UNIVERSITY OF CALIFORNIA, MERCED

Acute impact of air pollution on fetal and infant death in the San Joaquin Valley, California: A time-stratified case-crossover study

A Thesis submitted in partial satisfaction of the requirements for the degree of

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in

Public Health

by

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This Thesis of Sneha Ghimire is approved, and it is acceptable in quality and form for publication on microfilm and electronically:

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University of California, Merced

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Dedicated to

my loving uncle

Late Tara Prasad Ghimire

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LIST OF FIGURES

LIST OF TABLES

ABSTRACT

Acute impact of air pollution on fetal and infant death in the San Joaquin Valley, California: A time-stratified case-crossover study

by

Sneha Ghimire

Master of Science in Public Health

University of California, Merced 2024

Committee chair: Dr. Sandie Ha

Background: Despite the decrease in overall pollution levels across California, the San Joaquin Valley (SJV) remains a non-attainment area for pollutants including $PM_{2.5}$ and O_3.

Objectives: We assessed the association between acute exposure to $PM_{2.5}$ and O_3 and the risk of fetal and infant death in the SJV.

Methods: This time-stratified case-crossover analysis includes 1,343 singleton fetal deaths and 1,097 singleton infant deaths in the SJV from 2016 to 2019. Daily O_3 and $PM_{2.5}$ data were geospatially linked to the residential zip codes of mothers at delivery. We examined critical exposure windows including the day of death (lag 0) and the 14 days leading up to it (lag 1 - lag 14). We used conditional logistic regression models to calculate the odds ratio (OR) and 95% confidence intervals for each 10-unit increase in pollutants.

Results: $PM_{2.5}$ during cold (November-April) and O_3 during warm seasons (May-October) were positively associated with higher odds of fetal and infant deaths. O_3 was positively associated with both fetal and infant deaths across all lags while no significant associations were observed between infant death and $PM_{2.5}$ exposure. Associations also varied across racial, and socio-economic divisions and maternal ages.

Conclusions: As climate change escalates, rising air pollution may contribute to perinatal mortality.

TABLE OF CONTENTS

1. Introduction and Background

1.1.Public health significance of fetal and infant death

Fetal death refers to the demise of a fetus at any point during pregnancy (Barfield et al., 2016; Gregory et al., 2022). The categorization of fetal death varies according to the timing of the event within the gestational period. Death of a fetus that occurs before 20 weeks of full gestation is generally classified as pregnancy loss or spontaneous abortion. On the other hand, the terminology "stillbirth" is often employed to describe fetal death that occurs from the 20th week of gestation until birth, where the fetus displays no evidence of life (Barfield et al., 2016; CDC, 2022b). For international comparative studies, the World Health Organization standardizes the definition of stillbirth as a death of a fetus occurring at a gestational age of 28 weeks or later (World Health Organization). In 2020, the US witnessed 20,854 instances of fetal death, reflecting a rate of 5.74 per 1,000 live births. This closely aligns with the 2019 rate of 5.70 per 1,000 live births indicating no significant change. However, a broader look at the past three decades reveals a marked improvement, with a 24.3% reduction in fetal death rates from 1990 to 2020, dropping from 7.49 to 5.74 per 1,000 live births (Efflein, 2022; Gregory et al., 2022). It is crucial to recognize that while most fetal deaths happen early in pregnancy, many US states only mandate the reporting of those occurring at 20 weeks of gestation or later (Gregory et al., 2022), leading to potential underestimation. Fetal death, despite being a serious public health issue, is often underestimated due to a lack of understanding about its true incidence, underlying causes, and preventive measures (Gregory et al., 2022). The repercussions of fetal death are not constrained to economically poor nations but are observed globally (Lawn et al., 2016). Some known risk factors for fetal death include pregnancy complications (e.g., preeclampsia), complicated childbirth, genetics, fetal growth restriction, and congenital anomalies (Gardosi et al., 2013). However, the causes of many fetal deaths are often unclear and other risk factors responsible for fetal death remain unexplored (Gardosi, 2001; Williams et al., 2018).

Meanwhile, infant death refers to the decease of an infant during the first year following birth (CDC, 2022a). In 2020, the US recorded approximately 20,000 infant deaths, corresponding to a rate of 5.4 deaths per 1,000 live births (CDC, 2022a). These statistics emphasize the pressing necessity for ongoing initiatives to uncover and confront the factors leading to infant death. Some of the known leading causes of infant death in the US are birth defects, sudden infant death syndrome (SIDS), premature birth, low birth weight, maternal complications during pregnancy, and injuries such as suffocation (CDC, 2022a; UNICEF, 2019). Meanwhile, it is important to acknowledge that numerous unidentified or under-researched factors contribute to infant deaths. Despite the recent reduction in infant deaths, the US still recorded a total of 20,927 infant deaths in 2019, corresponding to 5.74 infant deaths per 1,000 live births. This rate remains significantly higher than what has been achieved in other developed nations **(Figure 1)** (OECD, 2023). Studies suggest that the decline in infant death rates in recent years is primarily due to a reduction in fatalities caused by congenital disorders and SIDS (Khan et al., 2018). However, there exist additional possible contributors to infant deaths that necessitate

deeper exploration. For instance, the possible impacts of environmental exposures, such as exposure to air pollution, have not been fully investigated.

