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Multigenerational Impacts of Childhood Access to the Safety Net: Early Life Exposure to Medicaid and the Next Generation's Health

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Abstract

We examine multi-generational impacts of positive in utero health interventions using a new research design that exploits sharp increases in prenatal Medicaid eligibility that occurred in some states. Our analyses are based on U.S. Vital Statistics Natality files, which enables linkages between individuals' early life Medicaid exposure and the next generation's health at birth. We find evidence that the health benefits associated with treated generations' early life program exposure extend to later offspring. Our results suggest that the returns on early life health investments may be substantively underestimated.

Keywords

I1; I13; I14

There is substantial evidence that health and socioeconomic inequalities persist across generations. A growing number of studies suggest that differences in early life health environments may causally contribute to these disparities. Negative shocks to the *in utero* environment, in particular, have been found to be harmful to individuals' later life health and earnings. A handful of studies also examine positive interventions and find that policies intended to improve early life experiences generate better adult outcomes (Almond and Currie, 2011; Almond, Currie and Duque, 2018). By extension, literatures in economics, epidemiology and child development predict that the causal impacts of these interventions should echo beyond the exposed generation, onto later offspring. Little is known, however, about the extent to which the early life environment affects future generations, or the potential for public policy to alter such linkages.

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We consider whether public health interventions experienced *in utero* affect the next generation's health. We focus on the impact of the largest source of health-related services for low-income individuals in the United States: the Medicaid program. Changes in program rules during the 1980s expanded access to low-income pregnant women who were not already connected to Medicaid through the welfare system, and led to a dramatic increase in prenatal coverage. There was considerable variation in the timing and magnitude of these expansions across states, which prior empirical research has harnessed to document the program's beneficial effects on cohorts who gained *in utero* access. We build on this "first generation" research to investigate whether positive policy interventions in one generation transmit to the next generation.

Our study makes three contributions. First, within the active "early origins" literature, the vast majority of causal studies confine their analyses to treated cohorts. While an ever-expanding number of animal experiments provide compelling evidence that the effects of early life environments can be transmitted to later generations,[1] human studies are more rare. We contribute to this literature by using a quasi-experimental design to document multi-generational effects in humans.

Second, we focus on documenting generationally persistent effects of a widespread policy-driven intervention. Most of what we do know about how individuals' early life health experiences affect their later offspring comes from studies of exposure to disasters such as famine and disease outbreaks, which may not compare well to the effects of more common (and malleable) health experiences. This is an important gap – particularly in light of current political debates about the cost of publicly provided health insurance – as substantive multiplier effects to future generations would suggest that existing benefit-cost calculations underestimate the true value of government investments in children's health.

Third, we advance knowledge of the impacts of the 1980s Medicaid expansions by putting forward a new research design that explicitly addresses ongoing debates about policy endogeneity and the validity of using state-level variation in the expansions' timing and magnitude as a natural experiment. Following the pioneering work of Currie and Gruber (1996a,b), most investigations employ an instrumental variables model to isolate policy effects from other potentially correlated changes in state characteristics. However, lack of information on the pre-expansion period (before 1979) has left open the possibility that estimates of the program's benefits are contaminated by unobserved state pre-trends that were correlated with the expansions' timing and magnitude. We have collected four additional years of information on state Medicaid rules, which allows us to measure state eligibility going back to 1975. With these measures, we estimate an event study model that allows flexible estimation of the dynamic effects of changes in Medicaid, and a direct assessment of the extent to which both Currie and Gruber's estimates, and our own estimates of the program's effects on the next generation's health, reflect other underlying factors. To implement this, we classify states as either treated states or control states based on their expansion patterns across the 1975–1988 period, during which time prenatal Medicaid was

[1]Useful reviews of this literature include Nadeau (2009); Daxinger and Whitelaw (2010); Hochberg et al. (2011); Daxinger and Whitelaw (2012); Heard and Martienssen (2014).

made available to the lowest income families. Treated states are those that experienced sharp increases in Medicaid eligibility, while control states are those that trended more incrementally over time. We also use restricted data on Medicaid coverage among labor and delivery hospital discharges to validate the event study design, and provide important new evidence on the impacts of the expansions on actual take-up.

Our main analyses are based on information that is available in restricted-use versions of the U.S. Vital Statistics birth records. With these data, we analyze infant health outcomes among two generations. First, we consider those who were directly exposed to the expansions: infants born between 1975 and 1988. We call these cohorts the "first generation." Most previous research has focused on a subset of these cohorts, who were born after 1979. Then, we move on to investigate whether the benefits of *in utero* Medicaid exposure affected the next generation by looking at birth outcomes among the first generation's offspring. We call these infants the "second generation." For the first generation, we use state and year of birth to assign policy generosity, and, for the second generation, we use *mother's* state and year of birth to link to measures of mother's early life Medicaid exposure.

We estimate Medicaid's impacts on standard measures of infant health, including birthweight and low birthweight. These outcomes are strong candidates for a second generation exploration because previous research has shown that birthweight is tied to measures of maternal health such as obesity and diabetes, for which there is already evidence of long-term program effects (Miller and Wherry, 2019). Moreover, birthweight is highly predictive of later life health and economic outcomes, making it an outcome of particular interest. We also investigate other measures of second generation health, including variants of gestational length and weight-for-gestational age. Finally, we explore potential transmission mechanisms. In addition to confirming Medicaid's impacts on the first generation's health at birth as a potential pathway, we consider changes in first generation fertility patterns that might be indicative of selection, and changes in first generation health and health behaviors during pregnancy.

We find that the expansions increased the first generation's likelihood of being enrolled in Medicaid at birth, and we validate the past research finding that the expansions reduced the percent of the first generation who were born low birthweight. Then we go on to show the benefits of expanding early life access for the next generation: the first generation's *in utero* exposure to Medicaid leads to a statistically significant increase in the second generation's birthweight and reduces the incidence of very low birthweight and small for gestational age. We also observe less precisely estimated, but suggestive, declines in low birthweight and very preterm births.

Analyzing the effect of a policy across multiple generations is complicated because of the time that passes between the first generation's treatment and the second generation's outcomes. Our identification assumption requires that among cohorts born in treated states around the time of the expansions there are no other cohort-state varying economic or health shocks that also affect the health of the next generation. As with all such research designs, we cannot be 100% sure that we have eliminated all possible confounders; nevertheless, our event study approach offers a transparent way to assess the validity of our design and

our results. We also show that our results are robust to a variety of specification checks, including alternative state and year control variables, changes in the sample definition, alternative measures of eligibility, omitting the control states entirely, and versions of the model that use a synthetic control approach to construct the comparison group. They are also robust to new methods proposed by Callaway and Sant'Anna (2020) that account for biases that are sometimes found in traditional two-way fixed effects and event study models. In some cases, these alternative specifications provide wider confidence intervals than our baseline results or moderately change the size of the effect. When taken together, however, the pattern of results paints a compelling picture of improvements in second generation infant health.

Documenting the presence of multi-generational spillovers is an important contribution in its own right. Moreover, back-of-the envelope calculations suggest that the magnitude of the spillovers is economically important. Our point estimates of the effects of *in utero* Medicaid access on the second generation's birthweight suggest medical cost savings in the first year of life that are about 60 percent of the costs of providing the first generation with *in utero* coverage. Even the lower bound of the 95 percent confidence interval suggests cost savings of around 16 percent of initial investment costs. Importantly, this calculation does not include any other benefits that were likely accrued to the second generation, such as later life savings in medical costs or social supports. Nor does it include benefits associated with previously documented improvements in the first generation's health and economic outcomes. If these benefits were incorporated, the costs savings would be substantially larger.

We find no evidence that changes in overall fertility can explain the effects on infant health, but we do find a small shift in the racial composition of women giving birth: first-generation Medicaid exposure increases the percent of second-generation births that are to white women, and decreases the percent to Black women. Controlling for mothers' demographic characteristics does not change the baseline results, however, and results are similar when we focus on white births, a sub-group for whom we would not expect to see effects if the main estimates are driven by changes in racial composition. Therefore, we do not believe that selection is driving the results.

Our results indicate that public investments in prenatal health have persistent impacts beyond the treated generation. By quantifying these effects, we establish that benefit/cost ratios based only on outcomes directly experienced by cohorts who were immediately affected by the Medicaid expansions underestimate the program's overall efficacy. More broadly, our analyses suggest that even "long-run" studies of early-life interventions may fail to capture the full extent of benefits conferred.

The remainder of our paper proceeds in the following way: Section I provides further information about the existing literature on "early life" health and multigenerational processes. In Section II, we describe the Medicaid program and the nature of the 1980s expansions. Sections III and IV describe our empirical strategy and data. We present our results in Section V and conclude with a discussion in Section VI.

I. Background

More than 40 years ago, Forsdahl (1977) put forward a provocative hypothesis that the period of gestation has significant impacts on individual health that reach well into adulthood. This theory gained further traction following Barker (1990), and in recent years there has been growing scientific agreement that the time both before and immediately after birth are critical periods when the developing body takes adaptive cues from its surrounding environment. A key feature of the "fetal origins" hypothesis is that the health effects of the *in utero* environment can remain latent for many years. We have yet to achieve a full understanding of the processes underlying these phenomena, but a leading theory is that the fetus's surrounding environment alters genetic programming through the "switching on" of specific genes.

Numerous economists and epidemiologists have used quasi-experimental designs to test the fetal origins hypothesis, and have found that *in utero* and early life health experiences can have important effects on later life outcomes (Almond and Currie, 2011; Almond, Currie and Duque, 2018). Within this literature, a handful of studies consider the long term efficacy of wide-spread positive health interventions in the modern U.S. In particular, Bailey et al. (2020), Bitler and Figinski (2019) and Hoynes, Schanzenbach and Almond (2016) investigate the 1960's roll out of the Food Stamp program and find that access to the program in early childhood generated improvements in later life economic and health outcomes, including conditions related to cardiovascular disease such as obesity, high blood pressure, and diabetes. Similarly, using variation in Medicaid rules that expanded coverage to low-income pregnant women during the 1980s, Miller and Wherry (2019) find that *in utero* exposure to Medicaid reduced the likelihood of having metabolic-syndrome and circulatory-system linked chronic illnesses in adulthood. Importantly for our study, when these later life diseases are experienced during pregnancy, women and their children are put at risk for a variety of health problems, including an increased risk of gestational diabetes, complications related to high blood pressure, and preterm birth (Catalano and Ehrenberg, 2006). Moving beyond health, Miller and Wherry also document that *in utero* exposure to the program is associated with increases in educational attainment,[2] a finding that is echoed in research evaluating the long-term effects of the 1980s and 1990s expansions to broader age groups (Currie, Decker and Lin, 2008; Cohodes et al., 2016; Thompson, 2017; Wherry and Meyer, 2016; Wherry et al., 2017; Brown, Kowalski and Lurie, 2020).[3] The latter studies do not separately estimate the effects of *in utero* exposure from later childhood exposure, however, and Miller and Wherry find that this distinction is important, with the prenatal expansions generating substantially bigger impacts.

Taken as a whole, the existing literature has generated two broad conclusions. First, early life health shocks have long-term impacts on the health and economic outcomes of those who experience them. Second, many widespread public health interventions targeted at children have substantive positive benefits that last well into adulthood. A natural question is

[2]This is consistent with earlier work by Levine and Schanzenbach (2009) linking Medicaid-induced improvements in infant health to higher test scores in 4th and 8th grade.

[3]There is also similar evidence from studies of the Medicaid program's introduction between 1966 and 1970 (Boudreaux, Golberstein and McAlpine, 2016; Sohn, 2017; Goodman-Bacon, 2021).

whether these effects endure to the next generation. Economists have previously documented that health and economic status persist across multiple generations (Clark, 2014; Solon, 2018; Halliday, Mazumder and Wong, 2021), but quasi-experimental investigations are rare. We know little about what drives the correlations, or the potential for policy based treatments to alter them. The dearth of work among social scientists likely results from the multiple challenges of identifying exogenous variation in early life health environments and linking that variation to data that provides relevant information on multiple generations.

These challenges can be overcome in biological studies, where an accumulation of evidence based on animal experiments finds that prenatal health shocks have persistent generational effects. As an example, studies have documented that rats that are malnourished before or during pregnancy produce offspring with smaller brains and reduced cognition, even if the offspring receive sufficient nutrition after birth. Importantly, these effects are not only observed in the immediate offspring, but are present in later generations as well (Galler and Rabinowitz, 2014). Similar multi-generational patterns have been found with *in-utero* exposure to disease and stress (Babenko, Kovalchuk and Metz, 2015; Weber-Stadlbauer, 2017). One explanation for this pattern is that the biological predecessors of the ovaries and sperm cells, which produce the next generation, are already present at the fetal stage and are therefore exposed to any insult experienced by the fetus.

