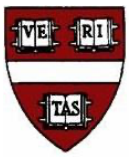
Housing instability, air pollution, and health: Three studies from the United States

HARVARD UNIVERSITY

Graduate School of Arts and Sciences



DISSERTATION ACCEPTANCE CERTIFICATE

The undersigned, appointed by the
Committee on Higher Degrees in Population Health Sciences,
have examined a dissertation entitled

"Housing Instability, Air Pollution, and Health: Three Studies from the United States"

presented by

Aayush Khadka

candidate for the degree of Doctor of Philosophy
and hereby certify that it is worthy of acceptance.

Dr. David Canning, Ph.D., Committee Chair, Harvard T.H. Chan School of Public Health
Dr. Margaret Anne McConnell, Ph.D., Harvard T.H. Chan School of Public Health
Dr. Marc G. Weisskopf, Ph.D., Sc.D., Harvard T.H. Chan School of Public Health

In lieu of all Dissertation Advisory Committee members' signatures,
I, Tyler J. VanderWeele, Ph.D., appointed by the Ph.D. in Population Health Sciences,
confirm that the Dissertation Advisory Committee has examined the above dissertation,
presented by Aayush Khadka, and hereby certify that it is worthy of acceptance as of 30 April 2021.

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Housing instability, air pollution, and health: Three studies from the United States

A dissertation submitted

by

Aayush Khadka

to

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Housing instability, air pollution, and health: Three studies from the United States

Abstract

The United States has consistently ranked poorly in terms of the maternal and child health outcomes relative to other economically developed countries. In addition, there are vast disparities in maternal and child health outcomes within the country by race, ethnicity, and class. A large and active body of literature suggests that differential exposure to social and environmental determinants across high income countries and within the United States may partly explain the existence of these disparities. This dissertation contributes to this field of social and environmental determinants of poor maternal and child health outcomes in the United States. Specifically, it investigates the role of housing instability – a social determinant – and air pollution – an environmental determinant – in impacting the risk of preterm birth, infant death, and pregnancy loss across the country.

Chapter 2 brings together lessons from the maternal health literature – which shows that prenatal psychosocial stress is a risk factor for preterm birth – and the housing literature – which demonstrates that threatened evictions are a major source of stress – to investigate if prenatal exposure to threatened evictions increases the risk of preterm birth. To answer this question, my co-authors and I combined over seven million live birth records from 1,633 counties between 2009 and 2016 with the largest, county-level dataset on threatened evictions from The Eviction Lab at Princeton University. Using a retrospective cohort study design, we fit regression models with several control variables including county fixed effects and find that increased prenatal

exposure to threatened evictions was positively associated with the risk of prematurity over the study period.

Chapter 3 analyzes the relationship of prenatal and post-birth air pollution exposure with infant death. Although this is a well-studied topic, the evidence base is mixed for a variety of reasons. My co-author and I contribute to the existing literature by using a Structural Equation Modeling framework to estimate direct paths from average prenatal and post-birth $PM_{2.5}$ exposure to infant mortality as well as indirect paths from prenatal $PM_{2.5}$ exposure to infant death via preterm birth and low birth weight. We fit the Structural Equation Model on over ten million linked birth-infant death records from 2011 to 2013 merged with daily, county-level average concentration of particulate matter less than 2.5 μm in diameter ($PM_{2.5}$). Our results suggest that increased exposure to $PM_{2.5}$ prenatally was positively associated with the risk of infant mortality with the majority of this association being driven by the direct path from prenatal air pollution to infant death. Our results for the association between post-birth $PM_{2.5}$ exposure and infant death were less precisely estimated in our primary analysis; however, robustness checks indicate a strong, positive association between post-birth air pollution exposure and infant death as well.

Chapter 4 investigates if higher levels of prenatal exposure to air pollution is associated with pregnancy loss. We use a novel analytic framework which allows us to infer the relationship between prenatal air pollution and pregnancy loss by instead analyzing the relationship between the same exposure and conceptions leading to live births, a metric which we can calculate using live birth records. To operationalize this framework, we used birth certificate data between 2001 and 2014 combined with daily, county-level concentration of $PM_{2.5}$, and daily, county-level data

on temperature, precipitation, and relative humidity. For our primary analysis, we fit quasi-Poisson models of the total number of conceptions leading to live births on average, month-by-month $PM_{2.5}$ exposure over a nine-month gestation period adjusting for county-month of year fixed effects and various meteorological and temporal confounders. We conducted several sensitivity analyses as well. Overall, we find inconclusive evidence of an association between prenatal $PM_{2.5}$ exposure and pregnancy loss at any point during gestation.

Table of contents

List of Tables

List of Figures

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For my mother and father

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1 Introduction

This dissertation investigates the relationship of exposure to housing instability and air pollution in the prenatal or post-birth periods with three key maternal and child health outcomes in the United States: preterm birth, infant death, and pregnancy loss.

1.1 The importance of the prenatal and post-birth environments

The impact of the prenatal environment on health and economic outcomes has been extensively documented across several disciplines. A consistent finding across these literatures has been that outcomes associated with fetal exposures manifest themselves throughout the life course in several ways.[1,2] At birth, they manifest themselves in the form of, inter alia, prematurity or low birth weight.[3–5] In the later stages of life, they manifest themselves in the form of lower educational attainment[6–13], lower earnings[7,14], lower height[15,16], as well as higher risk of morbidity and premature mortality[17–20].

There is an equally large literature on the health and economic impacts of various post-birth exposures. For instance, a wide body of work has demonstrated the role environmental exposures such as pesticides, tobacco smoke, and air pollution in impacting different infant and child health outcomes.[21–24] There is also a vast literature investigating challenges that mothers could face in the postnatal period, from insurance churn to lack of continuity of care to maternal death to poor mental health outcomes such as postpartum depression.[25–28]

The three research papers presented in this dissertation aim to contribute to the literature on the impact of exposures in the prenatal and post-birth periods. Chapter 2 focuses on prenatal exposure to an understudied but increasingly important form of housing instability – threatened evictions – and its relationship with preterm birth. Chapter 3 provides novel insight into a well-studied question by investigating the different pathways through which prenatal and post-birth air pollution exposures affect the risk of infant death. Finally, Chapter 4 attempts to identify key exposure windows in the prenatal period in terms of the relationship between air pollution and pregnancy loss using a novel analytic framework. Chapter 5 summarizes the findings from the three research papers and poses some questions for future research.

1.2 Preterm birth, infant mortality, and pregnancy loss in the United States

Preterm birth

Preterm birth, defined as being born before 37 completed weeks of gestation, is one of the leading causes of neonatal and infant death in the US.[29,30] Even when they survive, preterm babies face substantially higher risk of disability – such as cerebral palsy – and developmental delays relative to term babies.[30–34] In addition, prematurity can take a tremendous emotional and financial toll on the family while health systems too face much higher costs as a result of the additional care required for preterm babies.[35] For example, an Institute of Medicine report estimated that the societal cost of prematurity in the US in 2005 was approximately $26.2 billion or $51,600 per preterm infant.[30]

Prematurity is a relatively common outcome in the US, with approximately one in ten babies born preterm. This does not compare favorably to other high income countries such as Canada,

Great Britain, France, and Sweden where prematurity rates are over a percentage point lower relative to the US.[36] Within the country, there are vast disparities in the preterm birth proportion by race and ethnicity.[37] Vital records data from the National Center for Health Statistics (NCHS) show that the preterm birth rate is approximately 1.5 times higher among non-Hispanic Black women compared to non-Hispanic white women and Hispanic women.[38] In addition, since 2015, prematurity rates have been increasing for five straight years after demonstrating a decreasing trend for about a decade.[39]

The Centers for Disease Control and Prevention (CDC) attributes the declining preterm birth trend before 2015 to the declining teenage birth rate; however, we have a poorer understanding of what may be driving the increasing trend since 2015.[39–41] Part of the challenge in explaining this uptick is that we still have an incomplete understanding of the etiology of preterm birth. Prematurity can be the result of several, complex, interacting biological processes that can be influenced by many factors. Much better known are the risk factors associated with prematurity, which scholars have broadly classified into two categories: 1) characteristics of the pregnant woman – including their reproductive history; and, 2) characteristics of the current pregnancy.[42,43]

In terms of the characteristics of the pregnant woman, vulnerable socioeconomic conditions created by systemic racism in the US is associated with the higher rates of prematurity among Black women.[44] Age is another key risk factor for prematurity, with several studies from across the globe documenting a U-shaped relationship between maternal age and preterm birth risk.[45] Previous cases of preterm delivery, stillbirth, or induced abortions have also been identified as

potential risk factors for preterm birth. In terms of the characteristics of the current pregnancy, studies have identified several risk factors, including multiple gestation, vaginal bleeding, and experiencing high levels of psychosocial stress during pregnancy.[42,43]

Infant mortality

Infant mortality, which is defined as death in the first year of life, is considered to be an important marker of the health of a society.[29] In this regard, aggregate level US vital records data paint a relatively rosy picture of health in the US: the rate of infant death has nearly halved within a 34-year period between 1983 and 2017 from 10.9 deaths per 1,000 live births to 5.8 deaths per 1,000 live births.[46] Furthermore, infant mortality has been decreasing at a similar rate across all racial and ethnic groups in the country.[46]

However, despite this declining trend, the disparity in the infant mortality rate by race and ethnicity has remained static over time. For instance, in 1983, the infant mortality rate among non-Hispanic Black Americans was over two times the rate among non-Hispanic white Americans (19.2 deaths per 1,000 live births versus 9.3 deaths per 1,000 live births); in 2017, Black Americans still experienced twice the rate of infant deaths relative to white Americans (10.4 deaths per 1,000 live births versus 4.9 deaths per 1,000 live births).[46]

In addition, the infant mortality rate in the US does not compare favorably against other rich nations. In 2017, the US ranked 33rd in terms of infant mortality relative to the 43 member countries of the Organisation for Economic Co-operation and Development.[47] Other studies have shown that the US ranks poorly relative to some middle income countries as well: in 2013, for

instance, the US infant mortality rate ranked 51st in the world, which was comparable to Croatia, an upper middle income country.[48] A stark way of putting the US' relative infant mortality disadvantage compared to other rich nations is provided by Chen et. al. (2016) who claim that completely closing the infant mortality gap with Scandinavian countries would be worth approximately $84 billion annually to the US.[48]

There is a large literature investigating the reasons behind the within- and across-country disparities in the infant mortality rate. Black-white disparities in terms of infant health outcomes in the US reflect the legacy and persistence of structural racism in this country which rears its ugly head in several ways, including differential rates of adverse birth outcomes, income inequality, access to prenatal care, interaction with the healthcare system, neighborhood conditions, and significantly different socioeconomic conditions.[49–53] Studies have also argued that the higher rates of infant mortality in the US relative to other rich nations may be due to the relatively higher rates of preterm births and other adverse birth outcomes, although recent work suggests that the US experiences high levels of infant death among full term babies as well.[36,48,54–56] Poorer infant health relative to other countries may also reflect the US' fragmented health care system, wider within-country economic disparities, and a weaker social safety net to protect society's most vulnerable.[57]

Pregnancy loss

Pregnancy loss is defined as the loss of a nonviable intrauterine pregnancy, an embryo, or a fetus at any point during gestation. It is often a difficult outcome to measure because it requires a pregnancy to be recognized in the first place and because of the stigma associated with reporting

a loss. Furthermore, the diagnosis of pregnancy loss during the early stages of gestation is complex, which adds to the difficulties in accurately measuring it: for instance, the American College of Obstetricians and Gynecologists recommends that healthcare providers should make a diagnosis of early pregnancy loss only after doing a thorough medical history, physical exam, ultrasonography, and test for beta Human Chorionic Gonadotropin hormone.[58]

Perhaps because of the difficulties in measuring it, no national database exists for pregnancy loss. The National Center for Health Statistics maintains a dataset on deaths of fetuses that have, in most cases, completed at least 20 weeks of gestation; however, these data are known to have various quality issues and, in most cases, fail to capture all pregnancy losses in the country since most losses will have occurred by the end of the first trimester.[59] Despite the measurement challenges, a number of studies have attempted to calculate the incidence of pregnancy loss in the US and estimates vary from just under 20 percent to around 31 percent.[60,61] Studies have also shown that there exist wide disparities in the experience of fetal deaths by race and ethnicity: for instance, a recent analysis of vital records data showed that between 2015 and 2017, the fetal mortality rate among non-Hispanic Black women was twice that among non-Hispanic white and Hispanic women.[62]

Pregnancy loss can impose negative health consequences on both the pregnant woman and their partner. A review of 27 prospective studies from several countries including the US found that mental health disorders such as moderate depression and anxiety were reported by up to 32 percent of women within four to six weeks of experiencing a loss.[63] The same review also found that the partners of these women experienced similar symptoms, albeit at lower levels of

intensity.[63] Another case-control study among US women reported that those who experienced stillbirths – usually defined in the US as fetal death following 20 weeks of gestation – had higher odds of experiencing depression relative to women who did not experience a stillbirth, especially if the former group did not have a history of depression.[64] A systematic review of the international literature also found that stillbirths were associated with a variety of negative mental health effects for the pregnant woman and their partner.[65]

Like preterm birth, the etiology of pregnancy loss is complex, multifactorial, and not fully understood. Studies suggest that up to 50 percent of all cases of early pregnancy loss may be due to fetal chromosomal abnormalities; however, the cause of the remaining cases is less well known.[58,66] There are important differences between the etiology of stillbirth and the etiology of early pregnancy loss as well, with several studies suggesting that the causes of stillbirth range from asphyxia during labor to congenital anomalies to placental dysfunction and fetal growth restriction.[67]

1.3 Overview of research papers

Chapter 2: Evictions and preterm birth

As the rate of rentership climbed following the housing market crash in 2008, so did the risk of being evicted from rented properties.[68–70] Unsurprisingly, poorer households have faced the highest risk of eviction: data from the 2017 American Community Survey shows that among renters making less than $30,000, the rate of eviction was 2.7 percent, almost a percentage point higher than the eviction rate for the overall sample.[68] Emerging data from The Eviction Lab has also suggested that threatened evictions in the US are quite common: in 2016, approximately 6

percent of all renter occupied households had eviction cases filed against them.[71] Additional data from The Eviction Lab also suggests that there are seven eviction filings in local court every minute in the US.[72]

There is a small but fast developing literature on the health effects of experiencing an eviction or being threatened by one. Evidence from New York City suggests that evictions increase the likelihood of being hospitalized for mental health conditions and of being admitted to the emergency department.[73] Matthew Desmond, one of the pioneers of eviction research in the US, has documented that evicted mothers experience a higher likelihood of depression and worse self-reported health relative to mothers who do not experience evictions.[74] Another systematic review of studies on threatened evictions from the US and abroad has found strong evidence of a link between threatened evictions and mental illnesses such as depression, anxiety, suicide, and overall psychological distress.[75]

However, few studies have investigated if being threatened with evictions during pregnancy affects the health of the pregnant woman and their unborn child. Since threatened evictions have been demonstrated to be a source of stress and other poor mental health outcomes, they may be a risk factor for adverse birth outcomes such as preterm birth, especially since experiencing higher levels of psychosocial stress during pregnancy can increase the prematurity risk by almost two-fold.[43] Chapter 2 seeks to address this gap in the literature by investigating the association between in utero exposure to threatened evictions and preterm birth.

To answer this question, my co-authors and I combined over seven million live birth records from 1,633 counties in the US between 2009 and 2016 with the largest available, county-level dataset on threatened evictions from The Eviction Lab.[76] We fit linear regression models which adjusted for individual-level, county-level, and temporal confounders and find that women living in counties that experienced higher levels of threatened evictions during their pregnancy had an increased risk of delivering their child preterm over the study period. We also found some evidence to suggest that the risk of preterm birth may be sensitive to changes in the threatened evictions exposure in the second and third trimesters.

Chapter 3: Air pollution and infant death

Many studies investigating the different risk factors of infant mortality in the US have zoomed in on exposures experienced by the pregnant woman in the prenatal period and exposures experienced by the baby in the post-birth period. One exposure that has been well studied in this regard by scholars across several disciplines is air pollution. Part of the reason why air pollution is a compelling exposure to study in the context of infant death is because of its well-established association with adverse birth outcomes such as prematurity and because of the biological effects it has on the human body which could lead to death among infants due to their lack of a fully developed lung and immune systems.[77–79]

Despite the number of studies addressing this question, the evidence base, on the whole, is decidedly mixed. Results from Currie et. al. (2005), Currie et. al. (2009), and Son et. al. (2017) suggest that while post-birth exposure to increased air pollution increases the risk of infant death, prenatal air pollution exposure does not appear to be particularly impactful, at least at levels

experienced in the US.[80–82] In contrast, Son et. al. (2011) and Jung et. al. (2020) found that exposure to prenatal air pollution increased the risk of infant death but not post-birth exposure.[83,84] At the same time, other studies, such as those by Chay et. al. (2003) and Ritz et. al. (2006) provide evidence to suggest that exposures in both the prenatal and post-birth periods may be important in impacting the risk of infant death.[85,86] Several other studies have not contrasted the importance of prenatal and post-birth air pollution exposure in influencing infant death; however, these studies have consistently shown that increased air pollution – whether acute or chronic – appears to increase the risk of infant mortality.[79,87–90]

Part of the challenge with comparing across studies is that air pollution is not a singular entity in the sense that the air can be polluted by different types of pollutants and at different levels, all of which can have differing effects on infant, child, and adult health. For example, Currie et. al. (2005) and Currie et. al. (2009) investigated the relationship of infant mortality with exposure to particulate matter less than 10 μm (PM_{10}), carbon monoxide, and ozone; Son et. al. (2017) were concerned with exposure to particulate matter less than 2.5 μm ($PM_{2.5}$); and, Chay and Greenstone (2003) focused on exposure to total suspended particulates.[80–82,91] Furthermore, the US-based air pollution-infant mortality studies tend to be done in different and restricted geographical settings – such as California (Currie et. al., 2005), New Jersey (Currie et. al., 2009), and Massachusetts (Son et. al., 2017) – which may have important implications for comparability as well as generalizability.[80–82]

Chapter 3 aims to address some of the limitations of the current literature by focusing on $PM_{2.5}$ across the entire conterminous US. Beyond this, the novel contribution that this paper seeks to

make is by decomposing the association of prenatal $PM_{2.5}$ exposure with infant mortality into a direct path and an indirect path through two adverse birth outcomes. To achieve these aims, my co-author and I used data on over ten million linked birth-infant death records in the US between 2011 and 2013, merged these data with daily, county-level, population weighted average $PM_{2.5}$ concentration data from the CDC, and fit a Structural Equation Model to estimate the direct paths from prenatal and post-birth $PM_{2.5}$ exposure to infant death and indirect paths from prenatal $PM_{2.5}$ exposure through preterm birth and low birth weight. Our results suggest that increased prenatal $PM_{2.5}$ exposure was positively associated with the risk of infant death over the study period; however, our estimates for the relationship between post-birth $PM_{2.5}$ exposure and infant death were less precisely estimated. We also found that a majority of the association between the prenatal $PM_{2.5}$ exposure and infant death was attributable to the direct path from the exposure to the outcome.

Chapter 4: Air pollution and pregnancy loss

Studies that have attempted to investigate the air pollution-pregnancy loss relationship have tended to focus on the outcome of stillbirth. The US-focused evidence about this relationship is mixed. Faiz et. al. (2012) examined the association between in utero exposure to different pollutants and risk of stillbirth using vital records data from New Jersey and found evidence of a positive association for increased gestational exposure to nitrogen dioxide but not necessarily $PM_{2.5}$, sulfur dioxide, and carbon monoxide.[92] DeFranco et. al. (2015) conducted a similar study using Ohio vital records and found evidence for a positive association between third trimester $PM_{2.5}$ exposure and stillbirth but not for exposure over the entire duration of gestation.[93] Green et. al. (2015) conducted a similar study using California vital records data and found some

evidence to suggest that increased nitrogen dioxide exposure during pregnancy may increase the risk of stillbirth.[94]

Although characterizing the air pollution-stillbirth relationship is clearly important, a major limitation of these studies is that their results may not be generalizable for pregnancy loss at any point during gestation, primarily because the etiology of early pregnancy loss differs from the etiology of stillbirth. However, part of the challenge of studying air pollution's relationship with overall pregnancy loss is that losses in the early phases of gestation are not captured in vital records data. Furthermore, vital records on stillbirths are known to have many data quality issues which increases concerns about the reliability of results from studies using these data.[59]

Chapter 4 attempts to characterize the relationship between prenatal air pollution exposure and loss at any stage of gestation by using a novel analytic framework first developed by Kioumourtzoglou et. al. (2019).[95] This framework sidesteps traditional challenges of measuring pregnancy loss at any point during gestation and allows us to infer the relationship between prenatal air pollution and pregnancy loss by investigating a proxy outcome: the total number of live births occurring from conceptions in any given month. We call this proxy outcome live birth-identified conceptions. A major strength of this framework is that the proxy outcome of live birth-identified conceptions can be calculated from data on live births, which are generally recorded with a very high degree of accuracy in the US.

In Chapter 4, my co-authors and I adapt the Kioumourtzoglou et. al. (2019) analytic framework and focus specifically on the relationship between prenatal $PM_{2.5}$ exposure and pregnancy loss.

Our choice to study $PM_{2.5}$ was motivated by the plausible biological mechanism linking the pollutant with early pregnancy loss and because a cohort study with 344 singleton conceptions in Michigan and Texas by Ha et. al. (2018) found a positive association between chronic in utero $PM_{2.5}$ exposure and loss at any point during gestation.[96] We operationalized the analytic framework by linking live birth record data from all counties in the conterminous US between 2001 and 2014 with daily, county-level $PM_{2.5}$ concentration data from the CDC. We used distributed non-linear lagged models to estimate the association between month-by-month variation in $PM_{2.5}$ during gestation and live birth-identified conceptions. We fit all regression models adjusting for several meteorological variables as well as temporal and geographic controls. Results from our primary model suggests that $PM_{2.5}$ exposure in the fifth and sixth month of gestation was negatively associated with live birth-identified conceptions and consequently, positively associated with pregnancy loss. However, this result did not hold across all robustness checks which recommends caution in interpreting our primary findings.

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2 In utero exposure to threat of evictions and preterm birth: Evidence from the United States

Aayush Khadka[1,2], Günther Fink[3], Ashley Gromis[4], and Margaret McConnell[1]

[1]Department of Global Health and Population, Harvard T. H. Chan School of Public Health, Boston MA 02115
[2]Graduate School of Arts and Sciences, Harvard University, Cambridge MA 02138
[3]Swiss Tropical and Public Health Institute & University of Basel, Socinstrasse 57, 4051, Basel, Switzerland
[4]Department of Sociology, Princeton University, Princeton NJ 08544

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2.1 Abstract

Although they are becoming an increasingly common feature of the lives of low-income Americans, few studies have investigated if exposure to threatened evictions during pregnancy affects maternal and child health outcomes. Using live birth records from the National Center for Health Statistics from 2009 to 2016 and threatened evictions data from The Eviction Lab, we conducted a retrospective cohort analysis to investigate associations between in utero exposure to threated evictions and preterm birth (born <37 completed weeks of gestation) in the United States. We also investigated the relationship between prenatal threatened evictions exposure and three secondary outcomes: a continuous measure for gestational length, a continuous measure for birth weight, and an indicator for low birth weight (born <2,500 grams). To estimate these associations, we fit linear regression models that adjusted for individual- and time-varying county-level characteristics, county fixed effects, state-year-and-month-of-conception fixed effects, and a county-specific time trend. We found that exposure to increased levels of eviction case filings in utero was associated with an increased risk of prematurity and low birth weight. These associations appeared to be sensitive to exposure in the second and third trimesters. Associations with secondary outcomes and within various population sub-groups were, in general, imprecisely estimated.

2.2 Background

The threat of evictions, defined as filing for a landlord-initiated forced removal from a rental unit in local court, has become an increasingly common feature of the lives of low-income Americans. In 2016, approximately 2.4 million eviction cases were filed in the United States.[1] While this number represented approximately 6 percent of all renter-occupied households in the same year, many cities, such as North Charleston, South Carolina or Richmond, Virginia, experienced substantially higher risk of threatened evictions.[2,3]

A developing literature links eviction to harmful effects on health. Several studies have documented associations between increased eviction prevalence and elevated risk of experiencing stress, depression, anxiety, psychological distress, and drug use.[4–12] The evidence with respect to physical health outcomes is more mixed: some studies have demonstrated positive associations between evictions and chronic disease prevalence and emergency room use;[9,13] others, in contrast, have found no evidence of associations between evictions and poor health status.[4,10,14]

Despite mounting evidence of a link between eviction and health, there is less evidence on the relationship between threatened evictions and key maternal and child health outcomes. Evidence from the housing literature suggests that families with small children may be particularly likely to be threatened with evictions and, ultimately, be evicted.[4,15–17] Pregnant women and newborns threatened with eviction may be especially vulnerable to negative health effects as well.[18] We aimed to fill this gap in the literature by estimating associations between in utero exposure to

county-level eviction filings and adverse birth outcomes, particularly preterm births. We also investigated whether these associations varied by pregnancy trimester.

We analyzed preterm births as our primary outcome because it is the second largest contributor to infant mortality in the United States.[19] Furthermore, preterm babies face significantly higher risk of long-term morbidity and developmental challenges which lead to, among other things, substantially higher financial costs for all parties involved and a higher psychological toll for the caregivers.[20–24]

We hypothesized that increased prenatal exposure to threatened evictions would increase the likelihood of preterm birth and other adverse birth outcomes. This is because the threat of evictions is a key source of stress and other poor mental health outcomes, and a large literature demonstrates that high levels of prenatal stress is an important risk factor for poor birth outcomes.[20,25–28]

2.3 Methods

Study design and data

We conducted a retrospective cohort analysis by combining the largest county-level dataset on legal eviction case filings in the United States to date with restricted-use national birth records from the National Center for Health Statistics (NCHS).

Data on eviction filings were provided by The Eviction Lab at Princeton University and contained county-month-year-level counts of the number of eviction cases filed in local court for

1,633 counties between 2008 and 2016.[1] Temporal coverage varied across counties. As a measure of the quality of the threatened eviction estimates, the data also identified counties for which the number of case filings in any county-year fell within 85-115 percent of estimates obtained directly from the courts for the same county in the same year.

Live birth records from the NCHS represented the universe of live births in the United States between 2009 and 2016. These data contained individual-level information on each woman's demographics, delivery payment method, self-reported county of residence at delivery, completed weeks of gestation, birth weight, and day of week, month, and year of delivery.

We supplemented our analysis by using county-level data from two other sources. From the United States Census Bureau, we used data on annual, county-level 18-and-over population and annual, county-level poverty rate.[29] From the Bureau of Labor Statistics, we used information on monthly, unstandardized county unemployment rates.[30]

Exposure definition

For each pregnant woman, we constructed two exposure variables using data on county-month-year eviction case filings. The first exposure was defined over the duration of the pregnancy from the month of conception to the month of delivery. The second exposure was defined separately for the first trimester (month of conception to third month of gestation) and together for the second and third trimesters (fourth month of gestation to the month of delivery). We label the first exposure as EP (i.e., exposure during pregnancy) and the second exposure as ET (i.e., exposure by trimester).