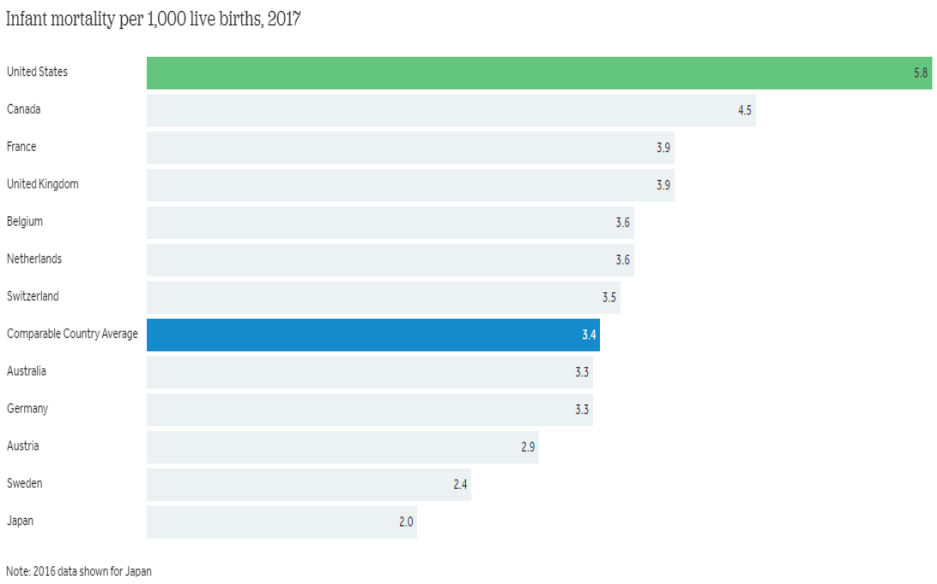

Figure 1: Infant mortality in the US compared to other developed countries in 2017 (Data source: KFF analysis of OECD data)

1.2. Disparities in fetal and infant deaths in the US

In the US, significant disparities exist in the rates of fetal and infant deaths across populations defined by numerous socioeconomic factors including race/ethnicity, healthcare access and quality, economic stability, neighborhood and built environment, discrimination leading to stress, education, and social and community context (OASH, 2021). In 2020, a disparate distribution of both fetal and infant death rates was observed across various racial and ethnic groups in the United States. For instance, the fetal death rate per 1,000 live births was reported as 4.73 among Non-Hispanic Whites, 10.34 for Non-Hispanic Blacks, 4.86 for Hispanics, 3.93 for Asians, 10.59 for Hawaiian/Pacific Islander, and 7.84 for American Indians/Alaskan Natives (Gregory et al., 2022). Similarly, the infant death rate per 1,000 live births was reported as 4.40 for Non-Hispanic Whites, 10.38 for Non-Hispanic Blacks, 4.69 for Hispanics, 3.14 for Asians, 7.17 for Hawaiian/Pacific Islander, and 7.68 for American Indians/Alaskan Natives (Ely & Driscoll, 2022). It is noteworthy that Non-Hispanic Black populations exhibited the highest mortality rates for both fetal and infant deaths, followed by the Hawaiian/Pacific Islander groups. These racial and ethnic disparities underscore the necessity for further research to identify the underlying factors contributing to these discrepancies.

1.3. Air pollution and its potential role

Population growth, industrialization, and an increasing number of petroleum-based transportation systems have resulted in increased air pollution and a degraded environment worldwide (Zhou, 2009). Ambient air pollution is a mixture of thousands of

components that can harm the environment and human health (Karimi et al., 2019). A large body of research has explored the effects of air pollution on both cardiovascular and respiratory health, drawing connections to an increased risk of mortality from all causes specifically from conditions like cardiovascular disease, respiratory disease, and lung cancer (Bevan et al., 2021; Chen & Hoek, 2020; Liu et al., 2019; Orellano et al., 2020; Schraufnagel et al., 2019; Shahi et al., 2014). These risks are notably associated with exposures to ambient $PM_{2.5}$ (Brook et al., 2010; Liu et al., 2019; Rajagopalan et al., 2018). Additionally, air pollution was the fourth-highest contributor to an untimely death in 2019, only surpassed by high blood pressure, tobacco consumption, and undernutrition **(Figure 2)** (Health Effects Insititue, 2020). Air pollution contributes to about 6.67 million deaths worldwide, accounting for around 12% of total deaths (Health Effects Insititue, 2020). The State of Global Air 2020 report states that approximately 500,000 infants didn't survive their first month in 2019 due to complications from premature birth and low birth weight attributed to air pollution exposure (Health Effects Insititue, 2020). This represented about 20% of the global 4.2 million infant fatalities within the first month of birth.

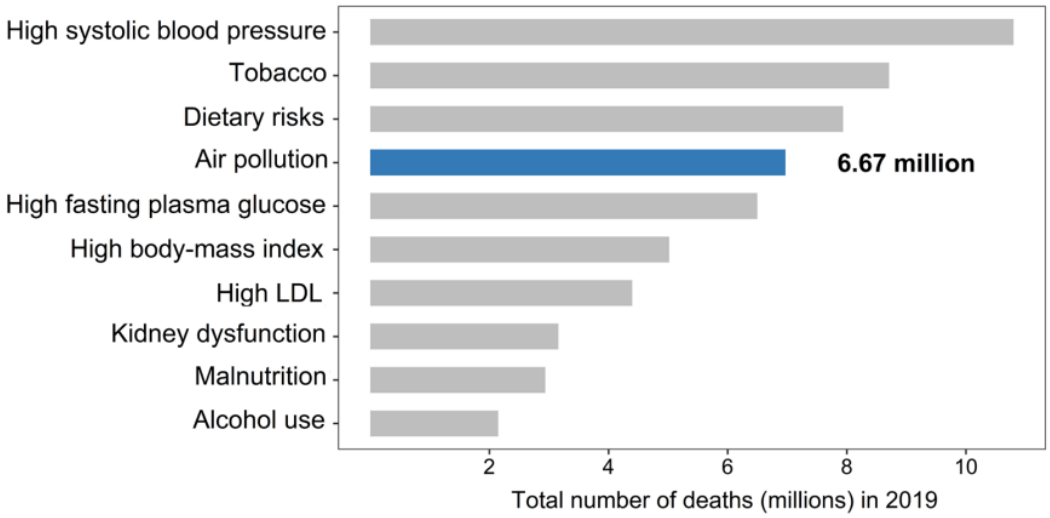

Figure 2: Global ranking of risk factors by total number of deaths from all causes in 2019 (Data source: State of Global Air 2020)

