Title

Essays on the effects of prenatal environmental factors on newborn’s health and the effectiveness of health care in lessening them.

Author

Ospino Rojas, Anderson

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Essays on the effects of prenatal environmental factors on newborn's health and the effectiveness of health care in lessening them.

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ANDERSON OSPINO ROJAS
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Approved:

_____
Marianne Bitler, Co-Chair

_____
Katrina Jessoe, Co-Chair

_____
Kevin Novan

Committee in Charge

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Abstract

Environmental and epidemiological research has linked exposure to air pollution and extreme temperatures during the prenatal period to the incidence of fetal and infant death, adverse birth outcomes, and worse outcomes in adulthood and across generations. To minimize these adverse effects, policymakers can reduce ambient exposures (i.e., mitigation) or intervene with some program that lessens the impacts (i.e., adaptation). The first two chapters focus on the effects of air pollution shocks and the efficacy of free prenatal care to lessen its adverse effects on health at birth. The last chapter focuses on the effects of extreme temperatures and the efficacy of free prenatal care and air conditioning to lessen its adverse effects on birth outcomes (e.g., birthweight, weeks of gestation, low birthweight rate).

The first chapter shows that the estimates of sulfur dioxide (SO_2) effects on birth outcomes are susceptible to the window used to measure exposure during the prenatal period. Measuring exposure from conception to birth, I find a negative impact of SO_2 on birthweight. In contrast, the estimate is positive when exposure is measured from conception to 39 weeks. Using each county's 52-week lagged SO_2 concentrations as a placebo, I find that using a fixed 39-week window from the date of conception is the most reliable methodology. However, this methodology's estimates indicate that higher SO_2 concentrations increase birth weight. I present evidence suggesting that this counterintuitive result is caused by livebirth bias (i.e., the infants that survive pollution shocks are positively selected). I overcome this problem by using the number of infants born with non-adverse outcomes per woman of reproductive age as the dependent variable instead of traditional outcomes (e.g., birthweight, low birthweight, or preterm birth rate). Applying this transformation, I find that SO_2 worsens health at birth, and its effects increase with the pollutant's concentration (i.e., the SO_2-birth outcome damage function is convex). Furthermore, the effects are more prominent for blacks than whites.

The second chapter tests whether access to free prenatal care lessens the adverse health effects of exposure to air pollution in utero. I study how the expansion of Medicaid (publicly-provided health insurance for low-income households) changed the effect of prenatal exposure to SO_2 on fetal

death and birth outcomes. Theoretically, the effect is ambiguous: Even if free prenatal supplementation (i.e., vitamins, iron, calcium) lessens the biological impact of air pollution, there could be a substitution between access to prenatal care and pollution avoidance. High SO_2 concentrations increased fetal deaths, and Medicaid's expansion attenuated this effect. Estimating the impact of Medicaid on the SO_2-birth outcome relationship is empirically challenging because the infants marginally saved by Medicaid could be positively or negatively selected. The analysis of traditional outcome variables (e.g., birthweight, low birthweight rate) suggests that Medicaid had no impact or even intensified the damage of SO_2 on health at birth. To account for the possibility of livebirth (i.e, sample selection) bias, I instead analyzed the number of non-low birth weight (i.e., healthy) infants per woman of reproductive age (*nlbw/w*). Using this dependent variable, I find that Medicaid mitigated the effect of SO_2 on *nlbw/w* in low-pollution areas and at the national level. Furthermore, the reduction was larger for blacks than whites; thus, Medicaid improved environmental justice in the US by shrinking the gap in the health effects of in-utero air pollution between races.

The third chapter tests whether access to free prenatal care and air conditioning lessens the adverse health effects of extreme temperatures in a non-rural setting. I study how the expansion of Medicaid changed the effect of extreme in-utero temperatures on birth outcomes in the US. In developed countries, physiological stress is the primary mechanism through which temperature affects pregnancy outcomes. In rural areas of the developing world, it can also do so indirectly through changes in real income, increased incidence of maternal disease, or increased conflicts. The results suggest that access to prenatal care did not lessen the impacts of extreme temperatures on birth outcomes. However, the diffusion of air conditioning reduced the effects of extremely hot days.

Overall, the results of these chapters suggest that providing low-income women with free prenatal care is a promising intervention to lessen the health impacts of in-utero air pollution but not those of extreme temperatures. On the other hand, air conditioning is a promising intervention to lessen the health effects of extreme heat on birth outcomes.

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Contents

Essay 1

Does prenatal air pollution affect fetal death and birth outcomes?

1.1 Introduction

Estimating the effects of air pollution on infant health is critical to identifying the concentrations that put human health at risk and the design of environmental regulation. The effects of prenatal air pollution on infants' health have been studied using various methodologies; however, there is little research on how sensitive the pollutant effects' estimates are to the window used to measure exposure. The results from this paper show that the estimates can be highly sensitive to the methodology used to measure exposure during the prenatal period. Measuring exposure from conception to birth, I find a negative impact of sulfur dioxide (SO_2) on birthweight. In contrast, the estimate was positive when exposure was measured from conception to 39 weeks; both estimates are statistically significant. This paper explores the reasons behind this disparity and its implications.

This paper's main objective is to examine how sensitive the estimates of the pollutant's effects on birth outcomes are to the window used to measure exposure. As a complement to this objective, I also implemented a placebo test to choose the most reliable methodology. This test revealed that measuring exposure from conception to birth did not generate reliable estimates of the pollutant's effects. However, using the most reliable methodology (i.e., conception-39 weeks), I found an

unanticipated and counterintuitive result: high SO_2 concentrations were associated with higher birthweight. In theory, this result could be explained by at least two factors: endogeneity or livebirth (i.e., sample selection) bias. As a secondary objective, I try to determine the source of this bias and address it. I claim that the livebirth bias is the cause and present supporting evidence, and I propose a novel methodology to overcome the livebirth bias.

The empirical strategy relies on restricted US birth certificate data from the Center for Disease Control and Prevention (CDC) for 1982-1991, daily air pollution monitoring data from the Environmental Protection Agency (EPA), daily weather data taken from Deschenes & Greenstone (2011), and the monthly state unemployment rate from the Bureau of Economic Analysis (BEA). The effects of air pollution on fetal death and birth outcomes are identified using a panel fixed effects model that exploits changes in exposure to different concentration bins during the prenatal period. I include county-year-race fixed effects— thus, the effects are identified from changes across infants conceived within the same county, year, and race. Seasonal county-quarter-race and month-race fixed effects are also included. I control for in-utero weather (temperature and precipitation), the state's unemployment rate during the pregnancy's first trimester, and demographics (mother's pregnancy history, marital status, high school dropout status, and newborn's gender). For the birth outcome model, pollution is measured from the presumed date of conception1 to birth or over a fixed 39-week window starting from conception. For the fetal death model, only the latter is considered.

[1] Conception is assumed to be 15 days after the last menstrual period. It is the standard assumption in this literature.

As a first result, I find that sulfur dioxide (SO2), a precursor of fine particulate matter (PM2.5), measured over a fixed 39-week window increased fetal deaths in low-pollution areas2 but not in highly polluted ones. In low-pollution counties, an additional day in SO2's second most polluted concentration bin3 during the pregnancy decreased the fertility rate by 0.029%, implying 1.53 fewer annual births per county.4 The back-of-the-envelope calculation suggests that for the typical low-pollution county, there were 46 fetal deaths associated with SO2 every year. There were no significant effects in highly polluted counties. The null effect for the latter suggests that other factors mediate air pollution's effect on fetal death. For instance, high-pollution counties have more hospital beds per capita and fewer high school dropout women. Both could explain pollution's smaller effect on this group. Alternatively, households in highly polluted counties may be better adapted to pollution. For example, buildings could be better insulated from outdoor pollution, or avoidance may always be higher.

The estimates of the effect of SO_2 on birthweight change drastically with the window used to measure exposure. If measured from conception-birth, an increase of 1 part per billion in the average SO_2 concentration during the prenatal period decreases birth weight by 0.24% (Approximately 8 grams). In contrast, if measured from conception to 39 weeks –as suggested by the fetal shocks literature[5]— birthweight increases by 0.014%. In theory, this latter result could be explained by at least two factors: endogeneity or livebirth bias.

[2] Counties were categorized into high vs. low pollution categories based on the average level of SO_2 over the first two years of data availability in the AQS (Air Quality System).
[3] SO_2 concentration range per quintile (parts per billion): $q1\ \ (0 \leq SO_2 < 1.84)\ \ q2\ \ (1.84 < SO_2 \leq 4.39)$, $q3\ \ (4.39 < SO_2 \leq 7.66)$, $q4\ \ (7.66 < SO_2 \leq 13)$, $q5\ \ (SO_2 > 13)$.
[4] For comparison, there are 4316 annual births per county in the low-pollution group.
[5] See for example, Persson & Rossin-slater (2018), Currie & Rossin-Slater (2013), and Currie et al. (2013)

The livebirth bias – an understudied sample selection bias— arises when the infants that survive prenatal pollution shocks are positively or negatively selected. I find that this bias affects the estimates of Sulfur dioxide's effects on birthweight during 1981-1991 in the US. However, it is uncovered only when pollution exposure is measured from conception to 39 weeks instead of up to the birthdate. The analysis of the fertility rate and left tail of the birthweight distribution were consistent with the hypothesis that the fetuses who die due to SO_2 shocks are negatively selected (i.e., would have been born with low weight). Furthermore, the point estimates associated with low SO_2 concentrations (and potentially lower income) were positive, inconsistent with an omitted-variable-bias story. Thus, it was concluded that livebirth bias is the cause behind the counterintuitive results.

I propose a novel approach to eliminate the livebirth bias from the estimate.[6] The methodology consists of using the number of infants born with non-adverse birth outcomes per woman of reproductive age as a dependent variable –instead of traditional birth outcomes (e.g., birthweight, low birthweight, or premature rates)— and weighting the regressions by the number of women of reproductive age. Using this approach, I find that high SO_2 concentration during the prenatal period worsens health at birth.

Sulfur dioxide concentrations above 7.66 parts per billion (ppb) damaged newborn health outcomes in the US during 1981-1991, and the effects were stronger in low-pollution counties. At the national level, one additional day in the most polluted quintile (SO_2>13 ppb) decreased the rate of non-low birth weight (i.e., healthy) infants per woman of reproductive by 0.0122%.

[6] I also tried to use a bounding approach analogous to Lee (2009), but it was ineffective in this context. After excluding the top and bottom 2% or 5% of observations by birthweight, I still found a positive estimate for the impact of SO_2 on birth outcomes (See section 1.5.2.3).

Consequently, a county's annual number of infants born with a weight above 2500 grams decreased by 38.76 due to SO_2.[7] For counties with an initial low baseline concentration, the rate of non-low birth weight infants per woman of reproductive age decreases by 0.0231% per one additional day in the second dirtiest quintile ($7.66<SO_2<13$ ppb), and there were 73.3 fewer infants born with weight above 2500 grams due to SO_2.

I also estimated the impacts across races and found that the damage caused by SO_2 was larger for black than white newborns. For low pollution counties, one additional day in the fourth and fifth quintiles decreases the rates of non-low birth weight (i.e., healthy) infants per woman of reproductive by 0.0237% and 0.0562% for whites and blacks, respectively. These results are consistent with previous environmental research that found larger air pollution effects on African Americans' infant health than whites.[8]

This paper makes three contributions. First, it contributes to the environmental literature showing that the window used to measure exposure to air pollution during the prenatal period has critical implications for the estimates of the effects of on birth outcomes. This result could be relevant for other pollutants or prenatal shocks in general. Moreover, from the fetal shocks literature, we previously knew that endogeneity could arise when the shock affected the duration of the pregnancy (Persson & Rossin-slater, 2018), or because the probability of experiencing a negative shock during the pregnancy is an strictly increasing function of its duration when prenatal exposure is measured by a binary variable (Currie & Rossin-Slater, 2013; Currie et al., 2013). In this paper,

[7] As a reference, there were 4423 annual births per county at the national level.
[8] See Chay & Greenstone(2003a), Currie & Walker (2011)

exposure was measured by the mean of a continuous variable, and daily air pollution levels can move in either direction; however, endogeneity can still arise because the environmental factor has a trend. Thus, newborns with longer gestation are – on average— mechanically assigned a lower prenatal exposure to the pollutant.

Second, I propose a novel approach to address the livebirth (i.e., sample selection) bias. The methodology consists of using the number of infants born with non-adverse birth outcomes per woman of reproductive age (*nlbw/w*) as dependent variables and weighting the regressions by the number of women of reproductive age. Intuitively, the advantage of this variable is that pollution's effects through the extensive and intensive margin go in the same direction. We start from the assumption that air pollution decreases birthweight (intensive margin). Let us suppose that pollution causes fetal deaths (extensive margin). If the infants who die due to pollution shocks were positively selected, *nlbw/w* would decrease due to the effects through both margins. On the other hand, if the infants were negatively selected, *nlbw/w* would decrease due to the intensive margin effect and would be unaffected by the extensive one.

Third, I departed from the linear effects assumption in the pollution-infant health relationship (Chay & Greenstone., 2003a, 2003b; Currie & Walker, 2011; Currie & Neidell, 2005; Currie et al., 2009) and estimated non-parametric effects by concentration quintiles. Doing so, I uncovered convex pollution-fetal death and pollution-birthweight damage functions (i.e., marginal damages increase with the pollutant's concentration). Additionally, to my knowledge, this paper is the first to analyze the effects of prenatal exposure to SO_2 on health at birth (fetal death and neonatal outcomes) for counties of all sizes in the US. Previous studies have analyzed the impacts of

pollution on infant health only in the largest counties (Woodruff et al., 1997; Woodruff et al., 2008). I could include the small counties in the analysis by using restricted vital statistic data.

The remainder of the paper provides background (Section 2), describes the data used to estimate the results (Section 3), discusses the empirical methods (Section 4), lays out the results (Section 5), and concludes (Section 6).

1.2 Background

This section summarizes the relevant concepts to understand air pollution and its societal impact. It discusses why finding unbiased estimates of the effects of pollution on human health is essential, reviews the methodological approaches to measure prenatal exposure, discusses the potential biases when estimating the effects of air pollution, and reviews previous literature on the effects of sulfur dioxide and particulate matter on infant health.

1.2.1 Air pollution, societal impacts and regulation

Air pollution has significant impacts on society, including adverse effects on human health and the economy. Exposure to polluted air has been linked to respiratory diseases, cardiovascular problems, and increased cancer risk (World Health Organization, 2016). These impacts on health result in higher healthcare costs, reduced productivity, and premature deaths. Vulnerable populations, such as children, the elderly, and low-income communities, are particularly affected (Bell & Ebisu, 2012). Economically, air pollution leads to decreased labor productivity, increased healthcare expenses, and damage to agriculture. The World Bank estimated that the global cost of

air pollution in 2016 amounted to $5.7 trillion, equivalent to 4.4% of global GDP (World Bank, 2016).

Many governments around the world regulate air pollution. In the US, the Environmental Protection Agency (EPA) regulates air pollution primarily to protect public health and the environment. The EPA uses the estimates of the impacts of air pollutants on human health as input for designing environmental regulations (US EPA, 1982). Thus, biased estimates could impose high costs on human health.

1.2.2 Methodologies to measure prenatal exposure

This section introduces the methodologies most commonly used by researchers to measure ambient pollution exposure during the gestational period. The universe of methodologies can be summarized across two dimensions: Functional form and the window to measure exposure. Broadly, two functional forms have been used: parametric approaches assuming linearity in the pollutant's effects and non-parametric approaches allowing for non-linear effects. As for the window, three major approaches have been used: from conception to birthdate,[9] from conception to full length,[10] or backward 39 weeks from the birthdate. In many cases, the choice has been driven by data availability since the date of the last menstrual period (LMP) is not always observed by the researcher. The date of conception is typically assumed to be 15 days after the LMP. Importantly, to the best of my knowledge, there is no previous research comparing the estimates of air pollution effects across these methodologies.

[9] This methodology also includes hybrid approaches like inferring the conception date from the birthdate and the doctor's estimated gestational age and measuring exposure from such presumed gestational age to the birthdate.
[10] A 39-week window was assumed for a full length pregnancy.

A large share of the epidemiological literature estimates exposure from conception to birthdate; however, the fetal origins literature suggests using a 39-week window, irrespective of the newborn's birthdate, because prenatal shocks could affect the duration of the pregnancy. Thus, the estimate of the shock's effect would be biased. This latter strategy has been used in multiple studies to analyze the effects of shocks during the prenatal period on birth outcomes (Persson & Rossin-Slater, 2018; Currie & Rossin-Slater, 2013; Currie et al., 2013).

In addition, even if pregnancy duration is not affected, the estimate can be biased when there is a trend in the pollutant's concentration. Measuring exposure from conception to birthdate can induce a mechanical correlation between gestational age and average exposure. For example, suppose the pollutant concentration has a decreasing trend. If this is the case, newborns with ex-ante better health (thus, a longer gestational age) will be assigned a lower level of in-utero pollution. Therefore, gestational age and prenatal pollution would be negatively correlated by construction. This would lead to overestimating the pollutant's effect on neonatal outcomes directly proportional to the gestational age (e.g., birth weight). Likewise, measuring exposure 39 weeks backward from the birthdate would be subject to the same bias because newborns with ex-ante better health would be assigned a lower exposure.[11]

On the other hand, using a 39-week window could introduce a different bias because some pregnancies don't reach full term; so, they will be assigned pollution measures that contain some days (or weeks) after birth. Ultimately, there is ex-ante a tradeoff when choosing the window to

[11] The point estimates generated using the conception -full length and 39-weeks backward from the birthdate methodologies are very similar. results not shown, available upon request.

measure exposure: When the conception-birthdate window is used, pollution exposure could be endogenous. On the other hand, the noise introduced in pollution exposure when a fixed 39-week window is used for all newborns could also bias the estimate. In this paper's empirical strategy section, I present the test used as a criterion to choose between both methodologies.

1.2.3 Potential biases estimating the health effects of pollution

Estimating the effects of air pollution on health outcomes is subject to at least two sources of bias: (i) omitted variable bias due to unobserved determinants of health correlated with pollution and (ii) bias due to measurement error. Moreover, estimating the effects of prenatal pollution on birth outcomes adds another source of bias: the livebirth bias is a type of sample selection bias that arises when pollution affects the fetuses' survival rate.

Studying the effects of prenatal interventions or shocks on infant health is challenging due to the livebirth bias. The problem arises because the intervention (or shock) can affect birth outcomes through two channels. On the extensive margin, it can change the number of pregnancies ending in live births; on the intensive one, it can affect the health outcomes of the newborns that would have been born, irrespective of the intervention. If an intervention (shock) reduces the incidence of fetal death and the marginal infants saved are positively or negatively selected, the estimate will be biased. It can even lead to a non-causal association between exposure and outcomes (Nguyen, 2020).

This problem is relevant for applied public and environmental economics. For instance, Currie & Moretti (2006) found that food stamps – a program that provides low-income households with

vouchers to buy food -increased the probability of low birth weight in California from 1960 to 1974. Adverse environmental shocks have also been associated with health improvements; for example, Raz et al. (2018) found that prenatal air pollution reduced the risk of autism. Both findings are counterintuitive, raising concerns that another type of bias is at play (e.g., omitted variable bias). The potential positive correlation between income and pollution is a significant concern in environmental-health economics because income data are generally unavailable to the researcher.

Previous research estimating the effects of prenatal air pollution on birth outcomes has focused on addressing omitted variable and attenuation bias. Different quasi-experimental research designs (i.e., difference-in-difference, instrumental variables, regression discontinuity) have been used to exploit plausible sources of exogenous variation in pollution. The possibility of livebirth bias has been acknowledged but has yet to be addressed. It is particularly challenging to do so because fetal death data in the US was underreported during the early 80s (NCHS, 1985; Kleinman, 1986). Moreover, traditional bounding methodologies to address sample selection, such as Lee (2009), are better suited for parametric functional forms that impose linearity in the effects. Thus, there is no straightforward recipe for addressing sample selection bias when using a non-parametric approach to estimate the non-linear effects of pollution. In the empirical strategy section, I propose a new approach to address a potential livebirth bias while simultaneously estimating the non-linear effects of the pollutant on birth outcomes.

1.2.4 Effects of sulfur oxides and particulate matter on infant health

Sulfur oxide (SO_2) is an umbrella term used to designate many oxygen-sulfur compounds, some gaseous and other solid; they are typically produced by burning fossil fuels (e.g., coal, oil). In the US, sulfur dioxide, SO_2, is the only compound occurring at significant atmospheric concentrations (US EPA, 1982). Hence, SO_2 has been measured and regulated as a surrogate for all sulfur oxides. In addition, SO_2 reacts with other compounds in the atmosphere and contributes to acid rain and particulate matter[12] formation. Therefore, in 1982, the EPA, following the recommendations of the World Health Organization, started updating the air quality criteria of particulate matter and sulfur oxides jointly. This decision was justified for two reasons. First, chemical processes in the atmosphere transform significant amounts of gaseous sulfur dioxide into particulate sulfate.[13] Second, it is difficult to separate the relative contributions of particulate matter and sulfur oxide on the mortality and morbidity effects observed in epidemiological studies (US EPA, 1982).

The difficulty separating sulfur oxide from particulate matter's effects remains because few stations monitored particulate matter during the early 80s. The first records for PM_{10} appeared in 1983 (21 counties). The monitoring network kept growing until reaching 48 in 1984 and 244 in 1985. Thus, sulfur dioxide is a surrogate for sulfur oxides and particulate matter derived from its oxidation in the atmosphere in this paper.

Prenatal exposure to sulfur dioxide affects health at birth; however, the direct biological pathways are not well understood. Previous research found effects on preterm birth (Sagiv et al., 2015), low

[12] Also known as particulate pollution. Defined as airborne solid particles and low vapor pressure liquid droplets with an effective diameter lower than a few hundred micrometers (US EPA , 1982)

[13] Sulfates are a family of chemicals that contain the fully oxidized ionic form of sulfur (SO_4^{-2}), in combination with metal and/or hydrogen ions (US EPA, 1982)

birth weight (Maisonet et al., 2001; Bell et al., 2007), very low birth weight (Rogers et al., 2000), and fetal death (Faiz et al., 2012). Most of these papers have analyzed small geographical areas of the US.[14] In addition, experiments randomizing pollution exposure among mice have found that high concentrations of sulfur dioxide during the gestational period were associated with decreased offspring birthweight (Singh, 1982). In humans, inhaled sulfur is known to enter the blood rapidly after exposure, but there is no evidence of placental transfer of sulfur inhaled as SO_2 (Rogers et al., 2000). Thus, sulfur dioxide may indirectly hurt the fetus through the health of the mother.

Prenatal exposure to particulate matter[15] also worsens birth outcomes. Previous research has found effects on birthweight (Colmer et al., 2021; Basu et al., 2004), preterm birth (Ritz et al., 2000; Huynh et al., 2006; Sagiv et al., 2015), low birth weight (Parker et al., 2005), very low birth weight (Rogers & Dunlop., 2006), and fetal death (Ebisu et al.,2018; DeFranco et al., 2015). Prenatal exposure to total suspended particles (TSP) has been linked to post-natal outcomes: infant mortality (Chay & Greenstone, 2003a, 2003b). Fine particulate matter ($PM_{2.5}$) is the most damaging to human health because these smaller particles can enter the bloodstream and spread through the whole body (Xu et al., 2008; Wang et al., 2013). However, the biological mechanisms that affect a fetus's health are poorly understood (Feng et al., 2016; Kanaan et al., 2006).

$PM_{2.5}$ is a mixture of many chemical constituents with different toxicities, which vary spatially and temporally (Bell et al., 2007). Cutting-edge research on the effects of $PM_{2.5}$ on human health focuses on analyzing the effects of its constituents separately. Among them, the sulfate ion (SO_4^{-2}),

[14] Mainly cities and counties of the eastern US. SO_2 concentrations tend to be higher in the eastern coast due to utilities' burning of fossil fuels for electricity generation.

[15] Different measures have been used: total suspended particles (TSP), particulate matter with diameter less than 10 micrometers (PM_{10}), less than 2.5 micrometers ($PM_{2.5}$), and less than 1 micrometer (PM_1)

derived from the oxidation of sulfur dioxide in the atmosphere, has been linked to fetal death (Ebisu et al., 2018). Secondary ammonium sulfate[16] ($(NH_4)_2SO_4$) has been linked to term low birth weight[17] (Bell et al., 2010; Ng et al., 2017) and increased risk of anemia in pregnant women during the third trimester (Xie et al., 2022).

1.3 Data

The empirical analysis relies on multiple data sources. First, birth outcomes and mother's demographics were taken from restricted versions of the national vital statistical files from the National Center for Health Statistics (NCHS). Additionally, data from the Surveillance, Epidemiology and End Results Program (SEER) were used to create measures of birth outcomes per woman of reproductive age (15-44 yr old). Second, I created SO_2, CO, NO_2, O_3, and PM_{10} measures by forecasting and imputing missing obsenrvatios[18] in the Air Quality System (AQS) raw monitoring data from the Environmental Protection Agency (EPA) and aggregating it at the county-day level. Finally, temperature and precipitation in-utero measures were built using county-day weather measures following the methodology by Deschenes & Greenstone (2011), and additional controls at the county level were constructed using data from the Bureau of Economic Analysis (BEA).

1.3.1 Vital statistics.

Natality data files consist of the universe of all live births in the US. These databases are publicly available at the Center for Disease Control and Prevention (CDC). Birthweight, weeks of gestation,

[16] $PM_{2.5}$ secondary ammonium sulfate is not directly emitted, it is formed through photochemical reactions of constituents that are mostly emitted by traffic and power plants (Hasheminassab et al., 2014a).
[17] Defined as birthweight less than 2500 grams, but conditional on the newborn reaching full term (37 weeks or more)
[18] Detailed methodology is presented in Appendix A.

last menstrual period and birth dates, and mother's demographics (age,[19] educational attainment,[20] pregnancy history,[21] marital status, and race (black, white, and other)) were obtained from individual birth certificates. However, from 1989 onwards, the public files do not disclose the county code for counties with a population smaller than 100 thousand according to the last available census.[22] This paper uses the restricted version of the data files for 1989-1991; which was provided by CDC by signing a data use agreement. Hence, the county code is observed for all counties throughout the entire period of analysis. County-level is the smallest geographical unit provided by the CDC; mother's zip code or address are not available in the restricted versions. Observations with missings in the date of conception, birth weight, or gestational age were dropped from the study (3.2%). In addition, non-singleton births were dropped from the sample (2.19%). The national sample contains about 35.6 million births during 1981-1991; Around 49% of them could be assigned prenatal exposure to SO_2 based on the county of maternal residence and conception week. Pollution data was not available for the rest. Summary statistics for birth certificate data is summarized in Table 1.1.

Four sets of measures of health at birth were built using this dataset. First, the fertility rate corresponds to the number of live births per county and week of conception divided by the number of women of reproductive age (i.e., women 15-44 yrs old) per county and year; summarized in Table 1.2. Second, traditional birth outcomes: birthweight, low birth weight (LBW) defined as a

[19] Age was modeled non-linearly with three dummy variables: teen pregnancy (age<=19), middle-age (20<= age<35), risky pregnancy (age>=36).
[20] Not all states reported education since the beginning of the period; consequently, there are many missings. A categorical variable with four categories was created: Highschool dropout (yes, no, not reported, not answered).
[21] Pregnancy history consists of 6 dummy variables based on parity (the total number of deliveries excluding the current one) and the individual fetal death rate (FDR): (1) first delivery, (2) 2nd delivery & FDR=0, (3) parity>=3 & FDR=0, (4) parity>=3 & FDR<=0.5, (5) parity<=2 & FDR=1, (6) parity>=3 & FDR>0.5.
[22] The CDC changed its policy in 1989 to prevent the unintentional disclosure of individuals or institutions. The main changes were: (1) the actual date of birth is not available (just the month and year), and (2) the county code is only reported for counties with a population larger than 100 thousand. In this paper, the exact day of birth is randomly generated from 1989 onwards. This could introduce classical measurement error in pollution exposure and bias the results downward. Robustness tests excluding the period 1989-1991were run, and the results hold.

dummy=1 when birth weight is less than 2500 grams, preterm birth (PTB) defined as a dummy=1 when the gestational age is less than 37 weeks , and small for gestational age (SGA) defined as a dummy=1 when the birth weight is below the 10th percentile of the weight distribution for the respective gestational age, gender, and maternal race of the newborn. All variables are summarized in Table 1.1. Third, health-at-birth measures robust to livebirth bias: *nlbw/w*, *nptb/w*, and *nsga/w* correspond to the number of non-adverse cases (Non-LBW, Non-PTB, and Non-SGA) per county, race, and week of conception divided by the number of women of reproductive age (15-44 years old) in the respective county, race, and year; summarized in Table 1.4. Lastly, the number of infants born with adverse health outcomes per woman of reproductive age (*lbw/w, ptb/w, and sga/w*) are summarized in Table 1.5.

Due to the low quality of fetal death data during the early 80s, the baseline empirical strategy to estimate the effect of pollution uses the fertility rate as a surrogate to indirectly estimate SO_2's effects on the incidence of fetal death. Microdata on fetal death were obtained from the NCHS. This data contains the universe of clinically reported fetal deaths of 20 or more weeks of gestation; however, it is very noisy during the 80s. Fetal deaths of less than 20 weeks of gestation need not be reported, but it is estimated that 70% of all fetal deaths in the US occurred before 20 weeks during the early 80s (NCHS, 1985). Furthermore, there is substantial underreporting of fetal deaths between 20-27 weeks of gestation, and limiting the sample to late fetal death (28 or more weeks of gestation) is not satisfactory (Kleinman, 1986).

Nevertheless, for supplementary analysis, I computed the fetal death rate as the ratio of clinically reported deaths to the sum of live births and fetal deaths conceived in the same county and week.

Table 1.3 presents the summary statistics. The supplementary analysis is discussed in the results section.

Figures 1.1-1.4 show the time series of fertility, low birthweight, preterm birth, and small for gestational age by conception month at the national level and for the counties with a balanced panel of SO_2 data through 1981-1991. The trends and seasonality are very similar across both samples.

1.3.2 Air pollution.

Air pollution measures were created using raw data from the Air Quality System (AQS) at the monitor-day level. The first step consisted of imputing the missings by making forecasts using the rest of the monitors in the same county.[23] The second step consisted of creating county-day measures of pollution based on a balanced panel of monitors within the county. Appendix A describes the methodology used to create county-day CO, SO_2, NO_2, O_3, and PM_{10} measures from 1981-1991. The final step consisted of assigning the prenatal exposure to each pollutant at the individual level based on the county of maternal residence and the newborn's presumed week of conception. Two windows were used to measure exposure : (i) from conception to birthdate, summarized in Table 1.6, and (ii) from conception to 39 weeks, summarized in Table 1.7. The correlations between both measures are shown in Table 1.8. I created two functional forms: (1) the average and (2) the fraction of days in which the county's daily concentration falls in each quintile bin of the national distribution over the respective window. Quintile cutoffs[24] were generated using

[23] This procedure builds up from the strategy used by Auffhammer & Kellogg (2011) to fill the missings at the station-day level in the weather monitoring network

[24] For SO_2, the following cutoffs were used: q_1 ($SO_2 \leq 1.84\ ppb$), $q_2(1.84\ ppb < SO_2 \leq 4.39\ ppb)$, $q_3(4.39\ ppb < SO_2 \leq 7.66\ ppb)$, $q_4(7.66\ ppb < SO_2 \leq 13\ ppb)$, and $q_5(SO_2 > 13\ ppb)$).

all county-day records from 1981-1991. The latter is used to estimate the non-linear effects of air pollution. Lastly, Table 1.12 shows SO_2 concentrations by race.

Figure 1.5 shows the counties with SO_2 data, and Figure 1.6 shows the set of counties with a balanced panel[25] of SO_2 data through 1981-1991. Figure 1.7 shows the average prenatal exposure to SO_2 of a typical 39-week pregnancy nationally and by counties' SO_2 baseline level (low vs. high). Figures 1.8.1 and 1.8.2 show the average time a pregnancy spends in each of SO_2's concentration quintiles by baseline SO_2 level and race, respectively. Figures 1.9.1 and 1.9.2 show the average fraction of days a 39-week pregnancy spends in the fourth and fifth concentration quintiles of SO_2.

1.3.3 Additional controls

Exposure to in-utero temperature and precipitation are modeled non-linearly using the county-day level series from Deschênes, Greenstone & Guryan (2009). For temperature, bins were created using the same thresholds (<25 F, 25-45 F, 45-65 F, 65-85 F, and >85 F); Table 1.11 summarizes the correlations between temperature and SO_2 bins. For precipitation, quintiles were created based on the distribution of daily rainfall during 1981-1991 for the entire US. In-utero weather variables were assigned based on the mother's county of residence and the newborn's week of conception. Exposure is measured by the fraction of days during the chosen window in which the county's mean falls in each bin. Finally, the unemployment rate during the pregnancy's first trimester was built using state-level data from the Bureau of Economic Analysis.

[25] A county belongs to the SO_2 Balanced panel if the fraction of daily missing observations for a 39-week forward looking rolling window was less than 50% every week-year during 1981-1991. This threshold is more stringent than the one used in other studies. For instance Bell et al. (2007) uses 75%.

1.4 Empirical strategy

Broadly, the empirical strategy to identify the effects of air pollution on the fertility rate and birth outcomes relies on the changes in the distribution of ambient levels of air pollution during the pregnancy across newborns conceived in the same county and year. I present two figures to visualize the variation used to identify the effects of air pollution on health. First, Figure 1.10 shows the seasonal variation in sulfur dioxide by conception month. Then, Figure 1.11 shows the variation I used to identify the effects of SO_2 (i.e., residual variation after removing county-year, county-quarter, and calendar month fixed effects).

One crucial remark, this paper does not attempt to estimate the biological effect of air pollution on infant health. Instead, the estimate should be interpreted as the reduced-form effect of ambient air pollution on health after factoring in any possible behavioral response (e.g., pollution avoidance) Thus, the estimates will be smaller than the pollutant's biological effects.

1.4.1 Methodology to estimate the effect on fetal death

$$FR_{cwy} = \alpha_{cy} + \alpha_m + \alpha_{cQ} + \sum_{j\ 1..5,\ \neq 3} \beta_j\ q_j\left(P_{cwy}^{39}\right) + \delta W_{cwy} + \theta\ U^{Q1}{}_{smy} + v_{cwy} \quad [1.1]$$

This model uses the fertility rate (FR) instead of the fetal detah rate (FDR) to indirectly estimate the pollutant's effects on the incidence of fetal death. FR is defined as the number of live births per county (*c*) and week(*w*)-year(*y*) of conception divided by the number of women of reproductive

age (i.e., women 15-44 yrs old) per county and year.[26] FDR corresponds to the ratio of clinically reported fetal deaths to the sum of live births and clinically reported fetal deaths conceived in the same county and week-year. Model [1.1] recovers the effect of pollution on fetal death under some conditions. First, pregnancy decisions in *t* do not react to sulfur dioxide shocks in $t+s$, $s>0$. Second, abortions do not correlate with the pollutant's concentration after controlling for the unemployment rate during the first trimester.

Model [1.1] includes a wide range of fixed effects: County-by-year fixed effects (α_{cy}) control for fixed unobserved factors and limit the potential bias caused by migration driven by environmental quality concerns.[27] Monthly seasonal fixed effects (α_m) control for national-level seasonality in fertility decisions and environmental factors (Currie & Schwandt., 2013; Bodnar & Simhan., 2008). County-by-quarter fixed effects (α_{cQ}) guarantee that the pollutant's effect is estimated from shocks beyond the regular (expected) local seasonal variation.[28] Table 1.13 shows the remaining variation in the fertility rate after removing all the fixed effects.

$q_j\left(P_{cwy}^{39}\right)$ corresponds to the fraction of days during a 39-week pregnancy in which the mean daily concentration of pollutant P in county *c* for a pregnancy started in week *w* of year *y* fell in the *j-th* concentration bin. The third quintile corresponds to the omitted category. Table 1.14 summarizes the remaining variation after removing all the fixed effects in [1.1]. W_{cwy} corresponds to a vector of weather controls (temperature and precipitation) in county *c* during a 39-week window started

[26] Also known as conception survival rate in epidemiological research. Corresponds
[27] Previous research has shown that higher-income households migrate in response to environmental quality in the US (Crowder & Downey, 2010; Pais, Crowder & Downey, 2014).
[28] For example, suppose costal county A experiences both an increase in local income and pollution during the summer; while county B, in the interior, may experience it during the fall driven by their local industry (e.g., smeltery).

on week w of year y. $U^{Q1}{}_{smy}$, the state-level unemployment rate[29] during a pregnancy's first trimester controls for the correlation between the unemployment rate and abortions (Blank et al., 1996). I used the first trimester only because, during the early 80s, about 90% of all abortions occurred during the first 12 weeks of gestation (Atrash et al., 1986). Mothers' demographic controls were excluded from [1.1] because they may be affected by air pollution shocks (i.e., bad control in econometric jargon).

1.4.2 Methodologies to estimate effects on neonatal outcomes.

For neonatal outcomes (Birthweight, low birth weight (LBW), small for gestational age (SGA), and preterm birth (PTB)), two models were used: in [1.2] pollutant exposure is assigned at the newborn's level and measured from the presumed date of conception[30] to the birthdate, in [1.3] it is assigned by county and week of conception and measured over a fixed 39-week window starting from the presumed week of conception. Therefore, any two pregnancies in the same county whose last menstrual period dates fall in the same week were assigned the same exposure.

$$y_{ickb} = \alpha_{cyr} + \alpha_{mr} + \alpha_{cQr} + \sum_{j\ 1..5,\neq 3} \beta_j\, q_j(P_{ckb}) + \delta W_{ckb} + \theta\, U^{Q1}{}_{smy} + \gamma X_{ickb} + u_{ickb} \quad [1.2]$$

where y_{ickb} represent the neonatal outcome for newborn i, conceived in date k and born on date b and county c.[31] $q_j(P_{ckb})$ corresponds to the fraction of days in which the pollutant's concentration in county c fell in the *j-th* concentration quintile of pollutant P during dates k and b. X_{ickb}

[29] Ideally, the unemployment rate at the county level would have been used, but this variable is only produced at the state-month level, for the period under study, by the bureau of economic analysis.
[30] Two weeks after the last menstrual period.
[31] The observations for which the county of residence and birth differ were dropped from the analysis.

correspond to a vector of individual-level controls from the birth certificates (mother's age, pregnancy history, marital status, high school dropout, and gender[32]). α_{cyr}, α_{mr}, $and\ \alpha_{cQr}$ correspond to county-year-race, conception month-race, and county-trimester-race fixed effects. Three racial categories were included: white, black, and other. u_{ickb} correspond's to the error term. The unemployment rate during the pregnancy's first trimester[33] was included in this model because the economic environment affects birth outcomes through compositional changes (i.e., women self-selecting into pregnancy) and improvements in health behaviors in periods of high unemployment (Dehejia & Lleras-Muney, 2004). Standard errors are clustered at the county level.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{mr} + \alpha_{cQr} + \sum_{j\ 1..5,\ \neq 3} \beta_j\ q_j\left(P_{cwy}^{39}\right) + \delta W_{cwyr} + \theta\ U^{Q1}{}_{smy} + \gamma X_{xwyr} + u_{cwyr} \quad [1.3]$$

where y_{cwyr} represent the average neonatal outcome of newborns whose mothers reside in county *c*, were conceived in week *w* of year *y* and who belong to racial group *r* (i.e., white, black, other). $q_j\left(P_{cwy}^{39}\right)$ corresponds to the fraction of days in which the pollutant's concentration in county c fell in the *j-th* concentration quintile of pollutant P during a fixed 39-week window starting from week w of year y. X_{xwyr} correspond to the mean of a vector of individual-level controls from the birth certificates (mother's age, pregnancy history, marital status, high school dropout, and gender ratio) by county, week-year, and race cells. u_{cwyr} corresponds to the error term. Regressions are weighted by the number of births in each cell, and the standard errors clustered at the county level.

[32] Sanders & Stoecker (2015) found that increases in Total Suspended Particles –which can be formed by the oxidation of SO_2 in the atmosphere— decrease the fraction of male newborns. For the pollutants analyzed in this paper, I did not find a significant impact on the gender ratio. The results for models [1.2] and [1.3] are essentially identical when the gender ratio is excluded.

[33] Controlling for the unemployment rate during the second or third trimesters does not alter the results in a significant way.

1.4.3 Choosing the most reliable methodology for birth outcomes

As previously discussed, this paper's main objective is to establish how sensitive the pollutant effect's estimates are to the window used to measure exposure. In [1.2], exposure is measured from conception to birthdate, and in [1.3] is measured using a 39-week fixed window starting from conception. The results differ across methodologies, but how do we choose the best one? In section 1.2.2, I argued that there is an ex-ante tradeoff when choosing the window to measure exposure: When the conception-birthdate window is used, the estimate could be biased due to the induced endogeneity in pollution. If a fixed 39-week window is used, there could be a bias because not all pregnancies reach full term.

In order to test how reliable both methodologies are, I used a placebo test. For each window, I built measures of exposure using each county's 52-week lagged pollutant concentration to estimate the effect on birth outcomes. Tables 1.9 and 1.10 show the correlation between the real and placebo measures when exposure is measured from conception-birth and conception-39 weeks, respectively.

Lastly, to evaluate the potential bias caused by using a fixed 39-week window for all newborns, I ran a robustness test measuring exposure from conception to 26 weeks. Notably, only 72.47% of the births reached 39 or more weeks; however, 99.6 % of all births reached at least 26 weeks.

1.4.4 Strategy to address the livebirth bias

As discussed in section 1.2.3, the livebirth bias poses a potential threat when estimating [1.2] and [1.3]. The pollutant effect's estimate could be biased because birth outcomes are observed only for the population that survives the shocks. Appendix B explains this problem in greater detail. Equation [B1] decomposes SO_2's reduced-form coefficient separately for two populations. First, the fetuses that transition from live birth to fetal death due to the shock (i.e., extensive margin). Second, the fetuses that would have been born irrespectively of the shock; this population would be affected only through the intensive margin (i.e., decrease in weight or the gestational age). The estimate of air pollution's effect in [1.2] and [1.3] could be negative if the fetuses surviving the shock were positively selected.

In order to overcome this bias, I use the number of non-adverse (i.e., healthy) birth outcomes per woman of reproductive age (*nlbw/w*, *nptb/w*, *nsga/w*). Intuitively, the advantage of this variable is that pollution's effects through the extensive and intensive margin go in the same direction[34]. For example, let us start by assuming that pollution affects health negatively through the intensive margin (e.g., decreases birthweight) and causes fetal deaths (extensive margin). Then, if the infants who die due to pollution shocks were positively selected, *nlbw/w* would decrease due to the effects through both margins. On the other hand, if the infants were negatively selected (i.e., infants who die would have belonged to the left tail of the birthweight distribution), *nlbw/w* would decrease due to the intensive margin but would not be affected by the extensive one. Appendix B discusses this approach in greater detail.

[34] With traditional outcome variables (e.g., birthweight) the impacts through the intensive and extensive margin can cancel eath other out.

Finally, the effects of pollution on health at birth are estimated using *nlbw-w, nptb-w, nsga-w* as outcome variables in [1.3]. The regressions are weighted by the number of women 15-44 years old from race *r* in the respective county and year, and the standard errors are clustered at the county level.

1.5 Results

This section presents the estimates of sulfur dioxide's effect on fetal death and birth outcomes in sections 1.5.1 and 1.5.2, respectively. In the latter, I compare the estimates of the effect of SO_2 on birthweight when exposure is measured from conception to birth vs. conception to 39 weeks. I selected the most robust methodology based on a placebo test. Finally, I show the estimates of the effect of SO_2 on newborns' health using a new approach robust to livebirth bias (i.e., sample selection bias).

1.5.1 Fetal death

Table 1.16. shows the results of estimating [1.1]. The first (last) three columns impose a linear (non-linear) functional form in SO_2's effects. Under the assumptions described in section 1.4.1, a negative coefficient is interpreted as evidence that high SO_2 concentrations increase fetal death. Column (5) shows that ambient concentrations of SO_2 between 7.66 and 13 ppb (fourth quintile) increased fetal deaths in counties with a low baseline level of SO_2 (i.e., low-pollution counties[35]). On the contrary, there is no significant effect in high-pollution counties (Col 6). This counterintuitive result suggests that other factors mediate air pollution's health effect. For instance,

[35] Low pollution counties correspond to those whose average SO_2 concentration during the first two years of available data is lower that the median of all counties with available data.

low-pollution counties have a larger fraction of pregnant women who are white and high school dropouts and have fewer hospital beds per capita. Alternatively, there could be an adaptation story: perhaps individuals in high-pollution counties are better insulated from outdoor air pollution.

The result in column 5 suggests that an additional day in SO_2's second-highest concentration bin (4^{th} quintile) during the pregnancy decreased the fertility rate by 0.029%[36], implying 1.53[37] fewer annual births per county.[38] I transformed this estimate to measure the effect of one additional day in the fourth quintile of SO_2 on the relative odds of fetal death, and the odds of fetal death increase by 0.17%.[39] Similarly, Sarovar et al. (2020) estimate that, in California, a 3 parts per billion (ppb) increase in SO_2 during one day increases the odds of fetal death between 0.15% and 5.45% four days later.