To construct EP and ET, we estimated each woman's date of last menstrual period (LMP) using information on the obstetric/clinical estimate of gestational length and the day of week, month, and year of delivery. Specifically, we estimated the date of birth of each newborn by assuming a uniform probability distribution over the day of week of birth within the month and year of delivery for each live birth following which we backed out the LMP date by subtracting the gestational length from the estimated date of birth. Next, we assigned a date of conception for each woman in our sample by assuming that conception occurs two weeks after the estimated date of LMP. Finally, we identified the number of eviction cases filed in each month of a woman's pregnancy by using information on her month-year of conception and county of residence at delivery.

Having assigned eviction cases to each pregnant woman for each month of their pregnancy, we constructed EP and ET in three steps. First, we normalized the number of cases filed for each county-month-year by the county's 18-and-over population for the same year. Next, we estimated the average of the population-normalized eviction case filings over the duration of each woman's pregnancy as well as separately for the first trimester and second and third trimesters combined. Finally, we standardized the average, normalized cases to define a z-score ($cases_{i,t}^{zscore}$) as

$$cases_{i,t}^{zscore} = \frac{cases_{i,t}^{avg,\,norm} - \overline{cases_{t}^{avg,norm}}}{sd(cases_{i,t}^{avg,\,norm})} \qquad [1]$$

where $cases_{i,t}^{avg,\,norm}$ represents the average normalized case filings for each pregnant woman i over duration of pregnancy/trimester t, $\overline{cases_t^{avg,norm}}$ represents the mean of the average normalized case filings in the entire analytic sample over time period t, and $sd(.)$ represents the standard deviation operator.

Outcome definition

We defined our primary outcome, preterm birth, as an indicator variable which equaled one if completed weeks of gestation for a newborn was reported as less than 37 weeks and zero otherwise. We used the obstetric/clinical estimate of gestation as our preferred measure for gestational length following NCHS recommendations.[31]

We also estimated associations between threatened evictions and three secondary pregnancy outcomes: 1) completed weeks of gestation defined as a continuous variable and measured using the obstetric/clinical estimate; 2) birth weight (in grams) defined as a continuous variable; and 3) low birth weight defined as an indicator variable which equaled one if birth weight was less than 2,500 grams and zero otherwise.

Analytic strategy

We constructed our analytic sample by restricting live birth observations based on the following inclusion criteria: 1) had obstetric/clinical estimate of gestation reported; 2) mother resided in a county for which eviction case filing data were available; 3) eviction case filing data were available for each month of gestation; 4) delivery occurred in a state that had adopted the 2003 revised birth certificate in the year of delivery; and 5) the live birth was singleton.

To prepare the data for analysis, we merged birth records with the eviction case filing data using information on the estimated month-year of conception associated with each live birth and each pregnant woman's county of residence at delivery. We similarly merged supplementary datasets on county-level population, unemployment, and poverty. Finally, for each observation in our data, we estimated eviction exposures EP and ET using Equation 1. We then constructed the analytic sample by applying the study inclusion criteria.

We computed descriptive statistics of the outcome and covariates of interest in the analytic sample by tertiles of exposure EP. We assumed that covariate data were missing at random and accounted for them by constructing five imputed datasets under the assumption that the observed and unobserved data together followed a multivariate normal distribution. In each imputed dataset, we winsorized exposures EP and ET at the 1st and 99th percentile to reduce the influence of extreme values of the exposure.[32] We then estimated the following equation:

$$y_{i,c,m,t} = \alpha + \beta zscore_{i,c} + \lambda' X_{i,c,t} + \delta_c + \theta_{s(c),t} + time_c + \epsilon_{i,c,m,t} \qquad [2].$$

In Equation 2, β represents the association between eviction filings and adverse birth outcomes, $X_{i,c,t}$ represents individual- and county-level covariates, δ_c represents county fixed effects defined based on each woman's self-reported county of residence at delivery, $\theta_{s(c),t}$ represents state-of-residence-year-and-month-of-conception fixed effects, and $time_c$ represents a county-of-residence-specific linear time-trend. Equation 2 allowed us to make within-county contrasts while flexibly controlling for temporal trends in the outcome at the state and county levels. The

individual-level covariates we controlled for in this specification were mother's age, a quadratic age term, race, highest level of education, parity, child's sex, and method of payment for delivery. Although tobacco use during pregnancy or gestational diabetes are known risk factors of adverse birth outcomes, we did not control for these variables since they plausibly lie on the causal pathway between threatened eviction exposure and birth outcomes. At the county-level, we controlled for a county's urban-rural classification based on the NCHS classification system, average unemployment rate of each woman's county of residence over the duration of the pregnancy, and poverty rate of the county of residence for the year of conception.[33,34] The NCHS classifies counties into six urban-rural categories: large central metro, large fringe metro, medium metro, small metro, micropolitan, and noncore.[33,34]

We estimated Equation 2 using Linear Probability Models (LPM) for the preterm birth and low birth weight outcomes. Although these are binary outcomes, we use the LPM because the model provides unbiased estimates of the marginal association between eviction filings and the outcome averaged over the distribution of the exposure variable.[35] We also used Ordinary Least Squares to estimate associations of the eviction case filing exposure with gestational length and birth weight. We accounted for correlated outcomes within counties by using Huber's cluster-robust standard errors at the county-level across all regression models.[36] Finally, we pooled estimates of the coefficient and standard errors across the five imputed datasets using Rubin's rules.[37]

We assessed the robustness of our results in several ways. We defined a separate exposure variable using data on eviction filings in the nine months prior to conception and used this exposure as a negative control to conduct a falsification test.[38] Evidence of a relationship

between eviction filings that pre-date pregnancy and pregnancy outcomes might indicate that our analytical model is identifying spurious relationships or pre-existing trends between county-level filings and county-level outcomes. To assess if our results are sensitive to exposure misclassification, we restricted the eviction case filing data to only those counties for which annual reported case filing counts were between 85 – 115 percent of external estimates ("verified cases"). Furthermore, to determine if our association estimates are affected by the lack of complete county time series, we restricted the analytic sample to women who lived in counties for which we had a complete panel ("complete time series"). Finally, we restricted our analytic sample to counties that had eviction data for five or more years over the study period ("five-year time series"). In all robustness tests, we imputed the missing data following the same procedure as in our primary analysis, winsorized the relevant exposure variables at the 1^{st} and 99^{th} percentiles, and estimated Equation 2.

To check for association heterogeneity, we separately re-estimated Equation 2 in the analytic sample amongst White non-Hispanic women, Black non-Hispanic women, Hispanic women, women of other races, and women who paid for their deliveries using Medicaid. We used payment for deliveries using Medicaid as a proxy for being low-income. We winsorized exposures EP and ET at the 1^{st} and 99^{th} percentiles within each sub-group and estimated Equation 2.

Software

We used Stata/MP 15.1 to clean the data, conduct descriptive analyses, and estimate all regression models.[39] We used "Amelia II" in RStudio to conduct multiple imputations of the analytic sample.[40,41]

Ethical statement

This study was deemed to be exempt from human subjects review by the Office of Human Research Administration at the Harvard T. H. Chan School of Public Health.

2.4 Results

Our analytic sample consisted of 7,324,812 live births from 1,633 counties in 39 states and the District of Columbia between 2009 and 2016. This sample was constructed from 31,950,741 live births across all counties in the United States over the same time period (Figure 2.1). The primary reason for not including live birth observations in the analytic sample was the lack of eviction case filing data at the county level. Appendix Figure 2.1 shows the states that were included in our analysis and the year from which they adopted the 2003 birth certificate revision while Appendix Figure 2.2 shows the counties that appear in our analytic sample and the number of years for which we have eviction data for each county. Counties from the Midwest are best represented in our analytic sample in terms of their frequency and time series length. In contrast, we have relatively few counties from the Northeast and the length of the time series of these counties is also relatively short. Finally, Appendix Table 2.1 shows that only six variables in our analytic sample had any item non-response and that the frequency of missing data in these six variables was very low.

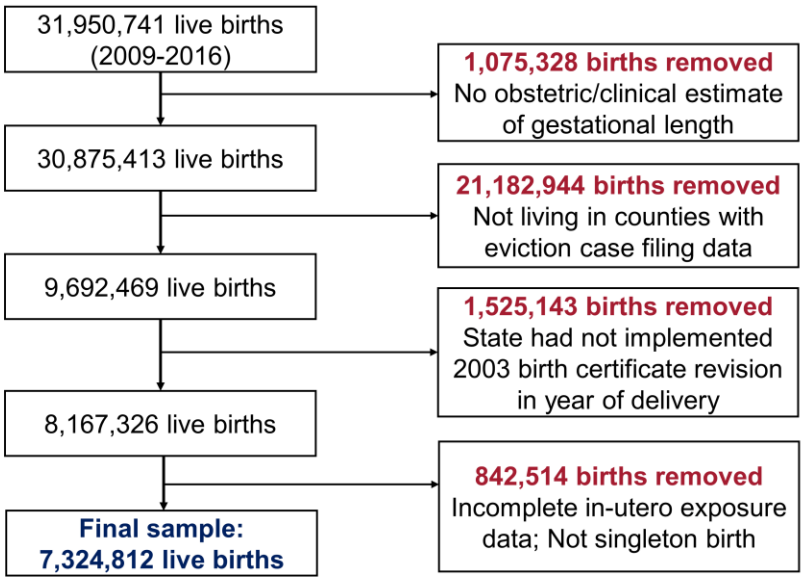

Figure 1 Construction of analytic sample

Figure 2.2 illustrates unadjusted, yearly averages of the four study outcomes by tertiles of exposure EP between 2009 and 2015. Panel (a) shows that the unadjusted preterm birth proportion was substantially lower among women who resided in counties with low levels of eviction filings ("low exposure tertile") relative to women who resided in counties with high levels of eviction filings ("high exposure tertile") in all years between 2009 and 2015. Similarly, panel (b) and panel (c) respectively show that average gestational length and average birth weight were consistently higher in the low exposure tertile relative to the high exposure tertile over the same time period. Finally, panel (d) suggests that the proportion of low birth weight newborns was consistently lower in the low exposure group relative to the high exposure group between 2009 and 2015.

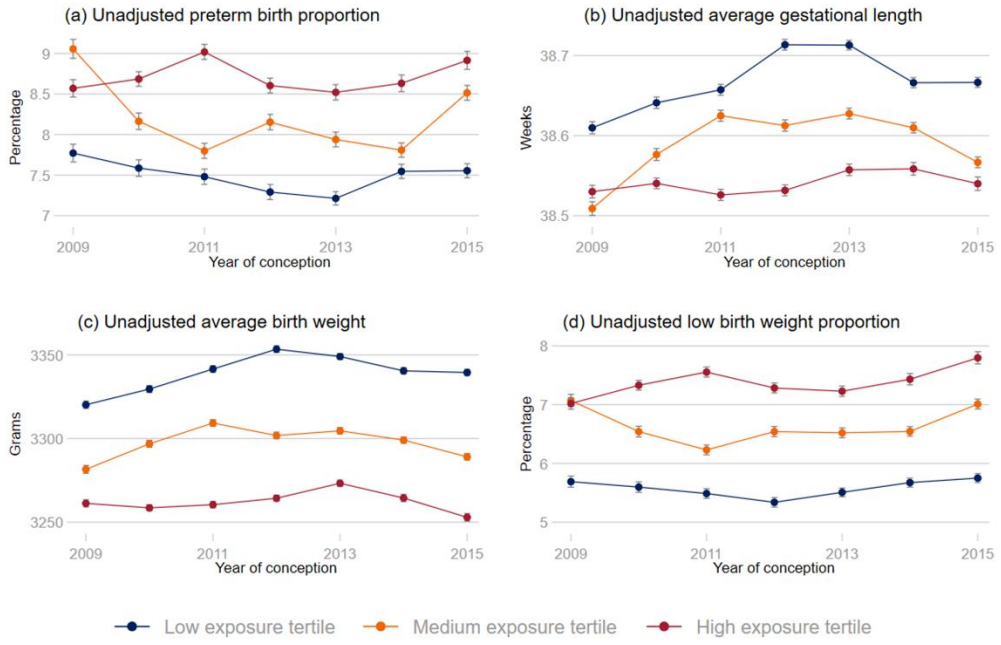

Figure 2 Unadjusted, annual average outcome by tertile defined using exposure to average eviction case filings over the duration of the pregnancy (exposure EP) and year of conception

Differences in unadjusted average outcomes across exposure categories presented in Figure 2.2 may reflect underlying compositional differences in these groups. Table 2.1 presents differences in individual-level socioeconomic characteristics and county-level characteristics across the three tertiles of exposure EP. Relative to women in the low exposure group, a higher proportion of women in the high exposure group reported not having a high school degree. Similarly, the proportion of Black non-Hispanic and Hispanic women was substantially higher in the high exposure group relative to the low exposure group as was the proportion of women who reported paying for their deliveries using Medicaid. Counties represented in the high exposure group were also more likely to have higher unemployment and poverty rates, and to be classified as metropolitan relative to counties in the low exposure group. Appendix Table 2.1 shows that these differences between the high and low exposure groups were consistent over time.

Table 2.1 Distribution of individual and county-level covariates over the study period by exposure tertiles defined using average eviction case filings over the pregnancy (exposure EP)

	Low exposure tertile	Medium exposure tertile	High exposure tertile
Individual-level			
Mean age (years)	27.96 [27.95, 27.96]	27.93 [27.92, 27.94]	27.76 [27.75, 27.77]
Percent of women with no High School	14.24 [14.2, 14.29]	16.59 [16.54, 16.63]	19.46 [19.41, 19.51]
Percent of women with High School but no tertiary degree	46.13 [46.06, 46.19]	46.74 [46.68, 46.81]	46.51 [46.45, 46.57]
Percent of women with a tertiary degree	39.63 [39.57, 39.69]	36.67 [36.61, 36.73]	34.03 [33.97, 34.09]
Percent White (non-Hispanic)	72.02 [71.96, 72.08]	55.67 [55.61, 55.73]	40.61 [40.55, 40.67]
Percent Black (non-Hispanic)	6.16 [6.13, 6.19]	17.36 [17.31, 17.41]	28.71 [28.65, 28.76]
Percent Hispanic	14.19 [14.15, 14.24]	21.08 [21.03, 21.13]	24.49 [24.44, 24.55]
Percent Other races	7.63 [7.6, 7.66]	5.89 [5.86, 5.92]	6.19 [6.16, 6.22]
Percent paying for delivery using Medicaid	38.29 [38.23, 38.35]	43.16 [43.1, 43.23]	44.67 [44.6, 44.73]
County-level			
Average unemployment rate	6.70 [6.7, 6.7]	7.08 [7.08, 7.08]	7.61 [7.6, 7.61]
Average poverty rate	13.74 [13.73, 13.74]	15.73 [15.73, 15.74]	17.21 [17.2, 17.21]
Percent metropolitan counties	65.95 [65.89, 66.01]	93.48 [93.45, 93.51]	97.58 [97.56, 97.6]

Note: 95 percent confidence intervals in brackets

Table 2.2 presents our main results of the association between exposure EP and birth outcomes. We estimated that a standard deviation increase in eviction case filings was associated with a 1.09 percentage point increase in the risk of preterm birth over the study period ($\beta = 1.09$

percentage point; 95 percent confidence interval: 0.05, 2.13). Since the effective exposure variation is constrained by our primary specification – that is, we analyze the variation that remains after accounting for county-level fixed effects, state-year-and-month-of-conception fixed effects, and a linear county-level time trend – an association of 1.09 percentage point corresponds to a 0.08 percentage point increase, on average, in the risk of preterm birth (Appendix Figure 2.3). Relative to the sample average preterm birth proportion of 8.18 percent, a 0.08 percentage point increase in risk corresponds to an approximately 1 percent increase.

Table 2.2 Associations between exposure to average eviction case filings over the duration of the pregnancy (exposure EP) and birth outcomes

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing over pregnancy	1.09% point [0.05, 2.13]	-0.05 weeks [-0.13, 0.03]	-11.96 grams [-28.75, 4.82]	0.72% point [0.02, 1.43]
Average value of the outcome in the analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	7,324,812	7,324,812	7,324,812	7,324,812

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

We also estimated a positive association between exposure EP and risk of low birth weight: a standard deviation increase in average case filings over a pregnancy was associated with a 0.72 percentage point increase in risk of low birth weight ($\beta = 0.72$ percentage point; 95 percent confidence interval: 0.02, 1.43). Given the effective variation we are working with, a 0.7 percentage point increase in risk corresponds to a 0.05 percentage point increase in the risk of newborns being born low birth weight (Appendix Figure 2.3). Associations between exposure EP and length of gestation as well as birth weight were negative but imprecisely estimated.

Table 2.3 presents associations between exposure ET and all study outcomes. In terms of the primary outcome, we estimated that a standard deviation increase in threatened evictions in the second and third trimesters was associated with a 1.02 percentage point increase in the risk of preterm birth over the study period ($\beta = 1.02$ percentage point; 95 percent confidence interval: 0.015, 1.90). After adjusting for the effective variation, this risk difference represented a 0.08 percentage point increase in the risk of preterm birth (Appendix Figure 2.4). Our estimate of the association between threatened evictions in the first trimester and preterm birth risk was imprecisely estimated.

Table 2.3 Associations between exposure to average eviction case filings by pregnancy trimester (exposure ET) and birth outcomes

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing in the first trimester	-0.06% points [-0.49, 0.37]	0.004 weeks [-0.03, 0.04]	1.51 grams [-5.2, 8.21]	-0.01% points [-0.33, 0.31]
Z-score of average case filing in the second and third trimesters	1.02% points [0.15, 1.9]	-0.05 weeks [-0.13, 0.03]	-11.70 grams [-26.76, 3.36]	0.69% points [0.03, 1.34]
Average value of the outcome in the analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	7,324,812	7,324,812	7,324,812	7,324,812

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

In terms of the secondary outcomes, we estimated a positive association between threatened evictions in the second and third trimesters and risk of low birth weight ($\beta = 0.69$ percentage point; 95 percent confidence interval: 0.03, 1.34). The estimated association corresponds to a 0.06 percentage point difference in the risk of low birth after accounting for the effective variation (Appendix Figure 2.4). The association between eviction case filings in the first trimester and the risk of low birth weight was imprecisely estimated. Associations with gestational length and birth weight for threatened evictions in the first trimester as well as the second and third trimesters were also imprecisely estimated.

Appendix Table 2.3 presents results from the falsification test using pre-conception exposure as a negative control and shows that associations of this variable with all four outcomes were small in magnitude and imprecisely estimated. Appendix Table 2.4 – Appendix Table 2.6 present results from estimating Equation 2 using exposure EP in the three robustness samples and show that results from the verified cases sub-sample (Appendix Table 2.4) and the five-year time series sub-sample (Appendix Table 2.6) were consistent with our main results.[‡] Appendix Table 2.7 – Appendix Table 2.9 present results from estimating Equation 2 in the three robustness sub-samples using exposure ET and show that results from the five-year time series sub-sample (Appendix Table 2.9) is consistent with our primary results.

Results from analyses checking for association heterogeneity by racial sub-groups and Medicaid payment status were largely consistent with the primary results (Appendix Table 2.10 – Appendix Table 2.19). We estimated a positive association between prematurity and exposure EP among White non-Hispanic women (Appendix Table 2.10). We also estimated a positive association between risk of being born low birth weight and exposure EP among White non-Hispanic and Black non-Hispanic women (Appendix Table 2.13). Increased exposure to threatened evictions in the second and third trimesters was also associated with an increased risk of prematurity among women of other races (Appendix Table 2.15) and women who paid for their deliveries using Medicaid (Appendix Table 2.19). We also estimated a positive association between second and third trimester threatened evictions exposure and risk of delivering a low birth weight newborn among White non-Hispanic women (Appendix Table 2.18).

[‡] The verified cases sub-sample included 7,027,351 live birth observations from 1,632 counties across 39 states and the District of Columbia. The complete time series sub-sample consisted of 3,254,301 live birth observations from 617 counties from 30 states. Finally, the five-year time series sub-sample had 6,364,818 observations from 1,156 counties across 37 states.

2.5 Discussion

In this study, we investigated the relationship between prenatal exposure to threatened evictions and adverse birth outcomes across 1,633 counties in 39 states and the District of Columbia between 2008 and 2016. We also studied if these associations varied by pregnancy trimester. We found that increased exposure to eviction filings during a pregnancy was associated with an increased risk of prematurity and being born low birth weight. We also found some evidence to suggest that risk of preterm birth and low birth weight were particularly sensitive to eviction filings in the second and third trimesters of a pregnancy.

Results from various robustness checks largely supported our primary results. Coefficients on the pre-pregnancy exposure variable, which we used as a negative control to conduct a falsification analysis, were small and their associated 95 percent confidence interval included the null. Although pre-pregnancy threatened evictions exposure may not be the ideal negative control because of its potential direct effect on birth outcomes through pre-pregnancy health, the falsification check results do provide suggestive evidence to support the claim that our primary results are not driven by spurious correlations. Results from estimating Equation 2 across the three robustness sub-samples also support our primary results, especially for exposure EP. Finally, we were largely unable to estimate precise associations between threatened evictions and adverse birth outcomes by racial sub-groups or among women who paid for their delivery using Medicaid insurance.

To the best of our knowledge, our study is one of the first to demonstrate that increased within-county exposure to eviction filings increases the risk of preterm birth. The positive associations between eviction filings – a source of prenatal stress – and risk of prematurity and low birth weight are consistent with studies that have investigated the impact of prenatal stressors on pregnancy outcomes. For instance, Gemmill and colleagues (2019) found that election of Donald Trump as president of the United States was associated with an increased risk of premature births among Latina women who were pregnant at the time of the election.[27] Similarly, Currie and colleagues (2019) demonstrated that exposure to assaults in utero was associated with a higher risk of adverse birth outcomes in New York City.[42]

However, the associations we estimate for preterm birth risk and low birthweight risk have relatively wide confidence intervals. For the association between exposure EP and preterm birth, for example, our results are compatible with a 0.05 to 2.13 percentage point increase in the risk of prematurity. This relatively wide range of estimates may in part reflect the ecological nature of our analysis which analyzes an exposure at the county-level, i.e., our analysis treats all pregnant women as exposed to a given level of eviction filings regardless of whether they personally received an eviction notice or were affected by one indirectly. This means that our associations likely capture some combination of the exposure of living in a community where evictions are common combined with the direct experience of receiving an eviction filing. Individual level data on eviction filings would allow for the estimation of these associations with a higher degree of precision and could consequently find substantially larger effects for pregnant women who were directly affected by the threat of evictions over the study period. The potential for community-level effects of housing instability resulting from increased eviction filings would

also be consistent with the housing and social epidemiology literatures which have shown the impact of neighborhood quality on health outcomes.[43–46]

Another strength of our study is that we analyze the relationship between threatened evictions and physical health outcomes using data that has wide geographic and temporal coverage. Prior studies looking at associations between threatened evictions, actual evictions, and health outcomes have either come from other countries such as Spain or from very localized geographies within the United States.[4,9,10,13] Niccolai and colleagues (2019) use similar data to estimate associations between evictions and sexually transmitted diseases across the country but limit their analysis to only 2014.[47]

A final strength of our analysis is that it adds to the literature on the importance of the timing of in utero stressors on the risk of preterm birth and other pregnancy outcomes. In this regard, our finding regarding the relative importance of eviction case filing exposure in the second and third trimesters is consistent with some recent studies such as that by Gemmill and colleagues (2019).[27] Earlier studies have suggested that exposure to traumatic events such as natural disasters in the first trimester is associated with poor birth outcomes although few previous studies have used national data and those that have were not focused on the United States.[48–51]

A key limitation of our analysis is that we did not have complete geographical and temporal coverage in terms of the eviction case filing data. The United States has over 3,000 counties but we only had threatened evictions data from 1,633. Lack of data from all counties is explained by the fact that collection of data on eviction cases is more difficult in some areas due to incomplete

or non-standardized electronic case management systems, limitations on access to paper case records, and restrictions on bulk records requests. It is unclear how these barriers to record collection are associated with case filing volumes, which limits the generalizability of our results. Furthermore, generalizability of our results may also be limited because we could only use approximately 57 percent of the possible 176,364 county-month-year observations, not only due to a lack of county-level data but also because we restricted our analysis to those state-years in which the 2003 birth certificate revision had been implemented.

Another limitation of our analysis is that we only observe residence at time of delivery, and we are therefore unable to determine whether a pregnant woman moved across counties over the course of her pregnancy. Movement across counties would mean that our exposure variable would be subject to measurement error that we expect would attenuate our results. However, there are reasons to believe that movement across counties may be limited in scope: the literature on evictions suggests that individuals who are evicted generally tend to move into worse quality neighborhoods and not necessarily across counties.[52] A review of the residential mobility literature by Bell and Belanger (2012) also finds that the distance moved during pregnancy when changing residences is often very short (median distance <10 kilometers).[53] In addition, a recent study by Garboden and Rosen (2019) also finds that threatened evictions do not always lead to actual evictions and therefore a change in residence.[54]

A final limitation of our analysis is the possibility of selection bias due to exposure to higher levels of threatened evictions resulting in greater loss of fetuses that would have been born

premature relative to lower levels of threatened eviction exposure.[55,56] We expect such fetal selection to attenuate our results.

Conclusions

The threat of evictions has been increasing over the past two decades, particularly for low-income Americans. Our analysis shows that for pregnant women, higher levels of threatened evictions are associated with increased risk of adverse birth outcomes in general and premature deliveries in particular. Across the United States, several policies are currently being enacted to offset the threat of evictions – our study suggests that evaluating the causal impact of these policies on parental well-being and child health is an important area of inquiry.

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3 Early life air pollution exposure and infant mortality in the United States: understanding the pathways

Aayush Khadka[1,2] and David Canning[1]

[1] Department of Global Health and Population, Harvard T. H. Chan School of Public Health, Boston MA 02115, USA
[2] Graduate School of Arts and Sciences, Harvard University

3.1 Abstract

We study the relationship of prenatal and post-birth exposure to particulate matter less than 2.5 μm in diameter ($PM_{2.5}$) and infant mortality in the US for all births occurring between 2011-2013 in the conterminous United States. We estimated direct paths from the prenatal and post-birth exposures to infant death as well as indirect paths from the prenatal exposure to the outcome through preterm birth and low birth weight. To do so, we linked over 10 million restricted access linked live birth-infant deaths data with daily, population-weighted, county-level $PM_{2.5}$ concentration data and fit a Structural Equation Model. In all equations, we controlled for a variety of individual- and count-level confounders of the exposure-outcome, mediator-outcome, and exposure-mediator relationship, including county and month-of-year fixed effects. We found that increased prenatal $PM_{2.5}$ exposure was associated with increased risk of infant death although evidence for the relationship between post-birth exposure and infant death was less precisely estimated. Most of the total association between prenatal $PM_{2.5}$ exposure and infant death was explained by the direct pathway from the exposure to the outcome rather than the indirect pathway through adverse birth outcomes.