The United States Environmental Protection Agency (EPA) regulates six air pollutants recognized for causing the most harm to human health and the environment. These pollutants are commonly known as "criteria pollutants (USEPA, 2022a) and include particulate matter (PM), nitrogen oxides (NO_x), sulfur oxides (SO_x), lead (Pb), carbon monoxide (CO), and ground-level ozone (O_3). These criteria pollutants are used to set the National Ambient Air Quality Standards (NAAQS) and are updated periodically using emerging scientific benchmarks and knowledge regarding health effects (USEPA, 2022a). Criteria air pollutants emerge from various sources, encompassing automobiles, industrial processes, construction activities, wildfires, and other forms of human involvement. PM can be grouped into either primary or secondary classification (USEPA, 2022c). Primary PM is directly emitted from sources and can vary

in size from coarse to fine. Interestingly, it is the only primary pollutant regulated under Section 109 of the Clean Air Act without a specific chemical identity definition (Ullrich, 2003). PM and O_3 are subsets of the criteria air pollutants, and their regulation is vital due to their profound effects on public health and the environment, along with the diverse origins from which they can emerge (USEPA, 2022b). Despite the strides like technological innovation, a shift toward renewable energy, increased air quality monitoring, and the implementation of more stringent laws to improve air quality since 1970, certain regions still consistently observe levels of PM and ground-level O_3 surpassing the NAAQS. As a result, these regions are identified as "nonattainment areas" (Congressional Research Service, 2020). Their toxicity and ubiquity make particulate matter less than 2.5 micrometers in diameter ($PM_{2.5}$) and O_3 among air pollutants most scrutinized with possible effects on health (American Lung Association, 2023b; USEPA, 2022b) and unfavorable birth outcomes (Ha et al., 2019; Heft-Neal et al., 2018; Nyadanu et al., 2022; Siddika et al., 2019).

1.4. $PM_{2.5}$ and epidemiologic associations with fetal and infant death

PM refers to tiny solid or liquid materials dispersed in the air and can originate from multiple sources (USEPA, 2022c). A schematic of the relative sizes of PM is presented in **Figure 3**. The chief constituents of $PM_{2.5}$ include organic carbonaceous mass, ammonium sulfate, ammonium nitrate, elemental carbon, and earthy substances, which are primarily derived from sources such as vehicles, power production, and airborne soil and metalworking processes (Adams et al., 2015; USEPA, 2022c). $PM_{2.5}$ often varies by region depending on local sources (Frank, 2006). Research consistently shows that exposures to smaller particles such as $PM_{2.5}$ are associated with more health hazards compared with larger particles (Brook et al., 2010; Rajagopalan et al., 2018). This is because these particles are more easily inhaled, can reach deeper into the lungs, and potentially access the bloodstream (Anwar et al., 2021). Although there have been substantial reductions in the national average concentrations of $PM_{2.5}$ following the implementation of the Clean Air Act, $PM_{2.5}$ levels in the United States have started to exhibit an annual increase of 5.5% since 2016 (Berkley, 2022). This rise can be in part attributed to more frequent wildfires (Burke et al., 2022) and other factors such as increased dependence on fuel oil or coal in certain areas (Berkley, 2022).

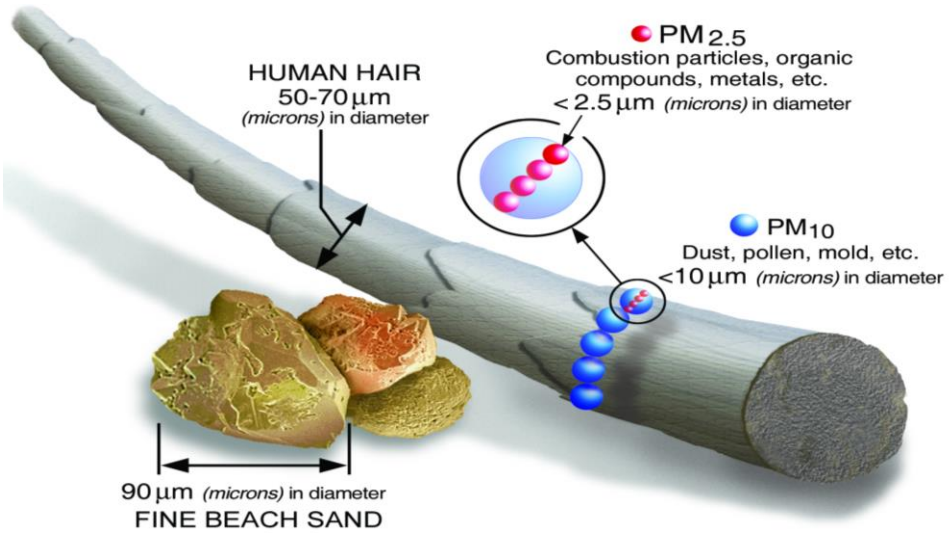

Figure 3: The analogy schematic of particulate matters ($PM_{2.5}$) (Adapted from US EPA)

$PM_{2.5}$ presents substantial public health risks globally, including in developed nations such as the US, and is particularly pronounced in underdeveloped and developing countries in Asia where $PM_{2.5}$ concentrations are notably higher (Fleischer et al., 2014). It was found that every one-unit increase in $PM_{2.5}$ annually is associated with a 14.5% increase in deaths of children under the age of five in Asian countries (Anwar et al., 2021). In developed nations such as the US, even with structural and regulatory attempts to curve $PM_{2.5}$ levels, climate change, topography, and increased dependence on fossil fuel are contributing to high exposures in some regions (Lelieveld et al., 2015).

$PM_{2.5}$ represents a toxic hazard to fetal development, impacting not only via chronic exposure but also through acute, short-term exposure. Research has demonstrated adverse health outcomes in fetuses and infants resulting from transient exposures to air pollution (Dastoorpoor et al., 2021; Faiz et al., 2013; Mendola et al., 2017; Sarovar et al., 2020b). While existing studies demonstrate a positive association, further research is required due to variations in geographical context, design methodology, sample size, pollution levels, population attributes, exposure metrics, and confounding across studies. Current literature primarily targets chronic exposures, with limited studies on the impact of transient short-term exposures. Moreover, prior studies on air pollution and fetal outcomes are limited from confounding as there are multiple differences between exposed and unexposed individuals that can also affect fetal outcomes. A study design that can allow us to study short-term exposures and adjust for confounding while accommodating time-variable elements like seasons, temperature, and humidity, can improve our estimation of the relationship between $PM_{2.5}$ and fetal/infant death. It is of paramount importance to prioritize research in an underserved area, as these locales often encompass susceptible racial and ethnic groups such as Black and Hispanic communities with low socioeconomic standing. These populations tend to inhabit areas with higher pollution levels compared to their White counterparts, resulting in them bearing an unequal burden of the harmful effects of air pollution (Woodruff et al., 2003).