Additionally, the back-of-the-envelope calculation suggests that 46[40] fetal deaths were associated with the fourth quintile of SO_2 every year for the typical low-pollution county . In contrast, the linear model predicts 27.14[41] for the same group (Col 2), although not statistically significant. Nevertheless, it is essential to highlight that these estimates are upper bounds since they implicitly assume that 1-day effects can be extrapolated to longer periods. However, we would expect larger behavioral responses (i.e., adaptation) to longer pollution episodes. Therefore, the marginal effect of one additional high-pollution day is likely decreasing. I did not find evidence that the effects

[36] 0.029%=(0.1/273)/1.25. Where 0.1 is the coefficient for the 4^{th} quintile of SO_2 in Table 1. 1.25 corresponds to the average weekly birth rate per thousand woman of reproductive age in low-pollution counties (See Table 1)

[37] 1.53= (0.1/273)*80.38*52. Where 80.38 corresponds to the average women of reproductive age (in thousadns) in low-pollution counties in Table's 1 sample. The expression is multiplied by 52 to get an annual estimate (i.e., the coefficients were estimated with weekly data)

[38] For comparison, there are on average 4316 annual births per county in the low-pollution group.

[39] $0.17\% = 100 * \left(\frac{OR^{high} - OR^{o}}{OR^{o}}\right)$. $OR^{o} = \frac{1061}{7075-1061}$. $OR^{high} = \frac{FD^{high}}{7075-FD^{high}}$. FD^{high}=1061+1.53. where 1061 is taken from footnote 66 and 1.53 from footnote 60.

[40] 46=0.1*0.11*80.38*52. Where 0.11 corresponds to the average time a pregnancy in low-pollution counties is exposed to SO_2 concentrations in the fourth quintile.

[41] 27.14=0.00158*4.11*80.38*52. Where 4.11 corresponds to the mean SO_2 concentration a pregnancy in low-pollution counties is exposed to.

were statistically different for one high-pollution day in isolation vs. in a 3-day episode of high pollution.[42] However, not many of these longer episodes were in the data, so this result should not be considered robust evidence against decreasing effects during longer pollution episodes.

Lastly, the magnitude of the back-of-the-envelope estimate presented above leads to sizeable estimates of the fraction of fetal deaths caused by sulfur dioxide. In 1990, 61% of all pregnancies ended in a live birth, 15% in fetal loss, and 24% in induced abortions in the US (Ventura et al., 2008). Consequently, there would have been 1061[43] annual fetal deaths per county for the low-pollution group. Therefore, based on estimates from Column 5, SO_2 would have caused 4.33% of all fetal deaths for this set of counties.

Table 1.17 shows the effect of SO_2 on the fetal death rate are consistent with the SO_2 effects on the fertility rate (Table 1.16). As discussed in the empirical strategy section, the fertility rate is used instead of the fetal death rate because fetal death data were very noisy during the 80s. Since reporting fetal deaths before 20 weeks of gestation is not mandatory, the preferred specifications in this table would be (1)-(3); however, the result in column (3) is counterintuitive. On the other hand, it is reassuring that the estimates in column (5), where fetal deaths of all gestational ages are included, are similar in magnitude to those of Col 5 of Table 1.16.

Table 1.18 presents the results of the placebo test; which show that the 39-week window used to measure exposure to SO_2 generates reliable estimates. In columns 2,4, and 6, I estimated the

[42] Results not shown, available upon request.

[43] 1061=(N Pregnancies)*0.15. $N\ Pregnancies = \frac{4316}{0.61} = 7075$. Where 4316 corresponds to the mean annual number of births per county for the low-pollution group (See Table A2).

impact of 52-week lagged SO_2 concentrations on the fertility rate for [1.1]. The estimates are statistically insignificant and much smaller in magnitude when the placebo measures are used. The estimates are also robust to controlling by co-pollutants (Carbon monoxide (CO), Nitrogen dioxide (NO_2), Ozone (O_3), and 10-microgram particulate matter (PM_{10})).[44]

1.5.2 Birth Outcomes

This section is split into four sub-sections. In the first one, I compare SO_2's estimates when exposure is computed from conception to birth vs. conception to 39 weeks. Additionally, I test the robustness of both methodologies using a placebo test. In the second section, I present and discuss the estimates for SO_2's effect on traditional birth outcomes (*birthweight, LBW, PTB, SGA*) when exposure is measured from conception to 39 weeks— the preferred methodology based on the placebo test. In the third section, I estimate the effects of SO_2 on newborn's health using a novel approach robust to livebirth bias. The last section shows the heterogeneity analysis by race.

1.5.2.1 Comparing results across windows to measure exposure

In this section, I contrast the results from the two models to estimate SO_2 effects on birthweight. Model [1.2] measures exposure from conception to birth and is estimated in Columns 1-2 of Table 1.19; Columns 3-4 are presented for comparison purposes. The estimate in Column 1, which imposes linear effects, suggests that an increase of 1 part per billion in the average SO_2 concentration during the prenatal period decreases birth weight by 0.24% (Approximately 8 grams[45]). The non-linear effects estimates in Column 2 suggests one additional day in the most

[44] Regression tables not shown, available upon request.
[45] 8=0.24% *3362.5. where 3362.5 corresponds to the average birthweight in counties with SO2 data (seet Table 1.1)

polluted quintile of SO_2 decreases birth weight by 0.022%[46]. The back-of-the-envelope computation suggests that birthweight decreases on average 1.1 %[47] due to SO_2.

Notably, we observe that the sign of the effect of SO_2 on birthweight switches from negative to positive in the last two columns when exposure is measured from conception to 39 weeks. Thus, the window chosen to measure exposure during the prenatal period is critical for the estimates. At this point the reader may be wondering which is the most reliable methodology to measure exposure. I used the placebo test explained in section 1.4.3 to answer this question.

Model [1.3] measures exposure from conception to 39 weeks and is estimated in Columns 3-4 of Table 1.20. In this table, data has been collapsed in cell by county, week of conception and race. Columns 1-2, are presented for comparison purposes. The estimate in Column 3, which imposes linear effects, suggests that an increase of 1 part per billion in the average SO_2 concentration during the prenatal period *increases* birthweight by 0.014%. The non-linear effects estimates in Column 4 suggest that one additional day in the second most polluted quintile of SO_2 *increases* birthweight by 0.0021%. The back-of-the-envelope computation suggests that birthweight *increases* on average by 0.1 % due to SO_2.

Using aggregated data, the sign of both estimates is positive irrespective of the window used to measure exposure. The change in the sign of the estimate in Column 1 between tables 1.19 and 1.20 raises concerns about the reliability of the conception-birth methodology. Furthermore, Table

[46] 0.022%=100(0.06/273).
[47] 1.1%=100(0.06*0.18). where 0.18 corresponds to the fraction of time the average pregnancy is exposed to the fith concentration quintile (see Table 1.6)

1.15 shows evidence of an intra-annual negative trend in the concentration of sulfur dioxide. Gestational age and prenatal pollution could negatively correlate by construction when exposure is measured from conception to birth (see section 1.2.2).

The reliability of both methodologies was tested with the placebo test discussed in section 1.4.3. In Table 1.21, exposure was measured from conception to birth. We observe that placebo estimates are statistically significant and about half the size in magnitude compared to the estimates with the actual concentration (Column 1 vs. 3). This suggests that measuring pollution from conception to birth leads to biased estimates. On the other hand, In Table 1.22, exposure was measured from conception to 39 weeks, and the placebo effects (Columns 3 and 4) were close to zero and statistically insignificant.

Lastly, to evaluate the potential bias caused by using a fixed 39-week window, I ran a robustness test measuring exposure from conception to 26 weeks in Table 1.23. Notably, only 72.47% of the births reached 39 or more weeks; however, 99.6 % of all births reached at least 26 weeks. The 26-week estimates were smaller in magnitude and statistically insignificant, which suggests that the 39-week estimates are not attenuated relative to the 26-week ones. Additionally, it could be hypothesized that the effects of high SO_2 days are larger during the third trimester.[48]

Based on the previous analysis, I selected the 39-week window as the preferred methodology to measure exposure.

[48] Effects were not disaggregated by trimester due to the high collinearity of the different SO_2 bins and lack of power to identify the additional parameters when disaggregating by trimester.

1.5.2.2 Estimates for traditional birth outcomes

The estimates of the effect of SO_2 on birthweight using a 39-week window to measure exposure (preferred empirical strategy) are counterintuitive. The results suggest that increases in SO_2 improve birth outcomes. As discussed in section 1.4.4 (Empirical strategy), this counterintuitive result might be caused by livebirth bias. Alternatively, it could be caused by omitted variable bias (e.g., income), as discussed in section 1.2.3 (Background). Nevertheless, as discussed below, a holistic review of the results indicates that the livebirth bias drives the positive correlation between sulfur dioxide and birthweight.

Tables 1.24 and 1.25 estimate [1.3] for birthweight and the fraction of adverse birth outcomes across several samples based on the county's average SO_2 concentration. The national-level estimates –driven by low-pollution counties— show that higher SO_2 prenatal concentrations are associated with better neonatal outcomes. It is important to recall that high SO_2 concentrations increase fetal death in low-pollution counties (Table 1.16). Thus, the results from Tables 1.24 and 1.25 vs. the ones from Table 1.16 are consistent with the possibility that the fetuses who survive pollution shocks are positively selected.

Admittedly, there may be lingering concerns regarding the exogeneity of air pollution due to omitted variable bias (e.g., income). However, these concerns are lessened once we consider that a significant fraction of local concentrations of pollutants come from sources elsewhere. For instance, in the eastern US, around 77% of each state's Ozone and $PM_{2.5}$ concentrations are caused by NO_x and SO_2 emissions in other states (Bergin et al., 2007). In addition, it is essential to consider the sources of emissions. According to the EPA, around 97% of man-made annual SO_2 emissions

during 1978 in the US were from stationary point sources. From these, 70% were from utilities burning coal and oil for electricity generation, and 20% were from industrial processes (e.g., smeltery). We would expect this variation to be captured by the local seasonal effects and weather controls in [1.3]. Therefore, the main concern would be a nationwide income shock that systematically correlates income and SO_2 emissions. For example, demand for electricity in the commercial and industrial sectors falls during economic downturns (Thoma, 2004). Nevertheless, using county-year-race fixed effects and controlling for the state-level unemployment rate during the pregnancy's first trimester mitigates this concern[49].

The joint review of the fetal death and birth outcome estimates indicates that the livebirth bias causes the positive correlation between sulfur dioxide and birthweight. First, we observe that SO_2 increases fetal death in low-pollution counties (Col 4 of Table 1.16,), then we observe that low-pollution counties are the source of the positive correlation between SO_2 and birthweight in Table 1.24. Additionally, I estimate [1.3] using the number of adverse cases per woman of reproductive age as the outcome variable in Table 1.26. Altogether, these results are consistent with the hypothesis that fetuses who die due to SO_2 shocks are negatively selected (i.e., thus surviving are positively selected) in low-pollution counties and nationally. Lastly, if income and pollution were still correlated despite the controls in [1.3], we would expect the estimates from the first and second quintiles of SO_2 to be negative in Table 1.24. However, we observe the opposite; low SO_2 concentrations are associated with better birth outcomes in low-pollution counties and nationally, although the estimates are not statistically significant.

[49] Controlling for the unemployment rate during the entire pregnancy leads to quantitatively similar estimates of SO_2's effects.

1.5.2.3 Estimates robust to livebirth bias

The analysis from the previous section indicates that the estimates of the effects of SO_2 on birth outcomes are affected by livebirth bias (i.e., sample selection). I attempted to address this problem using a bounding approach, as in Lee (2009), but the results were unsatisfactory. Thus, I implemented the novel approach proposed in section 1.4.4 (empirical strategy).

Table 1.27 shows the results from the bounding approach. In Columns 1-2 (3-4), the top and bottom 2% (5%) of observations were excluded. The point estimates decreased in magnitude, suggesting a sample selection problem, but were still positive and statistically significant.

Table 1.28 shows the results of the novel approach to overcome the livebirth bias. [1.3] is estimated using the number of non-adverse cases per woman of reproductive age as the dependent variable and weighting the regressions by the number of women of reproductive age from the respective race, county, and year. The results show that high SO_2 levels decrease the number of infants born with non-adverse (i.e., healthy) neonatal outcomes. These estimates are robust to controlling by Co-pollutants (CO, NO_2, O_3, and PM_{10}).[50] Notably, I also find that high SO_2 concentrations decrease the number of infants born non-premature (i.e., with 37 or more weeks of gestation[51]). Which is an additional reason against measuring exposure from conception to birth (see section 1.2.2)

[50] Regression tables not shown, available upon request.
[51] There is no significant impact on the gestational age, most likely caused by livebirth bias. Results not shown, available upon request.

The estimates in Column 1 suggest that the rate of non-low birth weight (i.e., healthy) infants per woman of reproductive decreases by 0.0122%[52] for one additional day in the fifth quintile (most polluted). Consequently, a county's annual number of infants born with a weight above 2500 grams decreases by 38.76[53] due to SO_2 . As a reference, at the national level, there were 4423 annual births per county (see Table 1.2). The effect is larger in low-pollution counties. The rate of non-lbw infants per woman of reproductive age decreases by 0.0231% per one additional day in the fourth quintile (second dirtiest), and there are 73.3 fewer infants born with weight above 2500 grams due to sulfur dioxide.

1.5.2.4 Heterogeneous effects by race

Table 1.29 shows the estimates of the effect of SO_2 on the number of infants born non-low birthweight (birthweight>2500g) per thousand woman of reproductive age (15-44 yrs. old) by race. We observe that the point estimate for the fifth quintile (i.e., most polluted) is larger for blacks than whites, although statistically significant only for low-pollution counties. The standard errors are much larger for blacks than whites, which is likely a consequence of the smaller sample size. For low pollution counties, one additional day in the fourth and fifth quintiles decreases the rates of non-low birth weight (i.e., healthy) infants per woman of reproductive by 0.0237%[54] and 0.0562%[55] for whites and blacks, respectively. These results are consistent with previous

[52] -0.0122%= 100*((-0.0487/293)/1.363). Where 0.0487 is the coefficient associated with the 5th quintile of SO_2 in Col 1, and 1.363 corresponds to the mean of the dependent variable.
[53] -38.76= -0.0487*0.18*85.03*52. Where 0.18 correspond to the fraction that the average 39-week pregnancy spends in the 5th quintile (see col1 of Table 1.7). 85.03 corresponds to the number of women of reproductive age. 52 corresponds to the number of weeks in a year.
[54] 0.0237% =100*(0.0826/293)/1.189) Where 0.0826 is the coefficient associated with the 4th quintile of SO_2 in Col 2, and 1.189 corresponds to the mean of the dependent variable for whites.
[55] 0.0562% =100*((0.218/293)/1.324)Where 0.218 is the coefficient associated with the 5th quintile of SO_2 in Col 4, and 1.324 corresponds to the mean of the dependent variable for blacks.

environmental research that found larger air pollution effects on African Americans' infant health than whites (Chay & Greenstone, 2003a; Currie & Walker, 2011).

1.6 Conclusion

This paper's objective is to test how sensitive are the estimates of the effects of air pollution on birth outcomes to the window used to measure exposure during the gestational period. To do so, I compared the results of two methodologies to measure prenatal exposure to sulfur dioxide (SO_2). The results show that the estimates of the effects of SO_2 on birth outcomes are highly sensitive to the window chosen to measure exposure. When exposure was measured from conception to birth, I found a negative and significant impact of SO_2 on birthweight. In contrast, the estimate was positive and significant when exposure was measured from conception to 39 weeks.

In order to choose the most reliable methodology, I created placebo measures using each county's 52-week lagged SO_2 concentrations to compute prenatal exposures. When exposure was measured from conception to birth, SO_2's estimate from the placebo was significant and about half the size compared to the estimate from the actual exposure. In contrast, when exposure was measured from conception to 39 weeks, the estimates from the placebo were close to zero and statistically insignificant.

Measuring exposure from conception to birth leads to endogenous measures of prenatal exposure that overestimate the effect of SO_2 on birth outcomes. This bias can be explained by the decreasing trend in SO_2 concentrations during 1981-1991. This trend holds intra-annually for the average county. Consequently, infants with ex-ante better health are assigned lower measures of prenatal

exposure. Thus, gestational age –or any other birth outcome directly proportional to it (e.g., birth weight)—and prenatal pollution are negatively correlated by construction. This mechanical correlation disappears when exposure is measured over a fixed 39-week window. Furthermore, the results show that the number of infants born non-preterm (i.e., with 37 or more weeks of gestation) per woman of reproductive age decreases high with SO_2 concentrations. Thus, the date of birth is itself an outcome affected by pollution.

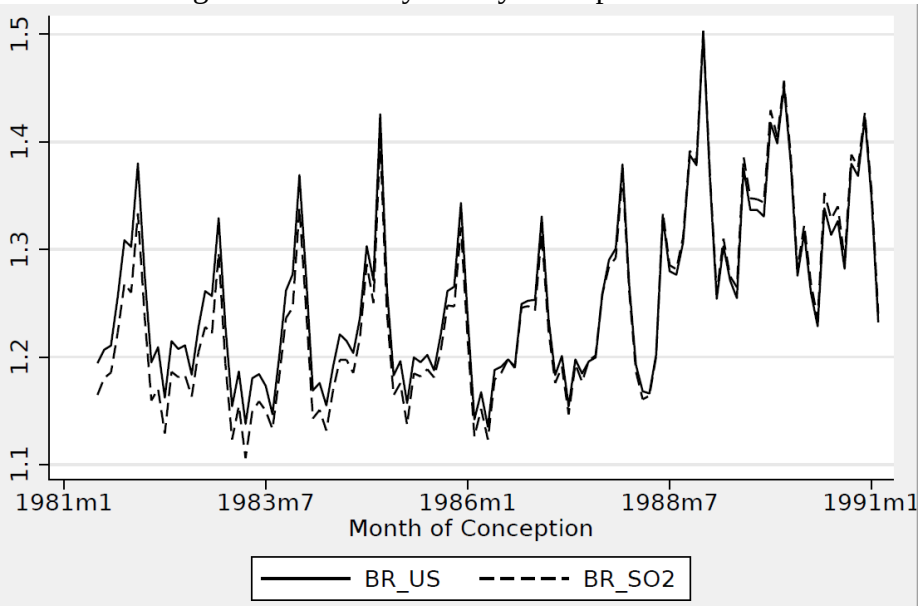
Using the most reliable methodology (i.e., conception-39 weeks), I found an unanticipated and counterintuitive result: A positive estimate of the effect of SO_2 on birthweight (i.e., high pollution associated with better health outcomes). Understanding this results was a challenge, and it required developing a new methodology to address the sample selection bias (i.e., livebirth bias) in this context. In theory, this result could be caused by at least two factors: (i) endogeneity bias, perhaps due to omitted variables (e.g., income), or (ii) the livebirth bias. The analysis of the fertility rate and left tail of the birthweight distribution were consistent with the hypothesis that the fetuses who die due to SO_2 shocks are negatively selected (i.e., would have been born with low weight). Furthermore, the point estimates of low SO_2 concentrations were inconsistent with an omitted-variable-bias story. Thus, it was concluded that livebirth bias was the cause behind the counterintuitive results.

The livebirth bias was addressed by using the number of infants born with non-adverse birth outcomes per woman of reproductive age as the dependent variable and weighting the regressions by the number of women of reproductive age. Using this approach, I find that high SO_2 concentration during the prenatal period worsens health at birth. Finally, I estimate the impacts

separately across races and find that the damage caused by SO_2 was larger for black than white newborns.

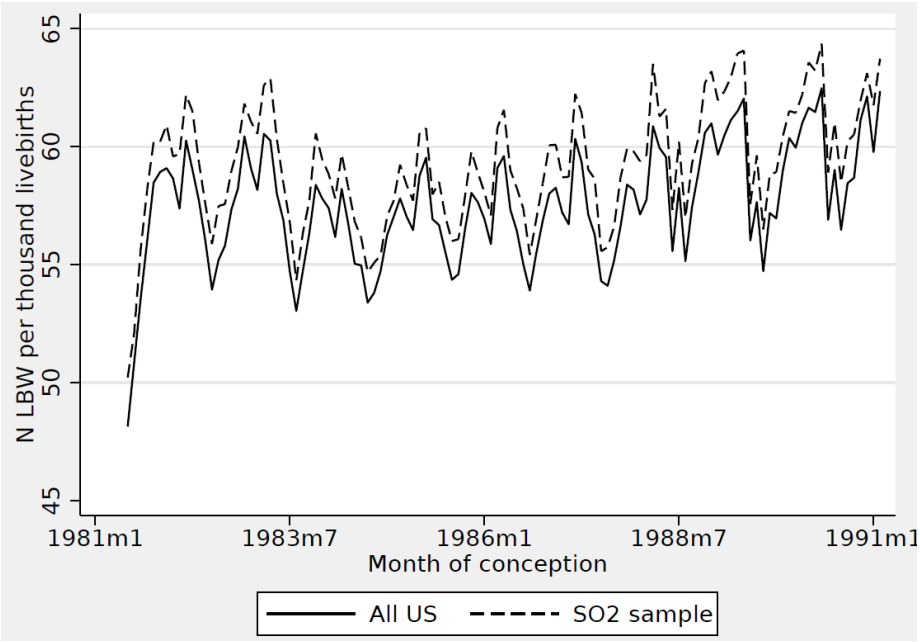
1.7 Figures

Figure 1.1: Fertility rate by conception month



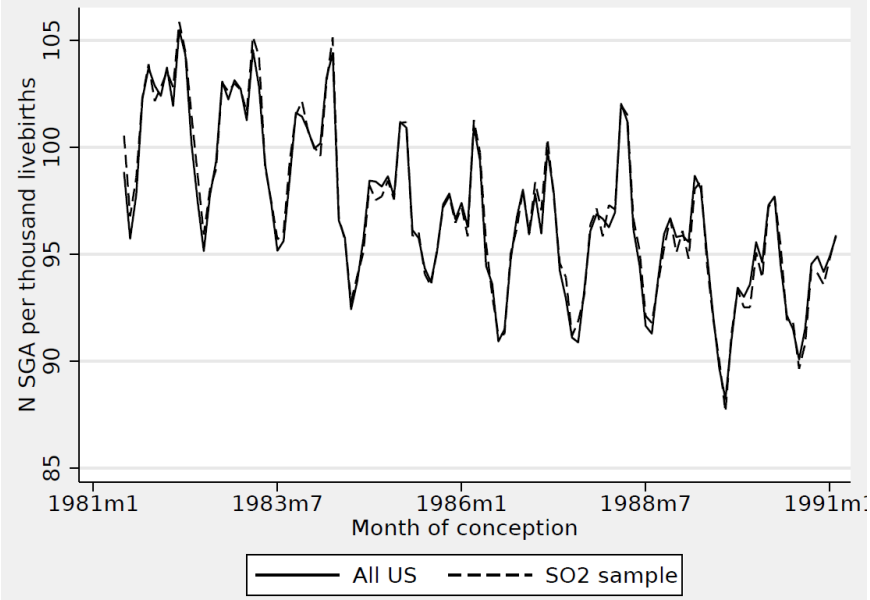
Note: Units on y-axis correspond to 1000*(N live births)/ N women 15-44 yr. old. BR_US (All counties in the US), BR_SO_2 (Counties with a balanced sample of SO_2 data through 1981-1991)

Figure 1.2: Low birthweight rate (LBW) by conception month.



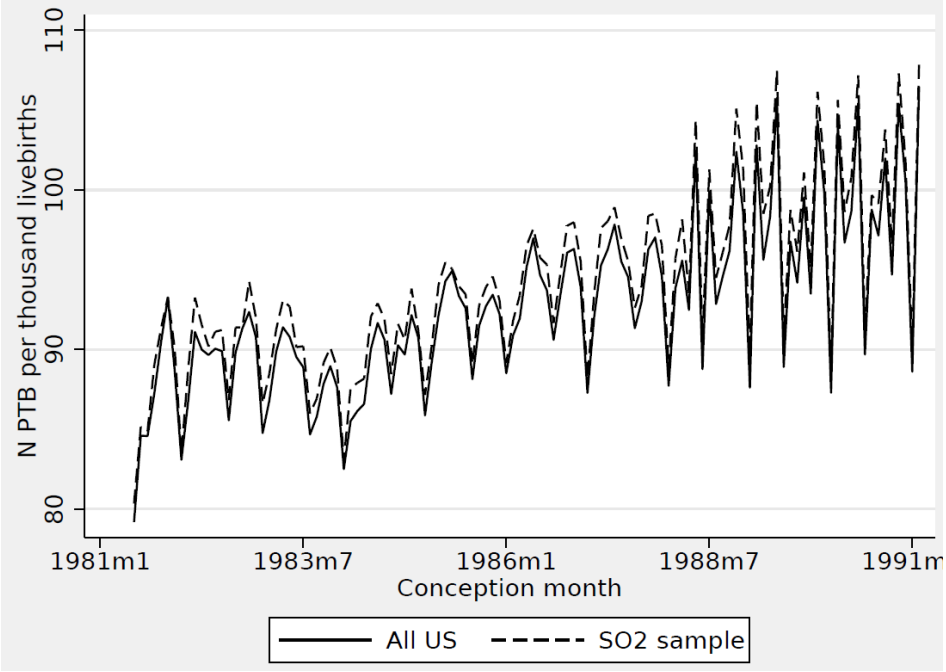
Note: A newborn is considered low birthweight if its weight at birth is 2500 grams or less. All US (All counties in the US), SO_2 sample(Counties with a balanced sample of SO_2 data through 1981-1991)

Figure 1.3: Small for gestational age rate (SGA) by conception month.
All US vs counties with SO_2 data.



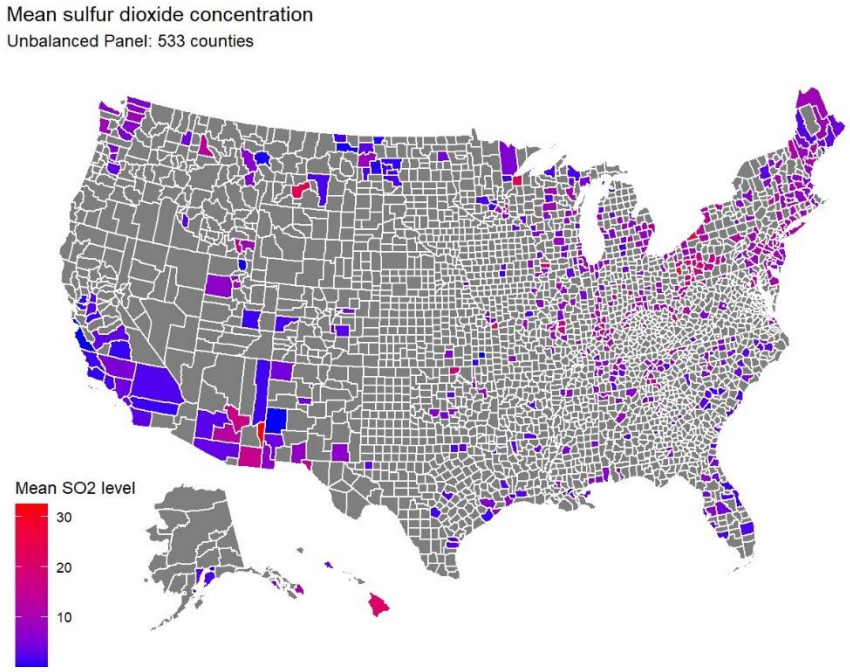
Note: A newborn is considered small for its gestational age if the weight at birth is less than the 10th percentile of birthweight for its gestational age, gender, and maternal race. Cutoff values for the 10th percentile were generated using all births in the US during 1982-1991. All US (All counties in the US), SO2 sample(Counties with a balanced sample of SO_2 data through 1981-1991)

Figure 1.4: Preterm birth rate (PTB) by conception month.



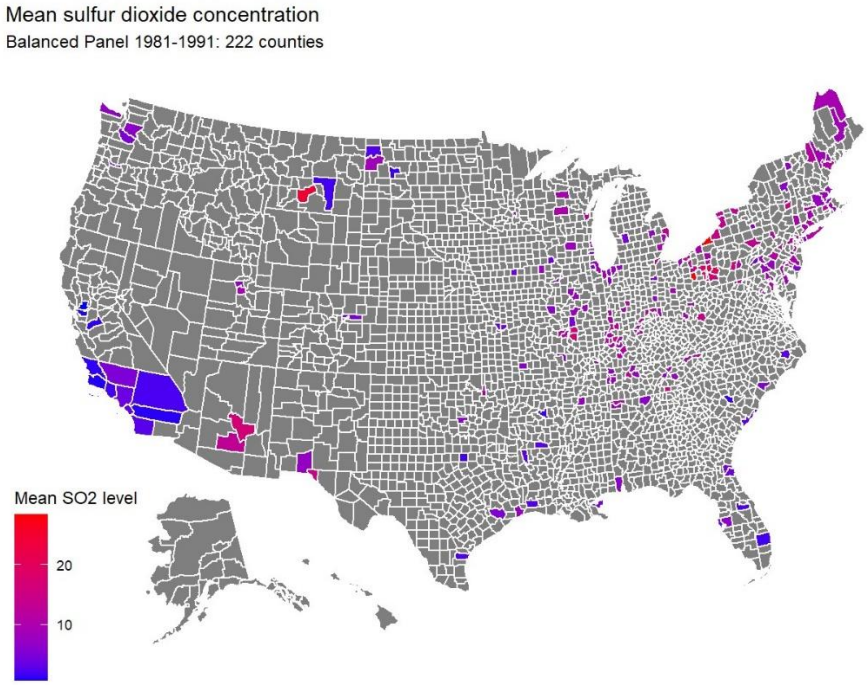
Note: A newborn is considered preterm if is born with less than 37 weeks of gestation. All US (All counties in the US), SO_2 sample(Counties with a balanced sample of SO_2 data through 1981-1991)

Figure 1.5: Mean sulfur dioxide concentration for all counties with monitoring data



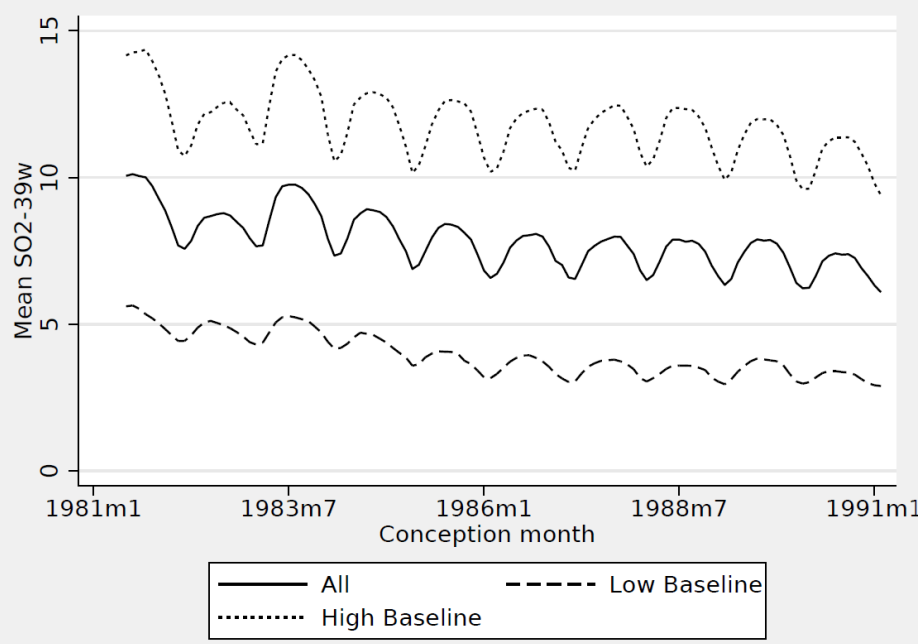
Note: Author's calculations from AQS (pollution monitoring data). Concentrations measured in parts per billion (ppb)

Figure 1.6: Mean sulfur dioxide concentration for counties in the balanced panel.



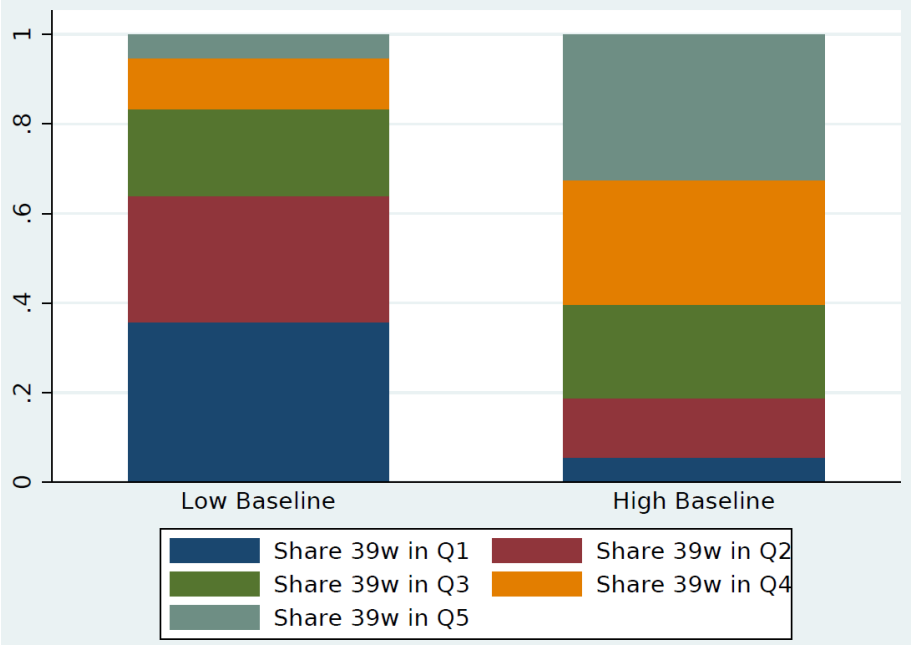
Note: Author's calculations from AQS (pollution monitoring data). Concentrations measured in parts per billion (ppb). A county belongs to the SO_2 Balanced panel if the fraction of daily missing observations for a 39-week forward looking rolling window was less than 50% every week-year during 1981-1991.

Figure 1.7: Average concentration of SO_2 (ppb) in the US during 1981-1991



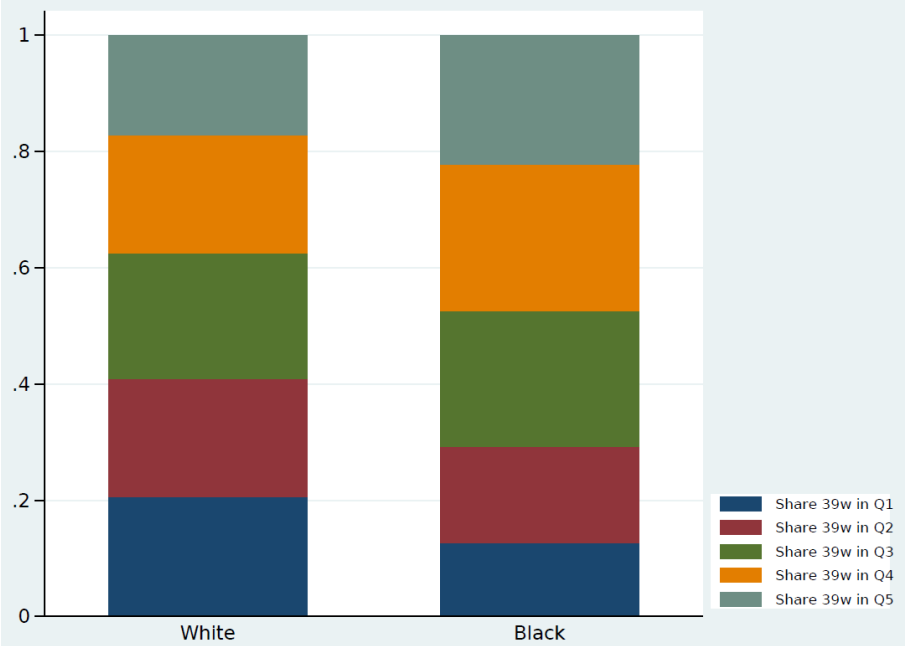
Note: Author's calculations from AQS (pollution monitoring data). Counties whose average level of SO_2 during their first two years was lower than the median in the SO_2 Balanced panel were assigned to the low Basleine category.

Figure 1.8.1: Distribution of SO_2 concentration for a standard 39-week pregnancy



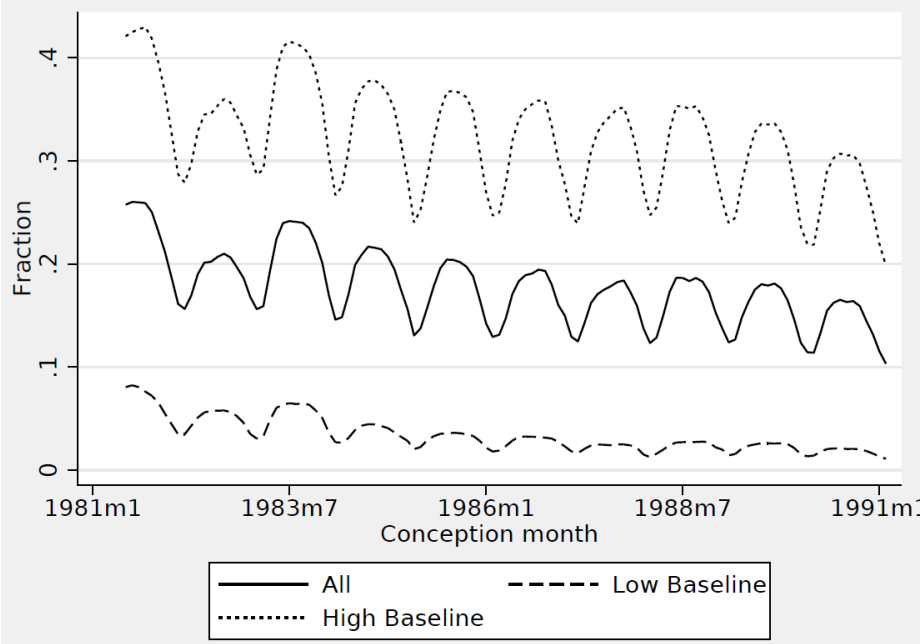
Note: Author's calculations from AQS (pollution monitoring data). Counties whose average level of SO_2 during their first two years was lower than the median in the SO_2-Balanced panel were assigned to the low basleine category. Concentration range per quintile: Q_1 ($SO_2 \leq 1.84\ ppb$), $Q_2(1.84\ ppb < SO_2 \leq 4.39\ ppb)$, $Q_3(4.39\ ppb < SO_2 \leq 7.66\ ppb)$, $Q_4(7.66\ ppb < SO_2 \leq 13\ ppb)$, and $Q_5(SO_2 > 13\ ppb)$).

Figure 1.8.2: Distribution of SO_2 concentration for a standard 39-week pregnancy by race



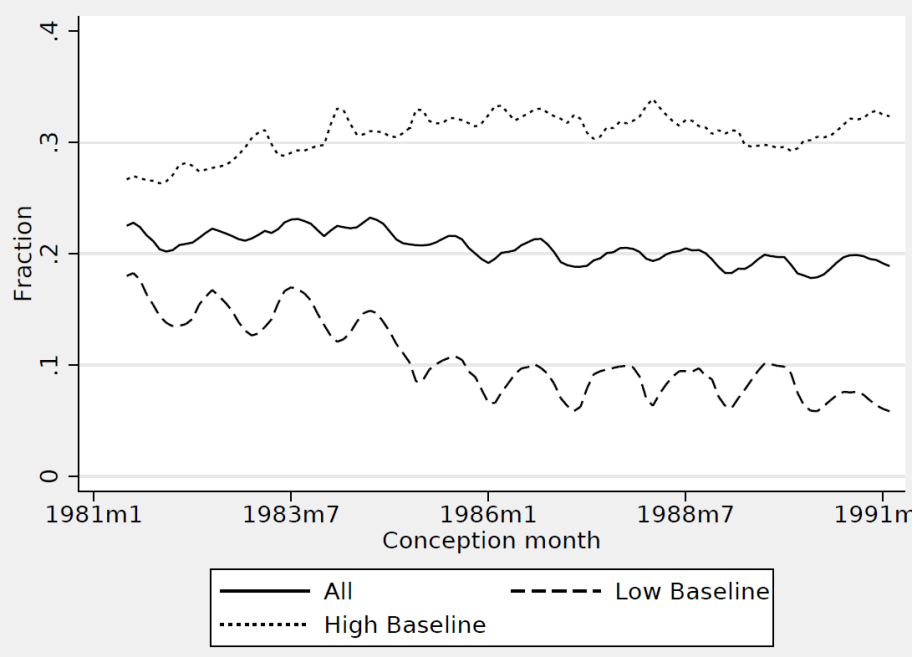
Note: Author's calculations from AQS (pollution monitoring data), CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Concentration range per quintile: Q_1 ($SO_2 \leq 1.84\ ppb$), Q_2($1.84\ ppb < SO_2 \leq 4.39\ ppb$), Q_3($4.39\ ppb < SO_2 \leq 7.66\ ppb$), Q_4($7.66\ ppb < SO_2 \leq 13\ ppb$), and Q_5($SO_2 > 13\ ppb$)).

Figure 1.9.1: Average fraction of a 39-week pregnancy spent in the fifth concentration quintile (SO_2>13 ppb) in the US by county's baseline pollution level



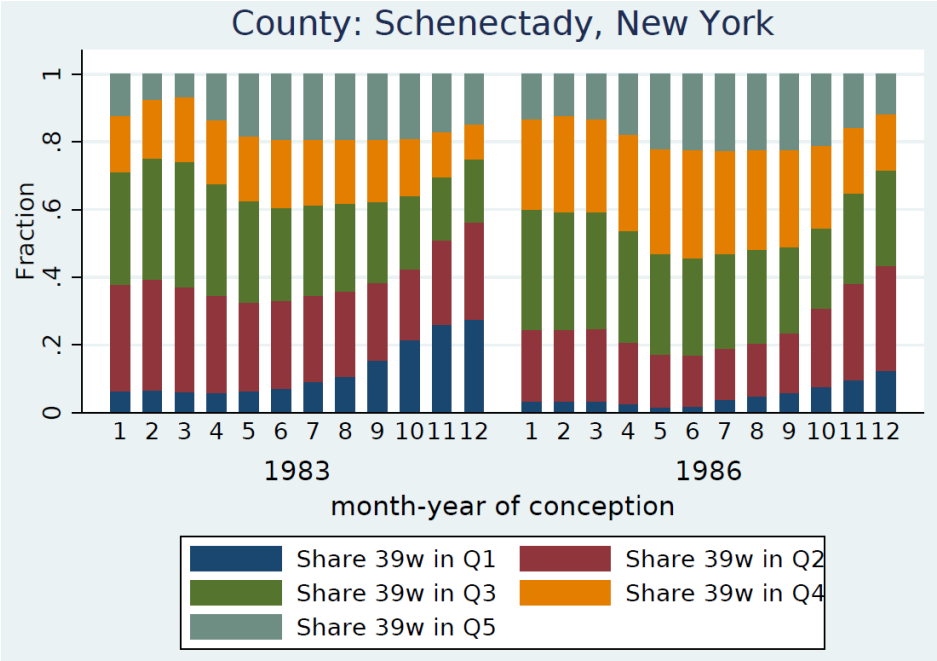
Note: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Counties whose average level of SO_2 during their first two years was lower than the median in the SO_2 Balanced panel were assigned to the low basleine category.

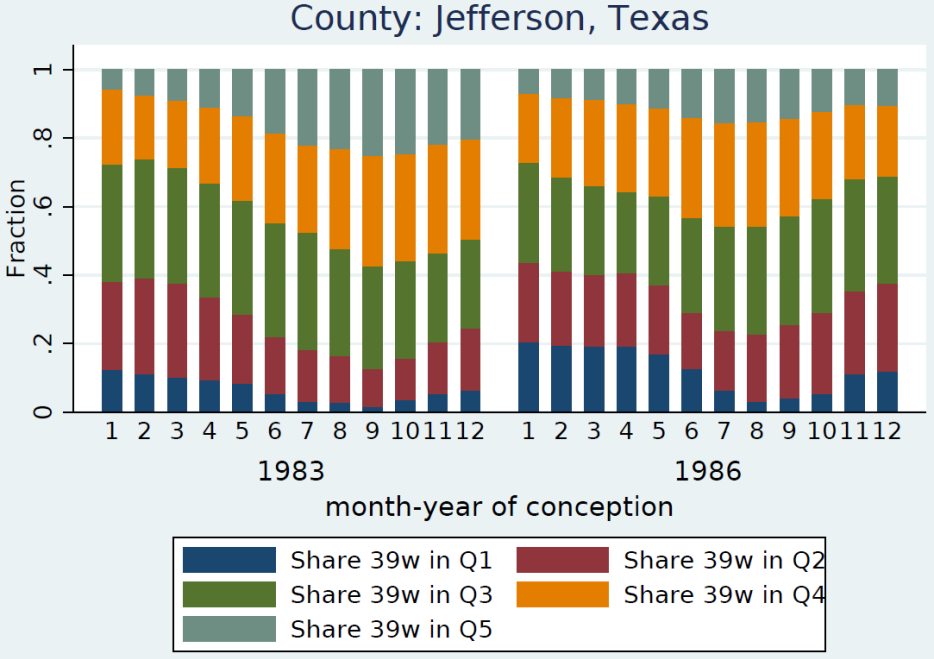
Figure 1.9.2: Average fraction of a 39-week pregnancy spent in the fourth concentration quintile ($7.66<SO_2<13$ ppb) in the US by county's baseline pollution level



Note: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Counties whose average level of SO_2 during their first two years was lower than the median in the SO_2 Balanced panel were assigned to the low Basleine category.