3.2 Background

Although several studies have investigated the relationship between early life ambient air pollution exposure and infant mortality, the evidence about the importance of prenatal and post-birth exposure in mixed.[1,2] Some studies have found that exposure to higher levels of post-birth air pollution increases the risk of infant death but increased prenatal air pollution exposure does not.[3,4] In contrast, other studies have found that higher levels of prenatal air pollution exposure increases the risk of infant death but post-birth air pollution does not.[5,6] Furthermore, some studies have also suggested that both prenatal and post-birth air pollution exposure may increase the risk of infant death.[7,8]

There are plausible biological mechanisms to link both prenatal and post-birth air pollution exposure to infant death. For instance, there is very strong evidence linking prenatal air pollution exposure with increased risk of adverse birth outcomes such as preterm birth and low birth weight.[9] Such a relationship would suggest that prenatal air pollution may be linked to infant death because prematurity and low birth weight are two of the most important drivers of infant mortality in the US.[10] At the same time, there are biologically plausible direct pathways from prenatal air pollution exposure to infant mortality as well: for example, exposure to fine particulate matter can cause imbalances in an individual's autonomic nervous system and result in oxidative stress, both of which when experienced by pregnant women can affect maternal and fetal health, which in turn can have implications for infant health.[11,12] Fine particulate matter can also traverse the placental barrier, thus directly affecting the health of the developing fetus which in turn could lead to worse infant health outcomes and, subsequently, infant death.[13] In terms of post-birth exposure, infants who are exposed to high levels of air pollution may demonstrate

similar pathophysiological responses as adults which, in combination with their more immature immune and lung systems, could increase their risk of death as well.[14]

Given the biological plausibility, the mixed nature of the evidence on the relationship of prenatal and post-birth air pollution exposure with infant death may be due to several reasons. First, studies are often conducted in different geographic settings and over different time periods which have important implications for the type and level of pollution exposure. Second, the exposure may be measured differently across different studies, with some using modeled estimates while others using distance-weighted estimates of pollution directly from monitors. Finally, analytic strategies may be different across different studies which could potentially affect the results. For example, variables on the causal pathway, such as gestational length and birth weight, may be adjusted for without accounting for potential collider stratification bias.[1,9,15–17]

In this study, we revisit the relationship between early life ambient air pollution exposure and infant mortality in the context of the US with regard to particulate matter less than 2.5 μm in diameter ($PM_{2.5}$). Specifically, we seek to characterize the relationship of prenatal $PM_{2.5}$ with infant death in terms of both a direct pathway and an indirect pathway through preterm birth and low birth weight. We also aim to estimate the direct relationship between post-birth $PM_{2.5}$ exposure and infant death. $PM_{2.5}$ is an important pollutant to study in this context because of its known association with various indicators of fetal health such as fetal growth and organ development as well as its relationship with infant morbidity, especially respiratory diseases.[18–20]

3.3 Methods

Data sources

We used modeled estimates of daily, population-weighted mean $PM_{2.5}$ concentration at the county-level within the conterminous US between 2009 and 2014. These publicly available estimates are provided by the Centers for Disease Control and Prevention and are based on the Environmental Protection Agency's (EPA) Downscaler model.[21,22] The Downscalar model fuses together modeled estimates of air pollution concentration from the EPA's Community Multi-Scale Air Quality model with data directly from air pollution monitors.[22,23]

We used restricted access, cohort-linked birth-infant death data between 2011 and 2013 for the outcome and mediators. These data are provided by the National Center for Health Statistics and represent the universe of live births and infant deaths for children born between 2011 and 2013. These data also contain information on the pregnant woman's county of residence at delivery as well as characteristics related to parental demographics, pregnancy, delivery, and infant death.

We collected information on county-level confounders from several publicly available data sources. We extracted information on monthly average temperature and rainfall from the National Oceanic and Atmospheric Administration.[24] From the US Census Bureau, we extracted information on annual county-level demographic and economic variables including racial composition, total population, poverty rate, and median household income.[25,26] Similarly, from the Bureau of Labor Statistics, we extracted information on monthly unstandardized unemployment rate at the county-level.[27] Finally, from the Centers for Medicare & Medicaid Services, we extracted information on the annual number of physicians in a county.[28]

Outcome definition

Our primary outcome was infant death which we defined as an indicator variable which takes the value one if a baby dies in the first year of life for any reason and zero otherwise.

Exposure definition

We defined prenatal air pollution exposure as the average $PM_{2.5}$ concentration in the pregnant woman's county of residence over a nine-month period from the date of conception. We defined the exposure for a nine-month period as opposed to the actual length of gestation because the latter is a function of prenatal air pollution exposure.[9]

Similarly, we defined post-birth exposure as the average $PM_{2.5}$ concentration in the pregnant woman's county of residence in the 12-month period following the end of the nine-month prenatal period. We defined post-birth exposure over a 12-month period since the number of days alive in the first year of life may be a function of air pollution exposure. We also defined the post-birth exposure from the end of the nine-month prenatal period as opposed to the date of birth to ensure that the prenatal and post-birth exposures did not overlap temporally.

Mediator definition

Preterm birth and low birth weight were the two mediators in our analysis. Preterm birth was coded as an indicator variable which equals one if the obstetric/clinical estimate of gestational length was less than 37 weeks and zero otherwise. Low birth weight was coded as one if birth weight was reported to be less than 2,500 grams and zero otherwise.

Constructing the analytic dataset

To construct our analytic sample, we applied the following inclusion criteria to the cohort-linked birth-infant death records: 1) used the 2003 birth certificate revision; and 2) pregnant woman's county of residence had daily $PM_{2.5}$ concentration information available.

To merge the birth-infant death records with the exposure data, we first estimated the date of last menstrual period (LMP) associated with each live birth observation by using information on the year, month, day of week of birth, and the obstetric/clinical estimate of gestational length. Specifically, using information on the day of week, month, and year of birth, we randomly assigned each birth to a date of birth by assuming a uniform probability distribution over the day of week within any given month-year. Then, we subtracted the length of gestation from the estimated date of birth to get our estimate of the LMP date. Finally, we assigned the date of conception for each live birth observation by adding two weeks to the LMP date under the assumption that conception occurs, on average, two weeks after the LMP.

We then merged the birth-infant death records with the $PM_{2.5}$ data based on each pregnant woman's reported county of residence and the date of conception. Similarly, we merged the monthly temperature, rainfall, and unemployment data with the live birth-infant death records based on the pregnant woman's county of residence and month and year of conception. Finally, we merged data on annual population, poverty rate, housing value, and healthcare access based on the pregnant woman's county of residence and year of conception.

Structural Equation Model

We applied a Structural Equation Modeling (SEM) framework to estimate the direct and indirect pathways from prenatal air pollution exposure as well as the direct path from post-birth air pollution exposure to infant death. We also modeled a direct path from prematurity to low birth weight. Although these two mediators are technically measured at the same time (i.e., at birth), prematurity is a coarse measure of gestational length, and birth weight is a function of gestational length.[29] A SEM framework is appropriate for this analysis because it allows us to estimate all pathways simultaneously which improves statistical power and allows us to estimate total direct and indirect effects easily.[30] A graphical representation of our SEM is presented in Appendix Figure 3.1.

To identify the direct path from prenatal $PM_{2.5}$ exposure to adverse birth outcomes, we controlled for a variety of individual, delivery, and county-level covariates. At the individual-level, we controlled for the pregnant woman's age, race, highest level of education, marital status, parity, pre-pregnancy smoking behavior, and average $PM_{2.5}$ exposure in the nine months prior to conception. We also controlled for father's age, race, and highest level of education. In terms of delivery characteristics, we controlled for method of payment for delivery, plural delivery, and child's sex. Finally, at the county-level, we controlled for average temperature, precipitation, and unemployment rate in the nine months following conception. We also controlled for annual county racial composition, poverty rate, median housing value, and number of physicians per 1,000 individuals. We controlled for the same variables to identify the direct path from post-birth $PM_{2.5}$ exposure to infant mortality.

To identify the direct path from adverse birth outcomes to infant mortality as well as the path from preterm birth to low birth weight, we additionally controlled for cigarette smoking during pregnancy. We did not control for other variables, such as number of prenatal care visits, because they may plausibly fall on the direct pathway from prenatal air pollution exposure to infant death.

Finally, to account for unobserved time-invariant county-specific sources of confounding, we controlled for county fixed effects across all models by demeaning the data at the county-level. In addition, we flexibly modeled time trends in the outcome in all models by including month-of-year fixed effects and a linear, county-specific time trend over the study period. Appendix Table 3.1 presents detailed definitions of all covariates used in the SEM.

Statistical analysis

Before fitting the SEM, we estimated summary statistics of the exposures, mediators, covariates, and outcome. To prepare our data for the SEM, we imputed missing values in the analytic sample five times by assuming that the data were missing at random and that the observed and unobserved data followed a multivariate normal distribution. We estimated the SEM in each imputed dataset using the standard maximum likelihood method with an identity link function in all equations. Using the identity link function allowed us to easily estimate direct, indirect, and total associations. We corrected our standard errors by using Huber's cluster-robust variance estimator at the county-level which allowed us to account for correlated outcomes within a county.[31] We combined the estimates from the SEM across all imputed datasets by using Rubin's rules.[32]

The literature on air pollution and infant mortality suggests that the relationship between the exposure and the outcome may be non-linear.[33] To account for potential non-linearity, we categorized both the prenatal and post-birth exposure as follows: <8 μg/m^3; 8-10 μg/m^3; 10-12 μg/m^3; and, ≥12 μg/m^3. These cutoffs reflected the distribution of the exposure variables in our dataset (Appendix Figure 3.2) and captured the annual air quality guidance of the World Health Organization (WHO; threshold = 10 μg/m^3) and the EPA (threshold = 12 μg/m^3).[34,35]

Robustness checks

We conducted three robustness checks in this analysis. For the first two robustness checks, we redefined the post-birth exposure variable and re-fit the SEM. Specifically, we redefined the post-birth exposure as average $PM_{2.5}$ concentration in the one month and two months following the end of the nine-month prenatal period. This reflected the fact that over three-quarters of all infant deaths in the US occurred within the first two months of life (Appendix Table 3.2). In the third robustness check, we disaggregated the prenatal $PM_{2.5}$ exposure into trimester-wise exposure while defining the post-birth exposure over a 12-month period following the end of the nine-month prenatal period. Disaggregating the prenatal exposure by trimester reflected results from the literature which suggest that the prenatal air pollution-adverse birth outcome relationship varies by pregnancy trimester.[36]

Software

We used Stata/IC 15 to clean the data, create the analytic sample, and estimate the SEM model.[37] We used RStudio to estimate values of the exposure in the preconception, prenatal, and post-birth periods, create all the figures, and impute the data using the Amelia II package.[38,39]

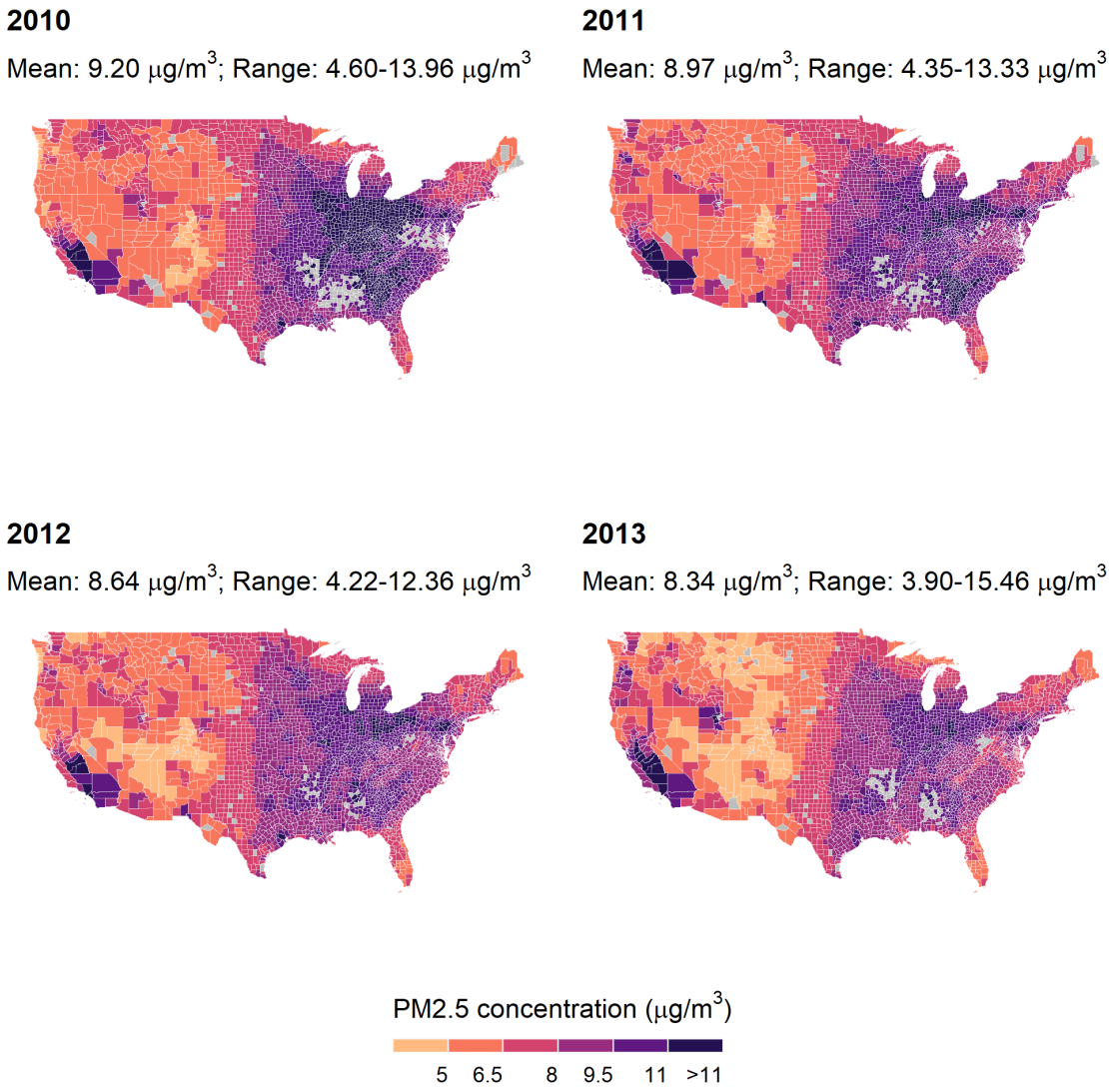
Ethical statement

This study was exempted from human subjects review by the Institutional Review Board at the Harvard T. H. Chan School of Public Health as per regulations found at 45 CFR 46.104(d) (4).

3.4 Results

Our analytic sample consisted of 10,017,357 live births and 58,913 infant deaths in 3,053 counties across 48 states in the conterminous US. Besides Alaska and Hawaii, Washington DC was also excluded from our analysis due to a lack of air pollution data. Appendix Figure 3.3 shows that the primary reason for excluding live birth and infant death observations from our analytic sample was births not being recorded using the 2003 revision of the birth certificate. Missing observations were relatively rare (<10 percent) except in the case of the father's age, race, and education, where missingness was between 12-15 percent (Appendix Table 3.3).

Figure 3.1 shows the annual average $PM_{2.5}$ concentration in counties where a conception occurred between 2010 and 2013, the earliest and latest conception years in our analytic sample. Average annual $PM_{2.5}$ concentration decreased over the study period from 9.20 $\mu g/m^3$ in 2010 to 8.34 $\mu g/m^3$ in 2013. $PM_{2.5}$ concentration also varied substantially by region: counties in the Interior Midwest, the South, and the Southwest experienced the highest levels of air pollution while counties in the Great Plains experienced the lowest levels of air pollution.

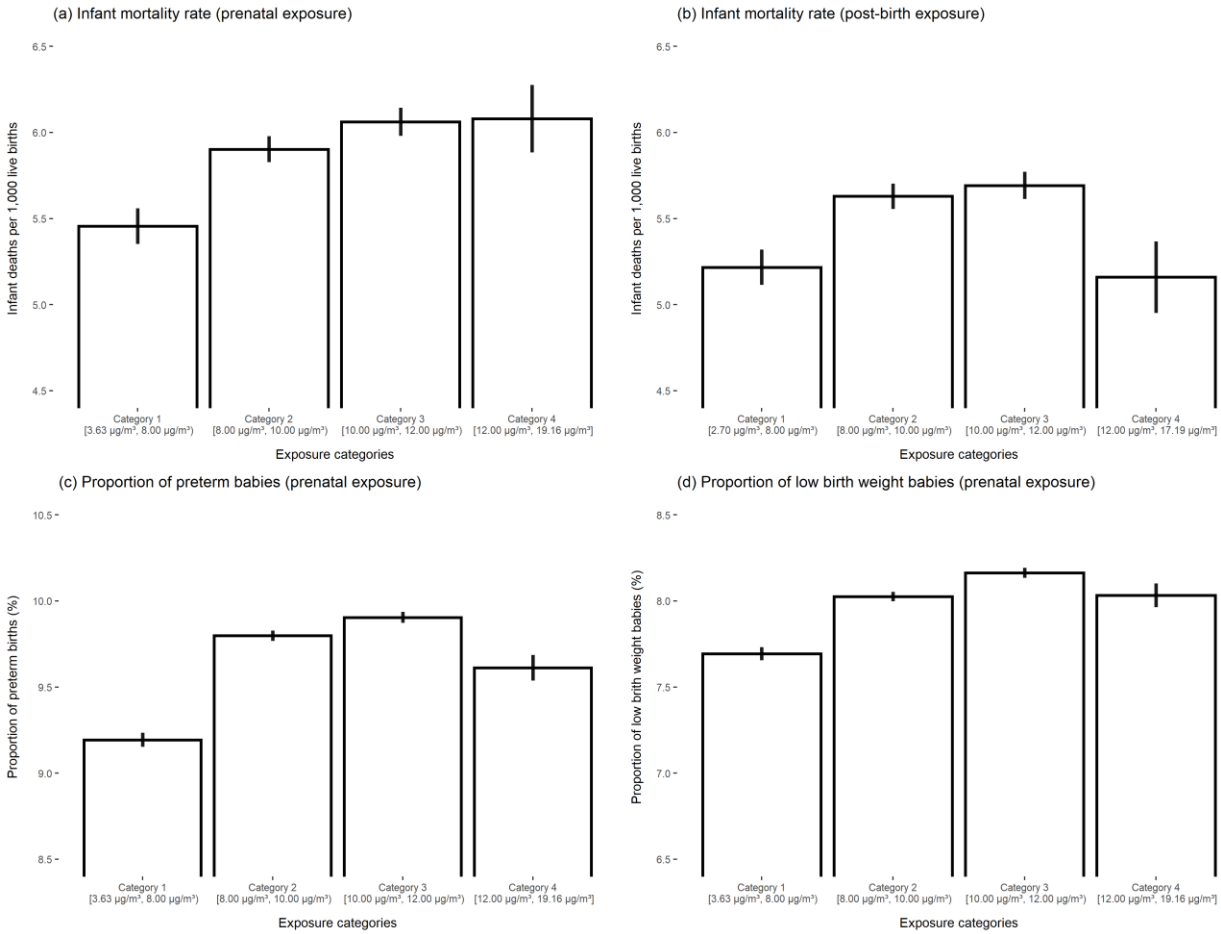


Note: Gray colored counties are counties where no birth occurred in the respective year of conception.

Figure 3 Average annual PM2.5 concentration by conception year in counties in which a conception occurred

In Figure 3.2, panels (a) and (b) respectively show unadjusted averages of infant mortality by the four categories of prenatal and post-birth $PM_{2.5}$ exposure. Infant mortality was increasing with prenatal air pollution over the study period; however, in terms of the post-birth exposure, infant mortality increased over the first three air pollution categories and decreased in the final category. Panels (c) and (d), which present unadjusted proportions of preterm birth and low birth

weight by categories of the prenatal exposure variable, follow a similar pattern with the proportion of these two mediators increasing over the first three air pollution categories and then decreasing.



Notes: The categories of both prenatal and post-birth exposure are: <8 µg/m^3; 8-10 µg/m^3; 10-12 µg/m^3; and, ≥12 µg/m^3. Panel (a) shows unadjusted infant deaths per 1,000 live births by categories of the prenatal exposure variable. Panel (b) shows unadjusted infant deaths per 1,000 live births by categories of the post-birth exposure variable. Panels (c) and (d) show unadjusted averages of preterm birth and low birth weight by categories of the prenatal exposure variable.

Figure 4 Unadjusted averages of infant mortality, preterm birth, and low birth weight by categories of the prenatal and post-birth PM2.5 exposure

Approximately three-quarters of the pregnant women in our sample were between 20-34 years, a majority were Non-Hispanic White, and more than 80 percent had a high school degree or higher

(Table 3.1). In addition, approximately 48 percent and 43 percent of mothers paid for their deliveries using private insurance and Medicaid respectively. When disaggregating these covariates by categories of the prenatal and post-birth exposure, we find that relative to the lowest air pollution category, the highest category had a lower proportion of individuals with a high school degree or more, higher proportion of deliveries paid for using Medicaid, and a higher average poverty rate (Appendix Table 3.4 and Appendix Table 3.5). Additionally, in terms of the prenatal exposure, the highest air pollution category had a higher proportion of Non-Hispanic Blacks and Hispanics relative to the lowest category (Appendix Table 3.4).

Table 3.1 Distribution of individual, delivery, and county-level covariates in the analytic sample

	Mean (SD)
	Overall
Individual-level variables	
Preconception $PM_{2.5}$ concentration ($\mu g/m^3$)	9.67 (1.76)
Mother's age	
<= 19 years	7.83 (26.86)
20-24 years	23.23 (42.23)
25-29 years	28.65 (45.21)
30-34 years	25.59 (43.64)
35-39 years	11.81 (32.27)
40-44 years	2.7 (16.21)
>= 45 years	0.19 (4.36)
Mother's race	
Non-Hispanic White	55.33 (49.72)
Non-Hispanic Black	14.42 (35.13)
Non-Hispanic Other	6.75 (25.09)
Hispanic	23.5 (42.4)
Mother's education	
No high school	16.8 (37.38)
High school / some college	46.47 (49.87)
College or more	36.74 (48.21)
Mother is married	59.54 (49.08)
Mother smoked cigarettes pre-pregnancy	11.71 (32.16)
Parity	
First child	32.97 (47.01)
Second child	28.44 (45.11)
Third or more child	38.59 (48.68)

	Mean (SD)
	Overall
Payment source for delivery	
Medicaid	43.16 (49.53)
Private insurance	47.59 (49.94)
Self-pay	4.25 (20.16)
Other	5 (21.8)
Child born female	48.82 (49.99)
Singleton delivery	96.56 (18.22)
Father's age	
<= 19 years	3.05 (17.2)
20-24 years	15.3 (36)
25-29 years	25.55 (43.61)
30-34 years	28.41 (45.1)
35-39 years	17 (37.56)
40-44 years	7.29 (26)
>= 45 years	3.41 (18.14)
Father's race	
Non-Hispanic White	56.08 (49.63)
Non-Hispanic Black	12.75 (33.36)
Non-Hispanic Other	7.44 (26.24)
Hispanic	23.73 (42.54)
Father's education	
No high school	15.96 (36.62)
High school / some college	48.62 (49.98)
College or more	35.43 (47.83)
County-level variables	
Average temperature during pregnancy	58.05 (9.54)
Average precipitation during pregnancy	3.06 (1.54)
Average unemployment during pregnancy	8.51 (2.48)
County racial composition	
Non-Hispanic White	61.99 (22.11)
Non-Hispanic Black	12.3 (12.62)
Non-Hispanic other	5.6 (6.14)
Hispanic	18.26 (18.22)
Average poverty rate	16.1 (5.47)
Median household income (USD)	52,311.3 (13,252.36)
Physicians per 1,000 individuals	0.35 (0.85)

Notes: SD = standard deviation. USD = United States dollars.

Table 3.2 presents the primary results from our analysis. The average Standardized Root Mean Square Residual (SRMR) of the overall model across all five imputed datasets was less than 0.01, which suggests a good fit with the data. Panels (a) and (b) show that the risk of preterm birth and low birth weight increased approximately linearly with increasing prenatal air pollution exposure. Panel (d) also shows that increased prenatal exposure to $PM_{2.5}$ over the study period increased the risk of infant mortality approximately linearly. Specifically, relative to the lowest exposure category, being exposed to, on average, 8-10 $\mu g/m^3$, 10-12 $\mu g/m^3$, and $\geq$12 $\mu g/m^3$ of $PM_{2.5}$ prenatally was associated with a 0.05 percentage point [95 percent confidence interval: 0.02, 0.07], 0.07 percentage point [95 percent confidence interval: 0.03, 0.10], and 0.1 percentage point [95 percent confidence interval: 0.06, 0.15] increase in the risk of infant death respectively. In terms of the post-birth $PM_{2.5}$ exposure, we estimated an increased risk of infant death between 8-10 $\mu g/m^3$ average air pollution exposure relative to the reference category of <8 $\mu g/m^3$. However, our estimates for the risk difference in the higher air pollution categories were less precise and included the null within the 95 percent confidence intervals.

Table 3.2 Estimates of the association between prenatal PM2.5 exposure, post-birth PM2.5 exposure (defined over 12 months), preterm birth, low birth weight, and infant mortality from the Structural Equation Model

	Percentage point change	95% confidence interval
Panel A: Direct association between prenatal $PM_{2.5}$ exposure and preterm birth		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.57	[0.43, 0.71]
[10.00 μg/m3 - 12.00 μg/m3)	0.95	[0.72, 1.18]
[12.00 μg/m3 - 19.16 μg/m3]	1.11	[0.72, 1.49]
Panel B: Direct association between prenatal $PM_{2.5}$ exposure and low birth weight		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.14	[0.06, 0.22]
[10.00 μg/m3 - 12.00 μg/m3)	0.23	[0.13, 0.34]
[12.00 μg/m3 - 19.16 μg/m3]	0.29	[0.14, 0.45]
Panel C: Direct association between preterm birth and low birth weight		
Preterm birth	49.67	[49.29, 50.05]
Panel D: Direct association of prenatal and post-birth $PM_{2.5}$ exposure with infant death		
Prenatal exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.05	[0.02, 0.07]
[10.00 μg/m3 - 12.00 μg/m3)	0.07	[0.03, 0.1]
[12.00 μg/m3 - 19.16 μg/m3]	0.1	[0.06, 0.15]
Post-birth exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.04	[0.01, 0.07]
[10.00 μg/m3 - 12.00 μg/m3)	0.01	[-0.03, 0.05]
[12.00 μg/m3 - 17.19 μg/m3]	0.01	[-0.06, 0.07]
Panel E: Direct association of preterm birth and low birth weight with infant death		
Preterm birth	2.00	[1.93, 2.07]
Low birth weight	3.64	[3.55, 3.73]
Number of observations	10,017,357	
Average SRMR	0	

Notes: All coefficients are expressed as percentage point changes in the respective outcomes. 95 percent confidence intervals were estimated using standard errors that were clustered at the county-level. The post-birth $PM_{2.5}$ exposure is estimated over a 12-month period following the end of the nine-month prenatal period. SRMR = Standardized Root Mean Square Residual. The average SRMR was calculated as the average of the SRMR of the Structural Equation Model fit in each of the five imputed datasets.

