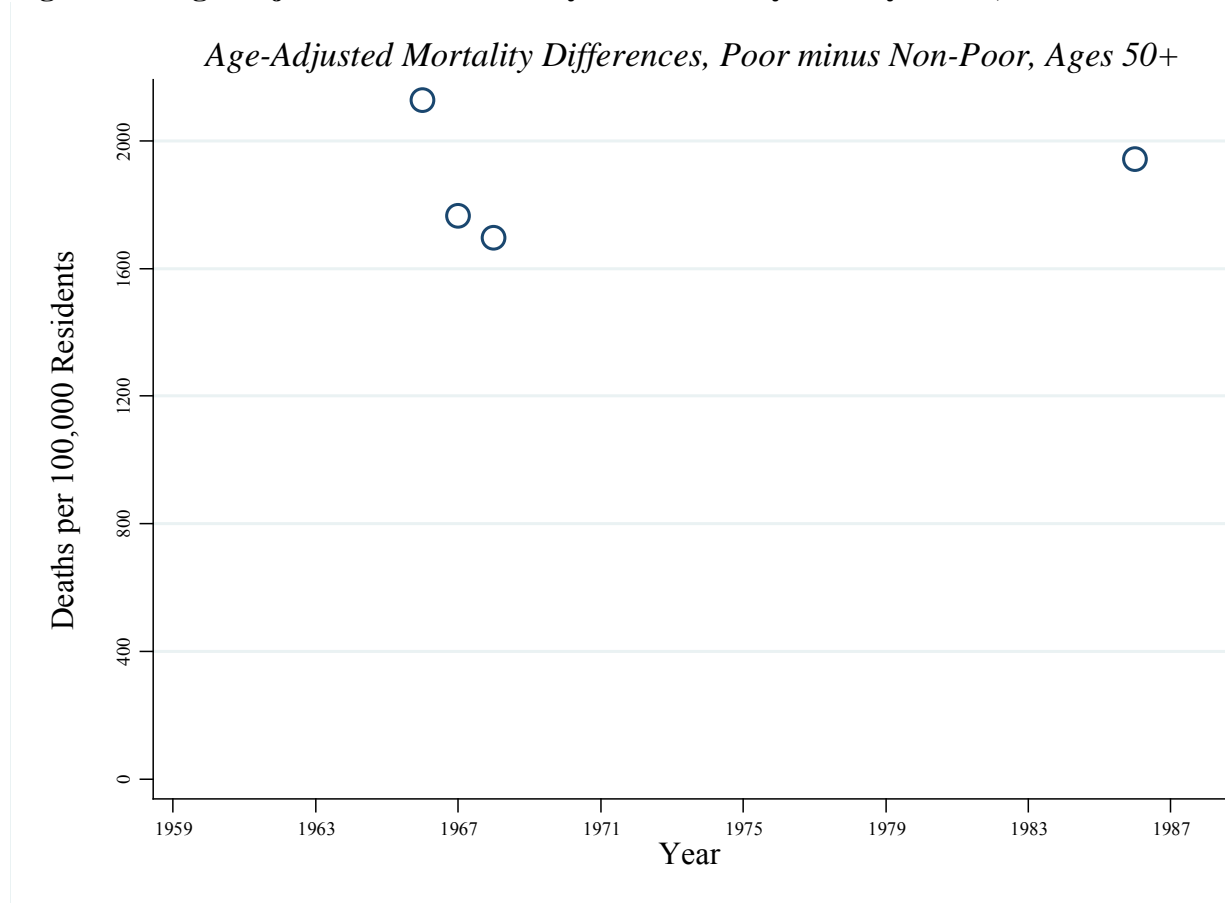
Notes: See notes to figure 3-1 and appendix A.

**Figure H-3 Age-Adjusted Older Adult Mortality by Cause (Ages 50 and Older), 1959 to 1988**



Notes: See notes to figure 3-1 and appendix A.

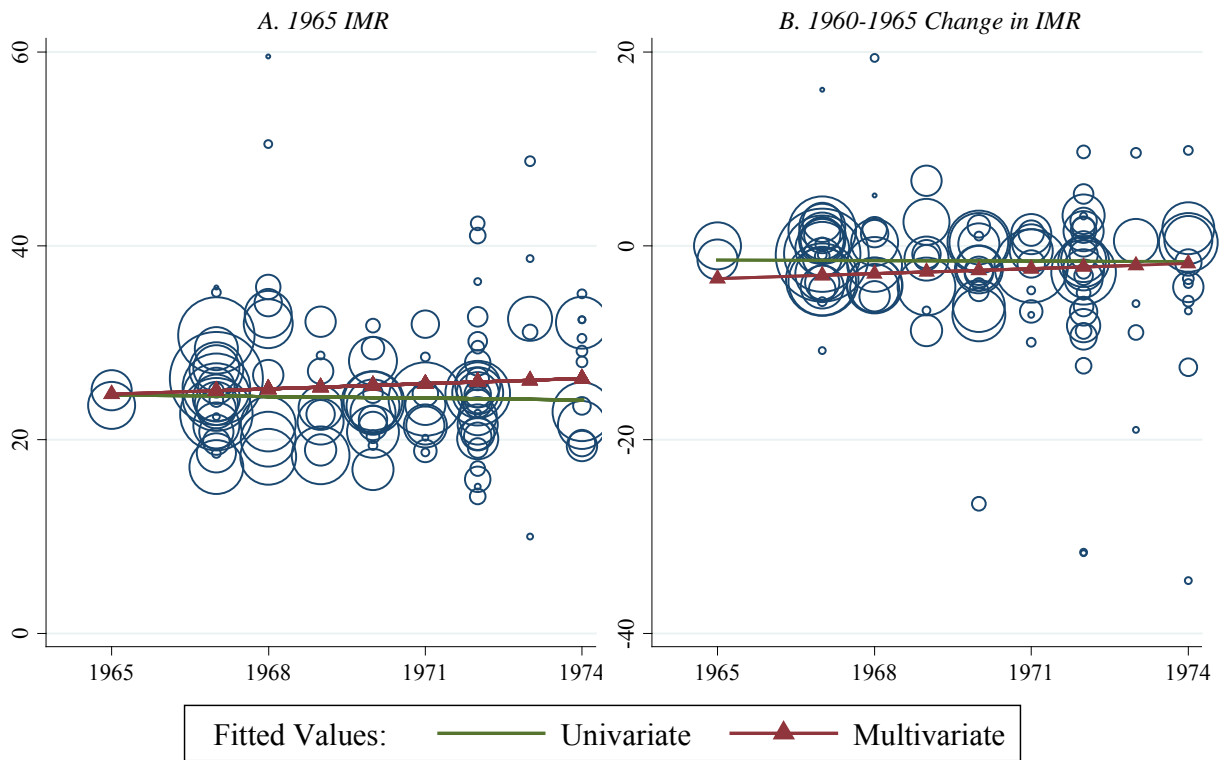
**Figure H-4 Age-Adjusted Adult Mortality Differences by Poverty Status, 1966-1986**



Notes: The figure plots the difference in age adjusted mortality rates for decedents in poor and non-poor families. The number of deaths by family poverty status are calculated from the National Mortality Followback Surveys (USDHHS, NCHS 1990 and 1986), population denominators by family poverty status are calculated from the Current Population Survey (King et al. 2010), and the weights used in the age adjustment are the national population share in 1960 (Haines and ICPSR 2005).

**Appendix I ADDITIONAL EVIDENCE ON EXOGENEITY AND EMPIRICAL SPECIFICATION**

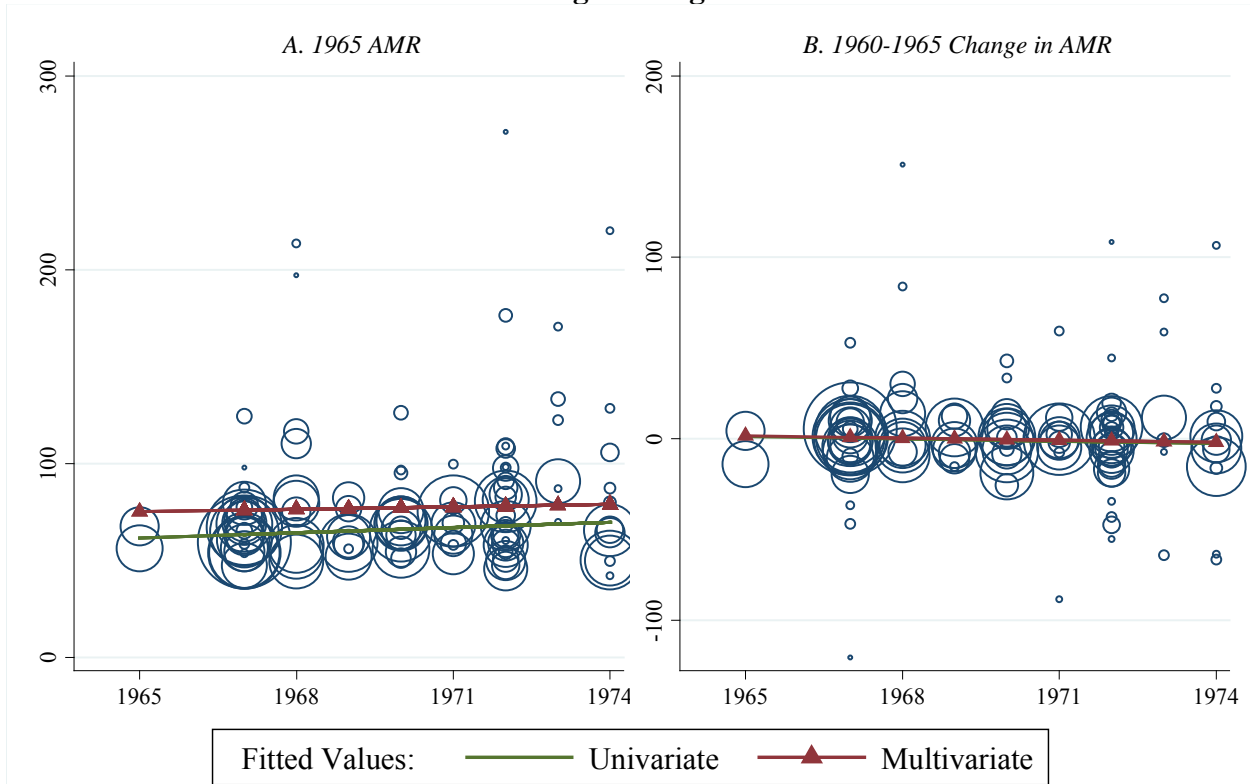
**Figure I-1 Infant Mortality Rates before the Community Health Center Program Began**



Notes: The dependent variable refers to levels of (A) or changes in (B) infant mortality rates (deaths per 1,000 live births). Univariate fitted values are from regressions of the dependent variable on the year CHCs were established for the 114 treated counties in the estimation sample. The estimated univariate slopes are  $-0.06$  (s.e. = 0.17) for panel A, and  $-0.02$  (s.e. = 0.12) for panel B. Multivariate fitted values are from regressions that also include the 1960 share of the county population that is urban, rural, between ages 0 and 4, older than 64, nonwhite, has more than 12 years of education, has less than 4 years of education, has family income less than \$3,000, has family income more than \$10,000; and the per-capita number of physicians (see table 1). The estimated multivariate slopes are  $0.17$  (s.e. = 0.13) for panel A and  $0.17$  (s.e. = 0.11) for panel B. Source: See figures 3 and 4.

