

DISCUSSION PAPER SERIES

IZA DP No. 13200

**The Value of Health Insurance during a Crisis:
Effects of Medicaid Implementation on
Pandemic Influenza Mortality**

Karen Clay
Joshua Lewis
Edson Severnini
Xiao Wang

APRIL 2020

DISCUSSION PAPER SERIES

IZA DP No. 13200

The Value of Health Insurance during a Crisis: Effects of Medicaid Implementation on Pandemic Influenza Mortality

Karen Clay

Carnegie Mellon and NBER

Xiao Wang

Carnegie Mellon University

Joshua Lewis

Universite de Montreal

Edson Severnini

Carnegie Mellon and IZA

APRIL 2020

Any opinions expressed in this paper are those of the author(s) and not those of IZA. Research published in this series may include views on policy, but IZA takes no institutional policy positions. The IZA research network is committed to the IZA Guiding Principles of Research Integrity.

The IZA Institute of Labor Economics is an independent economic research institute that conducts research in labor economics and offers evidence-based policy advice on labor market issues. Supported by the Deutsche Post Foundation, IZA runs the world's largest network of economists, whose research aims to provide answers to the global labor market challenges of our time. Our key objective is to build bridges between academic research, policymakers and society.

IZA Discussion Papers often represent preliminary work and are circulated to encourage discussion. Citation of such a paper should account for its provisional character. A revised version may be available directly from the author.

ISSN: 2365-9793

IZA – Institute of Labor Economics

Schaumburg-Lippe-Straße 5–9
53113 Bonn, Germany

Phone: +49-228-3894-0
Email: publications@iza.org

www.iza.org

ABSTRACT

The Value of Health Insurance during a Crisis: Effects of Medicaid Implementation on Pandemic Influenza Mortality*

This paper studies how better access to public health insurance affects infant mortality during pandemics. Our analysis combines cross-state variation in mandated eligibility for Medicaid with two influenza pandemics — the 1957-58 “Asian Flu” pandemic and the 1968-69 “Hong Kong Flu” — that arrived shortly before and after the program’s introduction. Exploiting heterogeneity in the underlying severity of these two shocks across counties, we find no relationship between Medicaid eligibility and pandemic infant mortality during the 1957-58 outbreak. After Medicaid implementation, we find that better access to insurance in high-eligibility states substantially reduced infant mortality during the 1968-69 pandemic. The reductions in pandemic infant mortality are too large to be attributable solely to new Medicaid recipients, suggesting that the expansion in health insurance coverage mitigated disease transmission among the broader population.

JEL Classification: I13, I18, N32, N52

Keywords: public health insurance, medicaid, influenza pandemics

Corresponding author:

Edson Severnini
Carnegie Mellon University
4800 Forbes Avenue
Pittsburgh PA 15213
USA

E-mail: ersevernini@gmail.com

* We thank Lowell Taylor, Maureen Cropper, Tatyana Deryugina, Raphael Godefroy, and Nick Kuminoff for insightful suggestions, and seminar participants at Carnegie Mellon University, McGill University, and Université de Montréal and conference participants at the 66th Annual North American Meetings of the Regional Science Association International, the ASSA meetings, and the Southern Economics Association meeting for valuable comments. The authors gratefully acknowledge financial support from the Center for Electricity Industry Studies, Heinz College, and the Berkman fund at Carnegie Mellon University, from the National Science Foundation Grant SES-1627432, and from the Université de Montréal.

1 Introduction

Does improved access to public health insurance save lives during a pandemic? On the one hand, the health benefits from extending medical services to uninsured populations may be especially large during a health crisis. On the other hand, the high case volume caused by an infectious disease outbreak may overwhelm medical resources, lowering the quality of care that patients receive.

This paper studies how better access to public insurance affects infant mortality during a pandemic. Our analysis combines the expansion in public insurance following the introduction of Medicaid in 1965 with two influenza pandemics – the 1957-58 “Asian Flu” pandemic and the 1968-69 “Hong Kong Flu” – that arrived shortly before and after the program’s introduction. Each outbreak was responsible for more than 100,000 deaths in the United States (Glezen, 1996; Simonsen et al., 1997), although pandemic severity varied widely across localities.

Our empirical strategy combines *cross-state* variation in Medicaid eligibility with *cross-county* differences in underlying size of the health shock to estimate the impact of Medicaid on pandemic-related infant mortality. We use panel data on county infant mortality for 1950-1979. Infant health was acutely sensitive to pandemic influenza, which affected mortality through both post-birth infection and prenatal exposure.¹ To measure state eligibility for insurance under the Medicaid program, we use information on the share of women receiving benefits under the Aid to Families with Dependent Children (AFDC) program in 1965, which generated wide cross-state differences in eligibility for Medicaid. Variation in AFDC rates across states stemmed from long-standing institutional differences in welfare programs, and we confirm that outcomes in high- and low-AFDC states trended similarly prior to 1965 (Goodman-Bacon, 2018).

¹Pregnant mothers were particularly susceptible to pandemic infection, and in utero exposure has been linked to a range of negative short- and long-run outcomes in infants.

To measure cross-county heterogeneity in the magnitude of the health shocks, we focus on two predictors of pandemic severity: urbanization – measured by the county urban population share, and local air pollution – measured by total capacity of coal-fired power plants, which were the leading source of air pollution by mid-century (see Figure A.2). Both factors have been linked to pandemic severity (Clay, Lewis and Severnini, 2018, 2019; Aiello et al., 2010; Goscé, Barton and Johansson, 2014), and we document a strong relationship between each county-level measure and excess infant mortality during both pandemics.

We adopt a triple-difference estimation strategy that compares the deviation from trend in infant mortality in pandemic years (first difference) across counties that were more or less exposed to the shock (second difference) across states with higher or lower AFDC-based Medicaid eligibility (third difference). Additionally, we explore how these relationships differ across the two pandemics that occurred before and after Medicaid implementation (fourth difference).

We find that expansions in healthcare access from Medicaid substantially mitigated the severity of the 1968-69 pandemic. The point estimates for infant mortality are large, negative, and statistically significant. The effects are stable across various specifications and unaffected by county-level controls. In contrast, we find no relationship between future Medicaid expansions and infant mortality during the 1957-58 influenza pandemic, supporting our identifying assumption that the 1968-69 outbreak would have been similarly severe across states absent the expansion in health insurance under Medicaid.

The effects are quantitatively meaningful. We estimate that better access to public health insurance in high-AFDC states led to a 6 to 7 percent decrease in infant mortality during the 1968-69 pandemic, averting between 2,646 and 2,777 infant deaths. These effects represent mortality reductions over and above the health benefits of public

insurance in non-pandemic years. Comparing the size of the mortality effects to the expansion in coverage under Medicaid, we find that the health improvements were too large to have accrued solely to newly insured households. Instead, the results are consistent with a local health externality, in which improved healthcare access among a subset of households reduced disease transmission to the broader population.

Why did expansions in insurance eligibility mitigate pandemic-related infant mortality? The results appear to have been driven by improved access to physician services and hospital care, consistent with experimental evidence on the effects of Medicaid expansions in the 2000s (Finkelstein et al., 2012; Baicker et al., 2013; Taubman et al., 2014; Finkelstein et al., 2016; Baicker et al., 2017). Comparing the effects by age of death within the first year, we find that health benefits from Medicaid were concentrated during the first hours after birth. These patterns could reflect impacts on newborn health through improved maternal health or better access to acute care during and immediately after delivery (Currie and Schwandt, 2013; Schwandt, 2018; Almond et al., 2010). We also estimate differential effects for non-white relative to white infant mortality that are roughly proportional to the racial differences in categorical eligibility for Medicaid.

This paper contributes to the literature on pandemics. The risk of global pandemics represents a substantial cost to societies due to both the economic disruption and the loss of life (Fan, Jamison, and Summers, 2016). Scholars have focused on the 1918 Spanish Flu Pandemic, and a large medical literature has sought to understand the characteristics of the H1N1 strain responsible for the pandemic (see Taubenberger and Morens, 2006). Economists have also explored the long-run health and economic effects of in utero exposure (Almond, 2006; Beach et al., 2017). Much less is known about what can be done to mitigate the threat posed by pandemics. Researchers generally consider public interventions to have had little impact on transmission during the 1918

Spanish Flu (Brainerd and Siegler, 2003; Crosby, 1989).² Our results suggest that improved access to medical care, through expansions in public insurance, may play an important role in reducing mortality during an outbreak.

This paper also contributes to the literature on the impact of public insurance on health. Despite extensive research, evidence on the health benefits of public health insurance has been mixed. Goodman-Bacon (2018) and Currie and Gruber (1996) find that the introduction of Medicaid in 1965 and subsequent expansions in eligibility in the 1980s led to improved health outcomes. In contrast, evidence from the Oregon Health Insurance Experiment shows increased health care utilization, and improved self-reported health, but no effects on clinical measures or one-year mortality (Baicker et al., 2013; Finkelstein et al., 2012). Whereas studies typically focus on the health impacts immediately following eligibility expansions, our results show that the benefits may be especially large during periodic health crises.

The paper proceeds as follows. Section 2 provides a historical background on the flu pandemics of 1957-58 and 1968-69 as well as on the roll out of Medicaid. Section 3 describes the data used in the analysis, and discusses some descriptive statistics. Section 4 presents the empirical strategy; Section 5 reports the results; Section 6 explores the quantitative impacts of Medicaid on pandemic mortality; and Section 7 concludes.

2 Background

2.1 The Influenza Pandemics of 1957-58 and 1968-69

Influenza pandemics are infrequent events that spread on a global scale. In February 1957, a new influenza A (H2N2) virus emerged in East Asia, triggering the “Asian Flu

²In contrast, Bootsma and Ferguson (2007) and Markel et al (2007), find evidence that non-pharmaceutical interventions reduced pandemic mortality.

Pandemic.” The virus reached U.S. coastal cities in the summer of 1957. Initially, there were few infections, although there was an upsurge in cases during a second pandemic wave that struck in October and November of 1957. An estimated 25% of the U.S. population was infected during this two month period (Henderson et al., 2009; Trotter Jr et al., 1959). By March, 69,800 pandemic-related deaths had occurred in the U.S., and by its end the pandemic is estimated to have killed 116,000 Americans (CDC and Diseases, 2018).

A second influenza pandemic hit the U.S. roughly a decade later. The “Hong Kong Influenza Pandemic” of 1968-69 was a global outbreak that originated in China in July 1968. The pandemic was caused by the influenza A (H3N2) virus. It arrived in the United States in September 1968. Although the virus was highly contagious, the case-fatality rates were significantly lower than the Asian Flu, and overall U.S. mortality rates were estimated to be 100,000 (CDC and Diseases, 2018).