Moreover, a systematic review conducted earlier concluded that the evidence linking PM with infant death isn't substantial or consistent (Glinianaia et al., 2004). Yet, the same study also hinted at the presence of some steady evidence demonstrating a link between PM and post-neonatal death. Research assessing the influence of regional differences in PM has shown detrimental effects on post-neonatal death, as evidenced in specific cohort and case-control studies (Ritz et al., 2006; Woodruff et al., 2008b; Woodruff et al., 1997). Nonetheless, research exploring the effects of brief periods of exposure to PM on infant death, using methodologies such as time-series and case-crossover analysis, have also yielded inconclusive findings (Carbajal-Arroyo et al., 2011; Ha et al., 2003; Hajat et al., 2007; Loomis et al., 1999; Scheers et al., 2011; Son et al., 2008; Tsai et al., 2003; Yang et al., 2006). Given the scarcity of existing on this topic, the relationship between PM and infant death is still unclear and warrants more attention.

1.5. Ozone and epidemiologic associations with fetal and infant death

Another common air pollutant is O_3, a gas shown to be harmful to pregnant people (Gao et al., 2022). Several studies indicated positive associations between acute O_3 exposure and adverse birth outcomes. For instance, in a US multi-center study, short-term exposure to O_3 in the week leading up to delivery was associated with the risk of fetal death (Mendola et al., 2017). Similarly, exposure to O_3 during the preceding three to five days was associated with infant death in Mexico City (Loomis et al., 1999). Even after accounting for other pollutants such as $PM_{2.5}$ and NO_2 in the same study, the impact of O_3 on fetal death remained consistent. Moreover, chronic exposure to O_3 was associated with a 9% increased risk of fetal death per 3.6-ppb increase in ozone concentration (Rammah et al., 2019) . A cohort study conducted in California found that exposure to O_3 during the third trimester was associated with fetal death risk, but exposures during earlier trimesters or averaged over the entire pregnancy did not show an association (Green et al., 2015). In contrast, a population-based case-control study in Taiwan showed no association between exposure to O_3 and fetal death after controlling for other pollutants (Hwang et al., 2011). Similar findings were obtained from a population-based prospective cohort study conducted in Wuhan, China, which reported no association between O_3 and fetal death at any stage of pregnancy (Yang et al., 2018).

Current research predominantly focuses on the effects of chronic exposure to air pollutants, often looking the role of short-term increases in O_3 levels. This oversight is compounded by the scarcity of studies specifically examining the relationship between O_3 exposure and fetal/infant outcomes. Furthermore, existing studies vary in study design, exposure and outcome assessments, ambient air pollutant concentrations, and statistical methodologies, making comparisons difficult, preventing a clear understanding of the role of O3 on early life mortality. Investigating the short-term impacts of spikes in O_3 and $PM_{2.5}$ concentrations is essential for elucidating the mechanisms through which short-term exposure might induce adverse outcomes. This approach is critical for highlighting the pathways of harm that can manifest within brief exposure periods, thereby emphasizing the considerable risks and potentially fatal consequences of transient air pollution exposure.

1.6. Biological Mechanism

Mounting data indicates that the biological mechanisms underlying the associations between air pollution and adverse pregnancy outcomes encompass various processes such as oxidative stress, blood clotting, maternal placental inflammation, hypertension, and possibly endocrine disruption (Slama et al., 2007). These processes may interfere with placenta growth, transplacental oxygen transport, and nutrient transport, which may promote fetal epigenetic changes, delayed growth, or increase infection susceptibility (Kannan et al., 2006; Slama et al., 2008) all of which have important contribution to both immediate and subsequent offspring health.

The mechanism by which environmental pollutants induce oxidative stress and inflammation is not fully understood, but it is believed that ROS levels can surge significantly in response to environmental stressors (Di Meo et al., 2016; Kannan et al., 2006; Li et al., 2003). Such stressors include exposure to air pollutants, chemicals, or toxins, which can trigger inflammation signals, that inhibit the function of antioxidant enzymes, resulting in substantial damage to cellular structures (Di Meo et al., 2016; Donaldson et al., 2001; Li et al., 2003; Rahman et al., 2006). Moreover, these conditions can induce harm to proteins, DNA, and lipids, which in turn can contribute to further inflammation and other detrimental biological impacts (Li et al., 2003; Li et al., 2019; Maccani & Marsit, 2009).

Recently it was discovered that $PM_{2.5}$ can cross the placenta in humans and can have direct exposure to the fetus during the most vulnerable stage of development (Bove et al., 2019), enter the fetal bloodstream, and harm the developing fetus through oxidative stress and inflammation (Janssen et al., 2012; Lodovici & Bigagli, 2011; Slama et al., 2008). Upon entering the fetal system, these particles can trigger oxidative stress and inflammation by amplifying the production of ROS and inflammatory cytokines (Dadvand et al., 2013). This can damage fetal tissues and impair normal fetal growth and development, potentially leading to adverse health effects such as fetal growth restriction, premature birth, and even death of the fetus (Dadvand et al., 2013). Likewise, exposure to $PM_{2.5}$ during the postnatal period may elicit comparable pathophysiological reactions, including the impact on oxidative stress, autonomic nervous system, and inflammation in mothers (Khadka & Canning, 2021). These adverse physiological changes, coupled with the immature immune and respiratory systems of infants, contribute to the risk of infant morbidity and mortality (Martinelli et al., 2013).

Exposure to O_3 has also been associated with elevated production of ROS and harm to cell structures, which consequently induces inflammation and impairs cellular function (Bai et al., 2022; Zhang et al., 2019). These changes may disrupt placental function and alter fetal development (Chung et al., 2021). Consistent with these mechanisms, research has indicated a connection between exposure to increased levels of O_3 during pregnancy and negative birth consequences, including premature birth and inhibited fetal growth (Rappazzo et al., 2021).