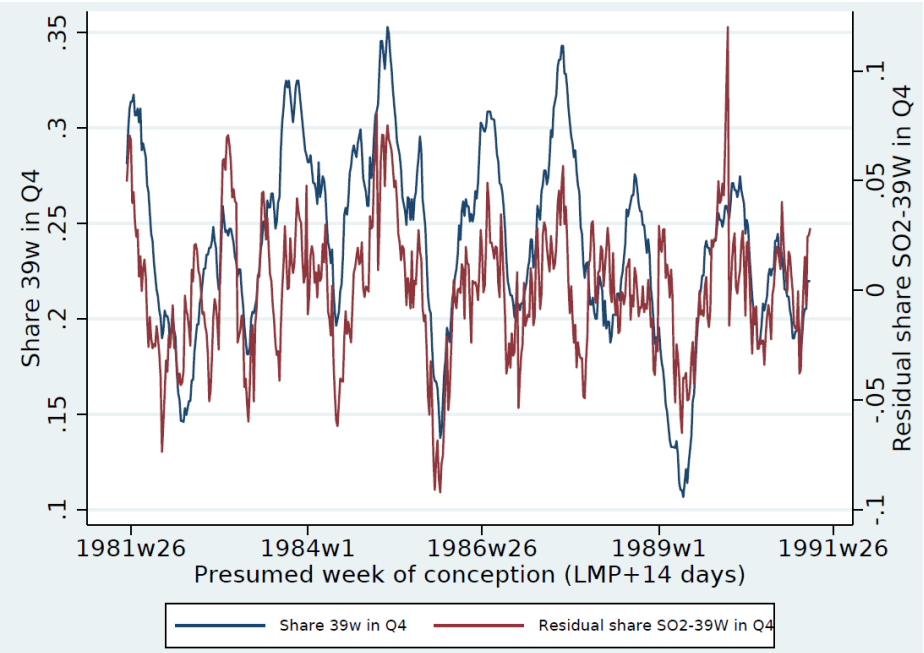
Figure 1.10: Distribution of SO_2 concentration for a standard 39-week pregnancy: two counties in two different years.





Note: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The thresholds for the quintiles were generated using all the county-day concentrations of SO_2 for all counties in the unbalanced SO_2 panel during 1981-1991.

Figure 1.11: Fraction of a 39-week pregnancy spent in the 4th quintile of the distribution of SO_2 for the county of Jefferson, Texas (Figure 6) vs residual variation.



Note: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The residual is computted by regression the original series (Share 39w in Q4) on the right hand side variables of model [1] (County-year FE, County-Quarter FE, month FE, in-utero temperature, in-utero precipitation, and unemployment rate during the first trimester of pregnancy)

1.8 Tables

Table 1.1: Summary statistics for birth certificate data

	US	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
Birth Outcomes (means)					
Birthweight (grams)	3369.8	3362.5	3358.8	3377.1	3347
Low Birth Weight (LBW)	57.87	51.72	52.83	47.43	55.62
Small for Gestational age (SGA)	98.98	90.64	91.89	88.52	92.57
Preterm birth (PTB)	93.02	94.56	95.35	92.77	96.45
Note: LBW, SGA, and PTB are expressed as number of cases per thousand births.					
Demographics of the mother (means)					
Share white (%)	80.0%	77.5%	77.1%	80.6%	74.7%
Share black (%)	15.9%	18.1%	18.3%	13.5%	22.3%
Share other (%)	4.2%	4.4%	4.6%	6.0%	3.1%
Share age<=19 (%)	12.9%	12.1%	11.8%	12.4%	11.8%
Share 20<=age<=35 (%)	81.9%	82.3%	82.3%	82.1%	82.5%
Share age>=36 (%)	5.2%	5.7%	5.8%	5.5%	5.8%
Share unmarried (%)	24.0%	26.7%	27.3%	25.0%	28.3%
High school dropout (%) (means)					
No	64.04	59.91	59.52	43.56	74.81
Yes	17.8	16.75	17.07	14.08	19.18
Not reported	16.71	21.78	21.78	41.47	3.83
Not Answered	1.44	1.56	1.63	0.89	2.17
High school dropout from 1989 onwards (%) (means)					
No	72.12	70.40	69.80	67.62	73.04
Yes	22.59	23.70	23.95	27.84	19.77
Not reported	3.56	3.82	4.04	3.65	3.99
Not answered	1.73	2.07	2.22	0.88	3.20
Pregnancy history (means)					
First delivery	35.0	35.4	35.3	36.1	34.6
Second delivery & fetal death rate =0	25.2	24.7	24.5	25.7	23.8
Delivery>=3 & fetal death rate =0	18.1	18.0	18.0	19.5	16.7
Delivery>=3 & fetal death rate <0.5	12.0	11.9	12.0	10.7	13.0
Delivery=2 & fetal death rate =1	5.3	5.3	5.4	4.5	6.1
Delivery>=3 & fetal death rate>0.5	4.4	4.7	4.8	3.6	5.7
Controls at the county-year level (means)					
Per capita income	$ 15,548	$ 16,774	$ 17,007	$ 16,651	$ 16,887
Per capita Gov transfers (Except Medical & SSI)	$ 157.65	$ 164.47	$ 172.54	$ 146.42	$ 188.80
Per capita UI transfers	$ 81.93	$ 93.45	$ 95.08	$ 86.92	$ 100.16
Hospital beds per capita	4.8	5.1	5.1	4.6	5.7
Unemployment rate during Q1 (%)	7.1	7.1	7.0	7.0	7.1

Fraction of births by Urban- Rural Classification for counties (NCHS -1990) (means)					
Large central metropolitan	32.36%	47.58%	59.75%	46.09%	49.15%
Large fringe metropolitan	18.12%	18.06%	16.41%	11.53%	24.99%
Medium metropolitan	21.86%	25.06%	18.86%	31.31%	18.41%
Small metropolitan	7.97%	5.15%	3.66%	5.87%	4.38%
Micropolitan	9.10%	2.76%	0.81%	3.27%	2.22%
Non-core	10.59%	1.4%	0.51%	1.93%	0.84%
N births	35,610,573	17,443,247	14,064,208	8,071,791	9,371,456
Counties	3,138	533	222	294	239

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991.

Table 1.2: Summary statistics for the average annual fertility rate and number of births

	US	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
All races					
Fertility rate (mean)	58.1	53.1	55.6	55.1	51.4
N Births	1,047	4,423	5,993	4,316	4,525
Women 15-44 (mean)	18021	83311	107743	78269	88107
Whites					
Fertility rate (mean)	56.0	50.9	53.4	53.7	48.4
N Births	841	3426	4617	3475	3379
Women 15-44 (mean)	15027	67291	86500	64671	69784
Blacks					
Fertility rate (mean)	70.2	63.3	66.2	62.8	63.5
N Births	167	802	1097	584	1009
Women 15-44 (mean)	2380	12672	16582	9292	15887

Note: Fertility rate corresponds to the ratio between the number of births and thousand women 15-44 yrs olds. Statistics at the county-year level. Author calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Women 15-44 year old per county-year was sourced from SEER data.

Table 1.3: Summary statistics for the fetal death rate

	US	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
FDR 20+ (mean)	0.0059	0.0057	0.0057	0.0055	0.0059
FDR All (mean)	0.012	0.0114	0.0115	0.0075	0.0146
County-Week Cells	1,647,521	193,392	118,249	92,591	100,801

Notes: Author calculations from CDC public fetal death files 1982-1991. FDR All (FDR 20+) corresponds to all fetal deaths (fetal deaths of 20 or more weeks of gestation) per week conception week and thousand women in reproductive age in the respective county. These statistics were computed after collapsing fetal death data by county and year.

Table 1.4: Average annual number of infants born with non-adverse health outcomes per woman of reproductive age

	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
			All races (means)	
Nlbw-w	49.33	51.60	51.49	47.51
Nsga-w	46.98	49.18	48.96	45.31
Nptb-w	47.83	49.97	49.78	46.19
			White (means)	
Nlbw-w	47.92	50.19	50.61	45.55
Nsga-w	45.07	47.23	47.70	42.75
Nptb-w	46.67	48.82	49.08	44.55
			Black (means)	
Nlbw-w	55.43	57.86	55.57	55.35
Nsga-w	55.84	58.38	55.78	55.87
Nptb-w	52.68	54.95	52.83	52.59

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Women 15-44 year old per county-year was sourced from SEER data. Nlbw/w , nsga/w, and nptb/w correspond to the number of non-low birthweight (birthweight>2500g), non-small for gestational age (birthweight > P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race, and non-preterm birth (gestational age>=37 weeks) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Obsrevations for which SO_2 data was missing more than 50% of the time during the 39-week window to measure exposure were excluded.

Table 1.5: Average number of infants born with adverse health outcomes per woman of reproductive age

	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
			All races (means)	
Lbw-w	2.68	2.87	2.55	2.79
Sga-w	5.04	5.29	5.09	4.99
Ptb-w	4.19	4.50	4.27	4.12
			White (means)	
Lbw-w	1.96	2.09	2.04	1.88
Sga-w	4.81	5.05	4.95	4.68
Ptb-w	3.21	3.46	3.57	2.88
			Black (means)	
Lbw-w	6.47	6.89	5.98	6.75
Sga-w	6.06	6.38	5.78	6.22
Ptb-w	9.22	9.81	8.72	9.50

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Women 15-44 year old per county-year was sourced from SEER data. Lbw/w , Sga/w, and Ptb/w correspond to the number of Low birthweight (birthweight<=2500g), Small for gestational age (birthweight <= P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race, and Preterm birth (gestational age<37 weeks) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Obsrevations for which SO_2 data was missing more than 50% of the time during the 39-week window to measure exposure were excluded.

Table 1.6: Summary statistics for SO_2 concentrations
Exposure measured from conception to birth

	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
Mean SO_2	7.935	8.208	4.106	11.424
SO_2-Sh(q1) (%)	0.230	0.216	0.383	0.091
SO_2-Sh(q2) (%)	0.182	0.176	0.234	0.134
SO_2-Sh(q3) (%)	0.203	0.205	0.193	0.212
SO_2-Sh(q4) (%)	0.206	0.215	0.135	0.271
SO_2-Sh(q5) (%)	0.179	0.189	0.056	0.292
N (live births)	17,584,134	14,177,608	8,384,824	9,199,310
Counties	533	222	294	239

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. Statistics computed using newborn-level data. Units: parts per billion (ppb). Observations for which SO_2 was missing more than 50% of the days were dropped from the analysis. Mean SO_2 corresponds to the average SO_2 concentration from the newborn's presumed date of conception to the date of birth. SO_2-Sh(q j) (%) corresponds to the fraction of days between the conception and birth dates in which the county's SO_2 concentration lies in the j-th quintile of national distribution of SO_2.

Table 1.7: Summary statistics for SO_2 concentrations
Exposure measured from conception to 39-weeks

	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
Mean SO_2	7.935	8.208	4.103	11.427
SO_2-Sh(q1) (%)	0.194	0.167	0.360	0.043
SO_2-Sh(q2) (%)	0.198	0.196	0.278	0.125
SO_2-Sh(q3) (%)	0.219	0.229	0.211	0.226
SO_2-Sh(q4) (%)	0.210	0.222	0.114	0.298
N (live births)	17,584,134	14,177,608	8,384,824	9,199,310
Counties	533	222	294	239

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. Statistics computed using newborn-level data. Units: parts per billion (ppb). Observations for which SO_2 was missing more than 50% of the days were dropped from the analysis. Mean SO_2 corresponds to the average SO_2 concentration from the newborn's presumed week of conception to 39 weeks. SO_2-Sh(q j) (%) corresponds to the fraction of days during the conception-39 weeks window in which the county's SO_2 concentration lies in the j-th quintile of national distribution of SO_2.

Table 1.8: Correlations between SO_2 Measures.
Conception to Birth vs. Conception to 39 weeks.

Mean SO_2	0.9985
SO_2-Sh(q1) (%)	0.8977
SO_2-Sh(q2) (%)	0.6499
SO_2-Sh (q3) (%)	0.6172
SO_2-Sh(q4) (%)	0.8182
SO_2-Sh(q5) (%)	0.9575

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. Correlations estimated using individual-level data for the unbalanced SO_2 panel. Observations for which SO_2 was missing more than 50% during either window to measure exposure were dropped from the analysis (N=17,973,734)

Table 1.9: Correlations - placebo test for conception-birth

	Raw correlation	Partial correlation
Mean SO_2	0.9453	0.0802
SO_2-Sh(q1) (%)	0.831	-0.092
SO_2-Sh(q2) (%)	0.5743	-0.0009
SO_2-Sh(q3) (%)	0.5081	0.0129
SO_2-Sh(q4) (%)	0.7301	-0.02
SO_2-Sh(q5) (%)	0.9028	0.0934

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. Correlations estimated using individual-level data for the unbalanced SO_2 panel. Observations for which SO_2 was missing more than 50% of the days in either series (Current or Lagged SO_2 Concentrations) were dropped from the analysis (N=16,802,912). The value reported in "Raw correlation" is the pearson correlation coefficient between the newborn's actual in-utero exposure to SO_2 and the analogous placebo (one-year lagged SO_2 concentration in the same county) measure. Partial correlation refers to the correlation of the residuals from regressing each SO_2 variable on county-year-race FE, county-quarter-race FE, month-race FE, mother's demographics, newborn's gender, in utero weather, and the unemployment rate during the pregnancy first trimester. Lagged values of temperature and precipitation were used to estimates the residuals for placebo measures. Mean SO_2 corresponds to the average SO_2 concentration from the newborn's presumed date of conception to the date of birth. SO_2-Sh(q j) (%) corresponds to the fraction of days between the conception and birth dates in which the county's SO_2 concentration lies in the j-th quintile of national distribution of SO_2.

Table 1.10: Correlations - placebo test for conception-39 weeks

	Raw correlation	Partial correlation
Mean SO_2	0.9451	0.0708
SO_2-Sh(q1) (%)	0.9210	-0.1283
SO_2-Sh(q2) (%)	0.8349	-0.0233
SO_2-Sh(q3) (%)	0.8392	-0.0374
SO_2-Sh(q4) (%)	0.8673	-0.0013
SO_2-Sh(q5) (%)	0.9323	0.0883

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. Correlations estimated using data aggregated by county-race-year and weighted by the number of obsrevations in each cell. Observations for which SO_2 was missing more than 50% of the days in either series (Current or Lagged SO_2 Concentrations) were dropped from the analysis (Cells 342,840). The value reported in "Raw correlation" is the pearson correlation coefficient between the newborn's actual in-utero exposure to SO_2 and the analogous placebo (one-year lagged SO_2 concentration in the same county) measure. Partial correlation refers to the correlation of the residuals from regressing each SO_2 variable on county-year-race FE, county-quarter-race FE, month-race FE, mother's demographics, newborn's gender, in utero weather, and the unemployment rate during the pregnancy first trimester. Regressions were weighted by the number of livebirths in each cell. Lagged values of temperature and precipitation were used to estimates the residuals for placebo measures. Mean SO_2 corresponds to the average SO_2 concentration from the newborn's presumed week of conception to 39 weeks. SO_2-Sh(q j) (%) corresponds to the fraction of days during the conception-39 weeks window in which the county's SO_2 concentration lies in the j-th quintile of national distribution of SO_2.

Table 1.11: Correlations between SO_2 and Temperature bins
Exposure measured from conception to 39-weeks

	SO_2-Q1	SO_2-Q2	SO_2-Q3	SO_2-Q4	SO_2-Q5	T<=25F	25<T<=45F	65<T<=85F	T>85F
SO_2-Q1	1.00								
SO_2-Q2	0.18	1.00							
SO_2-Q3	-0.54	0.16	1.00						
SO_2-Q4	-0.71	-0.58	0.27	1.00					
SO_2-Q5	-0.53	-0.62	-0.27	0.41	1.00				
T<=25F	-0.37	-0.26	0.06	0.37	0.34	1.00			
25 F<T<=45F	-0.53	-0.37	0.08	0.48	0.53	0.63	1.00		
65 F<T<=85F	0.29	0.27	-0.02	-0.28	-0.35	-0.55	-0.76	1.00	
T>85F	0.28	0.06	-0.09	-0.21	-0.19	-0.22	-0.29	0.21	1.00

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. All counties with SO_2 data were included (i.e, unbalanced panel of SO_2). SO_2-Qj corresponds to the fraction of days during the conception-39 weeks window in which the county's SO_2 concentration lies in the j-th quintile of national distribution of SO_2. A <T<= b corresponds to the fraction of days during the conception-39 weeks window in which the county's average temperature falls between a and b Fahrenheit degrees.

Table 1.12: Summary statistics for SO_2 concentrations by race.
Exposure measured from conception to 39-weeks

	SO_2 Unbalanced		SO_2 Balanced		Low SO_2 Baseline		High SO_2 Baseline	
Maternal race	White	Black	White	Black	White	Black	White	Black
Mean SO_2	7.72	9.22	8.05	9.28	4.08	4.45	11.30	11.85
SO_2-Sh(q1) (%)	20.56%	12.60%	17.65%	10.80%	36.50%	31.19%	4.86%	2.40%
SO_2-Sh(q2) (%)	20.38%	16.53%	20.31%	15.94%	27.63%	28.32%	13.24%	10.00%
SO_2-Sh(q3) (%)	21.60%	23.36%	22.56%	24.41%	20.77%	23.50%	22.42%	23.28%
SO_2-Sh(q4) (%)	20.26%	25.24%	21.38%	26.82%	11.27%	12.87%	29.11%	32.04%
SO_2-Sh(q5) (%)	17.18%	22.23%	18.08%	21.97%	3.81%	4.09%	30.35%	32.21%
N (live births)	13,916,363	3,247,815	11,160,795	2,644,179	6,902,615	1,153,437	7,013,738	2,094,378

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. Mean concentration is measured as parts per billion (ppb). Mean SO_2 corresponds to the average SO_2 concentration from the newborn's presumed week of conception to 39 weeks. SO_2-Sh(q j) (%) corresponds to the fraction of days during the conception-39 weeks window in which the county's SO_2 concentration lies in the j-th quintile of national distribution of SO_2.

Table 1.13: Remaining variation in birth outcomes

	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
Log(birthweight)	0.719	0.749	0.654	0.753
LBW rate	0.338	0.373	0.213	0.392
PTB rate	0.368	0.392	0.272	0.437
Fertility Rate (FR)	0.656	0.687	0.634	0.628
Nlbw/w	0.840	0.748	0.706	0.890

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Reported values correspond to the R-squared of a regression of each variable on county-year-race FE, county-quarter-race FE, and month-race FE. Regressions were weighted by the number of livebirths in each cell in rows 1-3 and by the number of women or reproductive age per county, race and year in rows 4-5. Log(birthweight) is the natural logarithm of weight at birth; LBW and PTB rate correspond to the fraction of infants born with weight below 2500 grams and less than 37 weeks of gestation, respectively; The fertility rate corresponds to the number of livebirths per thousand woman of reproductive age. Nlbw/w corresponds to the number of non-low birthweight infants per thousand woman of reproductive age.

Table 1.14: Remaining variation in SO_2 and temperature

	SO_2 Unbalanced	SO_2 Balanced	SO_2 Low Baseline	SO_2 High Baseline
Mean SO_2	0.973	0.982	0.968	0.967
SO_2-Sh(q1) (%)	0.974	0.978	0.962	0.937
SO_2-Sh(q2) (%)	0.939	0.938	0.917	0.922
SO_2-Sh(q3) (%)	0.936	0.943	0.944	0.924
SO_2-Sh(q4) (%)	0.952	0.957	0.932	0.922
SO_2-Sh(q5) (%)	0.975	0.975	0.955	0.959
Mean Temperature	0.985	0.984	0.986	0.974
T<=25F (%)	0.934	0.927	0.950	0.908
65 F<T<=85 F (%)	0.961	0.965	0.959	0.954
T>85 F (%)	0.919	0.820	0.925	0.811

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Air pollution data was sourced from the AQS. temperature data was sourced from Deschênes et al. (2009). Reported values correspond to the R-squared of a regression of each variable on county-year-race FE, county-quarter-race FE, and month-race FE. Regressions were weighted by the number of livebirths in each cell.

Table 1.15 Intra-annual trend in Sulfur dioxide

	(1)	(2)	(3)
Trend	-0.00485***	-0.00536***	-0.00242***
	(7.08e-05)	(5.72e-05)	(0.000213)
Constant	9.302***	9.394***	8.865***
	(0.0148)	(0.0119)	(0.0388)
Observations	1,548,981	1,548,980	1,548,979
R-squared	0.003	0.373	0.443
County-year FE	No	Yes	Yes
County-Qrter FE	No	No	Yes

Notes: Author's calculations using air pollution data from the AQS from years 1981-1991. The dependent variable is SO_2 concentration at the county-day level. The trend corresponds to a running variable from 1 to 365.

Table 1.16: Effect of sulfur dioxide (SO_2) on fetal death

	(1)	(2)	(3)	(4)	(5)	(6)
Baseline SO_2	All	Low	High	All	Low	High
Mean (FR)	1.177	1.250	1.117	1.177	1.250	1.117
Women 15-44	85.03	80.38	89.30	85.03	80.38	89.30
Cells	180,217	86,550	93,667	180,217	86,550	93,667
R-squared	0.660	0.637	0.633	0.660	0.638	0.633
Mean SO_2-39w	-0.000902	-0.00158	-0.000665			
	(0.000788)	(0.00152)	(0.000873)			
SO_2-39w-Sh(q1)				-0.0261	-0.0478	-0.0339
				(0.0255)	(0.0386)	(0.0418)
SO_2-39w-Sh(q2)				-0.0273	-0.0647	0.0321
				(0.0474)	(0.0648)	(0.0504)
SO_2-39w-Sh(q4)				-0.0523	-0.100*	-0.0117
				(0.0355)	(0.0580)	(0.0359)
SO_2-39w-Sh(q5)				-0.0438	-0.0923	-0.0172
				(0.0285)	(0.0562)	(0.0272)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is FR(fertility rate) = 1000*(N live birth/ N women 15-44) by county and week-year of conception cells. LB (HB) corresponds to counties whose average SO_2 concentration during the first two years in sample is below (above) the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year FE, county-Quarter FE, and month FE. Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy. Regressions were weighted by the number of women of reproductive age in each county-year. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.17: Effect of sulfur dioxide (SO_2) on the fetal death rate (FDR).

	(1)	(2)	(3)	(4)	(5)	(6)
	FDR 20+	FDR 20+	FDR 20+	FDR All	FDR All	FDR All
Baseline SO2	All	Low	High	All	Low	High
Mean (FDR)	0.0057	0.0055	0.0059	0.0114	0.0075	0.0146
Women 15-44 (Thousands)	85.03	80.38	89.30	85.03	80.38	89.30
Cells	180,217	86,550	93,630	180,217	86,550	93,630
R-squared	0.214	0.194	0.234	0.737	0.565	0.786
SO2-39w-Sh(q1)	0.000130	0.00123	0.000950	0.000459	0.00111	0.00404
	(0.00102)	(0.00141)	(0.00171)	(0.00110)	(0.00167)	(0.00259)
SO2-39w-Sh(q2)	0.000759	0.00381	-0.00526*	0.00152	0.00402	-0.00254
	(0.00241)	(0.00245)	(0.00290)	(0.00220)	(0.00270)	(0.00236)
SO2-39w-Sh(q4)	0.000275	0.00319	-0.00371*	0.00136	0.00498	-0.00291
	(0.00189)	(0.00314)	(0.00213)	(0.00202)	(0.00341)	(0.00220)
SO2-39w-Sh(q5)	0.000907	0.00342	-0.00182	-0.00206	0.00799*	-0.00573
	(0.00124)	(0.00224)	(0.00149)	(0.00387)	(0.00468)	(0.00435)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. FDR 20+ (fetal death rate) = 1000*(N fetal death of 20 or more weeks/ N women 15-44). FDR All (fetal death rate) = 1000*(N fetal death of all gestations / N women 15-44) by county and week-year of conception cells. LB (HB) corresponds to counties whose average SO_2 concentration during the first two years in sample is below (above) the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is kept as the omitted category. All regressions include county-year FE, county-Quarter FE, and month FE. Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy. Regressions are weighted by the number of women in reproductive age (thousands) in each county-year. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.18: Placebo test for SO_2's effect on fetal death

	(1)	(2)	(3)	(4)	(5)	(6)
Year of data for SO_2	Current	Lagged	Current	Lagged	Current	Lagged
Counties	All	All	LB	LB	HB	HB
Cells	160,683	160,683	75,041	75,041	85,642	85,642
R-squared	0.666	0.666	0.644	0.644	0.639	0.638
SO_2-39w-Sh(q1)	-0.0349	-0.00799	-0.0587	-0.0182	-0.0313	-0.0117
	(0.0213)	(0.0178)	(0.0385)	(0.0210)	(0.0423)	(0.0288)
SO_2-39w-Sh(q2)	-0.0462	-0.0393	-0.0862	-0.0383	0.0170	-0.0523
	(0.0477)	(0.0280)	(0.0675)	(0.0334)	(0.0332)	(0.0401)
SO_2-39w-Sh(q4)	-0.0626*	-0.0184	-0.113**	-0.0454	-0.0193	-0.0157
	(0.0331)	(0.0288)	(0.0499)	(0.0338)	(0.0290)	(0.0388)
SO_2-39w-Sh(q5)	-0.0538*	-0.0175	-0.0961*	0.0188	-0.0250	-0.0308
	(0.0288)	(0.0210)	(0.0555)	(0.0398)	(0.0250)	(0.0265)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is FR(fertility rate) = 1000*(N live birth/ N women 15-44) by county and week-year of conception cells. The baseline level of SO_2 was computed for each county based on the average concentration during the first two years of data availability. LB (HB) corresponds to counties whose average SO_2 concentration during the first two years in sample is below (above) the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is kept as the omitted category. All regressions include county-year FE, county-Quarter FE, and month FE. Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy. Columns (2), (4), and (6) correspond to the placebo tests in which prenatal exposure to all environmental variables (SO_2, temperature, and precipitation) were computed for a standard 39-week pregnancy starting in the respective county but a year before its actual week-year of conception. Regressions were weighted by the number of women of reproductive age in each county-year. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.19: Estimates of SO_2 effects on birthweight across different methodologies to measure exposure Results using newborn-level data

	(1)	(2)	(3)	(4)
Window to Measure exposure	Conception-Birth	Conception-Birth	Conception-39 w	Conception-39 w
Mean SO_2	-0.00238***		0.000152*	
	(0.000324)		(7.96e-05)	
SO_2-Sh(q1)		0.00757		0.000646
		(0.00958)		(0.00214)
SO_2-Sh(q2)		0.00553		0.00558
		(0.00591)		(0.00392)
SO_2-Sh(q4)		-0.00466		0.00805**
		(0.00752)		(0.00331)
SO_2-Sh(q5)		-0.0600***		0.00333
		(0.00697)		(0.00255)
Dep variable mean	8.10	8.10	8.10	8.10
Observations	17,398,536	17,398,536	17,398,536	17,398,536
R-squared	0.068	0.068	0.068	0.068

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is natural logarithm of birthweight. Pollution and weather (temperature and precipitacion) exposure are computed using a window from conception to birth in cols 1-2 and from conception to 39 weeks in cols 3-4. Mean SO_2 corresponds to sulfur dioxide's mean concentration during each window. SO_2-Sh(q j) corresponds to the fraction of days, during the respective window, in which the county's SO2 concentration lies in the j-th quintile of national distribution of SO2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Additional Controls: mother's demographics (high school dropout, marital status, pregnancy history, age), newborn's gender, in-utero weather (temperature and precipitation), and the state's unemployment rate during the pregnancy's first trimester. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Individual observations for which SO_2 data was missing more than 50% of the time were excluded.

Table 1.20: Estimates of SO_2 effects on birthweight across different methodologies to measure exposure
Results using data aggregated by county-conception week-race

	(1)	(2)	(3)	(4)
Window to Measure exposure	Conception-Birth	Conception-Birth	Conception-39 w	Conception-39 w
Mean SO_2	0.000153**		0.000139*	
	(7.61e-05)		(7.67e-05)	
SO_2-Sh(q1)		-0.00264		0.000374
		(0.00264)		(0.00217)
SO_2-Sh(q2)		-0.00234		0.00131
		(0.00378)		(0.00233)
SO_2-Sh(q4)		-0.00184		0.00563**
		(0.00389)		(0.00236)
SO_2-Sh(q5)		0.00325		0.00182
		(0.00267)		(0.00186)
Dep variable mean	8.10	8.10	8.10	8.10
Observations	17,506,986	17,506,986	17,506,986	17,506,986
R-squared	0.725	0.725	0.725	0.725

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is natural logarithm of birthweight. Pollution and weather exposure are computed using a window from conception to birth in cols 1-2 and from conception to 39 weeks in cols 3-4. Mean SO_2 corresponds to sulfur dioxide's mean concentration during each window. SO_2-Sh(q j) corresponds to the fraction of days, during the respective window, in which the county's SO2 concentration lies in the j-th quintile of national distribution of SO2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Additional Controls: mother's demographics (high school dropout, marital status, pregnancy history, age), newborn's gender, in-utero weather (temperature and precipitation), and the state's unemployment rate during the pregnancy's first trimester. Regressions are weighted by the number of births in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Cells for which the average fraction of missings in SO_2 was larger than 50% were excluded.

Table 1.21: Placebo test for conception-birth window

	(1)	(2)	(3)	(4)
SO_2 Series	Current	Current	Lagged	Lagged
Mean SO_2	-0.00157***		-0.000745***	
	(0.000183)		(0.000179)	
SO_2-Sh(q1)		0.00525		0.0107
		(0.00621)		(0.00651)
SO_2-Sh(q2)		0.00470		0.00584
		(0.00473)		(0.00503)
SO_2-Sh(q4)		-0.00414		-0.00142
		(0.00485)		(0.00533)
SO_2-Sh(q5)		-0.0378***		-0.00491
		(0.00427)		(0.00421)
Observations	16,802,912	16,802,912	16,802,912	16,802,912
R-squared	0.070	0.070	0.070	0.070

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable corresponds to the natural logarithm of birthweight. . Mean SO_2 corresponds to sulfur dioxide's mean concentration over the conception-birth window. SO_2-Sh(q j) corresponds to the fraction of days, from conception to birth, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-(bins), precipitation-(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, Unmarried). Columns (3) and (4) correspond to the placebo tests in which prenatal exposure to all environmental variables (SO_2, temperature, and precipitation) were computed from conception to birth, but using each county's 1-year lagged series. All regressions are weighted by the number of births in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.22: Placebo test for conception-39 week window

	(1)	(2)	(3)	(4)
SO_2 Series	Current	Current	Lagged	Lagged
Mean SO_2	0.000140*		-7.26e-05	
	(7.69e-05)		(7.46e-05)	
SO_2-Sh(q1)		0.00108		0.000528
		(0.00212)		(0.00186)
SO_2-Sh(q2)		0.00295		1.53e-05
		(0.00228)		(0.00217)
SO_2-Sh(q4)		0.00687***		0.000751
		(0.00249)		(0.00242)
SO_2-Sh(q5)		0.00244		-2.56e-05
		(0.00192)		(0.00188)
Observations	16,278,033	16,278,033	16,278,033	16,278,033
R-squared	0.730	0.730	0.730	0.730

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable corresponds to the natural logarithm of birthweight. . Mean SO_2 corresponds to sulfur dioxide's mean concentration over the 39-week window. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, Unmarried). Columns (3) and (4) correspond to the placebo tests in which prenatal exposure to all environmental variables (SO_2, temperature, and precipitation) were computed for a standard 39-week pregnancy using each county's 1-year lagged series. All regressions are weighted by the number of births in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.23: Robustness test using 26-week window

	(1)	(2)	(3)	(4)
Window to Measure exposure	Conception-39 w	Conception-39 w	Conception-26 w	Conception-26 w
Mean SO_2	0.000139*		6.67e-05	
	(7.67e-05)		(5.77e-05)	
SO_2-Sh(q1)		0.000374		-0.00159
		(0.00217)		(0.00173)
SO_2-Sh(q2)		0.00131		0.000801
		(0.00233)		(0.00181)
SO_2-Sh(q4)		0.00563**		0.00171
		(0.00236)		(0.00172)
SO_2-Sh(q5)		0.00182		0.000418
		(0.00186)		(0.00154)
Observations	17,506,986	17,506,986	17,506,986	17,506,986
R-squared	0.725	0.725	0.725	0.725

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is natural logarithm of birthweight. Pollution exposure was computed using a window from conception to 39 weeks in Cols 1-2 and from conception to 26 weeks in cols 3-4. Mean SO_2 corresponds to sulfur dioxide's mean concentration during each window. SO_2-Sh(q j) corresponds to the fraction of days, during the respective window, in which the county's SO2 concentration lies in the j-th quintile of national distribution of SO2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Additional Controls: mother's demographics (high school dropout, marital status, pregnancy history, age), newborn's gender, in-utero weather (temperature and precipitation) through a 39-week window, and the state's unemployment rate during the pregnancy's first trimester. Regressions are weighted by the number of births in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.24: Effects of Sulfur dioxide (SO_2) on birthweight.

	(1) BW	(2) BW	(3) BW	(4) Log(BW)	(5) Log(BW)	(6) Log(BW)
Baseline SO_2	All	Low	High	All	Low	High
Observations	17,506,986	8,383,192	9,123,794	17,506,986	8,383,192	9,123,794
R-squared	0.729	0.673	0.761	0.663	0.725	0.758
SO_2-39w-Sh(q1)	1.955	4.333	-1.157	0.00120	0.000374	-0.000468
	(5.840)	(7.380)	(9.007)	(0.00276)	(0.00217)	(0.00291)
SO_2-39w-Sh(q2)	3.776	6.697	2.508	0.00263	0.00131	0.000162
	(6.741)	(8.819)	(11.39)	(0.00304)	(0.00233)	(0.00409)
SO_2-39w-Sh(q4)	15.59**	27.37***	11.80	0.00923***	0.00563**	0.00424
	(6.594)	(10.09)	(8.192)	(0.00347)	(0.00236)	(0.00294)
SO_2-39w-Sh(q5)	4.903	8.823	2.124	0.00448	0.00182	0.000323
	(5.329)	(14.19)	(6.393)	(0.00473)	(0.00186)	(0.00228)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. BW corresponds to the weight at birth (grams). Log(BW) corresponds to the natural logarithm of the birthweight. Low (High) Baseline SO2 correspond to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, high school dropout, and unmarried). newborn-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of livebirths in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.25: Sulfur dioxide (SO_2) effects on the fraction of adverse birth outcomes.

	(1) LBW_r	(2) LBW_r	(3) SGA_r	(4) SGA_r	(5) PTB_r	(6) PTB_r
Baseline SO_2	All	Low	All	Low	All	Low
Mean (Y)	51.72	47.43	90.64	88.52	94.56	92.77
Observations	17,506,986	8,383,192	17,506,986	8,383,192	17,506,986	8,383,192
R-squared	0.452	0.358	0.146	0.117	0.526	0.451
SO_2-39w-Sh(q1)	-0.534	-1.331	-1.580	-3.231	0.493	1.611
	(1.501)	(1.927)	(2.002)	(2.667)	(2.428)	(2.902)
SO_2-39w-Sh(q2)	-1.695	-3.576	-5.474	-4.353	-1.850	-4.863
	(2.243)	(3.036)	(3.990)	(5.555)	(4.901)	(5.676)
SO_2-39w-Sh(q4)	-4.428*	-6.576**	-5.626*	-6.315	-2.152	-2.393
	(2.534)	(3.001)	(2.828)	(4.584)	(3.667)	(5.275)
SO_2-39w-Sh(q5)	-3.610	-8.325	-3.826	-4.688	-6.034*	-5.799
	(2.327)	(5.023)	(3.137)	(3.902)	(3.135)	(7.478)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. LBW: Low birthweight (birthweight<=2500g). SGA: Small for gestational age (birthweight < P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race. PTB: preterm birth (gestational age<37 weeks). All outcome variables correspond to the number of adverse cases per thousand livebirths. Low (High) Baseline SO2 correspond to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is kept as the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, High school dropout, and unmarried). newborn-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of livebirths in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.26: Sulfur dioxide (SO_2) effects on the number of adverse birth outcomes

	(1) Lbw/w	(2) Lbw/w	(3) Sga/w	(4) Sga/w	(5) Ptb/w	(6) Ptb/w
Baseline SO_2	All	Low	All	Low	All	Low
Mean (Y)	0.075	0.074	0.135	0.136	0.102	0.102
Women 15-44 (1k)	85.03	80.38	85.03	80.38	85.03	80.38
Cells	343,023	160,124	343,023	160,124	343,023	160,124
R-squared	0.525	0.430	0.297	0.259	0.608	0.54
SO_2-39w-Sh(q1)	-0.00144	-0.00460	-0.00388	-0.00888*	-0.00160	-0.00346
	(0.00269)	(0.00318)	(0.00356)	(0.00479)	(0.00354)	(0.00423)
SO_2-39w-Sh(q2)	-0.00223	-0.00858*	-0.00959*	-0.0131	-0.00322	-0.0127
	(0.00376)	(0.00446)	(0.00579)	(0.00809)	(0.00731)	(0.00884)
SO_2-39w-Sh(q4)	-0.00643*	-0.0133**	-0.0111**	-0.0169**	-0.00577	-0.0124**
	(0.00372)	(0.00630)	(0.00507)	(0.00790)	(0.00428)	(0.00619)
SO_2-39w-Sh(q5)	-0.00605*	-0.0152**	-0.00890*	-0.0142	-0.0108**	-0.0164*
	(0.00314)	(0.00647)	(0.00454)	(0.00887)	(0.00471)	(0.00993)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. LBW: Low birthweight (birthweight<=2500g). SGA: Small for gestational age (birthweight < P_{10}) where P_{10} is the 10th percentile of birthweight computed for every gestational age-gender-maternal race. PTB: preterm birth (gestational age<37 weeks). All outcome variables in columns 3-8 correspond to the number of adverse cases per thousand woman of reproductive age (15-44) of the respective race. Low (High) Baseline SO2 correspond to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is kept as the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Newborn-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.27: Addressing the livebirth bias using a bounding approach

	(1)	(2)	(3)	(4)
Fraction of observations excluded on each tail	2%	2%	5%	5%
Mean SO_2	6.74e-05		8.25e-05*	
	(5.43e-05)		(4.87e-05)	
SO_2-39w-Sh(q1)		0.00107		0.000488
		(0.00160)		(0.00120)
SO_2-39w-Sh(q2)		0.00318		0.00223
		(0.00258)		(0.00211)
SO_2-39w-Sh(q4)		0.00416*		0.00321*
		(0.00232)		(0.00179)
SO_2-39w-Sh(q5)		0.00212		0.00196
		(0.00180)		(0.00153)
Observations	16,785,251	16,785,251	15,784,824	15,784,824
R-squared	0.070	0.070	0.061	0.061

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. This table emulates the bounding approach used by Lee(2009). The dependent variable is the natural logarithm of the birthweight. SO2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Cols 1-2 exclude the infants with birthweight below the 2nd percentile or above the 98th percentile. Cols 3-4 exclude the infants with birthweight below the 5th percentile or above the 95th percentile All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, high school dropout, and unmarried). Newborn-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of livebirths in each cell. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.28: Addressing the livebirth bias using novel approach

	(1) Nlbw/w	(2) Nlbw/w	(3) Nsga/w	(4) Nsga/w	(5) Nptb/w	(6) Nptb/w
Baseline SO_2	All	Low	All	Low	All	Low
Mean (Y)	1.363	1.386	1.303	1.325	1.336	1.359
Women 15-44 (1k)	85.03	80.38	85.03	80.38	85.03	80.38
Cells	343,023	160,124	343,023	160,124	343,023	160,124
R-squared	0.652	0.643	0.660	0.645	0.627	0.619
SO_2-39w-Sh(q1)	-0.0282	-0.0503	-0.0258	-0.0461	-0.0281	-0.0515
	(0.0239)	(0.0342)	(0.0225)	(0.0317)	(0.0226)	(0.0329)
SO_2-39w-Sh(q2)	-0.0285	-0.0675	-0.0211	-0.0630	-0.0275	-0.0633
	(0.0416)	(0.0564)	(0.0396)	(0.0523)	(0.0383)	(0.0522)
SO_2-39w-Sh(q4)	-0.0453	-0.0921*	-0.0407	-0.0884*	-0.0460	-0.0929*
	(0.0326)	(0.0528)	(0.0311)	(0.0517)	(0.0321)	(0.0548)
SO_2-39w-Sh(q5)	-0.0487*	-0.0781	-0.0458*	-0.0791	-0.0440*	-0.0769
	(0.0258)	(0.0512)	(0.0242)	(0.0482)	(0.0246)	(0.0491)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The novel approach consists of transforming the dependent variable and weighting the regressions by a variable that is not affected by the pollution shock (more details in section 1.4.4). Newborn-level observations were collapsed into cells by county-week-year-race. The regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Nlbw/w , nsga/w, and nptb/w correspond to the number of non-low birthweight (birthweight>2500g), non-small for gestational age (birthweight > P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race, and non-preterm birth (gestational age>=37 weeks) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Low Baseline SO2 correspond to counties in the bottom 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, High school dropout, and unmarried). Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.29 : Heterogeneous effects of SO_2 by maternal race

	(1)	(2)	(3)	(4)	(5)	(6)
Race	White	White	Black	Black	Other	Other
Baseline SO_2	All	Low	All	Low	All	Low
Mean Birthweight	3439.54	3445.87	3162.2	3178.15	3287.84	3299.73
Mean (Y)	1.097	1.189	1.274	1.324	1.305	1.334
Women 15-44 (1k)	73.58	72.37	21.39	15.69	7.10	9.64
Cells	161,102	74,929	103,500	48,788	78,421	36,407
R-squared	0.721	0.727	0.440	0.374	0.529	0.553
SO_2-39w-Sh(q1)	-0.0182	-0.0281	-0.0344	-0.110	-0.157	-0.226
	(0.0204)	(0.0242)	(0.0704)	(0.0894)	(0.163)	(0.183)
SO_2-39w-Sh(q2)	-0.0414	-0.0638	0.0377	-0.0785	0.0473	-0.0364
	(0.0386)	(0.0523)	(0.0795)	(0.0888)	(0.154)	(0.192)
SO_2-39w-Sh(q4)	-0.0575**	-0.0826*	0.0448	-0.0998	0.00526	-0.0643
	(0.0265)	(0.0422)	(0.0738)	(0.110)	(0.164)	(0.198)
SO_2-39w-Sh(q5)	-0.0424*	-0.0455	-0.0734	-0.218**	-0.0405	-0.465*
	(0.0241)	(0.0470)	(0.0484)	(0.0940)	(0.109)	(0.269)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable, Nlbw/w , correspond to the number of non-low birthweight (birthweight>2500g) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Low Baseline SO2 correspond to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). newborn-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of women 15-44 yrs. old per county-race-year.. Standard errors are clustered at the county level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

1.9 Appendix A: Methodology to create pollution measures at the county-day level.

Algorithm:

1. Start from the AQS database of pollutant *X* at the monitor-day level for year 1980
2. Append the years 1981-1991 for the same pollutant.
3. Drop the monitors with fewer than 10 months of data for the whole period (19080-1991).
4. The following steps (a-d) will select a basket of monitors and an initial and final month year for each county.
    a) For each monitor, define the initial month-year in which the pollutant starts being measured. At least 50% of the observations during the initial month must be non-missing.
    b) For each monitor, define the last month-year in which the pollutant was measured
    c) For each county, define the length as the number of periods between the first and last month as defined in the previous two steps.
    d) Drop the monitors for which more than X% of the period (as a fraction of the length defined in the previous step) must be forecasted. Three different thresholds were used: 30%, 40%, and 50%.
    e) Repeat steps (b and c) until the basket of monitors is stable.

Notes:

1. Be aware that these steps are implemented for each county; therefore, the initial and final dates will differ across counties.
2. The previous steps could have generated up to 3 different baskets (and initial and final

dates) for each county. one for each of the thresholds (30%, 40%, 50%)

Note: Up to this point, a basket of monitors has been selected for each county and threshold (30, 40% or 50%). The following part of the algorithm will fill the missings at the monitor-day level. A balanced panel at the monitor-day level will be created for each county with two or more monitors. Missings in counties with a single monitor are not filled-up. The observations corresponding to extraordinary events (e.g.: wildfires) have been kept in the database.

5. For counties with 2 or more monitors, the missings are filled with the following algorithm:

    a. All the missing patterns of the remaining monitors within the county are identified for each county-monitor. For example: if there are three monitors in the county: a, b, and c; there will be four possible missing patterns for monitor a: (b non-missing, c non-missing), (b non-missing, c missing), (b missing, c-non-missing) , (b missing, c missing). The following regression is used to predict the missings in each monitor.

$\forall\ k\ in\ the\ county's\ \ basket$:

$$y_d^k = \alpha_o + \sum_{j \neq k}^{N} \sum_{p} \alpha_{pj}^k \left(d_{pj}^k * y_j\right) + \mu_p^k + \mu_{month-year}^k + \mu_{day}^k + u_t^k$$

Where $d_{pj}^k =$

$$\begin{cases} 1\ if\ y_j\ is\ not\ missing\ under\ the\ missing\ pattern\ p\ of\ regressors\ of\ monitor\ k \\ 0\ otherwise \end{cases}$$

$$\mu_{day}^k : dummies\ for\ 1\ up\ to\ 365$$

$$\mu_p^k : missing\ pattern\ fixed\ effects$$

a. Now set bounds for the predictions:
    i. Any negative prediction is replaced by zero
    ii. For each county, find the maximum reading of any monitor. Any prediction 20% larger than the maximum is replaced by the maximum.

The final step consists of creating a pollution measure at the county-day level: A county-day measure is created for a given day only if all the monitors within the county's basket are non-missing on that day (or have been filled up) . Otherwise, a missing is imputed for the county-day average. Notice that three different series were created for each county: one for each of the thresholds described in step (4d). There may be identical in some cases.

Final Note: The 50% threshold produces longer time series; however, the degree of imputation is higher. It is recommended to use the 50% threshold for the baseline empirical strategy and test the robustness of the results using the indices generated with the 40% and 30% thresholds.

1.10 Appendix B: Conceptual framework for the livebirth bias

This methodological appendix introduces a conceptual model to analyze how livebirth bias (i.e., sample selection) affects the estimates of the polluntants' effects in [1.2] and [1.3].