Vaccination had little impact on the spread of either pandemic (WHO, 2009). An effective vaccine was not developed during the 1968-69 pandemic. During the “Asian Flu” pandemic vaccines were developed by the summer of 1957, but by the pandemic’s peak, there was enough supply for just 17 percent of the U.S. population. For those who received a vaccine, the effectiveness ranged from 53-60%. Similarly, preventative public health measures such as quarantines and closures were not widely implemented during either outbreak, and they had minimal influence on disease transmission.³

Pandemic severity varied across locations within the United States and worldwide. Two factors were important determinants of severity during the 1918-1919 pandemic: urban density and air pollution (Clay, Lewis and Severini, 2018, 2019). Greater

³The Association of State and Territorial Health Officers (ASTHO) stated that “there is no practical advantage in the closing of schools or the curtailment of public gatherings as it relates to the spread of this disease. (Henderson et al., 2009, p. 267). Quarantines were viewed as ineffective given “the large number of travellers and the frequency of mild or inapparent cases” (Trotter Jr et al., 1959, p. 36).

interactions in densely populated areas increased rates of transmission (Aiello et al., 2010; Goscé, Barton and Johansson, 2014). Meanwhile, air pollution can increase susceptibility to viral infection and heighten the risk of severe complications, post-infection (Jakab, 1993; Jaspers et al., 2005).⁴ As we show below, these factors also contributed to the severity of the two mid-century pandemics.⁵

Infants were acutely sensitive to pandemic influenza through both in utero exposure and post-birth infection, and infant mortality rose sharply during both pandemic periods (Figure 1). In utero exposure to influenza has been linked to decreased birth-weight (Currie and Schwandt, 2013; Schwandt, 2018), and prenatal exposure to the 1918-19 pandemic had negative effects on long-run health and labor market outcomes (Almond, 2006; Nelson, 2010; Neelsen and Stratmann, 2012; Lin and Liu, 2014).⁶ Kelly (2011) finds negative effects of the 1957-58 pandemic on birth outcomes and cognitive development in Britain. Infants were also susceptible to post-neonatal influenza infection, particularly through secondary bacterial pneumonia infection, which was often deadly without intravenous antibiotic and fluid treatment in hospital (Almond, Chay and Greenstone, 2006).

2.2 Medicaid

The Social Security Amendments of 1965 established the Medicaid program, with the goal to improve medical access to the poor and reduce inequalities in health. Since the 1950s, the federal government had provided matching grants to states to provide medical care to the poor. Nevertheless, these payments were limited and states var-

⁴Air pollution also appears to contribute to mortality during the ongoing COVID-19 pandemic (Wu et al., 2020).

⁵Humidity has also been identified as a determinant of seasonal influenza mortality in urban counties (Barreca and Shimshack, 2012), however, we find no evidence that it contributed to mortality during either pandemic period.

⁶Williams and Mackenzie (1977) find adverse effects of exposure to influenza in mice including increased maternal and neonatal mortality and decreased growth rates of neonates.

ied widely in their funding for low-income individuals. The introduction of Medicaid program increased access to medical services among the nation's poor, especially for children and pregnant women.

Under Medicaid, the federal government expanded payments to states for the costs of providing health services to eligible individuals. The program eliminated caps on federal financing and increased the federal reimbursement rate. While there was considerable latitude in how states set up their medical assistance programs, states were required to extend coverage by 1970 or else lose federal reimbursements for existing medical programs. Twenty-six states adopted Medicaid in 1966, 11 in 1967, and the rest between 1968 and 1970, except Alaska (1972) and Arizona (1982). In the five years after Medicaid implementation, the share of children with public insurance increased by 10 percentage points, and the share of adults increased by 2 percent. (Goodman-Bacon, 2018).

The Medicaid program mandated coverage for recipients of federally funded welfare programs, which led to a close link between welfare program participation and Medicaid eligibility. As a result of underlying state-specific demographics and welfare program funding, there were significant cross-state differences in the size of the population eligible for Medicaid beginning in 1965. Given the low employment rates among the eligible population, Medicaid coverage represented new access to insurance as there was little scope for crowd-out of existing private insurance. Previous research has documented a close link between state AFDC reciprocity rates and subsequent increases in public health insurance following Medicaid implementation (Goodman-Bacon, 2018).

There are a number of channels through which access to public health insurance may have mitigated pandemic infant mortality. For infants who contracted influenza after birth, there were effective treatments for secondary bacterial pneumonia. These treatments included intravenously antibiotics and fluids which were administered in

hospitals and may have directly reduced post-neonatal mortality.⁷ Medical interventions may also have mitigated the effects of in utero exposure by reducing complications during pregnancy, thereby improving infant health at birth. Conditional on maternal health, Medicaid may have decreased neonatal mortality rates during the pandemic. Given the heightened risk of low-birthweight deliveries due to influenza infection, infant health may have improved due to better care during delivery and access to postnatal treatments such as oxygenated incubators (Richards et al., 2013). Because of this, our analysis focuses on pandemic and non-pandemic years, and on pandemics that occurred shortly before and after the introduction of Medicaid.

3 Data

To study the effects of Medicaid eligibility on pandemic mortality, we combine annual county-level health outcomes, state-level information on insurance eligibility, and underlying county-level characteristics that influenced pandemic severity.

Our main health outcome is the infant mortality rate, measured as the number of infant deaths per 1,000 live births. We obtain annual county-level infant mortality from 1950 to 1979 from the *Vital Statistics* (Bailey et al., 2018).

To measure eligibility for coverage under the Medicaid program, we use state-level information on the share of women receiving benefits under AFDC in 1965 (Goodman-Bacon, 2018). Given the close link between welfare participation and Medicaid enrollment, this variable captures cross-state differences in the size of the population eligible for the program. We focus on female AFDC participation, given its importance for both prenatal and postnatal healthcare access. We construct an indicator above- ver-

⁷Infants rarely contracted influenza during the first month of life. Regarding the prognosis for pneumonia, a 1964 pediatrics textbook stated: “The outcome is dependent on early diagnosis and the appropriateness of treatment” (Nelson, 1964, pp.847-848).

sus below-median state Medicaid eligibility based on this variable. Figure A.1 displays states with above- and below-median AFDC-based eligibility, with diagonal lines identifying states that implemented Medicaid prior to 1968.

To identify heterogeneity in the size of the health shocks, we focus on two predictors of pandemic severity across counties: air pollution and urbanization. We proxy local air pollution by total capacity of coal-fired power plants within the county boundaries (Clay, Lewis and Severnini, 2016).⁸ Coal-fired electricity generation was the leading source of air pollution by mid-century (Figure A.2). The dispersion of power plant emissions was localized, with more than 90 percent of particulate matter falling within a 30-mile radius of the plant (Levy et al., 2002).

Our second county-level predictor of pandemic severity is the urban population share, given the link between crowding and disease spread (Clay, Lewis and Severnini, 2019; Aiello et al., 2010; Goscé, Barton and Johansson, 2014). Both county-level predictors are measured in 1965.⁹ Figure A.3 shows the distribution of coal capacity and percent urban across counties.¹⁰

We draw on several additional sources for county-level covariates. These include baseline county demographic and economic characteristics from Haines and ICPSR (2010); transportation infrastructure (miles of railway lines in 1911 whether the county was intersected by the 1944 Interstate Highway System Plan) from Baum-Snow (2007) and Michaels (2008); and the number of hospital beds per capita from (Chung, Gaynor and Richards-Shubik, 2017).

⁸Direct measures air pollution are limited through the 1960s. Data for a sample of 85 counties with air quality monitoring show a strong relationship between local coal-fired capacity and TSP concentrations (Table A.1).

⁹Results based on 1955 values for coal capacity and percent urban are similar in magnitude.

¹⁰Table A.2 also provides summary statistics for the all the main variables in the analysis.

4 Empirical Strategy

To examine the role of Medicaid in offsetting the impacts of health shocks, we estimate the following triple difference regression:

$$\begin{aligned}
 IMR_{ct} = & \beta_1(Pand57_t \times Mod_c) + \beta_2(Pand68_t \times Mod_c) \\
 & + \beta_3(Pand57_t \times Mod_c \times HighAFDC_s) + \beta_4(Pand68_t \times Mod_c \times HighAFDC_s) \\
 & + \beta_5(Mod_c \times Post65_t) + \eta_c + \lambda_{st} + \psi X_{ct} + \theta_t Z_{c,baseline} + \epsilon_{ct}
 \end{aligned} \tag{1}$$

where IMR_{ct} denotes infant mortality rate per 1,000 live births in county c in year t . The variables $Pand57_t$ and $Pand68_t$ are dummies for the 1957-58 Asian Flu and the 1968-69 Hong Kong Flu pandemics. The term Mod_c denotes county-level modifiers (coal capacity, percent urban) that may have contributed to the underlying severity of the two pandemics, while $HighAFDC_s$ is an indicator for states that had above-median AFDC-based Medicaid eligibility.

The regression includes controls for county fixed effects, η_c , and state-by-year fixed effects, λ_{st} , and annual climatic variables, X_{ct} , that may have influenced disease spread (precipitation, average temperature, days above 29 degrees Celsius, and days below 10 degrees Celsius). We also include a vector of linear trends based on baseline county characteristics, $\theta_t Z_{c,baseline}$. The factors include baseline socioeconomic conditions (population density, percent white, percent aged 25 plus with a high school degree, and median family income, all measured in 1950). In addition, $Z_{c,baseline}$ includes several factors that have been specifically linked to pandemic severity including 1) baseline population health (measured as the infant mortality rate from 1927 to 1945), which captures potential susceptibility to a negative health shock,¹¹ 2) manu-

¹¹This extended time horizon is meant to capture underlying maternal health, which may have

facturing employment in 1950, which was an alternate source of local air pollution, 3) transportation infrastructure (mileage of railways, rivers, and the 1944 planned Interstate highway system), which may have contributed to the spread of the virus, and 4) healthcare infrastructure (per capita hospital beds in 1948).

The coefficients β_1 and β_2 identify the relationship between county-level modifiers and pandemic infant mortality in low-AFDC eligibility states. These coefficients capture the extent to which within-state variation in excess pandemic mortality was systematically related to baseline coal capacity and percent urban. The estimated coefficients provide a measure of underlying heterogeneity in severity of the health shocks according to these two county-level predictors.