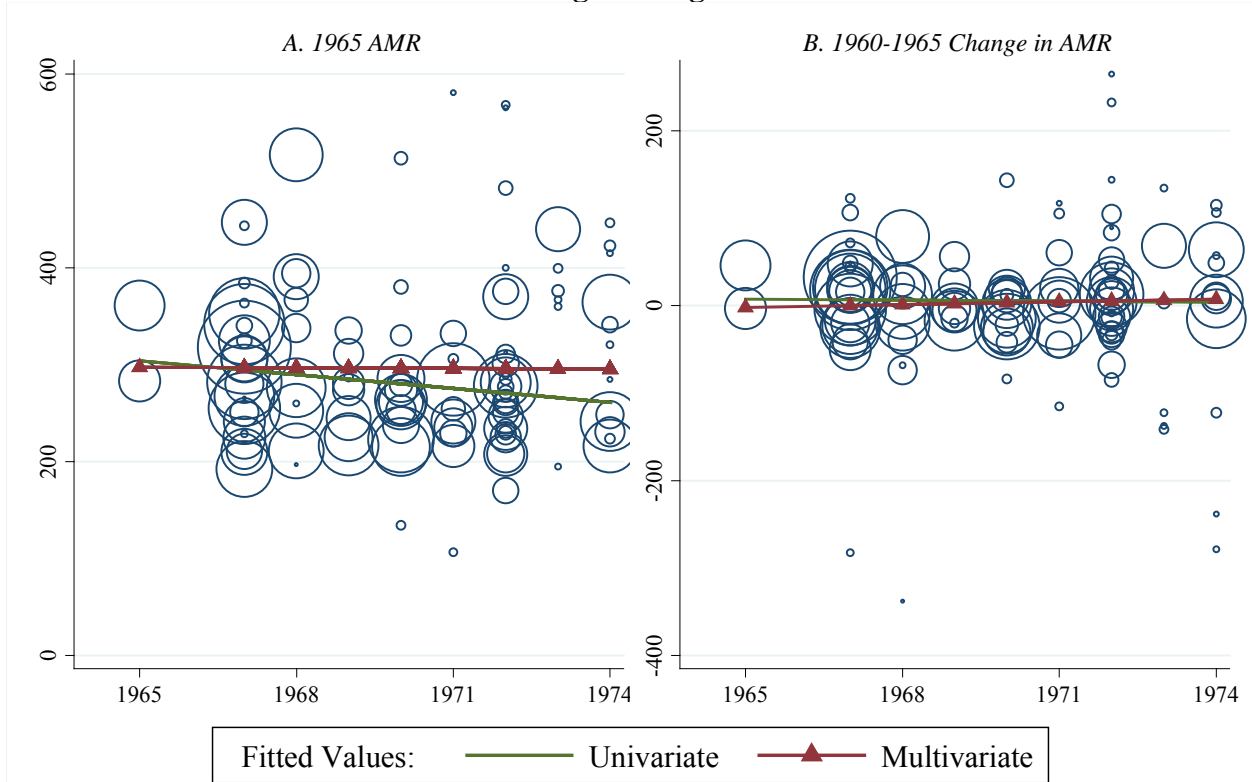


**Figure I-2 Age-Adjusted Child Mortality Rates before the Community Health Center Program Began**



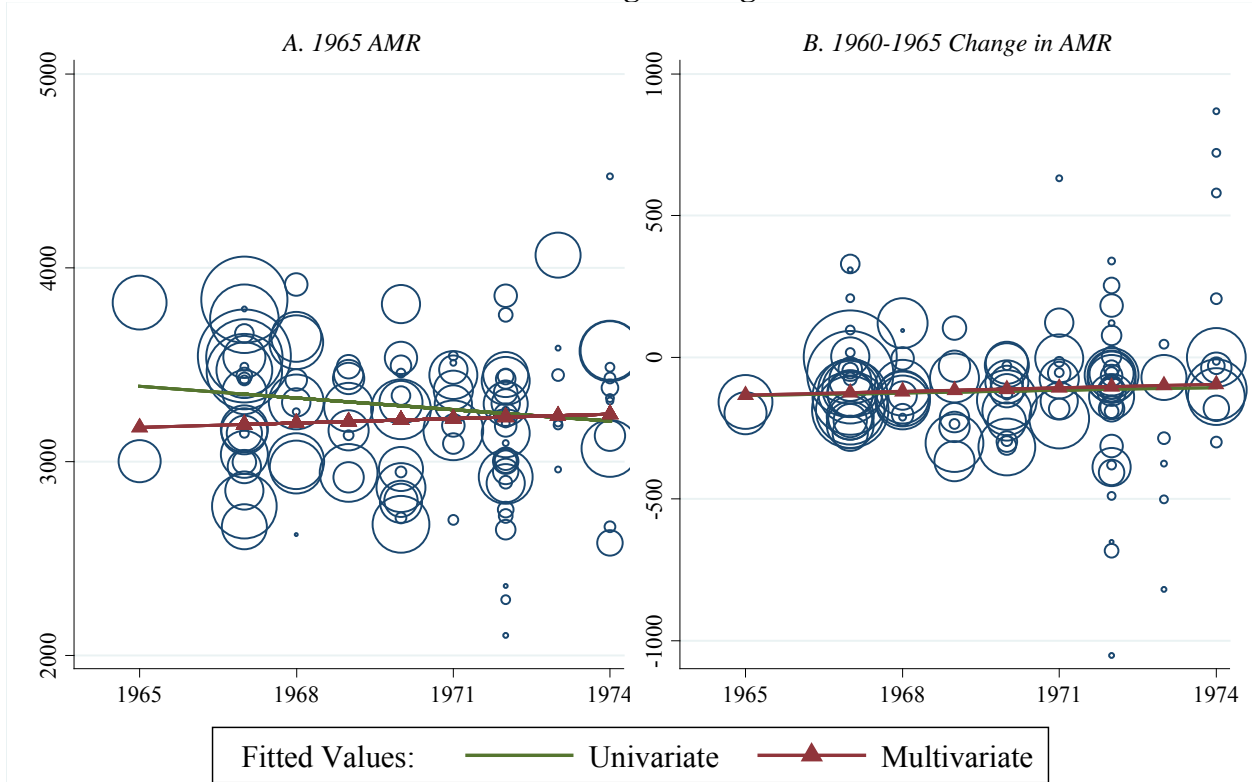
Notes: See figure I-1.A. The estimated univariate slopes are 0.8 (s.e. = 0.5) for panel A and -0.5 (s.e. = 0.4) for panel B. The estimated multivariate slopes are 0.4 (s.e. = 0.3) for panel A, and -0.4 (s.e. = 0.4) for panel B. Source: See figures 3-3 and 3-4.

**Figure I-3 Age-Adjusted Adult Mortality Rates before the Community Health Center Program Began**



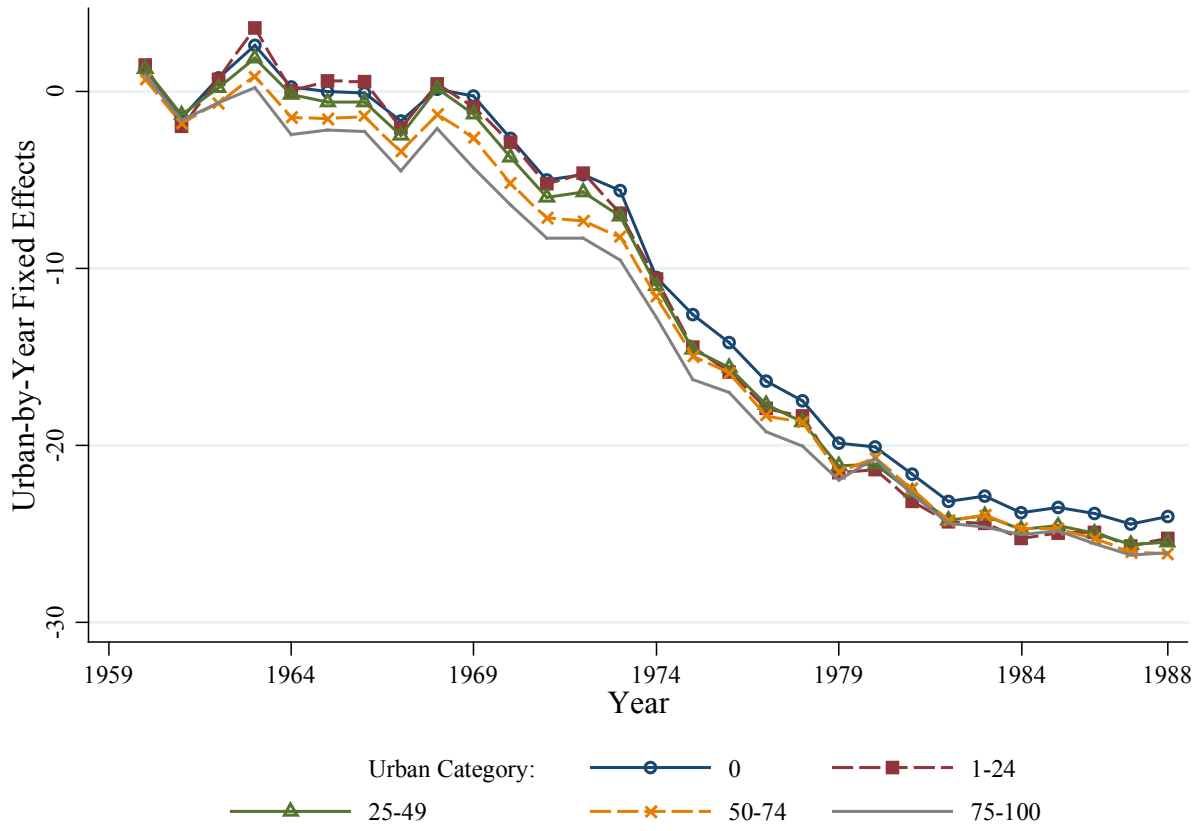
Notes: See figure I-1.A. The estimated univariate slopes are -4.8 (s.e. = 2.5) for panel A and -0.4 (s.e. = 1.2) for panel B. The estimated multivariate slopes are -0.3 (s.e. = 1.8) for panel A, and 1.0 (s.e. = 1.5) for panel B. Source: See figures 3-3 and 3-4.

**Figure I-4 Age-Adjusted Older Adult Mortality Rates before the Community Health Center Program Began**



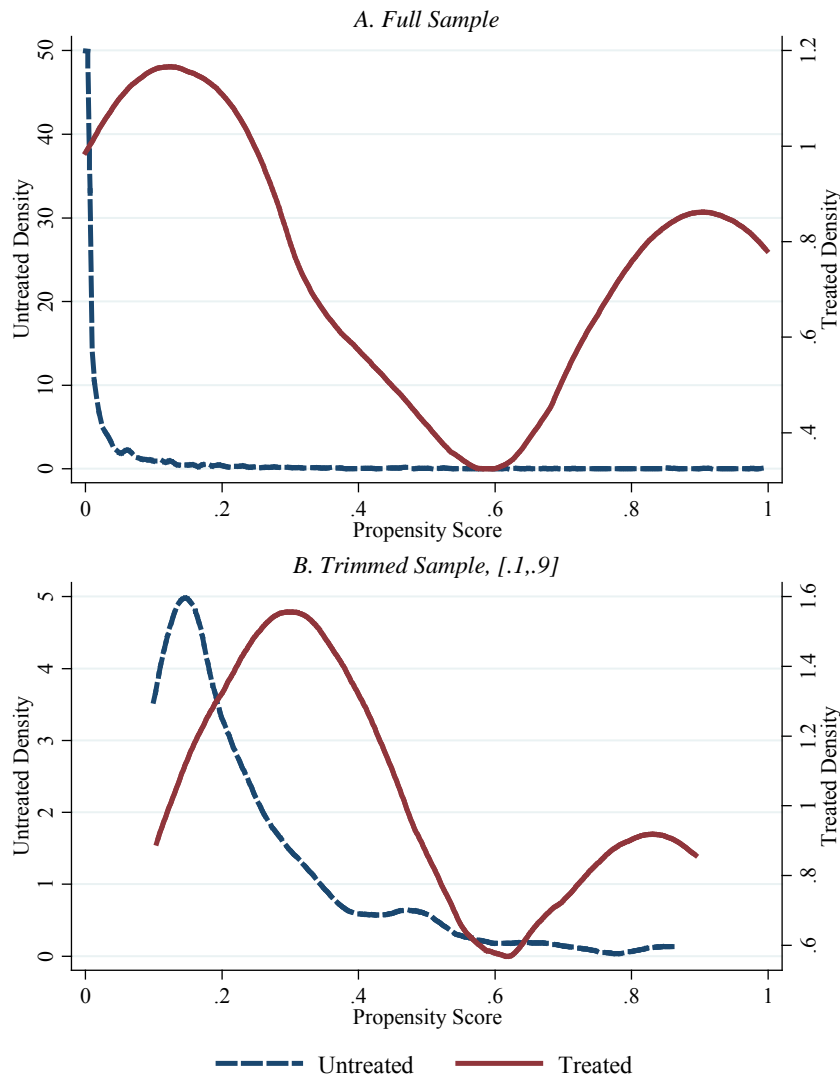
Notes: See figure I-1.A. The estimated univariate slopes are -20.9 (s.e. = 12.9) for panel A and 3.2 (s.e. = 4.3) for panel B. The estimated multivariate slopes are 7.4 (s.e. = 10.1) for panel A, and 4.2 (s.e. = 5.2) for panel B. Source: See figures 3-3 and 3-4.