1.7. Windows of susceptibility

The idea of "windows of susceptibility" implies that the timing of exposure is crucial in deciding the influence on subsequent health outcomes (Fudvoye et al., 2014; Wells, 2014). It is defined as the windows of exposure specific to certain periods in a person's life, such as pregnancy, prenatal, and post-natal periods, during which the vulnerability to specific environmental or lifestyle influences is notably higher (Gómez-Roig et al., 2021). Studies indicate that exposure to environmental factors during key developmental stages —in utero, early childhood, puberty, and pregnancy — can elicit enduring effects on both mother and offspring (Gómez-Roig et al., 2021; Miguel et al., 2019; Newkirk, 2017). These phases, often termed "windows of susceptibility," represent times of significant bodily changes, thereby increasing sensitivity to environmental impacts (Fitzgerald et al., 2020). With the fast progression of fetal development, the windows of susceptibility can be overlooked if exposure assessments are not carried out within the appropriate timeframe. The most susceptible groups in a population, such as children, are the first to feel the impact due to their distinctive physical, biological, and social traits (Bearer, 1995). As the boundaries of these windows of exposure are not always clear in fetuses and children, researchers are left to speculate about when they occur (Wright, 2017).

Long-term exposures to air pollutants such as $PM_{2.5}$ and O_3 have been linked to fatal outcomes in fetuses and infants. Studies focused on exposures during specific trimesters suggest that exposure to $PM_{2.5}$ during the first trimester and throughout pregnancy can lead to an increased risk of fetal deaths (Hwang et al., 2011; Wainstock et al., 2021). $PM_{2.5}$ exposures during any or all stages of pregnancy can elevate the likelihood of other adverse pregnancy outcomes like preterm births and low birth weights (Chersich et al., 2020; Kirwa et al., 2021; MacDorman, 2011; Padula et al., 2014; Rosa et al., 2017; Siddika et al., 2019), both of which significantly contribute to infant death (Huynh et al., 2006; Mekonnen et al., 2021). Similarly, O_3 levels during the first trimester are associated with a greater risk of pre-eclampsia and early delivery (Olsson et al., 2013). A study by Olsson et al. estimated that O_3 exposure could be responsible for about 5% of pre-eclampsia instances, which may also cause fetal death before birth (Olsson et al., 2013). Additionally, babies born prematurely may face a higher likelihood of infant death (Khadka & Canning, 2021). Various studies have examined exposures during specific trimesters as potentially vulnerable windows for fetal and infant deaths, with the majority indicating that the third trimester is the most sensitive period for exposure (deSouza et al., 2022; Mendola et al., 2017; Son et al., 2017). Investigating critical exposure windows specific to each trimester is commonly preferred due to its straightforward implementation and interpretation. Recently a study suggested that associations with trimester-specific exposures may not capture the whole picture because biological changes are continuous and do not strictly adhere to these three-month intervals (Wilson et al., 2017). In addition, given that some fetal deaths occur before reaching the third trimester, the understanding of trimester-specific windows of exposure may not always provide an accurate representation for fetal deaths because such analyses would exclude those born early without a third trimester.

In addition to the long-term chronic exposures, some studies also explored acute exposures (Chen et al., 2023; Mendola et al., 2017; Sarovar et al., 2020; Thurston & Ito, 2001; Yorifuji et al., 2016). Among the few studies on acute exposures to air pollutants, a case-crossover study by Sarovar et al., explored the link between short-term exposure to air pollutants such as $PM_{10-2.5}$ and O_3 and fetal death (Sarovar et al., 2020). They found that the critical windows of exposure were approximately two days and four days before fetal death for $PM_{10-2.5}$ and O_3, respectively (Sarovar et al., 2020). These findings suggest a relatively quick impact after prenatal exposure. The study by Loomis et al. provides additional evidence of the acute effects of prenatal and postnatal exposure to $PM_{2.5}$ on fetal and infant death.

1.8. Susceptible populations

Evidence suggests that certain groups within the population may be more susceptible to the health impacts of air pollution (Wang et al., 1997; Wilhelm & Ritz, 2003). For example, at the same level of exposure, males have a higher likelihood of having adverse birth outcomes such as premature birth and low birth weight birth in comparison to females (Ghosh et al., 2007; Zeitlin, 2002). Research also suggests that boys may be more vulnerable to the effects of air pollution on fetal development (Bertin et al., 2015). Similarly, the impacts of prenatal exposure to air pollution at 6–12 weeks of gestation on forced expiratory volume are more apparent in boys (Bose et al., 2018). The differential outcomes observable in intrauterine growth and vulnerability to oxidative stress are notably pronounced in male fetuses (Bolton et al., 2014; Jarvis, 2005). This disparity may be attributed to the variations in the levels and roles of sex hormones (Bertin et al., 2015). A study shows that among preterm cases, specifically those born before the 32nd week of gestation, chronic inflammation, measured by molecular markers of intrauterine inflammation was more frequently observed in the placentas of male fetuses compared with those of females (Goldenberg et al., 2006). Additional studies are needed to examine how sex-specific susceptibilities may affect the risk of fetal and infant deaths when exposed to air pollution.

There is also evidence that underserved populations may be affected more by air pollution (Bevan et al., 2021; Mekonnen et al., 2021; Woodruff et al., 2003). For example, individuals residing in lower-income areas have a higher likelihood of encountering detrimental birth outcomes, such as premature birth, restricted fetal growth, and shorter gestation periods, even when exposed to comparable levels of air pollution to more affluent areas (do Nascimento et al., 2022). Another study found that mothers who were Hispanic, Asian, Pacific Islander, and African-American were more vulnerable to adverse pregnancy outcomes compared with mothers who were White even at the same level of exposure to air pollution (Woodruff et al., 2003). These differences are likely attributable to variations in structural differences and socioeconomic characteristics that could foster disparities (do Nascimento et al., 2022; Jabin et al., 2022; Jang & Lee, 2022; Jbaily et al., 2022), including inadequate healthcare access, discrimination, and other social stressors (Horbar et al., 2019). For instance, areas with high pollution often coincide with disadvantaged communities, which are simultaneously more likely to have limited healthcare, poorer nutrition, fewer job opportunities, and other stressors

(American Lung Association, 2023a). As such, there is a need for more studies focusing on the underserved population or groups of the population who are highly affected by air pollution. Such studies can contribute to understanding the widening gap in the deaths of fetuses and infants in the US.