The main coefficient estimates of interest are β_3 and β_4 . These coefficients capture the differential in the pandemic-modifier gradient in high AFDC-based eligibility states relative to low eligibility states during both the 1957-58 Asian Flu and the 1968-69 Hong Kong Flu pandemics. The estimates of β_4 capture the extent to which the relative expansion in AFDC-based public insurance under Medicaid mitigated infant mortality in counties that were exposed to particularly severe health shocks. Meanwhile, the estimates of β_3 allow us to test whether there were pre-existing differences between high- and low-AFDC states that contributed to within-state heterogeneity in the severity of the pandemic.

Our identification assumption is that within-state heterogeneity in pandemic severity would have been similar across high- and low-AFDC states absent the implementation of Medicaid. This assumption is supported by three pieces of evidence.

First, AFDC-based Medicaid eligibility was based on long-standing institutional and demographic differences across states. Factors that influenced state-level eligibility, including long-run institutional barriers, family structure, and household incomes,

influenced susceptibility to the pandemic shocks.

had differed across states since the 1930s (Alston and Ferrie, 1985; Moehling, 2007). Moreover, AFDC rates were stable across states in the decades prior to Medicaid, suggesting no anticipatory changes in welfare generosity (Goodman-Bacon, 2018).

Second, we find little evidence that state AFDC eligibility are correlated with levels or trends in state socioeconomic conditions. Table A.3 presents results from balancing tests for differences in levels and trends in pre-1965 characteristics across states with different rates of AFDC eligibility.¹² We find no evidence of differential trends according to state AFDC eligibility. The coefficient estimates for β_0 and β_1 are all small and (with the exception of percent white) statistically insignificant. The overall patterns are consistent with the results of Goodman-Bacon (2018), who finds that welfare-based eligibility is uncorrelated with either the levels or trends across a range of state characteristics.

Third, the estimates of β_3 from equation (1) allow us to directly test whether there were unobservable differences across high- and low-AFDC states that impacted within-state heterogeneity in pandemic-related infant mortality. The results (reported below in section 5), are all statistically insignificant and small in magnitude, supporting our research design.

Two final estimation details are worth mentioning. First, robust standard errors are clustered at the county-level to adjust for heteroskedasticity and within-county serial correlation.¹³ Second, all regressions are weighted by the number of live births.

¹²The results are based on the following regression: $y_{st} = \alpha + \beta_0 HighAFDC_s^* + \beta_1 HighAFDC_s^*(Year - Year_{pre}) + \mu_{st}$, where β_0 tests for differences in levels and β_1 tests for differences in trends.

¹³We also report results from specification that cluster standard errors at the state-level.

5 Results

5.1 Coal Capacity, Percent Urban, and the Impact of Medicaid on Pandemic Mortality

The first set of results explore heterogeneity in pandemic severity according to coal capacity and urbanization and evaluates the extent to which greater access to public insurance offsets excess mortality. Table 1 reports the estimates of $\beta_1 - \beta_4$ from different versions of equation (1). Columns 1-2 report the results for coal capacity, columns 3-4 report the results for percent urban. We report results from two different specifications. Odd columns report the estimates from models that include the baseline controls for county fixed effects, state-by-year fixed effects, temperature and precipitation, and linear time trends according to baseline county socioeconomic conditions. In even columns, we report results based on the full set of controls.¹⁴

The estimates of the coal-pandemic interactions terms, β_1 and β_2 , reveal substantial within-state heterogeneity in excess pandemic mortality according to baseline county coal capacity (cols. 1-2). The coefficient estimates are positive and statistically significant, consistent with previous research suggesting that poor air quality exacerbates influenza severity (Clay, Lewis and Severnini, 2018; Hanlon, 2018). We find similar differentials in pandemic mortality in counties with a larger percentage of urban residents (cols. 3-4). The coefficient estimates for β_1 and β_2 are positive, statistically significant, and similar in magnitude across the two pandemics. The patterns are consistent with previous research on heightened transmission in densely populated areas (Clay, Lewis and Severnini, 2019; Aiello et al., 2010; Goscé, Barton and Johansson, 2014).

The triple interaction estimates suggest that better access to public health insurance

¹⁴Table A.4 reports the corresponding results, with standard errors clustered at the state level. Table A.5 reports results that progressively add each group of covariates.

under Medicaid significantly reduced excess pandemic mortality in polluted and urban areas. For coal capacity, the coefficients on the 1968-69 pandemic triple interaction term, β_4 , are negative and statistically significant, and similar in magnitude to the main coal-pandemic interaction effects (cols. 1-2). The positive coal capacity-pandemic mortality gradient in low-AFDC states did not exist in high-AFDC states during the 1968-69 pandemic, suggesting that better access to healthcare substantially mitigated the impact of the severe health shock. For percent urban, the estimates on the 1968-69 triple interaction term are also negative and statistically significant, implying that better access to healthcare largely offset the differential impact of the shock in urban areas.

We also report the coefficient estimates for β_3 , the triple interaction term for the 1957-58 pandemic. These estimates identify average differences in the pandemic mortality gradient in high-AFDC states relative to low-AFDC states prior to the enactment of Medicaid. Across the various specifications the point estimates are small and statistically insignificant, and hypothesis tests for the equality of the two triple interaction coefficients are rejected at a 10 percent significance level. These findings provide further support for our identifying assumption that absent Medicaid implementation, within-state heterogeneity in pandemic severity would have been similar across high- and low-AFDC states.

Table 1 columns 5-6 report the results from horserace regressions that include interactions effects for both coal capacity and percent urban. These models capture the independent link between each factor and pandemic severity, and the extent to which these relationships were mitigated by the Medicaid implementation. The goal of this exercise is not to establish the relative impact of each factor on pandemic severity, but rather the extent to which both coal capacity and percent urban reflect

two independent sources of heterogeneity in pandemic severity.¹⁵ The main pandemic interaction effects for coal and percent urban remain positive, albeit smaller in magnitude, consistent with the concentration of coal-fired generation near urban areas. The triple interactions effects for 1968-69 pandemic for coal and percent urban are negative and statistically significant, and roughly two-thirds the magnitude of the previous estimates. In contrast, the triple interaction estimates for the 1957-58 pandemic are small and statistically insignificant. Together, these results suggest that better access to healthcare following Medicaid implementation significantly offset both sources of excess pandemic mortality in high-AFDC states.

5.2 Robustness Exercises

We assess whether the observed link between AFDC reciprocity and excess mortality during the 1968-69 pandemic might reflect the impact of other social policies that were adopted in the 1960s under the War on Poverty. We focus on two major policies – Head Start and the Food Stamps program – both of which have been linked to relative improvements in infant health (Ludwig and Miller, 2007; Almond, Hoynes and Schanzenbach, 2011). We estimate versions of equation (1) that include triple interaction terms for above median per capita state expenditure under Head Start (per 1,000 children aged 1 to 9) and the Food Stamps program along with the main AFDC-based term. To address concerns that implementation of these other two welfare programs was influenced by underlying state health conditions, we report all triple interaction effects in differences (post- versus pre-Medicaid): $\beta_4 - \beta_3$.

Table 2 reports the results. For reference, column 1 reports the results for AFDC, based on Table 1 (cols. 2 and 4). The inclusion of interactions based on per capita

¹⁵This decomposition will also allow us to estimate the quantitative impact of Medicaid on coal- and urban-based pandemic infant mortality (see Section 6).

spending on Head Start or the Food Stamps program has little impact on the main AFDC interaction effects, and the point estimates for these other programs are small in magnitude and statistically insignificant (cols. 2,3). Together, these results suggest that relative decreases in excess pandemic mortality in high-AFDC states cannot be attributed to contemporaneous adoption of War on Poverty programs.

Table A.6 reports the results from several additional robustness tests. For reference, column 1 presents the baseline results. Column 2 reports estimates for a restricted sample of states that had implemented Medicaid by 1967, prior to the onset of the pandemic.¹⁶ This restriction addresses concerns regarding endogeneity in state decisions to implement Medicaid, which may have been influenced by the pandemic itself. The results from these regressions are similar to the baseline findings in sign, significance, and magnitude. We also report estimates from regressions for sub-samples with positive coal capacity (col. 3) and with non-zero urban population (col. 4). Despite the decreases in sample size, the findings are similar to the baseline results. Column 5 reports results from an unbalanced sample that includes an additional 109 counties with incomplete data.¹⁷ The estimates are similar in terms of sign, significance, and size.

5.3 Pandemic Infant Mortality by Age and Race

Table 3 reports estimates for infant mortality by age at death (first day, days 2-27, post-neonatal, and first year) and by race.¹⁸ Panel A reports the effects by coal capacity; Panel B reports the effects by percent urban; and Panel C reports the results

¹⁶This restriction excludes 11 states that implemented Medicaid between 1968 and 1970, as well as Alaska (1972) and Arizona (1982).

¹⁷These counties either had missing information on infant mortality in at least one year or lacked information on hospital beds per capita or median household income.

¹⁸County-level infant mortality data by age of death and race are available beginning in 1960 and in 1962, respectively.

from regressions that include both factors.

Columns 1 to 4 report the interaction effects by age. The results show that nearly all of the impact of Medicaid on coal-related pandemic mortality occurred during the first day of life. The point estimates for first day mortality are negative and statistically significant, and similar in magnitude to the total effects on one year mortality. The expansion in Medicaid may have reduced day one mortality through a number of channels. Greater access to healthcare services may have improved in utero health by mitigating the severity of influenza and secondary pneumonia infection among pregnant mothers.¹⁹ Better access to acute care, such as early heartbeat detection and oxygenated respirators, may have increased survival conditional on health at birth.²⁰ Finally, better access to public insurance may have decreased local transmission of the virus, thereby reducing the likelihood of maternal infection even among non-Medicaid recipients.²¹

We find significant effects of Medicaid implementation on urban-related pandemic mortality for both day one mortality and post-neonatal mortality. The estimated effects on day one pandemic-related mortality could reflect a combination of improved in utero health and better access to healthcare technologies during and after delivery. Meanwhile, the significant effects on post-neonatal mortality also suggest improved survival among infants who directly contracted the virus. These findings are supported by evidence on the importance of improved hospital access for the treatment of post-neonatal infectious disease (Almond, Chay and Greenstone, 2006).

Table 3 (col. 5) reports results from regressions that allow the effects on infant

¹⁹We find no significant impact of Medicaid implementation on maternal mortality (Table A.7), consistent with the fact that excess mortality during the 1968-69 Pandemic was largely limited to infants and the elderly.

²⁰Because county-level information on measures of health at birth, such as birthweight or Apgar scores, are not available prior to the 1968 pandemic, we are unable to evaluate the relative importance of these two channels.