**Figure I-5 Urban-by-Year Fixed Effects**



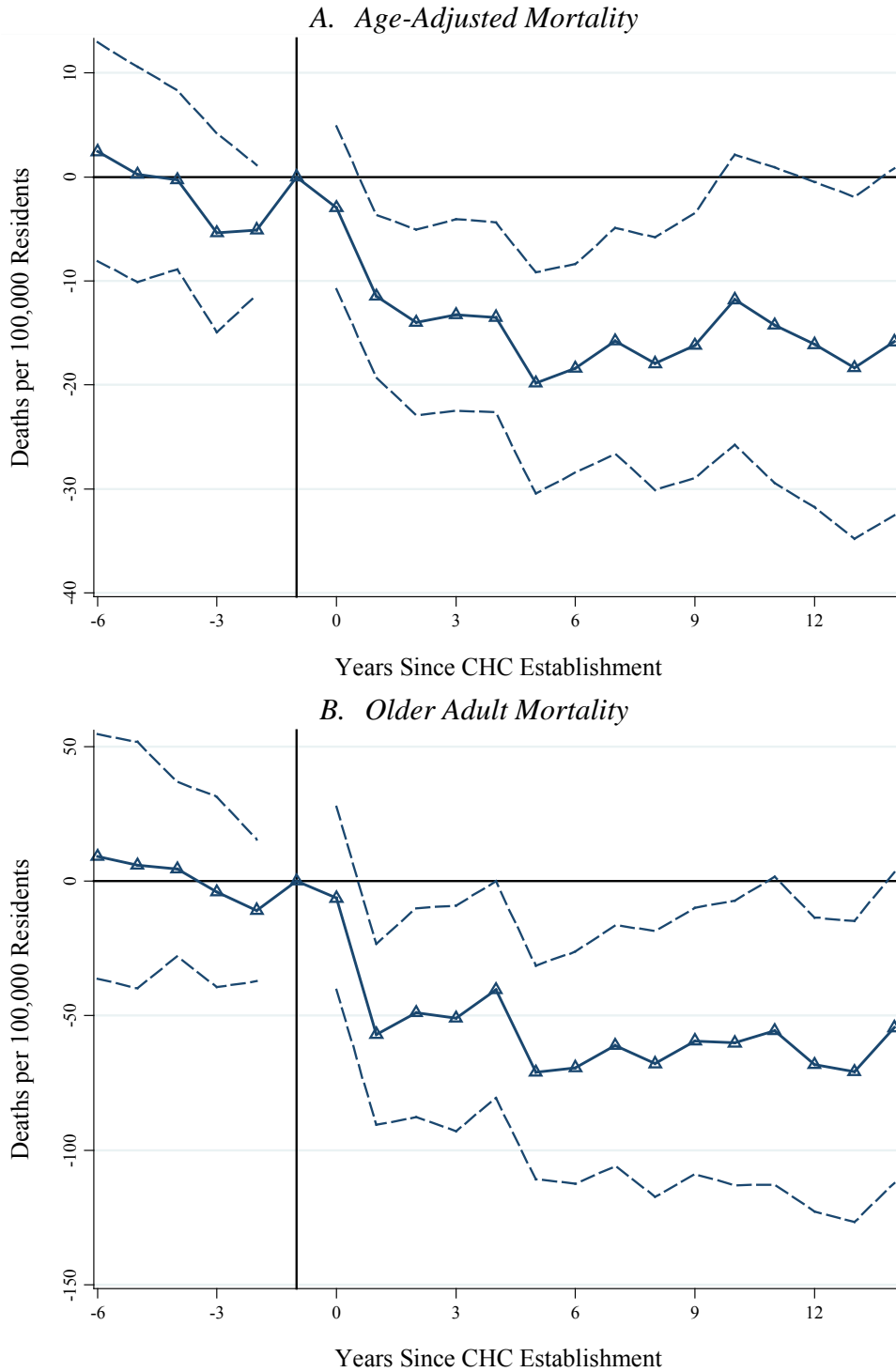
Notes: The figure plots the estimated urban-group-by-year fixed effects from the baseline specification presented in figure 3-5 and table 2.

**Figure I-6 Propensity Score Distributions**



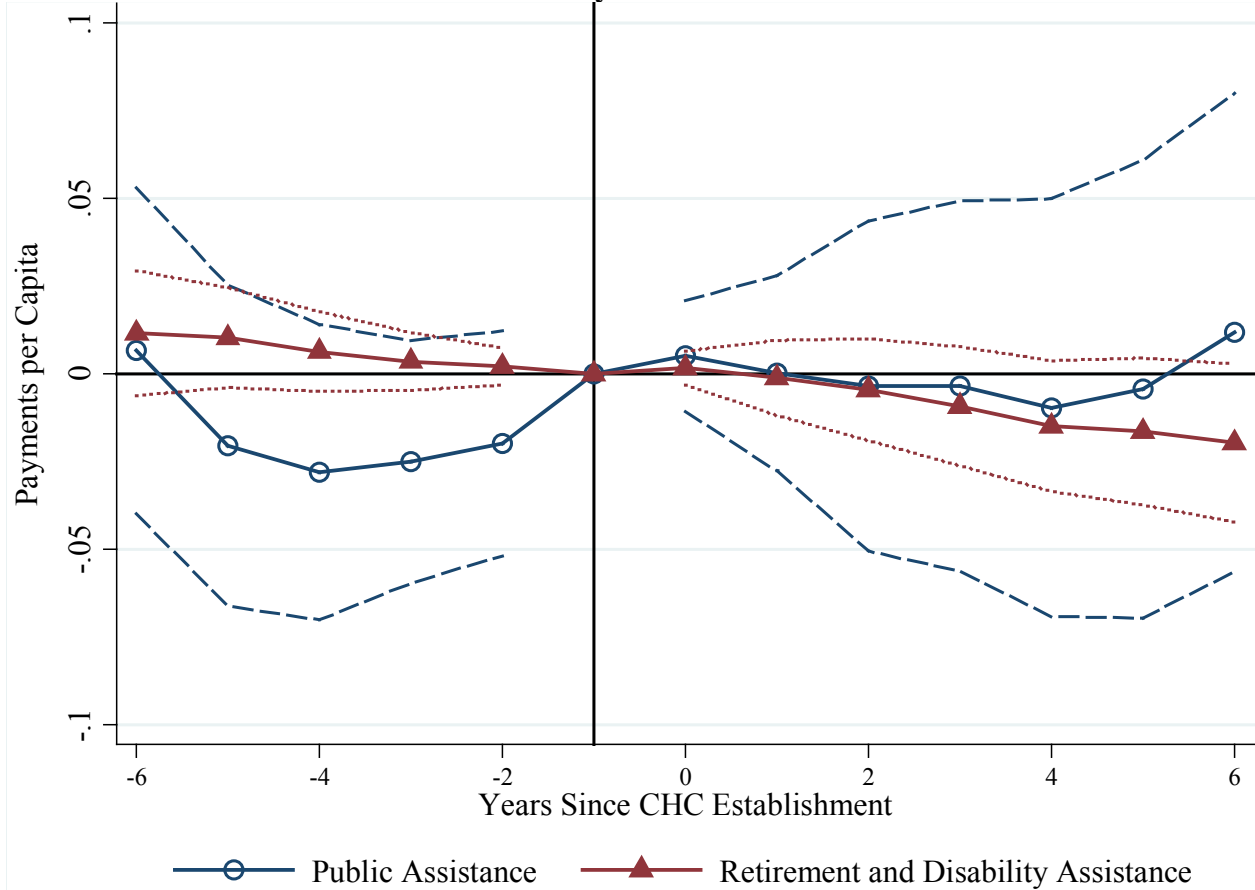
Notes: Figures show kernel density estimates using the Epanechnikov kernel for the full estimation sample (3,044 counties) and for a sample trimmed to include only propensity scores between 0.10 to 0.90 as suggested by Crump et al. (2009). The bandwidths for the untreated sample are .0026 and .0398 in the full and trimmed samples, respectively, and for the treated sample are .1388 and .0923 in the full and trimmed samples, respectively. We construct propensity scores by estimating a probit with the binary dependent variable equal to 1 if a county received a CHC from 1965 to 1974 using the following covariates: (1) Variables measured in 1960: population density and population density squared, 1950 to 1960 population growth, percent urban, percent rural, percent nonwhite, percent of population younger than 5, percent of population older than 21, percent of population older than 65, total housing units per 1000 population, civilian labor-force participation, fraction of housing units rented, median number of rooms per housing unit, percent of housing units with plumbing, share of housing units with a TV, share of housing units with a telephone, share of housing units with a car, the unemployment rate, share of the labor force that is male, fraction of the population 25 and older with less than 4 years of schooling, fraction of the population 25 and older with more than 12 or more years of schooling, number of MDs per 1,000 population. (2) Variables measured in 1959: fraction with family income below \$3,000, fraction with family income above \$10,000. (3) Variables measured in 1957: local government expenditures per 1000 population. (4) Other variables: dummy variables for the presence of a hospital in 1968 and for whether the county had a medical school in 1969, the total number of medical students in 1969, and four region dummies. This yields estimates of the propensity of treatment,  $\pi_i = P(D_i=1|X_i)$ . We then reweight untreated counties using the ratio,  $\frac{\pi_i(1-q)}{(1-\pi_i)q}$ , where  $q$  is the fraction of individuals over 50 in locations receiving CHCs, multiplied by the relevant population weight.

**Figure I-7 Changes in All-Cause Mortality Rates with the Establishment of a Community Health Center, Inverse Propensity Score Weighted Estimates, Propensity Score Trimmed Sample (0.1, 0.9)**



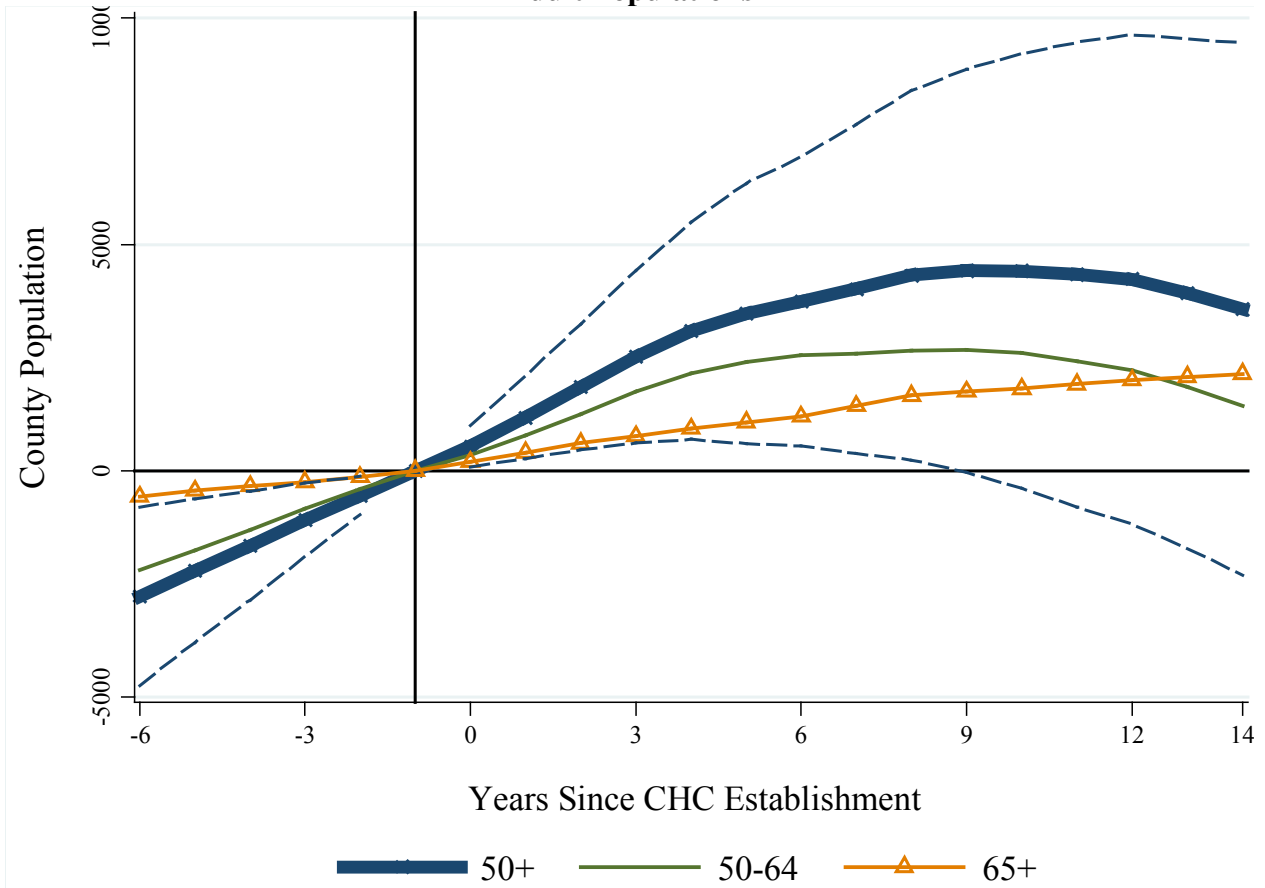
Notes: This is the event-study version of the table 2 column 4 DD specification. See table 2 and figure 3-5 notes. The sample includes only counties with estimated propensity scores between 0.1 and 0.9 (Crump et al. 2009).