1.9. The San Joaquin Valley, California

The San Joaquin Valley (SJV) is located in the central region of California with a population of 4.3 million and is widely recognized as one of the most productive agricultural areas globally. The valley stretches southward from San Joaquin County across Stanislaus, Merced, Madera, Fresno, Tulare, Kings, and Kern counties which constitutes approximately 11% of the total population in the state (Public Policy Institute of California, 2020). Meanwhile, it is considered one of California's most underserved areas with marked health and resource disparities. In contrast to the rest of the state, the SJV had a 25% higher infant death in 2018 (6 vs. 4 per 1,000) (Finocchio & Paci, 2020). In 2018, the median household income in the SJV was significantly lower ($52,621) compared with the rest of California ($75,277) (United States Census Bureau, 2018). Meanwhile, there are also severe healthcare shortages in the region (Finocchio & Paci, 2020). The proportion of mothers accessing prenatal care during the first trimester in the counties of the SJV is significantly lower when compared to the statewide average in California. Specifically, statewide data indicates that 86.8% of mothers receive prenatal care in the first trimester. In contrast, the counties within the SJV report lower percentages: Merced at 75.6%, Madera at 81.3%, San Joaquin at 81.2%, Kern at 82.1%, Tulare at 82.7%, Fresno at 85.3%, Kings at 85.8%, and Stanislaus at 85.9% (KidsData, 2021). This discrepancy highlights a regional disparity in the access to early prenatal care across the state indicating potential barriers to healthcare services for pregnant women.

The SJV experiences significant levels of $PM_{2.5}$ and O_3 pollution originating from various sources including residential wood burning, agricultural activities, and transportation (Chen et al., 2014). This is compounded by the presence of two major north-south highways (e.g. Interstate 5 (I-5) and State Route 99 (SR-99)) running through the region. The topography and weather patterns in the SJV make air pollution worse by preventing pollutants from being dispersed. The mountains surrounding the valley trap the pollutants, and the still air does not help to move them out of the area (SJVAPD, 2016). Due to the specific topography of SJV, it experiences some of the worst air quality in the states (Padula et al., 2014).. These conditions make it challenging to maintain healthy air quality levels in the SJV, particularly for vulnerable populations such as pregnant mothers and children.

1.10. Rationale of the study

Despite public health concerns, to date, no studies have evaluated the relationship between air pollution and fetal/infant death in the SJV. Nevertheless, studies have linked air pollution with adverse birth outcomes in this region (Ha, Martinez, et al., 2022; Padula et al., 2014; Padula et al., 2019). For example, an investigation was conducted on the impact of long-term air pollution on preterm birth in four SJV counties and found that being exposed to the highest quarter of air pollution levels was linked to an increased

likelihood of early preterm births, particularly during the second trimester and towards the end of the pregnancy period. The association was more pronounced for mothers residing in neighborhoods characterized by lower socioeconomic status (Padula et al., 2014, 2019). More recently, our team implemented the case-crossover design to investigate the short-term impacts of pollution on preterm birth and found that $PM_{2.5}$ and O_3 exposures were associated with preterm birth risks in the cold and warm seasons, respectively (Ha et al., 2022).

Given the lack of existing studies on fetal/infant death, further investigation of the risk of fetal and infant death in this highly polluted region is critical. Such knowledge can support strategies intended to decrease the burden of early-life fatalities. This understanding is also crucial for pollution management and planning strategies for a safer environment for vulnerable populations like pregnant women and their offspring representing racial minorities and belonging to low socioeconomic status. As mentioned above it is difficult to fully comprehend the relationship between air pollution and adverse health outcomes due to inconsistent findings, lack of identification of precise critical windows of exposure during pregnancy and after birth during early life, varying study designs, geographic locations, seasonal and time trends, population differences, and differences in the measurement of exposures, outcome, and confounding factors. Moreover, confounding is an inevitable issue in observational air pollution studies. To address this issue, an epidemiological design such as a time-stratified case-crossover analysis would be a significant addition to the literature as it can effectively address time-invariant and time-variant confounding.

1.11. Objectives and hypotheses

Given the knowledge gaps and evidence identified above, the overarching goal of this study was to understand the associations between exposure to acute air pollution such as $PM_{2.5}$ and O_3 and the odds of fetal and infant death in the SJV in California using a time-stratified case-crossover study design. The specific objectives of this study are to:

Objective 1: Investigate the relationship between acute exposure to air pollutants (particularly $PM_{2.5}$ and O_3) and the odds of fetal and infant death in the SJV.

> *Hypothesis 1.1:* Prenatal and postnatal exposure to air pollutants such as $PM_{2.5}$ and O_3 increase the risk of fetal and infant death.
>
> *Hypothesis 1.2:* $PM_{2.5}$ in the cold and O_3 in the warm seasons are associated with an increased risk of fetal and infant death.

Objective 2: Identify groups who may be more susceptible to the risk of fetal and infant death even at the same level of exposure.

> *Hypothesis 2.1:* The same level of air pollution exposure in communities of color and more disadvantaged populations may experience higher risk compared with predominantly White or economically more affluent populations.

Objective 3: Identify prenatal and postnatal critical acute windows of exposures across a 14-day lag period for fetal and infant death with $PM_{2.5}$ and O_3 exposures.

Hypothesis 3.1: Exposures to $PM_{2.5}$ and O_3 increase the risk of fetal and infant death within 14 days.