In spite of the methodological challenges, a few epidemiologists and economists have been able to shed light on this question by exploiting historical shocks. Painter et al. (2008) investigate the multi-generational impacts of the Dutch Hunger Winter of 1944–1945, which reduced the food consumption of a previously well-nourished population by more than 75 percent. They find that the offspring of those who were exposed to the famine *in utero* had worse health in later life. Similarly, Almond et al. (2010) find that fetal exposure to malnutrition resulting from the 1959–1961 Chinese famine increased low birthweight incidence in the next generation. Looking beyond the effects of extreme nutritional deprivation, Richter and Robling (2016) find that the children of those who were exposed *in utero* to the 1918–1919 influenza pandemic grew up to have lower levels of educational attainment. Similarly, Black et al. (2019) find that Norwegian cohorts exposed to radioactive fallout during the *in utero* period had children with lower cognitive ability.[4]

A small number of studies examine intergenerational health effects in the more modern U.S. context. Two studies relate local variation in infant mortality rates at the time of the mother's birth to her later offspring's health. Here, the infant mortality rate proxies for broad disease exposure, which can be driven by many factors, including access to medical care. Almond, Currie and Herrmann (2012) find that higher exposure to disease is associated with worse long-term outcomes and an increase in the probability that future offspring are below the low birthweight threshold. Almond and Chay (2006) focus on the dramatic improvements in Black infant mortality rates that coincided with the Civil Rights Act, and find that Black women born during the late 1960s had a reduced likelihood of giving birth to a low birthweight infant. A third study, Colmer and Voorheis (2020), documents improved

[4]A few studies also find that historical health shocks experienced at later ages have generationally persistent effects (van den Berg and Pinger, 2016; Bütikofer and Salvanes, 2020).

educational outcomes among the grandchildren of cohorts who benefited from reductions in pollution exposure driven by the 1970 Clean Air Act Amendments.[5]

We build on this small number of studies by harnessing a policy driven increase in access to a widespread public health program that is a critical component of the U.S. safety net. This allows us to establish multi-generational linkages associated with more common and contemporaneous variation in early life health experiences, while simultaneously quantifying long-term benefits of the Medicaid program that have not been previously considered. Medicaid may alter biological associations across generations by increasing the use of prenatal care, which provides nutrition and drug counseling, immunizations, early diagnoses and direct interventions.[6] Along these lines, Currie and Gruber (1996b) link changes in prenatal Medicaid access to reductions in treated cohorts' probability of being low birthweight, and maternal birthweight is predictive of later offspring's birthweight (e.g. Black, Devereux and Salvanes, 2007; Currie and Moretti, 2007; Royer, 2009). Access to Medicaid may also reduce maternal stress: in an analysis of the Oregon Health Insurance Experiment, Finkelstein et al. (2012) find that those who gained health insurance through the experiment experienced substantive improvements in mental health. Several studies have linked parental and *in utero* stress to children's well-being (Camacho, 2008; Valente, 2011; Mansour and Rees, 2012; Black, Devereux and Salvanes, 2016; Persson and Rossin-Slater, 2018), with possible ramifications for the next generation's health.

Besides biological pathways, Medicaid may of course affect the next generation's health through its documented impacts on the treated generation's human capital and earnings. As described earlier, numerous studies find that the Medicaid expansions had positive effects on treated cohorts' educational and economic outcomes in adulthood, and it is well known that children living in high income families are healthier than children living in low income families (Case, Lubotsky and Paxson, 2002; Case, Fertig and Paxson, 2005; Currie and Almond, 2011). This is intuitive, as parents with more income have more resources to invest in their children, experience lower stress levels (Evans and Garthwaite, 2014; Aizer, Stroud and Buka, 2016) and engage in healthier behaviors (Hoynes, Miller and Simon, 2015).

II. Medicaid and the 1980s Expansions

Medicaid currently provides health insurance coverage for nearly half of all births in the U.S. (Markus et al., 2013). Eligibility criteria for pregnant women were relatively restrictive until the 1980s, when a series of state and federal policy changes greatly expanded access. We describe the inception of the Medicaid program, including which groups were initially eligible, and the later policy changes that expanded eligibility, below.

Created in 1965 as part of the Social Security Amendments, Medicaid was initially available to low-income, non-disabled women of reproductive age who received cash assistance through the Aid to Families with Dependent Children (AFDC) program.[7] Eligibility for

[5]In addition, two recent studies document that the introduction of preschools (which included a health component) had positive effects on later generations' educational outcomes (Barr and Gibbs, 2019; Rossin-Slater and Wüst, 2020).

[6]Several studies of Medicaid's prenatal expansions document increased use or improved timing and adequacy of prenatal care (Currie and Gruber, 1996b, 2001; Dubay et al., 2001; Howell, 2001; Dave et al., 2008). The medical literature also hints that prenatal care may be a pathway for improving the intergenerational transmission of health (Lu et al., 2003).

AFDC was restricted to single women with at least one dependent child. Low-income women with first time pregnancies and those with marital partners did not qualify for coverage. Moreover, AFDC income eligibility thresholds were generally much lower than the federal poverty line. The average threshold was 61% of the federal poverty line (FPL) in 1979, and ranged from 24% to 99% across states.[8]

Because Medicaid is a joint federal-state program, the federal government sets mandatory eligibility requirements, but states have some flexibility to extend eligibility to other especially needy population groups. Dating back to 1966, a number of states exercised different options to extend eligibility to certain pregnant women not eligible for AFDC. The options offered coverage to specific groups of pregnant women, such as first-time pregnant women who would later qualify for AFDC, or pregnant women in two-parent families where the principal earner was unemployed and who met the income and resource requirements for AFDC.[9] Some options extended eligibility to all pregnant women who were financially eligible for AFDC, but who did not meet the program's family structure requirements (i.e. women who were *not* single mothers with dependent children). Adoption of these options was not uniform across states, and, prior to the 1980s, there were substantive differences in eligibility criteria that were applied to pregnant women.

Beginning in the early 1980s, there was emerging national consensus on the importance of prenatal care for pregnant women, which led to major changes to the Medicaid program (Howell, 2001). More states began to exercise options to expand Medicaid eligibility to pregnant women who did not meet the family structure requirements for AFDC, but who did meet the AFDC financial eligibility criteria. This wave of new optional state expansions was followed by two federal mandates requiring that *all* states expand eligibility to these women. These changes caused abrupt expansions in states with historically less generous eligibility rules and lower eligibility levels.

Figure 1 Panel (a), which is based on information we have gathered on state policy rules back to 1975, documents that these changes had a large effect: between 1975 and 1988 the share of women who qualified for pregnancy-related Medicaid climbed from about 12 to 20 percent.[10] Here, and throughout the rest of the paper, we multiply all proportions by 100 for ease of presentation. Panel (b), which uses data on hospital discharges for labor and delivery between 1979–1988, shows that this increase corresponded to a steep increase in the share of mothers who were enrolled in the Medicaid program at the time of their child's birth.[11] To our knowledge, this is the first time that administrative data have been used to document that take-up patterns among pregnant women mirrored these eligibility expansions.[12]

[7]Since Medicaid was initially tied directly to AFDC, many women received benefits from both programs. However, as we describe in detail in Appendix Section A.I, the policy variation we exploit is driven by expansions to Medicaid and not by changes in AFDC benefits.

[8]Authors' calculation based on payment standard for a family of 3 in 1979.

[9]Appendix Section A provides a detailed discussion of each of the different state options described in this section.

[10]Additional information about the calculator and data used to calculate eligibility in this figure is provided in Section III and Appendix Section A.

[11]While the changes track quite well across both of these measures, there are a few years in which coverage actually exceeds eligibility. We believe this is due to the fact that our eligibility estimates are based on women of child-bearing age, whereas our estimates of coverage are derived from a sample of women actually giving birth. These groups may differ on some important dimensions such as age and income.

Following the original terminology used by Currie and Gruber (1996b), we refer to these changes in eligibility as the "targeted" expansions. They differ from "broad" expansions that occurred later because they were aimed at very low income pregnant women (i.e. those with income levels below AFDC thresholds). Beginning in the late 1980s, the broad expansions allowed states to further expand coverage to pregnant women and children with higher incomes. We cannot make use of variation generated by broad changes in our analyses because most of the affected cohorts are still too young to allow for multi-generational analyses. We note, however, that previous studies have documented that the targeted expansions had stronger effects on treated cohorts' outcomes (Currie and Gruber, 1996b; Miller and Wherry, 2019), making them a preferred candidate to test for multi-generational effects. We also note that pregnant women who enrolled in Medicaid received coverage for prenatal care and services, hospital and postpartum care, and one year of Medicaid eligibility for their newborns (Congressional Research Service, 1988).

With this background in mind, we investigate multi-generational effects of *in utero* Medicaid access by focusing on the offspring of cohorts who were born between 1975 and 1988. We refer to the mothers in these cohorts as the "first" (exposed) generation, and to their later infants as the "second" generation.

III. Empirical Strategy

A. Background

Currie and Gruber examine the impact of the prenatal Medicaid expansions on first generation infant health by estimating a model that exploits variation in the magnitude of the expansions across states and over time. Specifically, they estimate:

$$y_{nb} = \alpha + \phi Elig_{nb} + \mu_n + \lambda_b + \gamma X_{nb} + \epsilon_{nb} \quad (1)$$

where y_{nb} is the fraction of infants born in state n and year b who were below the low birthweight threshold, μ_n and λ_b are state of birth and year of birth fixed effects, respectively, and X_{nb} is a vector of state-year control variables that reflect the demographic, economic, and policy environment. $Elig_{nb}$ is the percent of women of child-bearing age in the state-year who were eligible for Medicaid in the event of a pregnancy. Currie and Gruber's analyses, as well as later studies that build upon this research design, begin in 1979, on the eve of the Medicaid program's rapid expansion.

Acknowledging that changes in states' demographic and economic conditions could cause changes in the percent eligible for Medicaid even without a change in policy, Currie and Gruber pioneered a "simulated instrument" approach in which they instrument actual eligibility with a simulated measure that isolates changes driven only by changes in Medicaid rules and is independent of changes in states' demographic and economic characteristics. The simulated measure is constructed by applying Medicaid eligibility rules in each state and year to a *national* sample of potential mothers (women ages 15–44) drawn

[12]Dave et al. (2010) use this source of hospital discharge data to examine the effects of later Medicaid expansions (1985–1996) on the health insurance coverage of pregnant women at the time of child birth.

from each year of the Annual Social and Economic Supplement of the Current Population Survey (Flood et al., 2021). Our analyses also rely on simulated eligibility measures to capture changes in Medicaid policy, following the convention that has been adopted by much of the literature.[13] Appendix Figure A.1 shows that national trends in actual and simulated eligibility measures are very closely related.

Using this model, Currie and Gruber find that a 10 percentage point increase in prenatal Medicaid eligibility under the targeted expansions reduced the first generation's incidence of low birthweight by 2.6 percent.[14] Subsequent papers have extended their approach to look at outcomes associated with later childhood expansions by replacing $Elig_{nb}$ with a measure of cumulative or average childhood eligibility from birth to age 18 (e.g. Cohodes et al., 2016; Thompson, 2017; Brown, Kowalski and Lurie, 2020) and using a comparable simulated eligibility instrumental variables approach. Noting that the prenatal period is a particularly receptive stage of development, with the potential to yield large returns on investment, Miller and Wherry (2019) further extend this design by including separate measures of prenatal and later childhood eligibility. They find that this distinction is important, with most of the long-run health improvements driven by the expansions in prenatal access.

It would be natural to extend this framework to examine the expansions' impact on second generation outcomes.[15] As Currie and Gruber note, however, identification in this model rests on the assumption that state Medicaid policy was exogenous to treated cohorts' birth outcomes. Although previous analyses have included a large number of state and year varying controls, some researchers have argued that the changes are not "randomly occurring natural experiments," and they speculate that estimates of the expansions' impacts reflect other underlying state-level changes (Dave et al., 2008). The traditional research design has previously been estimated with very limited years of data from the pre-expansion era, which restricts researchers' ability to test for differential changes across states prior to the expansions. Therefore, we have collected additional years of information on state program rules, and use these data to document changes in state-level prenatal Medicaid eligibility back to 1975.[16] Then we implement a more transparent event study design that allows direct examination of the extent to which state-level pre-period outcomes varied with state-level Medicaid expansions, and addresses concerns about potential contaminants.

Our analyses start by documenting the evolution of state-level Medicaid eligibility. Appendix Figure A.2 shows the time path of eligibility from 1975–1988 for each of the

[13]To create the simulated *in utero* measure, we use a national random sample of 3,000 women from each year of the Annual Social and Economic Supplement of the Current Population Survey (CPS). Since childhood eligibility was also changing for these cohorts, we also examine whether the changes in prenatal eligibility were correlated with changes in childhood eligibility, and whether including controls for childhood eligibility affects the estimates. To construct measures of simulated childhood eligibility, we use national random samples of 1,000 children at each age between 1 and 18 in each survey year and calculate the percent of children in each state, year, and age that would be eligible for Medicaid based on state eligibility rules. We then add the estimates across ages for each cohort, to create a measure of cumulative simulated eligibility throughout childhood for each cohort and state and use this as a measure of generosity during childhood. We describe these calculations in more detail in Appendix Section A.

[14]We replicate this result using their empirical approach in Appendix Table A.1. Note that, for the purpose of this replication, we follow Currie and Gruber and express the outcome as the number of low birthweight births per 1000 infants. The first column displays the results reprinted from their paper and the second column is our replication exercise. The remaining columns update the analysis to cluster standard errors by state (column 3), add the state by year controls we implement in our model, described in detail below (column 4), and weight by the number of births in each cell (column 5).

[15]An earlier version of this paper used this approach. That version is available upon request.

[16]Information on eligibility prior to 1975 is very spotty, therefore we begin our sample period in 1975.

fifty states. It is immediately clear that states exhibit different expansion patterns, and that these patterns fall into two dominant types: many states experienced small, gradual increases in eligibility throughout the period (e.g. California and Rhode Island), while others exhibit flat, low levels of prenatal coverage during the 1970s that are later punctuated by a large, abrupt, increase (e.g. South Carolina and Maine).