The following equations model the effect of pollution on the incidence of low birthweight (LBW). However, the same framework can be applied to other adverse neonatal outcomes (e.g., preterm birth (PTB) and small for gestational age (SGA)).

Definitions and nomenclature:

There are three possible states: LBW and NLBW correspond to low birthweight and non-low birthweight and are observed in the data. In contrast, FD corresponds to fetal death and is unobserved in the birth certificate data. By Aggregating LBW and NLBW we obtain the universe of livebirths (LB).

Superscript "o" refers to the value of the respective variable absent the air pollution shock.

τ_{lbw}^{fd}: Number of fetuses that would have been low birthweight but die due to air pollution shocks.

τ_{nlbw}^{fd}: Number of fetuses that would have been born non-low birth weight (i.e., healthy weight) but die due to air pollution shocks.

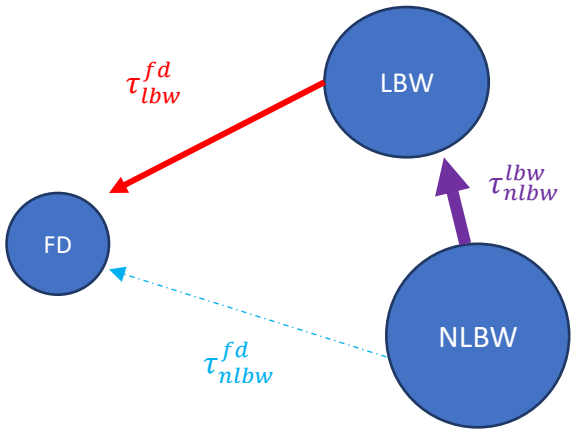
$\tau_{nlbw:}^{lbw}$ Number of infants that would have been born non-low birth weight (i.e., healthy weight) but are born low birthweight due to air pollution shocks.

$$\tau_{LB}^{fd} = \tau_{lbw}^{fd} + \tau_{nlbw}^{fd}$$

$$lbw = lbw^{o} - \tau_{lbw}^{fd} + \tau_{nlbw}^{lbw}$$

$$nlbw = nlbw^{o} - \tau_{nlbw}^{fd} - \tau_{nlbw}^{lbw}$$

Aggregating both states:



$$LB = lbw + nlbw$$

$$LB = (lbw^{o} + nlbw^{o}) - \left(\tau_{lbw}^{fd} + \tau_{nlbw}^{fd}\right)$$

$$LB = (LB^{o}) - \left(\tau_{LB}^{fd}\right)$$

As seen on the graph on the right, analyzing the effects of air pollution using the low birthweight rate is challenging because "LBW" state receives both outflows and inflows, and the magnitude of those inflows could be similar. Furthermore, both the numerator and denominator are affected by sample selection. Equation [B1] below shows the effect of pollution on the low birthweight rate; it shows that the marginal effect of an air pollution shock on the low birthweight rate can be negative when τ_{nlbw}^{lbw} is small relative to τ_{lbw}^{fd}.

$$lbw_r = \frac{lbw}{LB} = \frac{lbw^o - \tau_{lbw}^{fd} + \tau_{nlbw}^{lbw}}{LB^o - (\tau_{lbw}^{fd} + \tau_{nlbw}^{fd})}$$

By taking the derivative of the previous equation with respect to the air pollution variable and manipulating the expression, we obtain [B1]:

$$\frac{dlbw_r}{dP} = \frac{LB * \left(-\tau_{lbw}^{fd} + \tau_{nlbw}^{lbw}\right) - lbw\left(-\left(\tau_{lbw}^{fd} + \tau_{nlbw}^{fd}\right)\right)}{LB^2}$$

$$\frac{dlbw_r}{dP} = \frac{1}{LB}\left(-\tau_{lbw}^{fd} + \tau_{nlbw}^{lbw} + lbw_r(\tau_{lbw}^{fd} + \tau_{nlbw}^{fd})\right)$$

$$\frac{dlbw_r}{dP} = \frac{1}{LB}\left(\tau_{nlbw}^{lbw} + lbw_r\,\tau_{nlbw}^{fd} + (lbw_r - 1)\,\tau_{lbw}^{fd}\right)$$

$$\frac{dlbw_r}{dP} = \frac{1}{LB}\left(\tau_{nlbw}^{lbw} + lbw_r\tau_{nlbw}^{fd} - nlbw_r\tau_{lbw}^{fd}\right) \quad [B1]$$

As illustrated by the size of the arrows in the graph, this conceptual model assumes that $\tau_{nlbw}^{fd} \ll \tau_{lbw}^{fd}$. This makes sense because NLBW infants before the air pollution shock ($nlbw^o$) should have better underlying health than LBW infants before the air pollution shock (lbw^o). Hence, these infants should be more resilient to any shock. Thus, direct transitions from non-LBW to fetal death should be less frequent than from LBW. Consequently, a way to lessen this sample selection problem would be to analyze pollution's effects on the number of non-low birthweight infants.

$$\frac{dnlbw}{dP} = \left(-\tau_{nlbw}^{fd} - \tau_{nlbw}^{lbw}\right) \quad [B2]$$

Notice that while $\frac{dlbw_r}{dP}$ could be positive or negative when air pollution has a negative effect on both the fetal survival rate (i.e., extensive margin) and neonatal outcomes (i.e., intensive margin), $\frac{d\ nlbw}{dP}$ is unambiguously negative.

Essay 2

Does free health care mitigate the effect of prenatal air pollution on birth outcomes?

2.1 Introduction

Prenatal exposure to air pollution has been linked to fetal[56] and infant death,[57] adverse birth outcomes,[58] and worse outcomes in adulthood[59] and across generations.[60] Foremost, these impacts vary across social and economic factors. In particular, the health impacts vary by race and income (Chay & Greenstone, 2003a; Currie & Walker, 2011; Jayachandran, 2009; Arceo et al., 2016). Differences in pollution exposure and healthcare access have been proposed as sources of such disparities (Graff-Zivin & Neidell, 2013; Hsiang et al., 2019). Previous research has shown that higher pollution exposure contributes to larger marginal damages experienced by disadvantaged populations (Mohai et al., 2009; Banzhaf et al., 2019; Colmer et al., 2020; Currie et al., 2020), but there is little empirical evidence for the role of health care in mitigating the effects of air pollution. While previous research found evidence that medical care mitigates weather's effects on infant health (Banerjee & Maharaj, 2020; Gunnsteinsson et al., 2019), no previous research has examined the causal impact of healthcare on the pollution-infant health relationship.

[56] Effect on fetal death per pollutant: $PM_{2.5}$ (Ebisu et al., 2018; DeFranco et al.,2015), O_3 (Mendola et al., 2017), NO_2 (Green et al.,2015), and SO_2 (Faiz et al., 2012).
[57] See Chay & Greenstone., 2003a, 2003b; Currie & Walker (2011), Currie & Neidell (2005), Currie, Neidell & Schmieder (2009)
[58] See Currie & Walker (2011), Bell et al. (2007), Currie, Neidell & Schmieder (2009).
[59] See Isen, Rossin-Slater & Walker(2017), Almond & Currie (2011), Almond, Currie & Duque (2017).
[60] See Colmer & Voorheis (2020)

This paper examines and quantifies the impact of free health care in reducing air pollution's effects on fetal death and birth outcomes. I empirically examine these effects using restricted-use vital statistics data from 1981-1991, daily air pollution monitoring data, and policy variation from Medicaid's expansion during the 1980s. This expansion provided free access to prenatal health care, and at least one year after birth, to low-income women who became pregnant. Ex-ante, the sign of the effect is ambiguous. Access to free nutritional supplementation (e.g., vitamins, iron, folic acid) during prenatal controls could make the fetus more resilient to pollution shocks. However, pollution avoidance could decrease when healthcare becomes free (Graff-Zivin & Neidell., 2013). If the latter effect dominates, pollution's marginal damage would increase. Additionally, it is not clear how healthcare's efficacy changes with the pollutant's concentration.

I utilize spatial-temporal variation in Medicaid's expansion to estimate its impact on the effect of high pollution days on health outcomes. I build upon two empirical approaches: a panel-fixed effects model to identify the causal impact of air pollution on health and the empirical strategy used by East, Miller, Page, and Wherry (2023) to estimate the effect of Medicaid expansion on birth outcomes. Specifically, I interact Medicaid expansion, a binary variable at the state-year level, with exposure to the pollutant's concentration level bins, which vary by county of residence and week of conception.[61] Exposure was measured as the fraction of days during a 39-week window in which the county's daily concentration fell in each of the five quintiles of the pollutant's national distribution.[62] The pollutant's effects are identified using temporal variation in exposure through conception weeks within the same county and year, and controlling by county-quarter and

[61] Assumed to be two weeks after the last day of the Menstrual period. This date is reported on the birth certificates.

[62] Quintile cutoffs were generated using each pollutant's concentrations at the county-day level for the set of counties that had monitoring data in the AQS during 1981-1991. The effects in the two highest and lowest concentration bins are estimated relative to the third one (i.e., omitted category). SO2 concentration range per quintile (parts per billion): q_1 ($0 \leq SO_2 < 1.84$) q_2 ($1.84 < SO_2 \leq 4.39$), q_3 ($4.39 < SO_2 \leq 7.66$), q_4 ($7.66 < SO_2 \leq 13$), q_5 ($SO_2 > 13$).

calendar-month seasonal effects, the unemployment rate during the pregnancy's first trimester, in-utero weather (temperature and rainfall), and mothers' demographics.[63]

The results from the previous chapter show that high SO_2 concentrations during the prenatal period decreases the number of infants born with non-adverse outcomes per woman of reproductive age. In this chapter, I build upon those results and study if Medicaid's expansion mitigated SO_2 effects on fetal death and birth outcomes.

As a first result, I find that Medicaid's expansion decreased the fetal deaths caused by SO_2 in low-pollution counties but not in highly polluted ones, which is consistent with the previous chapter's finding of an insignificant effect of SO_2 on fetal death in highly polluted counties. The fertility rates associated with one additional day in the fourth and fifth quintiles of SO_2 exposure were 0.031% and 0.072% higher after Medicaid's expansion, respectively. Consequently, Medicaid's expansion increased a county's annual number of infants surviving an additional day in SO_2's fourth and fifth quintiles by 1.62 and 3.73, respectively. These results suggest that Medicaid is more valuable (i.e., saves more lives) at higher pollution concentrations. The back-of-the-envelope calculation suggests that Medicaid's expansion reduced the number of fetal deaths linked to SO_2 by 143,462 during 1981-1991 in the US. These pregnancies would otherwise have ended in fetal deaths due to SO_2.

As a second result, I find that Medicaid's expansion mitigated the effect of SO_2 on birthweight; however, this result is only uncovered when analyzing the number of non-low birthweight infants

[63] Mother's age, pregnancy history, marital status, and educational attainment. Also, the fraction of male newborns.

per woman of reproductive age, which is robust to livebirth bias –a type of sample selection bias. The estimates of the interaction terms between *Medicaid expansion* and *Pollution exposure*, which answer this paper's question, are potentially biased when traditional birth outcomes (e.g., birthweight, low birthweight[64] rate) are used as dependent variables. In principle, this bias can arise if access to prenatal healthcare reduces the fetal deaths caused by pollution, and the infants marginally saved are negatively selected.

Conceptually, prenatal care can impact the effects of pollution through two margins: (i) on the extensive margin, by preventing fetal deaths linked to pollution, and (ii) on the intensive margin, by mitigating pollution's effects on neonatal outcomes for the infants that would be born irrespective of the pollution shock. The bias arises when the infants marginally saved by Medicaid's expansion are negatively selected (i.e., born with adverse birth outcomes). This bias was overcome by using the number of infants born with non-adverse outcomes per woman of reproductive age as dependent variables.[65] Traditional bounding methodologies to address sample selection (i.e., adaptations of Lee (2009)) were ineffective in this context.

Using the number of infants born with a weight above 2500 grams per woman of reproductive age (*nlbw/w*) as the outcome variable, I find that Medicaid's expansion mitigated the impact of high SO_2 concentrations on birth outcomes in low-pollution counties and at the national level. For the national sample, Medicaid's mitigation of one-additional day in the fourth and fifth quintile of SO_2 exposure led to increases of 0.027% and 0.028% in the number of infants born non-low birth weight per woman of reproductive age. These results suggest that the impact of health care on the

[64] An infant is considered low birth weight if it is less than 2500 grams at birth.
[65] Non-low birthweight (nlbw/w), non-preterm birth (nptb/w), Non-small for gestational age (nsga/w).

SO_2 – birthweight relationship is homogeneous across high concentrations; however, in low-pollution counties, the effect in the fifth quintile was about twice the size of the fourth one. Similar results were obtained for other neonatal outcomes (e.g., preterm birth, small for gestational age). In addition, the back-of-the-envelope calculation for the national sample suggests that Medicaid's expansion increased the annual number of non-low birthweight infants by 176 per county, corresponding to an increase of 40 infants born with a weight above 2500 grams per thousand births. This estimate is an upper bound because it implicitly assumes that one-day effects can be extrapolated to longer periods.

Finally, I find that Medicaid's expansion contributed to closing the gap in the health effects of air pollution between blacks and whites. As Medicaid expanded, SO_2's impact on the number of infants born with a weight above 2500 grams per woman of reproductive age (*nlbw/w*) decreased more for blacks than whites. For blacks, *nlbw/w* associated with one additional day in the fourth quintile increased by 0.064% after Medicaid's expansion. For whites, the increase was 0.037% but linked to one additional day in SO_2's fifth quintile. These effects correspond to increases of 0.8 and 0.37 in non-low birthweight infants (i.e., healthy infants) per thousand births for blacks and whites, respectively.

This paper contributes to three sets of literature. The central contribution lies within the environmental literature attempting to identify the underlying mechanisms driving the link between environment and health. Previous research has exploited quasi-experimental variation in healthcare or income to study if they drive the heterogeneity in the effects of in-utero –or shortly after birth—environmental factors on health (Banerjee & Maharaj 2020; Gunnsteinsson et al.,

2019) and educational attainment (Aguilar & vicarelli, 2011; Adhvaryu et al., 2018). My results add to this literature by showing for the first time, to the best of my knowledge, that health care mediates in-utero air pollution impacts on infants' health. In Hsiang et al. (2019)'s terminology, my results show that the air pollution-birthweight damage curve is heterogeneous across levels of healthcare access. Previous research has addressed this question with an observational research design and concluded that prenatal care did not affect the air pollution-birthweight relationship (Colmer et al., 2021).

The second contribution lies within the health economics literature that studies the impacts of healthcare on infants' health. Previous literature has established that providing low-income women with health insurance (e.g., Medicaid in the US) reduced fetal deaths (Currie & Grogger, 2002), improved birth outcomes (Currie & Gruber, 1996a, 1996b; East et al., 2022), and reduced infant and child mortality (Goodman-Bacon, 2018). However, poor birth outcomes can occur due to many types of shocks (e.g., nutritional, income, environmental (e.g., temperature, rainfall, pollution), maternal disease, and/or stress), and it is unclear if healthcare effectively mitigates all of them. My results suggest that healthcare access during the prenatal period mitigates the effects of in-utero air pollution on health at birth.

The third contribution lies within the environmental justice literature. Previous environmental research has found larger air pollution effects on African Americans' infant health than whites (Chay & Greenstone, 2003a; Currie & Walker, 2011). Additionally, previous literature on Medicaid has established that expanding access to free health insurance improved infant health outcomes of African Americans more than for other races (Fisher,1992; Bhatt & Beck-Sagué,

2018). My results link both literatures and suggest that unequal access to healthcare contributes to the gap in the health effects of prenatal exposure to air pollution between blacks and whites in the US. Furthermore, Medicaid's expansion improved environmental justice by reducing gaps.

The remainder of the paper is laid out as follows: Section 2 reviews the background information about Medicaid and its effects on short and long-term outcomes. Section 3 introduces the conceptual framework to analyze prenatal care's potential in mitigating the impacts of in-utero air pollution. Section 4 describes the data. Section 5 explains the empirical strategy and robustness tests. Section 6 presents the results. Section 7 discusses the results and proposes desirable extensions. Finally, Section 8 concludes.

2.2 Background

This section provides background information about the policy variation I use to measure increases in healthcare access during the 1980s. Background information about sulfur dioxide and its impact on birth outcomes was provided in the previous chapter.

Medicaid provides access to health insurance for low- and moderate-income families in the US. This program covers the financial cost of the delivery, but also provides pregnant women with access to prenatal controls—which includes free nutritional supplementation (e.g., vitamins, iron, folic acid, etc.). The federal government and the states jointly finance this program, representing the second largest form of public-sector investment in children after K–12 education (Isaacs & Edelstein, 2017). In the early 1980s few low-income pregnant women were covered by Medicaid. Medicaid coverage for pregnant women was linked to Aid to Families with Dependent Children

(AFDC).[66] Only 4 out of 10 women of reproductive age with family income below the federal poverty line were covered by Medicaid in 1984 (Gold & Kenney, 1985). The expansions during the 1980s gave access to low-income women irrespective of family structure (i.e., marital status) and included childless pregnant women. It is estimated that Medicaid funded about 32 percent of all deliveries in 1991, compared with only 15 percent in 1985 (Singh et al., 1994).

Previous research has documented that increased Medicaid eligibility for low-income pregnant women during the 80s had positive effects on access to prenatal care (Currie & Gruber, 1996b, 2001; Dubay et al., 2001; Howell, 2001), birth outcomes (Currie & Gruber, 1996a, 1996b), infant and child mortality (Goodman-Bacon, 2018), long-term outcomes (Miller & Wherry, 2019), and even inter-generational effects on health (East et al., 2023).

Fertility was not affected by Medicaid's expansion during the 80s. Zadvoni & Bitler (2010) found no evidence that Medicaid affected birth or abortion rates, except for the birth rate among white women without a completed high school education. Similarly, DeLeire et al. (2011) concluded that no robust evidence indicates that Medicaid affected fertility. Lastly, Currie & Grogger (2002) found that higher income cutoffs for Medicaid and increased welfare caseloads reduced fetal deaths during the early 1990s in the United States.

Different measures of the generosity of Medicaid have been used. Previous researchers have designed measures that isolate variation driven by each state's changes in eligibility rules (i.e., policy variation) and exclude variation caused by social or economic shocks— potentially

[66] AFDC was a federal assistance program that provided financial assistance to children whose families had low or no income. Tying Medicaid coverage to being and AFDC beneficiary excluded low-income women who were married or first-time pregnant women in some states.

correlated with health outcomes. For example, seminal research on the effects of Medicaid on birth outcomes used simulated eligibility for Medicaid (Currie & Gruber, 1996a, 1996b). This continuous variable measures the fraction from a nationally representative sample of women 15-44 years old that would be eligible for Medicaid if they got pregnant each year and state. On the other hand, East, Miller, Page & Wherry (2023) used a binary variable to measure Medicaid's expansion beyond AFDC. This binary variable identifies for each state the year in which simulated eligibility had the largest increase. The baseline empirical strategy of this paper uses this binary variable. Estimates using simulated eligibility (continuous variable) are used for robustness tests.

2.3 Conceptual framework

This section introduces a theoretical framework to determine the direction of Medicaid's expansion impact on the pollution-birth outcome relationship. Three different channels were considered to determine, a priori, the sign of the interaction between Medicaid's expansion and air pollution: medical-biological, information, and an optimal response channel. Since the channels suggest opposite directions ex-ante, it is impossible to determine whether the expansion of Medicaid will mitigate or worsen pollution's health effects. However, the medical-biological channel, which suggests a decrease in the effect of pollution on neonatal outcomes, is believed to be the most relevant during the period analyzed.

The medical-biological channel refers to the physiological effect of receiving medical attention (nutrition and drug counseling, immunizations, and early diagnosis) during the prenatal controls. Based on this channel, we expect healthcare access to lessen air pollution's effect on birth outcomes via receiving free vitamins during the prenatal controls. First, several studies of

Medicaid's prenatal expansions document increased use or improved timing and adequacy of prenatal care (Currie & Gruber, 1996b, 2001; Dubay et al., 2001; Howell, 2001). Second, obstetricians routinely recommended nutritional supplementation to pregnant women during the 1980s (Hemminki, 1988). Third, previous research has found evidence that nutritional supplementation mitigates the effects of air pollution on health. For instance, Gunnsteinsson et al. (2022) found that vitamin supplementation at birth reduced the damage caused by environmental factors on an infant's health. Similarly, Zhong et al. (2017) found that ingesting vitamin B in adulthood attenuated air pollution's impact on the epigenome.[67] Therefore, it is reasonable to suspect that in-utero prenatal supplementation could mitigate pollution's effects.

Health care's effectiveness in mitigating pollution's health effects could also vary across the pollution distribution. For example, we do not know if health care is equally effective at high concentrations. There may be a threshold above which medical treatments are ineffective. Therefore, in the empirical analysis, I use different concentration bins to allow for potential non-linearities in the impact of Medicaid's expansion across the pollution concentration.

It is hypothesized that prenatal iron prescription could reduce sulfur dioxide's effect on the incidence of fetal death and adverse birth outcomes. The sulfate ion (SO_4^{-2}), a particle derived from the oxidation of SO_2, has been linked to the incidence of anemia during the third trimester of pregnancy[68] (Xie et al., 2022). Therefore, we would expect newborns of women who received iron during the prenatal controls to be more resilient to SO_2's shocks. This pathway is presumed to be

[67] Previous research suggests that air pollution modulates the epigenetic mark. Specifically, it interferes with DNA methylation (DNAm), a biological process required to produce proteins. (Rider et al., 2019)
[68] $PM_{2.5}$ has been linked to anemia in older adults in the US (Honda et al., 2017) and China (Elbarbary et al., 2020).

relevant for the US. For instance, during the early 2000s, it was estimated that 29.5% of pregnant women in the US had iron deficiency[69] during the third trimester, and the prevalence was higher among Mexican Americans and non-Hispanic blacks (Mei et al., 2011).

The information channel refers to the potential increase in pollution avoidance after receiving information about air pollution's adverse effects on health. Based on this channel, we would expect Medicaid's expansion to lessen the effect of air pollution on birth outcomes. For example, if pregnant women were unaware of air pollution's health impacts on their offspring and the doctors informed them when they got access to Medicaid, they may have reduced their exposure to outdoor air pollution. Reduced exposure would lead to a decrease in pollution's health impacts. Nevertheless, there is no evidence that obstetricians advised pregnant women to avoid pollution in the US during the 1980s (Hemminki, 1988).

Furthermore, the information channel could play a role only if individuals have access to air quality information or could tell by environmental conditions. Previous research has shown that individuals react to pollution alerts (Neidell, 2009; Janke, 2014); however, there was no official air quality information for any pollutant, nor pollution alerts, except for smog, in the US during the 80s. Absent pollution information, avoidance relies on how salient the pollutant is. Sulfur dioxide has a strong, pungent smell, and particulate matter (PM) can cause haze (US EPA, 1982). However, only individuals who personally experienced poor health in response to those olfactory or visual stimuli in the past would engage in pollution avoidance (Bresnahan et al., 1997).

[69] Iron deficiency anemia is a risk factor for perinatal complications like pre-eclampsia, low birthweight, prematurity, and perinatal mortality; preventive iron supplementation during pregnancy reduces the incidence of anemia in mothers and low birth weight in neonates (Imdad & Bhutta, 2012).

Therefore, we would not expect pregnant women to react to the smell of SO_2 or haze caused by PM unless they were highly susceptible to these pollutants before pregnancy.

Lastly, the optimal response channel refers to the theoretical model from Graff-Zivin & Neidell (2013). In this model, pollution avoidance and medical treatment can be substitutes in the health production function. Hence, pollution avoidance, which is costly to individuals, could decrease when medical treatment becomes cheaper. Consequently, pollution exposure could increase when individuals get access to free health insurance and eliminate the gains of the medical-biological channel. Nevertheless, the conditions required for this channel's feasibility are unlikely to hold during the 80s. Individuals must: (1) know that air pollution is bad for health, (2) be able to identify it, (3) rationally choose to avoid it—all of them before being granted access to Medicaid, and (4) re-optimize once they get access to free health insurance.

2.4 Data

The empirical analysis relies on multiple data sources. Section 1.3 (previous chapter) describes the sources and methodologies to build the health outcomes, measure SO_2, weather (temperature and precipitation), and unemployment rate during pregnancy. In this section, I complete the information needed for the empirical strategy by explaining how I measured increased access to health care during the prenatal period. Additionally, I describe the county-level economic controls used in the robustness tests.

Two measures of Medicaid were used: Simulated prenatal eligibility for Medicaid, a continuous variable; and Medicaid expansion, a binary one. The baseline empirical strategy relies on the latter one. Simulated prenatal eligibility, the continuous variable, is used for robustness tests.

Medicaid expansion, the binary variable, was taken from East, Miller, Page & Wherry (2023). This variable switches from zero to one when the states experience the largest jump in simulated prenatal eligibility for Medicaid. States with high levels of simulated eligibility before 1980 correspond to the control group and are coded as one always. Treated states experienced Medicaid expansion in different years throughout 1980-1985. Table 2.1 shows the number of states in the control group and each expansion cohort for different samples. Figure 2.1 shows the states that belong to each expansion cohort and the control group, and Figure 2.2 shows the fraction of newborns conceived in states that had expanded Medicaid.

Simulated prenatal eligibility for Medicaid was created using the methodology from Miller & Wherry (2019), which enhanced the seminal methodology designed by Currie & Gruber (1996a). Broadly, the methodology consists of two steps: First, building national representative samples of women 15-44 years old for each year during 1980-1991 using the Current Population Survey Annual Social and Economic Supplement (CPS-ASEC), and second, computing the fraction of women eligible for Medicaid if they got pregnant each year and state. I created measures for all women 15-44 years old and separately by race (white, black, and other). Figure 2.3 shows the temporal variation in simulated eligibility for Medicaid in treated vs. control states for the aggregate US, and Figure 2.4 disaggregates by race.

County-level controls to proxy for local economic conditions and government transfers were built using Bureau of Economic Analysis (BEA) data. The unemployment rate is available at the state level with monthly frequency. Per-capita income was taken from the deflated annual county-level BEA series. Government transfers were computed using county-year series from the Regional Economic Information System (REIS) sourced from the BEA[70]. Per capita government transfers were divided into unemployment insurance and non-medical welfare programs. The latter was computed excluding government expenditure on medical care and supplemental security income, divided by the population 64 years old or older. The number of women 15-44 years old was taken from the Surveillance, Epidemiology, and End Results Program (SEER) in the respective county and year. The number of hospital beds per capita per county was built using raw data from the Annual Hospital Association Survey (AHA) following Hoynes et al. (2016). Finally, I used the 1990 NCHS's urban-rural county classification for robustness tests.

2.5. Empirical strategy

This paper's empirical strategy aims to quantify the impact of Medicaid's expansion on the effects of air pollution on fetal death and birth outcomes. Two different empirical approaches have been proposed to test if a given factor (e.g., health care) mediates air pollution's effect. First, Hsiang, Oliva & Walker (2019) propose a strategy to simultaneously estimate the health effects of pollution and how much a factor impacts it. Their method requires exogenous variation in air pollution and the potential source of heterogeneity (e.g., health care) and orthogonality between the two. On the other side of the spectrum, Barwick et al. (2019) propose a strategy to identify, under certain

[70] Current transfer receipts of individuals from governments consist of: Retirement and disability insurance benefits, medical benefits, income maintenance benefits, unemployment insurance compensation, veterans' benefits, education and training assistance, and other transfer receipts of individuals from governments.

assumptions, the impact of a given factor on the pollutant's effects, even if the pollutant's variation were endogenous or the estimate of the pollutant's effect suffered from attenuation bias due to classical measurement error. In this paper, I aim to achieve the goals of the former strategy. Nevertheless, for the analysis by race, the estimates for blacks may be biased due to measurement error. This concern is further discussed in section 2.5.2.

Broadly, the empirical strategy relies on the interaction term of two variables (Medicaid and air pollution) in a panel data model. Interpreting interaction terms in this setting is challenging, and it is generally necessary to include additional interaction terms or fixed effects to draw causal inferences rather than estimating heterogeneous effects. The following section presents this discussion, and the following two sections describe the regression models.

2.5.1 Interaction terms in panel data models

As previously mentioned, this paper follows the framework presented by Hsiang et al. (2019). In principle, this method could be used in any setting with exogenous and orthogonal sources of variation in the environmental hazard and the factor modifying it (e.g., health care). Nonetheless, careful consideration is needed when both variables are non-constant, and the empirical strategy uses fixed effects (FE) over longitudinal data. Methodological Appendix C derives the analytical decomposition of the interaction term in such a setting while including fixed effects over the cross-sectional and temporal dimensions. From those derivations, we learn that the interaction term does not necessarily have a causal interpretation, even if each variable's remaining variation –net of all fixed effects— is exogenous. The interaction term can be analytically decomposed into five different terms in this general setting. Hence, when a single interaction term is included in the

regression model, identifying the source of variation driving the result is impossible. Consequently, neither is interpreting the coefficient.

The intuition behind the problem discussed above could be understand with the following example. Suppose we have a panel with two counties and two time periods. The counties have low and high baseline levels of healthcare access, and it increases in both of them in the second period. Suppose also that we run a regression model with a single interaction term between pollution and healthcare access and find a positive effect for the interaction term on birth outcomes. Two situations are possible: (a) counties with a higher baseline have smaller pollution effects (i.e, heterogeneity driven by between variation), or (b) the effect of pollution declines when healthcare access increase (i.e, heterogeneity driven by the within variation). A single interaction model does not allows us to identify the source of variation driving the results, and, arguably, the latter is more suitable for a causal interpretation.

I address this problem by making two changes from the baseline setting in Methodological Appendix C: (i) I use county-year FE instead of county and year FE to remove the potentially endogenous sources of variation from the interaction term. (ii) I used a binary variable to measure policy variation in Medicaid instead of simulated prenatal eligibility for Medicaid –which is continuous. Methodological Appendix D shows the analytical decomposition of the interaction term when these two changes are combined. Below, models [2.1] and [2.2] were estimated following this strategy.

Interacting a binary and a continuous variable is a standard method used by researchers analyzing the effects of an interaction term in a panel data model (Rossin-Slater & Wüst, 2015; Malamud et al., 2016; Gunnsteinsson et al., 2022; Mullins & White., 2020; Aguilar & Vicarelli., 2011; Adhvaryu et al., 2018). However, to the best of my knowledge, this is the first paper that zooms into the analytical decomposition of the interaction term and explicitly shows how to remove from the interaction term the variation that makes a causal interpretation infeasible.

Lastly, it is worth mentioning that given the variation in the timing of Medicaid's expansion (i.e., states expanded at different years), the interaction terms in models [2.1] and [2.2] could be subject to bias if Medicaid had heterogeneous effects across time or cohorts of expansion (Goodman-Bacon, 2021; Sant'Anna & Callaway, 2020; Roth et al., 2023). These concerns could be addressed using an event study framework as the one East et al. (2023) used. I did not do so because EPA air pollution monitoring data started in 1981. Thus, I could not have a balanced panel for the pre-period. However, East et al. (2023) reported that the estimates from Currie & Gruber (1996) fall within the 95 percent confidence interval of the event study's estimate. This suggests estimates from simple Difference in Difference models as [2.1] and [2.2] would be unbiased. Furthermore, I also estimate model [2.3], which builds up on Currie & Gruber's (1996) empirical strategy. This model analyzes how the effects of air pollution vary in response to changes in prenatal simulated eligibility for Medicaid.

2.5.2. Fetal death

The impact of Medicaid's expansion on SO_2's effect on fetal deaths is estimated using the following model.

$$FR_{cwyr} = \alpha_{cyr} + \alpha_{mr} + \alpha_{cQr} + \sum_{j\ 1..5,\neq 3} \rho_j\, q_j\left(SO_{2_{cwy}}\right) * D_{sy} + \sum_{j\ 1..5,\neq 3} \beta_j\, q_j\left(SO_{2_{cwy}}\right) + \delta W_{cwy}$$

$$+ \theta U^{Q1}{}_{smy} + v_{cwyr} \quad [2.1]$$

The previous model expands model [1.1] –described in section 1.4.1 in previous chapter— by adding the interaction term between SO_2 and D_{sy}. D_{sy} equals 1 if state s expands Medicaid during conception year y or has already expanded Medicaid in the past, 0 otherwise. Intuitively, the impact of Medicaid's expansion on the effect of a concentration bin on fetal death is identified by comparing the mean fertility rate associated with that concentration bin in treated states before the expansion with the mean fertility rate for the same concentration bin in control and treated states after the expansion. Robustness tests are discussed in section 2.6.

Lastly, a critical property of interaction terms— that applies to model [2.1], above, and [2.2] below— is that under some conditions $\rho\ in$ [2.1] $and\ \phi\ in$ [2.2] can be consistently estimated even if β is not (Barwick et al., 2019). For example, classical measurement error in pollution could bias $\hat{\beta}$ towards zero, and $\rho\ and\ \phi$ could still be consistently estimated if the nature and degree of measurement error does not change before and after the expansion. Empirical evidence to support the orthogonality between meausurement error in pollution and Medicaid's expansion is discussed in section 2.6.4. This property will be helpful in estimating heterogeneous effects by race because the effects of SO_2 on black infants' may be biased due to measurement error.

2.5.3 Neonatal outcomes.

The impact of Medicaid's expansion on SO_2's effect on neonatal outcomes is estimated using the following model.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{mr} + \alpha_{cQr} + \sum_{j\ 1..5,\neq 3} \phi_j\, q_j\left(SO_{2_{cwy}}\right) * D_{sy} + \sum_{j\ 1..5,\neq 3} \beta_j\, q_j\left(SO_{2_{cwy}}\right) + \delta W_{cwy} + \theta U^{Q1}{}_{smy} + \gamma X_{cwyr} + u_{cwyr} \quad [2.2]$$

The previous model expands model [1.3] –described in section 1.4.2 in previous chapter— by adding the interaction term between SO_2 and D_{sy}.

The main distinction between models [2.1] and [2.2] is that the interaction term coefficients, ϕ_j, in [2.2] could be biased downward due to livebirth bias (i.e., sample selection). The impact of Medicaid's expansion on SO_2's marginal effect on neonatal outcomes can be analytically decomposed into two margins. First, the expansion could affect neonatal outcomes by changing the number of fetuses that survive pollution shocks (i.e., extensive margin). Second, Medicaid could change SO_2's effects by improving neonatal outcomes for the infants that would have survived pollution shocks irrespectively of Medicaid's expansion (i.e., intensive margin). This distinction is crucial because the extensive margin response, captured by ρ_j, in [2.1], can bias ϕ_j , in [2.2], downwards. Intuitively, this would happen if the fetuses marginally saved by Medicaid are born with poor birth outcomes.

As discussed in methodological appendix F, the outcome variable for model [2.2] must be carefully constructed to obtain an estimate that can be interpreted easily. If the rate of adverse birth outcomes

were used (e.g., low birthweight rate), the coefficients ϕ_j in [2.2] would be described by equation [F1]. Thus, we would not be able to learn much from it because it is a non-linear combination of many parameters. On the other hand, if we use the number of non-adverse birth outcomes per woman of reproductive age (*nlbw/w, nptb/w, nsga/w*) as the outcome variables in [2.2], the coefficients can be used to approximate the additional number of infants born with healthy (i.e., non-adverse) outcomes due to Medicaid's mitigation of the effect of SO_2 on infant health (equation [F2]).

Intuitively, this strategy works because the intensive and extensive margin effects go in the same direction when *nlbw/w* is used as the dependent variable. For instance, suppose prenatal care prevents fetal deaths caused by air pollution and mitigates its effects through the intensive margin. Then, if the infants marginally saved by Medicaid's expansion were positively selected, *nlbw/w* would increase through the response on both margins. On the other hand, if the infants were negatively selected, *nlbw/w* would increase due to the intensive margin effect and would be unaffected by the extensive margin response.

Finally, in the spirit of Lee (2009)'s methodology to address sample selection, I attempted to find upper and lower bounds for Medicaid's impact on SO_2's effect on birthweight. I used a three-stage method. First, I used model [1.1] to estimate the additional number of live births per woman of reproductive age in response to Medicaid's expansion. Second, I did back-of-the-envelope calculations to find the percentage increase in live births, *s%* . Third, I sorted the observations by birthweight within each state-race-year, excluded the bottom (top) *s%* observations in the post-

expansion period, and used those samples to estimate [2.2] using log birthweight as the dependent variable and find the upper (lower) bound effects.

2.5.4 Robustness tests.

This section presents seven robustness tests for models [2.1] and [2.2]. Additionally, I introduce model [2.3], which interacts simulated eligibility, a continuous variable instead of binary, and pollution.

The first robustness test consisted of excluding control states. Models [2.1] and [2.2] rely on the (implicit) assumption that Medicaid's impact on air pollution health effects is homogeneous across treated and control states. However, different states have different demographics and generosity in social welfare programs—which could also mediate the pollutant's effect. Therefore, Medicaid's expansion could affect the marginal effect of pollution differently in treated and control states. By restricting the sample to treated states, Medicaid's impacts are identified by comparing the average effect of SO_2 in treated states before and after the expansion.

As a second robustness test, I allowed for heterogeneous effects of air pollution across states. As discussed in methodological appendix E, the impact of Medicaid's expansion on the marginal effect of pollution could be biased if Medicaid had heterogeneous impacts on the marginal effects of the pollutant across different expansion cohorts. This concern could be addressed by allowing for heterogeneous effects of pollution across cohorts of Medicaid expansion in models [2.1] and [2.2]. However, I used heterogeneous effects by state because they provide an additional advantage: Medicaid's impacts are identified by the average, across all treated states, of each

state's change in the pollutant's effect. In contrast, in [2.1] and [2.2], the impacts are identified by comparing only two effects: the pollutant's effect in treated states before the expansion and the pollutant's effects in control and treated states after the expansion.

The third robustness test added interaction terms between air pollution and non-medical government transfers. Since other social welfare programs were in place during the 80s and some states may have expanded them, Medicaid's impact could be overestimated. I constructed the transfer variable using county-level deflated annual series from the Bureau of Economic Analysis (BEA). Government transfers for medical care and Supplemental Security Income expenditures were excluded. I used the population of 0-64 years (sourced from SEER) to compute per capita values.

The fourth robustness test added interaction terms between in-utero temperature bins and Medicaid expansion. Since air pollution and temperature are correlated[71] and Medicaid may simultaneously change their effects, any impact of Medicaid on the temperature-health relationship could be captured in the interaction term between Medicaid and pollution. Notice also that models [2.1] and [2.2] assume that the health effects of air pollution and temperature are independent (i.e., pollution and temperature bins do not interact in any model).

The fifth robustness test used the SO_2 balanced panel to estimate models [2.1] and [2.2]. Notice that interaction models rely on the implicit assumption that the pollutant's effects are the same in counties entering the sample (i.e., counties with monitoring data) after the expansion and those

[71] High levels of SO_2 (fourth and fifth quintiles) are positively correlated with low temperatures (T<=45 F) and negatively correlated with high temperatures (T>=65 F).

already in the sample before the expansion. Using this balanced SO_2 panel, the number of counties falls from 533 to 222. This sample consists of the counties whose fraction of missing during a 39-week rolling window was less than 50% every week during 1981-1991.

The following two robustness tests build upon the previous one (i.e., use the SO_2 balanced panel) and add heterogeneous effects of pollution across time. Notice that Medicaid's impact on the marginal effect of pollution could be overestimated if the effects of air pollution decrease over time. Such a trend could be caused by technological change (e.g., houses better insulated from outdoor air pollution) or behavioral changes (e.g., pollution avoidance could have increased). I ran two robustness tests: I added heterogeneous effects of air pollution across years to [2.1] and [2.2]. I also used a less stringent version imposing a linear trend in the effects of air pollution.

The final robustness check consists of using simulated eligibility for Medicaid (SEM_{sy}), a continuous variable, instead of Medicaid's expansion, a binary variable, to analyze how the effect of SO_2 changes with healthcare access. In model [2.3], ε_j and ω_j capture the change in the pollutant's marginal effect when simulated eligibility is below or above each state's median.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{mr} + \alpha_{cQr} + \sum_{j\ 1..5,\ \neq 3} \varepsilon_j\, q_j\left(SO_{2_{cwy}}\right) * 1\left(SEM_{sy} < \widetilde{SEM_s}\right) * \left|SEM_{sy} - \widetilde{SEM_s}\right| +$$

$$\sum_{j\ 1..5,\ \neq 3} \omega_j\, q_j\left(SO_{2_{cwy}}\right) * 1\left(SEM_{sy} \geq \widetilde{SEM_s}\right) * \left|SEM_{sy} - \widetilde{SEM_s}\right| + \sum_{j\ 1..5,\ \neq 3} \beta_j\, q_j\left(SO_{2_{cwy}}\right) + \delta W_{cwy} +$$

$$\theta U^{Q1}{}_{smy} + \gamma X_{cwyr} + u_{cwyr} \quad [2.3]$$

In the previous equation, $\widetilde{SEM_s}$ corresponds to the median simulated eligibility in state s and $1\left(SEM_{sy} < \widetilde{SEM_s}\right)$ is a variable that indicates that simulated eligibility in state s and year y is below its median.

2.6 Results.

Sections 1 and 2 present the impacts of Medicaid's expansion on the effects of SO_2 on fetal death and birth outcomes, respectively. Section 3 presents the robustness tests, and the last section shows the results from estimating heterogeneous effects by race.

2.6.1 Fetal death

Table 2.2 shows the impact of Medicaid's expansion on the SO_2-fetal death damage function. Model [2.1] is estimated across different samples of counties based on the baseline concentration of SO_2. The results in column (2) show that Medicaid's expansion increased the number of infants surviving high prenatal exposure to SO_2 in low-pollution counties. The fertility rates associated with one additional day in the fourth and fifth quintile were 0.031%[72] and 0.072%[73] higher after Medicaid's expansion. Consequently, Medicaid's expansion increased a county's annual number of infants surviving an additional day in SO_2's fourth and fifth quintile by 1.62[74] and 3.73[75], respectively[76]. One important implication of these results is that health care saves more lives at higher pollution concentrations.

[72] 0.031% =(0.106/273)/1.25
[73] 0.072%=(0.244/273)/1.25
[74] 1.62= (0.106/273)*80.38*52.
[75] 3.73 =(0.244/273)*80.38*52.
[76] For comparison, for low-pollution counties, there were 4316 annual births per county (see Table 1.2).

The back-of-the-envelope calculation suggests that Medicaid's expansion reduced a county's annual fetal deaths linked to SO_2 from 90.1[77] to 39.5[78] in the fourth quintile and from 50.7[79] to 12.3[80] in the fifth. Consequently, Medicaid's expansion would have reduced the number of fetal deaths linked to SO_2 by 143,462[81] during 1981-1991 in the US. These pregnancies would otherwise have ended in fetal deaths due to SO_2. Nevertheless, since many of these marginally saved fetuses were negatively selected (results presented in the following section), how many survived their first year is unclear. Understanding the relevance of this number requires analyzing infant mortality as well.

The results from Table 2.2 could be used for a cost-benefit comparison between mitigation (i.e., reducing ambient air pollution levels) vs. adaptation (i.e., expanding healthcare access) policies. For instance, fetal deaths linked to SO_2 decreased by 2062[82] per 100 thousand live births as Medicaid Expanded; considering a 21 percentage point increase in simulated eligibility in treated states during this period, I estimate a decrease of 98 per 100 thousand live births for each percentage point increase in simulated eligibility. In contrast, Currie & Neidell (2005) estimated that each one-unit decline in CO[83] led to 13 fewer infant deaths per 100 thousand live births.

[77] $90.1 = \tau_{br} * 52 * 1.25 * 80.38$ and $\tau_{br} = \left(\frac{0.189}{1.25}\right) * 0.11$. Where 0.189 corresponds to the coefficient SO_2-39w-Sh(q4) on Col 2 of table 2.2. 1.25 corresponds to the average birth rate per week per thousand women of reproductive age (see table 2.2). 0.11 corresponds to the fraction of a 39-week pregnancy spent in the 4th quintile of SO_2 in counties with a low baseline (see col 3 of table 1.7). 80.38 corresponds to the average number of women of reproductive age (see table 2.2). 52 corresponds to the number of weeks in a year.

[78] $39.5 = \tau_{br}^{post} * 52 * 1.25 * 80.38$ and $\tau_{br}^{post} = \left(\frac{0.189-0.106}{1.25}\right) * 0.11$. where 0.106 corresponds to the coefficient Medicaid Exp*SO2-39w-Sh(q4) on col 2 of table 2.

[79] $50.7 = \tau_{br} * 52 * 1.25 * 80.38$ and $\tau_{br} = \left(\frac{0.322}{1.25}\right) * 0.04$. Where 0.322 corresponds to the coefficient SO2-39w-Sh(q4) on Col 2 of table 22. 0.04 corresponds to the fraction of a 39-week pregnancy spent in the 5th quintile of SO_2 in counties with a low baseline (see table col 3 of Table 1.3).

[80] $12.3 = \tau_{br}^{post} * 52 * 1.25 * 80.38$ and $\tau_{br}^{post} = \left(\frac{0.322-0.244}{1.25}\right) * 0.04$. where 0.244 corresponds to the coefficient Medicaid Exp*SO2-39w-Sh(q5) on col 2 of table 2.2.

[81] 143,462 =11*0.81*[(90.1-39.5) +(50.7-12.3)]*181. Where 181 corresponds to the number of counties with a low baseline level of SO_2 in treated states. The expression is multiplied by 11 to account for the full period 1981-1991, and 0.81 corresponds to the average value of the dummy variable for Medicaid expansion to take into account the staggered nature of the treatment.

[82] 2062=100,000*((90.1-39.5) +(50.7-12.3))/4,315. Where 4315 corresponds to the average number of livebirth per county-year for the sample of counties with a low baseline level of SO_2.