2. Materials and methods

2.1. Study design

The acute effects of air pollution on fetal and infant death were explored using a time-stratified case-crossover design, a study design widely and increasingly recognized for its utility in studying the association between short-term exposure to air pollution and acute health outcomes (Maclure, 1991). This analytical approach is a unique variation of the case-control study design and is particularly designed to adjust for confounders that do not change over time (Carracedo-Martinez et al., 2010). In contrast to traditional case-control designs, which compare exposure among cases with that of controls who have not encountered a particular outcome of interest, the case-crossover design compares exposure during a specific period when the outcome occurred (case period) to exposures during several control periods when the outcome did not occur (control periods). Through the comparison of an individual's exposure during the case period with their exposure during control periods, this approach allows each case to serve as its control and better control of time-invariant confounding factors that may impact the exposure-outcome relationship including genetic predispositions, chronic health conditions, and personal traits that remain stable over a relatively short span of a few weeks. We defined the time frame for case periods as the day of event (lag 0), extending back to the preceding fourteen days (lag 1–lag 14) following previous studies (Chen et al., 2021). Previous research has predominantly employed a lag duration ranging from one to seven days, coinciding with the week preceding the event, to explore the impacts of air pollution on diverse birth outcomes (Ha, Martinez, et al., 2022; Sarovar et al., 2020; Son et al., 2008) highlighting that while biological impacts are seen within a week, the precise timing of these effects remains uncertain. The impact of air pollution on birth outcomes may extend beyond the traditionally considered one-week window due to pollutants like $PM_{2.5}$ causing systemic inflammation and oxidative stress over longer periods. These effects, which arise from both direct and indirect exposure, might not manifest within a week as physiological changes develop over time. Considering the complexity of air pollution's effects, varying exposure levels, and individual differences, it appears reasonable to explore a 14-day window for adverse birth outcomes. This approach, advocating for expanded research into the timing and mechanisms of air pollution's impact on fetal development, suggests that a two-week exposure period could provide a clearer understanding of the risks to birth outcomes.

We used a method called time-stratified control selection to choose control periods **(Figure S1)**. In addressing the influence of time-related trends and variations in seasons, control intervals were determined for three weeks both before and following each instance of fetal and infant death. Acknowledging that the outcomes of case-crossover analyses can be heavily influenced by how control periods are chosen, we employed a two-sided control selection method known as bidirectional control sampling, involving the use of three to four varied control periods to accurately determine the

relationship between air pollution and fetal and infant death. For each defined case period, we selected all additional days within the same month that corresponded to the same weekday. For instance, if an event occurred on Friday, August 9th, 2019, then control periods would be all the other Fridays in that same month, including August 2nd, 16th, 23rd, and 30th. This strategy lets us match each case period with three to four control periods for the same person (Ha, Martinez, et al., 2022; Janes et al., 2005; Kojima et al., 2020; Nitta et al., 2010). One might wonder whether it's appropriate to choose control periods after the health event because the individuals are no longer at risk. However, this technique is considered appropriate and acceptable as individual events are unlikely to influence the collective exposure levels in the study group, and exposure to air pollution is typically perceived as an external variable to the individual. Therefore, selecting post-event control periods can still be an effective way to control for potential confounding factors (Mittleman & Mostofsky, 2014).

2.2. Study data and population

We obtained linked birth and death certificates of all births born in the state of California from 2016 to 2019 from the Office of Vital Records of the California Department of Public Health (CDPH). This comprehensive dataset includes all births and any subsequent death within one year of a live birth. The vital record files contain information about the newborn, mother, and father's demographics, along with some obstetric data related to pregnancy and delivery.

The initial sample included 1,881,390 participants from the state of California from 2016 to 2019. After limiting the sample to eight SJV counties (Kern, Kings, Fresno, Madera, Merced, Tulare, San Joaquin, and Stanislaus), the sample size was reduced to 244,140. After excluding observations with missing birth dates (n=141), those with gestation weeks <20 and >42 (n=607), multiple gestations (n=7214), and those with no zip-code for air pollution linkages (n=767), 235, 411 singleton births remained in our eligible population. As case-crossover design only utilizes cases, our final sample includes 1,343 fetal and 1,097 infant deaths **(Figure S2)**.

2.3. Exposure assessment

We obtained daily $PM_{2.5}$ (24-hour average) and O_3 (maximum 8-hour average) concentrations from the Valley (SJVAPCD, 2021). Air District utilizes a method rooted in regression analysis to establish the levels of air pollution within each zip code. This method integrates data from the Environmental Protection Agency's (EPA) Air Quality System monitors, as well as insights derived from the EPA's Community Multiscale Air Quality models. The latter considers emission metrics, meteorological data, and readings from local air quality monitoring systems. This modeling method has been previously validated and is known to replicate historical data effectively. Specifically, the R^2 values for the correlation between the modeled and observed data exceed 0.90 for both pollutants (Dunes D.A, 2015). To estimate individual exposures, we spatiotemporally linked participants with the daily air pollution data from the SJVAPCD. This was achieved by matching the mother's residential zip code at the time of birth, as stated on the birth certificate.

Numerous studies reported associations between exposure to temperature and fetal and infant death risk (Auger et al., 2015; Ha et al., 2017). We aggregated daily weather parameters, such as highest and lowest temperatures and relative humidity, for each zip code using data from gridMET (Abatzoglou, 2013). GridMET provides detailed weather estimates for 4km x 4km areas throughout the entire US and allows for comprehensive coverage in both space and time that is usually hard to achieve with data collected from individual weather stations. We then linked these weather variables with the zip codes of each participant. To assign these exposure levels, we calculated the average temperature and relative humidity exposure across each participant's zip code. This study examined the two-week windows of exposure before fetal and infant deaths, with "lag 0" representing the day of the event and lags 1-14 representing one to 14 days leading up to these events.

2.4. Time-invariant variables and effect modifiers

Time-invariant variables included in this study were maternal sociodemographic characteristics including educational status, age, maternal race/ethnicity, neighborhood income, prenatal care received, Women Infants and Children (WIC) program participation, principal source of payment for prenatal care, smoking status during pregnancy, maternal predelivery body mass index (BMI), and sex of the child.

To explore potential differential effects of air pollution across different groups, we considered effect modifiers including sex of the child, maternal race/ethnicity, maternal age, and neighborhood income. Median neighborhood income was obtained from the US American Community Survey 2010 data and linked to birth and death certificates at the zip-code level (American Community Survey, 2010).