This is exactly what the discussion in Section II should lead us to expect. Specifically, in early years, states that initially embraced strict eligibility criteria and did not avail themselves of the state options, exhibit low levels of eligibility. In later years, we observe discrete jumps in these states that correspond to a large, voluntary state-level expansion or one of the later federally mandated expansions. In contrast, states that took early advantage of the existing state options to cover low-income pregnant women have relatively high levels of eligibility from the beginning of the study period. As a result, these states exhibit smaller, more gradual increases that are less pronounced in the wake of the subsequent state and federal changes. These two different eligibility trajectories will be demonstrated visually in the next subsection.

B. Classification of Treatment and Control States

Using Appendix Figure A.2 as a guide, we organize states into “treated” states (those that experienced a sharp jump in eligibility) and “control” states (those for whom eligibility trended smoothly). Our event study is based on differences in the evolution of prenatal eligibility across these two groups. The treatment group consists of the 28 states in which eligibility was stagnant for a minimum of the five years between 1975 and 1979, and that later experienced a large positive shock. We focus on shocks that took place between 1980 and 1985 to ensure that we have at least four “post-event” years over which to observe the expansions’ effects. We include the 22 remaining states as “control” states to help identify secular changes that were separate from the Medicaid expansions.[17]

Figure 2 shows which states we classify as control states, which states we classify as treatment states, and the timing of the expansion for the treatment states. Given that “treatment” requires a large, abrupt increase in eligibility, it is unsurprising that treated states are disproportionately located in the South and Midwest; Medicaid was less generous in the pre-expansion period in these states, and transfer policies have historically been less generous in these parts of the country as well.[18]

[17]We did not have a specific algorithm to define treatment and control states or event time. Rather, each of the four authors individually classified states based on Appendix Figure A.2, and then we decided on a consistent classification as a group. We relied on measures of simulated eligibility, rather than actual eligibility, to ensure that changes in eligibility are driven by policy changes and not by demographic or economic shifts, and there were few classification discrepancies across the authors. States with large changes outside the 1980–1985 period are included in the control group, but the few states that fall into this category do not affect our results: we show that estimates are similar when we drop the control states from the analysis.

[18]Prior work has found that racism played a role in generating a less generous safety net (Lee and Roemer, 2006), and, Appendix Table A.2 shows that treated states have slightly higher Black populations than control states. We multiply all proportions by 100 for ease of presentation. There are a few small differences in other demographic and economic characteristics across treatment and control states. Our analyses include state fixed effects to account for any time-invariant differences, and we show that our estimates are robust to including a large number of state-year varying economic and policy controls, and region-year fixed effects that control for common shocks to outcomes within regions over time.

Figure 3 Panel (a) provides further insights into the sources of our identifying variation by combining all of the treated states together, and documenting how the changes in eligibility break down by source. The sources are shown additively from most to least restrictive (shown from lowest to highest lines on the graph), and include changes in eligibility driven by: changes in AFDC rules (solid blue line), targeted expansions occurring via state options (long dashed red line), targeted expansions occurring under federal mandates (short dashed green line), and broader income-based expansions (dash dot yellow line). The last category is concentrated at the end of the period, and reflects optional state expansions that included pregnant women with family incomes up to the poverty line.[19]

It is easy to see that prior to the 1980s, Medicaid eligibility in treated states was largely determined by state AFDC rules, and that these states were less likely to take up state options than the control states. Beginning in the early 1980s, a few treatment states increased eligibility through the state options. This was followed by a sizeable bump under the federal mandates. Between 1975 and 1988, prenatal eligibility roughly doubled in treatment states, from just under 9 percent to 18 percent. In contrast, Figure 3 Panel (b) shows that control states started off with higher eligibility, because they had already implemented the state options, and that eligibility gradually increased over time as more options were adopted. As a result, the federal mandates had smaller effects on eligibility.

Not only is the change in eligibility greater for treatment states than control states, but the women affected are more disadvantaged due to the more stringent baseline eligibility rules for these states. To demonstrate this, we compare the characteristics of women who gained eligibility over this period in both groups of states in Appendix Table A.3.[20] The women in treated states are noticeably more disadvantaged in terms of their family income and poverty rate than women in control states. Notice that nearly half of the women gaining eligibility in treatment states have family incomes below 50% of the FPL, versus only 13 percent in the control states. Women in the treatment states are also more likely to be single and non-white.

Finally, Figure 4 demonstrates that among treated states there is also variation in when the large jumps occur. States are grouped together by the initial year of the abrupt increase (the “expansion year”), and eligibility is plotted relative to the year prior to expansion, with the number of states expanding in each year shown in parentheses. While the most common expansion years are 1982 (11 states) and 1985 (7 states), when federal policy provided states with new options to expand coverage (1982), and mandated expansion of coverage to more women (1985), there are also states that experience large expansions in every other year between 1980 and 1985.[21] In our main event study framework, we rely on the differential timing of expansions across states, as well as differences between treated and control states, to identify the effects of Medicaid separately from time trends.

[19]Under the Omnibus Budget Reconciliation Act of 1986, states were allowed to expand Medicaid to pregnant women with family incomes below 100% FPL starting in April 1987. See additional discussion in Appendix Section A.I, along with more detailed breakouts of the different eligibility paths in Appendix Figures A.3 and A.4.

[20]Using the CPS, we calculate mean characteristics for the women in treatment states who are eligible during the post-period (defined using each state’s specific treatment timing), but who were not eligible under the rules that were in place during the last pre-treatment year. We calculate mean characteristics for women in control states who are eligible during 1982–1988 but were not eligible under the rules in place in 1981.

[21]See Appendix A.I for more details on these policy changes.

C. Event Study

The event study takes the following form:

$$y_{nb} = \alpha + \sum_{t=-5, t\neq -1}^{3} \beta_t 1\{b - e_n^* = t\} + \mu_n + \lambda_b + \gamma X_{nb} + \epsilon_{nb} \quad (2)$$

where y_{nb} is an outcome for individuals born in state n and year b. Initially, we estimate the expansions' effects on the first generation's *in utero* Medicaid eligibility and coverage at birth. Then, in line with the existing literature, we examine the first generation's health outcomes at birth. The primary goal of our study, however, is to understand whether the benefits associated with the first generation's prenatal access to Medicaid had spillover effects onto their offspring. To do so, we replace first generation outcomes with the outcomes of the offspring of women who were born in state n and year b.

The key regressors are the series of dummy variables $1\{b - e_n^* = t\}$ that take on a value of one for each event time year, where event time is defined for each treated state relative to the year in which it first experienced a discrete jump in eligibility (e_n^*). We omit the year before each state's large expansion $t = -1$, so the estimated β_t s are relative to the year before the expansion occurred. For example, β_1 is the effect one year after the discrete change in eligibility, relative to one year before the jump.[22] We do not define event time for the control states since they do not experience a large discrete expansion. The control states help estimate secular trends across cohorts and the effects of the control variables.[23]

As in previous studies, we include first generation state of birth fixed effects, μ_n, to account for fixed differences in the outcomes of mothers and their children across states, and first generation year of birth fixed effects, λ_b, to account for national changes over time. Our baseline model also follows the literature by incorporating a large number of first generation state and year of birth control variables including the unemployment rate, personal income per capita, maximum welfare benefit for a family of 4, indicators for state parental consent and notification laws, state Medicaid restrictions for abortion, and population demographic controls for each state and year. Sensitivity analyses include versions of the model that eliminate the control variables, or add additional controls, such as region-year fixed effects, and state-year controls at the time of the *second* generation's birth, which we discuss in more detail below. Our results are not sensitive to their inclusion. Our estimates are weighted by the size of the second generation birth cohort.[24] We cluster our standard errors by mothers' state of birth.

[22]By choosing to classify treated states as those with a large, abrupt eligibility change in the middle of the 1975–1988 period, we are able to observe at least 5 years before the event and 4 years after the event. We bin event time observations that are more than 5 years before the event and more than 4 years after the event. We estimate, but do not report these coeffcients, because they are based on an unbalanced sample. Binning allows us to separately identify treatment effects from secular time trends even when we do not include the control states in the model (Schmidheiny and Siegloch, 2019).

[23]A potential concern is that using partially treated states (those with smaller and less abrupt changes in eligibility) as a control group will lead to biased estimates (Schmidheiny and Siegloch, 2019). Figure 10 documents that the pattern of estimates is similar (albeit with larger standard errors) when the control states are not included in the analyses. We also note that there is little difference in the magnitude of the eligibility changes across treated states (Figure 4). Therefore, we do not use variation in the magnitude of the expansions as an additional source of identifying variation in our model.

[24]The results are robust to weighting by the size of the first generation cohort instead.

Appendix Figure A.5 plots second generation health outcomes by event time, where each treatment timing group is shown in a different color. Control states are shown in pink, with event time centered at 1982. It is easy to see that, consistent with the decades long national trend in infant health (Wang, 2010; United Health Foundation, 2020), and pre-dating the expansions, second generation health was declining. Linear trend estimates based on *pre-expansion* data are shown in solid lines, and clearly vary across state groups, possibly reflecting differing trends in the demographics of women giving birth, adoption of health technologies, or changes in state policies (including expansions of prenatal Medicaid eligibility that occurred before those that are the focus of this study). Appendix Figure A.6 documents that first generation health also trended differently across state groups. To ensure that our estimates are not contaminated by state differences in pre-expansion trajectories, we remove linear pre-trends following the two-step de-trending procedure implemented in Goodman-Bacon (2020) and described in more detail in Section 5.

This research design is a departure from the conventional approach, which exploits state and cohort variation in the timing and magnitude of the expansions but does not address potential differences in states' pre-period trajectories. Another advantage of our research design is that it provides non-parametric estimates of the expansions' dynamic effects. Assuming that treatment effects are homogeneous across treatment groups, estimates generated by the event study will be unbiased, even in the case of staggered treatment timing. As described below, we examine the importance of this assumption using the method proposed by Callaway and Sant'Anna (2020) and obtain similar results.

IV. Data

Our measures of Medicaid eligibility are based on data in the Annual Social and Economic Supplement of the Current Population Survey and are described in more detail in Section III and Appendix Section A. We also analyze Medicaid take-up among pregnant mothers using data from the National Hospital Discharge Survey (NHDS) (National Center for Health Statistics, 1988). Because the analysis requires the state in which the birth occurred, which is a restricted variable, these data were accessed through a Federal Statistical Research Data Center. The NHDS data provide discharge-level information for a nationally representative sample of non-Federal, short-stay hospitals, and include the expected payer for the hospital visit. This allows us to identify births paid for by Medicaid. Because the NHDS data are based on administrative hospital records, they are not subject to misreporting issues that are common in survey data (see Klerman, Ringel and Roth, 2005; Davern et al., 2009), but unlike our other data series, they are not available before 1979.[25] In our event study analyses, we estimate the pre-period coefficients using all available data. We also apply the NHDS survey weights. Using diagnosis and procedure codes based on the

[25]We acknowledge that if some pregnant women did not enroll in Medicaid until delivery then the NHDS data could produce an upper bound estimate of the percent of infants who gained *in utero* coverage. Using Medicaid claims data from four states, Ellwood and Kenney (1995) document that the share of women with Medicaid deliveries who enrolled during the *first trimester* were 50 percent (California), 52 percent (Georgia), 69 percent (Michigan), and 49 percent (Tennessee) in 1987. The authors also document higher first trimester enrollment among women receiving cash assistance, ranging from 66–79 percent. These take up rates were almost certainly higher at later stages of pregnancy, but before delivery, suggesting that the vast majority of women enrolled prior to delivery. Related to this, we remind the reader that all children who were covered at the time of delivery were eligible for coverage during their first year of life.

International Classification of Diseases coding system,[26] we identify 187,488 labor and delivery hospitalizations between 1979 and 1988 that include information on the expected payer.

To conduct our main birth outcome analyses, we use the 1975–2017 U.S. Vital Statistics Natality Data Files (National Center for Health Statistics, 1992, 2017).[27] These files contain individual birth records for the full census of U.S. births. The data include information on infants' health, gender, parity, and year and state of birth. They also include detailed demographic information about each infant's mother, including her state of birth and age, which allows us to approximate her year of birth.[28] The latter variables are critical to our second generation analyses, as they allow measures of *in utero* Medicaid eligibility to be matched to each mother in the first generation. In the second generation analyses we exclude infants whose mothers were born outside of the United States, as well as mothers born in Arizona, which did not adopt a state Medicaid program until 1982.

We begin by using the event study framework to reassess Currie and Gruber's first-generation results. Currie and Gruber focus on the incidence of low birthweight (less than 2500 grams) among infants born between 1979 and 1992. Appendix Table A.1 shows that we are able to replicate their results when we apply their research design to the same cohorts. Moving forward, our event study analysis changes the included cohorts in two ways. First, because a convincing event study requires pre-period data, we add the four cohorts born between 1975 and 1978. Second, the second generation analyses would ideally include all births to women born between 1975 and 1992, but many women born in the 1990s have not given birth by 2017, so we focus on cohorts born between 1975 and 1988. This ensures that all first generation cohorts that were affected by the targeted prenatal expansions are included, while allowing us to observe each cohort's fertility through age 28. The analyses of second generation outcomes is restricted to infants whose mothers meet this age criteria to ensure that each maternal cohort contributes equally to the identifying variation, and that cohort-level comparisons are across women who are giving birth at the same age. We further restrict the second generation analyses to first births only, as this generates a more representative sample of births across cohorts. During our time frame, 81% of first births, and 62% of all births, were to women aged 28 or younger.[29] We test the robustness of our results to both of these restrictions below.