Table 3.3 shows that for the prenatal exposure, the total increase in risk of infant death relative to the lowest exposure category was 0.07 percentage point [95 percent confidence interval: 0.04, 0.10], 0.11 percentage point [95 percent confidence interval: 0.07, 0.15], and 0.16 percentage point [95 percent confidence interval: 0.10, 0.22] for the 8-10 $\mu g/m^3$, 10-12 $\mu g/m^3$, and ≥ 12 $\mu g/m^3$ categories respectively. In addition, between 31-43 percent of the total association of the prenatal exposure with infant mortality was through the two mediators while the remaining association was captured by the direct path from the exposure to the outcome.

Table 3.3 Direct and indirect associations of prenatal and post-birth PM2.5 exposure (defined over 12 months) with infant mortality

	Direct association		Indirect association		Total association		Proportion mediated (%)
	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	
Panel A: Direct and indirect associations of the prenatal $PM_{2.5}$ exposure							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.05	[0.02, 0.07]	0.03	[0.02, 0.03]	0.07	[0.04, 0.1]	42.86
[10.00 μg/m3 - 12.00 μg/m3)	0.07	[0.03, 0.1]	0.04	[0.03, 0.06]	0.11	[0.07, 0.15]	36.36
[12.00 μg/m3 - 19.16 μg/m3]	0.1	[0.06, 0.15]	0.05	[0.03, 0.07]	0.16	[0.1, 0.22]	31.25
Panel B: Direct and indirect associations of the post-birth $PM_{2.5}$ exposure							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.04	[0.01, 0.07]	-	-	0.04	[0.01, 0.07]	-
[10.00 μg/m3 - 12.00 μg/m3)	0.01	[-0.03, 0.05]	-	-	0.01	[-0.03, 0.05]	-
[12.00 μg/m3 - 19.16 μg/m3]	0.01	[-0.06, 0.07]	-	-	0.01	[-0.06, 0.07]	-

Notes: Direct and indirect association estimates were estimated using the Structural Equation Model presented in Table 3.2. The indirect association from the prenatal $PM_{2.5}$ exposure to infant mortality reflects two paths: prenatal exposure → preterm birth → infant mortality; and, prenatal exposure → preterm birth → low birth weight → infant mortality. The post-birth $PM_{2.5}$ exposure is estimated over a 12-month period following the end of the nine-month prenatal period. Since there is only a direct path from the post-birth exposure to infant mortality, there are no results for the indirect association between the post-birth exposure and the outcome.

Appendix Table 3.6 and Appendix Table 3.7 present results from our robustness checks in which we redefined the time over which post-birth exposure is defined. In these models, we found a positive, precisely estimated relationship between post-birth exposure and infant death. In addition, the association of the prenatal exposure with the mediators and the outcome was approximately the same as our primary results. Appendix Table 3.9 and Appendix Table 3.10 present our estimates of the direct and indirect associations of prenatal and post-birth exposure with infant death when we redefined the post-birth $PM_{2.5}$ exposure. Like our primary results, we estimated that between 29-38 percent of the association between prenatal $PM_{2.5}$ and infant death was mediated by preterm birth and low birth weight.

Appendix Table 3.8 presents results from the robustness check in which we redefined the prenatal exposure by pregnancy trimester. In general, we found a stronger positive relationship between prenatal $PM_{2.5}$ exposure and infant death in the third trimester relative to the first and second trimesters. We also found evidence for a strong and positive direct relationship between $PM_{2.5}$ exposure in the second and third trimesters and the risk of prematurity. Appendix Table 3.11 presents the direct and indirect associations from the SEM with trimester-wise prenatal exposure. Like our main results, we found that less than 50 percent of the total association between prenatal $PM_{2.5}$ and infant death in any trimester was driven by the indirect path through preterm birth and low birth weight.

3.5 Discussion

We studied the associations of prenatal and post-birth exposure to $PM_{2.5}$ with the risk of infant death for all births which took place in the US between 2011 and 2013. We found that increased

prenatal exposure to $PM_{2.5}$ over the study period was associated with an increased risk of infant death, with much of this association driven by the direct path from the exposure to the outcome rather than the indirect paths through preterm birth and low birth weight. While our primary results for the association between post-birth $PM_{2.5}$ exposure and infant death were positive but less precisely estimate, results from our robustness checks suggested a strong positive association between post-birth exposure and infant death. We also found that prenatal $PM_{2.5}$ exposure in the second and third trimesters were particularly important in impacting infant mortality over the study period. Overall, our results showed an approximately linear relationship between the exposure and outcome, which appears to be consistent with the literature.[33]

The results from our study provide evidence in favor of increased air pollution exposure during gestation and possibly during the first year of life being harmful in terms of infant health, even at levels below the threshold set by the WHO and the EPA. This implication is consistent with recent findings from Di et. al. (2018) which suggested that increased $PM_{2.5}$ exposure below the EPA standard was associated with increased risk of death among older Americans.[40] It is also nominally consistent with the results from Chay and Greenstone (2003) who demonstrated a positive association between total suspended particulates and infant death at levels below the EPA mandated threshold, although their analysis used data from the early 1980s when the EPA threshold was different from what it is currently.[8]

Our result that less than 50 percent of the association between prenatal $PM_{2.5}$ exposure and infant death can be explained by the indirect pathways suggests that there are other important mechanisms by which in utero $PM_{2.5}$ exposure may affect the risk of infant death. Previous

studies have suggested that prenatal air pollution exposure may be linked with intrauterine growth retardation or congenital heart defects, all of which could affect the risk of infant mortality.[41,42] Future research should consider explicitly characterizing these pathways.

Finally, the discrepancy between the results from our primary and robustness checks for the post-birth exposure highlights the complexities in defining the exposure in studies investigating health outcomes in early life. A strength of our analysis relative to the literature is that we define our exposure variables independent of the actual length of gestation or time alive. While this allows us to avoid defining the exposure using metrics that themselves may be a function of the exposure, it also introduces error into the exposure variables which could affect both the point estimate and its associated standard error. Future research should consider determining the most appropriate methods of defining in utero and post-birth exposures.

Our study improves on the existing literature in several ways: first, we used high quality air pollution and infant death data with wide geographical coverage. Second, following lessons from the causal mediation literature, we carefully controlled for exposure-outcome, exposure-mediator, and mediator-outcome confounders.[43] We included county fixed effects in all our models which allowed us to net out any time-invariant sources of confounding at the county-level. We also modeled the time trend flexibly at the month-of-year level and the county-level which allowed us to net out any trends in the outcome. Third, we used a SEM framework to understand the different pathways from prenatal and post-birth $PM_{2.5}$ exposure to infant death. To the best of our knowledge, our paper is one of the first to use SEM to understand this system of relationships.

Nevertheless, our analysis is still subject to several limitations. First, we were unable to disaggregate the pollution measure by type of $PM_{2.5}$ pollutant. Previous studies have shown that there is a high degree of heterogeneity in terms of particulate matter composition in the US by region and season.[44] Furthermore, studies have also suggested that early life exposure to carbonaceous $PM_{2.5}$ changes the risk of infant death but sea salt or mineral dust does not.[45] Our use of county fixed effects somewhat addresses this issue by ensuring that we only use within-county variation in the exposure for our analysis.

Second, we did not include other pollutants in our model due to a lack of high quality, daily data on them. Pollutants such as ozone, carbon monoxide, and nitrogen dioxide have been shown to be associated with infant death, while other studies have indicated that these pollutants may be correlated with $PM_{2.5}$ levels as well.[46–54] Our inability to control for these pollutants therefore suggests that the associations we present in this analysis may reflect the relationship of general pollution with prematurity, low birth weight, and infant death.

Third, the fact that air pollution data was only available at the county-level may have introduced measurement error into our exposure variable. However, this issue is somewhat mitigated by the fact that the air pollution exposure is weighted based on the county's population.

Fourth, in defining the exposure variables, we assumed that women in our sample did not move across counties during pregnancy or after the birth of their child. This may not be true for every

woman in our analytic sample although the existing literature on movement during pregnancy suggests that when women do move, the median distance travelled is under 10 kilometers.[55,56]

Finally, a small but growing literature suggests that increased in utero air pollution exposure may increase the risk of pregnancy loss.[57,58] Because we use vital statistics data, our study effectively conditions on live births to analyze the relationship between prenatal air pollution exposure and infant death. As such, our estimates may be subject to a form of selection bias known as live birth bias.[59–61] Future studies should further investigate the degree to which differential fetal loss by levels of prenatal air pollution exposure impacts investigations of the relationship between early life air pollution exposure and infant health outcomes.

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4 Air pollution and pregnancy loss in the United States

Aayush Khadka[1,2], Marianthi-Anna Kioumourtzoglou[3], Marc Weisskopf[4,5]

[1] Department of Global Health and Population, Harvard T. H. Chan School of Public Health, Boston MA 02115
[2] Graduate School of Arts and Sciences, Harvard University, Cambridge MA 02138
[3] Department of Environmental Health Sciences, Columbia University Mailman School of Public Health, New York NY 10032
[4] Department of Environmental Health, Harvard T. H. Chan School of Public Health, Boston MA 02115
[5] Department of Epidemiology, Harvard T. H. Chan School of Public Health, Boston MA 02115

4.1 Abstract

Pregnancy loss at any point during gestation is estimated to affect up to 31 percent of recognized pregnancies in the United States. Although several studies have investigated the relationship between prenatal air pollution exposure and stillbirths – defined usually as fetal death after 20 weeks of gestation – few studies exist on the association between prenatal air pollution and loss at any point during a pregnancy. We used a novel analytic framework which involves proxying pregnancy loss using data on live births to investigate the relationship between monthly, prenatal exposure to particulate matter less than 2.5 μm in diameter ($PM_{2.5}$) and pregnancy loss. We operationalized the analytic framework by using data on the universe of live births from all counties in the conterminous US between 2001-2014 and daily, county-specific, population weighted data on $PM_{2.5}$ concentration. For our primary analysis, we estimated quasi-Poisson models of the total number of live births by conception month in each county on monthly $PM_{2.5}$ exposure over nine months of gestation in the same county. We also conducted several sensitivity analyses. Our primary results suggest that average $PM_{2.5}$ exposure in the fifth and sixth months of gestation was positively associated with pregnancy loss over the study period. However, this result was not robust across all sensitivity analyses.

4.2 Background

The loss of an embryo or fetus at any point during a pregnancy – which we will collectively refer to as pregnancy loss – is estimated to affect up to 31 percent of all identified pregnancies in the United States.[1–5] At its broadest level, pregnancy loss can be categorized as miscarriage and stillbirth, which generally refers to loss before and after 20 completed weeks of gestation respectively. The risk of loss differs dramatically by the stage of gestation, with the risk being highest in the first few weeks of pregnancy and being very low in the later stages of gestation.[6–9] Both miscarriages and stillbirths are, however, important outcomes because they have been demonstrated to negatively affect the mental health of both the pregnant woman who experiences the loss and their partner.[10–15]

The etiology of pregnancy loss is complex, multifactorial, differs by stage of gestation, and not fully understood. For example, up to 50 percent of early pregnancy loss is thought to be due to chromosomal abnormalities in the embryo or fetus, but the major causes driving the remaining 50 percent are not clear.[16,17] Stillbirth may occur through multiple different processes as well, including labor related asphyxia, placental dysfunction, fetal growth restriction, and systemic inflammation.[18]

Better understood are the risk factors associated with pregnancy loss.[9,19–21] Studies investigating these risk factors have often focused on examining anatomical, nutritional, lifestyle and morbidity-related characteristics of the pregnant woman such as uterine malformation, maternal infections, obesity, smoking during pregnancy, or having poorly controlled diabetes.[21–25] More recently, scholars have turned their attention to investigating the role of air pollution exposure in

the prenatal period as a risk factor for pregnancy loss, in particular because higher levels of air pollution is associated with chromosomal abnormalities, systemic inflammation in the pregnant woman, and poor fetal health because of pollutants entering the fetus' blood stream.[26]

A limitation of the existing air pollution-pregnancy loss literature is that a majority of studies focus on stillbirth as opposed to pregnancy loss at any point during gestation.[27–38] Stillbirth is clearly an important outcome and it is measured in vital records across all states in the US; however, an exclusive focus on the air pollution-stillbirth relationship may lead to an incomplete characterization of the association between prenatal air pollution and pregnancy loss overall because the etiology of loss in the earlier stages of gestation may be quite different from the etiology of stillbirth. Furthermore, fetal death records are known to have a number of data quality issues which may also affect the reliability of studies that use them.[39]

We aimed to address this gap in the literature by estimating the association between prenatal exposure to particulate matter less than 2.5 μm in diameter ($PM_{2.5}$) and pregnancy loss at any point during gestation. Beyond the biological plausibility of this association, we focus on $PM_{2.5}$ because a few, population-level studies have provided suggestive evidence of a positive association between prenatal $PM_{2.5}$ exposure and pregnancy loss. For example, a small cohort study from 16 sites in Michigan and Texas found that increased chronic $PM_{2.5}$ exposure during pregnancy increased the hazard of loss at any point during gestation.[31]

4.3 Methods

Analytic framework

Pregnancy loss is a difficult outcome to measure because it requires a pregnancy to first be recognized and because of the stigma associated with reporting it. Recognizing these challenges, Kioumourtzoglou et. al. (2019) developed a novel analytic framework to study pregnancy loss.[27] Their framework is grounded in two fundamental ideas: first, since a conception has only two end points – live birth or pregnancy loss – we know that conditional on total conceptions at any given time point, a change in the number of live births resulting from conceptions (which we call live birth-identified conceptions) will indicate a change in the number of pregnancy losses. Second, the total number of conceptions at any time point is plausibly independent of an exposure in the prenatal period – possibly conditional on preconception exposure – because the prenatal period occurs after a conception has taken place. Together, these two ideas imply that any association between a prenatal exposure and live birth-identified conceptions conditional on preconception exposure implies an association between the same exposure and pregnancy loss in the opposite direction. We adopted this framework in our analysis to investigate the relationship between prenatal $PM_{2.5}$ exposure and pregnancy loss in the US between 2001 and 2013.

Study design

To operationalize the analytic framework, we began by defining our basic unit of time as the month of conception and the basic geographical unit as a county. Then, for each county in our analysis, we used individual-level data on live births to calculate the total number of live birth-identified conceptions by conception month. In addition, we also estimated monthly average $PM_{2.5}$ concentration at the county-level. We merged the live birth-identified conceptions data with the monthly air pollution data and used a variant of a time series design to estimate the association of total live birth-identified conceptions in a given conception month with month-by-

month average $PM_{2.5}$ concentration in the following nine months in the same county, inclusive of the conception month. Appendix Figure 4.1 illustrates our study design.

Data sources

To create the monthly $PM_{2.5}$ exposure variable, we used modeled, population-weighted estimates of daily, average $PM_{2.5}$ concentration at the county-level in the conterminous US between 2001 and 2013. These data are publicly available from the Centers for Disease Control and Prevention and are based on the US Environmental Protection Agency's (EPA) Downscalar model.[40,41]

We estimated county-specific, monthly live birth-identified conceptions using restricted access birth certificate data from the National Center for Health Statistics. These data include the universe of live births across the entire US between 2001 and 2014. Although they do not include the newborn's exact date of birth, these data include information on the year, month, and day of week of birth. They also include information on the length of gestation, each pregnant woman's demographic characteristics, pregnancy characteristics, and their county of residence at the time of delivery.

We supplemented these data sources with information on daily temperature, precipitation, and relative humidity between 2001 and 2013. Temperature data were available through the Climate Prediction Center of the National Oceanic and Atmospheric Administration (NOAA) while precipitation and relative humidity data were available through the National Centers for Environmental Prediction at the NOAA.[42,43] The temperature, precipitation, and relative humidity data were available in raster format and had a spatial resolution of 0.5 x 0.5 degrees,

2.5 x 2.5 degrees, and 2.5 x 2.5 degrees respectively. We also supplemented the exposure and outcome data with information on the annual number of women aged 15-49 years in each county between 2001 and 2013 from the US Census Bureau.[44]

Constructing the analytic dataset

We used the live birth records to calculate monthly live birth-identified conceptions for each county in the conterminous US between 2002 and 2013 using a four-step process. First, using information on the year, month, and day of week of birth, we assigned a date of birth for each live birth by assuming a uniform probability distribution over the day of week within a given month-year. For example, suppose a newborn was born on Thursday in December 2009. Since there were five Thursdays in December 2009, we assumed that the probability of the newborn being born on any one Thursday was 20 percent and randomly assigned a date of birth based on this probability.

As our second step, we used the estimated date of birth and information on the obstetric/clinical estimate of gestation to back out the date of last menstrual period (LMP) associated with each live birth. Third, by assuming that conception occurs, on average, two weeks after LMP, we estimated the date of conception and, subsequently, the month of conception associated with each birth. Finally, we aggregated the data to the county-month level by calculating the total number of live birth-identified conceptions by month of conception within each county.

Since the month of conception for live births occurring in early 2002 and late 2013 may also include live births which occurred in late 2001 and early 2014 respectively, we incorporated

information on these births to construct our aggregate-level sample of total live birth-identified conceptions. Specifically, we estimated the aggregate number of live birth-identified conceptions by county-month for each live birth in 2001 and 2014 using the four-step process described above, merged these data with the aggregated live birth-identified conceptions data for 2002-2013, and re-calculated the total number of live birth-identified conceptions in each county-month. We discarded county-month observations from 2001 and 2014 in which no live birth took place in 2002 and 2013, respectively.

In constructing the final version of the aggregate-level live birth-identified conceptions dataset, we made three further considerations. First, because California only began reporting the obstetric/clinical estimate of gestation from 2006, we removed all live birth observations from the state which were conceived prior to 2006. Second, aside from the data from California before 2006, the live birth records had approximately 3 percent of the obstetric/clinical estimate of gestational length missing. Therefore, in order to not drop any live birth observations for reasons other than structural missingness as in the case of California, we used multiple imputation to impute the variable five times using a multivariate normal distribution-based expectation maximization procedure.[45] Multiple imputation has been demonstrated to provide consistent estimates when imputing missing data; however, it also required us to assume that the missingness mechanism was missing at random conditional on the demographic and socio-economic characteristics of the pregnant women as well as the LMP-based estimate of gestational length.[46] Third, the fact that we assumed a uniform probability distribution over the day of week in a given month-year when assigning a date of birth could also introduce error into our analysis. To account for this, we repeated the procedure of assigning the date of birth five

times using different seeds. Thus, in total, we constructed 25 aggregate level live birth-identified conceptions datasets.

To each of these 25 datasets, we merged data on monthly averages of $PM_{2.5}$ concentration, temperature, precipitation, and relative humidity at the county-month of conception level. Finally, we also merged data on annual population of females between 15-49 years at the county-year of conception level with all 25 datasets.

Statistical analysis

We used the distributed lag non-linear modeling framework to estimate associations between monthly $PM_{2.5}$ exposure and total number of live birth-identified conceptions in each county-month using a count model.[47] Specifically, for total live birth-identified conceptions in each county-month, we created county-specific unconstrained, linear leads of monthly $PM_{2.5}$ exposure, temperature, precipitation, and relative humidity over the subsequent nine months inclusive of the month of conception. We also estimated the average $PM_{2.5}$ concentration in the three months prior to each county-month observation to use as a confounder in our analysis. Preconception air pollution may be a confounder because it may be correlated with prenatal air pollution and because it may affect fecundity through its effects on, for example, gamete development and quality.[48,49]

For our primary analysis, we fit a quasi-Poisson model to estimate the association between monthly $PM_{2.5}$ concentration and live birth-identified conceptions. A quasi-Poisson model is more flexible than the traditional Poisson model for count data because it allows us to account

for overdispersion in our outcome.[50] In our primary model, we controlled for county-month of year fixed effects which allowed us to make inferences within-county-month of year for the association estimates (e.g., within Middlesex County, MA in the month of January over the study period). In addition, we adjusted for monthly averages of temperature, precipitation, relative humidity, as well as preconception $PM_{2.5}$. We also included the natural log of annual female population as an offset term which allowed us to interpret the coefficient estimates as rate ratios (RR). To additionally account for trends in the outcome over the study period, we included year-specific indicator variables in our model. Finally, we corrected our standard errors by clustering at the county-month of year level.[51]

We conducted three sensitivity analyses to check the robustness of our primary results. First, we re-fit a quasi-Poisson model with county-month of year fixed effects using month-by-month $PM_{2.5}$ exposure in the nine gestation months as well as in the five months prior to conception. This model allowed us to estimate the sensitivity of our primary estimates to explicitly modeling monthly preconception exposure in the analysis. In addition, this model also allowed us to investigate if there were any patterns to the potential association between monthly preconception $PM_{2.5}$ exposure and live birth-identified conceptions. We parametrized this model in the same way as our primary model and clustered the standard errors at the county-month of year level.

Second, we modified our primary model to include 12 months of post-conception exposure as opposed to nine months. This model allowed us to test the sensitivity of our primary estimates to the inclusion of additional post-conception exposure covariates. Furthermore, it also allowed us to test our analytic framework which suggests that the coefficients associated with exposure

variables in the time period when most live births have already occurred (e.g., month 11 after conception) should be close to the null. Like our primary model, we corrected the standard errors by clustering at the county-month of year level.

Third, to determine if our primary results were sensitive to model specification, we fit a quasi-Poisson model with county fixed effects and estimated associations of total live birth-identified conceptions with month-by-month prenatal $PM_{2.5}$ exposure over a nine-month period following conception. Like our primary model, we adjusted for monthly temperature, precipitation, and relative humidity, as well as average preconception $PM_{2.5}$ exposure in the three months before conception. Unlike our primary model, we adjusted for the meteorological variables using natural cubic splines with 3 degrees of freedom. We modeled these confounders using natural cubic splines as the air pollution-confounder relationship may be nonlinear when the primary source of variation is within-county as opposed to the more restrictive within-county-month of year. We accounted for temporal trends in two ways: first, we included year-specific dummy variables to account for trends over the study period. Second, we adjusted for temporal trends in the outcome within a given year by estimating county-specific natural cubic splines with knots at every other month. Finally, we clustered the estimated standard errors at the county-level.

We conducted the primary analyses and robustness checks on all 25 analytic datasets. For each analysis, we then combined the results using Rubin's Rules and presented the estimates and 95 percent confidence intervals graphically.[46]

Software

We used Stata/MP 16.1 to clean the data and construct the analytic sample.[52] We used RStudio to estimate daily averages of temperature, precipitation, and relative humidity at the county-level, and impute the missing gestational length data.[53] We created all figures and conducted all statistical analyses in RStudio as well.

Ethical statement

Our study was deemed exempt from human subjects review by the Institutional Review Board at the Harvard T. H. Chan School of Public Health.

4.4 Results

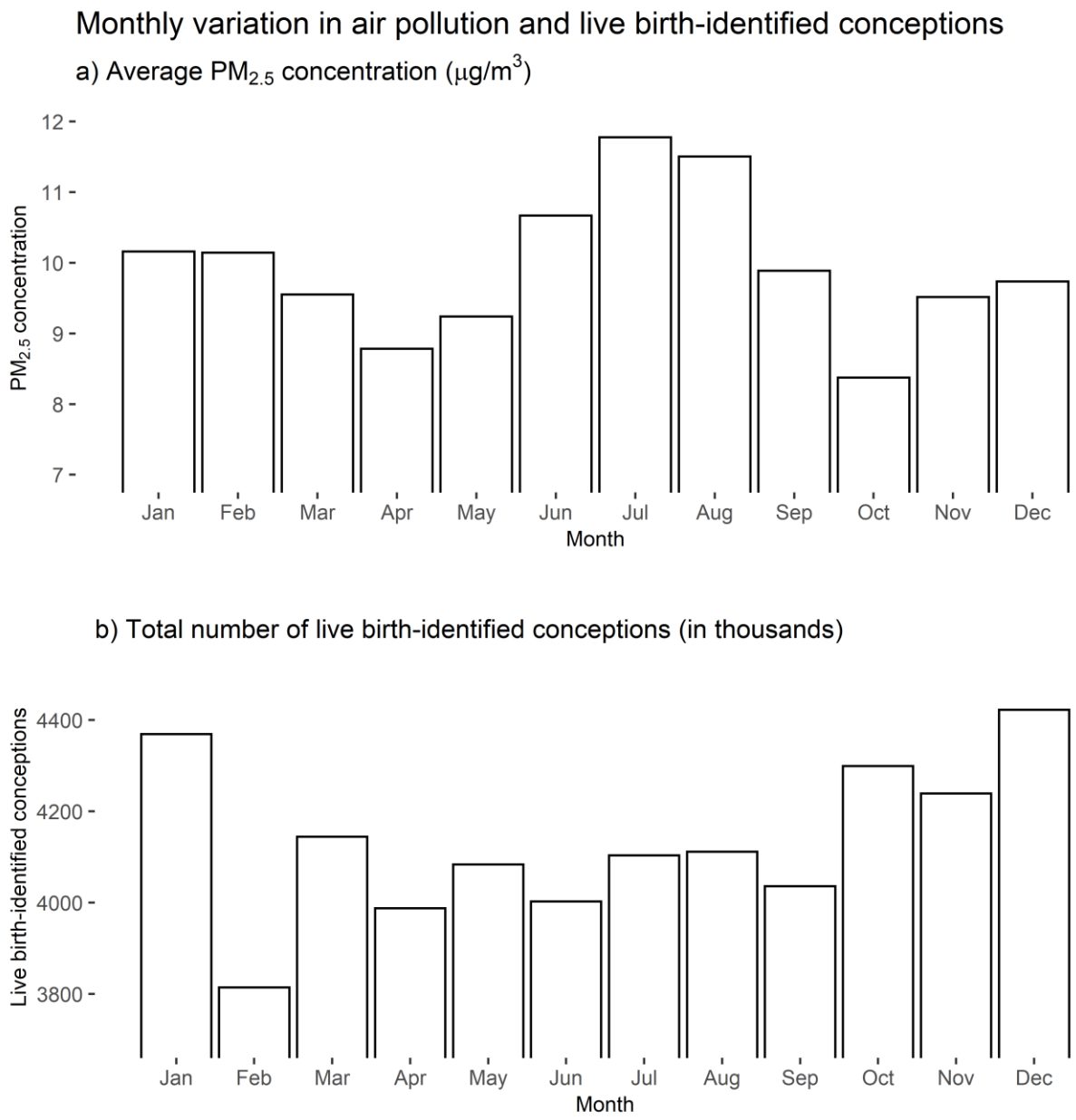
Our analytic datasets consisted of approximately 455 thousand county-month observations from 3,108 counties across the conterminous US between 2001 and 2013, the earliest and latest years in which a conception occurred in our individual-level birth records data. These county-month observations were created from approximately 47 million live birth observations. As Table 4.1 shows, the range of total live birth-identified conceptions was large with some counties having 0 births in certain county-months while others having as much as 13,683 live births. The average and median $PM_{2.5}$ concentration in our sample was relatively low at 10 $\mu g/m^3$ each. However, some county-months experienced especially high levels of air pollution since the maximum $PM_{2.5}$ concentration in our county-month level dataset was approximately 50 $\mu g/m^3$.