²¹We explore the role of local health externalities in Section 6.

mortality to differ by race.²² The estimates reveal systematically larger impacts of Medicaid eligibility on non-white infant mortality. In both the coal capacity and percent urban models, we estimate significantly larger reductions in nonwhite pandemic infant mortality in high-AFDC states. These differential impacts range from a factor of three in the coal capacity regression to a factor of six in the percent urban regression. Interestingly, these differential health impacts correspond roughly to the fivefold difference in AFDC eligibility across white and non-white households. The results also support previous research on the disproportionate impacts of Medicaid on nonwhite infant and child mortality (Goodman-Bacon, 2018).

Together, the results allow us to rule out hospital desegregation as an alternative explanation for the observed Medicaid impacts. First, excluding states that implemented Medicaid after 1967 – the majority of which were located in the South – has no impact on the main estimates (Table A.6, col. 2).²³ Second, the estimated decreases in infant mortality were concentrated among neonates (Table 3, cols. 1-4), whose health was largely unaffected by southern hospital desegregation (Almond, Chay and Greenstone, 2006). Third, the decreases in pandemic-related infant deaths occurred among both white and non-white infants in proportions that were roughly similar to their rates of AFDC eligibility (Table 3, col. 5).

²²White and Non-white infant mortality rates are calculated as the number of infant deaths per 1,000 live births, with both numerator and denominator measured separately by race.

²³Whereas hospital desegregation occurred shortly after 1965, most southern states implemented Medicaid in 1970. In fact, the only southern state with above median AFDC eligibility to implement Medicaid by 1967 was Oklahoma.

6 Medicaid during the 1968-69 Pandemic

6.1 Impacts on Pandemic Infant Deaths

In this section, we explore the quantitative implications of the findings to evaluate how the expansion in health insurance under Medicaid mitigated the infant mortality burden during the 1968-69 pandemic.

We calculate how better access to health insurance through Medicaid mitigated excess pandemic infant mortality associated with local coal capacity and urbanization across high- and low-AFDC states. To obtain the estimated pandemic mortality differentials, we multiply the triple interaction coefficients by the county-level means for coal capacity and urbanization. We report the results based on the 1968-69 triple interaction effect, β_4 . We also report estimates based on the difference in the interaction effects across the two pandemics, $\beta_4 - \beta_3$, to account for any underlying differences in the coal- and urban-pandemic mortality relationship across high- and low-AFDC states.²⁴

Table 4, Panel A reports the estimates for coal capacity. The preferred point estimates imply that better access to health insurance in high-AFDC states offset excess pandemic infant mortality rate by 0.26 ($= (0.071 - 0.023) \times 5.49$) to 0.39 (0.071×5.49) per 1,000 live births.²⁵ We combine these relative decreases in excess infant mortality with the size of the exposed population to calculate the number of infant deaths averted. The preferred estimates imply that the relative expansion of public insurance in high-AFDC states averted 514 to 760 infant deaths.²⁶

²⁴The two approaches yield similar quantitative findings, since the estimates for β_3 are all small and statistically insignificant (Table 1).

²⁵The county-level mean for coal capacity, 5.49, is weighted by total live births to capture average infant exposure across high-AFDC states.

²⁶These estimates are obtained by multiplying the implied infant mortality reductions by the total number of exposed infant in high-AFDC states. For example, the effect implied in col. 1 is given by $\beta_4/1,000 \times \text{Number of live births} = 0.00039 \times 1,950,135 = 760$.

Panel B reports the results for the combined effects of coal capacity and urbanization (Table 1, col. 6). The expansion in public health insurance significantly offset the pandemic mortality burden. We estimate relative decreases in excess pandemic mortality ranging from 1.36 to 1.42 per 1,000 live births, a 6 percent decrease in the overall infant mortality rate. By mitigating severe pandemic shocks in high coal and urban areas, the relative expansion of public insurance averted between 2,646 and 2,777 infant deaths in high-AFDC states.

6.2 Medicaid Recipients and Local Health Externalities

To conclude the analysis, we explore whether the effects on pandemic infant mortality can be attributed solely to new insurance coverage among the Medicaid eligible population. We estimate the average treatment effect on the treated (ATET), dividing the triple-difference estimates by the cross-state difference in insurance access implied by a first-stage regression of overall children’s insurance rates on the fraction of women age 15-44 on AFDC. The resulting estimates capture the pandemic mortality per program beneficiary.

We find that the effects on pandemic infant mortality are too large to be attributed solely to newly insured households. The ATETs for the joint effect of coal- and urban-related pandemic mortality range from 23.6 to 24.8 (Table 4 Panel C, cols. 3-4). Even adjusting for higher underlying infant mortality rates among the Medicaid eligible population, these estimates imply reductions in infant mortality of more than 70 percent among newly insured households. These effect sizes are implausibly large, and strongly suggest that the health benefits from Medicaid implementation extended beyond newly insured households.

Our pandemic ATET estimates are substantially larger than the average effects of Medicaid of infant health across both pandemic and non-pandemic years (Goodman-

Bacon, 2018). In normal times, Medicaid’s effects should be concentrated among recipient households whose access to medical services was directly affected by the program. During the pandemic, however, expansions in public insurance may influence local disease transmission and generate health externalities to non-recipient households. Better access to doctors may increase the likelihood that parents isolated sick children at home. Access to better healthcare may decrease viral load and shorten the period of contagion. The shift from home-based to hospital care for those with acute illnesses may further reduce transmission through an isolation effect. Understanding the role of the health system in influencing disease transmission may be a fruitful area of future research.

7 Conclusion

This paper provides new evidence on the role of public health insurance in mitigating pandemic severity. Our research strategy leverages cross-state variation in Medicaid implementation with two influenza pandemics that arrived shortly before and after the program’s passage. Prior to Medicaid implementation, we find no relationship between excess mortality during the 1957-58 “Asian Flu Pandemic.” After Medicaid implementation, we find that better access to healthcare significantly reduced infant mortality during the 1968-69 “Hong Kong Flu Pandemic.” The effects on mortality were sizeable and too large to be solely attributable to newly insured households. Instead, our findings suggest that better access to healthcare services for a subset of the population reduced local transmission more broadly.

Our findings provide new insights into the health benefits of public insurance. Whereas previous research on the health impacts of Medicaid have been mixed (Currie and Gruber, 1996; Goodman-Bacon, 2018; Baicker et al., 2013; Finkelstein et al.,

2012), our results show that the potential for public healthcare to save lives may be particularly large during periodic health crises. Because these episodes arrive infrequently, however, the benefits may not be captured by policy evaluations focused on immediate aftermath of implementation.

Pandemics pose a continued threat to population health. Despite modern testing capabilities and contact tracing, governments have struggled to contain the ongoing spread of the coronavirus disease 2019 (COVID-19). By demonstrating the value of improved healthcare access in reducing pandemic severity, this study's findings may have relevance for the mitigation of current and future outbreaks. Understanding how best to integrate the public and medical response to limit the spread and lethality of infectious disease outbreaks is a critical area for future research.

References

- Aiello, Allison E, Rebecca M Coulborn, Tomas J Aragon, Michael G Baker, Barri B Burrus, Benjamin J Cowling, Alasdair Duncan, Wayne Enanoria, M Patricia Fabian, Yu-hui Ferng, et al.** 2010. “Research findings from non-pharmaceutical intervention studies for pandemic influenza and current gaps in the research.” *American journal of infection control*, 38(4): 251–258.
- Almond, Douglas.** 2006. “Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population.” *Journal of political Economy*, 114(4): 672–712.
- Almond, Douglas, Hilary W. Hoynes, and Diane Whitmore Schanzenbach.** 2011. “Inside the War on Poverty: The Impact of Food Stamps on Birth Outcomes.” *Review of Economics and Statistics*, 93(2): 387–403.
- Almond, Douglas, Joseph J. Doyle, Jr., Amanda E. Kowalski, and Heidi Williams.** 2010. “Estimating Marginal Returns to Medical Care: Evidence from At-risk Newborns.” *Quarterly Journal of Economics*, 125(2): 591–634.
- Almond, Douglas, Kenneth Y Chay, and Michael Greenstone.** 2006. “Civil rights, the war on poverty, and black-white convergence in infant mortality in the rural South and Mississippi.”
- Alston, Lee J, and Joseph P Ferrie.** 1985. “Labor costs, paternalism, and loyalty in southern agriculture: A constraint on the growth of the welfare state.” *The Journal of Economic History*, 45(1): 95–117.
- Baicker, Katherine, , Heidi L. Allen, Bill J. Wright, and Amy N. Finkelstein.** 2017. “The Effect Of Medicaid On Medication Use Among Poor Adults: Evidence From Oregon.” *Health Affairs*, 36(12): 2110–2114.
- Baicker, Katherine, Sarah L. Taubman, Heidi L. Allen, Mira Bernstein, Jonathan H. Gruber, Joseph P. Newhouse, Eric C. Schneider, Bill J. Wright, Alan M. Zaslavsky, Amy N. Finkelstein, and Oregon Health Study Group.** 2013. “The Oregon Experiment – Effects of Medicaid on Clinical Outcomes.” *New England Journal of Medicine*, 368(18): 1713–1722.
- Bailey, Martha, Karen Clay, Price Fishback, Michael Haines, Shawn Kantor, Edson Severnini, Anna Wentz, and Inter-university Consortium for Political & Social Research ICPSR.** 2018. “U.S. County-Level Natality and Mortality Data, 1915-2007.” Inter-university Consortium for Political and Social Research, Ann Arbor, MI.
- Barreca, Alan I, and Jay P Shimshack.** 2012. “Absolute humidity, temperature, and influenza mortality: 30 years of county-level evidence from the United States.” *American journal of epidemiology*, 176(suppl.7): S114–S122.