[83] Their estimate is based on the effect of post-natal exposure to CO on the infant mortality rate (i.e., Number of newborns died during the first year of live as a fraction of the total number of livebirths) in California

Similarly, Chay & Greenstone (2003a) estimated that each one-unit decline in TSP decreased infant deaths by 4-7[84] per 100 thousand live births.

Thus, the decrease in fetal deaths as a fraction of the live births caused by a one percentage point increase in Medicaid's simulated eligibility was much larger than the decrease in the infant mortality rate caused by a one-unit decline in CO or TSP ambient levels. Previous estimates can be transformed into lives saved per thousand dollars using the average costs of a one-unit decline in the pollutant's ambient levels or a one percentage point increase in Medicaid's simulated eligibility. Nevertheless, applying this cost-benefit analysis would require analyzing the same outcome (e.g., infant death rate) and the same pollutant. This is a possible extension of this paper.

2.6.2 Birth outcomes

Table 2.3 shows the impact of Medicaid's expansion on the SO_2-birth weight damage function. Model [2.2] is estimated using log birthweight as the dependent variable. Column 3 shows that Medicaid's expansion increased birthweight by 2.39% for SO_2 concentrations between 8 and 13 ppb (4^{th} quintile) in highly polluted counties. Since the typical pregnancy is exposed to this concentration 30% of the time (Table 1.7), birthweight increased by 0.7%. The effects are an order of magnitude smaller in low-pollution areas and statistically insignificant. Nevertheless, based on the results from Table 2.2, the estimates for low-pollution counties could be biased toward zero to livebirth bias.

[84] Chay & Greenstone (2003b) estimated a one-unit decrease in TSP led to 5-8 fewer infant deaths per 100 thousand live births.

Results in Tables 2.4 and 2.5 are consistent with the hypothesis that a significant fraction of the infants saved by Medicaid in low-pollution counties were negatively selected (i.e., born with poor neonatal outcomes). Table 2.4 shows that Medicaid increased the fraction of low birthweight infants in low-pollution areas (Col 2). The estimate suggests that Medicaid's expansion was associated with an increase in the marginal effect of the fifth quintile of SO_2 on the low birthweight rate. Figure 2.7 repeats the regressions from Cols 2 and 3 of Table 2.4, changing the birthweight threshold. It shows the same story: As Medicaid expanded, the fraction of infants with low or very low birth weight increased in low-pollution counties. Similarly, Table 2.5 shows that the number of infants born with less than 2500 grams per woman of reproductive age increased in low-pollution counties as Medicaid expanded.

Table 2.6 shows the results from the bounding exercise to address the livebirth bias. This table estimates model [2.2] using log birthweight as the dependent variable over two samples to find upper and lower bounds for Medicaid's impacts on SO_2's effect on birthweight. As discussed in section 2.5.3, this bounding exercise resembles the idea behind Lee (2009)'s method of addressing sample selection. First, I used the results from Table 2.2 to estimate that live births increased on average by 2% in low-pollution areas. Then, I excluded the 2% largest (smallest) observation by birthweight and used the sample to estimate the lower (upper) bounds. The result shows that both bounds are statistically insignificant in low-pollution areas and nationwide.[85] Furthermore, the upper bound can be smaller than the lower bound for some ranges of SO_2. Overall, the results from this table show that traditional bounding exercises are not effective in this context.

[85] The same 2% threshold was used for the national sample.

The livebirth bias in low-pollution counties' estimates was addressed in Table 2.7. This table estimates [2.2] using the number of non-adverse cases per woman of reproductive age as the dependent variable. Results in all columns show that Medicaid's expansion improved neonatal outcomes at the national level and in low-pollution counties. For instance, at the national level, estimates from Column 1 show that the rate of non-low birth weight (i.e., healthy) infants per woman of reproductive age associated with one additional day in the fourth and fifth quintiles increased by 0.027%[86] and 0.028%[87], respectively, after Medicaid's expansion. Consequently, Medicaid's expansion increased a county's annual number of infants born with a weight above 2500 grams by 3.32.[88] For comparison, at the national level, there were 4423 annual births per county (see Table 1.2).

The back of the envelope calculation for Column 1 of Table 2.7 suggests that every year 176[89] additional infants were born non-low birth weight per county due to Medicaid's expansion. Therefore, relative to 4423 annual births per county at the national level, the rate of non-lbw infants per thousand live births increased by 40. Considering the 21 percentage points (pp) increase in Medicaid's simulated eligibility, the non-LBW rate increased by 1.9 per thousand live births for a one pp increase in simulated eligibility.

Figure 2.8 summarizes the impact of Medicaid on the number of infants born with weight above a given threshold per woman of reproductive age. These estimates were obtained by using different

[86] 0.027%=(0.0993/273)/1.363. where 1.363 corresponds to the dependent's variable mean
[87] 0.028%=(0.106/273)/1.363.
[88] 3.325=((0.0993 + 0.106)/273)*80.38*52
[89] $176 = 52 * (0.0993 * 0.21 + 0.106 * 0.18) * 85.03$. where 0.0993 and 0.106 correspond to the coefficients Medicaid Exp*SO2-39w-Sh(q4) and Medicaid Exp*SO_2-39w-Sh(q5) on col 1 of table 2.6. 0.21 and 0.18 correspond to the average fraction of a standard 39-week pregnancy spent in the fourth and fifth quintiles, respectively (see col1 of Table 1.7). 85.03 corresponds to the number of women of reproductive age (see table 2.7). 52 corresponds to the number of weeks in a year.

thresholds to construct the dependent variable. A threshold of 2500 grams is used in Columns 1-3 of Table 2.7. For figure 2.8, I compute the number of infants born with weight above T per woman of reproductive age, where T=1500, 1750 … 3500 grams. The results show that Medicaid's expansion improved infant health, as measured by birthweight. This improvement came via mitigation of the impact of high concentrations of sulfur dioxide (fourth and fifth quintile) on birthweight. Notably, the effect came from high pollution episodes in low-pollution counties and was larger for the highest concentration quintile as shown for the second panel in the figure.

2.6.3 Robustness tests

This section presents the robustness tests in two sub-sections. The first one shows that SO_2's variance has not decreased as Medicaid expanded, a pre-requisite to interpret the interaction terms in [2.1] to [2.3] as the causal impact of Medicaid on the Pollution-health damage functions. The second one implements seven different robustness tests, described in section 2.5.4, to show that the estimates in the baseline empirical strategy are unlikely to be biased.

2.6.3.1 Orthogonality between Medicaid expansion and sulfur dioxide.

In a model like [C1], described in Methodological Appendix C, the interaction term could be confounded if sulfur dioxide and Medicaid expansion were not orthogonal. For instance, the interaction term would be colinear with the non-interacted terms (Medicaid and SO_2) if changes in access to Medicaid are correlated with changes in the concentration of SO_2. In [2.1] – [2.3], this is not a concern because the effects of air pollution are identified from variation within each county-year and Medicaid varies at the state-year level.

However, a decrease in the variance of pollution would be a concern. For example, if the pollutant's variance decreased when Medicaid expands, the interaction term's estimate in [2.1] to [2.3] would suggest that Medicaid lessened the effects of air pollution even though there was no actual decrease in the pollutant's effect. Figures 2.5 and 2.6 show that the variation in high levels of sulfur dioxide (fourth and fifth quintile) has remained stable over time.

2.6.3.2 Robustness tests for Medicaid's impact on the pollutant's effect.

Table 2.8 shows the robustness test for the estimate of Medicaid's impact on SO_2's effects on fetal death, presented in Table 2.2. Seven robustness tests, described in section 2.5.4, were implemented. Col 1 restricts the sample to treated states only; hence, Medicaid's impacts on SO_2 effects are identified by comparing average SO_2 effects in treated states before and after the expansion. Column 2 allows for heterogeneous pollution effects across states; hence, Medicaid's impacts on SO_2 effects are identified by averaging within-state changes of SO_2 effects in treated states. Column 3 interacts SO_2 bins with non-medical per capita government transfers. Col 4 interacts with temperature bins with Medicaid expansion. Column 5 restricts the sample to the SO_2 balanced panel. Column 6 adds heterogeneous pollution effects by year, and Column 7 allows for a linear trend in the effects of pollution. Medicaid's expansion impacts on high SO_2 levels (fourth or fifth quintile) are generally robust; when Medicaid is interacted with in-utero temperature bins (column 4), the point estimate reduces by half and turns statistically insignificant. However, the estimate remains significant when SO_2's fourth and fifth quintiles are combined.[90]

[90] Results not show, available upon request.

Table 2.9 shows the robustness tests for the estimate of Medicaid's impact on SO_2's effects on the number of infants born with non-adverse outcomes, presented in Table 2.7. The same robustness tests used in Table 2.8 were applied. The sample reduces significantly, standard errors increase, and Medicaid's impacts turn insignificant when the balanced sample is used in Cols 5-6. By-year heterogenous SO_2 effects in Col 6 may be too stringent and absorb part of Medicaid's effect. On col 7, however, Medicaid's impact on the fourth quintile reduces to half its original, although not statistically significant due to bigger standard errors.

Table 2.10 shows how SO_2 effects on birth outcomes change with prenatal simulated eligibility for Medicaid. Model [2.3] is estimated using non-LBW infants per woman of reproductive age as the dependent variable. The results show that lower values of simulated eligibility for Medicaid increase the marginal damage of SO_2's highest concentration bin; however, this model also shows a non-symmetric effect. Higher values of simulated Medicaid eligibility— as compared to each state's median—do not mitigate the effects of SO_2. These results are displayed visually in Figure 2.9.

2.6.4 Heterogeneous effects by maternal race.

Table 2.11 shows the impact of Medicaid on the effect of SO_2 on fetal death by race Model [1.1] is estimated separately for blacks and whites in columns 1-2 and model [2.1] in columns 3-4. The results in columns 3 and 4 show that Medicaid's expansion increased the number of births surviving high-pollution shocks for both races. The expansion increased the annual number of black infants surviving an additional day in SO_2's 4^{th} quintile by 0.758[91] and the number of white

[91] 0.758=(0.314/273)*12.667*52. Where 12667 is the average number of black women of reproductive age per county (Table 1.2).

infants surviving an additional day in the 5th quintile by 1.177[92] per county. For comparison, at the national level, there were 802 and 3426 annual births per county from blacks and whites (see Table 1.2).

Table 2.12 shows the impact of Medicaid on the effect of SO_2 on the number of infants born non-low birthweight for white vs black. The results show that Medicaid's expansion increased the number of infants born with non-adverse outcomes (i.e., healthy) for both races, although the effect was stronger for blacks. Figure 2.10 displays th estimates from cols 1-2 for different birthweight thresholds.

The estimate of Medicaid's impact on the effect of the fourth quintile of SO_2 for blacks (column 2) seems odd because the magnitude of the mitigation (0.268) is larger than the original effect before the expansion (-0.202). Thus, by adding both coefficients, we would conclude that SO_2 improves blacks' birth outcomes after Medicaid's expansion. Consequently, we should consider the possibility that the pre-expansion coefficients, β_j in [2.2], for blacks are biased downward due to measurement error or omitted variable bias (e.g., income) and analyze the implications for the estimate of interest, ϕ_j in [2.2].

Let's start with the possibility of measurement error. The pollution measures were built by county and week of conception; therefore, since blacks are a smaller fraction of the population, blacks' exposure may be measured with more measurement error than whites. The larger standard errors for blacks in Tables 2.11 and 2.12 are consistent with this possibility. Nevertheless, as discussed

[92] 1.177=(0.0918/273)*67.291*52. Where 67291 is the average number of white women of reproductive age per county (Table 1.2).

in section 2.5.2, an advantageous property of interaction terms is that if the nature and degree of measurement error did not change with Medicaid's expansion, ϕ can be consistently estimated even though β were biased (Barwick et al., 2019). To explore the relationship between Medicaid expansion and measument error in pollution, I computed the standard deviation of daily SO_2 readings per county and year; then, I found an insignificant estimate of Medicaid expansion when controlling by county and year fixed effects.[93] There is also no correlation with the number of monitors per county-year. It must also be noted that I constructed pollution measures at the county-day level using a fixed set of monitors for each county. Previous analysis are not a perfect proof that measurement error in pollution is not correlated with Medicaid expansion, but there is no better option with the available data for these years.

Alternatively, suppose there remains some residual income variation in the error term of model [2.2] and it is correlated with the pollution level. Suppose that high (low) pollution concentrations are correlated with positive (negative) income shocks in the error term. Thus, in [2.2], β would be biased towards zero and ϕ would capure the join impact of Medicaid on SO_2 and income effects. Since we would expect Medicaid's expansion to reduce the effect of income shocks on birth outcomes, we should expect $\phi_4 \; and \; \phi_5$, the impacts on the fourth and fifth quintile effects, to be biased towards zero. On the contrary, we should expect $\phi_1 \; and \; \phi_2$, the impacts on the first and second quintile, to be biased upwards. Therefore, we should interpret the estimate of Medicaid's impact on the effect of the fourth concentration quintile on black infants' health (Col 2 of Table 2.12) as a lower bound. The same analysis can be applied to Cols 4 and 6.

[93] Results not shown, available upon request

Overall, the results indicate that Medicaid's expansion mitigated the effect of SO_2 on fetal death and birth outcomes for both races, but the effects were larger for blacks.

2.7 Discussion

Addressing the livebirth bias (sample selection) is critical to study how social welfare programs impact the marginal effects of environmental exposures during the gestational period on birth outcomes—the contrast between the estimates in tables 2.5 and 2.7 show it. More generally, the lessons from this paper apply to any case where the objective is studying how an intervention (*T*) changes the marginal effect of *x* on *y,* and $\hat{\beta}_x$ is affected by sample selection. If *T* changes the degree of sample selection, the estimate of the impact of T on β_x would be biased. In this paper, we observe this situation: A naïve interpretation of Table 2.5 would lead us to conclude that Medicaid's expansion increased the marginal damage of air pollution on birth outcomes. However, the conclusion flips once the outcome variable is properly transformed (Table 2.7). This problem is particularly critical in a context where the (ex-ante) theoretical prediction of the impact of *T* on β_x is ambiguous.

The results from this paper highlight that public health policy can be an effective instrument to mitigate the effects of air pollution on health, and it complements environmental policy to improve environmental justice. It is known that homogeneity in the air quality standard across heterogeneous populations results in heterogeneous marginal damages of air pollution on health. Furthermore, the marginal damages of air pollution are larger for the most disadvantaged households. This paper's results suggest that free health care for low-income households mitigates

the effects of prenatal air pollution on infant health; moreover, the impact was larger for blacks than whites. Thus, the program also improved environmental justice.

The discussion in the theoretical framework in section 2.3 makes this paper's limitation evident. The empirical strategy estimates the aggregate effects of the three channels: the medical-biological, the informational, and the optimal response channel. The first two suggest that Medicaid's expansion reduces the effect of air pollution on fetal death and birth outcomes. In contrast, the last one could lead to an increase in the marginal damages of air pollution. The second and third channels rely on the salience of air pollution, which is limited for SO_2 during the 1980s. Thus, the medical-biological channel (e.g., prenatal prescription of vitamins) is believed to drive the decrease in the marginal damage of SO2. Nevertheless, we cannot assert that the other two channels were entirely irrelevant.

There is also an alternative mechanism, not discussed in the theoretical framework, through which Medicaid's expansion could have impacted the marginal effects of air pollution on infant health. Access to Medicaid could have freed some resources that would have otherwise been used for healthcare expenditures (e.g., paying for prenatal controls out of pocket or private healthcare insurance). Thus, accessing Medicaid could be seen as a positive income shock. The empirical relevance of this channel is unclear. However, it could explain part of the decline in the marginal damage of air pollution. Importantly, this channel would not invalidate the conclusion that Medicaid’s expansion decreased the effects of air pollution. However, it raises questions as to what extent such a decrease can be attributed entirely to increased access to prenatal health care.

Finally, Medicaid could reduce prenatal air pollution health effects through compositional changes. For example, If Medicaid incentivized women ex-ante more resilient to pollution (e.g., higher income or educational attainment women) to get pregnant, the effects of ambient air pollution could decrease. Nevertheless, previous research has found no evidence of Medicaid on fertility, and I found no evidence of mothers' demographics changing in response to Medicaid's expansion, which mitigates this concern.

2.8 Conclusion

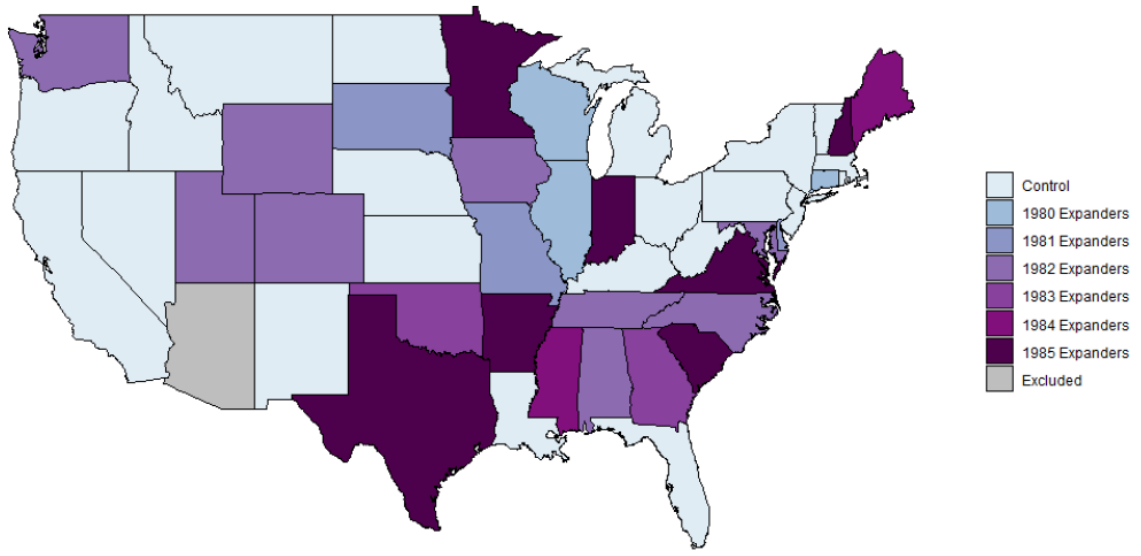
This paper aims to shed light on the effectiveness of health care as a tool to mitigate the impact of prenatal air pollution on infant health. The results suggest that the effect of prenatal air pollution on fetal death and birth outcomes decreased when low-income pregnant women gained access to free prenatal care. The number of live births and infants born with non-adverse neonatal outcomes (i.e., healthy) increased. Such improvements came via reductions in the health impacts of high SO2 concentrations. The prenatal prescription of vitamins and iron could have made the fetus more resilient to pollution shocks. Therefore, policymakers concerned with outdoor air pollution could increase low-income households' access to prenatal health care to lessen the impacts of pollution in-utero. This strategy can be helpful when governments do not have the institutional capacity to regulate pollution, especially if households' ability to avoid pollution is limited.

The analysis by race revealed that free health insurance for low-income households contributed to closing the gap in the effects of air pollution on infant health between blacks and whites. Therefore, increasing healthcare access for low-income households improved environmental justice.

The results of this paper also contribute to the debate on why developing countries have higher marginal damages of air pollution on infant health: non-linear effects or healthcare access? The results from this chapter and the previous one suggest that both factors contribute to the larger marginal damages. The previous chapter demonstrates that the marginal damages increase as the pollutant concentration rises. This chapter reveals that the marginal damages decrease when healthcare access improves. Moreover, the impact of prenatal healthcare is more significant at higher concentrations when the underlying health impacts are substantial.

2.9 Figures

Figure 2.1: Medicaid expansion during the 80s, treated vs. control states.

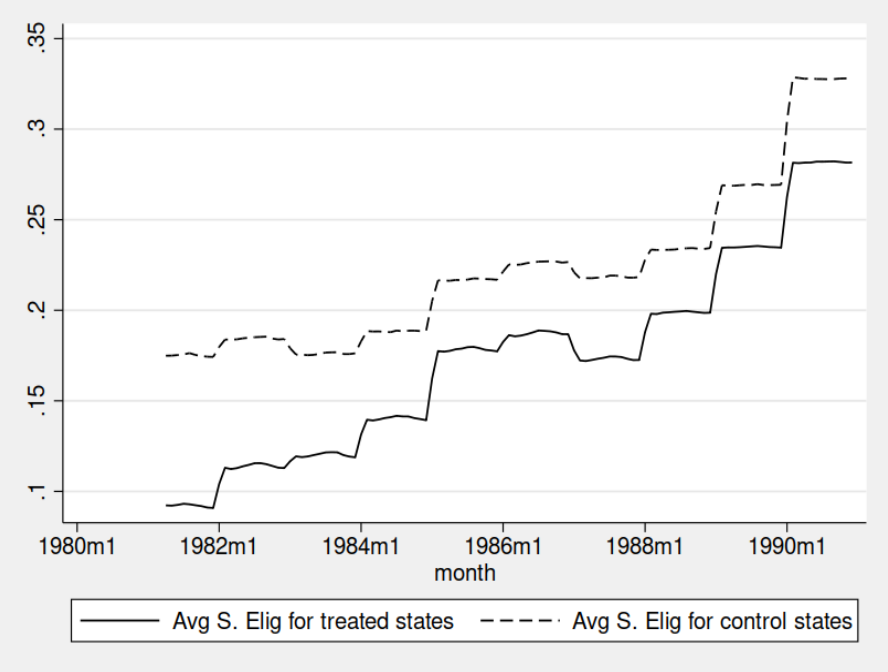


Source: East, Miller, Page & Wherry (2023). Alaska (1982 Expander) and Hawaii (Control) are not pictured. Arizona is omitted due to the late start date of their Medicaid program (1988).

Figure 2.2: Medicaid expansion (Binary variable)

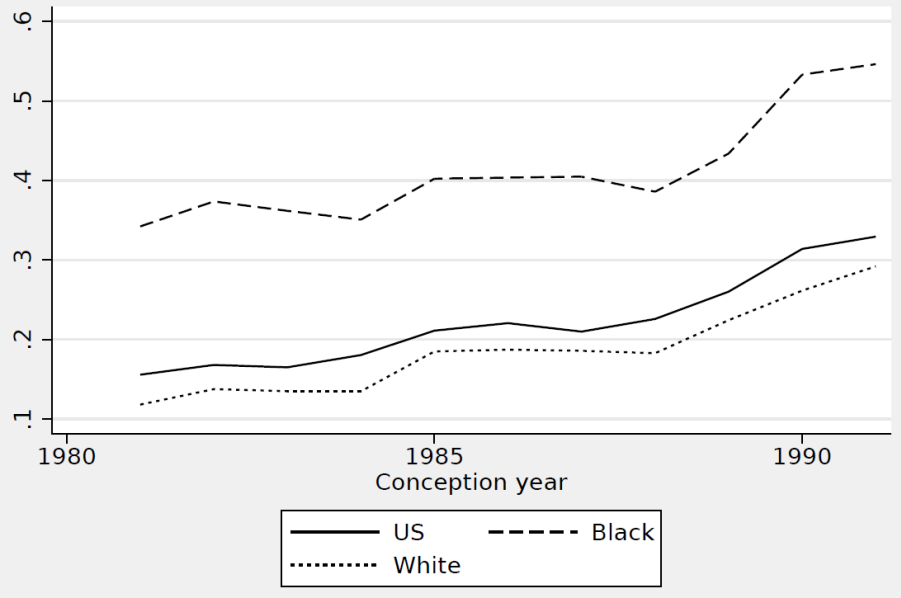
Note: Author's calculations. This graph shows the share of conceptions occurring in states that have expanded Medicaid

Figure 2.3: Prenatal simulated eligibility for Medicaid (continuous) for treated vs control states.



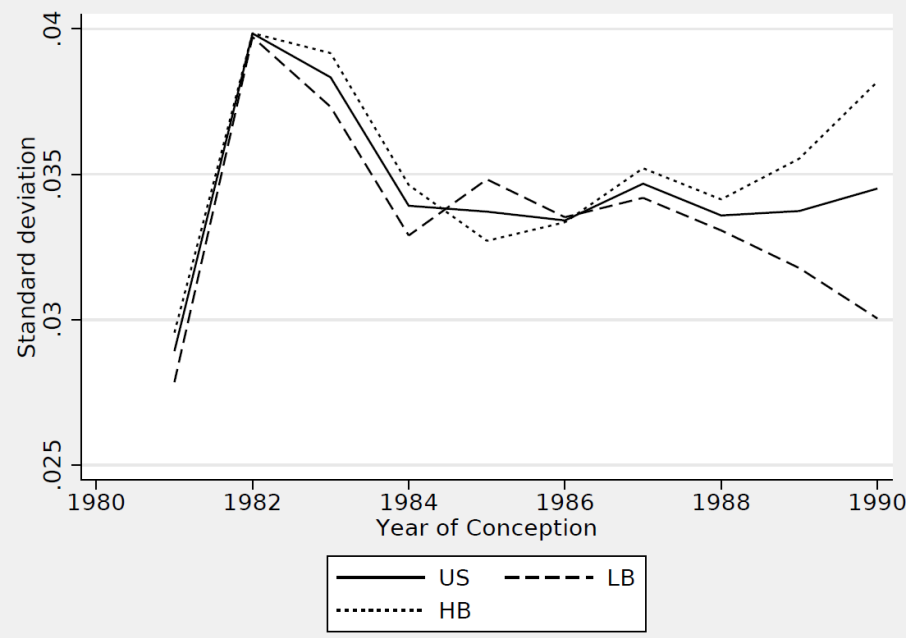
Note: Author's calculations. This graph shows the average values of simulated eligibility for Medicaid as weighted by the number of conceptions per month and state.

Figure 2.4: Prenatal simulated eligibility for Medicaid (Continuous) by Race.



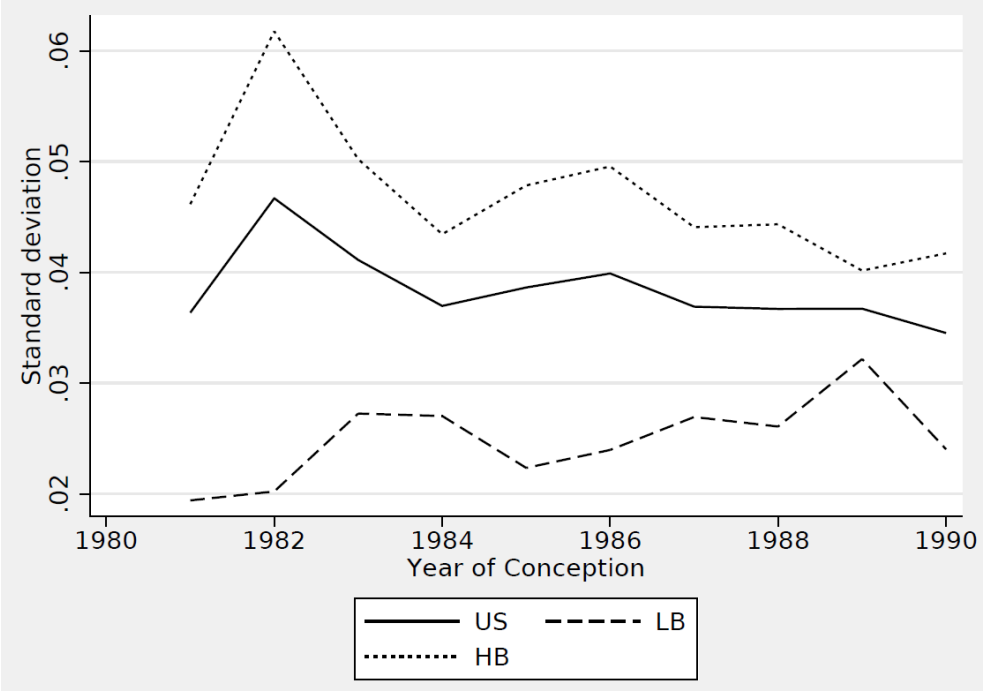
Note: Author's calculations. This graph shows the average values of simulated eligibility for Medicaid dissagregated by race. I weighted by the number of conceptions per month, state, and race.

Figure 2.5: Standard deviation of the remaining variation in the fourth quintile of SO_2 across years



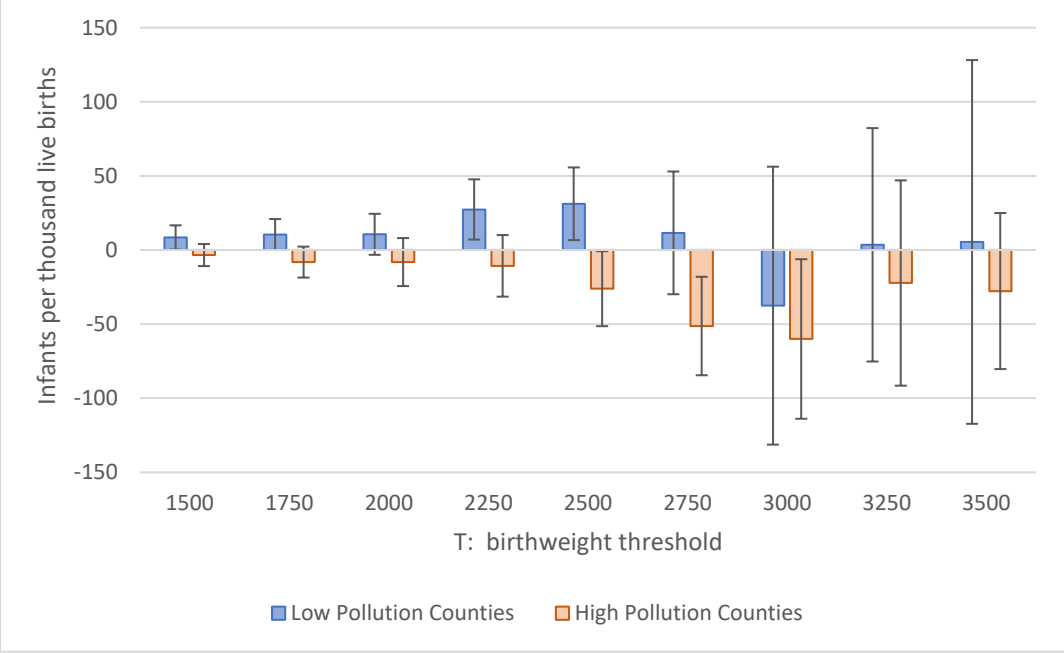
Note: Author's calculations. US: Counties with SO_2 data. LB=low baseline. HB=high baseline. This graph corresponds to the standard deviation of the residuals from regressing q_4 (SO_2) (i.e., fraction of a standard 39-week pregnancy spent in the fourth quintile of the national distribution) on county-year, county-quarter, and month FE.

Figure 2.6: Distribution of remaining variation in the fifth quintile of SO_2 across years.



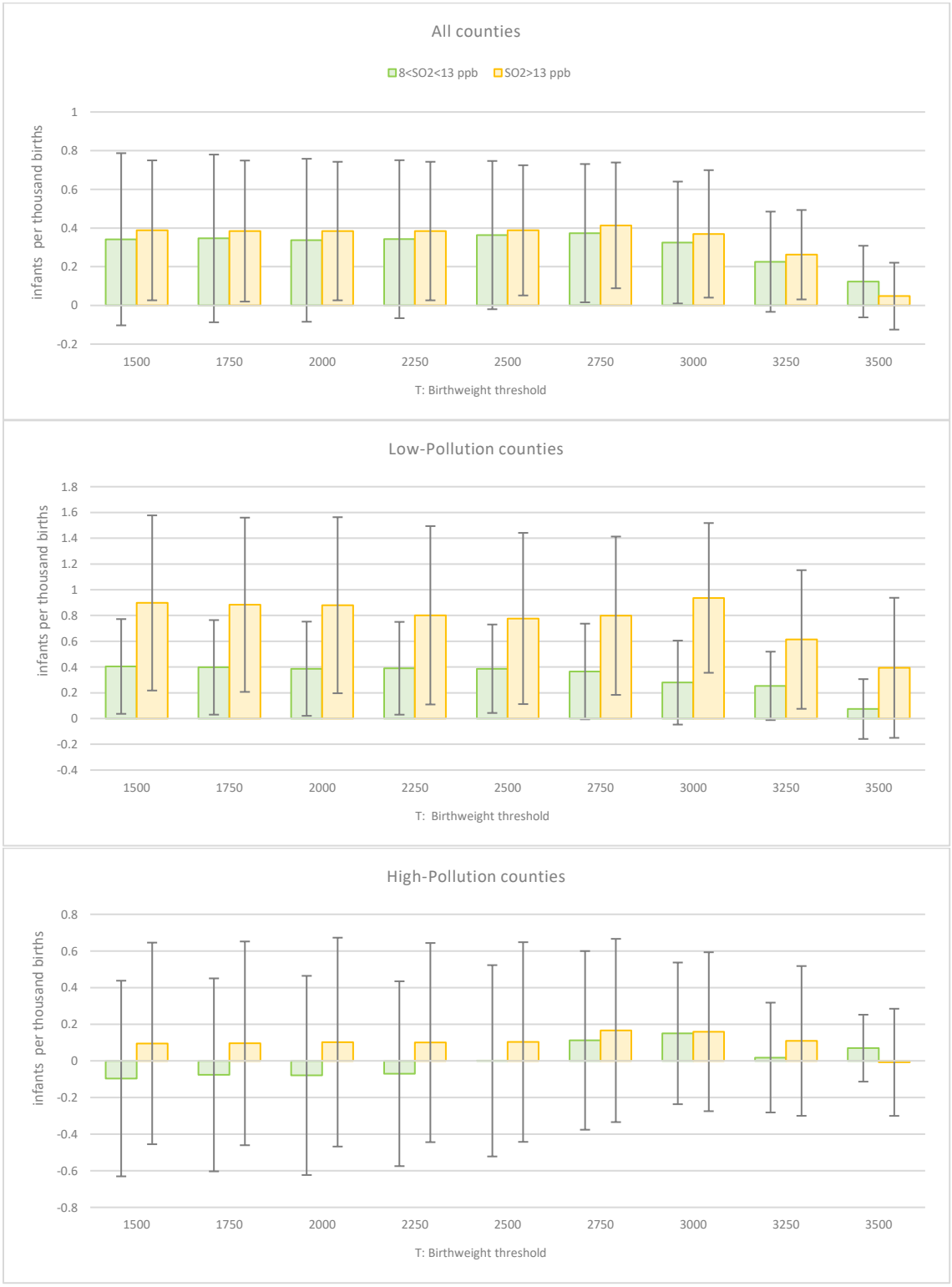
Note: Author's calculations. US: Counties with SO2 data. LB=low baseline. HB=high baseline. This graph corresponds to the standard deviation of the residuals from regressing q_5 (SO_2) (i.e., fraction of a standard 39-week pregnancy spent in the fifth quintile of the national distribution) on county-year, county-quarter, and month FE.

Figure 2.7: Medicaid's expansion impacts on the fraction of infants born with weight below threshold T.



Note: Author's calculations. Each bar in this figure corresponds to a different regression of model [2.2] changing the birthweight threshold to build the dependent variable $\frac{\sum_i 1(births<T)}{N\ births}$ The error bars correspond to the 95% confidence interval. The blue bars plot the impact of Medicaid on the fourth quintile of SO_2, Medicaid Exp*SO_2-39w-Sh(q4), in Column 2 of table 2.5 (low-pollution counties) and the orange bars plot the impact of Medicaid on the fifth quintile of SO_2, Medicaid Exp*SO_2-39w-Sh(q5), in Column 3 of table 2.5 (high-pollution counties).

Figure 2.8: Effects of Medicaid's expansion on the number of infants born with weight above threshold T.

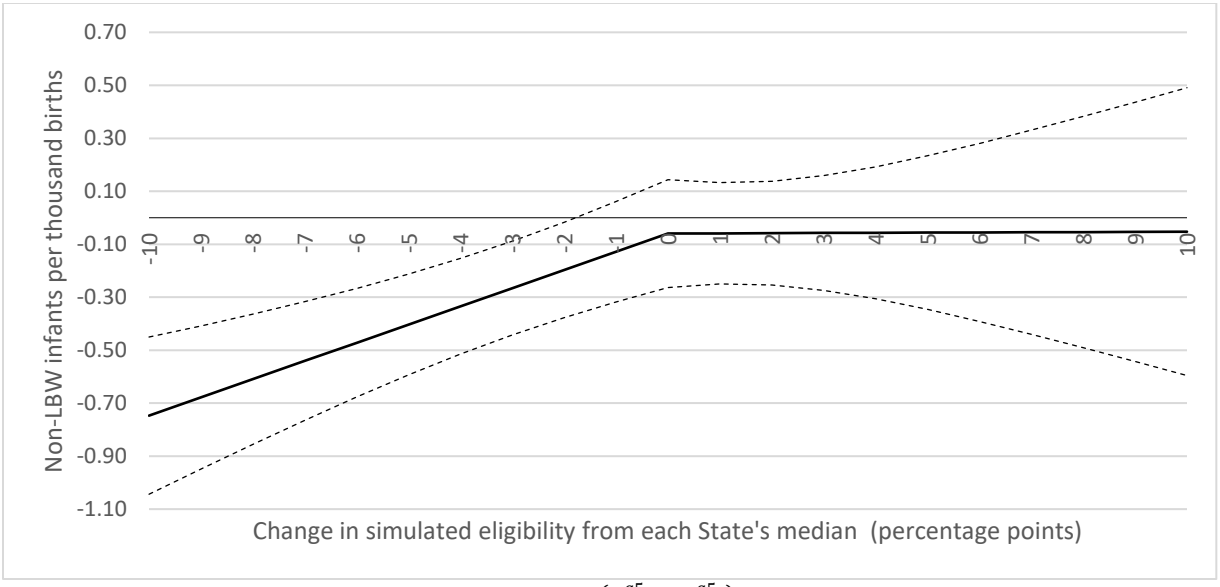


Note: Author's calculations. Each figure corresponds to a different sample (group of counties: all, low-pollution, high-pollution). Each pair of green and yellow bars correspond to a different regression. Each regression corresponds to [2.2] using the number of infants born with weight above threshold T (T=1500.. 3500) per woman of reproductive age. The green and yellow bars plot the impact of Medicaid on the effects of the fourth and fifth quintile of SO_2, Medicaid Exp*SO_2-39w-Sh(q4) and Medicaid Exp*SO_2-39w-Sh(q5) in Table 2.7. Notice that Table 2.6 only reports the estimates for a threshold of 2500 grams.

The height of the bars was computed as $h_T^{gj} = 1000 * (\frac{1}{273}) \left(\frac{\hat{\beta}_T^{gj} * Women_{15-44}^{g}}{(Nbirths^{g})} \right) * 52.$

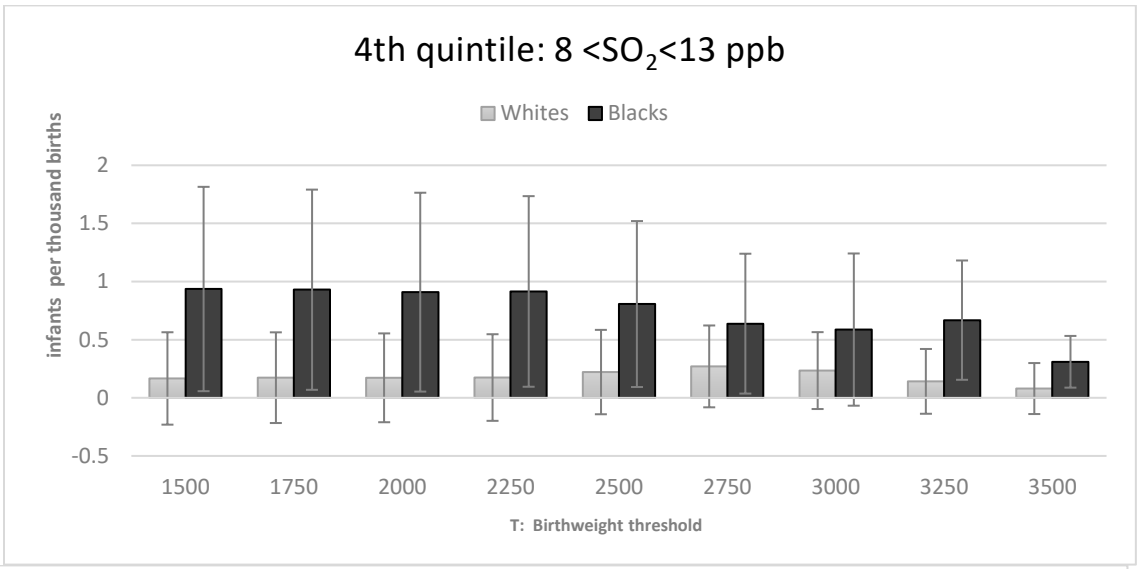
Where T indexes the birthweight threshold, g indexes the group of counties (all, low-pollution, high-pollution), and j indexes the concentration bin (4th quintile (8<SO_2<13), or 5th (SO_2>13 ppb)). $\hat{\beta}_T^{gj}$ corresponds to the coefficient of Medicaid Exp*SO_2-39w-Sh(q^j). The term (1/273) indicates that the impacts are estimated for 1 additional day in the j^{th} quintile as compared to the third one (omitted category). $Women_{15-44}^{g}$ and $Nbirths^{g}$ corresponds to the average number of women of reproductive age and annual births in group county g. Since the model is estimated using weekly data, I multiply by 52 to estimate the average number of births per year. The error bars correspond to the 95% confidence interval.

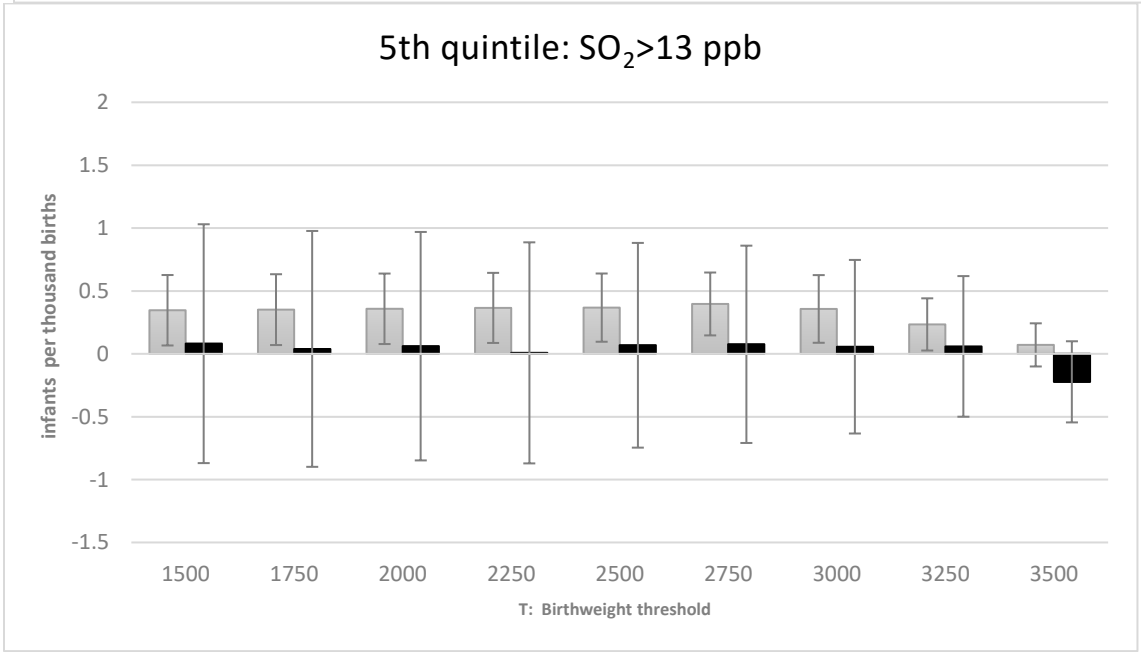
Figure 2.9: Marginal effect of one additional day in the highest concentration quintile of SO_2 as a function of simulated eligibility for Medicaid.



Notes:Author's calculations. This graph plots two estimates: $a\left(\beta_{SO2}^{q5} + \gamma_{SO2}^{q5}\right) * |x|$ when simulated eligibility is below the median (i.e, negative values on the x-axis) and $a\left(\beta_{SO2}^{q5} + \theta_{SO2}^{q5}\right) * (x)$ when simulated eligibility is above the median (i.e, positive values on the x-axis). Where β_{SO2}^{q5}, γ_{SO2}^{q5}, and θ_{SO2}^{q5} correspond to the coefficients SO_2-39w-Sh(q5), I(SEM<$\widehat{SEM_s}$)*|SEM-$\widehat{SEM_s}$|*SO_2-39w-Sh(q5), and I(SEM>$\widehat{SEM_s}$)*|SEM-$\widehat{SEM_s}$|*SO_2-39w-Sh(q5) in Col 1 of Table 2.10. The term a is a factor to transform the units into infants per thousand births, $a = 1000 * (\frac{1}{273}) \left(\frac{Women_{15-44}^{g}}{\left(\frac{Nbirths^{g}}{52}\right)} \right)$.

Figure 2.10: Effects of Medicaid's expansion on the number of infants born with weight above threshold T by race.





Notes:Author's calculations. The gray and black bars are estimated by running model [2.2] separately by birthweight threshold (T=1500.. 3500) and race (black and white). The error bars correspond to the 95% confidence interval.

The height of the bars was computed as $h_T^{rj} = 1000 * \left(\frac{1}{273}\right)\left(\frac{\hat{\beta}_T^{rj} * Women_{15-44}^{r}}{(Nbirths^{r})}\right) * 52.$

Where T indexes the birthweight threshold, r indexes the race (black, white), and j indexes the concentration bin (fourth (8<SO_2<13) , or fifth (SO_2>13 ppb)). $\hat{\beta}_T^{rj}$ corresponds to the coefficient of Medicaid Exp*SO_2-39w-Sh(q^j) in Columns 1 and 2 of Table 2.12. The term (1/273) indicates that the impacts are estimated for 1 additional day in the j-th quintile as compared to the third one (omitted category). $Women_{15-44}^{r}$ and $Nbirths^{r}$ corresponds to the average number of women of reproductive age and annual births of race r. Since the model is estimated using weekly data, I multiply by 52 to estimate the average number of births per year.