**Figure I-8 Changes in Per-Capita Public Assistance Payments with the Establishment of a Community Health Center**



Notes: The figure plots weighted least-squares estimates of  $\pi$  and  $\tau$  from equation 2 for model 2. The dependent variable equals the ratio of payments for each type of cash transfer program to county population (per 1,000). The public assistance variable contains the sum of per-capita expenditures on: Aid to Families with Dependent Children, emergency assistance programs, general assistance, SSI (and its predecessors Old Age Assistance, Aid to the Permanently and Totally Disabled, and Aid to the Blind), WIC, refugee assistance, foster home care and adoption assistance, earned income tax credit, and energy assistance. The retirement and disability assistance variable contains Old Age Survivors and Disability Insurance benefits, Railroad Retirement and disability benefits, Worker's Compensation benefits, and temporary disability payments, pension benefit guaranty payments, black lung payments, and Panama Canal construction annuity payments. Dashed lines are 95-percent confidence intervals using heteroskedasticity-robust standard errors clustered at the county level. See figure 3-4 notes for details on the specification and sample. Sources: NACAP and NAFO.

**Figure I-9 The Relationship between Community Health Center Establishment and Older-Adult Populations**



Notes: The specification is the same as in figure 3-5 but the dependent variable is the county population for the indicated age group.



**Table I-1 The Determinants of When Community Health Centers Were Established**

DV: Year CHC Grant Awarded	(1)	(2)
Proportion of Residents (1960)		
in urban areas	-0.05	-0.02
	[0.03]	[0.01]
in rural or farm areas	-0.02	0.05
	[0.08]	[0.04]
under 5 years of age	0.31	0.04
	[0.31]	[0.22]
over 64 years of age	0.12	-0.15
	[0.25]	[0.14]
nonwhite	-0.01	0.00
	[0.04]	[0.02]
with 12 years of education	0.08	0.02
	[0.07]	[0.05]
with less than 4 years of education	0.13	0.08
	[0.12]	[0.08]
in households with income <\$3,000	-0.06	-0.09
	[0.11]	[0.06]
in households with income >\$10,000	0.02	-0.04
	[0.15]	[0.09]
County Medical Resources		
Total Active MDs (per 1,000 residents)	-1.13	-0.66
	[0.48]	[0.26]
Mortality Variables		
1960 AMR	0.01	0.00
	[0.01]	[0.0]
1960-1965 Change in AMR	0.01	0.00
	[0.01]	[0.0]
Weighted?	Y	N
Observations	114	114
R2	0.22	0.21
p-value from F-test:		
H0: All Coefficients (w/o urban) =0	0.01	0.02
H0: All Coefficients (w/o urban and MDs)=0	0.44	0.49

Notes: Each column reports estimates from a separate linear regression. Robust standard errors are presented in brackets. Sample: 114 counties receiving a CHC between 1965 and 1974. Sources: See table 1.

**Table I-2 The Relationship Between Community Health Center Status in 1970 and the Probability of Changing Residence or State within Five Years**

<i>A. Lived in a Different House in 1965</i>		
	(1)	(2)
CHC by 1970	0.016	0.012
	[0.021]	[0.02]
Covariates?	N	Y
Sample Restriction	All, 50+	All, 50+
Observations	117,869	117,635
R2	<0.01	0.02
<i>B. Lived in a Different State in 1965</i>		
	(1)	(3)
CHC by 1970	-0.008	-0.008
	[0.012]	[0.011]
Constant	0.062	0.070
	[0.009]	[0.015]
Covariates?	N	Y
Sample Restriction	All, 50+	All, 50+
Observations	236,373	235,883
R2	0.00	0.01

Notes: The sample includes all identified counties in the 1970 Census (Ruggles et al 2010). Panel A includes respondents who filled out state and metro form 2, and panel B includes all state and metro respondents.

**Table I-3 Neighborhood Tenure and Differences in Self-Reported Health and Knowledge of Community Health Centers by Neighborhood Tenure, NHC Survey Respondents 50 and Older**

Neighborhood Tenure Categories:	< 1 Year	[1,3) Years	[3,5) Years	> 5 Years
	(1)	(2)	(3)	(4)
Share in Each Tenure Bin	0.05	0.08	0.07	0.80
Poor/Fair Subjective Health	0.39	0.40	0.45	0.39
p-value on difference from "<1 Year"		(0.76)	(0.11)	(0.98)
Knew about CHC Before Interview	0.31	0.35	0.38	0.37
p-value on difference from "<1 Year"		(0.21)	(0.02)	(0.01)

Notes: Data from the OEO's 11 City Survey.

**Table I-4 Estimated Marginal Effects from the Propensity Score Equation**

Independent Variable	Marginal Effect (x100)	Independent Variable	Marginal Effect (x100)
Pop. Density	-1.38E-04 [4.42E-03]	Houses per 1,000 Residents	0.11 [0.04]
(Pop. Density) <sup>2</sup>	-1.31E-07 [2.90E-07]	Share of Units Rented	86.40 [40.]
Population Growth, 1950-1960	-0.09 [0.08]	Share of Units with Plumbing	0.12 [0.32]
Labor Force Participation	-86.40 [74.5]	Median Numbers of Rooms	8.97 [8.62]
Unemployment Rate	2.98 [1.08]	Share of Families with TV	0.04 [0.38]
Male Share of Labor Force	-0.85 [0.73]	Share Families with Telephone	-0.51 [0.39]
Share of Residents in 1960: Nonwhite	0.55 [0.21]	Share of Families with a Car	0.50 [0.45]
Under Age 5	-2.72 [2.79]	Had a Hospital in 1968	-6.21 [6.43]
Under Age 21	-1.01 [1.58]	MDs per 1,000 Residents	1.77 [3.62]
Over Age 64	0.54 [1.53]	Government Expenditure per 1,000 Residents	-0.07 [0.06]
In Urban Area	0.57 [0.17]	Total Medical Students, 1969	0.01 [0.01]
In Rural Area	-0.44 [0.46]	Any Medical Students, 1969	49.50 [12.3]
with Family Income < \$3k	-0.69 [0.67]	Midwest	7.29 [9.04]
with Family Income >\$10k	1.08 [0.66]	Mid-Atlantic	-7.20 [6.33]
with < 4 Years of School	0.38 [0.61]	South	4.97 [10.6]
with > 12 Years of School	-0.43 [0.52]	West	37.50 [16.8]

Observations

3025

Notes: The table contains marginal effects (mean derivatives multiplied by 100) from a probit equation used to predict propensity scores. The dependent variable is a dummy equal to one for the 114 counties in the estimation sample that received CHCs before 1975.

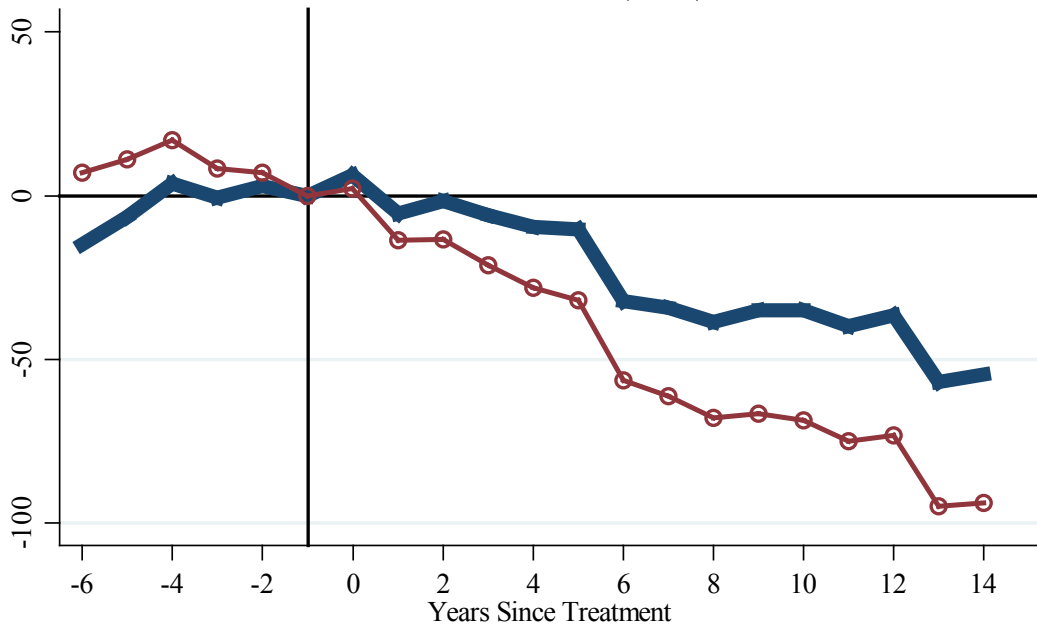
**Table I-5 Changes in All-Cause, Older-Adult Mortality Rates with the Establishment of a Community Health Center, Inverse Propensity Score Weighted Estimates**

	(1)	(2)	(3)
Years -6 to -2	4.623 [10.43]	4.984 [9.3]	2.029 [11.67]
Years 0 to 4	-33.52 [11.17]	-23.03 [14.89]	-28.99 [15.25]
Years 5 to 9	-52.12 [15.63]	-49.37 [19.17]	-65.02 [19.19]
Years 10 to 14	-61.41 [19.19]	-50.75 [21.46]	-67.75 [24.18]
Observations	91,290	91,320	9,810
Counties	3,043	3,044	327
R2	0.95	0.93	0.93
Specification and Sample	Baseline specification, P-weighted	Region-by-year effects specification, P-weighted	Region-by-year effects specification, P-weighted, Trimmed Sample

Notes: The first column reproduces column 4 of panel B of table 2. The second column replaces state-by-year effects with region-by-year effects. The third column trims the sample to those with estimated propensity scores between 0.1 and 0.9 (Crump et al. 2009) and includes region-by-year fixed effects.

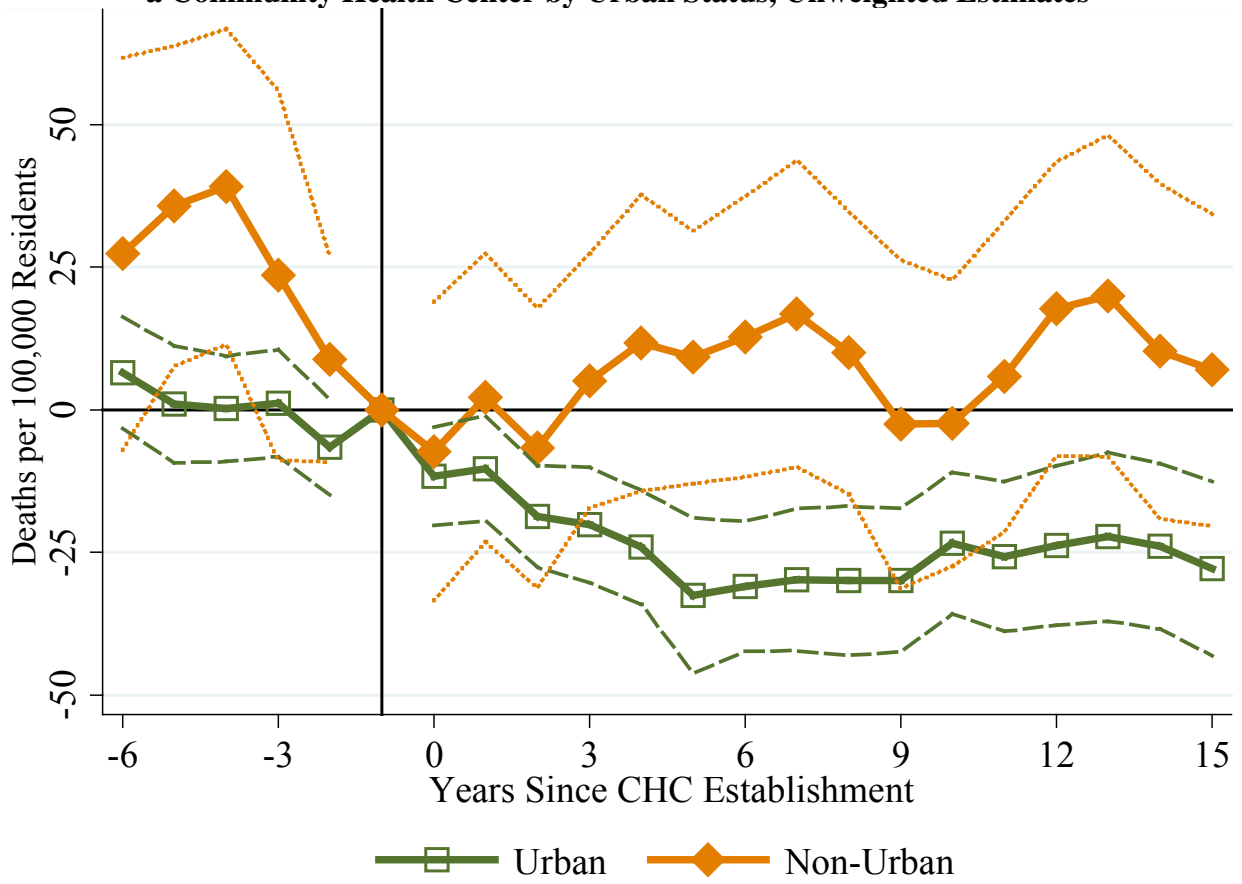
## Appendix J ROBUSTNESS CHECKS

**Figure J-1 The Relationship of All-Cause Mortality Rates and the Establishment of a Community Health Center, Treated Counties Only Adults 50 and Older**