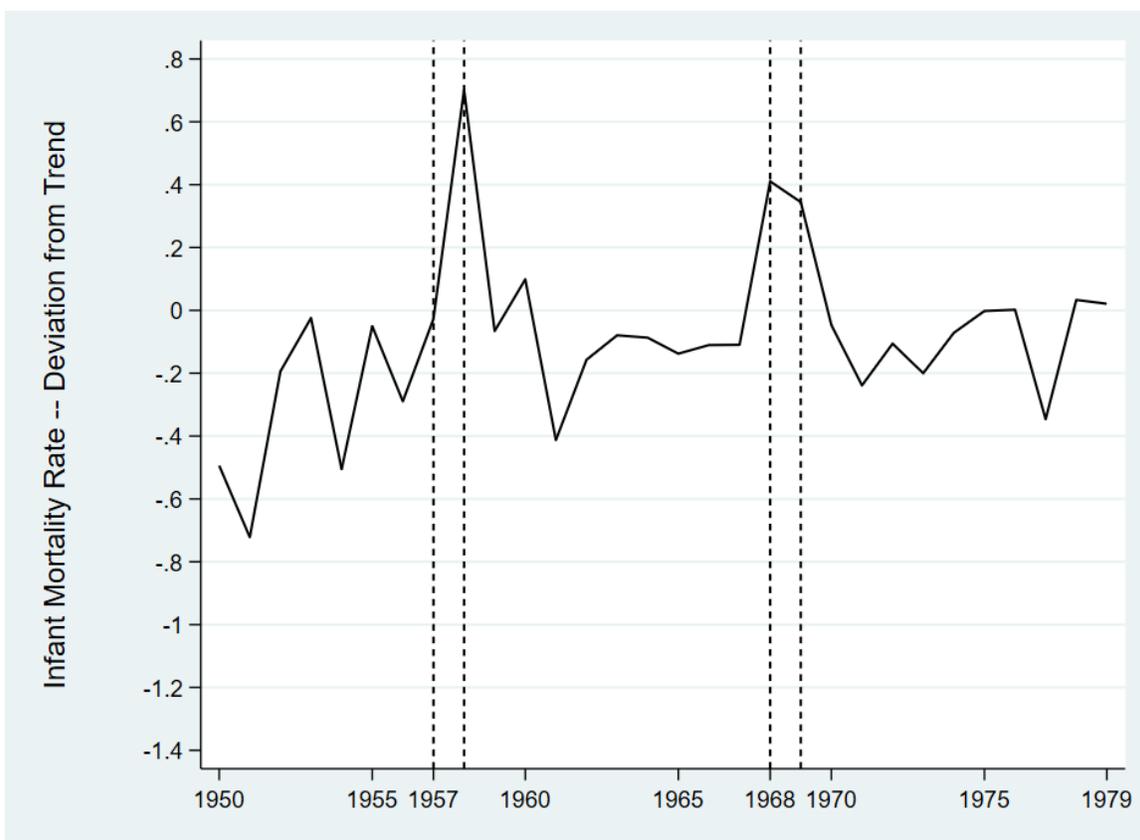
- Baum-Snow, Nathaniel.** 2007. “Did Highways Cause Suburbanization?” *Quarterly Journal of Economics*, 122(2): 775–805.
- CDC, National Center for Immunization, and Respiratory Diseases.** 2018. “Pandemic Influenza, Past Pandemics.” <https://www.cdc.gov/flu/pandemic-resources/basics/past-pandemics.html>.
- Chung, Andrea Park, Martin Gaynor, and Seth Richards-Shubik.** 2017. “Subsidies and structure: the lasting impact of the Hill-Burton program on the hospital industry.” *Review of Economics and Statistics*, 99(5): 926–943.
- Clay, Karen, Joshua Lewis, and Edson Severnini.** 2016. “Canary in a Coal Mine: Impact of Mid-20th Century Air Pollution Induced by Coal-Fired Power Generation on Infant Mortality and Property Values.” *NBER Working Paper No. 22155*.
- Clay, Karen, Joshua Lewis, and Edson Severnini.** 2018. “Pollution, Infectious Disease, and Mortality: Evidence from the 1918 Spanish Influenza Pandemic.” *Journal of Economic History*, 78(4): 1179–1209.
- Clay, Karen, Joshua Lewis, and Edson Severnini.** 2019. “What explains cross-city variation in mortality during the 1918 influenza pandemic? Evidence from 438 US cities.” *Economics & Human Biology*, 35: 42–50.
- Currie, Janet, and Hannes Schwandt.** 2013. “Within-mother analysis of seasonal patterns in health at birth.” *Proceedings of the National Academy of Sciences*, 110(30): 12265–12270.
- Currie, Janet, and Jonathan Gruber.** 1996. “Health Insurance Eligibility, Utilization of Medical Care, and Child Health.” *Quarterly Journal of Economics*, 111(2): 431–466.
- Finkelstein, Amy N., Sarah L. Taubman, Bill J. Wright, Mira Bernstein, Jonathan H. Gruber, Joseph P. Newhouse, Heidi L. Allen, Katherine Baicker, and Oregon Health Study Group.** 2012. “The Oregon Health Insurance Experiment: Evidence from the First Year.” *Quarterly Journal of Economics*, 127(3): 1057–1106.
- Finkelstein, Amy N., Sarah L. Taubman, Heidi L. Allen, Bill J. Wright, and Katherine Baicker.** 2016. “Effect of Medicaid Coverage on ED Use – Further Evidence from Oregon’s Experiment.” *New England Journal of Medicine*, 375(16): 1505–1507.
- Gartner, Scott Sigmund, et al.** 2006. In *Historical Statistics of the United States*, ed. Susan B. Carter, Scott Sigmund Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch and Gavin Wright. New York, NY:Cambridge University Press.

- Glezen, W Paul.** 1996. “Emerging infections: pandemic influenza.” *Epidemiologic reviews*, 18(1): 64–76.
- Goodman-Bacon, Andrew.** 2018. “Public Insurance and Mortality: Evidence from Medicaid Implementation.” *Journal of Political Economy*, 126(1): 216–262.
- Goscé, Lara, David AW Barton, and Anders Johansson.** 2014. “Analytical modelling of the spread of disease in confined and crowded spaces.” *Scientific reports*, 4: 4856.
- Haines, Michael R., and Inter-university Consortium for Political & Social Research ICPSR.** 2010. “Historical, Demographic, Economic, and Social Data: The United States, 1790-2002.” Inter-university Consortium for Political and Social Research, Ann Arbor, MI.
- Hanlon, W. Walker.** 2018. “London Fog: A Century of Pollution and Mortality, 1866-1965.” NBER Working Paper #24488.
- Henderson, Donald A, Brooke Courtney, Thomas V Inglesby, Eric Toner, and Jennifer B Nuzzo.** 2009. “Public health and medical responses to the 1957-58 influenza pandemic.” *Biosecurity and bioterrorism: biodefense strategy, practice, and science*, 7(3): 265–273.
- Jakab, George J.** 1993. “The toxicologic interactions resulting from inhalation of carbon black and acrolein on pulmonary antibacterial and antiviral defenses.” *Toxicology and applied pharmacology*, 121(2): 167–175.
- Jaspers, Ilona, Jonathan M Ciencewicki, Wenli Zhang, Luisa E Brighton, Johnny L Carson, Melinda A Beck, and Michael C Madden.** 2005. “Diesel exhaust enhances influenza virus infections in respiratory epithelial cells.” *Toxicological Sciences*, 85(2): 990–1002.
- Kelly, Elaine.** 2011. “The scourge of asian flu in utero exposure to pandemic influenza and the development of a cohort of british children.” *Journal of Human resources*, 46(4): 669–694.
- Levy, Jonathan I., John D. Spengler, Dennis Hlinka, David Sullivan, and Dennis Moon.** 2002. “Using CALPUFF to Evaluate the Impacts of Power Plant Emissions in Illinois: Model Sensitivity and Implications.” *Atmospheric Environment*, 36: 1063–1075.
- Lin, Ming-Jen, and Elaine M Liu.** 2014. “Does in utero exposure to illness matter? The 1918 influenza epidemic in Taiwan as a natural experiment.” *Journal of health economics*, 37: 152–163.

- Ludwig, Jens, and Douglas L. Miller.** 2007. “Does Head Start Improve Children’s Life Chances? Evidence from a Regression Discontinuity Design.” *Quarterly Journal of Economics*, 122(1): 159–208.
- Michaels, Guy.** 2008. “The Effect of Trade on the Demand for Skill: Evidence from the Interstate Highway System.” *Review of Economics and Statistics*, 90(4): 683–701.
- Moehling, Carolyn M.** 2007. “The American Welfare System and Family Structure An Historical Perspective.” *Journal of Human Resources*, 42(1): 117–155.
- Neelsen, Sven, and Thomas Stratmann.** 2012. “Long-run effects of fetal influenza exposure: Evidence from Switzerland.” *Social Science & Medicine*, 74(1): 58–66.
- Nelson, Richard E.** 2010. “Testing the fetal origins hypothesis in a developing country: evidence from the 1918 influenza pandemic.” *Health economics*, 19(10): 1181–1192.
- Richards, Jennifer L, Craig Hansen, Christine Bredfeldt, Robert A Bednarczyk, Mark C Steinhoff, Dzifa Adjaye-Gbewonyo, Kevin Ault, Mia Gallagher, Walter Orenstein, Robert L Davis, et al.** 2013. “Neonatal outcomes after antenatal influenza immunization during the 2009 H1N1 influenza pandemic: impact on preterm birth, birth weight, and small for gestational age birth.” *Clinical Infectious Diseases*, 56(9): 1216–1222.
- Schwandt, Hannes.** 2018. “The lasting legacy of seasonal influenza: In-utero exposure and labor market outcomes.”
- Simonsen, Lone, Matthew J Clarke, G David Williamson, Donna F Stroup, Nancy H Arden, and Lawrence B Schonberger.** 1997. “The impact of influenza epidemics on mortality: introducing a severity index.” *American journal of public health*, 87(12): 1944–1950.
- Taubman, Sarah L., Heidi L. Allen, Bill J. Wright, Katherine Baicker, and Amy N. Finkelstein.** 2014. “Medicaid Increases Emergency-Department Use: Evidence from Oregon’s Health Insurance Experiment.” *Science*, 343(6168): 263–268.
- Trotter Jr, Yates, Frederick L Dunn, Robert H Drachman, Donald A Henderson, Mario Pizzi, Alexander D Langmuir, et al.** 1959. “Asian influenza in the United States, 1957-1958.” *American journal of hygiene*, 70(1): 34–50.
- WHO, World Health Organization.** 2009. “Diarrhoea: Why Children Are Still Dying and What Can Be Done.” UN Children’s Fund and the World Health Organization.
- Williams, K, and JS Mackenzie.** 1977. “Influenza infections during pregnancy in the mouse.” *Epidemiology & Infection*, 79(2): 249–257.

Figures and Tables

Figure 1: Infant Mortality Rate and Influenza Pandemics



Notes: This figure displays deviations from trend in the infant mortality rate for the period 1950 to 1979. Deviations are constructed relative to a linear trends over each five-year interval during the sample period. The vertical short-dashed lines highlight the flu pandemics of 1957-58 and 1968-69.