Table 4.1 Distribution of PM2.5 concentration and meteorological variables in the analytic data at the county-month level (N = 455,586)

	Mean (SD)	Min	Q1	Median	Q3	Max
Count of live birth-identified conceptions	103 (344)	0	10	26	67	13,683
$PM_{2.5}$ concentration ($\mu g/m^3$)	10 (3)	2	8	10	12	50
Temperature (°C)	13 (10)	-21	6	14	21	36
Precipitation (inches)	3 (2)	0	1	2	4	28
Relative humidity (%)	69 (12)	10	65	72	77	99

Notes: Summary statistics presented in this table were estimated from data at the county-month level. SD = standard deviation. Q1 = 25^{th} percentile. Q3 = 75^{th} percentile. $\mu g/m^3$ = micrograms per cubic meter. °C = degrees Celsius.

Air pollution and live birth-identified conceptions both demonstrated substantial temporal and geographic variation in our analytic data. Figure 4.1 illustrates the within-year, month-by-month variation in average $PM_{2.5}$ concentration (panel a) and total live birth-identified conceptions (panel b). Average $PM_{2.5}$ concentration peaked during the summer months of July and August and were at their lowest levels in April and October. Live birth-identified conceptions were highest in the winter months of December and January and were relatively low during the late spring to early fall period. Live birth-identified conceptions were also low in February, although this reflects the fact that February has fewer days than the other months of the calendar.



Notes: $\mu g/m^3$ = micrograms per cubic meter.

Figure 5 Monthly variation in PM2.5 exposure and live birth-identified conceptions in the data averaged between 2001 and 2013

In terms of geographic variability, counties in the Midwest region had the lowest average $PM_{2.5}$ concentration over the study period while some counties in California, Indiana, and Ohio recorded the highest average air pollution (Figure 4.2, panel a). As a proportion of the annual

15–49-year-old female population, live birth-identified conceptions appeared to be lowest in densely populated counties in the east and west coasts (Figure 4.2, panel b). In contrast, the proportion of live birth-identified conceptions appeared to be highest in some counties in the Midwest.

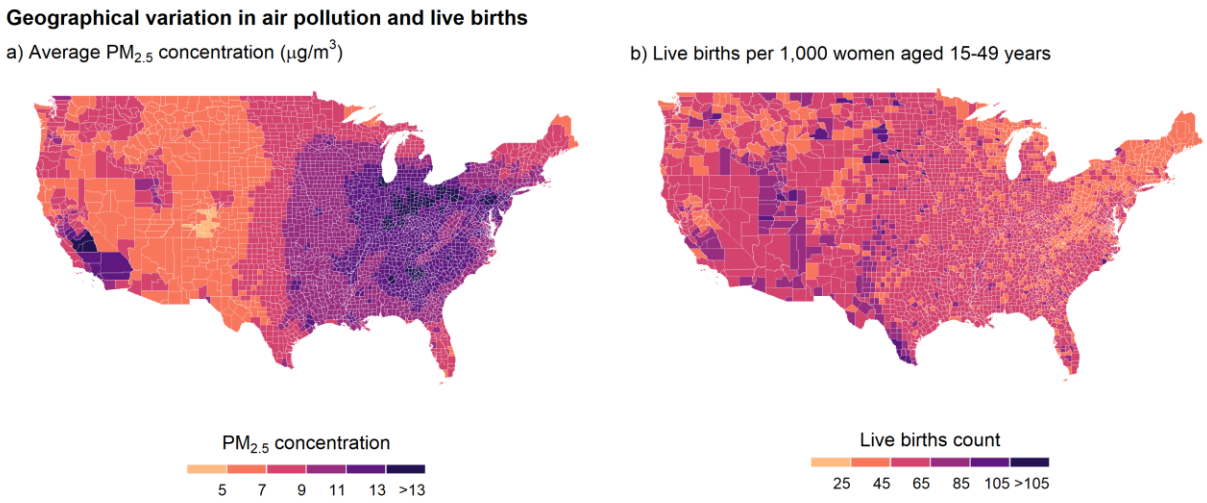
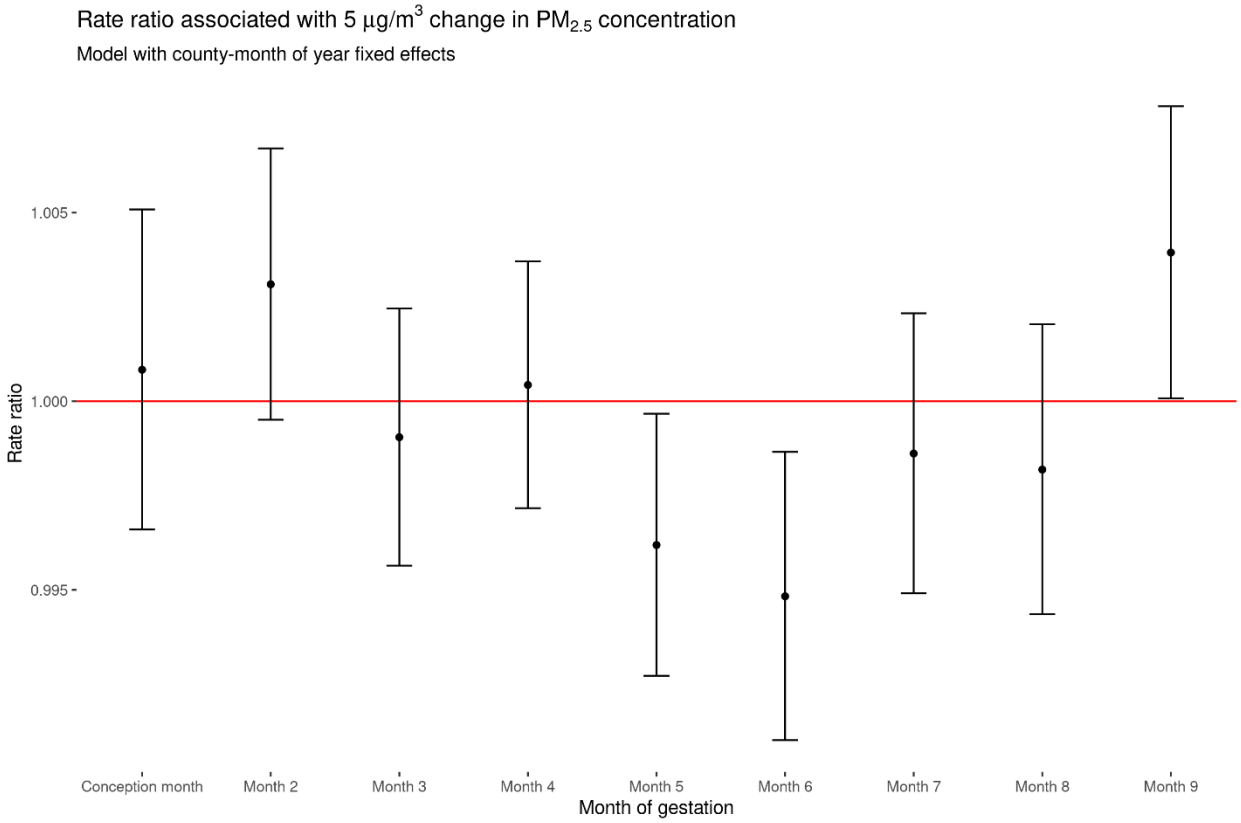


Figure 6 Average PM2.5 concentration and number of live birth-identified conceptions per 1,000 women aged 15-49 years between 2001 and 2013

Figure 4.3 presents the results from our primary model. There are three key take-aways from these results: first, these results provide some evidence to suggest that increased air pollution exposure in the fifth and sixth months of conception was associated with a decreased rate of live birth-identified conceptions over the study period. Second, these results also suggest a potential increase in the rate of live birth-identified conceptions because of increased air pollution exposure in the ninth month of conception. Third, the magnitude of the coefficient estimated for

all months, including the fifth, sixth, and ninth exposure months, are relatively small: for instance, our estimates suggest that a 5 μg/m^3 increase in $PM_{2.5}$ concentration in the sixth month of gestation was associated with a 0.5 percent decrease in the rate of live births (RR = 0.995; 95 percent confidence interval: 0.991, 0.999).

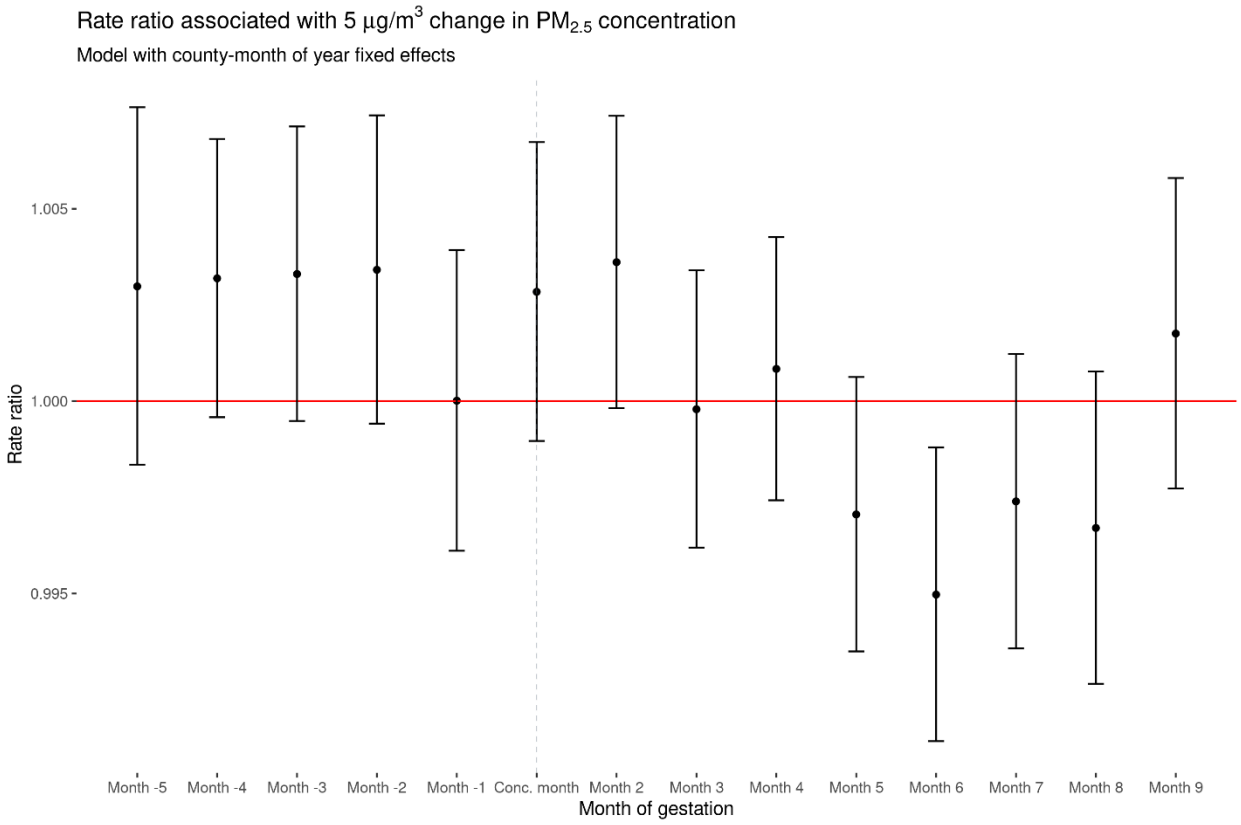


Notes: All estimates presented in the figure are from a quasi-Poisson model with county-month of year fixed effects. The model includes controls for temperature, precipitation, relative humidity, indicator variables for each year of conception in the data, and the log of county-specific annual female population between 15-49 years as an offset term. The red horizontal line indicates rate ratio = 1 (null). μg/m^3 = micrograms per cubic meter.

Figure 7 Rate ratio associated with 5 μg/m3 increase in PM2.5 concentration by month of gestation in a model with county-month of year fixed effects

Results from the first robustness check also suggests a small but negative association between exposure at month six of gestation and live birth-identified conceptions (Figure 4.4). Unlike results from the primary model, the 95 percent confidence intervals for exposures at both months

five and nine overlap the null. Interestingly, the point estimates in months five through two before conception have a positive point estimate for the association between the exposure and live birth-identified conceptions. However, the 95 percent confidence intervals of all these preconception exposure estimates overlap the null.



Notes: All estimates presented in the figure are from a quasi-Poisson model with county-month of year fixed effects. The model includes controls for temperature, precipitation, relative humidity, indicator variables for each year of conception in the data, and the log of county-specific annual female population between 15-49 years as an offset term. Conc. month = Conception month. Month -1 through -7 refers to months 1 through 7 before conception respectively. The red horizontal line indicates rate ratio = 1 (null). $\mu g/m^3$ = micrograms per cubic meter.

Figure 8 Rate ratio associated with 5 µg/m3 increase in PM2.5 concentration over nine months of gestation and five months before gestation in a model with county-month of year fixed effects

Appendix Figure 4.2 and Appendix Figure 4.3 present results from the remaining robustness checks. Appendix Figure 4.2 presents results from a model with county-month of year fixed effects and 12 months of gestational exposure. The point estimates – especially for gestational months five and six – are different from our primary results and, notably, the 95 percent confidence intervals on all point estimates overlap the null. In contrast, in a model with county fixed effects (Appendix Figure 4.3), we find some evidence to suggest a negative association between $PM_{2.5}$ exposure during the third month of gestation and live birth-identified conceptions but not in months five or six of gestation.

4.5 Discussion

In this study, we investigated the association between monthly variation in $PM_{2.5}$ exposure during gestation and total live birth-identified conceptions. From our primary model, we found some evidence to suggest that increased air pollution exposure in the fifth and sixth months of gestation were associated with a lower rate of live birth-identified conceptions and therefore an increased rate of pregnancy loss. The result for exposure at month six was robust to explicitly modeling monthly exposure over a five-month preconception period; however, the result for both months five and six were not robust to the inclusion of 12 months of post-conception $PM_{2.5}$ exposure. Our primary results were also not robust to modeling the relationship between live birth-identified conceptions and prenatal air pollution with county fixed effects as opposed to county-month of year fixed effects.

If we assume that the point estimates from our primary analysis are well identified and if we focus on month six of gestation – our most robust result – then a rate ratio of 0.995 for a 5 $\mu g/m^3$

increase in $PM_{2.5}$ concentration corresponds to approximately five fewer live births for every 1,000 conceptions since the offset population term remains constant within the county-month of year. However, given the fact that the robustness checks did not fully support the primary result, we must be careful not to overemphasize the relationship between prenatal $PM_{2.5}$ exposure and pregnancy loss. In fact, not over emphasizing the results would be somewhat in line with the published literature on gestational $PM_{2.5}$ exposure and stillbirth in the US, which has generally found a lack of association between the two. For instance, DeFranco et. al. (2015), Green et. al. (2015), and Faiz et. al. (2012) did not find evidence of an association between prenatal $PM_{2.5}$ exposure and stillbirth using vital records from Ohio, California, and New Jersey respectively.[30,32,34] Notably, Green et. al. (2015) and Faiz et. al. (2012) both found evidence of a positive association between prenatal exposure to nitrogen dioxide (NO_2) and stillbirth. This suggests that future analyses using the analytic framework in this paper may want to investigate the relationship between monthly, prenatal NO_2 exposure and live birth-identified conceptions.

It is possible, though, that our result for months five and six of gestation are what is being captured in a study by Ha et. al. (2018) who found a positive association between chronic prenatal exposure to $PM_{2.5}$ and pregnancy loss at any point during a pregnancy in a cohort of 344 singleton conceptions from 16 counties in Michigan and Texas.[31] Another study by Xue et. al. (2019) using Demographic and Health Surveys data from 33 African countries also found a positive association between average gestational $PM_{2.5}$ exposure and pregnancy loss, although their results may not be directly comparable to ours given the differing levels of air pollution exposure between African countries and the US.[38]

Despite the inconclusive results, our study extends the existing literature in two ways. Ours is one of the first studies to examine the relationship between $PM_{2.5}$ exposure and pregnancy loss at any time during a pregnancy across the entire conterminous US. Previous studies have tended to focus on narrower geographies such as states, which may limit their generalizability since pollutants demonstrate substantial geographic variability within the US.[54] Second, and most importantly, we demonstrate the application of an analytic framework that allows researchers to circumvent the traditional challenges involved in studying pregnancy loss at any point during gestation. Our study may serve as a guide for future analyses using this framework to study the role of different pollutants in impacting the risk of pregnancy loss.

However, our study is subject to some limitations as well. First, live birth-identified conceptions are used as a proxy for pregnancy loss but pregnancy loss may be spontaneous (e.g., miscarriages) or induced (e.g., induced abortions). Although we were primarily concerned with spontaneous loss in this analysis, we were unable to explicitly account for induced loss. However, since induced abortions are likely non-differential with respect to the prenatal $PM_{2.5}$ exposure, our inability to explicitly account for it may not bias our coefficient estimates although it may decrease precision. Furthermore, what is encouraging is that the analytic framework we employ in this paper can theoretically account for induced abortions, and researchers with access to better abortion data might want to do so in the future.

Second, our analytic framework requires the aggregation of individual-level data to a defined geographic and temporal level. While such aggregation allows us to proxy pregnancy loss using the count of live birth-identified conceptions, it also limits our ability to make claims about how

prenatal $PM_{2.5}$ exposure influences the risk of pregnancy loss at an individual level. This is especially important from the standpoint of healthcare providers who may want guidance on advising pregnant women about the risk of pregnancy loss at different levels of air pollution exposure. Future research may want to consider comparing the concordance of our aggregate level framework with individual-level processes either through prospective cohort analyses or simulation studies.

Third, we were unable to control for other pollutants in our model due to a lack of high quality, daily data on them. Existing multi-pollutant studies from several locations in the US have shown that pollutants such as nitrogen dioxide and sulfur dioxide may be associated with the risk of pregnancy loss.[32–34] These studies have also suggested that in some seasons, correlation between $PM_{2.5}$ and these pollutants can be positive and relatively strong (approximately 0.5).[32] Future research should consider applying our analytic framework within a multi-pollutant model context.

Finally, a key limitation of our data was the lack of an exact date of birth for each observation. We attempted to address this issue by assuming a uniform probability distribution over each day of week within a given month-year and then randomly assigning each live birth to a specific date by using information on its year, month, and day of week of birth. To account for any error introduced by this algorithm, we repeated it five times and combined our final regression results using Rubin's Rules. Nevertheless, analyses that use live birth data with the exact date of birth would provide more precise estimates of the association between prenatal $PM_{2.5}$ exposure and live birth-identified conceptions.

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5 Conclusion

The three research papers in this dissertation have aimed to understand the role of social and environmental exposures in affecting key maternal and child health outcomes in the US. Chapter 2 investigated the relationship between exposure to threatened evictions during pregnancy and the risk of prematurity. By combining birth certificate data with a novel dataset on threatened evictions, my co-authors and I demonstrated that increased exposure to threatened evictions in utero was associated with an increased risk of preterm birth. Chapter 3 examined the contribution of prenatal and post-birth $PM_{2.5}$ exposure to the risk of infant death. My co-author and I fit a Structural Equation Model using linked birth-infant death records merged with daily, county-level $PM_{2.5}$ concentration data. We showed that increased prenatal exposure was positively associated with infant mortality, with much of the association being driven by the direct path from the exposure to the outcome, while estimates for the relationship between post-birth $PM_{2.5}$ exposure and infant mortality were positive but less precisely estimated. Finally, in Chapter 4, my co-authors and I analyzed the relationship between prenatal exposure to $PM_{2.5}$ and the risk of pregnancy loss using a novel analytic framework that allowed us to sidestep some of the traditional challenges in measuring pregnancy loss. Results from our primary model suggested that increased $PM_{2.5}$ exposure in the fifth and sixth month of gestation may lead to fewer live birth-identified conceptions and hence more pregnancy loss. However, this result was not robust across all sensitivity analyses.

5.1 Contribution

This dissertation is located broadly within the literature on social and environmental determinants of maternal and child health outcomes in the US. However, all three research papers contribute to several specific sub-literatures as well.

The analysis in Chapter 2 is one of the first studies to investigate the role of threatened evictions on adverse birth outcomes at scale in the US. Chapter 2 also contributes to a dynamic literature on the various health and economic effects of evictions, which took off in the US in the late 2000's and has become especially relevant in the age of Covid-19 when millions of renters face the prospect of eviction without strong policy action from federal, state, and local governments.

There is, however, one important factor which separates Chapter 2 from the growing number of studies investigating the health effects of eviction. Unlike many of these studies, the analysis presented in Chapter 2 focuses on the threat of evictions as opposed to actual evictions.[1–4] While actual evictions as an exposure is undoubtedly important, our results suggest that earlier phases of the eviction process can be harmful for maternal and child health as well. The focus on the threat of evictions may be particularly relevant for the times we live in because, despite the federal moratorium on evictions during the Covid-19 pandemic, landlords are not restricted from filing eviction cases in local court.[5]

Chapters 3 and 4 both contribute to the literature on the role of air pollution in affecting maternal and child health outcomes. Although the analysis in Chapter 3 re-visits a question that has given rise to multiple studies, it is one of the first to investigate the air pollution-infant mortality

relationship at scale and to decompose the pathways through which prenatal air pollution exposure impacts infant mortality. A key strength of Chapter 3 is that it makes use of a very flexible statistical modeling framework (Structural Equation Models) and combines it with lessons from the causal mediation literature in Epidemiology to answer a question which has produced conflicting evidence in the literature so far.[6,7] The results from our analysis confirm many of the ideas already existing in the literature. For example, our results suggest that prenatal air pollution is an important risk factor for prematurity and low birth weight.[8] Our results also provide evidence in favor of low levels of air pollution being harmful for infant health, which is something that has been demonstrated more recently by other studies investigating the relationship between air pollution and adult mortality.[9] At the same time – and, in contrast to a number of studies in the literature – our analysis also provides robust evidence to suggest that prenatal air pollution affects infant death outside of its effect on adverse birth outcomes.

Like Chapter 3, Chapter 4 is also one of the first analyses to investigate the role of prenatal $PM_{2.5}$ exposure on pregnancy loss at any point during a pregnancy at a country-wide scale. Previous studies that have investigated this question have either limited themselves to very few study sites or have focused on understanding the prenatal air pollution-stillbirth relationship at the scale of a state (e.g., California, New Jersey, or Ohio).[10–13] Another key contribution of Chapter 4 is the use of a relatively new analytic framework to study pregnancy loss. This analytic framework, which involves proxying pregnancy loss with conceptions leading to live births, could potentially be used to study the relationship between various other exposures and pregnancy loss. As we discuss in the chapter itself, our analysis highlights both the strengths of this framework as well as its various limitations.

5.2 Future research

All three studies presented in this dissertation raise several new questions which may provide useful direction for future research.

Chapter 2: Evictions and preterm birth

One of the main questions which Chapter 2 raises is whether the associations we observed would remain unchanged, become stronger, or become weaker if we were able to accurately assign to each pregnant woman information about whether they themselves experienced a threatened eviction during pregnancy. After Chapter 2 was published, Leifheit et. al. (2020) published their results which attempted to answer this question using the Fragile Families Survey.[14] These scholars found a positive association between being threatened with evictions or homelessness and the risk of prematurity and/or low birth weight. Future research may want to expand on the analysis presented in Chapter 2 and by Leifheit et. al. (2020) to investigate the threatened evictions-preterm birth relationship at scale.[14]

Another question which Chapter 2 raises is about whether there are spillover effects of threatened evictions on adverse birth outcomes. For example, researchers could use data similar to those used by Currie et. al. (2019) to investigate if living nearby someone who is evicted affects one's own risk of delivering a baby prematurely.[15] Alternatively, one might ask if having a friend or a relative threatened with eviction also affects one's risk of delivering a baby premature or low birth weight. Answering these questions may have important implications for

the care and counseling we provide women during pregnancy to ensure that the health of the pregnant woman and the baby is not compromised.

A third question which Chapter 2 raises is whether policy and programmatic initiatives can help modify the threatened eviction-adverse birth outcome relationship. This may be a particularly fruitful line of inquiry for researchers because cities across the US have started implementing a variety of policies to combat the threat of eviction. For example, voters in San Francisco passed Prop F: Tenant Right to Counsel in 2018, which tenant rights advocates and eviction scholars argue may have a major beneficial impact on renters who have been threatened with legal eviction notices.[16,17] The right to counsel law ensures that tenants will be represented by a lawyer in local court, which many argue will automatically improve their chances of not being evicted from their current residence. Similarly, cities such as New York, San Francisco, and Seattle have some version of Just Cause Eviction laws – i.e., laws which protect tenants from being evicted at the whim of the landlord.[18] Both the Right to Counsel and Just Cause Eviction laws could provide researchers with natural experiments which they can then use to determine the moderating impact of such policies on the threatened eviction-adverse birth outcome relationship.

Chapter 3: Air pollution and infant mortality

One question which Chapter 3 raises is if the associations we estimated are being driven by specific types of $PM_{2.5}$ pollutants. Goyal et. al. (2019) have shown using data from several African and Asian countries that exposure to carbonaceous $PM_{2.5}$ is strongly associated with the risk of infant death but exposure to naturally occurring dust and sea salt is not.[19] As has been

well documented, $PM_{2.5}$ composition in the US demonstrates wide geographic and temporal variability, with black carbon concentrations being especially high in urban areas of the country.[20] Future studies may want to disentangle the differential effect of the different types of $PM_{2.5}$ pollutants on infant death in the US as well.

A related line of inquiry would be to investigate the role of pollutants other than $PM_{2.5}$ in affecting the risk of infant death. This question has been asked by numerous scholars – such as Currie et. al. (2011) who analyzed the relationship between traffic air pollution and infant health – but has not, to the best of my knowledge, been investigated at a national scale.[21] With the increasing availability of national air pollution data, it may be fruitful for researchers to adopt the approach we present in Chapter 3 using data on different pollutants.