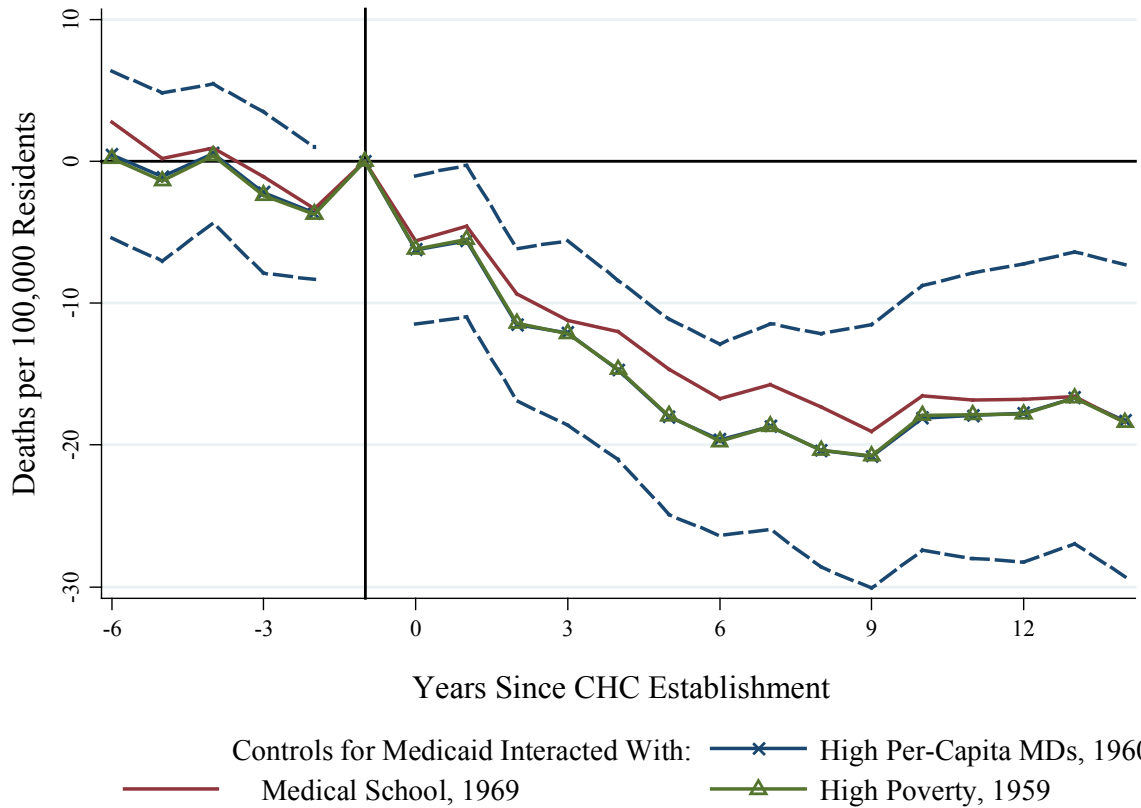
Notes: The specification in the solid line includes urban-by-year effects and region-by-year fixed effects. The series with open circles also include county-specific trends.

**Figure J-2 Changes in All-Cause, Age-Adjusted Mortality Rates with the Establishment of a Community Health Center by Urban Status, Unweighted Estimates**



Notes: See figure 3-5 and figure 3-6. The sample and specification are the same except that the estimates are not weighed by county populations.

**Figure J-3 Changes in All-Cause, Age-Adjusted Mortality Rates with Establishment of a Community Health Center with Controls for Medicaid Timing**



Notes: Here we present event-study estimates from model 2 of the effects of CHCs on AMR, which additionally control for local characteristics interacted with a binary variable for Medicaid start dates that vary across states. The idea behind this specification is that Medicaid may have had larger effects in places with different baseline characteristics (were poorer, had more physicians, or had a medical school). This specification controls for these potential effects of Medicaid by interacting dummy variables for years before and after Medicaid-implementation with county characteristics that may be correlated with stronger Medicaid effects. We estimate separate regressions that interact the Medicaid-timing dummies with an indicator for 1960 poverty rates greater than 45% (green open triangles), an indicator for whether a county had more than the median number of active MDs in 1960 (blue Xs), or an indicator for whether or not a county contained a medical school in 1969 (maroon, no markers). The estimated effects of CHCs are similar and statistically indistinguishable in all models.



## Appendix K SCALING AND MECHANISMS

**Table K-1 Calculation of Average Treatment Effect on the Treated for Older Adults**

A. Scaling by Share of Residents in Poverty	
1968 Poverty Rate (CPS)	0.22
ITT Estimate, Older Adults, Years 5-9 (average of 4 models)	-61.00
Implied ATET = ITT/Poverty	-278
B. Scaling by Estimate of CHC Users	
(1) National CHC Use (1970, SHSUE)	0.014
(2) Share of Sample Population in Treated Counties (1965, Census and SEER)	0.28
(3) Underreporting of Clinic Visits (Bound, Brown and Mathiowetz 2001)	0.39
(4) Share of MD Visits within 5 Years that took place last year (OEO Surveys)	0.76
(5) Inflation Factor = (1)/[(2)*(3)*(4)]	0.16
ITT Estimate, Older Adults, Years 5-9 (average of 4 models)	-61.00
Implied ATET = ITT/(5)	-381

**Table K-2 Potential Contribution of Anti-Hypertensive Medication to Estimated Effects for Older Adults using the Hypertension Detection and Follow-Up Program**

A. RCT Results for Anti-Hypertensive Drugs, Hypertension Detection and Follow-Up Program	
(1) ATET for 5-Year Mortality (HDFP 1979)	-2160 deaths per 100,000
(2) Share Using CHC (table F1)	0.16
(3) Share with Hypertension (NHES 197X)	0.26
(4) Implied ITT for 5-Year Mortality, (1)*(2)*(3)	-92 deaths per 100,000
B. CHC ITT Estimates	
(5) ITT Estimate, 1-Year Mortality (table 3)	-60 deaths per 100,000
(6) ITT Estimate, 5-Year Mortality = 100,000*[(1 - .0321) <sup>5</sup> - (1 - (.0321 - .0006)) <sup>5</sup> ]	-264 deaths per 100,000
(7) Share of 5-Year ITT accounted for by anti-hypertensive RCT estimates	0.35

**Table K-3 Knowledge of Community Health Centers by Age and Race, 11 City Survey**

	Nonwhite	White	p-value of difference
	(1)	(2)	(3)
Age 0	0.34	0.52	0.00
Ages 1-14	0.38	0.58	0.00
Ages 15-49	0.35	0.53	0.00
Ages 50+	0.32	0.42	0.00

Notes: The table presents means of the responses of household heads to the question “Had you heard of \_\_\_\_\_ health center, before this survey?” The question was not asked of respondents in the Eastern Montana Survey.

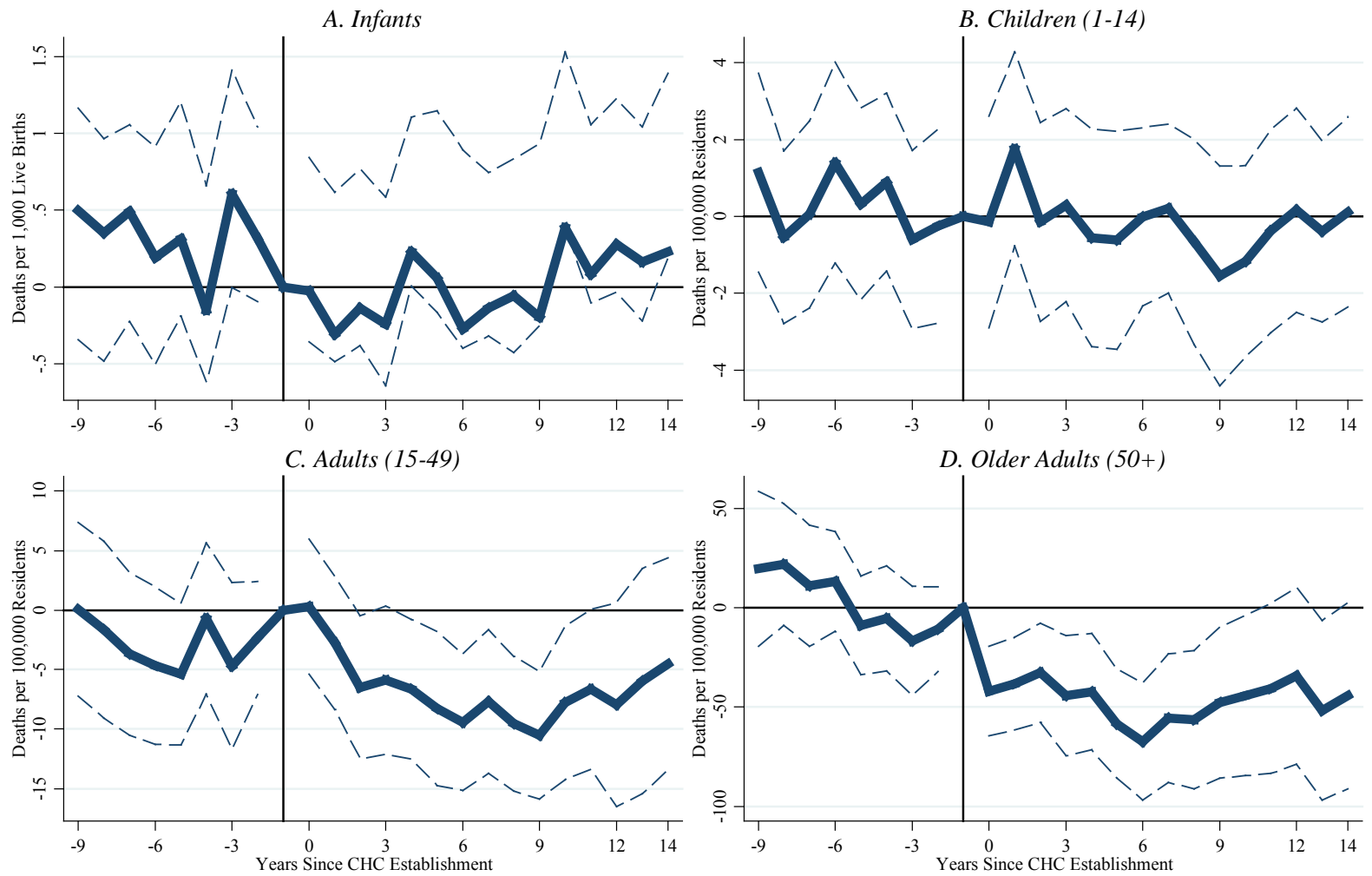
**Table K-4 Changes in Primary Care Use with the Establishment of a Community Health Center by Poverty Status, All Ages**

	(1)	(2)	(5)	(3)	(4)
	Regular Source of Care	Any Prescription Drug Exp.	Any Out-of-Pocket Prescription Drug Exp.	Scheduled Visits + Hosp. Admits	Saw Physician Last Year
A. Household Income Less than 100 Percent of Poverty Line					
Mean Dependent Variable in 1963 in Treated PSUs	0.75	0.27	0.27	4.71	0.51
CHC*1970	0.20 [0.09]	0.02 [0.04]	-0.03 [0.04]	2.58 [1.03]	0.07 [0.07]
Observations	4303	4303	4303	4303	4303
R2	0.11	0.18	0.22	0.15	0.16
B. Household Income between 100 and 299 Percent of the Poverty Line					
Mean Dependent Variable in 1963 in Treated PSUs	0.87	0.48	0.48	5.41	0.69
CHC*1970	-0.03 [0.04]	-0.03 [0.03]	-0.07 [0.03]	0.66 [0.49]	-0.02 [0.03]
Observations	10622	10622	10622	10622	10622
R2	0.08	0.11	0.12	0.06	0.09
C. Household Income over 300 Percent of the Poverty Line					
Mean Dependent Variable in 1963 in Treated PSUs	0.86	0.50	0.50	6.46	0.72
CHC*1970	-0.04 [0.03]	0.02 [0.04]	0.00 [0.03]	-0.61 [0.84]	0.02 [0.04]
Observations	4432	4432	4432	4432	4432
R2	0.13	0.11	0.10	0.06	0.08
H0: Coef. in Panel C = Coef. in Panel A (p-value)	0.01	0.95	0.55	0.02	0.56

Notes: See notes to table 5.