Table 1: Medicaid and Pandemic Infant Mortality

	Dependent Variable: Infant Mortality Rate					
	(1)	(2)	(3)	(4)	(5)	(6)
P 1957-58 x Coal	0.066*** (0.021)	0.066*** (0.021)			0.053** (0.024)	0.053** (0.024)
P 1957-58 x AFDC x Coal	-0.022 (0.024)	-0.023 (0.024)			-0.017 (0.027)	-0.018 (0.028)
P 1968-69 x Coal	0.049*** (0.015)	0.059*** (0.016)			0.036** (0.016)	0.050*** (0.018)
P 1968-69 x AFDC x Coal	-0.066*** (0.019)	-0.071*** (0.021)			-0.045** (0.020)	-0.050** (0.023)
P 1957-58 x Pct Urban			0.015*** (0.005)	0.015*** (0.005)	0.007 (0.005)	0.007 (0.005)
P 1957-58 x AFDC x Pct Urban			-0.004 (0.007)	-0.004 (0.007)	0.000 (0.007)	0.000 (0.007)
P 1968-69 x Pct Urban			0.017*** (0.005)	0.015*** (0.005)	0.010** (0.005)	0.007 (0.005)
P 1968-69 x AFDC x Pct Urban			-0.023*** (0.006)	-0.024*** (0.006)	-0.016** (0.006)	-0.016** (0.006)
$\beta_3=\beta_4$ (Coal)	.097	.065			.345	.277
$\beta_3=\beta_4$ (Urban)			.035	.019	.096	.076
Dep Var: Mean (S.D.)			23.6 (13.7)			
Coal Capacity: Mean (S.D.)			5.5 (8.8)			
Percent Urban: Mean (S.D.)			69.8 (28.2)			
Observations	83130	83130	83130	83130	83130	83130
Counties	2771	2771	2771	2771	2771	2771
Adj.R-Squared	.629	.632	.630	.633	.630	.633
Baseline Controls	Y	Y	Y	Y	Y	Y
Full Controls		Y		Y		Y

Notes: This table reports the main coefficients of interest estimated of equation (1) for coal capacity and percent urban. The baseline control include state-by-year and county fixed effects, temperature and precipitation variables (five bins each), and linear time trends in county-level baseline socioeconomic characteristics (population density, percent white, percent age 25+ with high school, and median family income (all measured in 1950). The full controls include the differential time trends according to manufacturing employment in 1950, and terciles of baseline IMR (averaged over the period 1927 to 1945), transportation infrastructure (mileage of navigable rivers, mileage of railroads, and an indicator for whether a county was supposed to receive a highway from the 1944 Interstate Highway System Plan), and hospital beds per capita in 1948. Standard errors clustered at the county level are reported in parentheses. *** denotes statistical significance at the 1 percent level, ** at the 5 percent level, and * at the 10 percent level. P-value of the t-test on the equality of β_3 , the coefficient of P 1957-58 x AFDC x Modifier, and β_4 , the coefficient of P 1968-69 x AFDC x Modifier are reported.

Table 2: Effects of AFDC and Other War on Poverty Programs

	Dependent Variable: Infant Mortality Rate		
	Baseline (1)	Head Start (2)	Food Stamps (3)
<i>Panel A: Effects by Coal Capacity</i>			
Difference in Triple Interactions: $\beta_4 - \beta_3$			
AFDC	-0.048* (0.026)	-0.053** (0.024)	-0.053* (0.031)
Head Start		-0.020 (0.046)	
Food Stamps			-0.032 (0.033)
<i>Panel B: Effects by Percent Urban</i>			
Difference in Triple Interactions: $\beta_4 - \beta_3$			
AFDC	-0.020** (0.008)	-0.021** (0.009)	-0.017** (0.008)
Head Start		0.003 (0.010)	
Food Stamps			-0.011 (0.009)
Observations	83130	83130	83130
Counties	2771	2771	2771
All controls	Y	Y	Y

Notes: This table reports the effects of the AFDC, Head Start, and Food Stamps programs on pandemic-related infant mortality. The table reports the difference in the triple interaction coefficient estimates ($\beta_4 - \beta_3$) based on equation (1). Head Start and Food Stamps are indicators for states with above median increases in per capita program funding from 1963 and 1970. All regressions include the full set of controls reported in Table 1. Standard errors clustered at the county level are reported in parentheses. *** denotes statistical significance at the 1 percent level, ** at the 5 percent level, and * at the 10 percent level.

Table 3: Pandemic Mortality by Age and Race

	Dependent Variable: Infant Mortality Rate				
	By Age				By Race
	Day 1 (1)	Day 2-27 (2)	Day 28+ (3)	Year 1 (4)	(5)
<i>Panel A: Effects by Coal Capacity</i>					
P 1968-69 x AFDC x Coal	-0.049*** (0.015)	-0.014 (0.009)	0.002 (0.007)	-0.055*** (0.018)	
x White					-0.037** (0.018)
x Non-white					-0.086** (0.039)
<i>Panel B: Effects by Percent Urban</i>					
P 1968-69 x AFDC x Pct Urban	-0.011** (0.004)	-0.001 (0.003)	-0.004* (0.002)	-0.023*** (0.006)	
x White					-0.011* (0.006)
x Non-white					-0.058*** (0.020)
<i>Panel C: Effects by Coal Capacity and Percent Urban</i>					
P 1968-69 x AFDC x Coal	-0.039** (0.016)	-0.014 (0.010)	0.011 (0.008)	-0.031 (0.020)	
x White					-0.038* (0.020)
x Non-white					-0.006 (0.046)
P 1968-69 x AFDC x Pct Urban	-0.005 (0.005)	0.000 (0.004)	-0.005* (0.003)	-0.018*** (0.006)	
x White					-0.007 (0.007)
x Non-white					-0.067*** (0.023)
Dep Var: Mean	7.7	5.9	4.3	23.6	16.8 (White) 27.6 (Non-white)
Coal Capacity: Mean (S.D.)			5.5 (8.8)		
Percent Urban: Mean (S.D.)			69.8 (28.2)		
Observations	49837	49837	49837	49837	70697
Counties	2769	2769	2769	2769	2769
Full Controls	Y	Y	Y	Y	Y

Notes: This table reports regressions on infant mortality by age of death and by race. County-level data on infant mortality by race and by age are available beginning in 1960 and 1962, respectively. Panel A reports the triple interactions estimates of β_4 for coal capacity, Panel B reports the estimates for percent urban, and Panel C reports the estimates from models that include both coal capacity and percent urban. All regressions report the full controls described in Table 1. Standard errors clustered at the county level are reported in parentheses. *** denotes statistical significance at the 1 percent level, ** at the 5 percent level, and * at the 10 percent level.

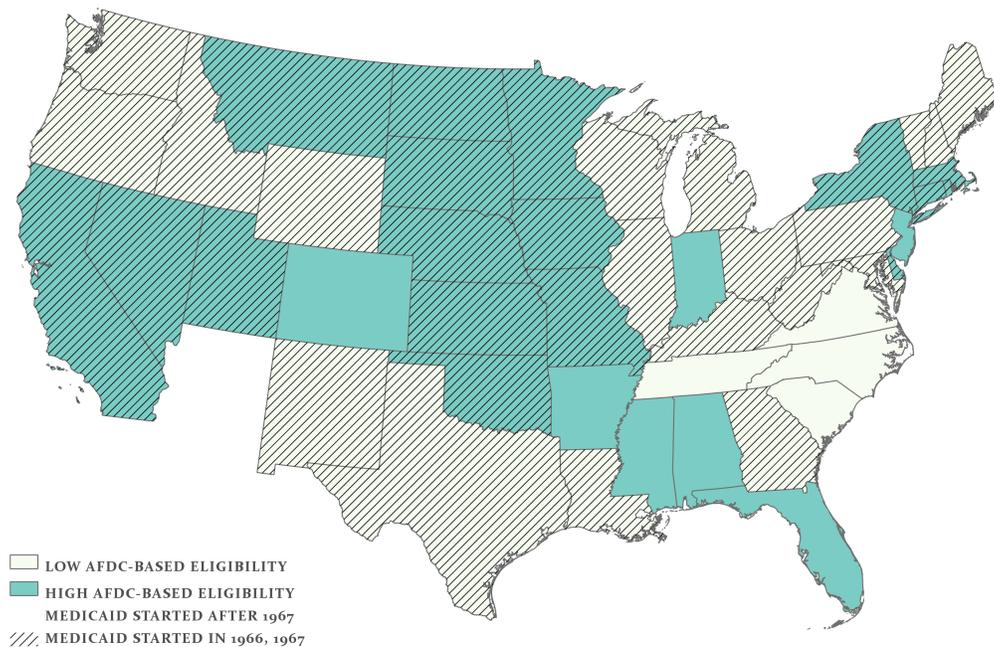
Table 4: Infant Death Averted and Average Treatment Effects on the Treated (ATETs)

	Coal		Coal + Urban	
	β_4	$\beta_4 - \beta_3$	β_4	$\beta_4 - \beta_3$
	(1)	(2)	(3)	(4)
<i>A. High- vs. Low-AFDC Pandemic Infant Mortality</i>				
Δ Infant Mortality Rate	-0.390 [-0.616, -0.164]	-0.264 [-0.543, 0.016]	-1.424 [-2.232, -0.616]	-1.357 [-2.553, -0.161]
<i>B. High- vs. Low-AFDC Pandemic Infant Deaths</i>				
Δ Infant Deaths	760 [319, 1201]	514 [-32, 1060]	2777 [1202, 4352]	2646 [315, 4798]
<i>C. Average Treatment Effect on the Treated (ATET)</i>				
Δ IMR per Newly Insured Household	6.79 [4.81, 11.40]	4.59 [3.26, 7.72]	24.79 [17.56, 41.63]	23.62 [16.74, 39.67]

Notes: Panel A reports the implied differentials in the infant mortality rate between high- and low-AFDC states due to the relative declines in excess pandemic infant mortality. Columns 1-2 report the effects based on coal capacity (based on Table 1, col. 2), columns 3-4 report the cumulative effects for coal capacity and percent urban (based on Table 1, col. 6). These estimates are derived by multiply the triple interaction coefficient estimates by average infant exposure to coal capacity and percent urban in high-AFDC states. Panel B reports the implied decrease in total number infant deaths in high-AFDC states based on the reported in Panel A. Panel C reports the proportional effects of Medicaid on the infant mortality rate of newly insured recipients: the average treatment effect on the treated (ATET). These effects were obtained by dividing the reduced form estimates in Panel A by the first-stage relationship between state-level AFDC eligibility and public insurance reciprocity: coefficient (s.e.) = 3.83 (0.94) (Goodman-Bacon, 2018). We derive confidence intervals for Panel C based on bootstrap draws from normal distributions with means and standard deviations equal to the coefficient estimates and standard errors from the reduced-form and first-stage regressions. The 95% confidence intervals are reported in square brackets.