A third line of inquiry which may be important is the use of causal mediation analysis methods to decompose the causal effect of prenatal air pollution on infant death into a direct path and indirect paths through adverse birth outcomes. There are two important strengths of the causal mediation methods relative to the Structural Equation Modeling framework we use. First, causal mediation methods allow us to model exposure-mediator interactions while also allowing us to estimate indirect effects. Second, causal mediation methods have a better developed set of sensitivity analyses, which can help us get a better sense of the robustness of our estimates. Yet causal mediation methods come at a cost as well. Perhaps most importantly, they do not allow us to estimate all paths from multiple exposure through multiple mediators to the outcome at once. However, it may still be of interest to estimate each mediated path separately, and in this case,

methods currently being developed by scholars such as Kara Rudolph and Mark van der Laan to identify stochastic direct and indirect effects may be of particular interest.[22]

Chapter 4: Air pollution and pregnancy loss

The results presented in Chapter 4 involved using a framework that aggregated live births to a specific temporal and geographic level and using it as a proxy outcome to understand the relationship between prenatal air pollution exposure and pregnancy loss. However, because of the aggregation, questions remain about how the results presented in Chapter 4 correspond to individual-level effects. To test the correspondence of the aggregate level study design to individual-level relationships, researchers could consider two types of studies: first, researchers could consider conducting prospective cohort studies along the lines of Ha et. al. (2018) by following individuals who are planning on conceiving over the study period.[23] Second, researchers could also consider doing simulation analyses where they specify individual-level survival models of conception, pregnancy loss, and live birth and then aggregate live birth data to understand how accurately the analytic framework we use in Chapter 4 captures these individual-level effects.

Separately, Chapter 4 also raises questions about the relationship between other pollutants and pregnancy loss. Some of the individual-level studies that have examined the relationship between nitrogen dioxide and stillbirth in the US have found a positive association.[24,25] Given this, it may be useful for researchers to investigate the relationship between nitrogen dioxide and pregnancy loss overall using the analytic framework presented in Chapter 4 and at scale.

5.3 References

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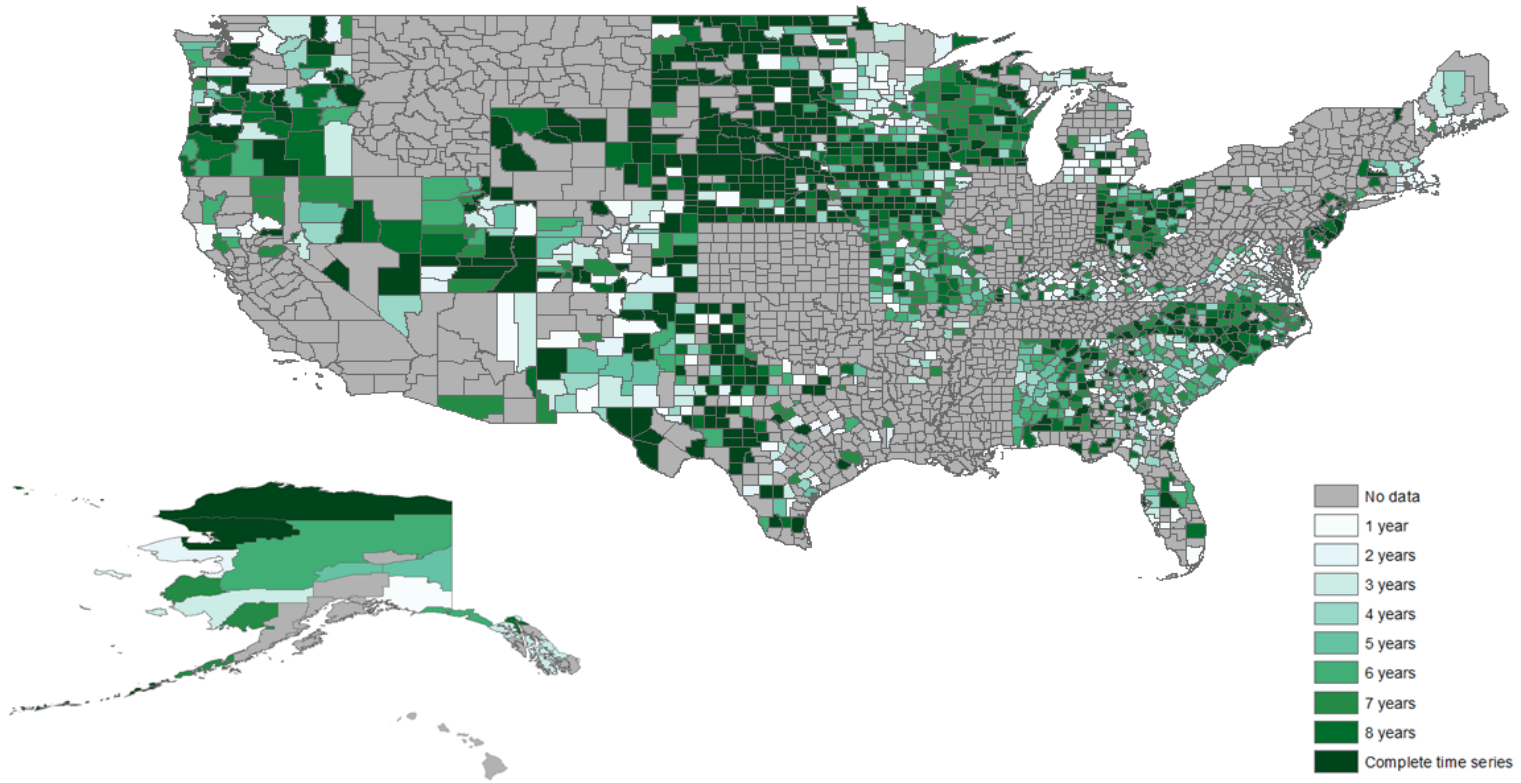
Supplementary materials for Chapter 2

Note: For states that adopted the 2003 birth certificate revision mid-year, we used birth records starting from the subsequent year. For example, Rhode Island adopted the 2003 birth certificate revision some time after January 1, 2014. Therefore, we used birth records from Rhode Island starting 2015 only.

Appendix Figure 2.1 States included in the analysis and the year from which they adopted the 2003 birth certificate revision



Notes: All counties colored in green are counties that have at least one year's worth of eviction case filing data. The darker the shade of green, the longer the length of the time series for a given county. The legend displays the total number of years for which we have eviction case filing data for any given county.

Appendix Figure 2.2 Counties represented in our analysis and the length of the time series associated with each county

Appendix Table 2.1 Frequency of missing data among variables in the analytic dataset

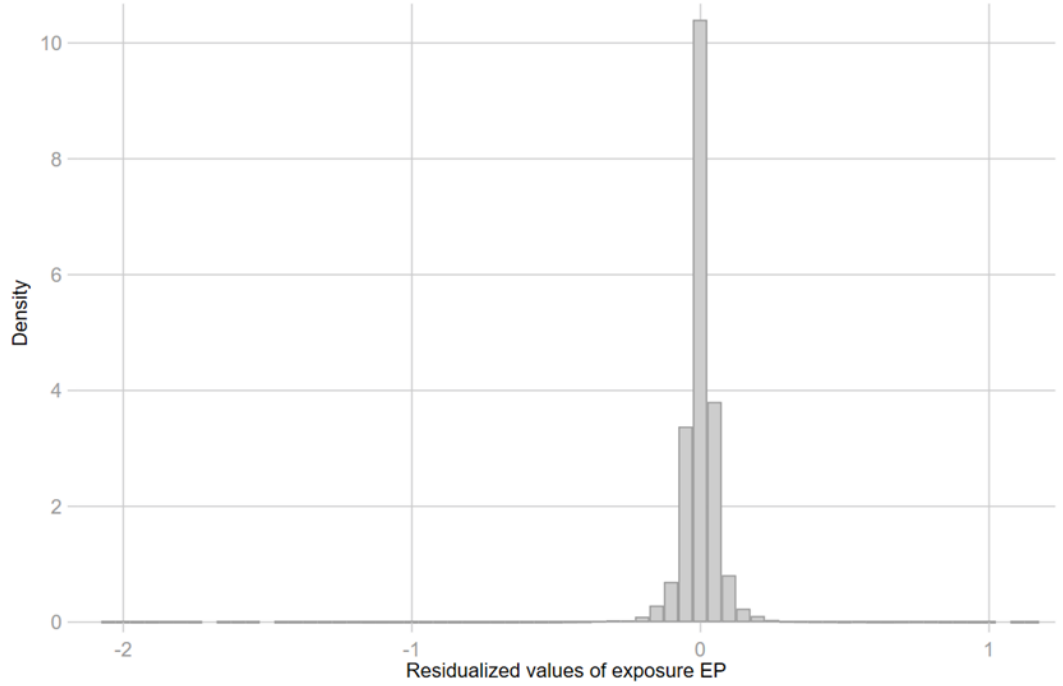
	Number of missing observations	Total number of observations	Percentage missing
Birth weight in grams[1]	5,994	7,324,812	0.08
Average eviction case filing during second and third trimesters	2,636	7,324,812	0.04
Average eviction case filing in the 9-month pre-pregnancy period	42,257	7,324,812	0.58
Parity	44,289	7,324,812	0.6
Method of payment	111,537	7,324,812	1.52
Average unemployment rate in county of residence during the entire pregnancy[*]	2,636	7,324,812	0.04
Obstetric/clinical estimate of gestational length in weeks[2]	0	7,324,812	0
Average eviction case filing during the entire pregnancy	0	7,324,812	0
Average eviction case filing during first trimester	0	7,324,812	0
Maternal age	0	7,324,812	0
Maternal education	0	7,324,812	0
Mother's race	0	7,324,812	0
Year of conception[3]	0	7,324,812	0
Month of conception[3]	0	7,324,812	0
County of residence[3]	0	7,324,812	0
State of residence	0	7,324,812	0
Child's sex	0	7,324,812	0
Delivery type (singleton only)	0	7,324,812	0
Average poverty rate in county of residence in the year of conception[*]	0	7,324,812	0
County of residence's urban/rural classification based on NCHS[*]	0	7,324,812	0

Notes: [1]The low birth weight indicator variable was defined based on the birth weight variable. [2]The preterm birth indicator variable was defined based on the obstetric/clinical estimate of gestational length. [3]These variables were used to define the county-specific linear time trend. [*]These variables were defined at the county-of-residence level.

Appendix Table 2.2 Distribution of individual and county-level characteristics in 2009 and 2015 by tertiles defined using average exposure to eviction case filings over the duration of the pregnancy (exposure EP)

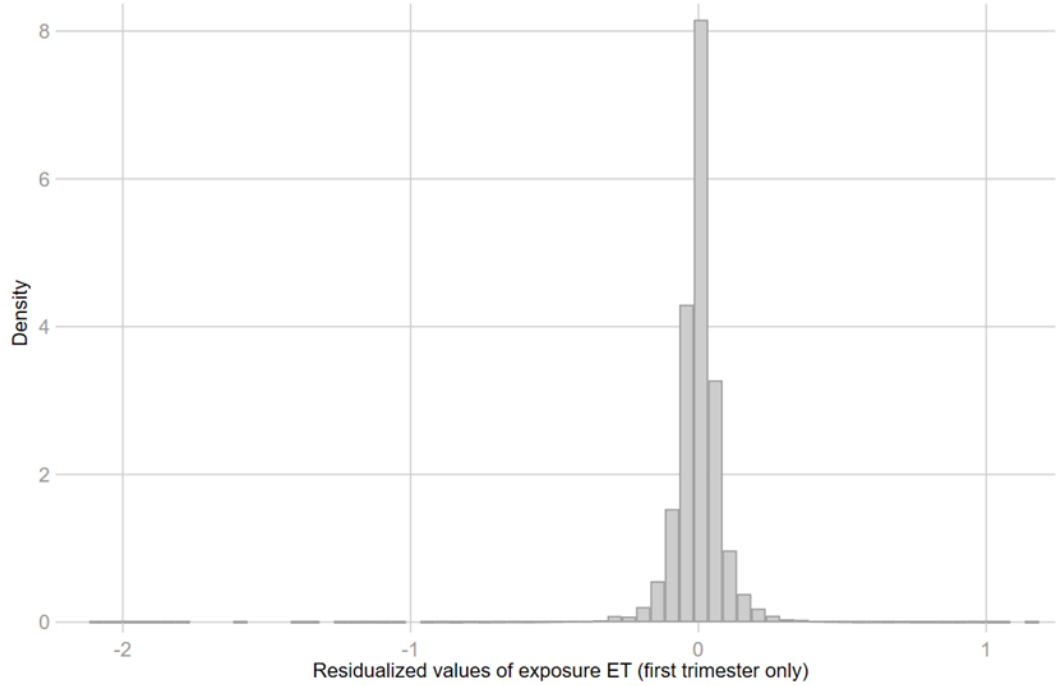
	2009			2015		
	Low exposure tertile	Medium exposure tertile	High exposure tertile	Low exposure tertile	Medium exposure tertile	High exposure tertile
Individual-level						
Mean age (years)	27.24 [27.21, 27.27]	27.20 [27.18, 27.23]	26.97 [26.95, 27]	28.26 [28.24, 28.28]	28.26 [28.25, 28.28]	28.29 [28.27, 28.32]
Percent of women with no High School	18.18 [18.01, 18.35]	20.43 [20.25, 20.61]	23.32 [23.14, 23.51]	12.59 [12.48, 12.7]	16.00 [15.88, 16.11]	16.17 [16.03, 16.3]
Percent of women with High School but no tertiary degree	47.6 [47.34, 47.78]	46.9 [46.68, 47.13]	48.4 [48.16, 48.6]	45.9 [45.69, 46.01]	47.2 [47.02, 47.34]	46.3 [46.12, 46.49]
Percent of women with a tertiary degree	34.3 [34.05, 34.47]	32.7 [32.46, 32.88]	28.3 [28.1, 28.5]	41.6 [41.4, 41.72]	36.8 [36.67, 36.97]	37.5 [37.35, 37.71]
Percent White (non-Hispanic)	69.8 [69.57, 69.96]	54.7 [54.47, 54.91]	39.4 [39.18, 39.61]	73.0 [72.84, 73.13]	50.7 [50.55, 50.86]	41.2 [40.99, 41.35]
Percent Black (non-Hispanic)	5.0 [4.94, 5.13]	14.6 [14.45, 14.77]	23.9 [23.75, 24.13]	7.3 [7.21, 7.38]	18.6 [18.45, 18.69]	31.5 [31.35, 31.69]
Percent Hispanic	18.0 [17.83, 18.16]	25.5 [25.33, 25.71]	31.1 [30.86, 31.27]	11.7 [11.6, 11.82]	24.9 [24.81, 25.08]	20.7 [20.57, 20.87]
Percent Other races	7.2 [7.1, 7.32]	5.2 [5.08, 5.28]	5.6 [5.5, 5.7]	8.0 [7.92, 8.09]	5.8 [5.71, 5.86]	6.6 [6.5, 6.68]
Percent paying for delivery using Medicaid	38.7 [38.53, 38.95]	42.4 [42.2, 42.64]	43.6 [43.36, 43.8]	37.8 [37.65, 37.97]	43.4 [43.27, 43.58]	44.8 [44.63, 45]
County-level						
Average unemployment rate	7.5 [7.48, 7.51]	7.8 [7.83, 7.85]	7.7 [7.64, 7.66]	5.0 [5.01, 5.02]	5.4 [5.36, 5.36]	5.7 [5.69, 5.69]
Average poverty rate	12.1 [12.06, 12.1]	13.5 [13.53, 13.57]	15.1 [15.08, 15.1]	14.2 [14.18, 14.21]	16.6 [16.59, 16.61]	17.4 [17.41, 17.43]
Percent metropolitan counties	65.1 [64.85, 65.26]	95.7 [95.66, 95.84]	98.5 [98.49, 98.6]	67.7 [67.53, 67.84]	93.8 [93.74, 93.9]	96.9 [96.84, 96.97]

Note: 95 percent confidence intervals in brackets



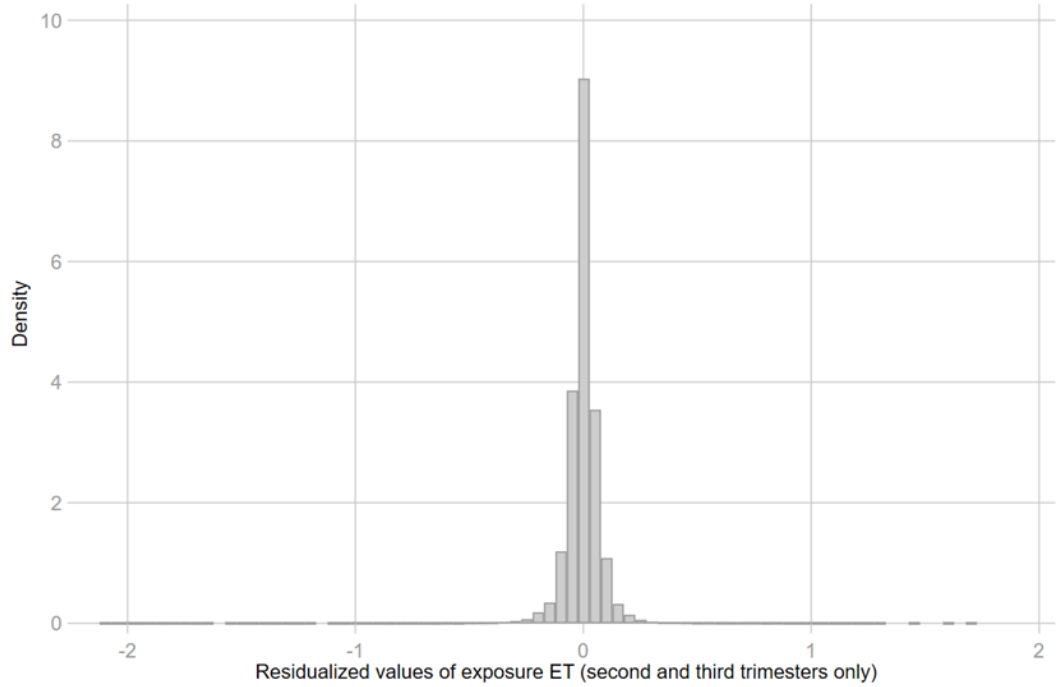
Mean	Standard deviation	Median
0	0.07	0

Appendix Figure 2.3 Distribution of residuals after conditioning average eviction case filings over the duration of a pregnancy (exposure EP) on county of residence fixed effects, state-of-residence-year-month fixed effects, and county-specific linear time trend



Mean	Standard deviation	Median
0	0.09	0

Appendix Figure 2.4 Distribution of residuals after conditioning average eviction case filings in the first trimester (exposure ET) on county of residence fixed effects, state-of-residence-year-month fixed effects, and county-specific linear time trend



Mean	Standard deviation	Median
0	0.08	0

Appendix Figure 2.5 Distribution of residuals after conditioning average eviction case filings in the second and third trimesters (exposure ET) on county of residence fixed effects, state-of-residence-year-month fixed effects, and county-specific linear time trend

Appendix Table 2.3 Results from falsification check using exposure to average eviction case filings in the nine months prior to conception as a negative control

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing in the nine months prior to conception	0.12% point [-0.55, 0.8]	0.01 weeks [-0.04, 0.07]	-1.15 grams [-9.57, 7.26]	0.13% point [-0.24, 0.5]
Average value of the outcome in the analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	7,324,812	7,324,812	7,324,812	7,324,812

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for threatened eviction exposure over the duration of the pregnancy, mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.4 Associations between exposure to average eviction case filings over the duration of the pregnancy [exposure EP] and birth outcomes in the verified cases sub-sample

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing over pregnancy	1.28% point	-0.06 weeks	-13.99 grams	0.87% point
	[0.07, 2.49]	[-0.16, 0.03]	[-33.45, 5.47]	[0.04, 1.7]
Average value of the outcome in the verified cases sample	8.20%	38.59 weeks	3,297.33 grams	6.58%
Average value of the outcome in the primary analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	7,027,351	7,027,351	7,027,351	7,027,351

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.5 Associations between exposure to average eviction case filings over the duration of the pregnancy [exposure EP] and birth outcomes in the complete time series sub-sample

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing over pregnancy	0.45% point [-0.25, 1.14]	-0.01 weeks [-0.06, 0.03]	-4.74 grams [-17.51, 8.03]	0.31% point [-0.24, 0.86]
Average value of the outcome among counties with complete panel of exposure data	7.89%	38.63 weeks	3,310.39 grams	6.34%
Average value of the outcome in the primary analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	3,254,301	3,254,301	3,254,301	3,254,301

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.6 Associations between exposure to average eviction case filings over the duration of the pregnancy [exposure EP] and birth outcomes in the five-year time series sub-sample

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing over pregnancy	1.42% point [0.5, 2.34]	-0.08 weeks [-0.14, 0.01]	-16.88 grams [-32.09, 1.67]	0.92% point [0.3, 1.55]
Average value of the outcome among counties with exposure information for five years or more	8.11%	38.60 weeks	3,301.84 grams	6.49%
Average value of the outcome in the primary analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	6,364,818	6,364,818	6,364,818	6,364,818

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.7 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and birth outcomes in the verified cases sub-sample

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing in the first trimester	-0.03% point [-0.47, 0.41]	0.000 weeks [-0.03, 0.03]	0.92 grams [-5.88, 7.72]	0.040% point [-0.31, 0.39]
Z-score of average case filing in the second and third trimesters	0.96% point [-0.02, 1.93]	-0.04 weeks [-0.13, 0.05]	-10.02 grams [-26.08, 6.05]	0.648% point [-0.07, 1.36]
Average value of the outcome in the verified cases sample	8.20%	38.59 weeks	3,297.33 grams	6.58%
Average value of the outcome in the primary analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	7,027,351	7,027,351	7,027,351	7,027,351

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.8 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and birth outcomes in the complete time series sub-sample

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing in the first trimester	-0.02% point [-0.33, 0.3]	-0.009 weeks [-0.04, 0.02]	-3.10 grams [-10.08, 3.87]	0.07% point [-0.19, 0.32]
Z-score of average case filing in the second and third trimesters	0.25% point [-0.28, 0.77]	0.01 weeks [-0.04, 0.05]	0.84 grams [-8.94, 10.63]	0.15% point [-0.31, 0.61]
Average value of the outcome among counties with complete panel of exposure data	7.89%	38.63 weeks	3,310.39 grams	6.34%
Average value of the outcome in the primary analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	3,254,301	3,254,301	3,254,301	3,254,301

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.9 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and birth outcomes in the five-year time series sub-sample

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing in the first trimester	0.08% point [-0.32, 0.48]	-0.005 weeks [-0.04, 0.03]	-0.24 grams [-7.24, 6.77]	0.06% point [-0.26, 0.38]
Z-score of average case filing in the second and third trimesters	0.88% point [0.15, 1.62]	-0.04 weeks [-0.1, 0.02]	-10.20 grams [-22.07, 1.66]	0.59% point [0.02, 1.15]
Average value of the outcome among counties with exposure information for five years or more	8.11%	38.60 weeks	3,301.84 grams	6.49%
Average value of the outcome in the primary analytic sample	8.18%	38.59 weeks	3,298.56 grams	6.57%
Average value of the outcome in the United States over the study period	9.81%	38.49 weeks	3,269.06 grams	8.07%
Observations	6,364,818	6,364,818	6,364,818	6,364,818

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.10 Associations between exposure to average eviction case filings over the duration of a pregnancy [exposure EP] and preterm birth by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing over pregnancy	1.06% point [0.17, 1.95]	2.13% point [-0.06, 4.33]	0.69% point [-1.05, 2.43]	1.27% point [-0.64, 3.18]
Average proportion of preterm birth in the analytic sample	7.18%	11.59%	8.14%	7.84%
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect percentage point change in the risk of preterm birth for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.11 Associations between exposure to average eviction case filings over the duration of a pregnancy [exposure EP] and gestational length by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing over pregnancy	-0.07 weeks [-0.14, 0.01]	-0.16 weeks [-0.35, 0.03]	0.01 weeks [-0.13, 0.14]	0.02 weeks [-0.17, 0.21]
Average length of gestation in the analytic sample	38.71 weeks	38.25 weeks	38.58 weeks	38.57 weeks
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect change in gestational length measured in weeks for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.12 Associations between exposure to average eviction case filings over the duration of a pregnancy [exposure EP] and birth weight by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing over pregnancy	-14.33 grams [-28.16, 0.51]	-27.38 grams [-60.3, 5.55]	-11.27 grams [-39.95, 17.41]	10.38 grams [-29.67, 50.43]
Average birth weight in the analytic sample [grams]	3,369.33 grams	3,111.65 grams	3,288.01 grams	3,223.35 grams
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect change in birth weight measured in grams for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.13 Associations between exposure to average eviction case filings over the duration of a pregnancy [exposure EP] and low birth weight by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing over pregnancy	0.88% point [0.25, 1.5]	1.69% point [0.16, 3.22]	0.12% point [-0.88, 1.11]	-0.57% point [-2.1, 0.96]
Average proportion of low birth weight in the analytic sample	5.19%	11.34%	6.11%	7.09%
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect percentage point change in the risk of low birth weight for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.14 Associations between exposure to average eviction case filings over the duration of the pregnancy [exposure EP] and birth outcomes among women who paid for their delivery using Medicaid

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing over pregnancy	1.03% point [-0.12, 2.18]	-0.03 weeks [-0.13, 0.06]	-8.43 grams [-28.75, 11.89]	0.63% point [-0.19, 1.45]
Average value of the outcome among women using Medicaid to pay for deliveries	9.37%	38.48 weeks	3,225.87 grams	8.20%
Observations in analytic sample	3,083,408	3,083,408	3,083,408	3,083,408

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.15 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and preterm birth by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing in the first trimester	0.18% point [-0.23, 0.6]	0.37% point [-0.51, 1.25]	-0.61% point [-1.65, 0.43]	-0.67% point [-1.76, 0.42]
Z-score of average case filing in the second and third trimesters	0.62% point [-0.07, 1.31]	1.86% point [-0.02, 3.73]	1.10% point [-0.05, 2.26]	1.96% point [0.16, 3.75]
Average proportion or preterm birth in the analytic sample	7.18%	11.59%	8.14%	7.84%
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect percentage point change in the risk of preterm birth for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.16 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and gestational length by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing in the first trimester	-0.01 weeks [-0.04, 0.02]	-0.04 weeks [-0.11, 0.04]	0.03 weeks [-0.04, 0.09]	0.04 weeks [-0.04, 0.12]
Z-score of average case filing in the second and third trimesters	-0.04 weeks [-0.1, 0.01]	-0.13 weeks [-0.32, 0.06]	0.00 weeks [-0.11, 0.11]	-0.05 weeks [-0.21, 0.12]
Average length of gestation in the analytic sample	38.71 weeks	38.25 weeks	38.58 weeks	38.57 weeks
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect change in gestational length measured in weeks for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.17 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and birth weight by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing in the first trimester	-1.43 grams [-8.74, 5.88]	-5.45 grams [-19.44, 8.53]	3.07 grams [-10.12, 16.26]	11.51 grams [-9.02, 32.05]
Z-score of average case filing in the second and third trimesters	-9.21 grams [-20.95, 2.53]	-23.61 grams [-56.64, 9.42]	-10.97 grams [-32.37, 10.43]	-3.03 grams [-37.66, 31.6]
Average birth weight in the analytic sample [grams]	3,369.33 grams	3,111.65 grams	3,288.01 grams	3,223.35 grams
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect change in birth weight measured in grams for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.18 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and low birth weight by racial sub-groups

Racial category	White non-Hispanic	Black non-Hispanic	Hispanic	Other races
Z-score of average case filing in the first trimester	0.19% point [-0.09, 0.46]	0.35% point [-0.41, 1.11]	-0.34% point [-0.96, 0.28]	-0.25% point [-1.24, 0.74]
Z-score of average case filing in the second and third trimesters	0.56% point [0.03, 1.09]	1.44% point [-0.12, 2.99]	0.38% point [-0.44, 1.2]	-0.24% point [-1.85, 1.37]
Average proportion of low birth weight in the analytic sample	5.19%	11.34%	6.11%	7.09%
Observations in analytic sample	4,100,995	1,277,406	1,463,664	482,747

Notes: All coefficient estimates reflect percentage point change in the risk of low birth weight for a standard deviation change in average case filings over a pregnancy. 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's highest level of education, parity, child's sex, method of payment for delivery, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

Appendix Table 2.19 Associations between exposure to average eviction case filings by pregnancy trimester [exposure ET] and birth outcomes among women who paid for their delivery using Medicaid

Outcome	Preterm birth	Gestational length	Birth weight	Low birth weight
Z-score of average case filing in the first trimester	-0.05% point [-0.61, 0.5]	0.02 weeks [-0.02, 0.06]	7.05 grams [-2.27, 16.38]	-0.26% point [-0.73, 0.21]
Z-score of average case filing in the second and third trimesters	1.06% point [0.05, 2.08]	-0.06 weeks [-0.15, 0.04]	-15.84 grams [-33.66, 1.98]	0.94% point [0.1, 1.78]
Average value of the outcome among women using Medicaid to pay for deliveries	9.37%	38.48 weeks	3,225.87 grams	8.20%
Observations in analytic sample	3,083,408	3,083,408	3,083,408	3,083,408

Notes: 95 percent confidence intervals in brackets constructed using clustered standard errors at the county-level. All results presented in the table are pooled estimates from Ordinary Least Squares regressions estimated on five imputed datasets. All models controlled for county of residence fixed effects, state-of-residence-year-and-month fixed effects, and a linear county-specific time trend. All models also controlled for mother's age, a quadratic age term, mother's race, mother's highest level of education, parity, child's sex, urban-rural classification of county of residence, county of residence's annual poverty rate, and county of residence's unstandardized, monthly unemployment rate.