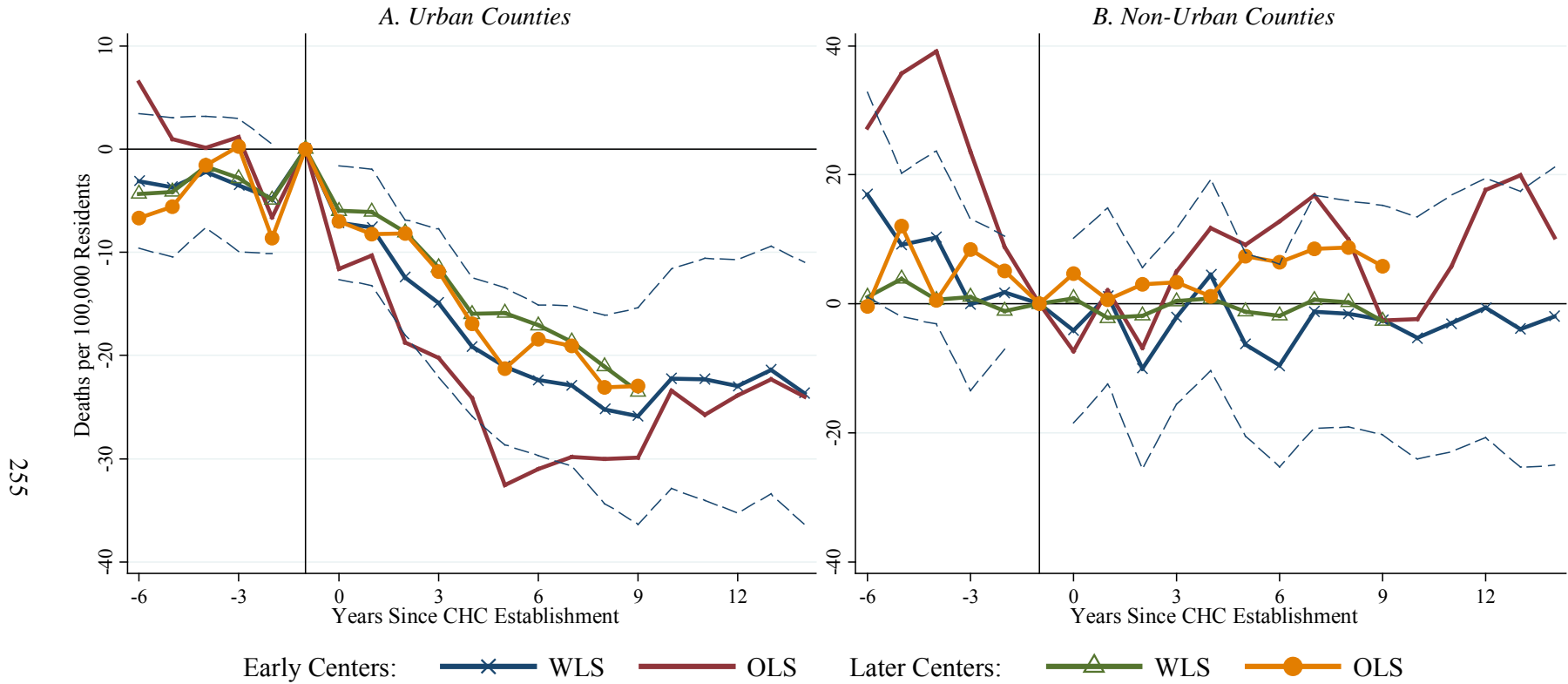
**Appendix L ADDITIONAL ESTIMATES**

**Figure L-1 Changes in All-Cause Mortality Rates with the Establishment of a Community Health Center, CHCs funded after 1967**



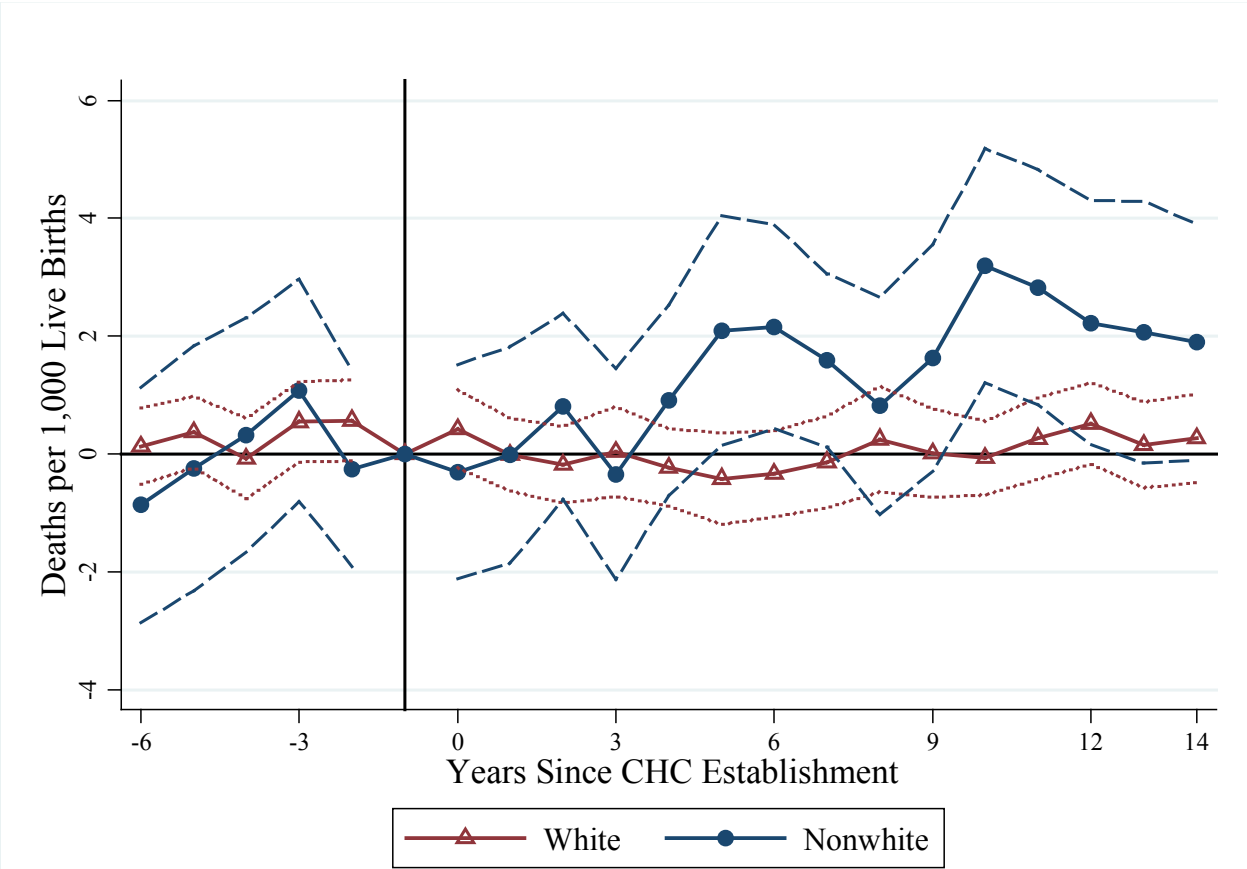
Notes: The figure presents results from model 2, but the sample of treated counties only includes the 88 counties funded after 1967 to show 9 years of pre-CHC results rather than 6.

**Figure L-2 Changes in All-Cause Mortality Rates with the Establishment of a Community Health Center, All Centers Funded between 1965 and 1980, by Urban Status**



Notes: The figure presents weighted and unweighted results from model 2. See notes to figure 3-5 and figure 3-6.

**Figure L-3 Changes in Infant Mortality Rates by Race with Establishment of a Community Health Center**



Notes: See notes to figure 3-7.

**Table L-1 Changes in All-Cause Mortality Rates with the Establishment of a Community Health Center, All Age Groups**

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	<i>A. DV: Deaths per 1,000 Infants</i>				<i>B. DV: Deaths per 100,000 Children</i>			
Mean at t*=-1	21.4				64.3			
Years -6 to -2	0.5 [0.2]	0.2 [0.2]	0.2 [0.2]	0.4 [0.3]	0.4 [2.2]	0.1 [2.2]	0.2 [2.3]	0.5 [2.1]
Years 0 to 4	0.1 [0.2]	0.1 [0.2]	0.1 [0.2]	0.3 [0.2]	1.3 [2.1]	2.0 [2.2]	1.9 [2.3]	2.6 [2.0]
Years 5 to 9	0.0 [0.3]	0.1 [0.2]	0.0 [0.3]	0.1 [0.4]	1.7 [2.8]	1.9 [2.4]	1.5 [2.9]	2.0 [2.3]
Years 10 to 14	0.4 [0.3]	0.5 [0.2]	0.4 [0.4]	0.4 [0.3]	1.2 [2.7]	1.8 [2.3]	1.4 [4.0]	2.3 [2.4]
R2	0.59	0.62	0.65	0.91	0.15	0.17	0.21	0.64
	<i>C. DV: Deaths per 100,000 Adults</i>				<i>D. DV: Deaths per 100,000 Older Adults</i>			
Mean at t*=-1	290.5				3212.8			
Years -6 to -2	-4.9 [2.6]	-3.2 [1.9]	-4.0 [2.1]	-3.0 [1.9]	11.1 [10.4]	-0.4 [8.1]	-1.7 [8.1]	5.6 [11.1]
Years 0 to 4	4.2 [2.9]	0.5 [2.2]	1.6 [2.5]	0.5 [2.9]	-29.7 [13.7]	-41.1 [9.6]	-38.1 [8.9]	-30.8 [11.4]
Years 5 to 9	6.1 [5.1]	-3.3 [2.6]	-0.1 [3.7]	0.7 [3.8]	-58.6 [17.3]	-70.6 [14.8]	-60.8 [11.6]	-49.6 [15.7]
Years 10 to 14	4.1 [5.9]	-7.4 [3.3]	-2.1 [4.9]	-3.3 [3.9]	-49.0 [21.1]	-62.7 [19.2]	-45.0 [15.4]	-61.6 [18.5]
R2	0.37	0.43	0.47	0.82	0.77	0.80	0.84	0.96
Covariates	C, U-Y	C, U-Y, S-Y, R, D-Year	C, U-Y, S-Y, R, C-Year	C, U-Y, S-Y, R, P-weights	C, U-Y	C, U-Y, S-Y, R, D-Year	C, U-Y, S-Y, R, C-Year	C, U-Y, S-Y, R, P-weights

See notes to table 2.