A Appendix

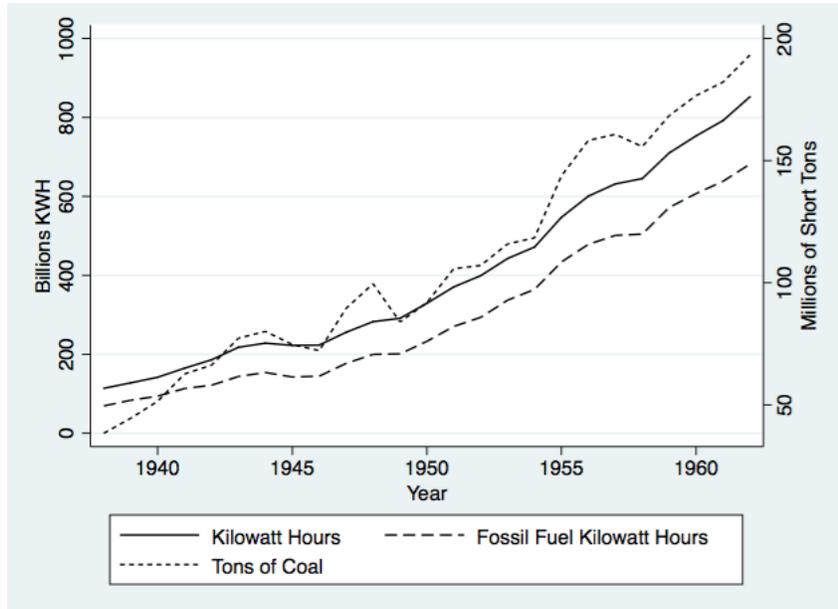
Figure A.1: Exposure to Medicaid Across U.S. States



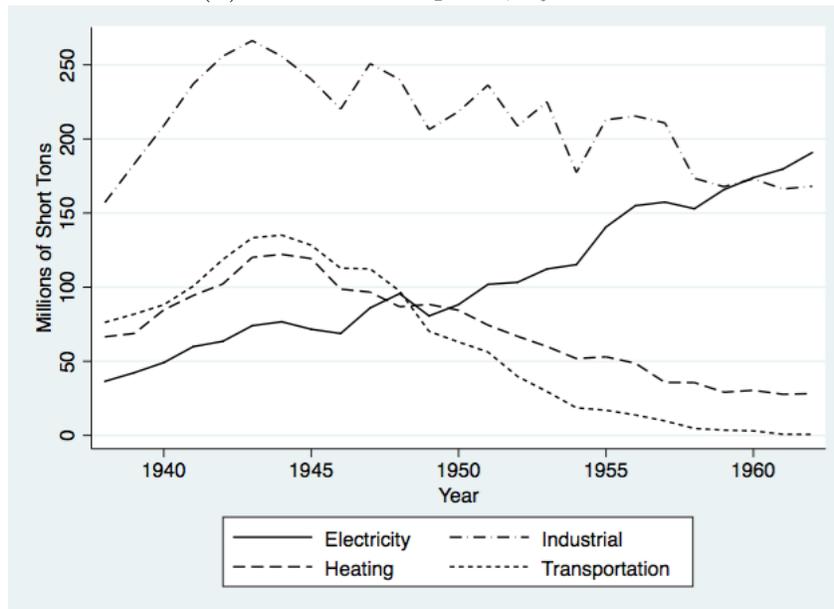
Notes: This map presents high-AFDC (green) and low-AFDC (grey) states by date of Medicaid implementation (dashed line = implementation by 1967, unmarked = implementation after 1967).

Figure A.2: Trends in U.S. Electricity Generation and Coal Consumption

(a) Trends in Electricity Generation



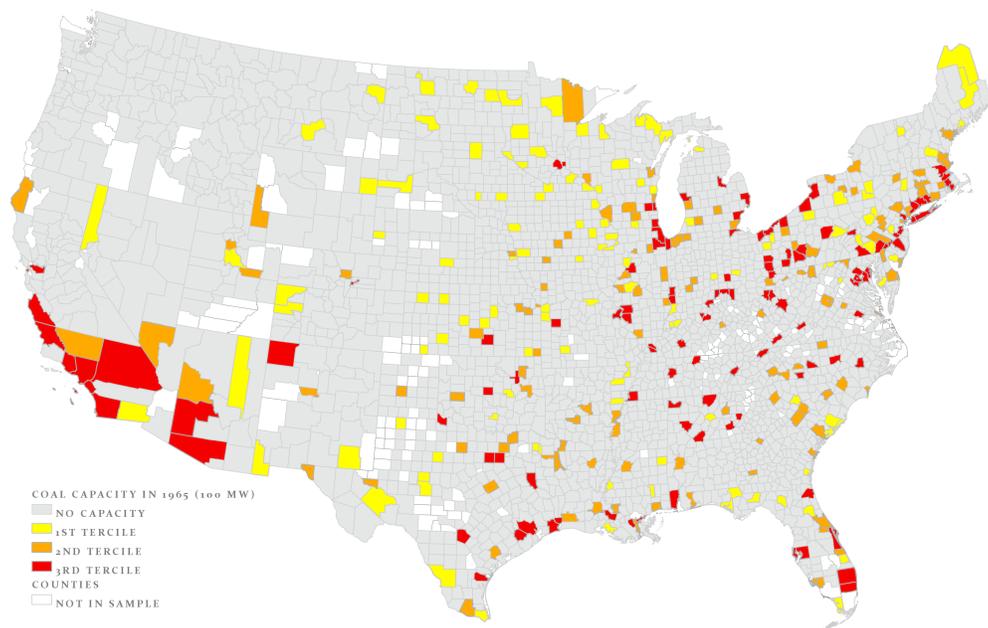
(b) Coal Consumption, by Source



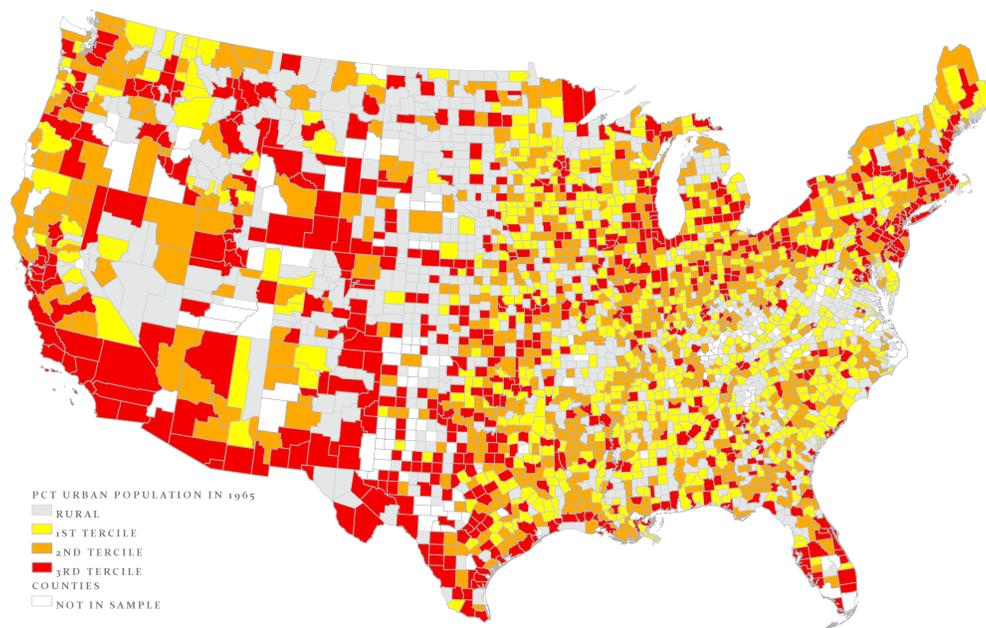
Notes: (a) Data from Gartner (2006), *Historical Statistics of the United States*, Table Db218-227. Electric utilities-power generation and fossil fuel consumption by energy source: 1920-2000. (b) Data from United States Bureau of Mines, *Minerals Yearbook* (various years).

Figure A.3: Variation in Coal-fired Electricity Generating Capacity and Percent Urban

(a) Coal Capacity



(b) Percent Urban



Notes: This map presents the sample counties identified by tertile of coal capacity (panel a) and tertile of percent urban (panel b) in 1965.

Table A.1: Total Suspended Particulates (TSP) Concentration and Coal Capacity

	Dependent variable: Total Suspended Particulates	
	(1)	(2)
Coal capacity (≤ 30 miles)	2.3245** (1.0228)	
Coal capacity (≤ 50 miles)		2.2378*** (0.6451)
Observations	433	433
Counties	85	85
R-squared	0.723	0.753
Mean dep var in 1957		141
Mean dep var in 1962		100
State-by-Year FE	Y	Y
Geographic Controls	Y	Y

Notes: This table reports the relationship between coal-fired electricity generating capacity and total suspended particulates (TSP), a measure of particulate matter collected by the EPA for the period 1957-1962. Coal capacity measures total coal-fired generating capacity within x miles of the county centroid. Geographic controls include percent urban, percent employed in manufacturing, percent non-white, and climatic controls.

Table A.2: Summary Statistics

	1950	1960	1970	All Years (1950-1979)
Infant Mortality Rate(per 1,000 births)				
All	29.34 (9.591)	25.81 (7.284)	19.93 (5.582)	23.60 (13.737)
Non-white			28.99 (34.29)	27.62 (26.67)
White			17.83 (4.793)	16.80 (5.980)
Day 1			8.819 (3.173)	7.703 (3.705)
Day 2-27			6.256 (2.629)	5.901 (2.979)
Post-neonatal			4.163 (2.046)	4.349 (2.712)
Maternal Mortality(per 100,000 adult women)				1.319 (3.619)
AFDC rate for Adult women in the year of Medicaid				
All				2.291 (0.974)
High AFDC states				3.066 (0.780)
Low AFDC states				1.529 (0.335)
Non-white				11.07 (4.241)
White				1.254 (0.675)
Coal Capacity(100 MW)	1.492 (2.985)	4.064 (7.378)	7.050 (11.60)	5.084 (9.659)
Pct Urban Population	60.53 (30.48)	68.75 (28.31)	72.48 (27.41)	68.84 (28.50)

Notes: This table presents summary statistics for variables included in the analysis. All variables are weighted by live births. AFDC Rates are women aged 18 to 44, evaluated at the year of Medicaid implementation.