The second generation analyses include infants born between 1990 and 2017.[30] We examine the impacts of the prenatal expansions on the next generation's average birthweight and incidence of low birthweight. These outcomes are standard measures of infant health and are highly predictive of later life health, cognitive and economic outcomes (e.g. Black, Devereux and Salvanes, 2007; Figlio et al., 2014). We also explore other outcomes

[26]Details are found in Appendix Section D.
[27]Beginning in 2005, mother's state of birth information is only available in the restricted access data, so we use the restricted data from 2005–2017.
[28]Miller and Wherry (2016) show that using age to impute birth year leads to nearly identical simulated eligibility assignments as using actual birth year.
[29]Authors' calculations from the Vital Statistics Natality Files.
[30]We restrict births to women ages 15 or older. The second generation sample, therefore begins in 1990, since this is year that the 1975 cohort turns 15.

available in the national natality data, including very low birthweight (weighing less than 1500 grams), gestational length (in weeks), preterm (born before 37 weeks gestation), and very preterm (born before 28 weeks gestation). As described in Kramer (1987b,a), birthweight is determined in part by gestational length. For this reason, we also consider a common measure of intrauterine growth: whether the infant is below the 10th percentile of birthweight for gestational age ("small for gestational age").[31]

We collapse the data into cells based on the first generation's state of birth and year of birth. We then merge each cell with corresponding measures of actual and simulated Medicaid eligibility, and with information on states' economic conditions (state unemployment rate and per capita income), demographic composition (age distribution, marital status, educational attainment and race), safety net generosity, and abortion policies. Additional details about these control variables and sources are provided in Appendix Section D.I.

As discussed in Section III, second generation outcomes were trending differently in treatment and control states in the pre-expansion era. To ensure that our estimates are not contaminated by differential linear pre-trends, we directly remove them using the two-step de-trending method implemented in Goodman-Bacon (2020). For each treated state and outcome,[32] we estimate a linear trend using data only from years prior to the expansion, then extrapolate this estimated trend through all years of data, and subtract the predicted values of each outcome from the observed values. Since control states do not have a clear expansion year, the linear "pre-trend" is estimated using data from 1975–1981, as 1982 is the year of the national trend break in eligibility (Figure 1). The underlying assumption in the event study model is therefore that outcomes would have continued to follow their linear pre-trends in the absence of the expansions. Since linear pre-trends are removed from our analysis, the event study provides information on any remaining non-linear differences across states in the pre-period.

V. Results

A. Eligibility Estimates

Figure 5 provides "first stage" event study estimates based on equation (2), where Panel (a) shows the estimated change in prenatal eligibility and Panel (b) shows the estimated change in simulated prenatal eligibility, both of which are calculated using the CPS, as described in Appendix Section A. Recall that both of these variables capture the percent of women who are *eligible* for Medicaid, rather than the percent who *received* Medicaid. The horizontal axis denotes the number of years before and after the expansion. Event time zero is the first year of the expansion. We omit event time -1, so all estimates are relative to the year before the expansion. The estimates are plotted along with their 95% confidence intervals.

It is immediately clear that after accounting for differences in linear pre-period trends there are no remaining pre-expansion differences between treatment and control states, and that in treated states, the period following the expansion is associated with an abrupt increase

[31]This variable is constructed using two potentially noisy variables, increasing the likelihood that the variable is measured with error.
[32]An exception is that we do not de-trend the NHDS payment data, given the limited number of pre-expansion years.

in both actual and simulated eligibility. This is expected because of the way in which we assign treatment, and is evidence of a strong first stage. Our estimates indicate that four years after the initial expansion, simulated eligibility had increased by 6.6 percentage points. Coefficient and standard error estimates are reported in Appendix Table A.4.

Appendix Figure A.7 shows the results of a variety of additional analyses, with the baseline estimates shown as solid black circles. Alternative specifications include models that eliminate the state-year controls (solid blue triangles), and add region-year fixed effects (solid green square). The estimates barely change across these specifications indicating that the eligibility expansions used for identification were not strongly related to other state-year policy, demographic, or economic changes, or to other changes within regions over time. The results are very similar when we do not de-trend (hollow gray circle), weight by the number of first generation (female) births rather than the number of second generation births (hollow purple square), and when we estimate the model without including the control states (hollow red triangle).[33]

B. Medicaid Coverage at Birth

Although Figure 5 confirms that eligibility increased in the wake of the expansions, we would not expect broader eligibility to translate into better infant health outcomes without a corresponding increase in program take-up. To investigate Medicaid take-up, we estimate the event study model using NHDS data on Medicaid coverage at the time of birth. Figure 6 shows estimates based on equation (2), where the dependent variable is the percent of hospitalized births that were covered by Medicaid. As in Figure 5, the pre-expansion estimates are close to zero, and there is a clear increase in coverage following the initial expansion.[34] Importantly, within four years of the initial expansion, the percent of births covered by Medicaid had increased by 4.6 percentage points, which, when compared to the 6.6 percentage point increase in simulated eligibility, implies a 70% take-up rate among newly eligible mothers. In contrast to eligibility in Figure 5, however, the increase in coverage phases in more slowly, suggesting that it took time for program take-up to fully respond. Given this, we anticipate a similar ramp-up pattern in the health estimates.

C. First Generation Estimates

Having established the event study's validity, we use the same model to reexamine Currie and Gruber's first generation results. Independent of our upcoming second generation analyses, here we make an important contribution to the literature by shedding light on the extent to which previous estimates of the expansions' effects may have been confounded by the presence of other changes that were occurring within states.

[33]In Appendix Figure A.8, we also document that our results are robust to an alternative measure of simulated eligibility that is based on a fixed national sample where the data are pooled across all of our sample years (1975–1988). This version of simulated eligibility ensures that any changes in national population demographics over time are not driving variation in the simulated measure. Appendix Figure A.9 shows that our estimates of the expansions' effects on eligibility are robust to the inclusion of additional control variables that account for factors that might change between the time the mother (first generation) was born and the child (second generation) was born. We discuss these robustness checks in more detail when we examine second generation health outcomes in Section V.D.

[34]Note, however, that as discussed in Section IV, the pre-expansion coefficients in this analysis are estimated using all available data, but necessarily include different treatment states at each event time (i.e. are \unbalanced") due to the limited years of pre-data available for the NHDS. This is not the case for any post-expansion coefficients.

Figure 7 and Appendix Table A.4 present our event study estimates of the expansions' effects on the percent of first generation newborns who were low birthweight. The estimates strongly support Currie and Gruber's original findings. Our pre-period estimates are close to zero and not distinguishable from each other, and there is an abrupt decrease in low birthweight in the treated states, compared to the control states, immediately following treated states' initial expansion. The magnitude of the point estimates grows over the post-expansion period, so that, four years post treatment, the incidence of low birthweight has declined by 0.25 percentage points ($p < 0.001$), or 3.3 percent of the treated states' pre-period mean (Appendix Table A.4).[35] Our confidence intervals include effects as small as a 0.09 percentage point reduction (1.2% when compared to the treated states' pre-expansion mean) and as large as a 0.40 percentage point reduction (5.4%). When coupled with our estimated 6.6 percentage point increase in prenatal eligibility, the results suggest that a 10 percentage point increase in eligibility reduces the incidence of low birthweight in the first generation by 0.37 percentage points, or about 5 percent.[36] As a point of comparison, Currie and Gruber estimate that a 10 percentage point increase in eligibility led to a 2.6 percent decline in low birthweight, which falls squarely within our 95% confidence interval.

To interpret these effects as causal, it must be the case that the timing of the expansions was unrelated to other factors that affect infant health. For example, one might be concerned that a state's decision to expand was influenced by the state of its economy. To check for this, we examine how our estimates change under the same set of alternative specifications described in Section V.A, which include models that eliminate the state-year controls or add region-year fixed effects, a version of the baseline model that does not remove linear pre-trends, and a version of the model that does not include the control states. Appendix Figure A.10 Panel (a) shows that estimates are similar across the specifications. The coefficient estimates during the post-period are smaller when we do not account for pre-existing linear trends, supporting the possibility that control states' early adoption of the expansion options may have already altered their infant health trajectories prior to the 1980s. Unsurprisingly, given the reduced sample size, confidence intervals are larger when we estimate the model without

[35]Recall that for ease of presentation, we multiply all variables with values between 0 and 1 by 100.

[36]Note that the baseline mean among women who gained eligibility may be substantially higher than the population average because those who gained eligibility were extremely disadvantaged. For example, among children living in the lowest income households in the 1981 National Health Interview Survey (under $3000 in annual income), the incidence of low birth weight was 17.4 percent, which is more than twice as large as the baseline incidence of low birthweight in the treated states (7.44 percent, see Appendix Table A.4). Even so, the effect sizes contained within the 95% confidence interval around the low birthweight estimate are sufficiently large to suggest that they may not arise from increases in prenatal care alone. A combination of pathways may contribute to the effects. For example, prenatal care may put mothers in touch with other social services: three quarters of women receiving Medicaid funded prenatal care in 1988 reported that they received WIC during their pregnancy (see Table 1 in Miller and Wherry, 2019), and forty percent reported that they learned about WIC through a doctor, nurse or health provider. Related to this, it is possible that prenatal counseling changes nutrition or substance use behaviors sufficiently to affect birth outcomes. Again, as discussed in Miller and Wherry (2019), 90% of women who obtained prenatal care through Medicaid received nutritional counseling, 95% were counseled to take vitamin supplements, 69% received counseling with respect to smoking, and 61% received counseling with respect to alcohol. These types of instructions may be more common among low-income women, or they may have a stronger impact on low-income women relative to the average woman who gets a prenatal care visit. Another potential pathway is through an increase in families' financial resources. Based on the following website, https://www.cryo-cell.com/blog/april-2017/when-childbirth-cost-100-dollars, out-of-pocket expenditures for an uninsured birth during this time period were about $1500 ($6500 in 2012$s). Multiple studies document that cash and near-cash transfers improve birth outcomes among infants born to low-income women (e.g. Kehrer and Wolin, 1979; Almond, Hoynes and Schanzenbach, 2011; Hoynes, Miller and Simon, 2015; East, 2020). Another possible pathway is that insurance may reduce maternal stress. A significant body of research links prenatal maternal stress to adverse birth outcomes, including low birthweight (Persson and Rossin-Slater, 2018). Moreover, poor urban women from minority backgrounds are twice as likely as middle-class women to meet diagnostic criteria for major and minor depression during pregnancy and the postpartum period (Grote et al., 2010). Other potential mechanisms include changes in maternal employment, or spillover effects onto non-participants.

including the control states. Even with the loss of precision that accompanies some of these checks, however, the pattern of results remains very similar.

Taken as a whole, our event study analysis bolsters the credibility of Currie and Gruber's landmark findings. The new research design also provides insights into the time path of effects, specifically, that the expansions' impact on the first generation's health took time to fully ramp up. A likely explanation is that it took time for pregnant women to learn about their eligibility and enroll in the program. Moreover, although improvements in the first generation's birthweight are not a necessary pre-condition for the presence of second generation effects, the results presented in Figure 7 allude to mothers' health at birth as a potential conduit, as previous studies have documented intergenerational linkages in low birthweight (e.g. Currie and Moretti, 2007).[37]

D. Second Generation Estimates

Figures 8 and 9 show the relationship between mothers' *in utero* Medicaid access and second generation birthweight and gestational length outcomes, respectively. Focusing first on birthweight (Panel (a) of Figure 8), we see evidence of an increase following the expansions. The point estimates grow over time, reaching 4.7 grams four years after expansion (see Appendix Table A.4). The 95% confidence interval allows us to rule out effects of more than 8.2 grams and less than 1.2 grams. Although the impacts on some of the other infant health outcomes are less precisely estimated, we see largely similar patterns, particularly at the lower end of the birthweight distribution (low birthweight and very low birthweight).[38] We also see evidence of a decrease in very preterm births in Panel (c) of Figure 9. As discussed in Section IV, low birthweight and prematurity are of particular interest because they are closely linked to other early and later life health and cognitive outcomes. Also, low birthweight and preterm births are associated with maternal health characteristics that are known to have been improved by the prenatal expansions (Institute of Medicine, 2017; Miller and Wherry, 2019).[39] We also see some evidence of improvements in small for gestational age in Panel (d) of Figure 9, which is indicative of gains in intrauterine growth.[40]

As with the first generation estimates, Figure 10 shows that the second generation results are, for the most part, similar when we employ the specification checks described in Section V.A. We only show these for the outcomes that seemed affected by Medicaid in the baseline

[37]We also consider the expansions' effects on average birthweight and the incidence of very low birthweight, which are not examined in the original Currie and Gruber paper. Event study estimates and robustness analyses are provided in Panels (b) and (c) of Appendix Figure A.10 and Appendix Table A.6. The estimated effects are suggestive of expansion related declines in very low birthweight. We do not examine changes in outcomes related to gestational length because prior to 1981 the birth certificate records have a high rate of missing values for this variable.

[38]The 95% confidence intervals around the year four point estimates for low birthweight (very low birthweight) allow us to rule out declines of more than 0.27 (0.14) percentage points and gains of more than 0.03 (declines of less than 0.02) compared to baseline means of 7.52 and 1.45.

[39]For example, low birthweight has been linked to chronic hypertension, pre-pregnancy diabetes, and maternal obesity (Institute of Medicine, US).