2.10 Tables

Table 2.1: Number of states and counties across cohorts of year of Medicaid expansion.

	States	All Counties	SO_2 UNB	SO_2 LB	SO_2 HB	SO_2 BLN
Control	22	1161	238	112	126	115
1980	3	182	45	26	19	21
1981	3	184	15	6	9	8
1982	11	563	93	66	27	34
1983	2	236	19	17	2	2
1984	2	98	18	12	6	5
1985	7	700	91	52	39	35
1988	1	14	7	3	4	2

Notes: Author's calculations. UNB (Unbalanced SO_2 panel), LB (Low baseline), HB (High baseline), BLN (SO_2 Balanced panel)

Table 2.2: Medicaid's expansion impact on the effect of SO_2's on fetal death.

	(1)	(2)	(3)
Baseline SO_2	All	Low	High
Mean (FR)	1.177	1.250	1.117
Women 15-44 (Thousands)	85.03	80.38	89.30
Cells	180,217	86,550	93,667
R-squared	0.660	0.638	0.633
Medicaid Exp*SO_2-39w-Sh(q1)	0.00266	0.0310	-0.0585
	(0.0529)	(0.0518)	(0.105)
Medicaid Exp*SO_2-39w-Sh(q2)	0.0568	0.0803	-0.0138
	(0.0756)	(0.0864)	(0.184)
Medicaid Exp*SO_2-39w-Sh(q4)	0.0675	0.106**	-0.0209
	(0.0533)	(0.0421)	(0.0744)
Medicaid Exp*SO_2-39w-Sh(q5)	0.0405	0.244*	-0.0111
	(0.0540)	(0.123)	(0.0964)
SO_2-39w-Sh(q1)	-0.0316	-0.0785	0.0213
	(0.0565)	(0.0634)	(0.108)
SO_2-39w-Sh(q2)	-0.0780	-0.135*	0.0451
	(0.0617)	(0.0719)	(0.192)
SO_2-39w-Sh(q4)	-0.112**	-0.189***	0.00798
	(0.0532)	(0.0386)	(0.0850)
SO_2-39w-Sh(q5)	-0.0805	-0.322**	-0.00660
	(0.0564)	(0.142)	(0.103)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is FR (fertility rate) = 1000*(N live birth/ N women 15-44) by county and week-year of conception cells. Low (High) corresponds to counties whose average SO_2 concentration during the first two years in sample is below (above) the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year FE, county-Quarter FE, and month FE. Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy. Regressions were weighted by the number of women of reproductive age in each county-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.3: Medicaid's expansion impact on the effect of SO_2 on birthweight.

	(1)	(2)	(3)
	Log (BW)	Log (BW)	Log (BW)
Baseline SO_2	All	Low	High
Observations	17,506,986	8,383,192	9,123,794
R-squared	0.725	0.663	0.758
Medicaid Exp*SO_2-39w-Sh(q1)	0.00515	0.000421	0.0148*
	(0.00797)	(0.00719)	(0.00859)
Medicaid Exp*SO_2-39w-Sh(q2)	0.00500	0.000734	0.0107
	(0.00865)	(0.00968)	(0.0190)
Medicaid Exp*SO_2-39w-Sh(q4)	0.0108	0.00374	0.0239**
	(0.00699)	(0.00346)	(0.0105)
Medicaid Exp*SO_2-39w-Sh(q5)	0.00279	-0.00626	0.00648
	(0.00689)	(0.0149)	(0.0101)
SO_2-39w-Sh(q1)	-0.00435	0.000829	-0.0143
	(0.00779)	(0.00734)	(0.00875)
SO_2-39w-Sh(q2)	-0.00319	0.00209	-0.00984
	(0.00819)	(0.00822)	(0.0190)
SO_2-39w-Sh(q4)	-0.00414	0.00613**	-0.0183*
	(0.00691)	(0.00273)	(0.0101)
SO_2-39w-Sh(q5)	-0.000657	0.0105	-0.00566
	(0.00725)	(0.0153)	(0.0100)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is the natural logarithm of birthweight. Low (High) corresponds to counties whose average SO_2 concentration during the first two years in sample is below (above) the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Regressions were weighted by the number of births in each cell. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.4: Medicaid's expansion impact on the effect of SO_2 on the fraction of adverse birth outcomes

	(1) LBW_r	(2) LBW_r	(3) LBW_r	(4) SGA_r	(5) SGA_r	(6) PTB_r	(7) PTB_r
Baseline SO_2	All	Low	High	All	Low	All	Low
Mean (Y)	51.72	47.43	55.62	90.64	88.52	94.56	88.52
Observations	17,506,986	8,383,192	9,123,794	17,506,986	8,383,192	17,506,986	8,383,192
R-squared	0.452	0.358	0.507	0.146	0.117	0.526	0.451
Medicaid Exp*SO_2-39w-Sh(q1)	-9.450	-4.540	0.653	-9.434	-12.57	-10.31	1.714
	(8.559)	(6.175)	(12.80)	(10.51)	(10.49)	(8.497)	(9.251)
Medicaid Exp*SO_2-39w-Sh(q2)	-14.69	-11.36	-10.61	-21.02*	-21.71	-17.25	-6.401
	(11.66)	(12.75)	(18.27)	(10.89)	(12.93)	(10.41)	(8.472)
Medicaid Exp*SO_2-39w-Sh(q4)	-12.17	-3.035	-26.08**	-17.72	-13.47*	-15.16	1.261
	(8.997)	(5.316)	(12.63)	(10.95)	(7.996)	(11.56)	(10.25)
Medicaid Exp*SO_2-39w-Sh(q5)	-5.640	31.42**	-6.419	-21.91*	-36.68	-5.325	39.57
	(8.569)	(12.53)	(11.51)	(12.16)	(28.23)	(8.705)	(29.64)
SO_2-39w-Sh(q1)	8.108	2.622	0.00935	7.377	8.129	9.993	0.110
	(8.258)	(5.542)	(12.74)	(8.854)	(9.332)	(7.972)	(7.765)
SO_2-39w-Sh(q2)	11.91	6.809	11.21	13.86	15.41	14.09	0.942
	(11.35)	(11.79)	(18.15)	(10.78)	(12.28)	(9.321)	(8.726)
SO_2-39w-Sh(q4)	6.696	-3.632	21.71*	10.33	5.658	11.67	-3.500
	(8.712)	(4.727)	(12.35)	(10.40)	(5.961)	(9.782)	(5.344)
SO_2-39w-Sh(q5)	1.542	-38.42***	4.786	16.64	30.07	-1.240	-43.69

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. LBW: Low birthweight (birthweight<=2500g). SGA: Small for gestational age (birthweight < P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race. PTB: preterm birth (gestational age<37 weeks). All outcome variables correspond to the number of adverse cases per thousand livebirths. Low (High) corresponds to counties whose average SO_2 concentration during the first two years in sample is below (above) the national median. SO_2-39w-Sh (q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Regressions were weighted by the number of births in each cell. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.5: Medicaid's expansion impact on the effect of SO_2 on the incidence of adverse birth outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
	Lbw/w	Lbw/w	Sga/w	Sga/w	Ptb/w	Ptb/w
Baseline SO_2	All	Low	All	Low	All	Low
Mean (Y)	0.075	0.074	0.135	0.136	0.102	0.102
Women 15-44 (1k)	85.03	80.38	85.03	80.38	85.03	80.38
Cells	343,023	160,124	343,023	160,124	343,023	160,124
R-squared	0.525	0.430	0.297	0.259	0.608	0.54
Medicaid Exp*SO_2-39w-Sh(q1)	-0.00824	-0.00176	-0.00478	-0.00726	-0.00838	0.00687
	(0.0133)	(0.00948)	(0.0157)	(0.0154)	(0.0130)	(0.0121)
Medicaid Exp*SO_2-39w-Sh(q2)	-0.0121	-0.00987	-0.0149	-0.0159	-0.0140	-0.00338
	(0.0176)	(0.0199)	(0.0160)	(0.0195)	(0.0183)	(0.0176)
Medicaid Exp*SO_2-39w-Sh(q4)	-0.00834	0.00403	-0.0107	-0.00294	-0.0108	0.00989
	(0.0143)	(0.00785)	(0.0165)	(0.0105)	(0.0171)	(0.0112)
Medicaid Exp*SO_2-39w-Sh(q5)	-0.00181	0.0462***	-0.0168	-0.0251	0.00182	0.0663
	(0.0109)	(0.0157)	(0.0143)	(0.0368)	(0.0102)	(0.0398)
SO_2-39w-Sh(q1)	0.00605	-0.00319	0.000807	-0.00226	0.00610	-0.00966
	(0.0134)	(0.00947)	(0.0148)	(0.0161)	(0.0132)	(0.0118)
SO_2-39w-Sh(q2)	0.00906	0.000595	0.00414	0.00165	0.00983	-0.00956
	(0.0168)	(0.0176)	(0.0177)	(0.0211)	(0.0146)	(0.0135)
SO_2-39w-Sh(q4)	0.00130	-0.0164**	-0.00143	-0.0140	0.00421	-0.0210**
	(0.0143)	(0.00693)	(0.0171)	(0.00986)	(0.0162)	(0.00784)
SO_2-39w-Sh(q5)	-0.00445	-0.0595***	0.00684	0.00982	-0.0127	-0.0801*
	(0.0113)	(0.0168)	(0.0155)	(0.0379)	(0.00994)	(0.0400)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. LBW: Low birthweight (birthweight<=2500g). SGA: Small for gestational age (birthweight < P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race. PTB: preterm birth (gestational age<37 weeks). All outcome variables correspond to the number of adverse cases per thousand woman of reproductive age (15-44) of the respective race. "Low" sample corresponds to counties whose average SO_2 concentration during the first two years in sample is below the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). . Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Newborn-level observations collapsed into cells by county-week-year-race. All regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.6: Bounding exercise for Medicaid's expansion impacts on the effect of SO_2 on birthweight.

	(1) Log (BW)	(2) Log (BW)	(3) Log (BW)	(4) Log (BW)
Baseline SO_2	All	All	Low	Low
Bound	Lower	Upper	Lower	Upper
Observations	17,505,957	17,506,647	8,382,696	8,383,043
R-squared	0.735	0.673	0.146	0.704
Medicaid Exp*SO_2-39w-Sh(q1)	0.00577	0.00560	0.00106	0.000354
	(0.00802)	(0.00788)	(0.00708)	(0.00687)
Medicaid Exp*SO_2-39w-Sh(q2)	0.00511	0.00643	0.000288	9.85e-05
	(0.00910)	(0.00818)	(0.0102)	(0.00833)
Medicaid Exp*SO_2-39w-Sh(q4)	0.0108	0.00790	0.00379	-0.000771
	(0.00699)	(0.00747)	(0.00373)	(0.00324)
Medicaid Exp*SO_2-39w-Sh(q5)	0.00178	0.00272	-0.00970	-0.00657
	(0.00696)	(0.00675)	(0.0153)	(0.0149)
SO_2-39w-Sh(q1)	-0.00479	-0.00423	0.000437	0.000958
	(0.00793)	(0.00755)	(0.00738)	(0.00687)
SO_2-39w-Sh(q2)	-0.00320	-0.00459	0.00233	0.000994
	(0.00861)	(0.00804)	(0.00856)	(0.00779)
SO_2-39w-Sh(q4)	-0.00436	-0.00352	0.00535*	0.00737***
	(0.00686)	(0.00756)	(0.00271)	(0.00254)
SO_2-39w-Sh(q5)	0.000332	-0.00107	0.0141	0.00816
	(0.00743)	(0.00690)	(0.0155)	(0.0149)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is the natural logarithm of birthweight. "Low" sample corresponds to counties whose average SO_2 concentration during the first two years in sample is below the national median. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Regressions were weighted by the number of births in each cell. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.7: Medicaid's expansion impact on the effect of SO_2 on the incidence of non-adverse birth outcomes

	(1) Nlbw/w	(2) Nlbw/w	(3) Nlbw/w	(4) Nsga/w	(5) Nsga/w	(6) Nptb/w	(7) Nptb/w
SO2 Baseline	All	Low	High	All	Low	All	Low
Mean(Y)	1.363	1.386	1.342	1.303	1.325	1.336	1.359
Women 15-44 yr. old	85.03	80.38	89.3	85.03	80.38	85.03	80.38
Cells	343,023	160,124	182,899	343,023	160,124	343,023	160,124
R-squared	0.652	0.643	0.622	0.660	0.645	0.627	0.619
Medicaid Exp*SO_2-39w-Sh(q1)	0.0515	0.0697	-0.0526	0.0480	0.0752	0.0516	0.0611
	(0.0483)	(0.0482)	(0.0835)	(0.0509)	(0.0473)	(0.0511)	(0.0501)
Medicaid Exp*SO_2-39w-Sh(q2)	0.120	0.139	-0.0583	0.122	0.145	0.121	0.132
	(0.0934)	(0.104)	(0.170)	(0.0980)	(0.108)	(0.0917)	(0.103)
Medicaid Exp*SO_2-39w-Sh(q4)	0.0993*	0.109**	3.97e-05	0.102*	0.116**	0.102*	0.103*
	(0.0534)	(0.0494)	(0.0709)	(0.0567)	(0.0514)	(0.0551)	(0.0545)
Medicaid Exp*SO_2-39w-Sh(q5)	0.106**	0.219**	0.0273	0.121**	0.290***	0.103**	0.199**
	(0.0469)	(0.0956)	(0.0740)	(0.0485)	(0.0926)	(0.0500)	(0.0860)
SO_2-39w-Sh(q1)	-0.0773	-0.115**	0.0218	-0.0720	-0.115**	-0.0773	-0.108**
	(0.0498)	(0.0500)	(0.0840)	(0.0506)	(0.0440)	(0.0530)	(0.0516)
SO_2-39w-Sh(q2)	-0.139	-0.193**	0.0888	-0.134	-0.194**	-0.139	-0.183*
	(0.0866)	(0.0945)	(0.177)	(0.0869)	(0.0905)	(0.0883)	(0.0968)
SO_2-39w-Sh(q4)	-0.135***	-0.187***	-0.000405	-0.133***	-0.190***	-0.138***	-0.183***
	(0.0454)	(0.0295)	(0.0760)	(0.0462)	(0.0279)	(0.0463)	(0.0302)
SO_2-39w-Sh(q5)	-0.148***	-0.286**	-0.0493	-0.159***	-0.355***	-0.139***	-0.265***
	(0.0440)	(0.111)	(0.0805)	(0.0429)	(0.107)	(0.0483)	(0.0925)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Nlbw/w , Nsga/w, and Nptb/w correspond to the number of non-low birthweight (birthweight>2500g), non-small for gestational age (birthweight > P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race, and non-preterm birth (gestational age>=37 weeks) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Low (High) Baseline SO2 corresponds to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). newborn-level observations collapsed into cells by county-week-year-race. All regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.8: Robustness tests for Medicaid's expansion impact on the effect of SO_2 on fetal death.

	(1) FR	(2) FR	(3) FR	(4) FR	(5) FR	(6) FR	(7) FR
SO2 Balanced panel	No	No	No	No	Yes	Yes	Yes
States	Treated	All	All	All	All	All	All
Het effects SO2 q4-q5-by-states	No	Yes	No	No	No	No	No
Interact SO2-Goverm Transfers	No	No	Yes	No	No	No	No
Interact Temperature bins* ME	No	No	No	Yes	No	No	No
Het effects SO2-q4-q5-by-year	No	No	No	No	No	Yes	No
Trends in SO2	No	No	No	No	No	No	Yes
Cells	49,877	86,550	85,940	86,550	42,108	42,108	42,108
R-squared	0.540	0.638	0.637	0.638	0.666	0.667	0.666
Medicaid Exp*SO_2-39w-Sh(q1)	0.0544	0.0565	-0.00623	0.0217	0.0640	0.0686	0.0699
	(0.0689)	(0.0486)	(0.0640)	(0.0378)	(0.104)	(0.118)	(0.120)
Medicaid Exp*SO_2-39w-Sh(q2)	0.0742	0.0981	0.108	0.106	-0.0123	-0.0377	-0.0219
	(0.134)	(0.0872)	(0.119)	(0.0814)	(0.178)	(0.216)	(0.210)
Medicaid Exp*SO_2-39w-Sh(q4)	0.163**	0.239***	0.0719	0.0418	0.205**	-0.0500	0.124
	(0.0602)	(0.0428)	(0.0864)	(0.0475)	(0.0765)	(0.157)	(0.138)
Medicaid Exp*SO_2-39w-Sh(q5)	0.254**	0.230	0.258**	0.0678	0.449**	0.464*	0.332*
	(0.101)	(0.149)	(0.116)	(0.0962)	(0.180)	(0.239)	(0.171)
SO_2-39w-Sh(q1)	-0.0753	-0.0975*	-0.0900	-0.0639	-0.161	-0.161	-0.170
	(0.0605)	(0.0561)	(0.0749)	(0.0388)	(0.128)	(0.134)	(0.123)
SO_2-39w-Sh(q2)	-0.124	-0.153**	-0.0904	-0.156***	-0.132	-0.115	-0.165
	(0.0743)	(0.0667)	(0.0904)	(0.0516)	(0.200)	(0.220)	(0.199)
SO_2-39w-Sh(q4)	-0.170***		-0.194***	-0.126***	-0.365***		-0.458***
	(0.0420)		(0.0635)	(0.0328)	(0.0798)		(0.107)
SO_2-39w-Sh(q5)	-0.270**		-0.320*	-0.153*	-0.527**		-0.566***
	(0.122)		(0.165)	(0.0873)	(0.196)		(0.193)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. FR (fertility rate) = 1000*(N live birth/ N women 15-44) by county and week-year of conception cells. The balanced panel corresponds to counties for which the fraction of missing in SO_2 was less than 50% for every week-year during 1981-1991. SO2-39w-Sh (q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year FE, county-Quarter FE, and month FE. Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy. Government transfers correspond to the county's transfer payments excluding medical care and supplemental security income divided by the population of 64yrs old or younger. For model (7), separate trends were allowed for each of the quintiles of SO_2 (q1,q2,q4,q5). Regressions were weighted by the number of women of reproductive age in each county-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.9: Robustness tests for Medicaid's expansion impact on the effect of SO_2 on the number of non-low birthweight infants.

	(1) Nlbw/w	(2) Nlbw/w	(3) Nlbw/w	(4) Nlbw/w	(5) Nlbw/w	(6) Nlbw/w	(7) Nlbw/w
SO2-Balanced panel	No	No	No	No	Yes	Yes	Yes
States	Treated	All	All	All	All	All	All
Het effects SO2 q4-q5-by-states	No	Yes	No	No	No	No	No
Interact SO2-Goverm Transfers	No	No	Yes	No	No	No	No
Interact Temperature bins* ME	No	No	No	Yes	No	No	No
Het effects SO2-q4-q5-by-year	No	No	No	No	No	Yes	No
Trends in SO2	No	No	No	No	No	No	Yes
Cells	100,125	343,023	340,775	343,023	233,366	233,366	233,366
R-squared	0.653	0.652	0.651	0.652	0.677	0.677	0.677
Medicaid Exp*SO_2-39w-Sh(q1)	0.0854	0.0488	-0.00797	0.0666**	0.00231	0.0135	-0.00232
	(0.0914)	(0.0587)	(0.0510)	(0.0287)	(0.0888)	(0.0905)	(0.0927)
Medicaid Exp*SO_2-39w-Sh(q2)	0.00797	0.129	0.100	0.170**	-0.0188	-0.0134	-0.0111
	(0.150)	(0.0901)	(0.103)	(0.0756)	(0.164)	(0.177)	(0.180)
Medicaid Exp*SO_2-39w-Sh(q4)	0.140*	0.167**	0.0575	0.0819*	0.106	-0.00930	0.0467
	(0.0789)	(0.0696)	(0.0529)	(0.0485)	(0.101)	(0.129)	(0.122)
Medicaid Exp*SO_2-39w-Sh(q5)	0.139**	0.0801	0.0963*	-0.00712	0.0906	-0.0118	0.0214
	(0.0504)	(0.0582)	(0.0551)	(0.0449)	(0.0593)	(0.0678)	(0.0734)
SO_2-39w-Sh(q1)	-0.0322	-0.0681	-0.121**	-0.0893***	-0.0474	-0.0554	-0.0549
	(0.0895)	(0.0597)	(0.0512)	(0.0222)	(0.0943)	(0.0951)	(0.0928)
SO_2-39w-Sh(q2)	-0.0192	-0.142	-0.168*	-0.187***	-0.0181	-0.0340	-0.0353
	(0.175)	(0.0855)	(0.0917)	(0.0592)	(0.173)	(0.184)	(0.172)
SO_2-39w-Sh(q4)	-0.127		-0.167***	-0.119***	-0.151		-0.200*
	(0.0797)		(0.0493)	(0.0359)	(0.0979)		(0.103)
SO_2-39w-Sh(q5)	-0.119*		-0.127**	-0.0390	-0.133**		-0.166**
	(0.0587)		(0.0530)	(0.0385)	(0.0587)		(0.0629)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Nlbw/w , Nsga/w, and Nptb/w correspond to the number of non-low birthweight (birthweight>2500g), non-small for gestational age (birthweight > P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race, and non-preterm birth (gestational age>=37 weeks) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Low (High) Baseline SO2 corresponds to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). newborn-level observations collapsed into cells by county-week-year-race. Government transfers correspond to the county's transfer payments excluding medical care and supplemental security income divided by the population of 64yrs old or younger. For model (7), separate trends were allowed for each of the quintiles of SO_2 (q1,q2,q4,q5). All regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.10: Effect of simulated eligibility for Medicaid on the effect of SO_2 on the number of non-low birthweight infants.

	(1) Nlbw/w	(2) Nlbw/w	(3) Nlbw/w	(4) Nlbw/w	(5) Nlbw/w
SO2 Baseline	All	Low	High	All	All
Race	All	All	All	White	Black
Mean (Y)	1.363	1.386	1.342	0.977	1.540
Cells	343,023	160,124	182,899	161,102	103,494
R-squared	0.652	0.643	0.623	0.722	0.440
I(SEM>$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q1)	0.137	0.0443	0.563	0.204	-1.167
	(0.719)	(0.998)	(0.886)	(0.731)	(1.635)
I(SEM>$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q2)	0.311	0.354	0.382	0.377	-0.341
	(0.558)	(0.745)	(0.961)	(0.546)	(1.254)
I(SEM>$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q4)	0.421	0.248	0.853	0.637	-1.701
	(0.700)	(0.964)	(0.924)	(0.709)	(1.692)
I(SEM>$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q5)	0.0188	1.016	-0.00186	0.168	-1.349
	(0.825)	(0.837)	(0.924)	(0.831)	(0.984)
I(SEM<$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q1)	0.279	-0.155	0.522	0.200	1.769
	(0.732)	(0.722)	(0.665)	(0.698)	(2.235)
I(SEM<$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q2)	-0.182	-0.709	0.937	-0.269	1.674
	(0.712)	(0.985)	(0.957)	(0.747)	(1.407)
I(SEM<$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q4)	-1.097	-2.228**	0.131	-1.080	-1.934
	(0.747)	(0.949)	(0.720)	(0.756)	(1.991)
I(SEM<$\widetilde{SEM_s}$)*|SEM-$\widetilde{SEM_s}$|*SO_2-39w-Sh(q5)	-1.916***	-1.516	-1.557***	-1.576***	0.805
	(0.481)	(1.260)	(0.530)	(0.472)	(1.633)
SO_2-39w-Sh(q1)	-0.0376	-0.0500	-0.0443	-0.0261	-0.0546
	(0.0324)	(0.0483)	(0.0460)	(0.0264)	(0.0884)
SO_2-39w-Sh(q2)	-0.0321	-0.0637	0.0174	-0.0421	0.0103
	(0.0473)	(0.0600)	(0.0504)	(0.0409)	(0.101)
SO_2-39w-Sh(q4)	-0.0286	-0.0399	-0.0119	-0.0409	0.120
	(0.0394)	(0.0542)	(0.0416)	(0.0344)	(0.0984)
SO_2-39w-Sh(q5)	-0.0166	-0.0643	0.00389	-0.0130	-0.0665
	(0.0289)	(0.0619)	(0.0244)	(0.0286)	(0.0649)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Nlbw/w corresponds to the number of non-low birthweight (birthweight>2500g per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. Low (High) Baseline SO2 corresponds to counties in the bottom (top) 5 deciles of SO_2 concentration during the first two years in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. SEM corresponds to simulated eligibility for Medicaid, and $\widetilde{SEM_s}$ corresponds to the median simulated eligibility for Medicaid in state *s*. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). newborn-level observations collapsed into cells by county-week-year-race. All regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.11: Medicaid's expansion impact on the effect of SO_2 on fetal death by race.

	(1) FR	(2) FR	(3) FR	(4) FR
Maternal race	White	Black	White	Black
Cells	161,155	103,632	161,155	103,632
Mean Y	1.011	1.672	1.011	1.672
R-squared	0.732	0.465	0.732	0.465
Medicaid Exp*SO_2-39w-Sh(q1)			0.0531	0.183
			(0.0494)	(0.1130)
Medicaid Exp*SO_2-39w-Sh(q2)			0.0375	0.056
			(0.0771)	(0.1650)
Medicaid Exp*SO_2-39w-Sh(q4)			0.0442	0.314*
			(0.0556)	(0.1670)
Medicaid Exp*SO_2-39w-Sh(q5)			0.0918**	0.0203
			(0.0382)	(0.1630)
SO_2-39w-Sh(q1)	-0.0183	-0.0535	-0.0653	-0.218*
	(0.0220)	(0.0743)	(0.0468)	(0.1240)
SO_2-39w-Sh(q2)	-0.0555	0.00701	-0.0902	-0.039
	(0.0476)	(0.0980)	(0.0696)	(0.1810)
SO_2-39w-Sh(q4)	-0.0682**	0.0174	-0.109**	-0.272
	(0.0319)	(0.0836)	(0.0505)	(0.1780)
SO_2-39w-Sh(q5)	-0.0523*	-0.102*	-0.139***	-0.117
	(0.0279)	(0.0575)	(0.0360)	(0.1680)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. The dependent variable is FR (fertility rate) = 1000*(N live birth/ N women 15-44) by county, race and week-year of conception cells. Model was estimated using all counties with available SO_2 data. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year FE, county-Quarter FE, and month FE. Controls included: Temperature-39w(bins), precipitation-39w(bins), and the unemployment rate during the first trimester of pregnancy. Regressions were weighted by the number of women of reproductive age in each county-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2.12: Medicaid's expansion impact on the effect of SO_2 on the number of infants born with non-adverse outcomes by race.

	(1) Nlbw/w	(2) Nlbw/w	(3) Nsga/w	(4) Nsga/w	(5) Nptb/w	(6) Nptb/w
Maternal race	White	Black	White	Black	White	Black
Cells	161,102	103,500	161,102	103,500	161,102	103,500
Mean Y	0.977	1.540	0.912	1.522	0.964	1.501
R-squared	0.721	0.440	0.719	0.450	0.699	0.423
Medicaid Exp*SO_2-39w-Sh(q1)	0.0657	0.139	0.0560	0.158	0.0624	0.139
	(0.0439)	(0.0931)	(0.0473)	(0.0984)	(0.0431)	(0.109)
Medicaid Exp*SO_2-39w-Sh(q2)	0.0665	0.0905	0.0722	0.0703	0.0641	0.118
	(0.0705)	(0.145)	(0.0754)	(0.142)	(0.0653)	(0.185)
Medicaid Exp*SO_2-39w-Sh(q4)	0.0592	0.268**	0.0586	0.299**	0.0530	0.327**
	(0.0495)	(0.121)	(0.0511)	(0.135)	(0.0501)	(0.133)
Medicaid Exp*SO_2-39w-Sh(q5)	0.0982**	0.0226	0.111***	0.0387	0.0973**	0.00336
	(0.0370)	(0.138)	(0.0373)	(0.148)	(0.0369)	(0.158)
SO_2-39w-Sh(q1)	-0.0770*	-0.160*	-0.0648	-0.182*	-0.0754*	-0.163
	(0.0400)	(0.0879)	(0.0409)	(0.100)	(0.0405)	(0.112)
SO_2-39w-Sh(q2)	-0.103	-0.0423	-0.0979	-0.0233	-0.0985	-0.0855
	(0.0629)	(0.150)	(0.0618)	(0.140)	(0.0603)	(0.192)
SO_2-39w-Sh(q4)	-0.112***	-0.202*	-0.102**	-0.243*	-0.108**	-0.262**
	(0.0398)	(0.113)	(0.0390)	(0.133)	(0.0401)	(0.120)
SO_2-39w-Sh(q5)	-0.135***	-0.0912	-0.143***	-0.105	-0.130***	-0.0705
	(0.0339)	(0.143)	(0.0305)	(0.150)	(0.0338)	(0.165)

Notes: Author's calculations from CDC public birth files 1982-1988 and restricted-use birth files 1989-1991. Nlbw/w , Nsga/w, and Nptb/w correspond to the number of non-low birthweight (birthweight>2500g), non-small for gestational age (birthweight > P_{10}) where P_{10} is the 10^{th} percentile of birthweight computed for every gestational age-gender-maternal race, and non-preterm birth (gestational age>=37 weeks) per thousand woman of reproductive age (15-44 yrs. old) from the respective race, county, and year. All counties with SO_2 data were included in the sample. SO_2-39w-Sh(q j) corresponds to the fraction of days, from a 39-week period started at the week of conception, in which the county's SO_2 concentration lies in the j-th quintile of national daily distribution of SO_2. The third quintile is the omitted category. Medicaid Exp is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past. All regressions include county-year-race FE, county-Quarter-race FE, and month-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Temperature-39w(bins), precipitation-39w(bins), the unemployment rate during the first trimester of pregnancy, and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). newborn-level observations collapsed into cells by county-week-year-race. All regressions are weighted by the number of women 15-44 yrs. old per county-race-year. Standard errors are clustered at the state level. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

2.11 Methodological Appendix C: Interaction terms in panel data models

This methodological appendix shows the analytical decomposition of the interaction term of two continuous variables in a panel data model. The baseline model consists of a two-way fixed effects balanced panel.

Suppose we estimate the following linear regression model:

$$Y_{it} = \mu_i + \mu_t + \delta M_{it} + \theta P_{it} + \phi M_{it} P_{it} + u_{it} \quad [C1]$$

Where Y_{it} is a health outcome in county i and period t. Medicaid (M) and Pollution (P) are two continuous variables and can be accurately described by a mean, a trend, and a shock as equations [C2] and [C3] show.

$$M_{it} = \bar{M}_i + \bar{M}_t + m_{it} \; [C2]$$

$$P_{it} = \bar{P}_i + \bar{P}_t + p_{it} \; [C3]$$

Suppose the air pollution and Medicaid shocks are orthogonal: $Cov(m_{it}, p_{it}) = 0 \; [C4]$

Also, since the panel is balanced, we expect:

$$Cov(\bar{M}_i, \bar{M}_t) = 0, \; Cov(\bar{P}_i, \bar{P}_t) = 0, \; Cov(\bar{M}_i, \bar{P}_t) = 0, \quad Cov(\bar{P}_i, \bar{M}_t) = 0 \; [C5]$$

Using [C2] and [C3], the interaction term can be expressed as:

$$MP_{it} = M_{it} * P_{it}$$

$$= \bar{M}_i\bar{P}_i + \bar{M}_t\bar{P}_t + \bar{M}_i\bar{P}_t + \bar{M}_t\bar{P}_i + p_{it}\bar{M}_i + p_{it}\bar{M}_t + m_{it}\bar{P}_i + m_{it}\bar{P}_t + m_{it}p_{it} \; [C6]$$

The first and second terms in [C6] are absorbed by county (μ_i) and time (μ_t) fixed effects, respectively.

The third and fourth term will converge to zero by [C5]. Hence, after adding county and fixed effects to the model, the remaining terms in the interaction are:

$$mp_{it} = p_{it}\bar{M}_i + p_{it}\bar{M}_t + m_{it}\bar{P}_i + m_{it}\bar{P}_t + p_{it}m_{it} \; [C7]$$

In this setting, the term mp_{it} is composed of five different terms. Therefore, its interpretation should be done carefully depending on the objective of the analysis.

If the objective is describing heterogeneity of pollution estimates, it would be enough to remove the third and fourth terms in [C7]. In the other hand, if the objective is testing if Medicaid changed pollution's effects, it would be better to keep only the last term in [C7]

Isolating this last term can be done by adding heterogeneous effects across both panel dimensions: (1) The terms $p_{it}\bar{M}_i$ and $p_{it}\bar{M}_t$ can be removed by estimating heterogeneous effects of p_{it} across counties and time, respectively. (2) the terms $m_{it}\bar{P}_i \; and \; m_{it}\bar{P}_t$ can be removed from the interaction by estimating heterogeneous effects of Medicaid across counties and time. Consequently, it would be sufficient to augment the regression model in [C1] to:

$$y_{it} = \mu_i + \mu_t + \delta_i M_{it} + \delta_t M_{it} + \theta_i P_{it} + \theta_t P_{it} + \phi MP_{it} + v_{it} \quad [C8]$$

Now, once the term $p_{it}m_{it}$ has been isolated in [C8], the challenge is its interpretation. Both m_{it} and p_{it} can be either positive or negative. In consequence, there are four different cases embedded into a single coefficient:

a) $m_{it} > 0\ ,\ p_{it} > 0$

b) $m_{it} > 0\ ,\ p_{it} < 0$

c) $m_{it} < 0\ , p_{it} < 0$

d) $m_{it} < 0\ , p_{it} > 0$

The interpretation of this term is not straightforward. There are many possible paths to disentangle and interpret this term; a clever one is to make one of the variables categorical. In this paper, simulated eligibility for Medicaid is turned into a binary variable; hence, m_{it} can only take on two values for each county (before and after the expansion of Medicaid). Doing so, ϕ, the coefficient of the interaction term, can be interpreted as the change in pollution's estimate when Medicaid expands.

A final remark: Turning one of the variables (M or P) into binary in [C1] does not necessarily grant a causal interpretation. The single interaction term's estimate in [C1] may be driven by either of the first four terms in [C7]. Ultimately, the researcher must decide which terms they want to keep in the interaction and interpret the estimate accordingly.

2.12 Methodological Appendix D: Modifying interaction terms in panel data models to guarantee a causal interpretation.

This methodological appendix informs the empirical strategy in models [2.1] and [2.2]. This framework is still a simplified version of those models because it omits the monthly nationwide seasonal fixed effects, the race dimension in the fixed effects, and the non-linear formulation in the effects of pollution; however, including them does not change the conclusions.

Objective: Derive, analytically, the residual variation contained in the interaction term when equations [2.1] and [2.2] are estimated to verify that only the plausibly exogenous variation in pollution is used to estimate the interaction term. This Methodological appendix builds upon Methodological appendix C.

Starting from the model in [C1]:

i. Suppose the outcome variable and pollution are measured at the county level with a weekly frequency.
ii. Medicaid is Measured as a binary variable, D_{sy}, that varies at the state-year level.

<u>In order to isolate the plausibly exogenous variation in pollution:</u>

iii. Use county-year (μ_{cy}) instead of county (μ_c) and year (μ_y) fixed effects.
iv. Add by-county-quarter seasonal effects to the model.

Hence the new model, analogous to models [2.1] and [2.2] is:

$$Y_{cwy} = \mu_{cy} + \mu_{cQ} + \theta P_{cwy} + \phi D_{sy} P_{cwy} + u_{cwy} \quad [D1]$$

Notice that Medicaid cannot be estimated in this model because it varies at the state-year level and we have imposed county-year fixed effects.

Additionally, suppose that Medicaid (M) and Pollution (P) are two continuous variables and the identifying assumption for a causal interpretation of the estimates rest of the exogeneity of d_{sy}, and p_{cwy} in equations [D2] and [D3] below. The term $\bar{P}_{CQ}$ denotes seasonal effects by county-quarter.

$$D_{sy} = \bar{D}_s + \bar{D}_y + d_{sy} \; [D2]$$

$$P_{cwy} = \bar{P}_c + \bar{P}_y + \bar{P}_{cQ} + p_{cwy} \; [D3]$$

Now, strictly from an algebraic point of view, equations $[D2]$ and $[D3]$ can be represented more flexibly with the following equations:

$$D_{sy} = \bar{D}_{sy} + d_{sy} \; [D4]$$

$$P_{cwy} = \bar{P}_{cy} + \bar{P}_{cQ} + p_{cwy} \; [D5]$$

Hence, the interaction term, the product of $[D4]$ and $[D5]$, corresponds to:

$$DP_{cwy} = D_{sy} * P_{cwy} = \bar{P}_{cy}\bar{D}_{sy} + \bar{P}_{cQ}\bar{D}_{sy} + p_{cwy}\bar{D}_{sy} \; [D6]$$

The first term in [D6] is absorbed by μ_{cy}.

If the local seasonality in pollution is not affected by Medicaid, as has been implicitly assumed, the second term will be absorbed by the county-quarter seasonal fixed effects.

Thus, for a large enough time series, so that $\bar{P}_{cQ} \longrightarrow \mu_{cQ}$, we should expect:

$$\left(\bar{P}_{cQ}\bar{D}_{sy} - \bar{P}_{cQ}\right) \longrightarrow 0$$

Nevertheless, the second term in [D6] can be fully removed by adding interaction terms between the county-quarter seasonal effects and D_{sy}. In this paper, adding interaction terms between seasonal effects and Medicaid does not change the results, but it may be relevant in other cases.

In conclusion, only one term remains in the interaction term in models [2.1] and [2.2] and corresponds to the interaction of two plausibly exogenous sources of variation.

$$DP_{cwy} = p_{cwy}\bar{D}_{sy} \; [D7]$$

This variation corresponds to the changes in air pollution across weeks of conception within the same county and year interacted with a binary variable (Medicaid's expansion). In this model, ϕ is identified by comparing the average effect of pollution when $\bar{D}_{sy} = 0$ (pre-expansion) vs $\bar{D}_{sy} = 1$ (post-expansion).

2.13 Methodological Appendix E: Refining the estimation of interaction terms in panel data models

This methodological appendix explains why it is desirable to allow for heterogeneous effects of air pollution across states in models [2.1] and [2.2].

Given the staggered nature of Medicaid's expansion, there is one last caveat with model [D1]. The coefficient of the interaction, ϕ, will not necessarily converge to the desire estimand if Medicaid's expansion has heterogeneous effects across states. Consider the following, over-simplified, example: Suppose we have a balanced sample of two counties belonging to different states and ten periods. Medicaid's expansion happened in year 4 in county A and year 6 in county B. Suppose that air pollution's effects before the expansion were 0.5 in county A and 0.7 in county B. After the expansion, they decreased to 0.3 and 0.4 in county A and B, respectively. Hence, the effects were $\tau_A = -0.2\,, \tau_B = -0.3$

For ease of explanation, suppose that the total number of births in county A and B during the 10 years is the same in both counties, and it was not affected by Medicaid's expansion. In consequence, the average effect of Medicaid expansion on the air pollution-birth outcome relationship is $\phi = \frac{1}{2}(\tau_A + \tau_B) = -0.25$. However, since model [D1] identifies the coefficient of the interaction from the comparison of the average effects of air pollution before and after the expansion, the estimate[94] would be $\hat{\phi} = -0.2875$.

[94] Notice that the weight of county A (B) in the pre-period is 3/8 (5/8) and 5/8 (3/8) in the post period. Then, the average effect of air pollution was (3/8)*0.5+(5/8)*0.7=0.625 in the pre-period, and (5/8)*0.3+(3/8)*0.4=0.3375 in the post-period. Thus $\widehat{\phi} = -0.2875$

The true estimate, $\phi = -0.25$, can be recovered by allowing heterogeneous effects of air pollution across states (θ_s) in [D1]. The model would be:

$$Y_{cwy} = \mu_{cy} + \mu_{cQ} + \theta_s P_{cwy} + \phi D_{sy} P_{cwy} + u_{cwy} \quad [E1]$$

One remark: if the effect of Medicaid on pollution's health effect was homogeneous (i.e., $\tau_A = \tau_B = -0.25$), we would not need heterogeneous effects of pollution across states to recover $\rho = -0.25$.

Finally, in [D1] ϕ is identified by comparing the average effect of pollution when $\overline{D}_{sy} = 0$ (pre-expansion) vs $\overline{D}_{sy} = 1$ (post-expansion). However, some states did not experience an expansion throughout the period analyzed. The control states had expanded Medicaid before 1981, for them $D_{sy} = 1 \; \forall y$. If we want to estimate ϕ using only the variation from the treated states before and after the expansion, we need to use model $[E1]$ or exclude the control states from the sample.

2.14 Methodological Appendix F: Livebirth bias in health care's impact

This methodological appendix is a continuation of Methodological Appendix B.

The conceptual model in Appendix B can be used to analyze how the interaction term between Medicaid's expansion and pollution in [2.2] is affected by sample selection. For this, we need to take the derivative of $\frac{dlbw_r}{dP}$ with respect to Medicaid's expansion (ME):

$$\frac{\partial\left(\frac{dlbw_r}{dP}\right)}{\partial ME} = \frac{1}{LB^2}\left(\frac{\partial\,\tau_{LB}^{fd}}{\partial ME}\left(lbw\; - \tau_{nlbw}^{lbw} + \tau_{lbw}^{fd}\right) + \left(\frac{\partial\,\tau_{nlbw}^{lbw}}{\partial ME} - \frac{\partial \tau_{lbw}^{fd}}{\partial ME}\right)\left(LB + \tau_{LB}^{fd}\right)\right.$$

$$\left. + 2\,\frac{\partial\,\tau_{LB}^{fd}}{\partial ME}\left(\frac{dlbw_r}{dP}\right)\,(LB)\right) \quad [F1]$$

We can observe in [F1] that if air pollution shocks cause fetal deaths ($\tau_{lbw}^{fd} > 0$ or $\tau_{nlbw}^{fd} > 0$), interpreting the interaction term in [1.2] is challenging.

The interaction term in model [2.1] gives us the impact of Medicaid expansion on pollution's effect on fetal deaths $\frac{\partial\,\tau_{LB}^{fd}}{\partial ME}$. On the contrary, the interaction term in model [2.2] is a combination of $\frac{\partial\,\tau_{LB}^{fd}}{\partial ME}$, $\frac{\partial\,\tau_{nlbw}^{lbw}}{\partial ME}$, $\tau_{nlbw}^{lbw}, and\ \tau_{lbw}^{fd}$.

Ideally, we would want to isolate the impact of Medicaid on pollution effects through the intensive margin, $\frac{\partial\, \tau_{nlbw}^{lbw}}{\partial ME}$. We can get close to this goal by using the number of non-low birthweight infants -as opposed to the low birthweight rate- as the outcome variable in model [2.2].

Thus, we can take the derivative of $\frac{d\ nlbw}{dP}$ with respect to Medicaid's expansion.

$$\frac{\partial\ \left(\frac{\partial\ nlbw}{\partial P}\right)}{\partial\ Med\ Exp} = -\frac{\partial \tau_{nlbw}^{fd}}{\partial\ ME} - \frac{\partial \tau_{nlbw}^{lbw}}{\partial\ ME} \quad [F2]$$

If $\frac{\partial \tau_{nlbw}^{fd}}{\partial\ ME}$ in [F2] is close to zero, we would effectively capture Medicaid's impacts through the intensive margin.

If $\frac{\partial \tau_{nlbw}^{fd}}{\partial\ ME}$ in [F2] were similar to $\frac{\partial \tau_{nlbw}^{lbw}}{\partial\ ME}$, the interaction term would capture Medicaid's impacts through the extensive and intensive margin; however, the effects of both margins go in the same direction.

Essay 3

Mitigating the impacts of extreme temperature on birth outcomes: Free prenatal care or air conditioning?

3.1 Introduction

Extreme temperatures during the gestational period have adverse short and long-term consequences that vary across socioeconomic factors. Since extreme-heat episodes are becoming more frequent due to climate change, understanding the causal mechanisms driving such heterogeneity is critical for designing policies that reduce climate change's health impacts cost-effectively. Besides increasing fetal death (Wilde et al., 2017) and deteriorating maternal health (Kim et al., 2021), extreme heat has also been linked to adverse pregnancy outcomes such as preterm birth (Basu et al., 2010; Dadvand et al., 2011; Andalón et al., 2016; Smith & Harderman, 2020), low birth weight (Deschênes et al., 2009; Molina & Saldarriaga, 2017), and infant mortality (Banerjee & Maharaj, 2020). Furthermore, it affects economic outcomes in adulthood (Isen et al., 2017). Importantly, previous research has documented heterogeneous impacts by mothers' race, age, and education (Banerjee & Maharaj, 2020; Anadalon et al., 2014; Deschênes et al., 2009; Basu et al., 2010; Smith & Harderman, 2020). However, the causal mechanisms behind such heterogeneity are poorly understood. For example, income, healthcare, access to air conditioning, or maternal behavior could be the drivers of such heterogeneity. Unveiling this is essential to identifying interventions that effectively mitigate the effects of extreme temperatures (Hsiang et

al., 2019) and lessen the health impacts of climate change cost-effectively (Mullins & White, 2020).

This paper examines and quantifies the extent to which prenatal care and air conditioning mitigate the adverse effects of extreme temperatures on birth outcomes in a developed country context. I examine the effects empirically using publicly available vital statistics data, daily temperature and rainfall data, spatial-temporal variation in residential air conditioning ownership, and spatial-temporal policy variation from Medicaid's expansion during the 1980s. This expansion to Medicaid gave free access to prenatal health care – and at least one year after birth— to low-income women who became pregnant. Recent experimental evidence by Shankar et al. (2023) suggests that the effects of heat on birth length were partially mitigated in women randomized to a comprehensive nutritional supplementation (e.g., vitamins, iron, calcium) program before pregnancy in Pakistan. Thus, nutritional supplementation received during prenatal care could offset the negative impacts of heat on birth outcomes.