**Table L-2 Changes in Cause-Specific Mortality Rates with the Establishment of a  
Community Health Center, Children and Adults**

DV Cause:	(1) Cardiovascular Disease	(2) Cerebrovascular Disease	(3) Cancer	(4) Infectious Disease	(5) Diabetes	(6) Accident
<i>A. DV: Deaths per 100,000 Children</i>						
Mean at t*=-1	1.4	1.1	6.9	5.4	0.2	28.5
Years -6 to -2	0.03 [0.11]	-0.06 [0.08]	0.57 [0.24]	-0.07 [0.28]	-0.01 [0.04]	-0.70 [0.52]
Years 0 to 4	-0.03 [0.11]	-0.09 [0.09]	0.65 [0.23]	-0.03 [0.27]	0.02 [0.04]	-0.48 [0.53]
Years 5 to 9	-0.05 [0.12]	-0.14 [0.09]	0.28 [0.24]	0.20 [0.29]	0.04 [0.04]	-0.66 [0.59]
Years 10 to 14	-0.08 [0.14]	-0.16 [0.09]	0.34 [0.25]	0.33 [0.28]	0.02 [0.04]	-1.10 [0.6]
R2	0.02	0.04	0.10	0.22	0.02	0.10
<i>B. DV: Deaths per 100,000 Adults</i>						
Mean at t*=-1	60.6	17.3	52.3	12.3	4.5	47.8
Years -6 to -2	0.37 [0.89]	0.31 [0.42]	0.85 [0.64]	0.37 [0.32]	-0.05 [0.21]	-0.70 [0.74]
Years 0 to 4	-0.65 [0.9]	-0.33 [0.4]	-0.20 [0.62]	-0.64 [0.35]	0.03 [0.21]	1.13 [0.72]
Years 5 to 9	-1.26 [1.03]	-0.87 [0.41]	-0.54 [0.65]	-1.07 [0.56]	-0.10 [0.2]	0.51 [0.87]
Years 10 to 14	-0.93 [1.08]	-1.04 [0.46]	0.11 [0.75]	-0.73 [1.32]	-0.24 [0.22]	0.21 [0.83]
R2	0.34	0.24	0.07	0.25	0.04	0.15

See notes to table 3.

**Table L-3 Effect of Cumulative Community Health Center Grant Funds on Age-Adjusted Mortality**

	(1)	(2)	(3)	(4)
	<i>DV: Age-Adjusted Mortality, All Ages</i>			
Mean at t*=-1	929.3			
Cumulative CHC Grant Amounts (Millions of 2010 Dollars)	-0.17	-0.36	-0.40	-0.36
	[0.06]	[0.07]	[0.09]	[0.07]
R2	0.82	0.85	0.96	0.87
Covariates	C, U-Y	C, U-Y, S- Y, R, D·Year	C, U-Y, S- Y, R, C·Year	C, U-Y, S- Y, R, P- weights

Notes: The table presents the estimated coefficient on the running sum of CHC grant dollars. For untreated counties this is zero. For treated counties, this is zero before CHC establishment and weakly increases in each year thereafter. The sums stop (and are constant) in 1974.

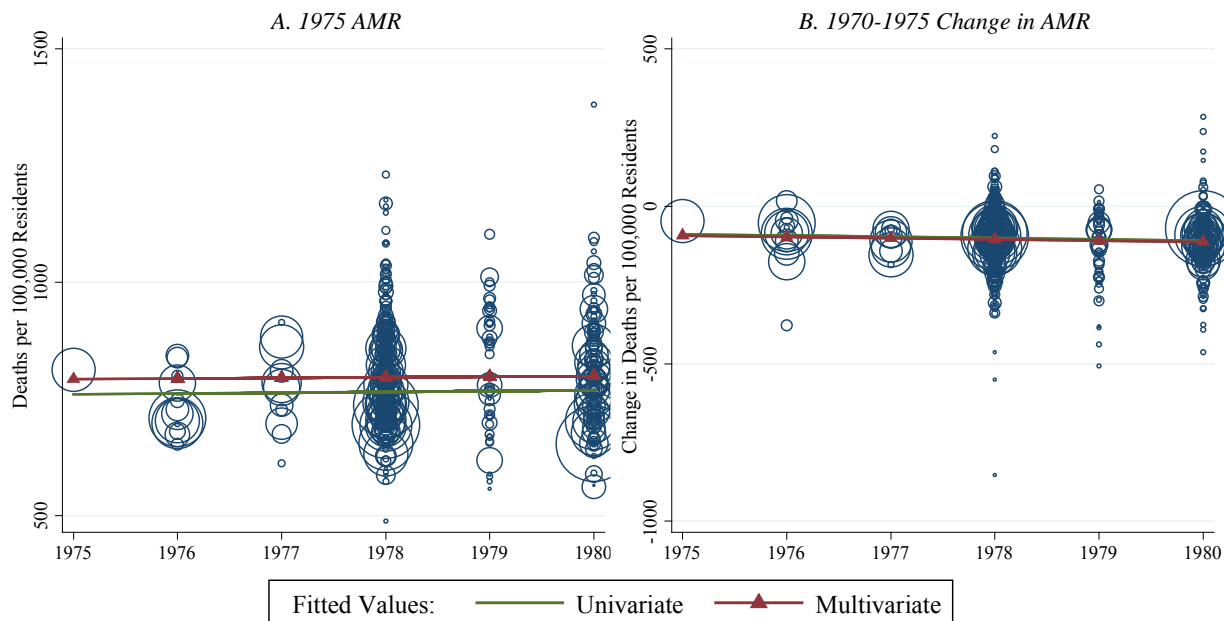
**Table L-4 Changes in Age-Specific Mortality Rates with the Establishment of a Community Health Center**

	(1)	(2)	(3)	(4)
<i>DV: Age-Adjusted Mortality Rates, Ages 50-64</i>				
Mean DV at t=-1:	1,482.0			
Years -6 to -2	-0.4 [7.0]	-2.9 [6.4]	-6.5 [7.1]	-3.2 [7.0]
Years 0 to 4	-3.3 [8.1]	-13.7 [6.5]	-8.3 [6.0]	-10.6 [7.5]
Years 5 to 9	-18.8 [10.0]	-31.5 [9.8]	-15.2 [7.7]	-15.2 [10.1]
Years 10 to 14	-18.6 [12.9]	-34.7 [12.9]	-8.6 [9.4]	-17.3 [11.7]
R2	0.56	0.60	0.65	0.89
<i>DV: Age-Adjusted Mortality Rates, Ages 65-79</i>				
Mean DV at t=-1:	4,627.3			
Years -6 to -2	17.2 [21.0]	-7.4 [17.0]	-4.5 [18.8]	5.9 [19.9]
Years 0 to 4	-61.7 [25.2]	-65.4 [18.6]	-67.0 [17.5]	-65.4 [20.2]
Years 5 to 9	-108.3 [31.0]	-108.8 [26.2]	-110.9 [21.3]	-92.3 [31.3]
Years 10 to 14	-87.5 [38.1]	-86.5 [32.9]	-88.5 [26.8]	-84.4 [36.5]
R2	0.68	0.72	0.75	0.93
<i>DV: Age-Adjusted Mortality Rates, Ages 80+</i>				
Mean DV at t=-1:	13,700.0			
Years -6 to -2	95.7 [70.7]	39.7 [64.0]	43.6 [65.0]	13.5 [79.7]
Years 0 to 4	-107.6 [78.0]	-153.0 [65.4]	-158.7 [68.9]	-82.3 [71.8]
Years 5 to 9	-185.2 [98.5]	-234.1 [81.6]	-246.2 [88.7]	-114.7 [84.0]
Years 10 to 14	-144.1 [116.4]	-200.5 [95.3]	-191.1 [116.5]	-170.0 [96.1]
R2	0.57	0.61	0.66	0.89
Covariates	C, U-Y	C, U-Y, S-Y, R, D·Year	C, U-Y, S-Y, R, C·Year	C, U-Y, S-Y, R, P-weights

Notes: See table 2 notes.

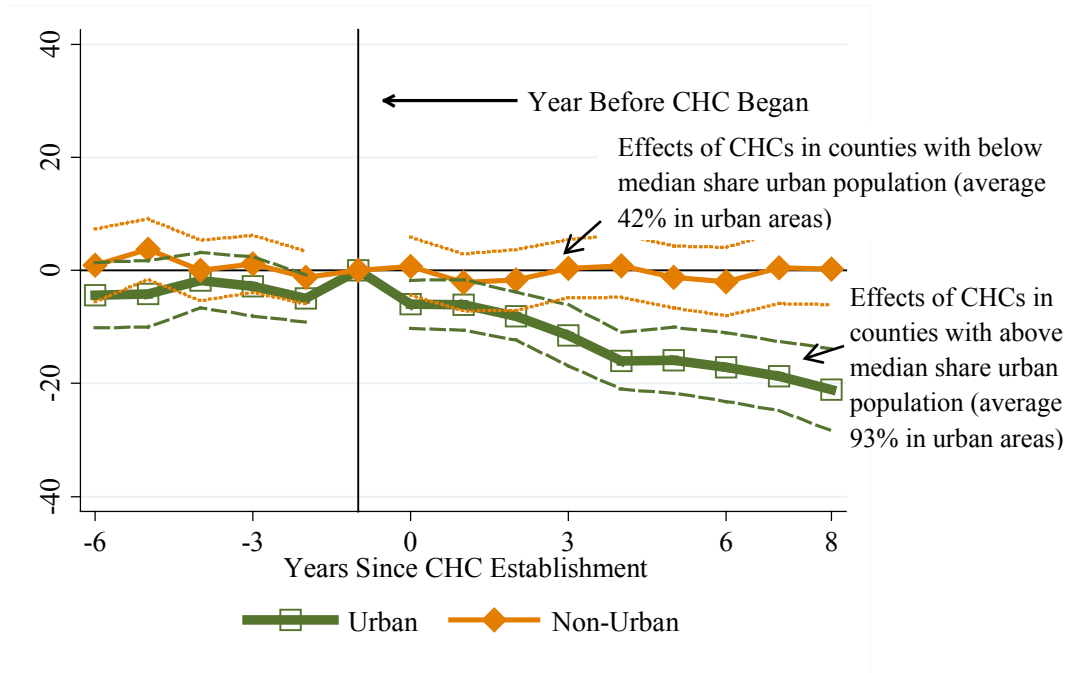
**Appendix M ESTIMATES AND FIGURES INCLUDING CHCs FIRST FUNDED FROM 1975-1980**

**Figure M-1 Age-Adjusted Mortality Rates before the Community Health Center Program Began, Centers Funded in 1975-1980**



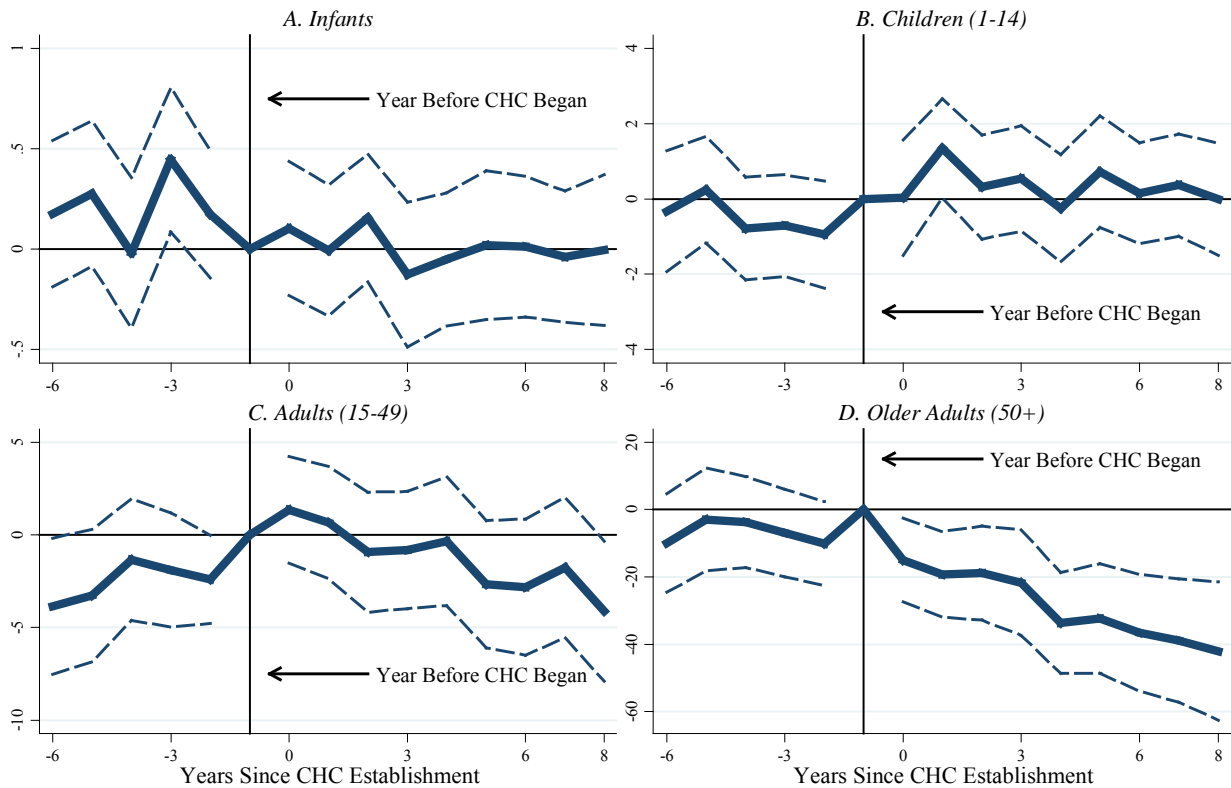
Notes: The dependent variable refers to (levels of or changes in) age-adjusted mortality rates (deaths per 100,000 residents). Univariate fitted values are from regressions of the dependent variable on the year CHCs were established for the 499 counties that first received CHCs between 1975 and 1980. The estimated univariate slopes are 1.7 (s.e. = 3.1) for panel A, and 1.3 (s.e. = 2.6) for panel B. Multivariate fitted values are from regressions that also include the 1960 share of the county population that is urban, rural, between ages 0 and 4, older than 64, nonwhite, has more than 12 years of education, has less than 4 years of education, has family income less than \$3,000, has family income more than \$10,000; and the per-capita number of physicians (see table 1). The estimated multivariate slopes are -4.2 (s.e. = 1.8) for panel A and -4.1 (s.e. = 2.3) for panel B. Source: See figures 1 and 2.