[40]The differences in outcomes between treated and control states are evident even in the raw means. In Appendix Figure A.11 we plot mean outcomes by event time, across treatment and control groups (solid black and orange lines). The trends are very similar prior to the expansions, and then deviate for the outcomes for which we find (at least suggestive) evidence of effects – birthweight, low birthweight, very low birthweight, very preterm, and small for gestational age. These same patterns persist after we residualize with the state-year controls in our baseline models – shown in the gray and orange dashed lines.

results and for gestational length and preterm, the robustness checks are shown in Appendix Tables A.10–A.11. Our second generation estimates often have large confidence intervals associated with them, but across all of these checks the patterns remain very stable, even if not always statistically different from zero at conventional significance levels. Excluding the state-year covariates, or adding region-year fixed effects does not change the results, which again demonstrates that these large expansions were not correlated in a meaningful way with state-level changes in demographics, policies, or economic conditions, or with time-varying changes within regions. The figure also documents similar patterns when we use different weights (mother rather than child cohort size). We see larger deviations from the baseline estimates when we do not account for pre-existing differences in state trends (gray circles in the figure). When we remove control states from our sample, the estimated intergenerational effects often become larger but more imprecise.[41]

Appendix Figure A.12 shows the sensitivity of the estimates to additional sets of state-year controls. During the 1980s and 1990s, Medicaid coverage was also extended to older and higher income children.If the prenatal expansions we focus on are correlated with these other childhood expansions, then we might be erroneously attributing the observed health improvements among second generation infants to their mothers' *in utero* coverage when, in fact, the improvements result from mothers' increased Medicaid access in later childhood. Most studies estimate the impact of expanding children's access to Medicaid using a single index that aggregates eligibility across the length of childhood, without evaluating linkages between the *in utero* and later childhood expansions. In Appendix Figure A.13, we use the same event study framework to directly estimate the relationship between the prenatal expansions and the childhood expansions, and find no evidence that they are correlated.[42] Therefore, it is unsurprising that our second generation estimates are very similar when we add measures of later childhood eligibility to our baseline model (shown in the solid purple triangles in Appendix Figure A.12).

Appendix Figure A.12 also documents that the results are similar when we control for mother's own eligibility during adulthood (open green circles), and when we control for the second generation's own prenatal eligibility (open pink triangles). They are also unaffected when we include the same set of state-year controls listed in Section III augmented with additional controls capturing the generosity of welfare and access to family planning services, but measured in the *child's* year of birth rather than the mother's year of birth (shown in the solid blue squares).[43]

A remaining concern with event study estimates is that they may be biased in the presence of heterogeneous treatment effects across expansion timing groups (Sun and Abraham, 2020). To alleviate this concern, we implement a new estimation method proposed by Callaway and Sant'Anna (2020) that is robust to heterogeneous treatment effects across groups. Essentially, this approach avoids using earlier treated units as controls for later treated units

[41]We report the coefficient and standard error estimates from all of these robustness checks and for each outcome in Appendix Tables A.7–A.13.
[42]We cannot rule out that changes in prenatal eligibility affected childhood take-up of Medicaid, however.
[43]See the discussion in Appendix Section A for detailed information about how we construct these other eligibility variables. Additional details on the other control variables are found in Appendix Section D.I.

(which can lead to biased estimates). The results are shown in Appendix Figure A.14.[44] Across all outcomes, the results are very similar to our baseline estimates.[45] This reassures us that the baseline event study results are robust to possible treatment effect heterogeneity.

As an alternative to our main event study model, we also evaluate the impact of the eligibility expansions using a synthetic control approach.[46] This method constructs a weighted average of untreated states to serve as a synthetic control for each treated state, with weights selected to minimize the pre-treatment differences in the outcome and control variables across the treated state and synthetic control units, as described in Abadie, Diamond and Hainmueller (2010). We select weights following the Abadie, Diamond and Hainmueller (2010) method to obtain a synthetic control unit for each treated state, then stack the observations for each treated state and its synthetic control, and estimate an event study model, following the methods described in Kleven (2021). The results of this exercise are reported in Appendix Figure A.15. While in some cases the synthetic control approach produces smaller point estimates, the overall patterns remain very similar.

Next, we verify that our results are not sensitive to our sample selection criteria. Appendix Figure A.16 shows estimates based on a second generation sample of higher parity births. This sample generates similar birthweight and low birthweight estimates as the main sample, but we no longer observe improvements in the incidence of very preterm or small for gestational-age. Tabulations we ran using pre-period data indicate that these outcomes are less common among higher order births, which may explain the difference. Appendix Figure A.17 shows what happens when we relax our baseline maternal age restriction from ages 15–28 to include births to older mothers (recall that this produces a sample that is unbalanced in maternal age). Again, the estimates are very similar to those produced by our main sample.

Finally, we repeat our analysis including only children of foreign-born mothers, who are excluded in the main analysis, and who we assume were unaffected by the first generation prenatal expansions, since the first generation was not born in the U.S. This placebo check is another way of addressing concerns about unexplained cohort by state specific changes in infant health that are correlated with, but separate from, exposure to the prenatal expansions. We use the state of residence at the time of the child's birth to assign policy and control variables. The results from this analysis are found in Appendix Figure A.18.[47] As expected, we do not detect any changes in infant health that correspond with the expansions' timing.

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[44]We implement the Callaway and Sant'Anna method using R code they provided. We use the same weighting as in our main analysis (by size of birth cohort) and cluster by state. To simplify comparisons across estimators, we do not include control variables in this model. Note that our application of this method continues to use the control states as a comparison group and that some of these states may have experienced similar expansions in eligibility (i.e. "treatment") prior to our study period.

[45]Specifically, with this method, we find that four years after the expansion average birthweight had increased by 4.2 grams, low birthweight had decreased by 0.09 percentage points, very low birthweight decreased by 0.08 percentage points, very preterm decreased by 0.05 percentage points, and small-for-gestation-age decreased by 0.14 percentage points.

[46]This method offers an alternative way to account for pre-trends; when using this method we do not de-trend the outcome variables and instead use the matching procedure to find control states that match the treated states' pre-trends. Appendix Section D.IV contains further details on our implementation of this method.

[47]We note that the sample of births to foreign-born women is much smaller than the sample of births to U.S.-born women. For example, in 1980 there are an average of 19,000 births per state to US-born women, compared to 4,500 births to foreign-born women. Not surprisingly, the estimates for the sample of births to foreign-born women are accompanied by larger confident intervals.

Magnitudes—Our results indicate that the benefits of prenatal Medicaid spill over onto later offspring. Taken at face value, the point estimate for average birthweight suggests that the 6.6 percentage point increase in *in utero* eligibility generated by the 1980s expansions increased the second generation's average birthweight by a statistically significant 4.7 grams, which corresponds to an increase per newly eligible woman of about 71 grams (2 percent relative to the mean).[48] This is a bit less than half of the observed gap in average birthweight between mothers with vs. without a high school degree at the beginning of our sample period. Comparable estimates for low birthweight and very low birthweight are 1.8 percentage points and 1.2 percentage points. Of course, the confidence intervals around these estimates include both much smaller and much larger effect sizes.

Not everyone in the first generation who became eligible for Medicaid under the expansions actually received coverage. We therefore interpret the estimated effects per newly eligible woman as "intent to treat" estimates, where treatment is defined as enrollment in the program. Assuming that the benefits of Medicaid eligibility accrue only to those who were actually enrolled *in utero*, and that there were no effects on those who were eligible but did not enroll, we can obtain the treatment effect of Medicaid enrollment by dividing the estimated expansion effects by the estimated increase in Medicaid coverage in Appendix Table A.4 (4.6 percentage points in the fourth year of expansion). Using the point estimates above, this calculation implies that among the offspring of women whose pregnant mothers enrolled in Medicaid, average birthweight increased by a little over 100 grams.[49] Similarly, the treatment effect estimates for low birthweight and very low birthweight are 2.6 and 1.8 percentage points, respectively. While some of these treatment effect estimates are large in comparison to our baseline means, it is important to recall that the early expansions targeted very poor pregnant women for whom the incidence of poor birth outcomes was substantially higher than in the full population.[50]

We can also compare our estimates of the expansions' effects across generations. We note that the first generation receives a direct and clearly defined treatment, which is access to Medicaid coverage during the *in utero* period. In contrast, the second generation's treatment is the bundle of biological and economic outcomes experienced by the mother as a result of her access to Medicaid, and subsequently passed on to her infant. It is therefore not clear *ex ante* which generation should experience larger effects, or whether effects must be present at birth in the first generation in order for the second generation to be plausibly affected. Comparing the point estimates in columns 4 and 6 in Appendix Table A.4 we see that the decline in low birthweight experienced by the second generation is about 48% as large as the effect in the first generation. This is consistent with Currie and Moretti (2007), who find that the probability of being low birthweight is nearly 50 percent higher among children whose mothers were themselves born below the low birthweight threshold.[51]

[48]Note that this calculation relies on the assumption that there are no spillover effects of the expansions onto women who did not gain eligibility. This assumption may be violated if, for example, those who gained prenatal eligibility shared health information with friends or neighbors who did not gain eligibility, or if the expansions induced changes in providers' behavior or policies that affected communities more broadly. If such spillovers exist, these treatment effect calculations may be biased upwards.

[49]We obtain this estimate by dividing 4.8 grams by the 4.6 percentage point increase in Medicaid coverage.

[50]As noted earlier, our calculations indicate that the incidence of low birthweight among children living in the lowest income households in the 1981 NHIS was more than twice that of the full population.

Finally, keeping in mind that there are wide confidence intervals around the estimates, we consider possible implications for the program's return on investment. Currie and Gruber (1996b) report that the targeted expansions increased Medicaid spending per eligible woman by $450 (this cost is estimated in 1981 and we inflate to 2011 dollars). We compare this with the medical cost associated with lower birthweight averted in the second generation. Based on estimates provided in Almond, Chay and Lee (2005), we calculate that each additional gram of birthweight reduces hospital costs by $8.29.[52] Our estimates suggest that first generation eligibility increases average second generation birthweight by 71 grams, resulting in about $589 in savings per first generation woman made eligible. Given the amount of time that has passed, we discount these cost savings back to the period of the initial Medicaid outlays (1981), using the discount rate recommended by the Department of Commerce for life-cycle studies (3%, see Lavappa and Kneifel, 2016). For this calculation, we assume cost savings accrue equally across all second generation birth years.[53] This calculation suggests that the expansions generated average cost savings of $287 per newly eligible woman, or more than 60% of the cost of the initial investment.[54] Even if we use a more conservative birthweight estimate from the bottom of the 95% confidence interval, we calculate an average cost savings of more than 16% of the cost of the initial investment. Notably, this calculation is focused solely on benefits that translate to the second generation, and ignores any improvements in the first generation's health and human capital. The calculation also ignores medical cost savings that result from any second generation health improvements in later life that are tied to better health at birth, as well as improvements in later life earnings (and tax revenues), which have also been tied to birthweight (e.g. Black, Devereux and Salvanes, 2007; Bharadwaj, Lundborg and Rooth, 2018).

It may also be reasonable to expect that health benefits accruing to the first two generations will continue to be passed on in the future, albeit incompletely. We could therefore model the intergenerational benefits of the Medicaid program as an annuity that pays off once each generation. We assume that half of the benefit to the mother's generation is transmitted to the child (as we observe for low birthweight), and then only half of that received benefit to the child's offspring, and so forth, such that the benefit of an investment decays exponentially across generations. Applying a 3% discount rate, we calculate that accounting for the intergenerational aspects of the program's effects in this way results in estimated benefits that are more than 30% higher than what would be observed if the analysis focused only on the first generation.[55]

[51]Other studies have estimated smaller intergenerational birthweight correlations (Black, Devereux and Salvanes, 2007; Royer, 2009) but importantly, Currie and Moretti (2007) find that poverty increases the transmission of low birthweight from mother to child.

[52]Almond, Chay and Lee (2005) estimate that an additional gram of birthweight reduces hospital charges by $22 for infants in the 2000–2100 gram range in 2000 dollars, or $28.60 in 2011 dollars. Since hospital charges do not accurately reflect hospital resource costs (due to markups), we deflate this estimate using national charge-to-cost ratios (Bai and Anderson, 2015) to arrive at $8.29 per gram.

[53]We estimate cost savings for each second generation birth year from 1995 to 2017 since the first expansions occurred in 1980 and first generation mothers born in that year reached age 15 in 1995.

[54]Using the discount rate recommended by the Office of Management and Budget of 0.5% instead, the discounted value of the benefits is $520 (U.S. Office of Management and Budget, 2016). Details of these calculations are in the Appendix D.V.

[55]Specifically, we assume the Medicaid investment generates a payoff that falls by 50% in value and is paid out every 25 years. The present value of such a payoff of size X would be:

Additional Outcomes—We extend our analysis of the intergenerational effects of the Medicaid expansions by looking at additional health indicators, including the presence of congenital anomalies, whether the birth was associated with any abnormal conditions, and the sex ratio at birth.[56] Congenital anomalies are more common among low birthweight and preterm infants, and have been linked to environmental factors that are often associated with low income, including poorer access to nutritious foods, health care and screening (World Health Organization, 2012). Similarly, some abnormal conditions such as NICU admission or surfactant replacement therapy could be indicative of underlying health conditions. Our examination of the sex ratio is motivated by the Trivers and Willard (1973) hypothesis, which suggests that, because male fetuses are more sensitive to negative health environments than female fetuses, improvements in maternal health might disproportionately reduce the number of *in utero* losses that are male. The results of these analyses are provided in Appendix Figure A.19 and lend further support to the hypothesis that the expansions' health benefits extended beyond the treated generation. Although there is no evidence that the expansions affected abnormal conditions at birth, the patterns of estimates for congenital anomalies and the sex ratio are similar to our main results.