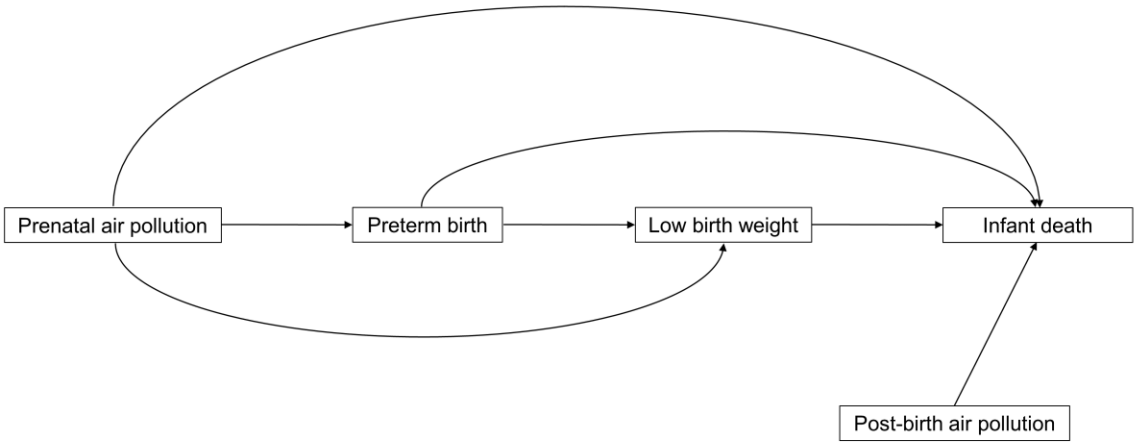
Supplementary materials for Chapter 3

Appendix Table 3.1 Covariate definition and data source

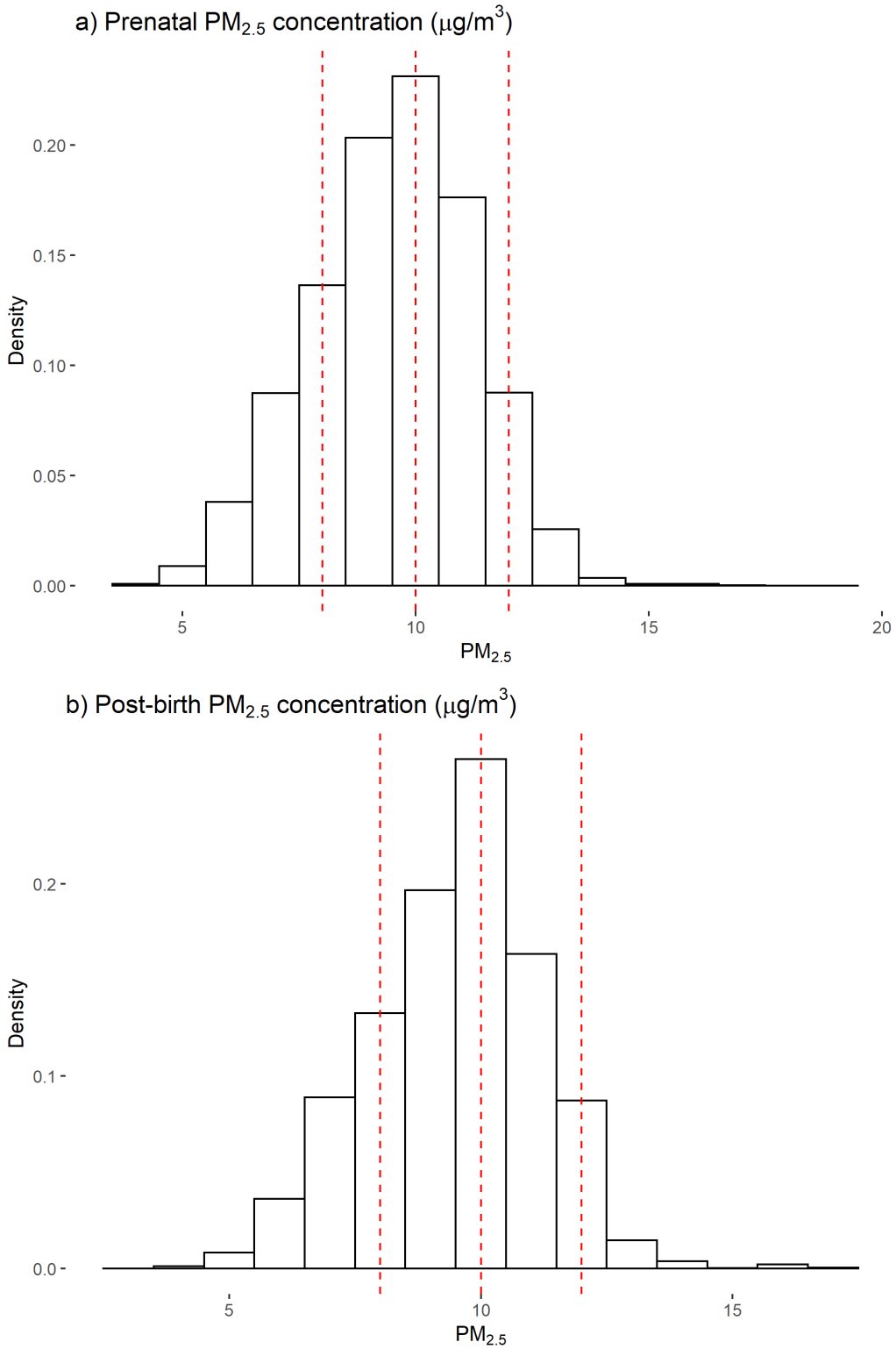
Covariate definition and categories	Source of data
Individual-level variables	
Mother's age (19 years or under; 20-24 years; 25-29 years; 30-34 years; 35-39 years; 40-44 years; 45 years and older)	NCHS linked birth-infant death records
Mother's race (non-Hispanic white; non-Hispanic Black; non-Hispanic other; Hispanic)	
Mother's marital status (Married; Unmarried)	
Mother's education (Without a high school degree, With a high school degree only, Any tertiary degree)	
Mother smoked before pregnancy (Yes; No)	
Mother smoked during pregnancy (Yes; No)	
Parity (First birth, Second birth, Three of more births)	
Birth plurality (Singleton; Multiple)	
Child's sex (Female; Male)	
Delivery payment source (Medicaid; Private insurance; Self-pay; Other)	
Father's age (19 years or under; 20-24 years; 25-29 years; 30-34 years; 35-39 years; 40-44 years; 45 years and older)	
Father's education (Without a high school degree; With a high school degree only; Any tertiary degree)	
Father's race (non-Hispanic white; non-Hispanic Black; non-Hispanic other; Hispanic)	
Conception month and year	
County-of-residence-level variables	

Average temperature during pregnancy (°F) Average precipitation during pregnancy (in)	NOAA
Annual racial composition of county (Proportion non-Hispanic white; proportion non-Hispanic Black; proportion non-Hispanic other; proportion Hispanic) Annual population Annual poverty rate Annual median household income	US Census Bureau
Monthly unemployment rate	Bureau of Labor Statistics
Number of physicians	CMS

Notes: NCHS = National Center for Health Statistics; NOAA = National Oceanic and Atmospheric Administration; °F = degrees Fahrenheit; in = inches; CMS = Centers for Medicare & Medicaid Services



Appendix Figure 3.1 Graphical representation of the Structural Equation Model

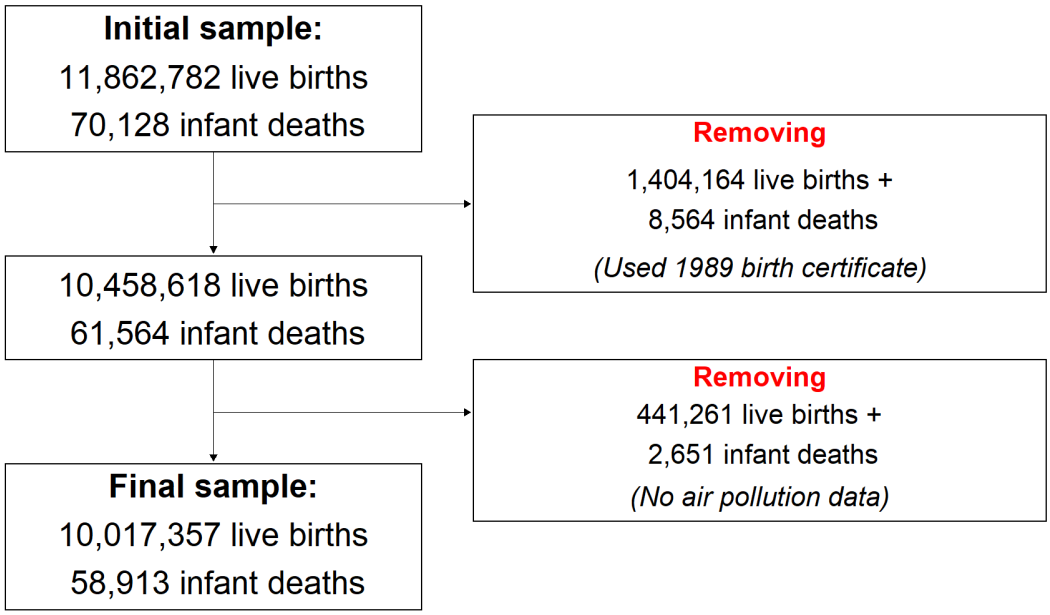


Notes: The red dashed lines on each histogram reflect the threshold values used to create the categorical exposure variable used in the Structural Equation Model. The first red line is at 8 μg/m^3; the second is at 10 μg/m^3; and the third is at 12 μg/m^3.

Appendix Figure 3.2 Distribution of average prenatal and post-birth PM2.5 concentration

Appendix Table 3.2 Frequency table of number of months alive conditional on dying in the first year of life over the study periods

Months alive	Frequency	Proportion
1	40,304	68.4%
2	4,833	8.2%
3	3,509	6.0%
4	2,586	4.4%
5	1,966	3.3%
6	1,525	2.6%
7	1,158	2.0%
8	850	1.4%
9	691	1.2%
10	555	0.9%
11	483	0.8%
12	453	0.8%
Total	58,913	



Appendix Figure 3.3 Constructing the analytic sample

Appendix Table 3.3 Frequency and proportion of missing data among variables in the analytic sample

	Number of missing observations	Total number of observations	Proportion missing
Father's education	1,533,908	10,017,357	15.31
Father's race	1,404,016	10,017,357	14.02
Father's age	1,287,549	10,017,357	12.85
Mother smoked cigarettes during pregnancy	608,210	10,017,357	6.07
Mother smoked cigarettes pre-pregnancy	606,380	10,017,357	6.05
Payment source for delivery	144,634	10,017,357	1.44
Post-birth PM2.5 exposure	130,515	10,017,357	1.3
Mother's education	120,953	10,017,357	1.21
Parity	113,745	10,017,357	1.14
Mother's race	71,191	10,017,357	0.71
Birth weight	30	10,017,357	~0
Prenatal PM2.5 exposure	0	10,017,357	0
Preconception PM2.5 concentration	0	10,017,357	0
Gestational length	0	10,017,357	0
Infant death	0	10,017,357	0
Mother is married	0	10,017,357	0
Mother's age	0	10,017,357	0
Singleton delivery	0	10,017,357	0
Child born female	0	10,017,357	0
Average temperature during pregnancy	0	10,017,357	0
Average precipitation during pregnancy	0	10,017,357	0
Average unemployment during pregnancy	0	10,017,357	0
Proportion of Non-Hispanic whites in county	0	10,017,357	0
Proportion of Non-Hispanic Blacks in county	0	10,017,357	0
Proportion of Hispanics	0	10,017,357	0
Proportion of Non-Hispanic other races	0	10,017,357	0
Average poverty rate	0	10,017,357	0
Median household income	0	10,017,357	0
Physicians per 1,000 individuals	0	10,017,357	0

Notes: The variables in the first column are arranged in descending order of missingness. They represent the variables that were used in the Structural Equation Model, either as the exposure, outcome, mediators, or confounders.

Appendix Table 3.4 Distribution of individual, delivery, and county-level covariates in the analytic sample by categories of prenatal PM2.5 exposure

	Mean (SD)			
	Prenatal $PM_{2.5}$ concentration			
	Category 1 [3.63μg/m^3, 8.00μg/m^3)	Category 2 [8.00μg/m^3, 10.00μg/m^3)	Category 3 [10.00μg/m^3, 12.00μg/m^3)	Category 4 [12.00μg/m^3, 19.16μg/m^3)
Individual-level variables				
Preconception $PM_{2.5}$ concentration (μg/m^3)	7.46 (1.19)	9.43 (1.15)	10.84 (1.15)	11.75 (1.39)
Mother's age				
<= 19 years	7.54 (26.4)	7.72 (26.69)	8.01 (27.14)	8.47 (27.84)
20-24 years	23.95 (42.68)	23.36 (42.31)	22.79 (41.95)	22.47 (41.74)
25-29 years	29.29 (45.51)	28.78 (45.28)	28.33 (45.06)	27.61 (44.7)
30-34 years	24.82 (43.2)	25.62 (43.65)	25.95 (43.83)	25.89 (43.8)
35-39 years	11.54 (31.95)	11.7 (32.14)	11.98 (32.48)	12.42 (32.98)
40-44 years	2.68 (16.14)	2.64 (16.04)	2.74 (16.32)	2.93 (16.87)
>= 45 years	0.18 (4.27)	0.19 (4.31)	0.2 (4.42)	0.21 (4.6)
Mother's race				
Non-Hispanic White	57.33 (49.46)	58.74 (49.23)	52.16 (49.95)	44.66 (49.71)
Non-Hispanic Black	9.8 (29.73)	13.95 (34.65)	16.86 (37.44)	18.43 (38.77)
Non-Hispanic Other	6.4 (24.48)	6.67 (24.94)	6.93 (25.4)	7.42 (26.21)
Hispanic	26.47 (44.12)	20.64 (40.47)	24.04 (42.73)	29.49 (45.6)
Mother's education				
No high school	15.94 (36.6)	16.07 (36.73)	17.68 (38.15)	19.31 (39.47)
High school / some college	49.08 (49.99)	46.22 (49.86)	45.53 (49.8)	44.96 (49.75)
College or more	34.98 (47.69)	37.71 (48.47)	36.79 (48.22)	35.73 (47.92)
Mother is married	59.98 (48.99)	60.95 (48.79)	58.37 (49.3)	55.66 (49.68)
Mother smoked cigarettes pre-pregnancy	11.22 (31.56)	12.44 (33)	11.52 (31.92)	9.62 (29.49)
Parity				
First child	32.25 (46.75)	33.01 (47.02)	33.17 (47.08)	34.01 (47.37)
Second child	28.28 (45.03)	28.64 (45.21)	28.38 (45.08)	27.89 (44.85)
Third or more child	39.47 (48.88)	38.35 (48.62)	38.45 (48.65)	38.1 (48.56)
Payment source for delivery				
Medicaid	44.39 (49.68)	41.88 (49.34)	43.13 (49.53)	47.92 (49.96)
Private insurance	44.7 (49.72)	49.02 (49.99)	48.12 (49.96)	44.39 (49.68)
Self-pay	5.17 (22.14)	4.01 (19.63)	4.16 (19.97)	3.28 (17.81)

Other	5.74 (23.25)	5.09 (21.98)	4.59 (20.93)	4.41 (20.54)
Child born female	48.74 (49.98)	48.85 (49.99)	48.83 (49.99)	48.88 (49.99)
Singleton delivery	96.78 (17.66)	96.5 (18.38)	96.5 (18.38)	96.64 (18.02)
Father's age				
<= 19 years	2.96 (16.95)	2.95 (16.92)	3.15 (17.48)	3.43 (18.19)
20-24 years	15.82 (36.49)	15.36 (36.06)	14.98 (35.69)	14.96 (35.67)
25-29 years	26.45 (44.11)	25.69 (43.69)	25.04 (43.32)	24.51 (43.02)
30-34 years	27.55 (44.68)	28.56 (45.17)	28.74 (45.26)	28.31 (45.05)
35-39 years	16.41 (37.04)	16.98 (37.55)	17.27 (37.8)	17.54 (38.03)
40-44 years	7.23 (25.9)	7.16 (25.79)	7.4 (26.18)	7.65 (26.59)
>= 45 years	3.57 (18.56)	3.3 (17.86)	3.41 (18.14)	3.6 (18.62)
Father's race				
Non-Hispanic White	57.48 (49.44)	59.3 (49.13)	53.23 (49.9)	46.43 (49.87)
Non-Hispanic Black	9.45 (29.26)	12.64 (33.23)	14.42 (35.13)	14.83 (35.54)
Non-Hispanic Other	6.87 (25.29)	7.39 (26.17)	7.72 (26.69)	8.04 (27.2)
Hispanic	26.2 (43.97)	20.67 (40.49)	24.63 (43.08)	30.7 (46.13)
Father's education				
No high school	15.45 (36.14)	15.2 (35.91)	16.7 (37.3)	18.37 (38.73)
High school / some college	51.55 (49.98)	48.73 (49.98)	47.25 (49.92)	46.06 (49.84)
College or more	33.01 (47.02)	36.07 (48.02)	36.06 (48.02)	35.57 (47.87)
County-level variables				
Average temperature during pregnancy	58.25 (12.4)	58 (8.86)	58.37 (8.79)	55.82 (6.52)
Average precipitation during pregnancy	3.12 (1.97)	3.24 (1.47)	2.89 (1.32)	2.67 (1.46)
Average unemployment during pregnancy	8.41 (2.58)	8.01 (2.4)	8.8 (2.3)	10.37 (2.53)
County racial composition				
Non-Hispanic White	64.46 (22.13)	64.9 (20.92)	59 (22.59)	52.06 (21.82)
Non-Hispanic Black	7.33 (8.52)	12.07 (13.08)	14.69 (12.96)	16.21 (13.56)
Non-Hispanic other	5.07 (7.49)	5.46 (6.4)	5.89 (5.08)	6.51 (4.82)
Hispanic	21.21 (19.51)	15.66 (17.43)	18.65 (17.45)	23.52 (20.36)
Average poverty rate	15.97 (4.98)	15.76 (6.03)	16.26 (5.07)	17.86 (4.91)
Median household income	50023.7 (11434.19)	53576.41 (15402.29)	52584.86 (11941.65)	49845.36 (8493.12)
Physicians per 1,000 individuals	0.36 (1.15)	0.35 (0.93)	0.33 (0.54)	0.39 (0.44)

Notes: SD = standard deviation. USD = United States dollars.

Appendix Table 3.5 Distribution of individual, delivery, and county-level covariates in the analytic sample by categories of post-birth PM2.5 exposure

	Mean (SD)			
	Prenatal $PM_{2.5}$ concentration			
	Cateogry 1 [2.70 μg/m^3, 8.00 μg/m^3)	Category 2 [8.00 μg/m^3, 10.00 μg/m^3)	Category 3 [10.00 μg/m^3, 12.00 μg/m^3)	Category 4 [12.00 μg/m^3, 17.19 μg/m^3)
Individual-level variables				
Preconception $PM_{2.5}$ concentration (μg/m^3)	7.4 (1.14)	9.38 (1.1)	10.95 (1.16)	11.82 (1.3)
Mother's age				
<= 19 years	7.78 (26.78)	7.89 (26.95)	7.83 (26.86)	7.8 (26.82)
20-24 years	24.39 (42.94)	23.55 (42.43)	22.49 (41.75)	21.43 (41.03)
25-29 years	29.38 (45.55)	28.88 (45.32)	28.33 (45.06)	26.49 (44.13)
30-34 years	24.46 (42.98)	25.39 (43.52)	26.3 (44.03)	26.48 (44.12)
35-39 years	11.23 (31.57)	11.52 (31.93)	12.11 (32.62)	14.02 (34.72)
40-44 years	2.59 (15.89)	2.6 (15.91)	2.75 (16.34)	3.5 (18.37)
>= 45 years	0.18 (4.18)	0.18 (4.2)	0.2 (4.48)	0.27 (5.21)
Mother's race				
Non-Hispanic White	57.47 (49.44)	59.92 (49.01)	52.31 (49.95)	30.43 (46.01)
Non-Hispanic Black	10.32 (30.43)	14.09 (34.79)	17.29 (37.82)	10.91 (31.18)
Non-Hispanic Other	5.75 (23.28)	6.65 (24.92)	6.87 (25.3)	10.8 (31.04)
Hispanic	26.46 (44.11)	19.33 (39.49)	23.52 (42.41)	47.86 (49.95)
Mother's education				
No high school	16.06 (36.71)	16.2 (36.84)	17.26 (37.79)	21.85 (41.32)
High school / some college	49.39 (50)	46.15 (49.85)	45.28 (49.78)	45.52 (49.8)
College or more	34.55 (47.55)	37.65 (48.45)	37.46 (48.4)	32.63 (46.89)
Mother is married	59.21 (49.14)	61.2 (48.73)	58.52 (49.27)	55.08 (49.74)
Mother smoked cigarettes pre-pregnancy	11.47 (31.86)	12.98 (33.61)	11.42 (31.8)	4.14 (19.91)
Mother smoked cigarettes during pregnancy	9.06 (28.7)	9.99 (29.98)	8.64 (28.09)	3.07 (17.24)
Parity				
First child	32.29 (46.76)	33.1 (47.06)	33.26 (47.11)	33.46 (47.19)
Second child	28.23 (45.01)	28.64 (45.21)	28.38 (45.08)	28.12 (44.96)
Third or more child	39.48 (48.88)	38.26 (48.6)	38.37 (48.63)	38.41 (48.64)
Payment source for delivery				

Medicaid	44.82 (49.73)	42.06 (49.37)	42.27 (49.4)	52.07 (49.96)
Private insurance	43.72 (49.6)	49.04 (49.99)	48.91 (49.99)	41.23 (49.23)
Self-pay	5.5 (22.8)	3.84 (19.21)	4.14 (19.91)	3.57 (18.56)
Other	5.96 (23.67)	5.07 (21.93)	4.68 (21.12)	3.13 (17.41)
Child born female	48.79 (49.99)	48.84 (49.99)	48.87 (49.99)	48.76 (49.98)
Singleton delivery	96.88 (17.39)	96.59 (18.14)	96.55 (18.26)	96.86 (17.43)
Father's age				
<= 19 years	3.05 (17.2)	2.98 (17)	3.08 (17.29)	3.51 (18.4)
20-24 years	16.12 (36.77)	15.5 (36.19)	14.69 (35.4)	14.85 (35.56)
25-29 years	26.68 (44.23)	25.88 (43.8)	24.88 (43.23)	23.38 (42.32)
30-34 years	27.37 (44.58)	28.48 (45.13)	29.03 (45.39)	27.37 (44.59)
35-39 years	16.15 (36.79)	16.82 (37.41)	17.47 (37.98)	18.28 (38.65)
40-44 years	7.09 (25.66)	7.1 (25.68)	7.44 (26.24)	8.43 (27.78)
>= 45 years	3.55 (18.51)	3.23 (17.69)	3.4 (18.13)	4.18 (20)
Father's race				
Non-Hispanic White	57.75 (49.4)	60.47 (48.89)	53.57 (49.87)	31.59 (46.49)
Non-Hispanic Black	9.86 (29.81)	12.67 (33.26)	14.75 (35.46)	9.39 (29.16)
Non-Hispanic Other	6.27 (24.24)	7.44 (26.24)	7.6 (26.49)	10.97 (31.25)
Hispanic	26.12 (43.93)	19.42 (39.56)	24.08 (42.76)	48.05 (49.96)
Father's education				
No high school	15.36 (36.05)	15.24 (35.94)	16.22 (36.86)	22.69 (41.88)
High school / some college	52.04 (49.96)	48.64 (49.98)	46.98 (49.91)	46.25 (49.86)
College or more	32.61 (46.88)	36.13 (48.04)	36.8 (48.23)	31.06 (46.27)
County-level variables				
Average temperature during pregnancy	58.67 (12.81)	57.64 (9.08)	58.09 (8.25)	57.56 (6.51)
Average precipitation during pregnancy	3.1 (1.92)	3.26 (1.44)	2.97 (1.37)	1.81 (1.29)
Average unemployment during pregnancy	8.45 (2.57)	8.06 (2.42)	8.76 (2.2)	11.13 (2.78)
County racial composition				
Non-Hispanic White	64.64 (22.69)	65.9 (20.65)	59.16 (21.91)	38.94 (15.62)
Non-Hispanic Black	7.57 (8.41)	12.32 (13.56)	15.03 (13.03)	10.21 (8.06)
Non-Hispanic other	4.49 (6.8)	5.37 (6.54)	5.87 (5.06)	10.04 (5.04)
Hispanic	21.46 (20.41)	14.47 (16.69)	18.16 (16.68)	39.04 (16.16)
Average poverty rate	16.08 (5.03)	15.72 (6.05)	16.14 (4.95)	19.23 (4.49)
Median household income	48923.01 (10438.92)	53399.45 (15823.55)	52909.86 (11474.82)	51726.89 (8044.06)

Physicians per 1,000 individuals	0.37 (1.18)	0.35 (0.96)	0.33 (0.46)	0.32 (0.25)

Notes: SD = standard deviation. USD = United States dollars.