I use spatial-temporal variation in Medicaid's expansion to estimate its impact on the effect of extremely hot and cold days on birth outcomes. I build upon the panel-fixed effects approach similar to the one used by Deschênes et al. (2009) to identify the causal impact of extreme weather on birth outcomes. Then, I expand the model by adding interaction terms between a binary variable to measure Medicaid expansion,[95] and each temperature bin— which varies by county of residence and week of conception.[96] Exposure was measured as the number of days during each trimester (i.e., 13-week window) in which the county's daily temperature falls in each bin (<25 F, 25-45 F,

[95] This variable varies at the state-year leven and was created by East, Miller, Page & Wherry (2023)
[96] Assumed to be two weeks after the last day of the menstrual period. This date is reported on the birth certificates

45-65 F, 65-85 F, and >85 F). The effects of temperature are identified using temporal variation in exposure through conception weeks within the same county and year and controlling by county-week-race fixed effects, in-utero rainfall by trimester, and mothers' demographics.

To estimate the impact of Air Conditioning (AC) on the temperature-birth outcome damage function, I build upon the empirical strategy used by Barreca et al. (2016) to study the impact of AC on the temperature-mortality relationship. I interact exposure to each temperature bin during the pregnancy with the state's mean residential air conditioning ownership rate.

The empirical analysis starts by finding evidence of a negative impact of extreme temperatures on birthweight in the US for the 1974-1988 period. As Deschênes et al. (2009) reported for the US during the 1972-1988 period, I also found negative impacts of days in the 65-85 F and T>85F range during the second and third trimesters. Additionally, I find that extreme cold and heat during the first trimester reduce the duration of the pregnancy. This first step sets the base to test if increased access to healthcare lessens the impact of extreme temperatures during the prenatal period on an infant's health.

As this paper's central result, I find that Medicaid's expansion did not mitigate the effects of extreme temperatures during the gestational period on birth outcomes. This result may be relevant for other developed countries or urban areas. As a second result, I found that increased access to residential air conditioning mitigated the effects of extreme heat on birthweight and gestational age.

This paper contributes to a growing literature that explores which interventions effectively help society adapt to climate change. Previous literature has suggested air conditioning (Barreca et al., 2016), health care (Mullins & White, 2020; Banerjee & Maharaj, 2020; Cohen & Dechezleprêtre, 2022), cash transfers (Garg et al., 2020), and access to banks (Burgess et al., 2017) as strategies that effectively lessen the health impacts of extreme temperatures. This paper expands this literature by evaluating the effectiveness of health care and air conditioning in mitigating the impacts of in-utero temperatures on birth outcomes (birthweight and gestational age) in a developed country context.

Banerjee & Maharaj (2020) found that medical care lessens the impacts of extreme heat during the gestational period on infant mortality in rural India, but no evidence exists for developed countries. The distinction is important because there are multiple channels through which temperature may affect health in developing countries (e.g., changes in real income, increased incidence of maternal disease, conflicts, or crime). On the other hand, in developed countries, physiological stress is the primary mechanism of temperature affecting fetuses' health, and air conditioning (AC) is more widely available.

This paper also explores the types of adverse prenatal shocks that Medicaid mitigates. Previous research shows that pregnancy outcomes improved when low-income women gained access to free prenatal care through Medicaid (Currie & Gruber, 1996a, 1996b; East et al., 2023; Goodman-Bacon, 2018). Many types of shocks could cause such poor outcomes (e.g., income, nutrition, pollution, temperature, rainfall, maternal disease, or stress). However, previous research has not established which of them are mitigated by prenatal care. Based on evidence from a developing

country, this paper's result suggests that Medicaid does not mitigate the negative impacts of extreme temperatures on birth outcomes.

The remainder of the paper is laid out as follows: Section 3.2 reviews previous research on the impacts of in-utero temperatures on pregnancy outcomes and presents the mechanisms through which prenatal care and air conditioning could mitigate the health impacts of extreme temperatures. Section 3.3 describes the data. Section 3.4 explains the empirical strategy. Section 3.5 presents the results and robustness tests. Finally, Section 3.6 concludes.

3.2 Background and Conceptual framework.

This section presents background information regarding the effects of prenatal temperature on pregnancy outcomes, as well as the potential impact of healthcare and air conditioning on the temperature-birth outcome relationship. First, section 3.2.1 reviews the background information about the impacts of in-utero temperature on fertility, fetal death, and birth outcomes. Next, section 3.2.2 analyzes the potential mechanisms through which prenatal care could impact the effects of temperature on birth outcomes. Finally, section 3.2.3 reviews previous research that suggests that air conditioning is an effective strategy to mitigate the health impacts of extreme heat. The background on Medicaid was presented in section 2.2 (previous chapter).

3.2.1 In-utero temperature and pregnancy outcomes

Extreme temperatures during conception and in-utero adversely impact short- and long-term outcomes. Extreme heat impacts reproductive health (Barreca et al.,2018), increases fetal death (Wilde et al., 2017), and deteriorates maternal health (Kim et al., 2021). Infants' health is also

affected. Extreme temperatures during pregnancy increase the incidence of preterm delivery (Basu et al., 2010; Dadvand et al., 2011; Andalón et al., 2016; Smith & Harderman, 2020), low birth weight (Deschênes et al., 2009; Andalón et al., 2016; Molina & Saldarriaga, 2017), and infant mortality (Banerjee & Maharaj, 2020). Furthermore, these impacts go beyond the infant's health and affect economic outcomes in adulthood (Isen et al., 2017).

Multiple channels could drive these effects. In developed countries, physiological stress is the primary concern. In agricultural economies, extreme heat could, in addition, (i) increase the likelihood of disease, (ii) decrease real income, and (iii) increase conflicts and crimes. Physiological stress is caused by a failure in the body's thermoregulatory mechanisms. Under extreme heat, blood flow is redirected from organs to the skin to keep a normal body temperature (Bouchama & Knochel, 2002). Pregnant women are particularly susceptible to extreme temperatures because their bodies' temperature is higher than usual. Animal studies have shown that extreme temperatures during the gestational period worsen birth outcomes and can cause congenital malformations (Edwards et al., 2002; Edwards et al., 2003). In addition, medical research suggests that blood flow to the uterus decreases under extreme temperatures, leading to reduced fetal nutrient uptake (Soultanakis-Aligianni, 2003).

In rural areas of the developing world, the relationship between weather and pregnancy outcomes is even more complex because the weather can affect health directly through physiological channels and indirectly through its effects on crop yields and increased food prices (Banerjee & Maharaj, 2020). Furthermore, the effects of temperature on infants' health may depend on local sensitivity to other environmental factors like rainfall and diseases linked to weather, such as

malaria (Kudamatsu et al., 2012). Additionally, extreme weather shocks could affect health indirectly through increased risk of conflict.[97] Extreme temperatures increase the onset of conflicts (Burke et al., 2015), and conflict-induced maternal stress may worsen birth outcomes. An advantage of this paper is that all these alternative mechanisms through which temperature can affect health are not present for the US during the 1980s.

The effects of in-utero temperature on birth outcomes differ over socioeconomic dimensions, but the sources of such heterogeneity are poorly understood. In India, the effects are large in rural areas but insignificant in urban ones; they are also larger for less educated mothers and in colder districts –although imprecisely estimated (Banerjee & Maharaj, 2020). In Colombia, the effects are larger for younger and less educated mothers (Andalon et al., 2014). In the US, the effects of extreme heat on birthweight are larger for blacks (Deschênes et al., 2009). Similarly, the impacts on preterm delivery are larger for young, black, and Asian mothers in California (Basu et al., 2010) and educated black mothers in Minnesota (Smith & Harderman, 2020). Nevertheless, the sources of such heterogeneity by race and education are poorly understood. Income, health care, or access to air conditioning could be the drivers behind them, among others.

3.2.2 Prenatal care as an adaptation to extreme temperatures.

The climate-change-driven increase in the frequency of extreme weather has stimulated a growing literature exploring the effectiveness of interventions in attenuating the temperature-health relationship. Such adaptation strategies consist of behavioral changes or interventions that reduce

[97] From a biological point of view, high temperature affects serotonin neurotransmission in the brain, which makes people prone to aggression and violent crimes (Dell et al., 2014). In contrast, non-biological mechanisms are derived from heat-induced lower economic growth. Including a lower opportunity cost of violence or protest, reduced state capacity to maintain security, higher food prices leading to food riots, and weather-induced migration (Dell et al., 2014).

the impacts of extreme weather events. For example, Mullins and White (2020) find that access to primary care services provided by community health centers lessened the heat-mortality relationship but did not mitigate the effects of cold in the US through the 60s and 70s. Similarly, Garg et al. (2020) find that cash transfers mitigate the effects of same-day temperatures on homicides in Mexico.

The effectiveness of interventions in mitigating the health-at-birth impacts of extreme in-utero temperatures has yet to be widely studied. The most comprehensive study, to my knowledge, comes from Banerjee and Maharab (2020), who found that a community healthcare worker program mitigates the impact of extreme temperature on infant mortality in rural India while an employment-guarantee program was ineffective in mitigating it. However, there is no previous research on the effectiveness of prenatal care in mitigating the effects of temperature in-utero on birth outcomes in developed countries. The distinction between developed and developing countries is essential because the predominant mechanism in developed countries is physiological. In developing countries, indirect effects through real income or increased incidence of disease and conflict are also significant (Banerjee & Maharab, 2020).

Previous evidence suggests that medical attention during prenatal care (nutrition and drug prescriptions, immunizations, screening and early diagnosis) could mitigate the physiological impact of extreme temperature on birth outcomes. As previously mentioned, extreme temperatures are believed to reduce the fetuses' nutritional uptake (Soultanakis-Aligianni, 2003); Thus, nutritional supplementation could counteract this effect. Furthermore, experimental research by Shankar et al. (2023) found that the negative impacts of heat on birth length were partially

mitigated in women randomized to a comprehensive maternal nutritional supplementation program (micronutrients plus balanced energy supplement[98]), before pregnancy in Pakistan.

Lastly, as discussed in section 2.3 (previous chapter), besides the medical-biological channel, there are two potential channels through which increased access to health care could impact the temperature-birth outcome relationship. (i) the information channel, and (ii) the optimal response channel. Nevertheless, there is no evidence that obstetricians recommended women avoid exposure to heat during pregnancy in the US during the 1980s (Hemminki, 1988). The optimal response channel is unlikely to apply for temperature because individuals avoid extreme temperatures not just to mitigate the possible impacts on health but also to avoid the discomfort caused by them.

3.2.3 Air conditioning as an adaptation to extreme heat

Air conditioning (AC) has been identified as an effective technology to mitigate the impacts of extreme heat on health. The seminal paper in this literature found that the diffusion of residential air conditioning explains the decline in the temperature-mortality relationship in the US during the 1960s (Barreca et al., 2016). In a similar line of research, Deschênes and Greenstone (2011) found that electricity consumption increased for daily temperatures above 90° F. One extra such day, relative to a day in the 50°–60°F range, leads to a 0.4 percent increase in annual consumption in the US during 1968-2002.

[98] The nutritional supplement consisted of a daily 20 g small quantity-lipid-based nutritional supplement (sqLNS) with 22 micronutrients in amounts appropriate for pregnancy and lactation. In addition to the multiple micronutrients and polyunsaturated lipids (linoleic 4.9 g and α-linolenic 0.59 g), the composition included dried skimmed milk, soybean and peanut extract, sugar, maltodextrin stabilizers, and emulsifiers (Nutriset, Malauney, France). An additional lipidbased balanced protein/energy supplement (300 kcal/d; 12% calories from protein and no added micronutrients; Nutriset, Maluaney, France) was provided to women with low BMI (≤20) or low gestational weight gain.

Both of these findings suggest that households in the US have used air conditioning to mitigate the discomfort caused by heat, and its use mitigated the impact of extremely hot days on the mortality rate. Therefore, AC could also have mitigated the impact of in-utero heat on birth outcomes. Nevertheless, to the best of my knowledge, no previous research has evaluated its effectiveness on birth outcomes.

3.3 Data

The empirical analysis relies on multiple data sources. Section 1.3 (previous chapter) describes the sources and methodologies to build the health outcomes and measure temperature and precipitation) in-utero. However, in this paper, the study period is 1974-1988. Table 3.1 presents the summary statistics for these years. Section 2.4 (previous chapter) describes the measures of access to prenatal care. This chapter's empirical strategy also uses the binary measure of Medicaid expansion created by East et al. (2023).

County-level controls proxying for local economic conditions and government transfers were built using Bureau of Economic Analysis (BEA) data. The unemployment rate is available at the state level with monthly frequency. Per-capita income was taken from the deflated annual county-level BEA series. Government transfers were computed using county-year series from the Regional Economic Information System (REIS) sourced from the BEA. Per capita government transfers were divided into unemployment insurance and non-medical welfare programs. The latter was computed excluding government expenditure on medical care and Supplemental Security Income (SSI) and dividing by the population 64 years old or younger. Population counts came from the

Surveillance, Epidemiology, and End Results Program (SEER) data. Air conditioning ownership rates by state, race, and year were computed using decennial Census data and following the methodology used by Barreca et al. (2016). For years prior to 1980, linear interpolation was used. After 1980, they linearly extrapolated state-year ownership rates between using the annual rate of change between 1970 and 1980 censuses and bounded AC rate ownership at 100%. Finally, I used USDA's 1986 county typology to characterize urban vs. rural counties. Summary statistics are presented in Table 3.1.

Figure 3.1 presents the mean birthweight during 1974-1988 with a monthly frequency. Figure 3.2 summarizes the distribution of in-utero temperature across different samples. Lastly, Figure 3.3 presents the fraction of households that had access to air conditioning from 1974 to 1988 in the US and separately by race.

3.4. Empirical strategy

This paper's empirical strategy aims to establish whether healthcare access for low-income pregnant women and air conditioning mitigate the effect of in-utero temperature on birth weight. To do so, I build upon the strategy proposed by Hsiang, Oliva & Walker (2019), whose framework corresponds to the state of the art to identify, empirically, the sources of heterogeneity of environmental damages. The following example explains the intuition behind the challenge of identifying the causal mechanisms driving heterogeneous environmental damages. Suppose an environmental factor, *x (e.g., temperature)*, has heterogeneous effects across a given dimension, *z (e.g., health care)*. This could be caused by: (i) a correlation between the baseline exposure

to *x* and *z* and a non-linear damage function[99] or (ii) a heterogeneous damage function across *z*.[100] *z* is the causal mechanism behind *x*'s heterogeneous effects across *z* only under the latter. Their method requires exogenous variation in weather, the potential source of heterogeneity (e.g., health care), and orthogonality between the two. The research design used in this paper guarantees that temperature shocks are orthogonal to Medicaid and air conditioning penetration rates.[101]

The empirical strategy is presented below in three sections. First, I introduce the model used to estimate the effects of in-utero temperature on birthweight. Second, I present the interaction and event study models to estimate how Medicaid's expansion changed the temperature-birthweight relationship. Third, I present the interaction model to estimate how residential air conditioning penetration rates contributed to mitigating temperature impacts on birth outcomes.

3.4.1 Effect of in-utero temperature on birthweight

Broadly, the empirical strategy relies on using unexpected temporal variation in temperature. To do so, I exploit temperature variation in the county of residence across weeks of conception within the same calendar year net of each county's seasonal variation per calendar week. The model presented below estimates the effects of in-utero temperature on birth outcomes.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{cwr} + \sum_{k=1}^{3} \sum_{j=1, j\neq 3}^{5} \delta_{jk} T_{cwy}^{jk} + \theta T_{c\, w-k\, y} + \gamma R_{cwy} + \beta X_{cwyr} + u_{cwyr} \quad [3.1]$$

[99] For example, access to healthcare could be negatively correlated with the mean temperature, and temperature's damage function could be convex.
[100] For example, the health impacts may be lower when individuals access health care.
[101] Explained in greater detal below. Since Medicaid and Air conditioning penetration rates vary at the state-year level in [3.2], the variation used to identify the effects of temperature is orthogonal to both of them.

Where y_{cwyr} corresponds to the mean birth outcome in county c, for a pregnancy started during calendar week w of year y from women of race r. α_{cyr} corresponds to county-race-year fixed effects. Thus, the effects are identified using in-utero weather variation across weeks of conception in the same county, year, and race. In addition, relative to county-race fixed effects, using α_{cyr} limits the potential bias caused by migration driven by environmental quality concerns.[102] α_{cwr} corresponds to seasonal effects by county, week, and race. Thus, the estimates won't be biased by any temporal sorting in conceptions along the year. This is a crucial concern in this literature, because temperature variation is higly stational and previous research has documented seasonal patterns in mother's demographics (Buckles & Hungerman, 2013;Wilde et al., 2014); furthermore, other unobservables determinants of health could also be correlated with the season of conception (Bodnar & Simhan., 2008; Currie & Schwandt., 2013).

T_{cwy}^{jk} corresponds to the number of days during the k-th trimester (13-week window) in which a county's daily temperature fell in the j-th bin. Five bins, described in section 3.2, were used. The number of days in the 45-65 F range of temperaure corresponds to the omitted category. In-utero rainfall vector, R_{cwy}, was defined analagously, but using five bins- each of them corresponding to a quintile. Similar models have been widely used to estimate the causal impact of temperature on health outcomes (e.g., Deschênes et al., 2009; Dell et al., 2014). $T_{c\,w-k\,y}$ corresponds to a vector of lagged mean temperatures; I used the mean weakly average for the first four lagged weeks relative to the week of conception, and the monthly average from (lagged) months 2- 7.[103] This

[102] Previous research has shown that higher-income households migrate in response to environmental quality in the US (Crowder & Downey, 2010; Pais, Crowder & Downey, 2014).
[103] Disaggregating the means from the 2nd-6th months into weekly means does not change the results.

vector was included because previous research suggests that lagged temperature affects fertility (Barreca et al., 2018) and first-trimester estimates were sensitive to includding temperature lags.

Lastly, some additional controls by county, race, and week of conception cells are included to improve the precision of the estimates. X_{cwyr} correspond to the mean of a vector of individual-level controls from the birth certificates (age of the mother, pregnancy history, marital status, high school dropout, newborn's gender).[104] The standard errors are clustered at the county level, and regressions are weighted by the number of births in each cell.

3.4.2 Impact of access to prenatal care on the effects of temperature.

This section describes the empirical strategy to estimate the impact of Medicaid's expansion on temperature's effects on birth outcomes. The empirical strategy relies on the interaction term of two non-constant variables in a panel data model. The interpretation of the effects of such interaction can be challenging because the coefficient(s) of interest could contain multiple sources of variation (e.g., between and within variation). While describing heterogeneous effects could be done by simply interacting the variables of interest (i.e., Medicaid and temperature), drawing causal inferences may require adding multiple interaction terms to isolate the variation with a causal interpretation. Section 2.5.1 (previous chapter) discusses this in greater detail.

The impact of Medicaid's expansion on the effects of temperature on birthweight is estimated by ϕ in the following model.

[104] These demographics were not affected by weather. Results not shown, available upon request.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{cwr} + \sum_{k=1}^{3}\sum_{j=1,j\neq 3}^{5}\left(\phi_{jk}T_{cwy}^{jk} * D_{sy}\right) + \eta\left(T_{c\,w-k\,y} * D_{sy}\right) + \sum_{k=1}^{3}\sum_{j=1,j\neq 3}^{5}\delta_{jk}T_{cwy}^{jk}$$

$$+ \theta T_{c\,w-k\,y} + \gamma R_{cwy} + \beta X_{cwyr} + u_{cwyr} \quad [3.2]$$

The previous model expands model [3.1] by adding: (i) interaction terms between in-utero temperature bins (T_{cwy}^{jk}) and Medicaid expansion (D_{sy}), and (ii) lagged temperature ($T_{c\,w-k\,y}$) and Medicaid expansion (D_{sy}). D_{sy} is a binary variable equal to one if state *s* expands Medicaid during conception year *y* or has already expanded it;[105] zero otherwise. In this model, the impact of Medicaid's expansion on the temperature-health damage function is identified by comparing the effect associated with a given temperature bin in treated states before the expansion with the effect for the same bin in control and treated states after the expansion. Standard errors are clustered at the state level, and regressions are weighted by the number of births in each cell.

In order to add heterogeneous effects of temperature across years while keeping the number of parameters to be estimated tractable, I switched to temperature bins during the entire pregnancy (T_{cwy}^{j}) instead of by trimesters. The following equation describes the new model. Model [3.3] uses the same cutoffs for temperature and precipitation bins as in [3.2] but uses a single window from conception to 39 weeks. Other control variables are identical.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{cwr} + \sum_{j=1,\neq 3}^{5}\left(\phi_{j}T_{cwy}^{j} * D_{sy}\right) + \eta\left(T_{c\,w-k\,y} * D_{sy}\right)$$

$$+ \sum_{l=1974}^{1988}\sum_{j=1,j\neq 3}^{5}\left(\delta_{jl}T_{cwy}^{j}\right) + \theta T_{c\,w-k\,y} + \gamma R_{cwy} + \beta X_{cwyr} + u_{cwyr} \quad [3.3]$$

[105] In particular, D_{sy}=1 always for control states (i.e, stated that had expanded Medicaid before 1980)

Lastly, equation [3.4] presents the event study for Medicaid's impact on the temperature-birthweight relationship by trimesters. Control states (i.e., states that did not expand Medicaid during the 1980s) were excluded from the sample to estimate this model.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{cwr} + \sum_{\tau=-5,\tau\neq-1}^{\tau=5,} \sum_{k=1}^{3} \sum_{j=1,j\neq3}^{5} \left(\phi_{jk\tau} T_{cwy}^{jk} * 1(y - e_s^* = \tau) \right) + \eta\left(T_{c\,w-k\,y} * D_{sy}\right)$$

$$+ \sum_{k=1}^{3} \sum_{j=1,j\neq3}^{5} \delta_{jk} T_{cwy}^{jk} + \theta T_{c\,w-k\,y} + \gamma R_{cwy} + \beta X_{cwyr} + u_{cwyr} \quad [3.4]$$

Following the empirical strategy from East et al., (2023), $1(y - e_s^* = \tau)$ correspond to a series of dummy variables 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_s^*). The year before each state's large expansion ($\tau = -1$) is omitted, so the estimated ϕs are relative to the effects of each temperature bin in the year before the expansion occurred (δ). For example $\phi_{65-85,k,1}$ corresonds to the effect of days between 65 and 85 F during the k-th trimester one year after the discrete change in eligibility, relative to one year before the jump. Similarly, $\phi_{65-85,k,0}$ corresponds to the effect on the year of the jump in eligibility, relative to one year before the jump[106].

[106] Event time obeservations that are more than five year before or after the event were binned together. I estimate but do not report these estimates, because they are based on an unbalanced sample. Binning allows to separately identify treatment effects from secular time trends even when control states are not included in the model (Schmidheiny & Siegloch, 2019).

3.4.3 Impact of air conditioning on the effects of temperature.

This section presents the empirical strategy to analyze how the effects of temperature change with residential air conditioning (AC) ownership.

In model [3.5], ϵ_j and ω_j capture the change in the effects of temperature's j-th bin when residential air conditioning ownership is below or above each state's median.

$$y_{cwyr} = \alpha_{cyr} + \alpha_{cwr} + \left(\sum_{j=1, j\neq 3}^{5} \epsilon_j T^j_{cwy} + \eta T_{c\, w-k\, y} \right) * 1\left(AC_{sy} \leq \widetilde{AC_s}\right) * \left(AC_{sy} - \widetilde{AC_s}\right)$$

$$+ \left(\sum_{j=1, j\neq 3}^{5} \omega_j T^j_{cwy} + \phi T_{c\, w-k\, y} \right) * 1\left(AC_{sy} > \widetilde{AC_s}\right) * \left(AC_{sy} - \widetilde{AC_s}\right) + \sum_{l=1974}^{1988} \sum_{j=1, j\neq 3}^{5} \left(\delta_{jl} T^j_{cwy}\right) + \theta T_{c\, w-k\, y}$$

$$+ \gamma R_{cwy} + \beta X_{cwyr} + u_{cwyr} \quad [3.5]$$

In the previous equation, $\widetilde{AC_s}$ corresponds to the median residential air conditioning ownership in state s. $1\left(AC_{sy} \leq \widetilde{AC_s}\right)$ and $1\left(AC_{sy} > \widetilde{AC_s}\right)$ are dummy variable equal to one when residential air conditioning ownership in state *s* and year *y* is below and above the state's median, respectively.

The previous model builds upon the empirical strategy used by Barreca et al. (2016) to estimate the impact of air conditioning on the temperature-mortality relationship in the US during the twentieth century. As discussed by these authors, the variation in AC diffusion is not experimental. It may not be orthogonal to the temperature-health relationship. For instance, AC diffusion may be higher in places where temperature has a more significant health impact. For this model, the biggest threat to identification would be AC diffusion to increase in years where the impacts of temperature on health are also increasing. It would lead to underestimating the impacts of AC on

the temperature-birth outcome relationship. However, as Barreca et al. (2018) argued, this concern is mitigated by allowing for heterogeneous effects of each temperature bin across years.

3.5 Results

Table 3.2 shows how the estimates of the effects of temperature on birthweight change when controls (rainfall, demographics, lagged temperatures, and lagged rainfall) are sequentially added to the regression model. Column 4 in Table 3.2 corresponds to the baseline empirical strategy described in model [3.1]. The contrast between columns 3 and 4 in Table 3.2 shows that controlling for lagged temperature is important for the first trimester estimates. On the other hand, controlling for lagged precipitation has no impact on the estimates.

Table 3.3 estimates temperature's effects on birthweight, the low birthweight rate, weeks of gestation, and preterm birth rate using model [3.1]. The results show that high temperatures during the second and third trimesters decrease birthweight and increase the fraction of infants born with less than 2500 grams. The impacts are larger for exposures during the second trimester and increase with temperature. One additional day above 85 F during the second and third trimester reduces birthweight by 0.013% and 0.00865%, respectively, relative to days between 45-65 F. Deschenes et al.(2009) study temperature's impacts with a similar empirical strategy for the US during 1972-1988 and find impacts on birthweight between 0.003% and 0.009% for hot days. On the other hand, the gestational age and preterm birth rate are affected mainly by extreme temperatures, both heat and cold, during the first trimester. Days above 85 F during the third trimester also decrease the pregnancy's duration. One additional day in this range decreases the weeks of gestation by

0.39%. The birthweight and gestational age estimates are displayed graphically in Figures 3.4 and 3.5, respectively.

Table 3.4 estimates the effects of temperature on birthweight separately for whites, black, cold and warm counties, and metropolitan counties.[107] These estimates correspond to model [3.1] across different samples. The effects of extremely hot days (i.e., T>85 F) are larger for blacks than whites. For moderately hot days (i.e., 65-85 F), the impacts are larger in cold counties compared to warm. This result is consistent with the idea that warmer counties are better adapted to heat. However, the impacts are larger for days above 85 F in warm counties. Lastly, we observe that metropolitan counties drive the effects in the US. Therefore, the mechanism behind these effects is most likely physiological. Indirect income shocks through the agricultural sector are significant in developing countries or rural areas.

Table 3.5 estimates the effects of temperature on birthweight and gestational age separately for whites, black, cold and warm counties, and metropolitan counties. However, the effects are estimated for the entire pregnancy (i.e., from conception to 39 weeks) instead of by trimesters. The estimates are consistent with the findings in Table 3.4 except for the estimate of the effect of days with T>85 F on the gestational age. This counterintuitive sign should not be believed because the average pregnancy is exposed to this temperature range for only 0.26 days in cold counties (See Figure 3.2). Table 3.5 is a baseline for the empirical strategy to estimate the impacts of Medicaid and air conditioning on the temperature-birth outcome relationship. Both strategies require

[107] Cold (Warm) counties corresponds to counties whose average temperature during 1970-1988 belongs to the bottom (top) 33% of the distribution. Metropolitan counties corresponds to Metropolitan counties according with USDA's 1986 County Typology Codes.

heterogeneous effects of temperature bins across years. However, the number of parameters to be estimated is vast when the effects of temperature are disaggregated by trimester.

Table 3.6 estimates Medicaid's impact on the temperature-birthweight and temperature-gestational age damage functions. The table shows the estimates of ϕ *and* δ in [3.2].These results suggest that Medicaid's expansion increased temperature damage on birth outcomes. For instance, the impact of days between 65-85 F during the second trimester on birthweight would have increased by 0.0057%. These counterintuitive results could be caused by another factor not included in [3.2]. For instance, suppose another factor mediates the impact of temperature on birth outcomes (e.g., income shocks). If, by coincidence, Medicaid changes are correlated with changes in this unobserved factor, ϕ would be biased. To account for this possibility, we should include heterogeneous effects of temperature across years. However, the number of parameters grew too much, and the model could not be estimated due to computational constraints.

Nonetheless, in Table 3.7, I present the results of model [3.3], which estimates the impacts on temperature bins during the entire pregnancy instead of by trimesters, and it includes heterogeneous effects of each temperature bin across years. Medicaid's impact is close to zero and statistically insignificant. Table 3.8 presents the estimates model [3.3] across different samples (whites, blacks, cold, warm, and metropolitan counties). The results show that Medicaid did not mitigate the effects of extreme temperatures on any demographic or subset of counties. The estimates for blacks and warm counties turn statistically insignificant when the sample is restricted to treated states only[108]. The results for the gestational age are qualitatively similar.[109]

[108] Results not shown, available upon request.
[109] Results not shown, available upon request.

Figure 3.6 presents the results from an event study of Medicaid's impact on the temperature-birthweight damage function. This event study corresponds to model [3.4]. In line with previous results, this event study shows no robust evidence that Medicaid's expansion mitigated the impacts of extreme temperatures on birth weight. Additionally, there seems to be a negative trend in the effects of days between 65 and 85 F during all trimesters before Medicaid's expansion; Therefore, allowing for heterogeneous effects of temperature bins across years, as done in [3.4], is a critical piece of this paper's empirical strategy. Similar results were obtained for the gestational age; these graphs were not presented but are available upon request.

Finally, Table 3.9 and Figures 3.7 and 3.8 present the estimates of the impact of air conditioning on the temperature-birthweight and temperature-gestational age relationships. These results suggest that increased access to air conditioning mitigates the impacts of extreme heat on birth outcomes. This result is consistent with research suggesting that AC diffusion in the US after the 1960s mitigated the impact of extreme heat on the mortality rate (Barreca et al., 2016) and with research showing that US electricity consumption increases with daily temperatures above 90 F (Deschênes & Greenstone, 2011)

The first graph of Figure 3.7 shows the impact of AC on the temperature-birthweight relationship for the entire US. We observe that an increase in AC was associated with an increase in the effect of temperature on birthweight (i.e., a reduction of temperature's negative effect), although not statistically significant at standard levels. However, for whites, the impact is statistically significant. An increase of 30 percentage points (pp) in AC is associated with an increase in

birthweight of about 0.1%. This increase is caused by a reduction in the effect of days with T>85 F. For blacks, AC mitigates the impacts of days between 65 and 85 F, a 30 pp increase in AC leads to an increase in birthweight of about 0.033%.

Figure 3.8 plots the estimates of AC diffusion on the temperature-gestational age relationship. For the entire US, the first graph shows that a 35 pp increase in AC is associated with an increase of about 0.02 weeks in the duration of the pregnancy. This increase is caused by a reduction in the impact of days with T>85. For whites and blacks, the impact of a 35 pp point increase is about 0.02 and 0.03 weeks, respectively. Both are associated with mitigation in the effects of days with T>85F. There is, however, an important difference between the estimates for blacks and whites. For whites, the impact is associated with years in which the AC penetration rate was below the state's median; for blacks, it is associated with years in which it was above. This suggests that the pattern of AC diffusion may be quite heterogeneous across races. The population of whites most affected by extreme heat may have gotten access to AC at lower aggregate penetration rates. In contrast, the blacks most affected by heat may have gotten access only after high aggregate penetration rates were achieved.

3.6 Conclusions

The results from the empirical analysis show that extreme temperatures worsen birth outcomes. Increased access to prenatal health care (i.e., prenatal eligibility for Medicaid) did not mitigate its effects, but increased access to air conditioning did.

Birthweight is affected primarily by extreme heat during the second and third trimesters. In contrast, gestational age is affected mainly by extreme temperatures, both hot and cold days, during the first trimester. These effects hold strongly in urban areas, which suggests that physiological impacts are the mechanism behind these effects. In contrast, previous research suggests that income shocks through agricultural output may be a mechanism in other settings in developing countries.

As a first result, I found no evidence that Medicaid's expansion mitigated the effects of temperature on either birthweight or gestational age. Previous experimental medical research found that improved nutrition before pregnancy mitigated in-utero extreme heat's effects on birth outcomes. Thus, ex-ante Medicaid's expansion could make the fetus more resilient to extreme temperature shocks. However, this is not the case for the US. Interestingly, the event study showed an increasing pattern in the effects of temperature between 65 and 85 Fahrenheit (F). Failing to account for this pattern causes the estimate of Medicaid's impact to be biased. When heterogeneous effects of temperature across years are excluded from the empirical strategy, I find that Medicaid's expansion increased the marginal damage caused by days between 65-85 F or above 85 F.

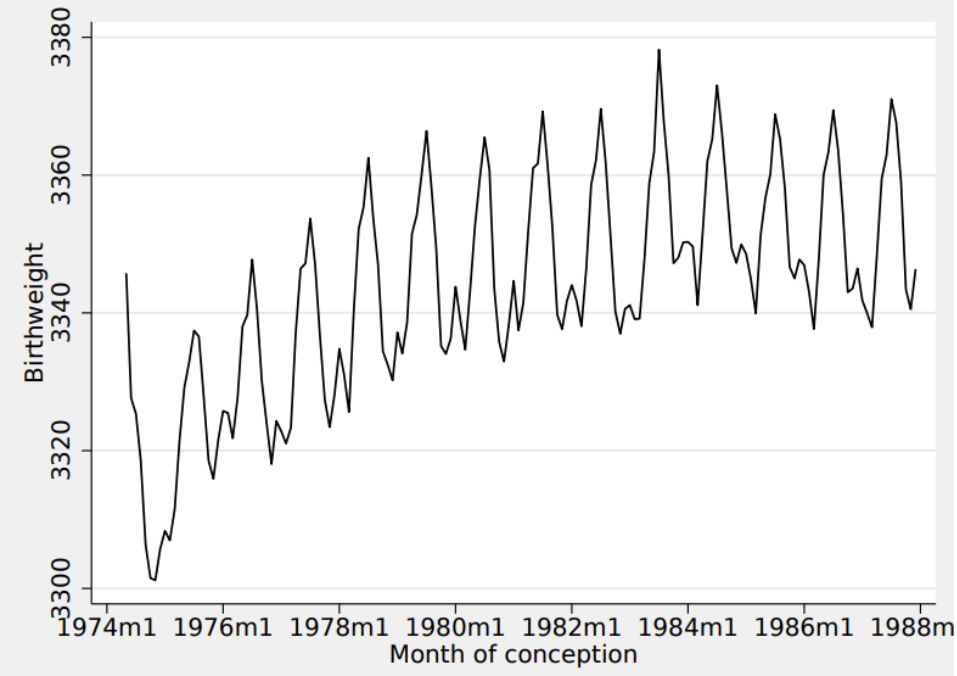
The heterogeneity analysis revealed that Medicaid did not mitigate temperature effects for either whites or blacks. It was also ineffective in Mitigating these effects in counties with cold or warm climates or metropolitan counties.

As a second result, I found that the diffusion of residential air conditioning during this period contributed to reducing the effects of extreme heat on birthweight and the duration of the pregnancy. This result is consistent with research suggesting that AC diffusion in the US after the 1960s mitigated the impact of extreme heat on the mortality rate (Barreca et al., 2016) and increased electricity consumption with days above 90 F (Deschênes & Greenstone, 2011).

The findings from this paper suggest that expanding access to free prenatal care to low-income women did not help to mitigate the impact of extreme temperatures in a setting where physiological mechanisms drive the impacts of extreme temperatures on birth outcomes (i.e., in a developed country and urban setting). In contrast, increased access to air conditioning effectively mitigated the effects of extreme heat on birth outcomes. These findings can guide policymakers in designing strategies to adapt to climate change.

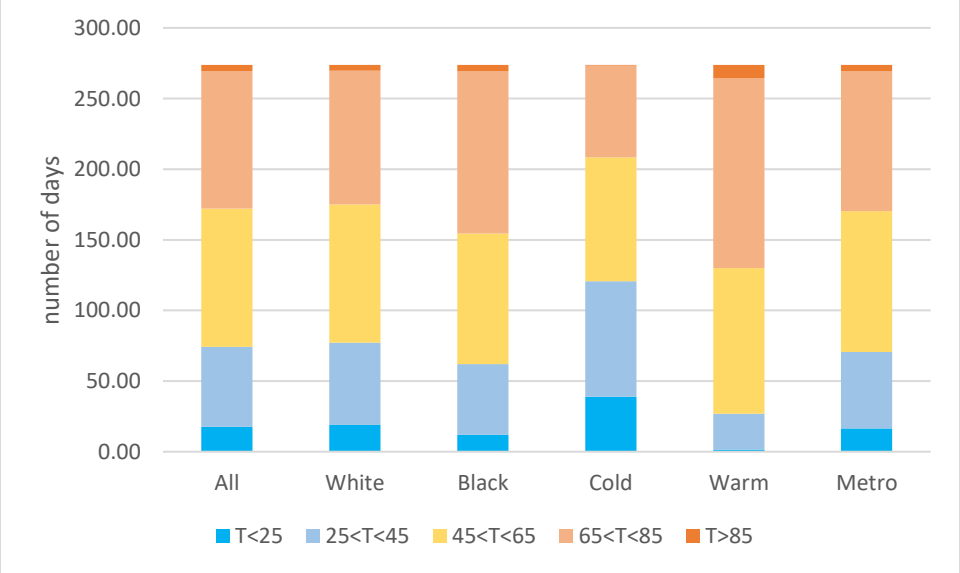
3.7 Figures

Figure 3.1: Birthweigth



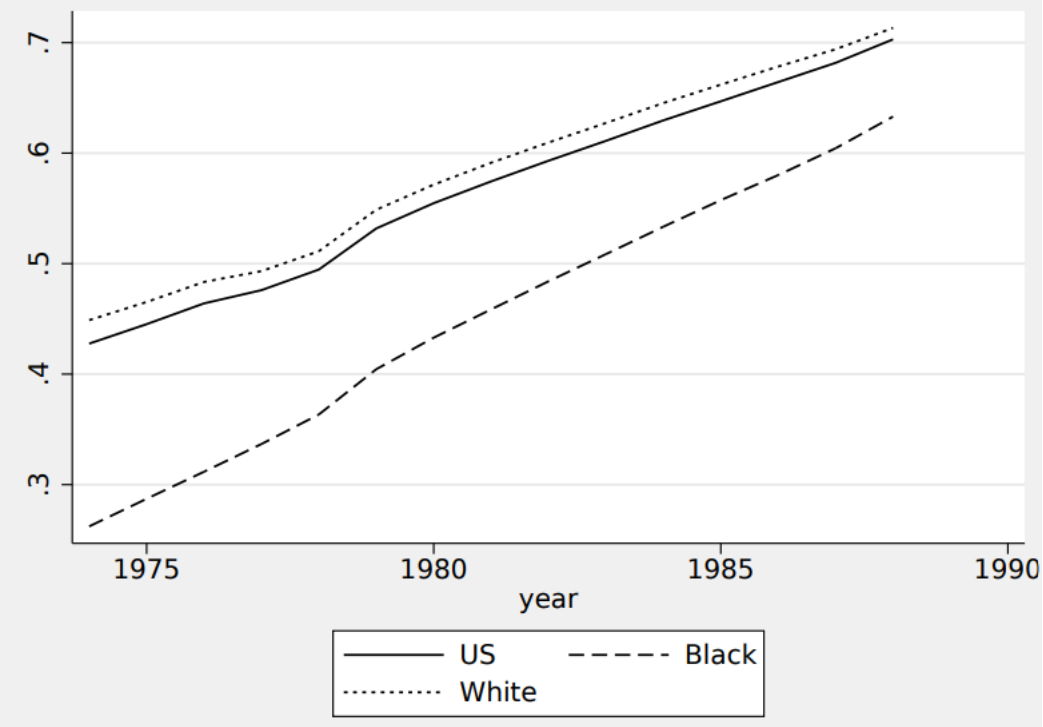
Notes: Author's calculations from CDC public birth files 1975-1988.

Figure 3.2: Distribution of in-utero temperature across different samples



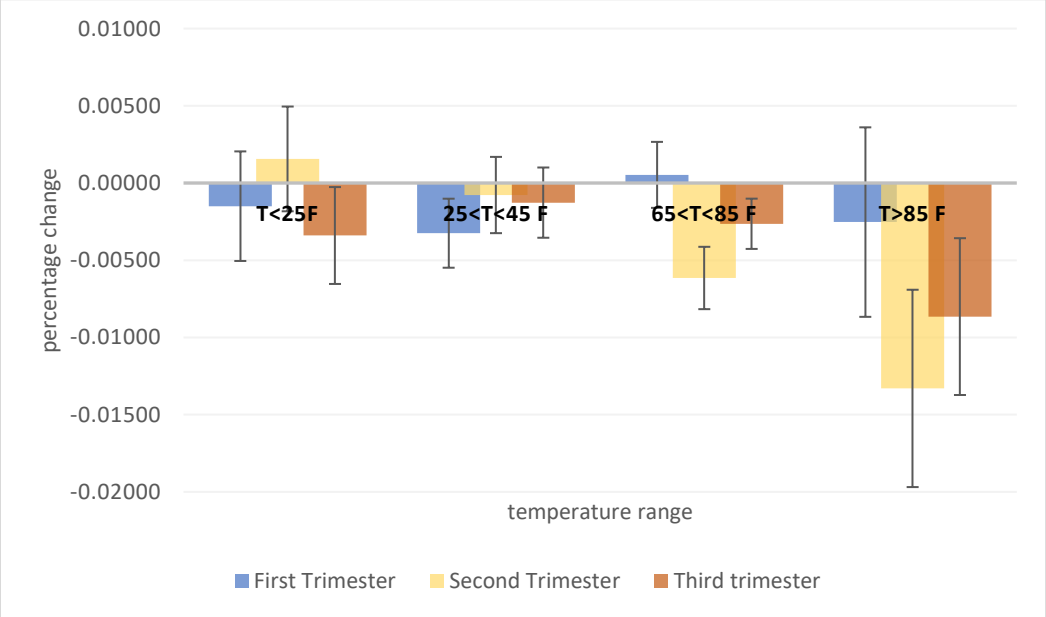
Notes: Author's calculations from CDC public birth files 1975-1988.

Figure 3.3 : Residential Air Conditioning Ownership



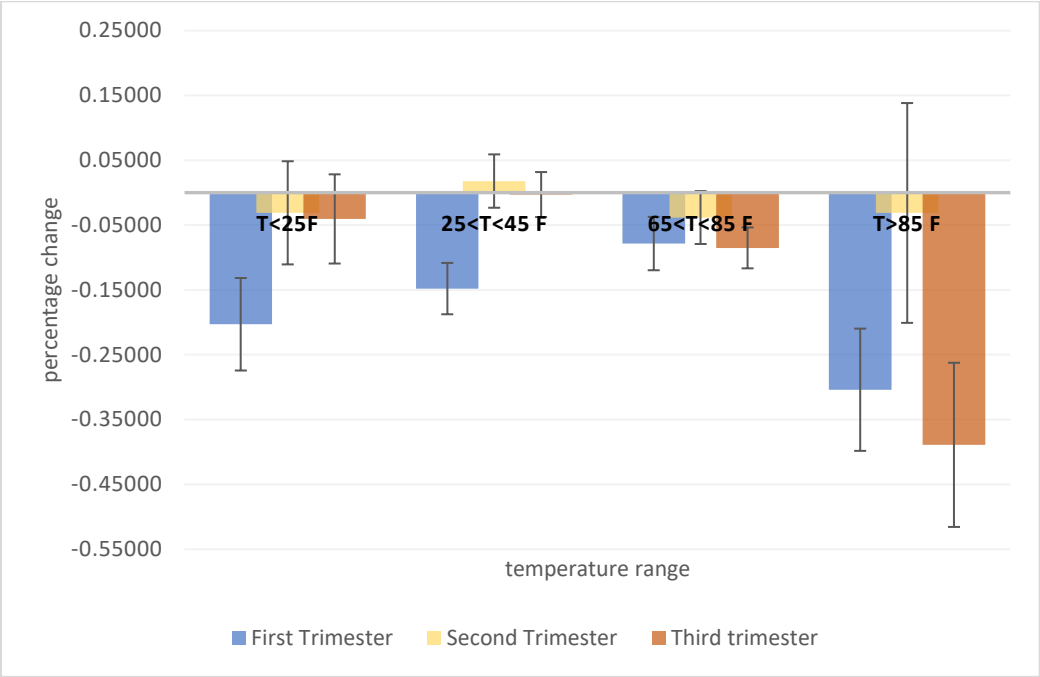
Notes: Author's calculations from Census data.