E. Mechanisms

Changes in Fertility or Maternal Characteristics—What are the mechanisms generating these intergenerational spillovers? We investigate potential pathways using additional information provided in the natality files. First, we consider changes in the first generation's fertility. The same (or related) biological processes that lead to improvements in the first generation's health may have also affected the first generation's fecundity.[57] We also consider whether the Medicaid expansions led to changes in the composition of women giving birth: if children are a normal good, then Medicaid induced increases in the first generation's earnings might also lead to increases in the desired number of children. On the other hand, improved economic opportunities might also lead to delays in childbearing (Brown, Kowalski and Lurie, 2020).[58]

We explore these potential mechanisms in Appendix Figure A20. We estimate regressions similar to equation (2), replacing the dependent variable with measures of fertility or maternal characteristics (age, educational attainment, marital status and race).[59] We find

$$\$X \times \sum_{n=0}^{\infty} \left(\frac{0.5}{(1+r)^{25}}\right)^{n}.$$

Since $\frac{0.5}{(1+r)^{25}} < 1$ we can apply the rules for geometric series. Plugging in $r = 0.03$ for a 3% discount rate, this simplifies to $\$X \times 1.31$.

[56]Examples of abnormal conditions include the provision of assisted ventilation, NICU admission, surfactant replacement therapy, antibiotics, seizures, or significant birth injury.

[57]We are not able to measure total fertility because we cannot observe most of our first generation cohorts throughout their childbearing years.

[58]Brown, Kowalski and Lurie (2020) estimate that each additional year of Medicaid eligibility from birth to age 18 is associated with a 1.2 percentage point reduction in the probability that a woman has her first child by age 28, but the authors do not examine the effect of *in utero* eligibility on fertility.

no evidence that the Medicaid expansions led to changes in fertility behavior; neither the likelihood of having a first birth, nor the age at first birth, are affected by the expansions. In addition to their statistical insignificance, the point estimates are very small (four years after the expansions, there is a 0.3% decrease in the first birth rate and a 0.1% increase in age at first birth, relative to the baseline means).

We do see some evidence of changes in the characteristics of mothers, particularly with respect to race. The expansions are associated with an increase in the percent of births to white mothers (0.7% relative to the pre-treatment mean) and a decrease in the percent of births to Black mothers (3% relative to the pre-treatment mean). Since white infants tend to be healthier on average than Black infants, we consider whether the shift towards white births explains the expansions' apparent effects on the second generation's health. To do this, we use the estimated effects four years after the primary expansion. Appendix Table A5 shows that by year four the percent of births to white mothers had increased by 0.54 percentage points, with a roughly similar decrease in the percent of births to Black mothers.[60] A back-of-the-envelope calculation based on this compositional shift suggests that selection can explain, at most, about 30 percent of the overall effect of the Medicaid expansion on average birthweight.[61] In addition, we re-estimate our main model directly controlling for mother's education, marital status, and race. The results, shown in Appendix Figure A22, are very similar to our baseline estimates. This is a strong indication that the observed health improvements are not driven by changes in the composition of mothers giving birth.[62]

Maternal Health and Behaviors—Finally, we consider the role of maternal health and maternal health behaviors. The results of these analyses should be interpreted cautiously, as health conditions reported on birth certificates are relatively limited and known to be under-reported (Lain et al., 2012). Nevertheless, Appendix Figure A23 hints at the presence of biological pathways. We see suggestive evidence that the prenatal expansions reduced the incidence of medical risk factors, which include diabetes, chronic hypertension, pregnancy-related hypertension, and eclampsia, among first generation mothers. Consistent with first generation studies that document a positive link between early-life Medicaid and later-life metabolic health (e.g. Boudreaux, Golberstein and McAlpine, 2016; Thompson, 2017; Miller and Wherry, 2019), the decline in maternal medical risk is driven by a reduction in pregnancy-related hypertension. As described in Section I, an improvement in this outcome

[59]Three of the outcomes analyzed in this section and the next (Section V.E) – mother's educational attainment, prenatal care utilization, and race – were affected by the introduction of the 2003 revision of the U.S. Standard Certificate of Live Birth. This revised version replaced the 1989 version that was in use during the remainder of the period covered by our analyses. See Appendix Section D for more details on how we account for this change in our analyses.

[60]We appreciate that by year four the estimated effects on racial composition are no longer statistically significant, but we use these estimates, which are very similar to the estimates in year 3, so as to be consistent with the focal event year used in the rest of the paper.

[61]If we apply these estimates to the race specific averages for birthweight (3329.6 for children of white mothers and 3080.5 grams for children of Black mothers), we predict an increase in average birthweight of 1.24 grams due solely to the expansion's effect on the racial composition of births (i.e. 3329.6*.005 – 3080.5*.005). This estimate is 26% of the program's estimated increase in the second generation's average birthweight of 4.7 grams in year 4 of the expansion. We also explore whether the observed changes in infant health persist when we restrict the sample of second generation births to white mothers (78%) of the sample. The results, shown in Appendix Figure A21, are very similar to those produced by the full baseline sample, indicating that selection based solely on race cannot explain all of our findings. Unfortunately, small sample sizes impede further subgroup analyses.

[62]It is also possible that marital matching changed. Unfortunately, there is limited information about fathers' characteristics provided on birth certificates (age, race, and Hispanic origin), which makes a direct examination of this hypothesis infeasible.

would be expected to reduce the likelihood of giving birth to a low birthweight or preterm infant.[63]

VI. Conclusion

Social scientists have long known that children's outcomes are predicted by their parents' socioeconomic status (e.g. Solon, 1992; Mazumder, 2005), and recent research suggests that health plays an important role in the intergenerational transmission process (Halliday, Mazumder and Wong, 2020, 2021). The extent to which the impacts of policy-driven health investments spill over onto later generations has been largely unexplored, however. Understanding such effects is important, both because it sheds light on how economic and health status are conferred across generations, and because it provides insights on the extent to which generationally persistent disadvantage can be ameliorated by policy choices. Furthermore, if safety net programs' benefits extend beyond treated generations, then those benefits need to be accounted for in order to accurately assess both net-costs to the government and their total value to recipients (direct and indirect). Neglecting policies' multi-generational effects could result in dramatic undervaluation.

Any exercise that seeks to quantify a policy's multigenerational effects will inherently face substantial uncertainty: multigenerational outcomes are measured generations away from the initial treatment, and the individuals who were directly affected by a policy early in life have been subject to a multitude of economic, social, educational and cultural exposures before having children themselves. Furthermore, the factors that generate maternal health accrue over a lifetime, and the decision whether and when to have children is multifaceted and varied. Given this, disentangling the exact mechanism(s) is practically very difficult. Nevertheless, such explorations are critical to the accurate assessment of interventions' benefits, and our results are an important contribution to this effort.

In this paper, we provide new evidence that expanding health related services to low-income pregnant women has persistent impacts on later generations' health. We introduce a new event study approach to analyze the effects of the 1980s Medicaid expansions that exploits large, discrete jumps in state eligibility resulting from a combination of state policy choices and federal mandates. Using this new approach, we confirm previous studies' findings that the targeted expansions generated improvements in the first generation's birth outcomes. Then we document that this treated generation went on to give birth to healthier offspring. Despite the large confidence intervals accompanying some of our estimates, they all point to a consistent pattern of improvement in second generation health. Moreover, these results do not appear to be driven by changes in the first generation's fertility behavior or selection into childbearing. Instead, the positive spillovers likely reflect improvements in the first generation's health and economic outcomes prior to giving birth. In particular, our analyses

[63]Surprisingly, we see some evidence that Medicaid eligibility is associated with reductions in the use of prenatal care during pregnancy. If anything, this would lead us to expect worse infant health outcomes, and suggests that the effects would be larger in the absence of this association. We do not examine changes in alcohol or tobacco use due to limited availability on the birth certificate records for all states and years. Alcohol use is only available through 2006. Information on tobacco use is available through 2008. This would only allow us to observe women through age 18 and age 20, respectively, if we were to balance the sample on mothers' age at birth.

suggest that Medicaid-induced health gains among first generation mothers may be an important underlying mechanism.

Our study offers a new perspective on health inequalities and the potential role for government intervention. Generational persistence in the impacts of early life environments suggest that historical differences in fetal health conditions between advantaged and disadvantaged groups may undermine contemporaneous efforts to close health and economic gaps. At the same time, our results indicate that early life health investments have payoffs that extend well beyond those that social policymakers usually consider. It is notable that Medicaid's second generation effects are observed among cohorts who were born during roughly the same time frame for which recent studies by Aizer and Currie (2014) and Currie and Schwandt (2016a,b) document large improvements in child health and declining health inequality. Investigating a more complete range of program benefits to later generations is an important goal of future work, and is critical in light of increasing debates about the efficacy of the U.S. safety net.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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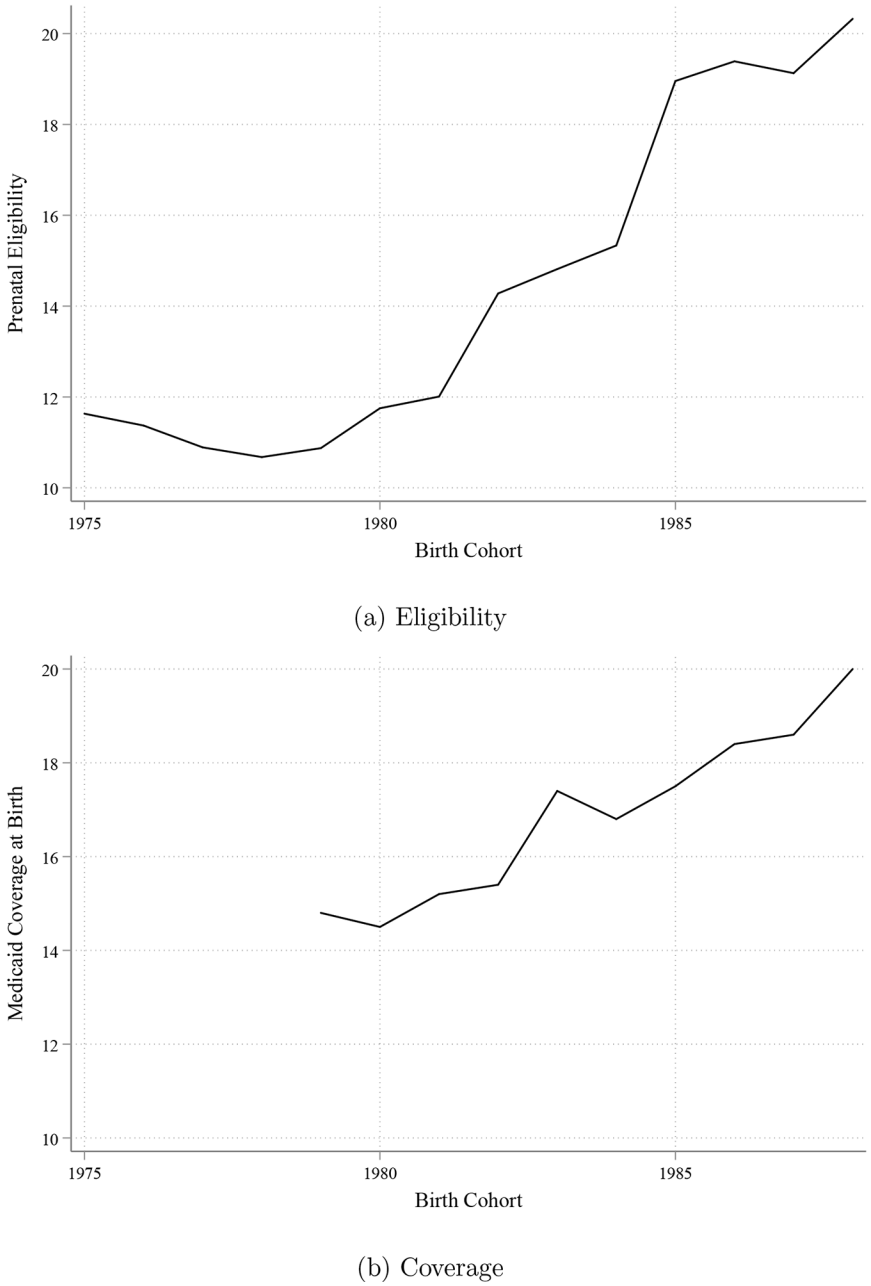


Figure 1.:

Trends in Prenatal Medicaid Eligibility and Medicaid Coverage at Birth, 1975 to 1988

Note: Authors' calculation from the Current Population Survey and Medicaid eligibility rules and from the National Hospital Discharge Survey. For eligibility estimates, state averages are weighted using the number of births in each state-year cohort. For coverage estimates, sample weights are applied. All estimates are reported in percents. See text for further details.