Appendix Table 3.6 Estimates of the association between prenatal PM2.5 exposure, post-birth PM2.5 exposure (defined over 1 month), preterm birth, low birth weight, and infant mortality from the Structural Equation Model

	Percentage point change	95% confidence interval
Panel A: Direct association between prenatal $PM_{2.5}$ exposure and preterm birth		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.57	[0.43, 0.71]
[10.00 μg/m3 - 12.00 μg/m3)	0.95	[0.72, 1.18]
[12.00 μg/m3 - 19.16 μg/m3]	1.11	[0.72, 1.49]
Panel B: Direct association between prenatal $PM_{2.5}$ exposure and low birth weight		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.14	[0.06, 0.22]
[10.00 μg/m3 - 12.00 μg/m3)	0.23	[0.13, 0.34]
[12.00 μg/m3 - 19.16 μg/m3]	0.29	[0.14, 0.45]
Panel C: Direct association between preterm birth and low birth weight		
Preterm birth	49.67	[49.29, 50.05]
Panel D: Direct association of prenatal and post-birth $PM_{2.5}$ exposure with infant death		
Prenatal exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.05	[0.02, 0.08]
[10.00 μg/m3 - 12.00 μg/m3)	0.08	[0.04, 0.12]
[12.00 μg/m3 - 19.16 μg/m3]	0.12	[0.07, 0.17]
Post-birth exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.01, 0.04]
[10.00 μg/m3 - 12.00 μg/m3)	0.05	[0.03, 0.06]
[12.00 μg/m3 - 17.19 μg/m3]	0.05	[0.02, 0.07]
Panel E: Direct association of preterm birth and low birth weight with infant death		
Preterm birth	2.00	[1.93, 2.07]
Low birth weight	3.64	[3.55, 3.73]
Number of observations	10,017,357	
Average SRMR	0	

Notes: All coefficients are expressed as percentage point changes in the respective outcomes. 95 percent confidence intervals were estimated using standard errors that were clustered at the county-level. The post-birth $PM_{2.5}$ exposure is estimated over a one-month period following the end of the nine-month prenatal period. SRMR = Standardized Root Mean Square Residual. The average SRMR was calculated as the average of the SRMR of the Structural Equation Model fit in each of the five imputed datasets.

Appendix Table 3.7 Estimates of the association between prenatal PM2.5 exposure, post-birth PM2.5 exposure (defined over 2 months), preterm birth, low birth weight, and infant mortality from the Structural Equation Model

	Percentage point change	95% confidence interval
Panel A: Direct association between prenatal $PM_{2.5}$ exposure and preterm birth		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.57	[0.43, 0.71]
[10.00 μg/m3 - 12.00 μg/m3)	0.95	[0.72, 1.18]
[12.00 μg/m3 - 19.16 μg/m3]	1.11	[0.72, 1.49]
Panel B: Direct association between prenatal $PM_{2.5}$ exposure and low birth weight		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.14	[0.06, 0.22]
[10.00 μg/m3 - 12.00 μg/m3)	0.23	[0.13, 0.34]
[12.00 μg/m3 - 19.16 μg/m3]	0.29	[0.14, 0.45]
Panel C: Direct association between preterm birth and low birth weight		
Preterm birth	49.67	[49.29, 50.05]
Panel D: Direct association of prenatal and post-birth $PM_{2.5}$ exposure with infant death		
Prenatal exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.05	[0.03, 0.08]
[10.00 μg/m3 - 12.00 μg/m3)	0.08	[0.05, 0.12]
[12.00 μg/m3 - 19.16 μg/m3]	0.12	[0.07, 0.17]
Post-birth exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.02, 0.05]
[10.00 μg/m3 - 12.00 μg/m3)	0.03	[0, 0.05]
[12.00 μg/m3 - 17.19 μg/m3]	0.04	[0.01, 0.07]
Panel E: Direct association of preterm birth and low birth weight with infant death		
Preterm birth	2.00	[1.93, 2.07]
Low birth weight	3.64	[3.55, 3.73]
Number of observations	10,017,357	
Average SRMR	0	

Notes: All coefficients are expressed as percentage point changes in the respective outcomes. 95 percent confidence intervals were estimated using standard errors that were clustered at the county-level. The post-birth $PM_{2.5}$ exposure is estimated over a two-month period following the end of the nine-month prenatal period. SRMR = Standardized Root Mean Square Residual. The average SRMR was calculated as the average of the SRMR of the Structural Equation Model fit in each of the five imputed datasets.

Appendix Table 3.8 Estimates of the association between prenatal PM2.5 exposure disaggregated by trimester, post-birth PM2.5 exposure (defined over 12 months), preterm birth, low birth weight, and infant mortality from the Structural Equation Model

	Percentage point change	95% confidence interval
Panel A: Direct association between prenatal $PM_{2.5}$ exposure and preterm birth		
First trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.15	[0.03, 0.26]
[10.00 μg/m3 - 12.00 μg/m3)	0.08	[-0.11, 0.27]
[12.00 μg/m3 - 19.16 μg/m3]	-0.08	[-0.33, 0.18]
Second trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.37	[0.25, 0.49]
[10.00 μg/m3 - 12.00 μg/m3)	0.62	[0.48, 0.77]
[12.00 μg/m3 - 19.16 μg/m3]	0.74	[0.56, 0.92]
Third trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.38	[0.26, 0.49]
[10.00 μg/m3 - 12.00 μg/m3)	0.71	[0.55, 0.87]
[12.00 μg/m3 - 19.16 μg/m3]	1.02	[0.82, 1.21]
Panel B: Direct association between prenatal $PM_{2.5}$ exposure and low birth weight		
First trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.07	[0.01, 0.12]
[10.00 μg/m3 - 12.00 μg/m3)	0.08	[0, 0.15]
[12.00 μg/m3 - 19.16 μg/m3]	0	[-0.09, 0.08]
Second trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.11	[0.06, 0.17]
[10.00 μg/m3 - 12.00 μg/m3)	0.15	[0.08, 0.23]
[12.00 μg/m3 - 19.16 μg/m3]	0.23	[0.14, 0.31]
Third trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.13	[0.07, 0.18]
[10.00 μg/m3 - 12.00 μg/m3)	0.22	[0.15, 0.29]
[12.00 μg/m3 - 19.16 μg/m3]	0.35	[0.26, 0.43]
Panel C: Direct association between preterm birth and low birth weight		
Preterm birth	49.67	[49.29, 50.05]
Panel D: Direct association of prenatal and post-birth $PM_{2.5}$ exposure with infant death		

Prenatal exposure		
First trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.01, 0.05]
[10.00 μg/m3 - 12.00 μg/m3)	0.04	[0.01, 0.06]
[12.00 μg/m3 - 19.16 μg/m3]	0.03	[0, 0.06]
Second trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.04	[0.02, 0.05]
[10.00 μg/m3 - 12.00 μg/m3)	0.04	[0.01, 0.06]
[12.00 μg/m3 - 19.16 μg/m3]	0.07	[0.04, 0.09]
Third trimester		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.02, 0.05]
[10.00 μg/m3 - 12.00 μg/m3)	0.07	[0.05, 0.09]
[12.00 μg/m3 - 19.16 μg/m3]	0.1	[0.06, 0.13]
Post-birth exposure		
Ref: <8 μg/m³		
[8.00 μg/m3 - 10.00 μg/m3)	0.04	[0.01, 0.07]
[10.00 μg/m3 - 12.00 μg/m3)	0.01	[-0.03, 0.05]
[12.00 μg/m3 - 17.19 μg/m3]	-0.01	[-0.07, 0.05]
Panel E: Direct association of preterm birth and low birth weight with infant death		
Preterm birth	2.00	[1.93, 2.07]
Low birth weight	3.64	[3.55, 3.73]
Number of observations	10,017,357	
Average SRMR	0	

Notes: All coefficients are expressed as percentage point changes in the respective outcomes. 95 percent confidence intervals were estimated using standard errors that were clustered at the county-level. The post-birth $PM_{2.5}$ exposure is estimated over a 12-month period following the end of the nine-month prenatal period. SRMR = Standardized Root Mean Square Residual. The average SRMR was calculated as the average of the SRMR of the Structural Equation Model fit in each of the five imputed datasets.

Appendix Table 3.9 Direct and indirect associations of prenatal and post-birth PM2.5 exposure (defined over 1 month) with infant mortality

	Direct association		Indirect association		Total association		Proportion mediated (%)
	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	
Panel A: Direct and indirect associations of the prenatal $PM_{2.5}$ exposure							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.05	[0.02, 0.08]	0.03	[0.02, 0.03]	0.08	[0.05, 0.1]	37.5
[10.00 μg/m3 - 12.00 μg/m3)	0.08	[0.04, 0.12]	0.04	[0.03, 0.06]	0.12	[0.08, 0.17]	33.33
[12.00 μg/m3 - 19.16 μg/m3]	0.12	[0.07, 0.17]	0.05	[0.03, 0.07]	0.17	[0.11, 0.23]	29.41
Panel B: Direct and indirect associations of the post-birth $PM_{2.5}$ exposure							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.01, 0.04]	-	-	0.03	[0.01, 0.04]	-
[10.00 μg/m3 - 12.00 μg/m3)	0.05	[0.03, 0.06]	-	-	0.05	[0.03, 0.06]	-
[12.00 μg/m3 - 19.16 μg/m3]	0.05	[0.02, 0.07]	-	-	0.05	[0.02, 0.07]	-

Notes: Direct and indirect association estimates were estimated using the Structural Equation Model presented in Appendix Table 3.6. The indirect association from the prenatal $PM_{2.5}$ exposure to infant mortality reflects two paths: prenatal exposure → preterm birth → infant mortality; and, prenatal exposure → preterm birth → low birth weight → infant mortality. The post-birth $PM_{2.5}$ exposure is estimated over a one-month period following the end of the nine-month prenatal period. Since there is only a direct path from the post-birth exposure to infant mortality, there are no results for the indirect association between the post-birth exposure and the outcome.

Appendix Table 3.10 Direct and indirect associations of prenatal and post-birth PM2.5 exposure (defined over 2 months) with infant mortality

	Direct association		Indirect association		Total association		Proportion mediated (%)
	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	
Panel A: Direct and indirect associations of the prenatal $PM_{2.5}$ exposure							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.05	[0.03, 0.08]	0.03	[0.02, 0.03]	0.08	[0.05, 0.11]	37.5
[10.00 μg/m3 - 12.00 μg/m3)	0.08	[0.05, 0.12]	0.04	[0.03, 0.06]	0.13	[0.08, 0.17]	30.77
[12.00 μg/m3 - 19.16 μg/m3]	0.12	[0.07, 0.17]	0.05	[0.03, 0.07]	0.17	[0.11, 0.23]	29.41
Panel B: Direct and indirect associations of the post-birth $PM_{2.5}$ exposure							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.02, 0.05]	-	-	0.03	[0.02, 0.05]	-
[10.00 μg/m3 - 12.00 μg/m3)	0.03	[0, 0.05]	-	-	0.03	[0, 0.05]	-
[12.00 μg/m3 - 19.16 μg/m3]	0.04	[0.01, 0.07]	-	-	0.04	[0.01, 0.07]	-

Notes: Direct and indirect association estimates were estimated using the Structural Equation Model presented in Appendix Table 3.7. The indirect association from the prenatal $PM_{2.5}$ exposure to infant mortality reflects two paths: prenatal exposure → preterm birth → infant mortality; and, prenatal exposure → preterm birth → low birth weight → infant mortality. The post-birth $PM_{2.5}$ exposure is estimated over a two-month period following the end of the nine-month prenatal period. Since there is only a direct path from the post-birth exposure to infant mortality, there are no results for the indirect association between the post-birth exposure and the outcome.

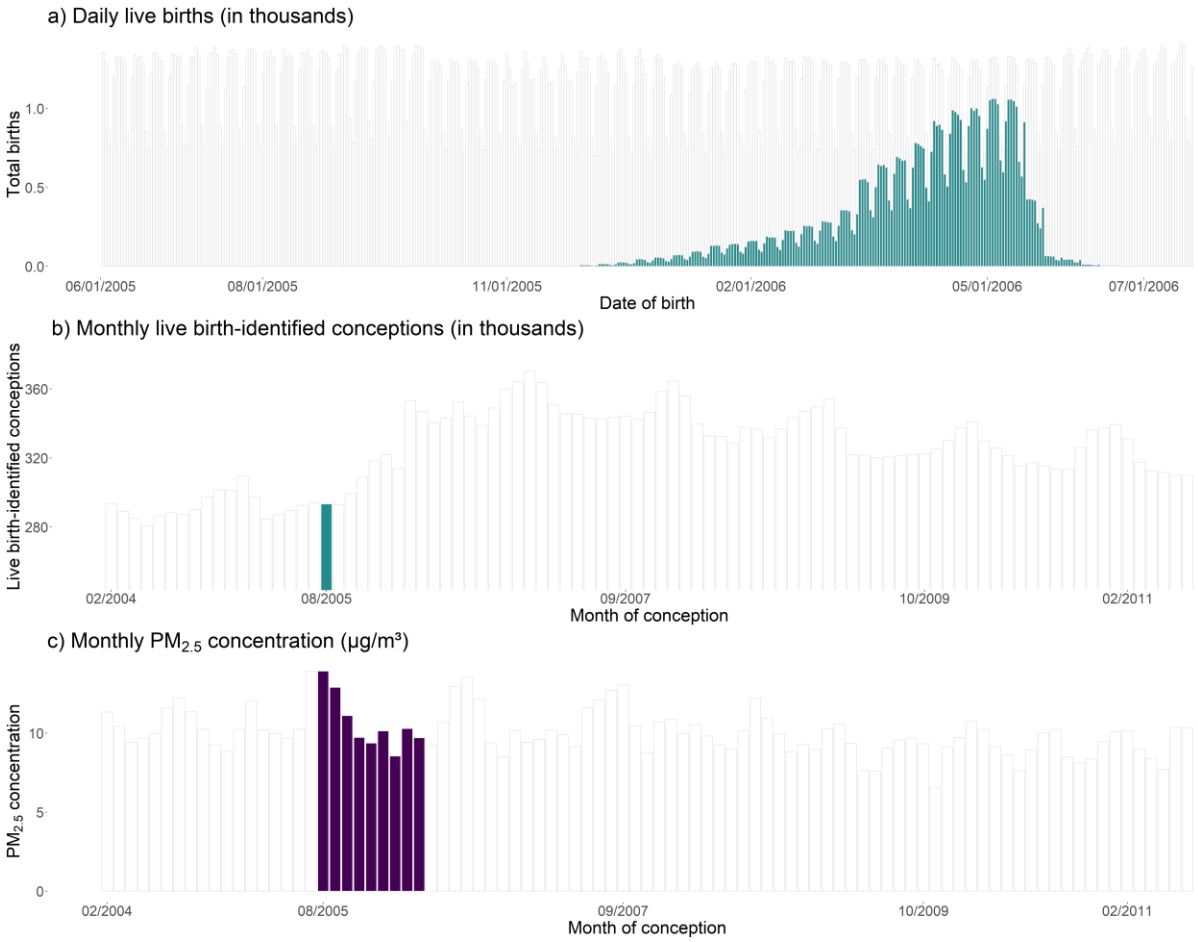
Appendix Table 3.11 Direct and indirect associations of prenatal PM2.5 exposure disaggregate by trimester and post-birth PM2.5 exposure (defined over 12 months) with infant mortality

	Direct association		Indirect association		Total association		Proportion mediated (%)
	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	Percentage point change	95% confidence interval	
Panel A: Direct and indirect associations of the prenatal $PM_{2.5}$ exposure							
First trimester							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.01, 0.05]	0.01	[0, 0.01]	0.04	[0.01, 0.06]	25
[10.00 μg/m3 - 12.00 μg/m3)	0.04	[0.01, 0.06]	0.01	[0, 0.01]	0.04	[0.01, 0.07]	25
[12.00 μg/m3 - 19.16 μg/m3]	0.03	[0, 0.06]	0	[-0.01, 0.01]	0.03	[-0.01, 0.07]	0
Second trimester							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.04	[0.02, 0.05]	0.02	[0.01, 0.02]	0.05	[0.03, 0.07]	40
[10.00 μg/m3 - 12.00 μg/m3)	0.04	[0.01, 0.06]	0.03	[0.02, 0.04]	0.07	[0.04, 0.09]	42.86
[12.00 μg/m3 - 19.16 μg/m3]	0.07	[0.04, 0.09]	0.04	[0.03, 0.05]	0.1	[0.07, 0.13]	40
Third trimester							
Ref: <8 μg/m³							
[8.00 μg/m3 - 10.00 μg/m3)	0.03	[0.02, 0.05]	0.02	[0.01, 0.02]	0.05	[0.03, 0.07]	40
[10.00 μg/m3 - 12.00 μg/m3)	0.07	[0.05, 0.09]	0.04	[0.03, 0.04]	0.1	[0.08, 0.13]	40
[12.00 μg/m3 - 19.16 μg/m3]	0.1	[0.06, 0.13]	0.05	[0.04, 0.06]	0.15	[0.11, 0.18]	33.33
Panel B: Direct and indirect associations of the postnatal $PM_{2.5}$ exposure							
Ref: <8 μg/m³							

[8.00 µg/m3 - 10.00 µg/m3)	0.04	[0.01, 0.07]	-	-	0.04	[0.01, 0.07]	-
[10.00 µg/m3 - 12.00 µg/m3)	0.01	[-0.03, 0.05]	-	-	0.01	[-0.03, 0.05]	-
[12.00 µg/m3 - 19.16 µg/m3]	-0.01	[-0.07, 0.05]	-	-	-0.01	[-0.07, 0.05]	-

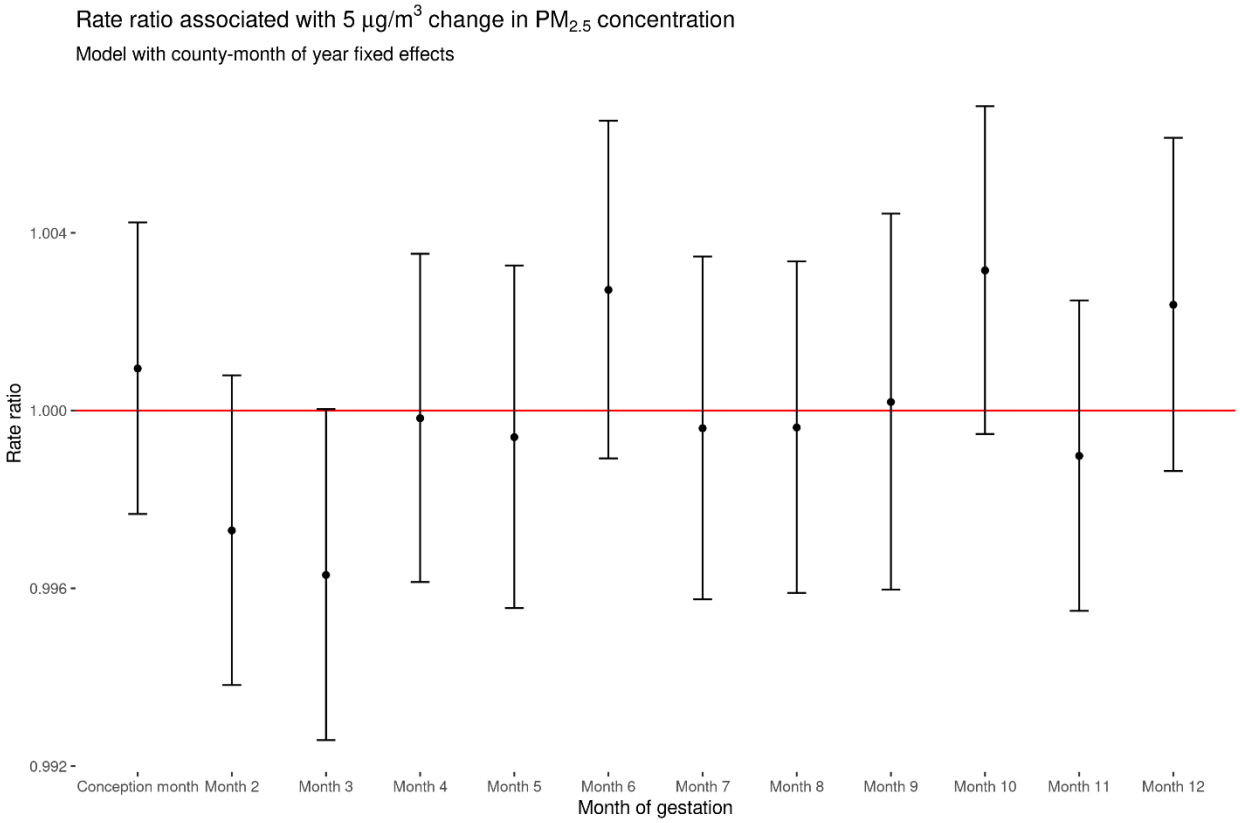
Notes: Direct and indirect association estimates were estimated using the Structural Equation Model presented in Appendix Table 3.8. The indirect association from the prenatal $PM_{2.5}$ exposure to infant mortality reflects two paths: prenatal exposure → preterm birth → infant mortality; and, prenatal exposure → preterm birth → low birth weight → infant mortality. The post-birth $PM_{2.5}$ exposure is estimated over a 12-month period following the end of the nine-month prenatal period. Since there is only a direct path from the post-birth exposure to infant mortality, there are no results for the indirect association between the post-birth exposure and the outcome.

Supplementary materials for Chapter 4



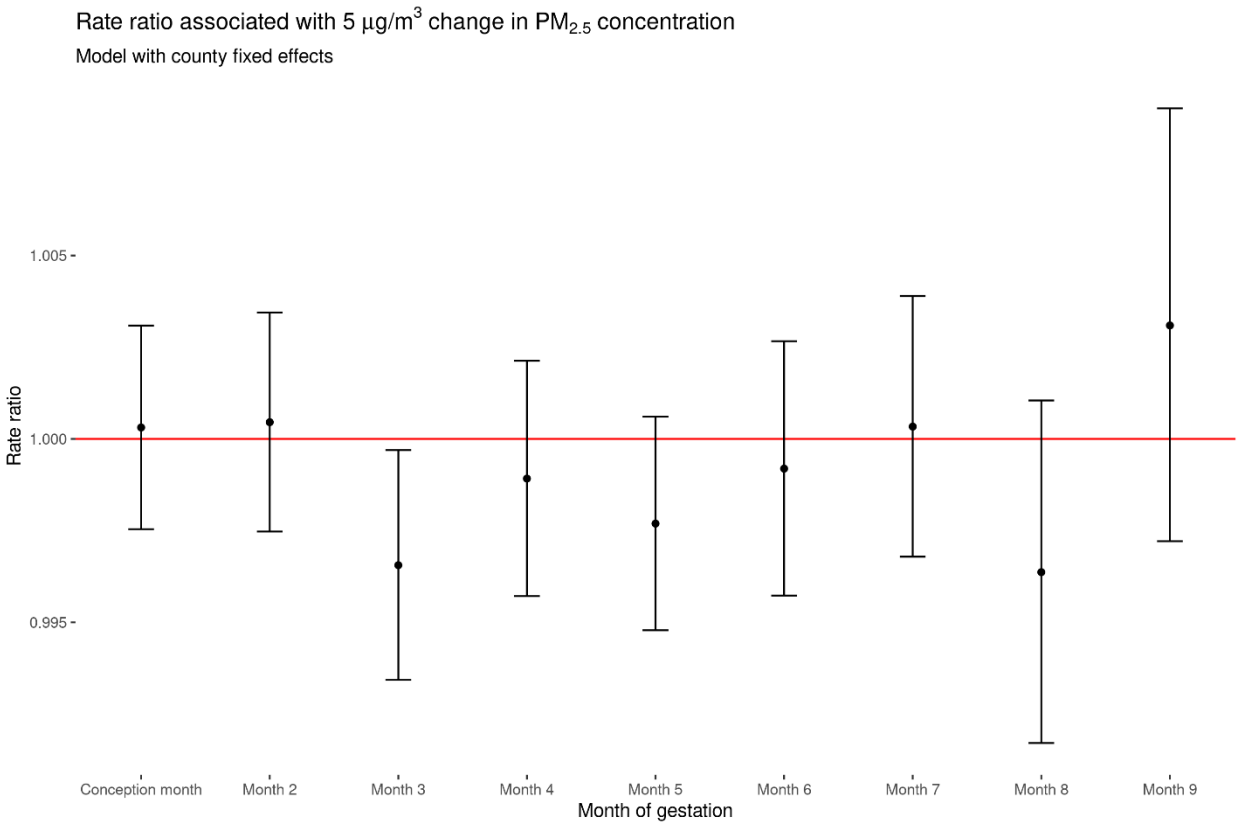
Notes: This figure illustrates our study design and is based on synthetic live birth data which follows the distribution in the birth records data. Panel (a) is a bar chart of the daily number of live births. Panel (b) is a bar chart of monthly live birth-identified conceptions, which is the outcome we use in our analysis. The turquoise highlights in the bars presented in panel (a) represent the live births that were conceived in the turquoise highlighted bar in panel (b). In the context of this specific figure, all the turquoise highlighted births in panel (a) were conceived in August 2005. Panel (c) presents average monthly $PM_{2.5}$ concentration. The purple highlighted bars represent those months which we use as the gestational exposure months for the turquoise highlighted bar in panel (b).

Appendix Figure 4.1 Illustration of the study design in any given county in the data



Notes: All estimates presented in the figure are from a conditional quasi-Poisson model with county-month of year fixed effects. This model includes controls for temperature, precipitation, relative humidity, indicator variables for each year of conception in the data, and the log of county-specific annual female population between 15-49 years as an offset term. Unlike the primary model, the model presented in the figure above includes 12 months of gestational exposure. The red horizontal line indicates RR = 1 (null).

Appendix Figure 4.2 Rate ratio associated with 5 µg/m3 increase in PM2.5 concentration by month of gestation over a 12-month period in a model with county-month of year fixed effects



Notes: All estimates presented in the figure are from a conditional quasi-Poisson model with county fixed effects. This model includes controls for temperature, precipitation, relative humidity, indicator variables for each year of conception in the data, a natural cubic spline to control for within-year temporal trends, and the log of county-specific annual female population between 15-49 years as an offset term. Unlike the primary model, the model presented in the figure above includes 12 months of gestational exposure. The red horizontal line indicates RR = 1 (null).

Appendix Figure 4.3 Rate ratio associated with 5 µg/m3 increase in PM2.5 concentration by month of gestation in a model with county fixed effects