**Figure M-2 Heterogeneity in the Relationship between Community Health Centers and Mortality Rates by Population Density, All CHCs 1965-1980**



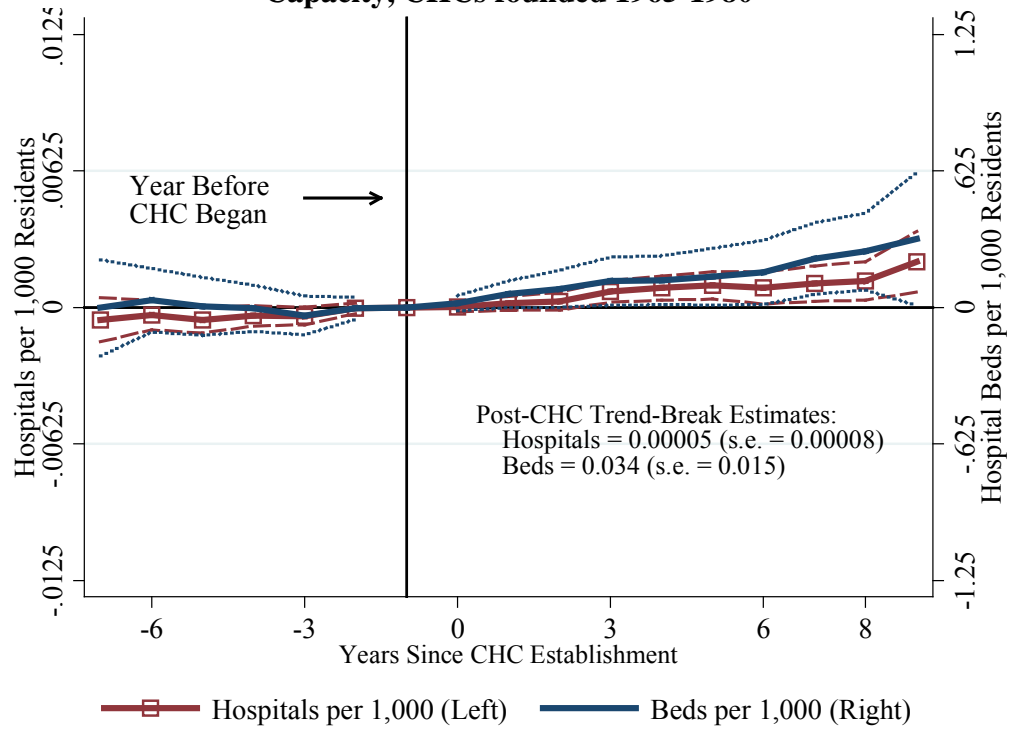
Notes: The coefficients are weighted, least-squares estimates of  $\pi$  and  $\tau$  from our baseline specification of equation 1 where the event-study dummies are estimated separately for areas with above (labeled “urban”) and below (labeled “non-urban”) the median urban share of the population among treated counties in 1960 (81%). See figure 3-5 notes for details on the specification and sources.

**Figure M-3 The Relationship between Community Health Centers and Age-Group Mortality Rates, All CHCs 1965-1980**



Notes: The dependent variable is the all-cause, age-adjusted mortality rate for the indicated age group. Infant mortality is measured per 1,000 live births and mortality rates for other groups are measured per 100,000 residents. Weights are the appropriate county populations in 1960. Infant sample: 2,963 counties with valid data on 1960 characteristics identified in both mortality and natality files (88,890 county-year observations). Mean of infant mortality rate in treated counties in t-1: 22.1. Non-infant sample: 3,044 U.S. counties with valid data on 1960 characteristics (91,320 county-year observations). Mean of AMR in treated counties in t-1 for children is 63.8; for adults is 287.6; and for older adults is 3225.9. See notes to figure 3-5 for details.

**Figure M-4 Relationship between Community Health Centers Establishment and Hospital Capacity, CHCs founded 1965-1980**



Notes: This figure includes all CHCs established from 1965 to 1980, whereas figure 3-9 in the text contains only CHCs established from 1965 to 1974. See figure 3-9 for specification notes and sources.

**Table M-1 Relationship between Community Health Centers and All-Cause Mortality Rates, CHCs founded 1965-1980**

<i>A. Age-Adjusted Mortality, All Ages</i>				
Mean at t*=-1	844.5			
Years -6 to -2	0.5 [1.8]	-2.4 [1.5]	-2.5 [1.6]	-2.2 [1.5]
Years 0 to 4	-3.5 [2.0]	-5.5 [1.5]	-7.5 [1.6]	-5.5 [1.5]
Years 5 to 8	-7.7 [2.6]	-10.0 [2.1]	-13.1 [2.3]	-9.9 [2.2]
R2	0.82	0.85	0.90	0.87
<i>B. Age-Adjusted Mortality, 50 Years and Older</i>				
Mean at t*=-1	2,914.9			
Years -6 to -2	5.2 [6.6]	-6.7 [5.4]	-6.9 [5.7]	-4.0 [5.4]
Years 0 to 4	-16.5 [8.1]	-21.4 [5.9]	-26.7 [6.5]	-23.4 [5.6]
Years 5 to 9	-33.8 [10.5]	-36.7 [8.4]	-44.3 [9.1]	-40.0 [7.6]
R2	0.78	0.80	0.88	0.84
Covariates	C, U-Y	C, U-Y, S-Y, R, D·Year	C, U-Y, S-Y, R, C·Year	C, U-Y, S-Y, R, P-weights

Notes: Models presented are weighted least-squares estimates of equation 1 using event-year categories. C: county fixed effects; U-Y: urban by year fixed effects; S-Y: state-by-year fixed effects; R: annual, county-level covariates; D·Year: 1960 characteristics interacted with linear time trends; C·Year: county-specific linear time trends; P-weights: uses an estimate of the propensity of receiving a CHC to reweight untreated counties. See text for more details. Weights are the appropriate county populations in 1960. See notes to figure 3-5 and 6 for details on sample and sources.



**Table M-2 The Relationship between Community Health Centers and Cause-Specific Mortality Rates for Older Adults, CHCs founded 1965-1980**

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
DV Cause:	All-Cause	Cardiovascular Disease	Cerebrovascular Disease	Cancer	Infectious Disease	Diabetes	Accident
A. Age-Adjusted Mortality, Older Adults (50+)							
Mean at t*=-1	2,915	1318.3	364.2	597.9	103.2	60.3	78.4
Years -6 to -2	-6.67 [5.4]	0.02 [4.07]	0.9 [1.83]	-5.91 [2.02]	1.37 [1.23]	-0.29 [0.7]	-1.33 [0.92]
Years 0 to 4	-21.38 [5.87]	-7.31 [3.52]	-4.85 [2.01]	-5.09 [2.08]	-0.31 [1.09]	-0.25 [0.67]	-0.73 [0.82]
Years 5 to 9	-36.73 [8.45]	-14.04 [5.01]	-6.77 [2.54]	-6.58 [2.78]	-0.4 [1.46]	-0.79 [0.77]	-0.97 [1.02]
R2	0.8	0.8	0.77	0.25	0.31	0.2	0.33
B. Age-Adjusted Mortality, Ages 50-64							
Mean at t*=-1	1,317	507.2	98.3	361.4	37.9	25.8	53.3
Years -6 to -2	-3.4 [4.36]	-1.09 [3.18]	0.27 [1.01]	-1.93 [1.94]	0.46 [0.73]	-0.31 [0.52]	-0.91 [0.85]
Years 0 to 4	-6.43 [4.0]	-3.33 [2.53]	-1.1 [0.97]	-0.55 [1.89]	-0.44 [0.65]	-0.65 [0.51]	-0.12 [0.86]
Years 5 to 9	-16.04 [5.38]	-4.84 [3.14]	-1.99 [1.17]	-2.79 [2.25]	-1.2 [0.72]	-0.82 [0.58]	-0.27 [0.88]
R2	0.58	0.71	0.71	0.18	0.17	0.11	0.25
C. Age-Adjusted Mortality, Ages 65+							
Mean at t*=-1	5,307	2,529.40	761.5	955.1	200.4	111.4	115.7
Years -6 to -2	-10.09 [11.52]	2.1 [9.53]	4.46 [4.1]	-10.03 [4.43]	5.56 [2.96]	-0.5 [1.46]	-0.57 [1.85]
Years 0 to 4	-41.74 [12.69]	-8.8 [10.93]	-8.66 [4.41]	-11.3 [4.67]	-2.21 [2.81]	0.23 [1.69]	-0.21 [1.39]
Years 5 to 9	-67.81 [16.91]	-23.58 [16.21]	-10.73 [5.94]	-12.09 [5.83]	-4.64 [3.61]	-1.03 [2.1]	-0.67 [1.83]
R2	0.76	0.71	0.71	0.18	0.17	0.11	0.25

Notes: The dependent variable is the age-adjusted, age-group specific mortality rate by cause for our baseline specification. See notes to figure 3-5 and table 3 for details on the sample and sources.

**Table M-3 Heterogeneity in the Relationship between Community Health Centers and Mortality Rates, All CHCs Begun 1965-1980**

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
<i>Mean at t*=-1</i>	2,685	3,231	2,503	3,011	2,847	3,470	2,917	2,855	2,933	2,956
Years -6 to -2	-16.3 [6.6]	5.6 [7.7]	-7.0 [11.2]	-7.1 [5.9]	23.2 [18.3]	-8.4 [6.0]	0.6 [6.0]	-9.2 [6.0]	-12.0 [6.8]	-6.1 [6.1]
Years 0 to 4	-10.1 [5.9]	-36.4 [10.4]	-7.9 [10.4]	-23.4 [6.9]	-7.9 [19.6]	-24.9 [6.2]	-19.9 [6.0]	-23.3 [7.0]	-19.7 [7.2]	-20.7 [6.4]
Years 5 to 8	-10.8 [8.8]	-70.7 [14.1]	-3.4 [12.4]	-41.5 [10.3]	-27.3 [24.9]	-39.9 [9.0]	-36.5 [9.1]	-35.1 [10.2]	-29.0 [9.7]	-41.2 [9.1]
R2	0.80		0.80		0.80		0.75	0.81	0.85	0.80
Characteristic defining stratification	1960 AMR		1960 MDs per capita		Race		Dropping One Region at a Time			
	Below Median	Above Median	Below Median	Above Median	Nonwhite	White	NE	MW	S	W
Mean characteristic in group	3,134	3,622	2.8	6.5	100	100				

The dependent variable is the AMR. This table reports model 2 estimates of the effects of  $\tilde{\pi}_y^k$  and  $\tilde{\tau}_y^k$  obtained by replacing equation 1's event-study dummies with  $\sum_k (\sum_{g=-2}^{-1} \tilde{\pi}_g^k D_j^k D_j^g + \sum_{y=0}^3 \tilde{\tau}_g^k D_j^k D_j^g)$ , where  $D_j^k$  is equal to 1 if the county received a CHC between 1965 and 1974 and belongs to group k. k is defined as the group of treated counties with the indicated characteristic. Columns (7)-(10) are from separate regressions, each dropping one region from the analysis at a time as indicated in the column header, and are for 2,423, 1,691, 1,418, and 2,367 counties, respectively.