Figure 3.4: Effects of temperature on birthweight



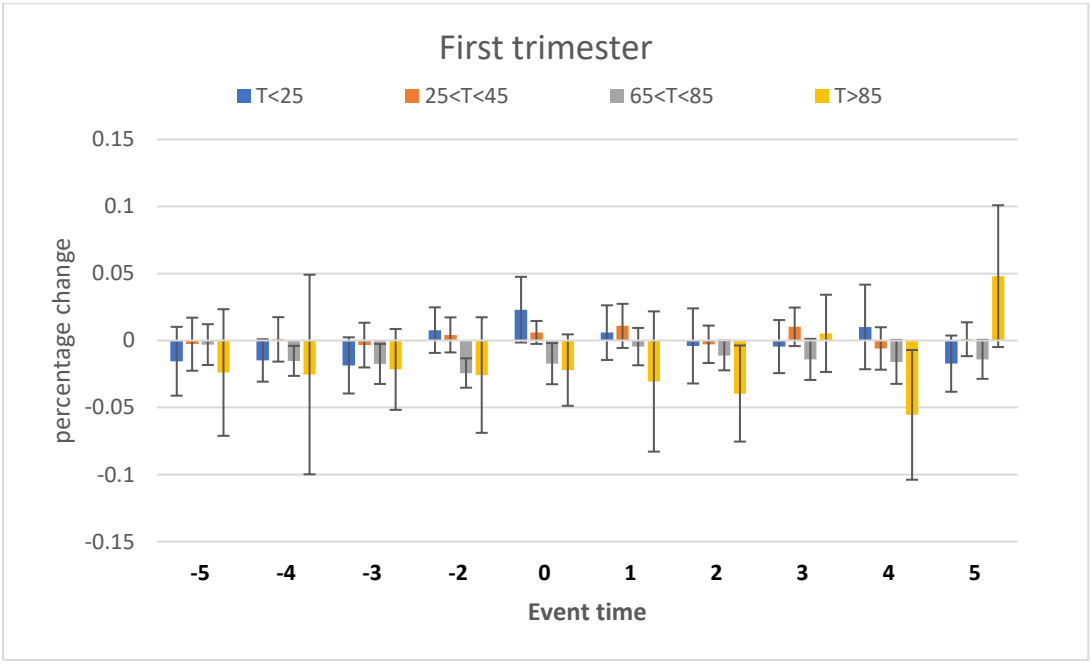
Notes: Author's calculations from CDC public birth files 1975-1988. This graph plots the effects of 1 additional day during the first, second, and third trimester in each temperature bin with respect to the 45-65 F range. These estimates are reported in Col 1 of Table 3.3.

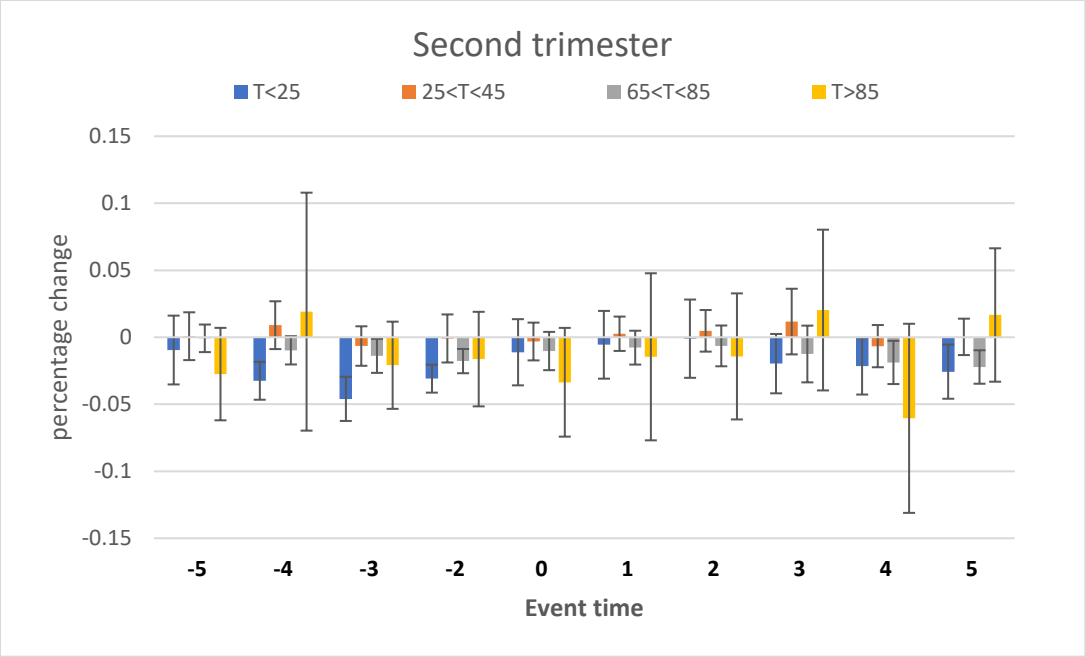
Figure 3.5: Effects of temperature on the gestational age

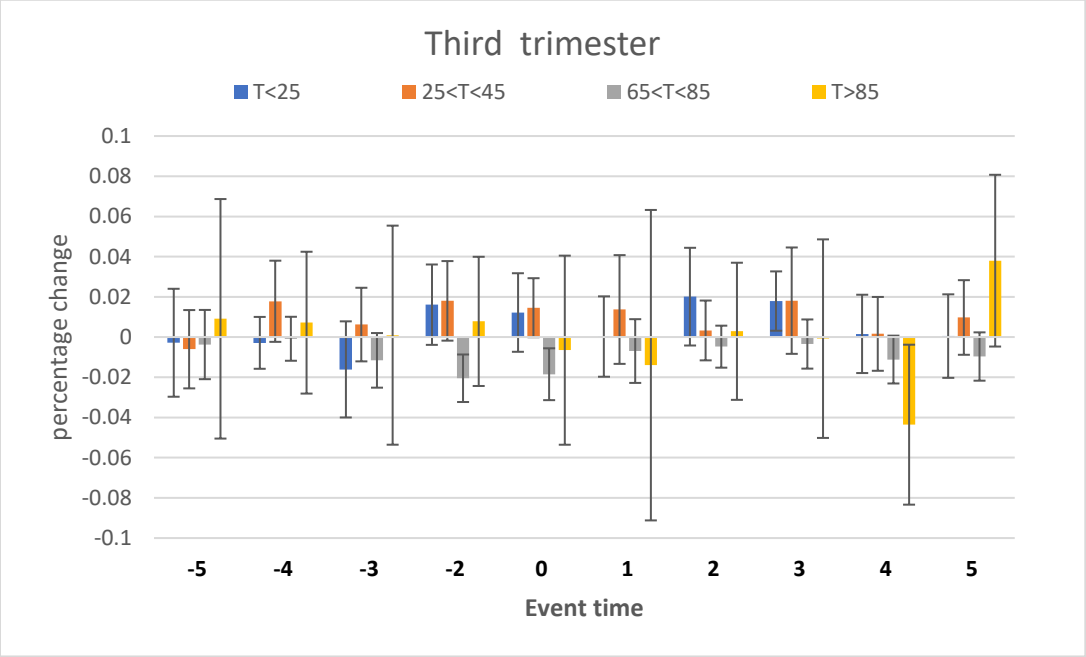


Notes: Author's calculations from CDC public birth files 1975-1988. This graph plots the effects of 1 additional day during the first, second, and third trimester in each temperature bin with respect to the 45-65 F range. These estimates are reported in Col 3 of Table 3.3.

Figure 3.6: Event study for Medicaid's expansion impact on the temperature-birthweight relationship

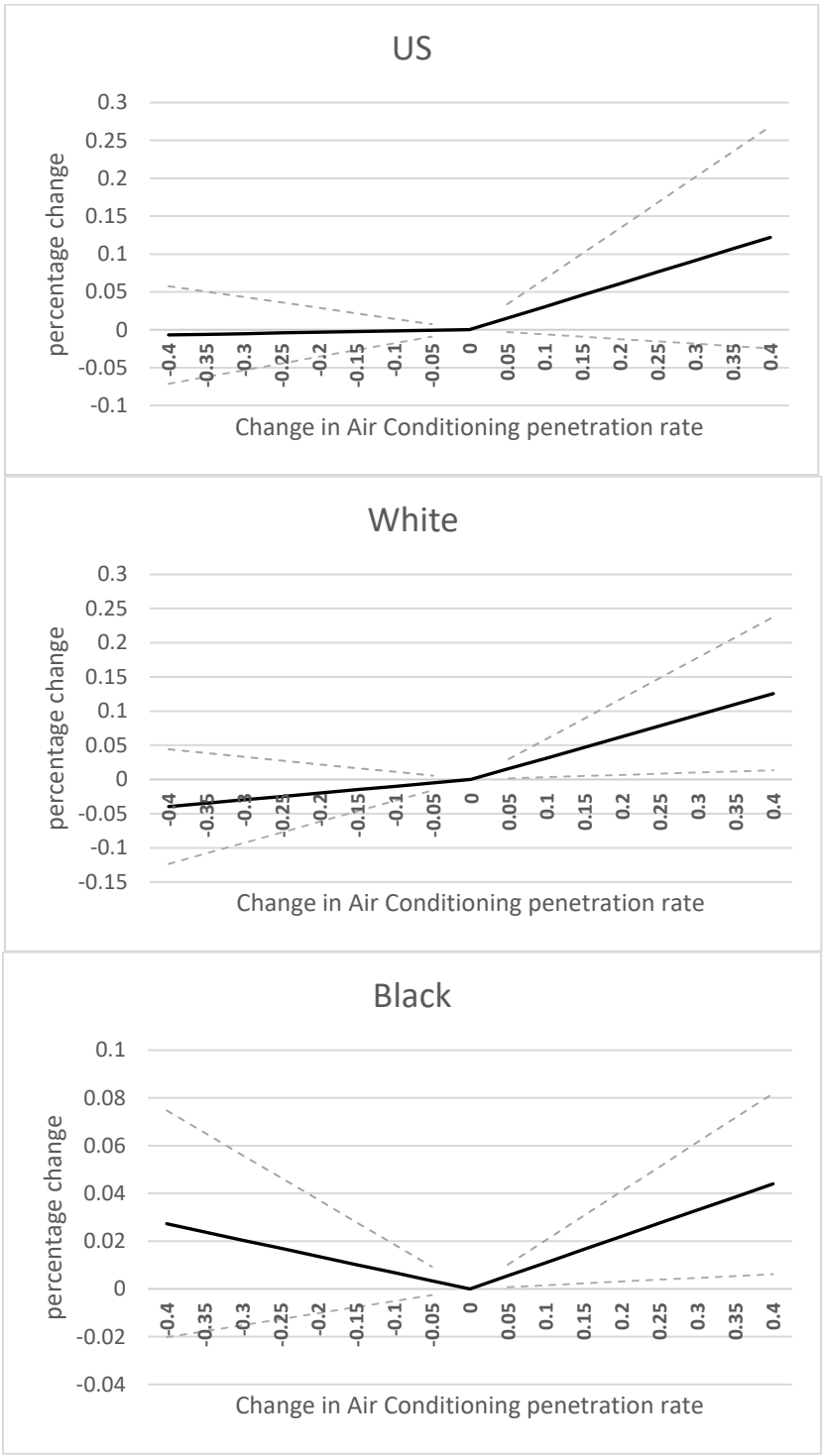






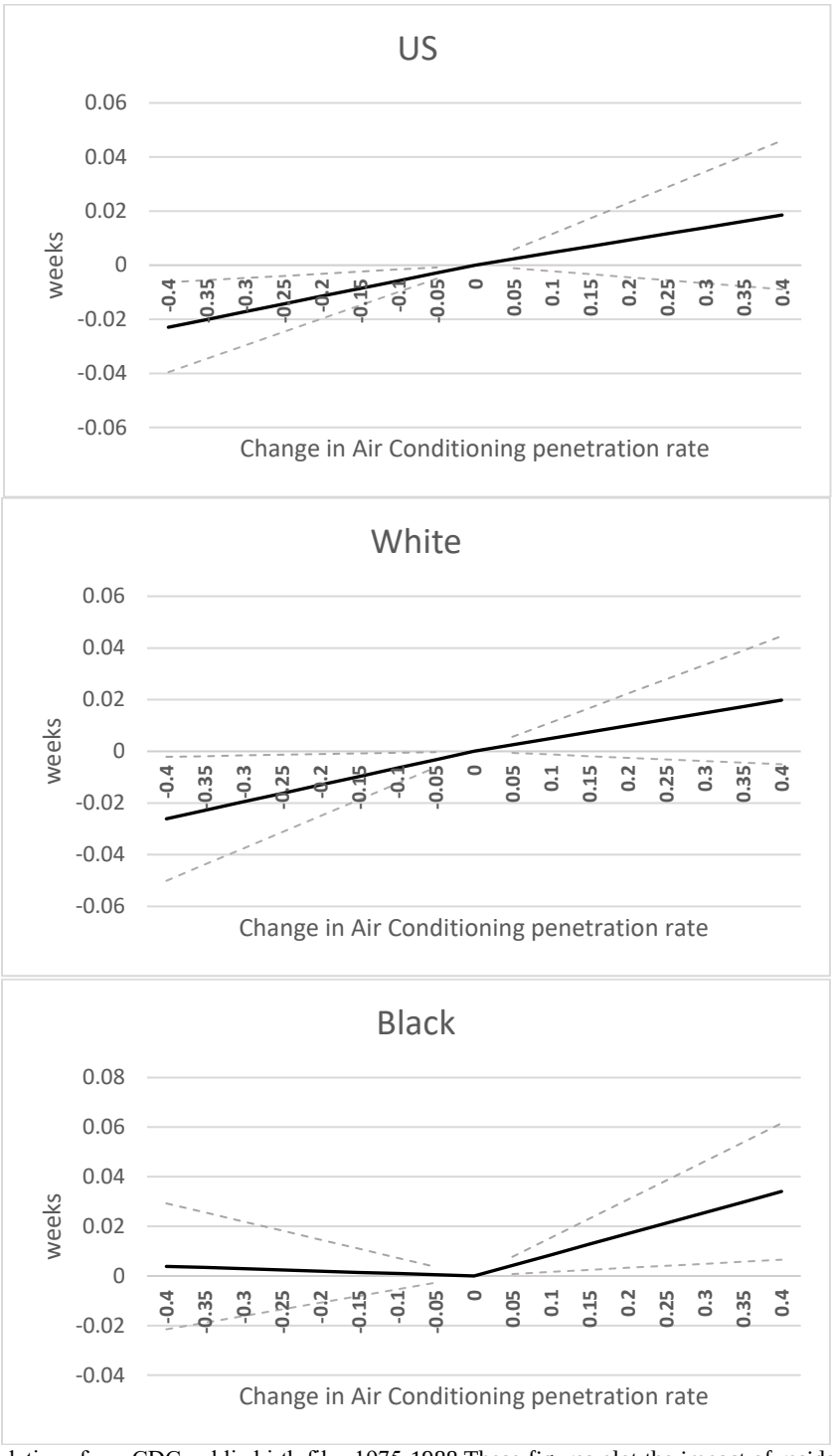
Notes: Author's calculations from CDC public birth files 1975-1988. These graphs plot the impact of Medicaid's expansion on the effects of 1 additional day during the first, second, and third trimester in each temperature bin with respect to the 45-65 F range. All event study estimates are relative to t=-1. Estmates for $t \leq -6$, $t \geq 6$ were included in the regression model but are not displayed. Control states (i.e. states who expaned Medicaid prior to 1980) were excluded from the sample. Only states from cohorts of expansion between 1980-1983 were included in the event study.

Figure 3.7: Impact of Air Conditioning on the temperature-birthweight relationship



Notes: Author's calculations from CDC public birth files 1975-1988. These figures plot the impact of residential air conditioning ownership (AC) on the effects of temperature on birthweight. For the US and whites, the figures plot the 95% confidence intervals (CI) for AC impact on days with T>85 F. For blacks, the figure plots the 90% CI for the impact on days with 65<T<85 F. These graphs plot the estimates from columns 1-3 of Table 3.9.

Figure 3.8: Impact of Air Conditioning on the temperature-gestational age relationship



Notes: Author's calculations from CDC public birth files 1975-1988.These figures plot the impact of residential air conditioning ownership (AC) on the effects of temperature on the gestational age. All figures plot the 95% confidence intervals (CI) for AC impact on days with T>85 F. These graphs plot the estimates from columns 4-6 of Table 3.9.

3.8 Tables

Table 3.1: Summary statistics

	US	White	Black	Cold County	Hot County	Metropolitan County
N births	42,039,568	34,415,283	6,329,139	13,235,743	16,448,708	31,532,601
Birth Outcomes						
Birthweight (grams)	3349.46	3396.10	3108.12	3368.82	3327.60	3345.15
Log birthweight	8.10	8.11	8.01	8.10	8.09	8.09
Low birthweight infants per thousand births	66.74	55.97	126.08	62.33	71.42	67.46
Gestational age (weeks)	39.44	39.60	38.62	39.53	39.34	39.42
Demographics of the mother						
race white (%)	81.86%	1.00%	0.00%	88.48%	74.71%	80.67%
race black (%)	15.06%	0.00%	1.00%	9.26%	21.26%	16.07%
race other (%)	3.08%	0.00%	0.00%	2.25%	3.68%	3.26%
share age<=19 (%)	14.45%	12.41%	26.48%	12.39%	16.65%	13.58%
share 20<=age<=35 (%)	81.65%	83.66%	70.34%	83.79%	79.54%	82.32%
share age>=36 (%)	3.91%	3.92%	3.18%	3.82%	3.82%	4.10%
Marital status						
Married	68.11%	73.73%	36.41%	69.36%	67.12%	67.74%
Unmarried	16.03%	10.33%	47.25%	13.49%	18.65%	16.82%
Not Reported	15.86%	15.94%	16.34%	17.15%	14.23%	15.43%
Highschool dropout (%)						
No	61.15%	63.15%	55.27%	77.82%	41.60%	58.97%
Yes	17.79%	15.48%	30.71%	18.00%	16.79%	15.89%
Not Reported	2.53%	2.67%	1.41%	0.48%	4.72%	3.12%
Not Answered	18.54%	18.70%	12.61%	3.70%	36.89%	22.02%
Pregnancy history (%)						
First delivery	36.10%	36.66%	33.24%	34.41%	37.24%	36.43%
2nd delivery & fetal death rate =0	26.23%	26.82%	23.15%	25.70%	26.74%	26.10%
delivery>=3 & fetal death rate =0	18.67%	17.76%	23.13%	18.35%	20.00%	18.27%
delivery>=3 & fetal death rate <0.5	10.58%	10.40%	11.71%	12.26%	8.89%	10.45%
delivery=2 & fetal death rate =1	4.58%	4.66%	4.34%	5.16%	3.75%	4.71%
delivery>=3 & fetal death rate>0.5	3.17%	3.16%	3.30%	3.61%	2.49%	3.35%
Missing	0.66%	0.54%	1.14%	0.51%	0.89%	0.68%
Controls at the county-year level						
Gov transfers (Exc Medical & SSI) per population 0-64 yr. old	127.61	120.43	162.46	140.49	121.34	131.49
UI transfers per population 0-64 yr. old	81.55	82.01	79.11	100.71	62.35	81.87

Notes: Author's calculations from CDC public birth files 1975-1988.

Table 3.2: Selecting model to estimate the effects of temperature on birth outcomes.

	(1)	(2)	(3)	(4)	(5)
Precipitation controls	No	Yes	Yes	Yes	Yes
Demographic controls	No	No	Yes	Yes	Yes
Lagged Temperature	No	No	No	Yes	Yes
Lagged precipitation	No	No	No	No	Yes
R-squared	0.382	0.382	0.392	0.392	0.393
Observations	42,001,350	42,001,350	42,001,350	42,001,350	42,001,350
Temp-Q1(T<25F)	-2.52e-06	-1.13e-05	-4.94e-05***	-1.50e-05	-1.43e-05
	(1.33e-05)	(1.37e-05)	(1.42e-05)	(1.81e-05)	(1.85e-05)
Temp-Q1(25<T<45F)	-4.05e-05***	-4.28e-05***	-5.03e-05***	-3.25e-05***	-3.15e-05***
	(9.45e-06)	(9.56e-06)	(9.75e-06)	(1.14e-05)	(1.15e-05)
Temp-Q1(65<T<85F)	1.04e-05	7.00e-06	2.82e-05***	5.28e-06	4.60e-06
	(7.44e-06)	(7.56e-06)	(7.74e-06)	(1.09e-05)	(1.10e-05)
Temp-Q1(T>85F)	3.77e-06	-9.32e-06	1.69e-05	-2.53e-05	-2.68e-05
	(2.89e-05)	(3.01e-05)	(2.84e-05)	(3.13e-05)	(3.17e-05)
Temp-Q2(T<25F)	3.09e-05**	2.18e-05*	-1.66e-05	1.56e-05	1.82e-05
	(1.21e-05)	(1.23e-05)	(1.27e-05)	(1.73e-05)	(1.73e-05)
Temp-Q2(25<T<45F)	-2.32e-05**	-2.61e-05***	-2.42e-05**	-7.78e-06	-7.06e-06
	(9.57e-06)	(9.88e-06)	(1.02e-05)	(1.26e-05)	(1.27e-05)
Temp-Q2(65<T<85F)	-4.31e-05***	-4.77e-05***	-4.30e-05***	-6.15e-05***	-6.09e-05***
	(7.77e-06)	(8.24e-06)	(8.49e-06)	(1.03e-05)	(1.03e-05)
Temp-Q2(T>85F)	-0.000109***	-0.000122***	-9.83e-05***	-0.000133***	-0.000136***
	(3.05e-05)	(3.28e-05)	(3.04e-05)	(3.26e-05)	(3.35e-05)
Temp-Q3(T<25F)	2.00e-05	1.34e-05	-3.60e-05***	-3.40e-05**	-3.02e-05*
	(1.26e-05)	(1.33e-05)	(1.37e-05)	(1.60e-05)	(1.60e-05)
Temp-Q3(25<T<45F)	4.80e-06	2.61e-06	-1.14e-05	-1.27e-05	-1.18e-05
	(9.97e-06)	(9.94e-06)	(1.02e-05)	(1.16e-05)	(1.16e-05)
Temp-Q3(65<T<85F)	-2.86e-05***	-3.30e-05***	-2.24e-05***	-2.64e-05***	-2.63e-05***
	(7.33e-06)	(7.57e-06)	(7.79e-06)	(8.30e-06)	(8.50e-06)
Temp-Q3(T>85F)	-9.20e-05***	-0.000107***	-7.87e-05***	-8.65e-05***	-8.94e-05***
	(2.44e-05)	(2.48e-05)	(2.56e-05)	(2.59e-05)	(2.65e-05)

Notes: Author's calculations from CDC public birth files 1975-1988.The dependent variable corresponds to Log(bw): Natural logarithm of birthweight. Temp-Qj(x<t<y) corresponds to the number of days during the j-th pregancy trimester in which the county's temperature is between x and y. The omitted category corresponds to days with temperatures between 45-65F . All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-Q1(bins), Rainfall-Q2(bins), Rainfall-Q3(bins), and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarriedIndividual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell.). Standard errors are clustered at the county level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.3: Effects of temperature on birth outcomes.

	(1) Log(bw)	(2) LBW	(3) Gestational age	(4) PTB
Observations	42,001,350	42,001,350	41,907,577	41,907,577
R-squared	0.392	0.244	0.291	0.268
Temp-Q1(T<25F)	-1.50e-05	0.0211	-0.00203***	0.0843***
	(1.81e-05)	(0.0204)	(0.000364)	(0.0298)
Temp-Q1(25<T<45F)	-3.25e-05***	0.0328**	-0.00148***	0.0976***
	(1.14e-05)	(0.0130)	(0.000202)	(0.0175)
Temp-Q1(65<T<85F)	5.28e-06	-0.00968	-0.000785***	0.0402***
	(1.09e-05)	(0.0123)	(0.000210)	(0.0146)
Temp-Q1(T>85F)	-2.53e-05	0.0406	-0.00304***	0.143***
	(3.13e-05)	(0.0356)	(0.000481)	(0.0378)
Temp-Q2(T<25F)	1.56e-05	0.0155	-0.000311	-0.0273
	(1.73e-05)	(0.0192)	(0.000406)	(0.0318)
Temp-Q2(25<T<45F)	-7.78e-06	0.0155	0.000179	0.00876
	(1.26e-05)	(0.0139)	(0.000210)	(0.0180)
Temp-Q2(65<T<85F)	-6.15e-05***	0.0268**	-0.000384*	0.0180
	(1.03e-05)	(0.0131)	(0.000208)	(0.0148)
Temp-Q2(T>85F)	-0.000133***	0.103***	-0.000313	0.0662
	(3.26e-05)	(0.0314)	(0.000865)	(0.0546)
Temp-Q3(T<25F)	-3.40e-05**	0.0458**	-0.000405	-0.0139
	(1.60e-05)	(0.0188)	(0.000351)	(0.0274)
Temp-Q3(25<T<45F)	-1.27e-05	0.0278**	-2.93e-05	-0.00544
	(1.16e-05)	(0.0129)	(0.000177)	(0.0158)
Temp-Q3(65<T<85F)	-2.64e-05***	0.0107	-0.000852***	0.0154
	(8.30e-06)	(0.0114)	(0.000161)	(0.0138)
Temp-Q3(T>85F)	-8.65e-05***	0.0726***	-0.00389***	0.162***
	(2.59e-05)	(0.0254)	(0.000646)	(0.0368)

Notes: Author's calculations from CDC public birth files 1975-1988. Log(bw): Natural logarithm of birthweight. LBW corresponds to the number of low birthweight infants per thousand births. Gestational age corresponds to the newborn's weeks of gestation at birth. PTB corresponds to the number of infants born premature (i.e., gestational age<37 weeks) per thousand births. Temp-Qj(x<t<y) corresponds to the number of days during the j-th pregancy trimester in which the county's temperature is between x and y. The omitted category corresponds to days with temperatures between 45-65F . All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-Q1(bins), Rainfall-Q2(bins), Rainfall-Q3(bins), and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Standard errors are clustered at the county level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.4: Heterogeneous effects of temperature by trimester on birthweight

	(1)	(2)	(3)	(4)	(5)
Sample	Whites	Blacks	Cold counties	Warm counties	Metro counties
Observations	34,414,304	6,316,941	13,221,099	16,438,735	31,525,162
R-squared	0.194	0.181	0.405	0.391	0.536
Temp-Q1(T<25F)	-2.94e-05	4.14e-05	-4.36e-05*	0.000222**	2.41e-05
	(1.91e-05)	(5.99e-05)	(2.65e-05)	(9.29e-05)	(2.23e-05)
Temp-Q1(25<T<45F)	-4.68e-05***	-3.15e-05	-7.62e-05***	-6.83e-06	-8.36e-06
	(1.19e-05)	(3.68e-05)	(1.99e-05)	(2.11e-05)	(1.36e-05)
Temp-Q1(65<T<85F)	7.51e-06	3.26e-05	1.22e-05	-1.32e-05	-4.35e-06
	(1.29e-05)	(3.15e-05)	(2.30e-05)	(2.06e-05)	(1.40e-05)
Temp-Q1(T>85F)	1.04e-05	-8.96e-05	5.73e-05	-5.97e-05	-4.42e-05
	(3.05e-05)	(7.80e-05)	(0.000294)	(4.09e-05)	(3.79e-05)
Temp-Q2(T<25F)	1.85e-05	-3.03e-05	-6.73e-05***	0.000262**	3.99e-05*
	(1.80e-05)	(6.00e-05)	(2.38e-05)	(0.000111)	(2.12e-05)
Temp-Q2(25<T<45F)	-1.31e-05	-5.62e-05	-7.97e-05***	-3.26e-06	9.85e-06
	(1.33e-05)	(3.89e-05)	(1.73e-05)	(2.78e-05)	(1.52e-05)
Temp-Q2(65<T<85F)	-6.12e-05***	-2.11e-05	-0.000118***	-7.91e-05***	-6.89e-05***
	(1.11e-05)	(3.21e-05)	(2.85e-05)	(1.63e-05)	(1.21e-05)
Temp-Q2(T>85F)	-0.000102***	-0.000193***	0.000322	-0.000172***	-0.000156***
	(3.19e-05)	(7.17e-05)	(0.000299)	(4.03e-05)	(3.91e-05)
Temp-Q3(T<25F)	-3.32e-05**	-5.06e-05	-0.000102***	1.23e-05	-9.67e-06
	(1.69e-05)	(5.14e-05)	(2.25e-05)	(0.000103)	(1.96e-05)
Temp-Q3(25<T<45F)	-2.33e-05**	-1.24e-05	-8.43e-05***	-2.82e-06	6.08e-06
	(1.18e-05)	(4.18e-05)	(1.84e-05)	(2.94e-05)	(1.39e-05)
Temp-Q3(65<T<85F)	-1.70e-05*	-4.41e-05	-6.33e-05***	-4.70e-05***	-3.41e-05***
	(9.26e-06)	(2.99e-05)	(1.75e-05)	(1.53e-05)	(9.83e-06)
Temp-Q3(T>85F)	-5.24e-05*	-0.000205***	2.02e-05	-0.000117***	-0.000107***
	(2.70e-05)	(6.17e-05)	(0.000136)	(3.41e-05)	(3.13e-05)

Notes: Author's calculations from CDC public birth files 1975-1988. The dependent variable corresponds to Log(bw): Natural logarithm of birthweight. Cold (Warm) counties corresponds to counties whose average temperature during 1970-1988 belongs to the bottom (top) 33% of the distribution. Metro corresponds to Metropolitan counties according with USDA's 1986 County Typology Codes. Temp-Qj(x<t<y) corresponds to the number of days during the j-th pregancy trimester in which the county's temperature is between x and y. The omitted category corresponds to days with temperatures between 45-65F . All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-Q1(bins), Rainfall-Q2(bins), Rainfall-Q3(bins), and a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Standard errors are clustered at the county level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.5: Heterogeneous effects of temperature during entire pregnancy on birthweight

	(1)	(2)	(3)	(4)	(5)	(6)
Sample	All	White	Black	Cold county	Warm county	Metro county
			Log(birthweight)			
Observations	42,001,350	34,414,304	6,316,941	13,221,099	16,438,735	31,525,162
R-squared	0.392	0.194	0.181	0.405	0.391	0.536
Temp- EP(T<25F)	4.51E-06	2.35e-06	4.18e-06	-2.95e-05*	0.000173**	3.58e-05**
	-0.0000236	(1.43e-05)	(4.39e-05)	(1.73e-05)	(8.60e-05)	(1.68e-05)
Temp-EP(25<T<45F)	-1.00E-05	-1.98e-05**	-2.11e-05	-5.64e-05***	-1.37e-06	1.04e-05
	-1.21E-05	(9.53e-06)	(3.00e-05)	(1.35e-05)	(2.06e-05)	(1.12e-05)
Temp-EP(65<T<85F)	-2.58e-05**	-2.33e-05***	-2.57e-06	-1.27e-05	-4.22e-05***	-3.22e-05***
	-1.08E-05	(7.64e-06)	(2.32e-05)	(1.21e-05)	(1.45e-05)	(8.45e-06)
Temp-EP(T>85F)	-8.92e-05**	-5.91e-05**	-0.000165***	3.30e-05	-0.000116***	-0.000107***
	-4.32E-05	(2.39e-05)	(5.05e-05)	(0.000108)	(3.27e-05)	(3.00e-05)
			Gestational Age			
Observations	41,907,577	34,378,277	6,275,780	13,201,016	16,391,763	31,498,781
R-squared	0.291	0.170	0.179	0.279	0.297	0.386
Temp- EP(T<25F)	-0.00106	-0.000857***	-0.00219**	-0.000765*	-0.00245*	-0.000600
	-0.000909	(0.000309)	(0.000920)	(0.000417)	(0.00144)	(0.000435)
Temp-EP(25<T<45F)	-0.000642	-0.000440***	-0.00245***	-3.24e-05	-0.00163***	-0.000111
	-0.000395	(0.000165)	(0.000519)	(0.000232)	(0.000388)	(0.000206)
Temp-EP(65<T<85F)	-0.00121***	-0.00116***	-0.00161***	-0.00167***	-0.00117***	-0.00128***
	-0.000401	(0.000181)	(0.000493)	(0.000288)	(0.000393)	(0.000244)
Temp-EP(T>85F)	-0.00342***	-0.00291***	-0.00540***	0.00278*	-0.00378***	-0.00376***
	-0.00124	(0.000540)	(0.00105)	(0.00153)	(0.000852)	(0.000695)

Notes: Author's calculations from CDC public birth files 1975-1988. Log(BW): Natural logarithm of birthweight.. Gestational age corresponds to the newborn's weeks of gestation at birth. Temp-EP(x<t<y) corresponds to the number of days during the entire pregancy in which the county's temperature was between x and y . The omitted category corresponds to days with temperatures between 45-65F. All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-EP(bins)), a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried). Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Errors are clustered at the state level . Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.6 Correlation between Medicaid's expansion and temperature-birthweight relationship

	(1)	(2)	(3)	(4)
	Log(bw)	LBW	Gestational Age	PTB
Observations	42,001,350	42,001,350	41,907,577	41,907,577
R-squared	0.393	0.244	0.291	0.268
Temp-Q1(T<25F)*ME	-5.36e-06	0.0176	0.00205	-0.140
	(6.37e-05)	(0.0528)	(0.00132)	(0.107)
Temp-Q1(25<T<45F)*ME	-1.95e-05	0.0173	0.00109	-0.0226
	(4.33e-05)	(0.0397)	(0.000890)	(0.0824)
Temp-Q1(65<T<85F)*ME	-3.67e-05	0.0392*	-0.00172**	0.126***
	(2.27e-05)	(0.0208)	(0.000678)	(0.0458)
Temp-Q1(T>85F)*ME	-9.05e-05*	0.0323	-0.00287***	0.248***
	(5.05e-05)	(0.0483)	(0.000987)	(0.0730)
Temp-Q2(T<25F)*ME	2.79e-06	0.0120	-2.76e-05	-0.0539

	(6.57e-05)	(0.0606)	(0.00205)	(0.116)
Temp-Q2(25<T<45F)*ME	-8.16e-06	0.0157	-0.000818	0.0736
	(4.13e-05)	(0.0370)	(0.00157)	(0.0907)
Temp-Q2(65<T<85F)*ME	-5.73e-05**	0.0522*	-0.00159**	0.125**
	(2.71e-05)	(0.0283)	(0.000647)	(0.0591)
Temp-Q2(T>85F)*ME	-0.000139	0.118	-0.00609***	0.423***
	(8.64e-05)	(0.0773)	(0.00203)	(0.142)
Temp-Q3(T<25F)*ME	3.65e-05	-0.0300	0.00134	-0.118
	(5.56e-05)	(0.0532)	(0.00138)	(0.0716)
Temp-Q3(25<T<45F)*ME	2.51e-05	-0.00446	0.000614	-0.0455
	(3.48e-05)	(0.0324)	(0.000792)	(0.0373)
Temp-Q3(65<T<85F)*ME	-1.20e-05	0.0106	-0.00110	0.0735**
	(2.14e-05)	(0.0205)	(0.000739)	(0.0357)
Temp-Q3(T>85F)*ME	-2.81e-05	-0.0207	-7.95e-05	0.0416
	(6.06e-05)	(0.0554)	(0.00135)	(0.0870)
Temp-Q1(T<25F)	-8.07e-06	0.00901	-0.00348**	0.180*
	(5.82e-05)	(0.0524)	(0.00134)	(0.106)
Temp-Q1(25<T<45F)	-2.00e-05	0.0242	-0.00222***	0.110
	(3.50e-05)	(0.0335)	(0.000803)	(0.0710)
Temp-Q1(65<T<85F)	3.94e-05**	-0.0482**	0.000740	-0.0691*
	(1.94e-05)	(0.0194)	(0.000453)	(0.0370)
Temp-Q1(T>85F)	6.49e-06	0.0265	-0.00151	0.0229
	(4.60e-05)	(0.0535)	(0.00101)	(0.0807)
Temp-Q2(T<25F)	1.57e-05	0.00627	-0.000230	0.00773
	(5.89e-05)	(0.0521)	(0.00206)	(0.111)
Temp-Q2(25<T<45F)	-6.37e-06	0.00881	0.000658	-0.0368
	(3.59e-05)	(0.0310)	(0.00135)	(0.0774)
Temp-Q2(65<T<85F)	-1.32e-05	-0.0182	0.00102*	-0.0881*
	(2.18e-05)	(0.0223)	(0.000558)	(0.0487)
Temp-Q2(T>85F)	-7.69e-05	0.0555	0.00176	-0.0942
	(5.05e-05)	(0.0426)	(0.00209)	(0.119)
Temp-Q3(T<25F)	-6.27e-05	0.0736	-0.00156	0.0800
	(5.10e-05)	(0.0472)	(0.00139)	(0.0705)
Temp-Q3(25<T<45F)	-3.45e-05	0.0373	-0.000610	0.0314
	(3.24e-05)	(0.0293)	(0.000641)	(0.0300)
Temp-Q3(65<T<85F)	-1.80e-05	0.00264	3.00e-05	-0.0396
	(1.88e-05)	(0.0187)	(0.000525)	(0.0273)
Temp-Q3(T>85F)	-5.94e-05	0.0627	-0.00316*	0.0935
	(6.31e-05)	(0.0450)	(0.00182)	(0.0866)

Notes: Author's calculations from CDC public birth files 1975-1988. Log(bw): Natural logarithm of birthweight. LBW corresponds to the number of low birthweight infants per thousand births. Gestational age corresponds to the newborn's weeks of gestation at birth. PTB corresponds to the number of infants born premature (i.e., gestational age<37 weeks) per thousand births. Temp-Qj(x<t<y) corresponds to the number of days during the j-th pregancy trimester in which the county's temperature is between x and y. The omitted category corresponds to days with temperatures between 45-65F. Medicaid Expansion (ME) is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past . All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-Q1(bins), Rainfall-Q2(bins), Rainfall-Q3(bins), a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried), and interactions between lagged temperature and Medicaid expansion (ME). Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Errors are clustered at the state level . Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.7: Impact of Medicaid's expansion on the temperature-birthweight relationship

	(1) Log(bw)	(2) Log(bw)	(3) Gestational Age	(4) Gestational Age
Observations	42,001,350	42,001,350	41,907,577	41,907,577
R-squared	0.392	0.393	0.291	0.291
Heterogeneous effects of temperature across years	No	Yes	No	Yes
Temp- EP(T<25F)*ME	1.65e-05	-6.10e-05	0.000749	-0.000842
	(5.76e-05)	(3.99e-05)	(0.00163)	(0.00136)
Temp-EP(25<T<45F)*ME	4.24e-06	-2.34e-05	0.000597	-5.51e-05
	(3.46e-05)	(2.67e-05)	(0.000990)	(0.000802)
Temp-EP(65<T<85F)*ME	-2.17e-05	-4.44e-08	-0.000831	4.43e-06
	(1.88e-05)	(1.46e-05)	(0.000617)	(0.000559)
Temp-EP(T>85F)*ME	-8.26e-05*	-4.53e-05	-0.00255***	-6.63e-06
	(4.26e-05)	(5.16e-05)	(0.000856)	(0.000974)
Temp- EP(T<25F)	-3.91e-06		-0.00154	
	(5.39e-05)		(0.00168)	
Temp-EP(25<T<45F)	-1.54e-05		-0.00114	
	(2.99e-05)		(0.000916)	
Temp-EP(65<T<85F)	-6.86e-06		-0.000498	
	(1.59e-05)		(0.000359)	
Temp-EP(T>85F)	-5.15e-05		-0.00223*	
	(4.82e-05)		(0.00130)	

Notes: Author's calculations from CDC public birth files 1975-1988. Log(bw): Natural logarithm of birthweight.. Gestational age corresponds to the newborn's weeks of gestation at birth. Temp-EP(x<t<y) corresponds to the number of days during the entire pregancy in which the county's temperature was between x and y . The omitted category corresponds to days with temperatures between 45-65F. Medicaid Expansion (ME) is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past . All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-EP(bins)), a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried), and and interactions between lagged temperature and Medicaid expansion (ME). Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Errors are clustered at the state level . Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.8: Heterogeneous impacts of Medicaid's expansion on the temperature-birthweight relationship

	(1)	(2)	(3)	(4)	(5)
Sample	White	Black	Cold county	Warm county	Metro county
Observations	34,414,304	6,316,941	13,221,099	16,438,735	31,525,162
R-squared	0.194	0.181	0.406	0.391	0.537
Temp- EP(T<25F)*ME	-6.02e-05	-0.000139*	4.32e-06	-0.000523*	-5.19e-05
	(4.10e-05)	(7.12e-05)	(5.14e-05)	(0.000267)	(4.76e-05)
Temp-EP(25<T<45F)*ME	-2.57e-05	-3.34e-05	2.99e-05	-5.67e-05	-2.40e-05
	(2.63e-05)	(6.18e-05)	(3.11e-05)	(7.24e-05)	(3.13e-05)
Temp-EP(65<T<85F)*ME	-4.92e-06	7.45e-05	8.62e-06	2.64e-05	-1.20e-06
	(1.67e-05)	(5.43e-05)	(2.51e-05)	(2.89e-05)	(1.85e-05)
Temp-EP(T>85F)*ME	-4.99e-05	0.000149	-0.000238	1.81e-06	-6.15e-05
	(5.01e-05)	(9.51e-05)	(0.000700)	(7.07e-05)	(5.13e-05)

Notes: Author's calculations from CDC public birth files 1975-1988. The dependent variable corresponds to Log(bw): Natural logarithm of birthweight. Temp-EP(x<t<y) corresponds to the number of days during the entire pregancy in which the county's temperature was between x and y . The omitted category corresponds to days with temperatures between 45-65F. Medicaid Expansion (ME) is a dummy variable at the state-year level =1 if Medicaid is expanded in the current year or has been expanded in the past . All regressions include county-year-race FE and county-

week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-EP(bins), a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried), interactions between lagged temperature and Medicaid expansion (ME), and heterogeneous effects of each temperature bin across years. Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Errors are clustered at the state level . Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 3.9: Impact of air conditioning on the temperature-birthweight relationship

	(1) Log(bw)	(2) Log(bw)	(3) Log(bw)	(4) Gestational Age	(5) Gestational Age	(6) Gestational Age
Sample	All	White	Black	All	White	Black
Observations	42,001,350	34,414,304	6,316,941	41,907,577	34,378,277	6,275,780
R-squared	0.393	0.194	0.181	0.292	0.171	0.181
Temp- EP(T<25 F)*$I(AC_{sy} \leq AC_s) * (AC_{sy} - AC_s)$	-6.64e-07	-0.000400	0.00140	-0.000620	-0.00572	0.0481
	(0.000458)	(0.000473)	(0.00131)	(0.0154)	(0.0151)	(0.0347)
Temp- EP(T<25 F)*$I(AC_{sy} > AC_s) * (AC_{sy} - AC_s)$	-0.000725	3.00e-05	-0.00244	-0.00941	0.00126	-0.0607
	(0.00105)	(0.00118)	(0.00181)	(0.0142)	(0.0151)	(0.0431)
Temp- EP(25<T<45 F)*$I(AC_{sy} \leq AC_s) * (AC_{sy} - AC_s)$	0.000233	0.000339	9.60e-05	0.0111	0.00507	0.0286
	(0.000453)	(0.000539)	(0.000949)	(0.0124)	(0.0135)	(0.0194)
Temp- EP(25<T<45 F)*$I(AC_{sy} > AC_s) * (AC_{sy} - AC_s)$	0.000241	0.000461	0.000545	-0.00532	0.00697	-0.00610
	(0.000335)	(0.000369)	(0.000719)	(0.0106)	(0.0113)	(0.00867)
Temp- EP(65<T<85 F)*$I(AC_{sy} \leq AC_s) * (AC_{sy} - AC_s)$	-0.000384	-0.000277	-0.000681	-0.00939	-0.00320	-0.0363
	(0.000408)	(0.000520)	(0.000719)	(0.0213)	(0.0208)	(0.0228)
Temp- EP(65<T<85 F)*$I(AC_{sy} > AC_s) * (AC_{sy} - AC_s)$	0.000452	0.000359	0.00110*	0.0167	0.0193	0.0255**
	(0.000448)	(0.000465)	(0.000573)	(0.0217)	(0.0233)	(0.0126)
Temp- EP(T>85 F)*$I(AC_{sy} \leq AC_s) * (AC_{sy} - AC_s)$	0.000170	0.000992	-0.00272	0.0574***	0.0653**	-0.00975
	(0.000823)	(0.00107)	(0.00182)	(0.0212)	(0.0306)	(0.0324)
Temp- EP(T>85 F)*$I(AC_{sy} > AC_s) * (AC_{sy} - AC_s)$	0.00305	0.00314**	0.00379	0.0464	0.0495	0.0852**
	(0.00187)	(0.00143)	(0.00252)	(0.0351)	(0.0316)	(0.0351)

Notes: Author's calculations from CDC public birth files 1975-1988. Log(bw): Natural logarithm of birthweight.. Gestational age corresponds to the newborn's weeks of gestation at birth. Temp-EP(x<t<y) corresponds to the number of days during the entire pregancy in which the county's temperature was between x and y . The omitted category corresponds to days with temperatures between 45-65F. $I(AC_{sy} \leq AC_s)$ is an indicator variable equal to one when air conditioning ownership in state s and year y is below or equal to the state's median. All regressions include county-year-race FE and county-week-race FE. Three categories of maternal race were used (white, black, and other). Controls included: Current week's mean temperature, lagged weekly mean temperature for up to four weeks, lagged monthly mean temperature from t-2 to t-7, Rainfall-EP(bins)), a vector of demographics (newborn's gender, teenage mother, age mother>=35, pregnancy history, Highschool dropout, and unmarried), interaction terms between lagged temperature and $I(AC_{sy} > AC_s) * (AC_{sy} - AC_s)$, interaction terms between lagged temperature and $I(AC_{sy} < AC_s) * (AC_{sy} - AC_s)$, and heterogeneous effects of each temperature bin across years. Individual-level observations were collapsed into cells by county-week-year-race. All regressions are weighted by the number of births in each cell. Errors are clustered at the state level . Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

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