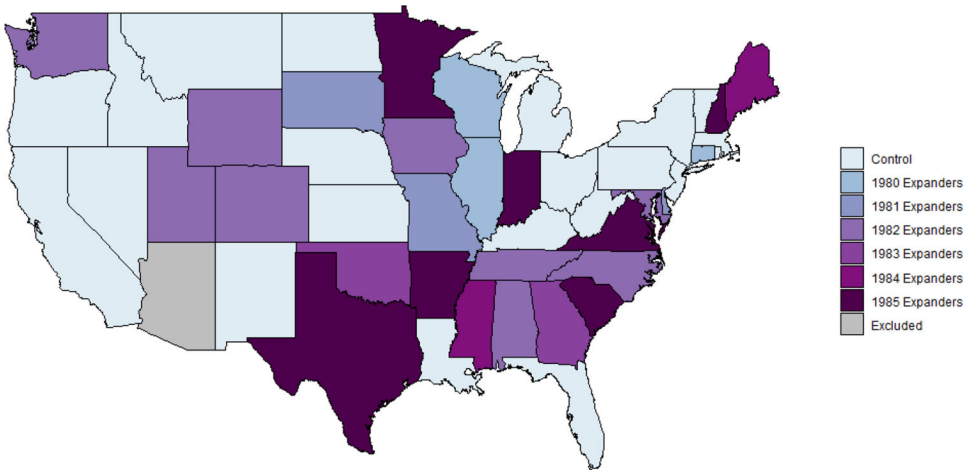
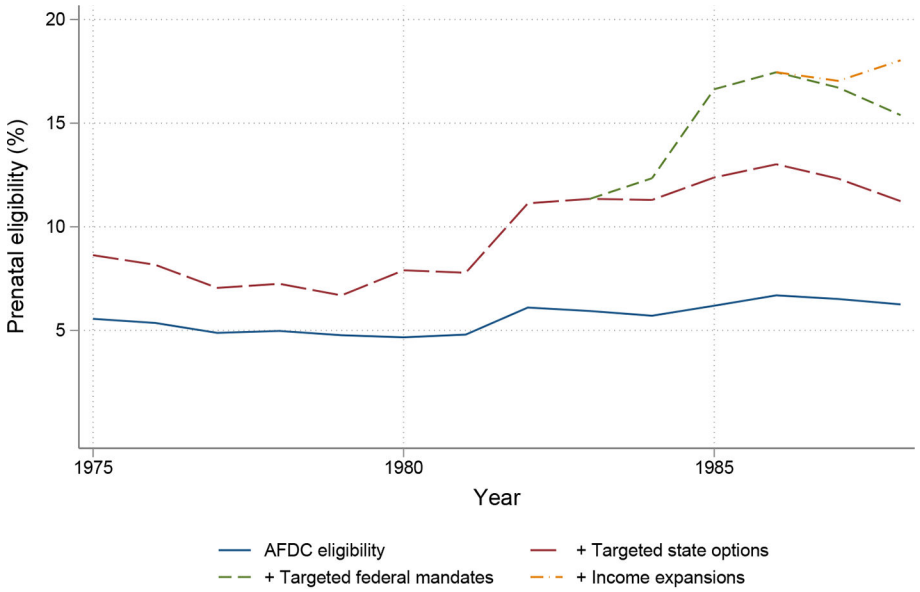
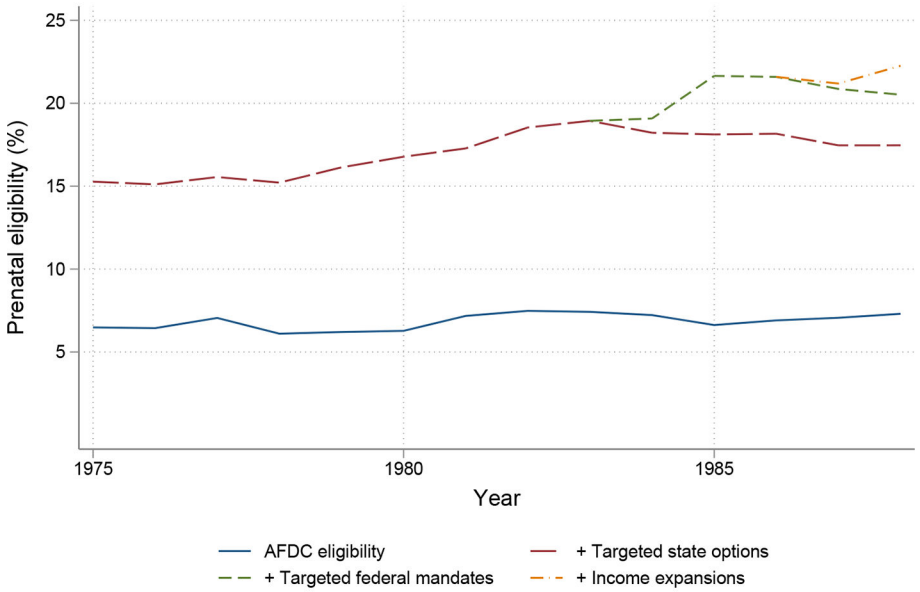


Figure 2.:
Treatment and Control States
Note: See text for further details. Not pictured is Alaska (1982 Expander) and Hawaii (Control). Arizona is omitted due to the late start date of their Medicaid program.



(a) Treated States



(b) Control States

Figure 3.:

Prenatal Eligibility by Source and Treatment Status, 1975–1988

Note: Authors' calculation from the Current Population Survey and Medicaid eligibility rules. Each line represents Medicaid eligibility through each of the state options and federal mandates. Specifically, we construct this figure by calculating the percent of women who would be eligible under each pathway in an additive fashion that reflects the order of eligibility pathways from lowest to highest pathway on the figure. These pathways are discussed in more detail in Appendix Section A. All estimates are reported in percents.

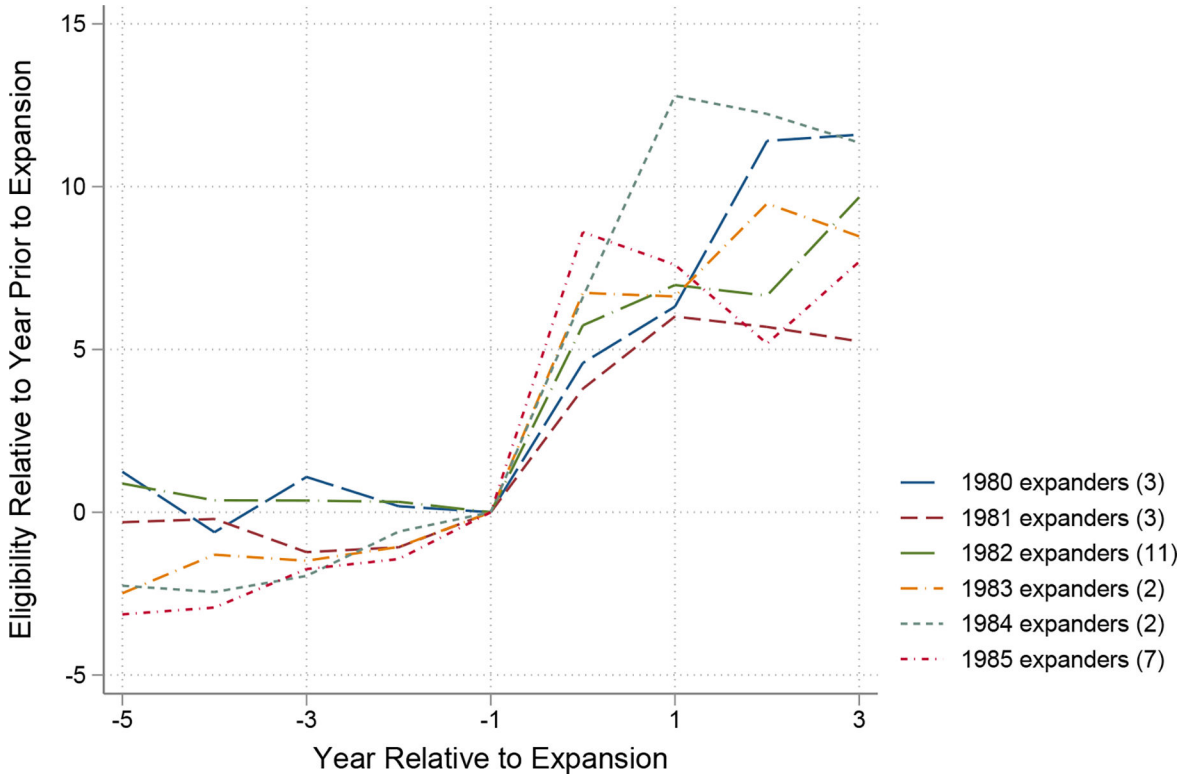
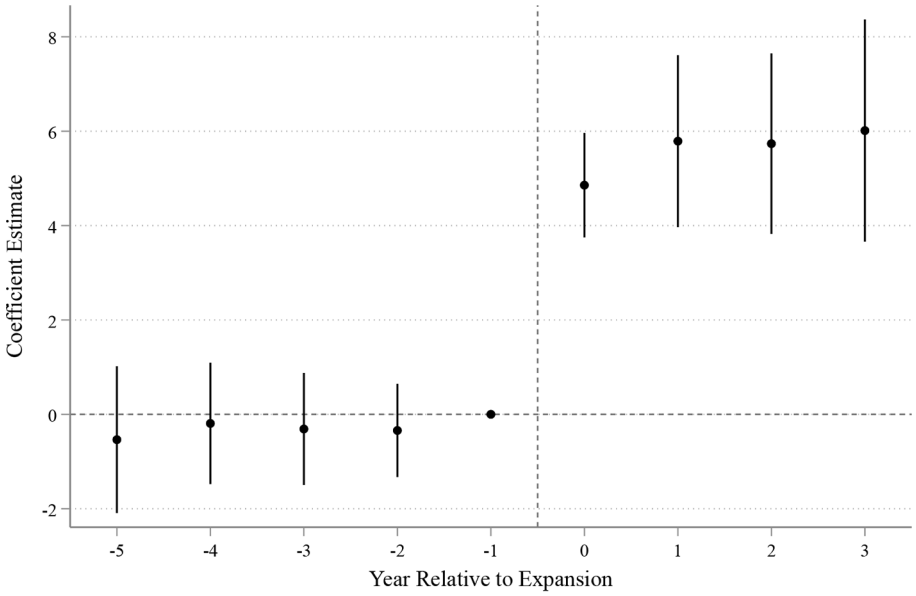


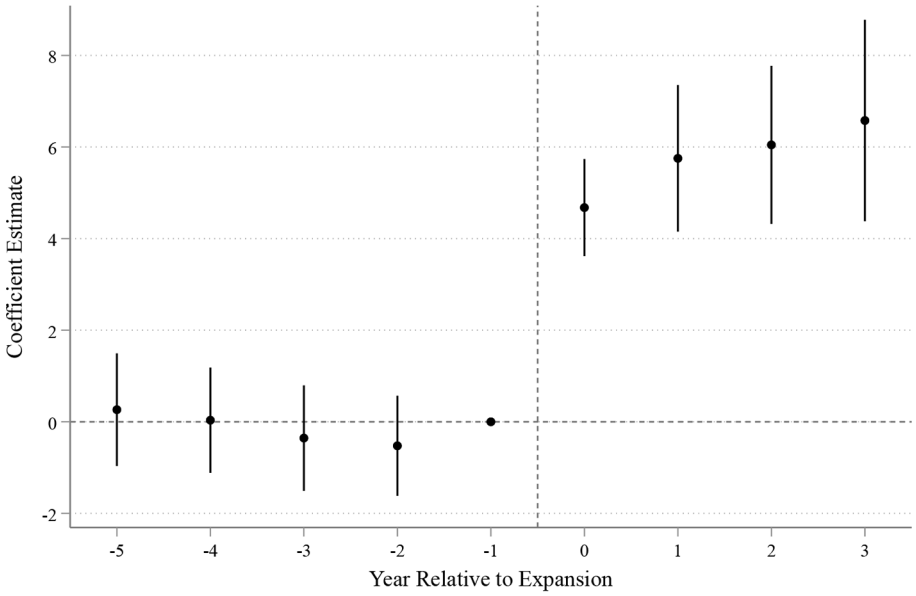
Figure 4.:

Trends in Simulated Prenatal Medicaid Eligibility Relative to Year Prior to Large Expansion

Note: Authors' calculation from the Current Population Survey and Medicaid eligibility rules. Difference in simulated eligibility from the year prior to the large expansion in each group of treated states is depicted. The number of treated states in each group is listed in parenthesis. All estimates are reported in percents. See text for further details.



(a) Actual Eligibility



(b) Simulated Eligibility

Figure 5.:
Event Study Coefficients for Prenatal Eligibility

Note: Coefficient estimates are reported in percentage points. Estimated for first-born infants of mothers born in 1975–1988 and ages 15–28. Pre-period trend is estimated and removed from all observations for each state prior to the event study estimation. For treated states, this is estimated using all pre-period years for each state. For control states, we use the period 1975–1981 to estimate this trend. Regressions are weighted by second generation birth cohort size and include mother's state of birth and mother's year of birth fixed

effects and controls for state-year variables (unemployment rate, personal income per capita, maximum welfare benefit for a family of 4, indicators for state parental consent and notification laws and state Medicaid restrictions for abortion, and demographic controls for each state and year). Standard errors are clustered by mother's state of birth.

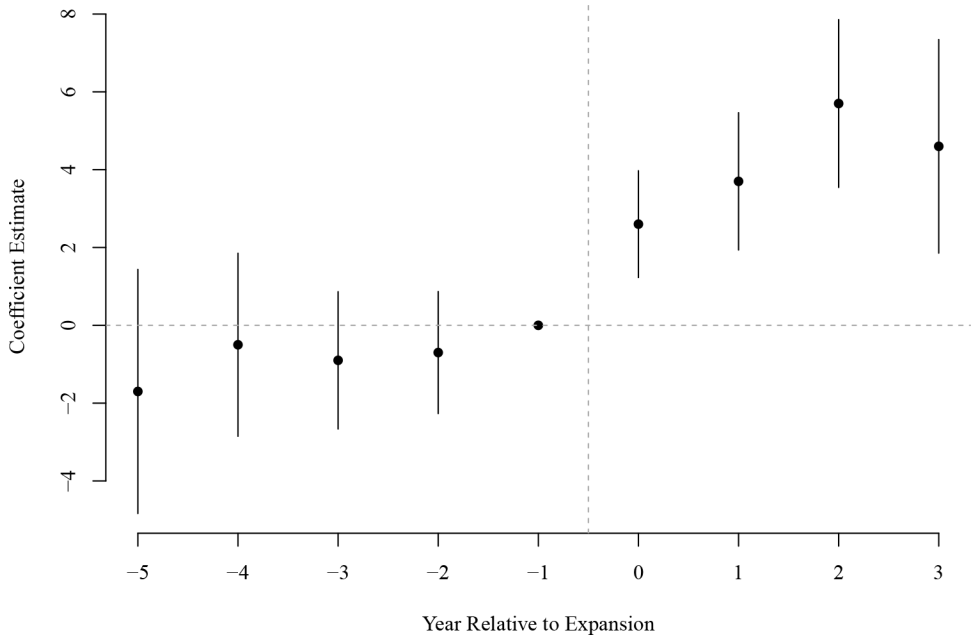


Figure 6.:

Event Study for Medicaid Coverage Among Labor and Delivery Hospital Discharges

Note: Coefficient estimates are reported in percentage points. Estimated for hospital discharges for labor and delivery between 1979–1988. Regressions are weighted by NHDS sample weights and include state of birth and year of birth fixed effects and controls for state-year variables (unemployment rate, personal income per capita, maximum welfare benefit for a family of 4, indicators for state parental consent and notification laws and state Medicaid restrictions for abortion, and demographic controls for each state and year). Standard errors are clustered by state.