Table A.3: Pre-trend Analysis

	(1)	(2)	(3)	(4)
	Obs	Mean	Pre-Med	AFDC
<i>A. IMR, Coal, Climate Var (1950-1965)</i>		at 1965	AFDC	x (Yr - 1965)
IMR (per 1,000 births)	630	25.0	1.603 (1.042)	0.024 (0.039)
Coal Capacity (100 MW)	630	1.0	0.248 (0.172)	0.017 (0.011)
Annual Average Temperature (F)	630	12.2	0.949 (0.932)	-0.010 (0.010)
Annual Precipitation (mm)	630	887.3	56.32 (49.69)	-2.60 (2.24)
Annual Absolute Humidity (g/kg)	630	6.4	0.308 (0.351)	0.008 (0.010)
Per capita Hospital Beds	630	3.1	-0.024 (0.135)	0.003 (0.004)
		Mean	Pre-Med	AFDC
<i>B. Census Demographics (1950, 1960)</i>		at 1960	AFDC	x (Yr - 1960)
Pct Manufacturing Employment	90	22.5	0.118 (1.888)	-0.016 (0.066)
Population Density	90	152.0	49.15 (53.70)	1.54 (1.30)
Pct Urban Pop	90	68.0	0.150 (3.433)	0.129 (0.097)
Pct White	90	89.7	-2.716 (1.826)	0.080* (0.047)
Pct 25yrs+ w/ High School	90	0.55	-0.022 (0.025)	-0.001 (0.002)
Median Housing Income	90	37000	212.8 (2016.3)	96.6 (65.4)

Notes: The table presents results from balancing tests for correlation between baseline AFDC rates and trends and levels in pre-1965 state outcomes. The model is: $y_{st} = \alpha + \beta_0 AFDC_s^* + \beta_1 AFDC_s^* \times (Year - Year_{pre}) + \mu_{st}$. Year 1965 is the latest pre-Medicaid year ($Year_{pre}$) except in panel B (1960) and panel C (1950). β_0 is the relationship between pre-Medicaid AFDC level and levels of each characteristics. β_1 is the relationship between pre-Medicaid AFDC and linear trends of each variable.

Table A.4: Main Estimates with Standard Errors Clustered at the State Level

	Dependent Variable: Infant Mortality Rate					
	(1)	(2)	(3)	(4)	(5)	(6)
P 1957-58 x Coal	0.066*** (0.022)	0.066*** (0.023)			0.053* (0.028)	0.053* (0.029)
P 1957-58 x AFDC x Coal	-0.022 (0.020)	-0.023 (0.022)			-0.017 (0.026)	-0.018 (0.028)
P 1968-69 x Coal	0.049** (0.023)	0.059** (0.025)			0.036 (0.027)	0.050* (0.029)
P 1968-69 x AFDC x Coal	-0.066*** (0.024)	-0.071** (0.028)			-0.045 (0.028)	-0.050 (0.032)
P 1957-58 x Pct Urban			0.015*** (0.004)	0.015*** (0.004)	0.007 (0.006)	0.007 (0.006)
P 1957-58 x AFDC x Pct Urban			-0.004 (0.007)	-0.004 (0.007)	0.000 (0.008)	0.000 (0.008)
P 1968-69 x Pct Urban			0.017** (0.006)	0.015*** (0.005)	0.010** (0.004)	0.007* (0.004)
P 1968-69 x AFDC x Pct Urban			-0.023*** (0.006)	-0.024*** (0.007)	-0.016** (0.007)	-0.016** (0.008)
$\beta_3=\beta_4$ (coal)	.006	.005			.139	.108
$\beta_3=\beta_4$ (urban)			.083	.058	.152	.135
Observations	83130	83130	83130	83130	83130	83130
Counties	2771	2771	2771	2771	2771	2771
Adj. R-Squared	.629	.632	.630	.633	.630	.633
Baseline Controls	Y	Y	Y	Y	Y	Y
Full Controls		Y		Y		Y

Notes: This table reports the coefficient estimates from Table 1 with standard error clustered at the state level. *** denotes statistical significance at the 1 percent level, ** at the 5 percent level, and * at the 10 percent level.

Table A.5: Medicaid and Pandemic Infant Mortality: Progressive Inclusion of Covariates

	Dependent Variable: Infant Mortality Rate							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
P 1957-58 x Coal	0.070*** (0.021)	0.067*** (0.020)	0.066*** (0.021)	0.066*** (0.021)	0.056** (0.025)	0.053** (0.023)	0.053** (0.024)	0.053** (0.024)
P 1957-58 x AFDC x Coal	-0.034 (0.025)	-0.025 (0.024)	-0.022 (0.024)	-0.023 (0.024)	-0.028 (0.029)	-0.019 (0.027)	-0.017 (0.027)	-0.018 (0.028)
P 1968-69 x Coal	0.030** (0.014)	0.030** (0.014)	0.049*** (0.015)	0.059*** (0.016)	0.033** (0.015)	0.034** (0.015)	0.036** (0.016)	0.050*** (0.018)
P 1968-69 x AFDC x Coal	-0.056*** (0.019)	-0.062*** (0.019)	-0.066*** (0.019)	-0.071*** (0.021)	-0.038* (0.020)	-0.043** (0.020)	-0.045** (0.020)	-0.050** (0.023)
$\beta_3 = \beta_4$ (coal)	.401	.155	.097	.065				
P 1957-58 x Pct Urban Pop	0.016*** (0.005)	0.015*** (0.005)	0.015*** (0.005)	0.015*** (0.005)	0.007 (0.005)	0.007 (0.005)	0.007 (0.005)	0.007 (0.005)
P 1957-58 x AFDC x Pct Urban	-0.005 (0.007)	-0.005 (0.007)	-0.004 (0.007)	-0.004 (0.007)	0.000 (0.007)	-0.000 (0.007)	0.000 (0.007)	0.000 (0.007)
P 1968-69 x Pct Urban	0.004 (0.004)	0.004 (0.004)	0.017*** (0.005)	0.015*** (0.005)	-0.002 (0.004)	-0.002 (0.005)	0.010** (0.005)	0.007 (0.005)
P 1968-69 x AFDC x Pct Urban	-0.025*** (0.006)	-0.026*** (0.006)	-0.023*** (0.006)	-0.024*** (0.006)	-0.019*** (0.006)	-0.019*** (0.006)	-0.016** (0.006)	-0.016** (0.006)
$\beta_3 = \beta_4$ (coal)					.753	.416	.345	.277
$\beta_3 = \beta_4$ (urban)	.023	.017	.035	.019	.039	.047	.096	.076
Observations	83130	83130	83130	83130	83130	83130	83130	83130
Counties	2771	2771	2771	2771	2771	2771	2771	2771
State x Year, County FE	Y	Y	Y	Y	Y	Y	Y	Y
Annual Climate Vars		Y	Y	Y		Y	Y	Y
Socioeconomic			Y	Y			Y	Y
Full Controls				Y				Y

Notes: This table reports estimates of equation (1), progressively including covariates. *** denotes statistical significance at the 1 percent level, ** at the 5 percent level, and * at the 10 percent level.

Table A.6: Robustness Exercises

	Dependent Variable: Infant Mortality Rate				
	Baseline estimates	Medicaid implementation by 1967	Coal capacity > 0	Urban pop. > 0	Unbalanced sample
	(1)	(2)	(3)	(4)	(5)
P 1957-58 x Coal	0.066*** (0.021)	0.063*** (0.021)	0.084*** (0.023)	0.066*** (0.021)	0.065*** (0.021)
P 1957-58 x AFDC x Coal	-0.023 (0.024)	-0.015 (0.024)	-0.053* (0.030)	-0.022 (0.024)	-0.027 (0.025)
P 1968-69 x Coal	0.059*** (0.016)	0.059*** (0.017)	0.075*** (0.020)	0.059*** (0.016)	0.056*** (0.016)
P 1968-69 x AFDC x Coal	-0.071*** (0.021)	-0.070*** (0.022)	-0.103*** (0.024)	-0.068*** (0.021)	-0.067*** (0.022)
$\beta_3=\beta_4$.065	.036	.093	.068	.115
P 1957-58 x Urban	0.015*** (0.005)	0.016*** (0.005)	0.028*** (0.008)	0.016*** (0.006)	0.015*** (0.005)
P 1957-58 x AFDC x Urban	-0.004 (0.007)	-0.010 (0.007)	-0.011 (0.012)	0.001 (0.008)	-0.000 (0.007)
P 1968-69 x Urban	0.015*** (0.005)	0.016*** (0.005)	0.026*** (0.008)	0.015*** (0.005)	0.012*** (0.004)
P 1968-69 x AFDC x Urban	-0.024*** (0.006)	-0.025*** (0.007)	-0.034*** (0.011)	-0.021*** (0.007)	-0.022*** (0.006)
$\beta_3=\beta_4$.019	.117	.123	.026	.010
Observations	83130	62790	20010	56010	86305
Counties	2771	2093	667	1867	2880
All Controls	Y	Y	Y	Y	Y (partial)

Notes: This table reports the coefficients of several robustness exercises. Column (1) reports the baseline estimates from column 2 and column 4 in Table 1. Column 2 reports results for the subsample of 34 states that implemented Medicaid by 1967. Columns 3 and 4 shows results for counties with positive coal capacity and positive urban population. Column 5 includes an additional 109 counties (in Ma and VA) which either have missing information on infant mortality (in various non-pandemic years) or lack information on hospital beds per capita or median family income. These regressions exclude the latter two variables as covariates.

Table A.7: Medicaid and Pandemic Maternal Mortality

	Dependent Variable: Maternal Mortality Rate					
	(1)	(2)	(3)	(4)	(5)	(6)
P 1968-69 x Coal	-0.003 (0.010)	0.003 (0.010)			-0.010 (0.011)	-0.009 (0.010)
P 1968-69 x AFDC x Coal	0.014 (0.011)	0.008 (0.011)			0.018 (0.012)	0.014 (0.012)
P 1968-69 x Pct Urban			0.003 (0.003)	0.004 (0.003)	0.004 (0.003)	0.005 (0.003)
P 1968-69 x AFDC x Pct Urban			0.002 (0.004)	0.002 (0.004)	-0.000 (0.004)	0.000 (0.004)
Dep Var: Mean (S.D.)			1.3 (3.6)			
Coal Capacity: Mean (S.D.)			5.5 (8.8)			
Percent Urban: Mean (S.D.)			69.8 (28.2)			
Observations	49842	49842	49842	49842	49842	49842
Counties	2769	2769	2769	2769	2769	2769
Adj. R-Squared	.151	.172	.151	.173	.151	.173
Baseline Controls	Y	Y	Y	Y	Y	Y
Full Controls		Y		Y		Y

Notes: This table reports regressions for maternal mortality, defined as the number of women's death related to pregnancy per 100,000 women aged 15 to 54. Data on maternal mortality is available beginning in 1962. The specifications correspond to Table 1.