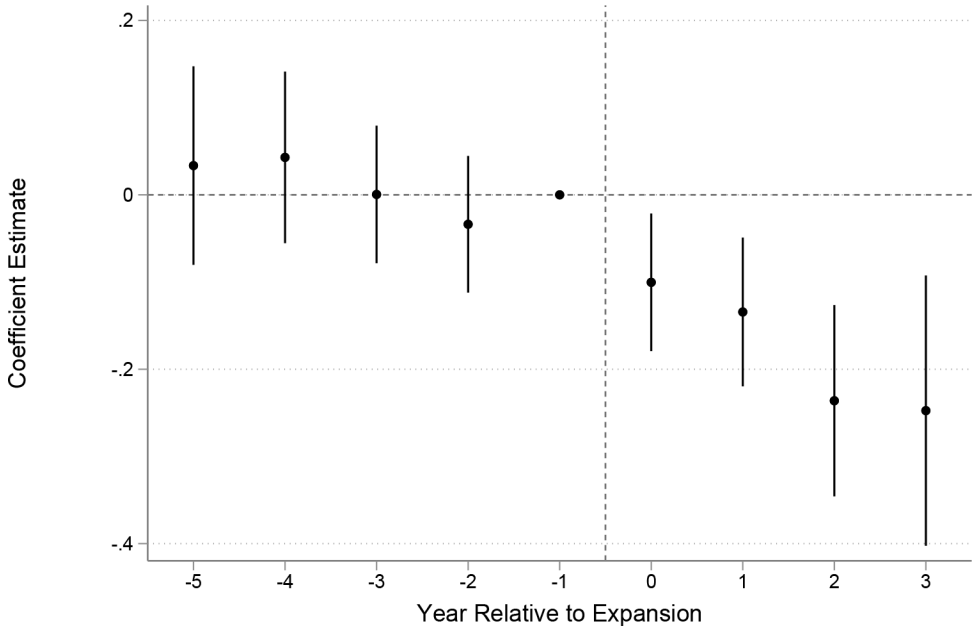
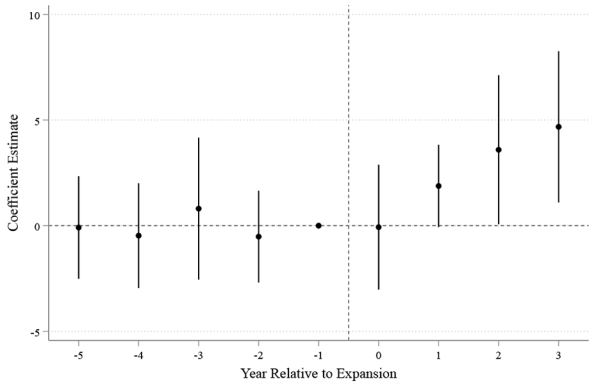


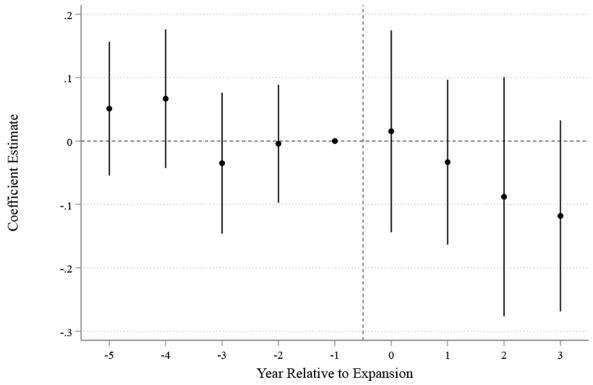
Figure 7.:

Event Study for First-Generation Outcome: Low Birth Weight

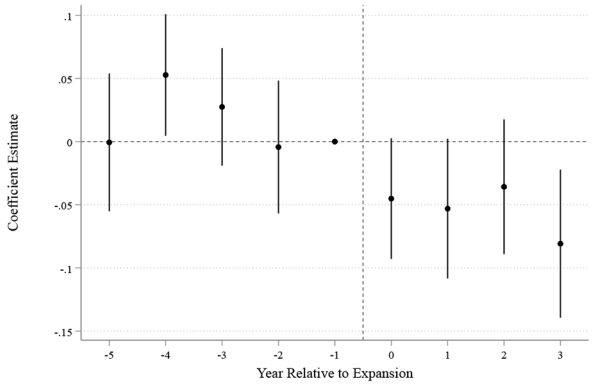
Note: Coefficient estimates are reported in percentage points. Estimated for infants born in 1975–1988. Pre-period trend is estimated and removed from all observations for each state prior to the event study estimation. For treated states, this is estimated using all pre-period years for each state. For control states, we use the period 1975–1981 to estimate this trend. Regressions are weighted by birth cohort size and include state of birth and year of birth fixed effects and controls for state-year variables (unemployment rate, personal income per capita, maximum welfare benefit for a family of 4, indicators for state parental consent and notification laws and state Medicaid restrictions for abortion, and demographic controls for each state and year). Standard errors are clustered by infant's state of birth.



(a) Birthweight



(b) Low birthweight



(c) Very low birthweight

Figure 8.:

Event Study for Second Generation Birthweight Outcomes

Note: Coefficient estimates for (b) and (c) are reported in percentage points. Estimated for first-born infants of mothers born in 1975–1988 and ages 15–28. Pre-period trend is estimated and removed from all observations for each state prior to the event study estimation. For treated states, this is estimated using all pre-period years for each state. For control states, we use the period 1975–1981 to estimate this trend. Regressions are weighted by second generation birth cohort size and include mother's state of birth and mother's

year of birth fixed effects and controls for state-year variables (unemployment rate, personal income per capita, maximum welfare benefit for a family of 4, indicators for state parental consent and notification laws and state Medicaid restrictions for abortion, and demographic controls for each state and year). Standard errors are clustered by mother's state of birth.

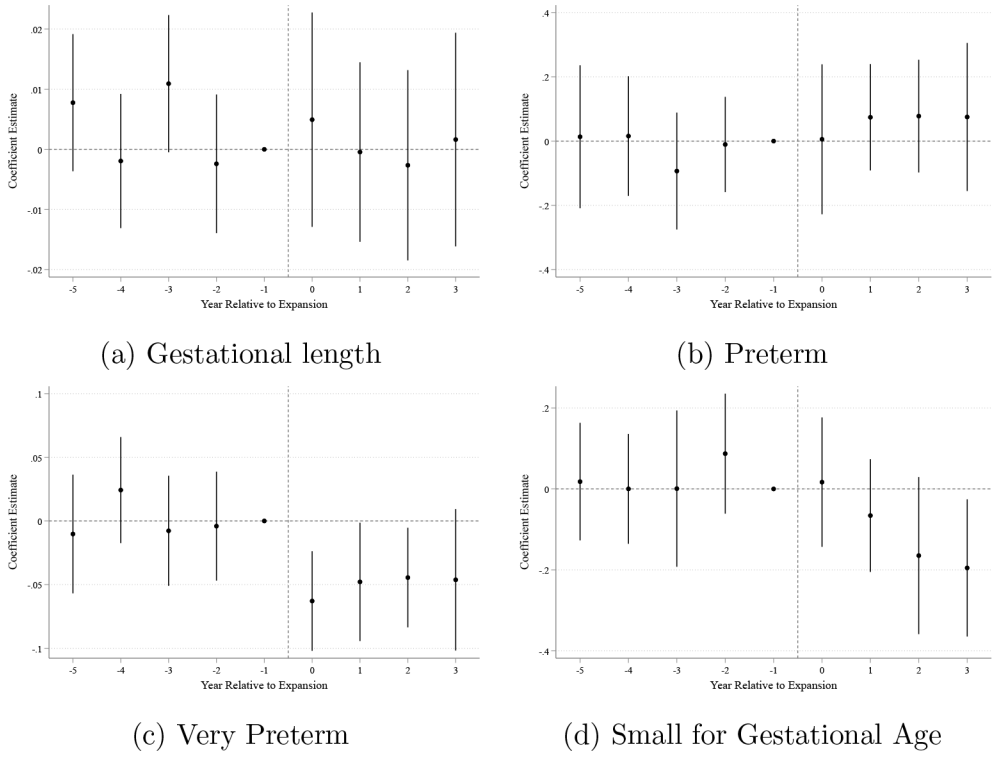
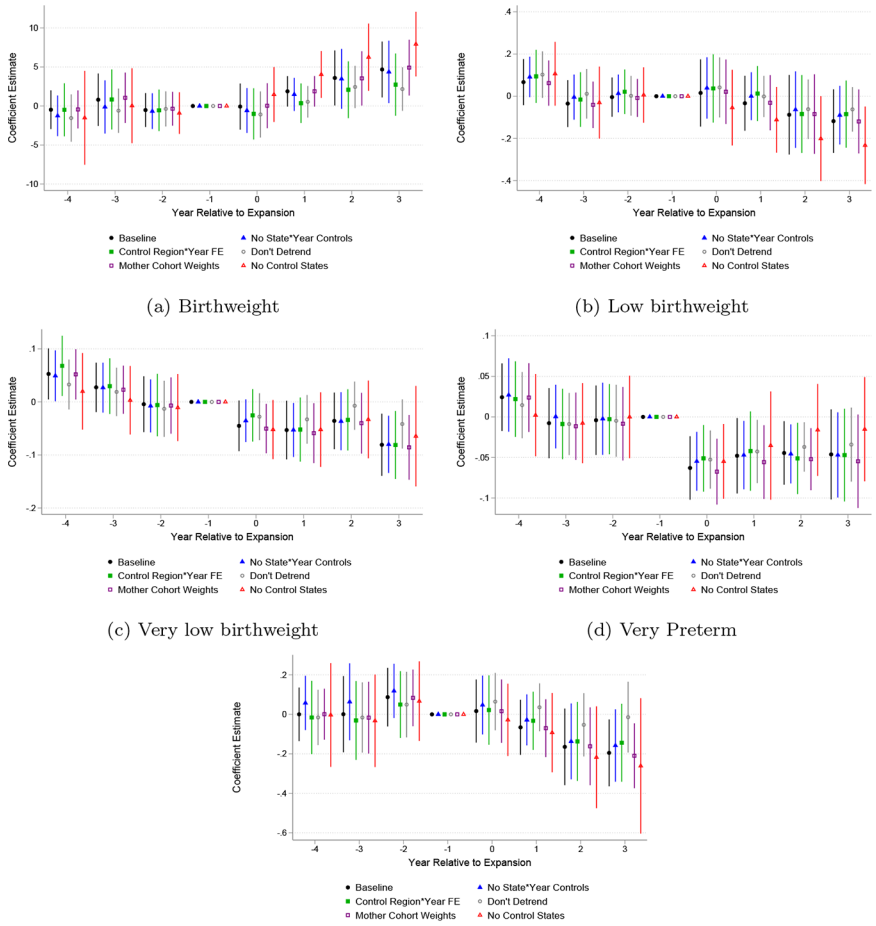


Figure 9.:

Event Study for Second Generation Gestational Length Outcomes

Note: Coefficient estimates for (b), (c), and (d) are reported in percentage points. Estimated for first-born infants of mothers born in 1975–1988 and ages 15–28. Pre-period trend is estimated and removed from all observations for each state prior to the event study estimation. For treated states, this is estimated using all pre-period years for each state. For control states, we use the period 1975–1981 to estimate this trend. Regressions are weighted by second generation birth cohort size and include mother's state of birth and mother's year of birth fixed effects and controls for state-year variables (unemployment rate, personal income per capita, maximum welfare benefit for a family of 4, indicators for state parental consent and notification laws and state Medicaid restrictions for abortion, and demographic controls for each state and year). Standard errors are clustered by mother's state of birth.



(a) Birthweight

(b) Low birthweight

(c) Very low birthweight

(d) Very Preterm

(e) Small for Gestational Age

Figure 10.:

Event Study for Second Generation Outcomes, Robustness to Alternative Controls and Specifications

Note: Coefficient estimates for (b), (c), (d), and (e) are reported in percentage points. Estimated for first-born infants of mothers born in 1975–1988 and ages 15–28. Pre-period trend is estimated and removed from all observations for each state prior to the event study estimation. For treated states, this is estimated using all pre-period years for each state. For control states, we use the period 1975–1981 to estimate this trend. Regressions are weighted by second generation birth cohort size and include mother's state of birth and mother's year of birth fixed effects. Unless indicated otherwise, regressions also include state-year control variables. Standard errors are clustered by mother's state of birth.