2.5. Outcome assessment

The main outcomes of interest, fetal and infant deaths, were assessed using linked birth and death certificates from the California Office of Vital Statistics. We defined fetal death as any death of an unborn fetus occurring between 20 to 42 weeks of gestation. Infant death was defined as the death of a live-born infant within the first year of life. While we were able to follow each live birth to document death within one year, given the nature of birth certificate data, it was not feasible to establish a longitudinal connection between pregnancies of the same mother. Consequently, the analysis was not conducted based on individual women but rather on specific singleton pregnancies/babies affected by the outcomes.

2.6. Statistical analysis

Descriptive analyses were conducted on all time-invariant variables to provide an overview of the characteristics of the cases under study. Following this, an examination of the distribution of air pollutants and weather variables during the study period (2016 – 2019) was undertaken to contextualize the environmental conditions. To extend this analysis, we presented the distribution of air pollutants among participants, comparing both case and control periods across all lag days. Additionally, we also conducted Spearman correlation coefficient analysis to understand relationships between air

pollutants in our study across time. This analysis was conducted for all pollutants between lags, as well as directly between the air pollutants and weather variables themselves across all lags. We tested the normality of our exposure variables. The results showed that O_3 and temperature had a normal distribution. $PM_{2.5}$ and relative humidity also had a normal distribution, but they were skewed to the right.

We used conditional logistic regression models to compare air pollution exposures during the case and control periods within the same person **(Figure S3)**. For each pollutant of interest, we tested square and higher-order polynomial terms and visually plotted pollutant concentrations against the odds of the event. These exploratory analyses did not yield any indication of a non-linear association between air pollutants and the logarithmic odds of fetal and infant death cases. As such, we assumed a linear relationship between the pollutants and death onset, and calculated the odds ratio (OR) for each $10\mu g/m^3$ increase in $PM_{2.5}$ (Woodruff et al., 2006; Yorifuji et al., 2016) and 10-ppb increase in O_3 exposure (Green et al., 2015) with its respective 95% confidence intervals. Due to strong seasonality of air pollution in the SJV, we stratified our analyses by season of delivery for fetal death and season of death for infant death. We defined cold season as November through April and the warm season as May through October (Ha, Martinez, et al., 2022). Prior studies have also used these seasonal definitions and stratification of pollutants by season under their analysis (Basu et al., 2016; Jhun et al., 2015). As the case-crossover design already inherently adjusts for time-invariant confounding, we only modelled time-varying confounding factors such as temperature, humidity, and co-pollutant as covariates. Further analyses included a) whole-year analysis model b) a single pollutant model for each of the pollutants c) models stratified by neighborhood income, maternal age, race/ethnicity, and sex of the child. These stratified analyses aimed to investigate whether contextual and individual characteristics had a potential influence in modifying the relationship between air pollution and the outcomes. While our analyses did reveal a few effect modifiers for air pollution that were statistically significant, the reliability of the odds ratio estimates was compromised due to the limited sample size, yielding no coherent pattern of observation. For instance, maternal race, an examined variable in this study, had a very small sample size for significant race/ethnicity (e.g., Asian and Hawaiian/PI). Despite having statistical significance, it did not demonstrate any stable estimates and meaningful observations. Thus, stratification by maternal race is not depicted in the results. The final statistical model included O_3, $PM_{2.5}$, temperature, humidity, season of birth/death (depending upon the outcome in the model), lag effect, interaction terms between each pollutant and lag day, interaction terms between each pollutant and season of birth/death. We also performed a normality test for the air pollutants and weather variables, it was observed that the variables O_3 and temperature were normally distributed. Similarly, $PM_{2.5}$ and relative humidity followed normal distribution; however, they were right-skewed. We also conducted several additional analyses to further explore the relationship between air pollution and fetal and infant death. All analyses were performed utilizing SAS version 9.4 (Cary, NC).

2.6.1. Additional analysis

We also conducted additional testing adjusting for other lag days to assess the temporal dependance between lags, given their high correlation. The estimates were found to be consistent with those in the primary model but with wider confidence intervals. This analysis confirms that the estimates were indeed independent of the temporal dependance between lags within the primary models.

In our analysis, we observed inverse associations within the models that prompted further methodological refinement. We implemented several strategies, including log-transformation and z-standardization of the pollutants. Despite this exploration, the inverse associations persisted. Consequently, we adhered to our original modeling approach, as it provided the most consistent and interpretable framework for our study.

2.7. Missing data

In the study, participants with missing data on maternal characteristics were categorized as 'unknown'. While there was some missing data, the extent was generally low below 7%. All observations containing missing data on time-invariant factors were retained, as these variables were not required in the model. This approach ensured that valuable information could still be obtained from the available data, without compromising the integrity of the main analysis.

2.8. Ethics

The research protocol of this study was approved by the review board at the UCM (IRB number: UCM2017-128 and UCM2022-69). The approval from this ethical review board ensured that the study adhered to the requisite ethical principles and regulatory guidelines. This study was also approved by the California Committee for the Protection of Human Subjects.

3. Results

3.1.Population statistics

The prevalence of fetal death during the study period was 0.57% while that of infant death was 0.47%. **Table 1** provides an overview of the characteristics of the participants (only cases) involved in the study. A higher proportion of cases occurred during the warm season and were male. The majority of the babies had mothers between 26-35 years old, were Hispanic, had some college, had public insurance, and were non-smokers. In addition, fetal deaths were most likely in neighborhoods with median household income in the third quartile, while infant deaths were more likely to come from neighborhoods in the lowest quartile (26.89%).

Table 1: Characteristic of fetal and infant deaths in the SJV, California, 2016-2019 (N=2440)

Demographic characteristics	Fetal death (N = 1343)	Infant death (N=1097)

	Frequency (N)	Percentage (%)	Frequency (N)	Percentage (%)
Sex of child				
Male	698	51.97	Missing # here	55.61
Female	634	47.21	486	44.30
Undetermined	11	0.82	1	0.09
Age of mother				
Less than 18 years	27	2.01	17	1.55
Between 18-25 years	388	28.89	409	37.28
Between 26-35 years	628	46.76	478	43.57
Greater than 35 years	293	21.82	192	17.50
Missing	7	0.52	1	0.09
Race of mother				
Non-Hispanic White	308	22.93	238	21.70
Non-Hispanic Black	109	8.12	92	8.39
Hispanic	779	58.00	619	56.43
Asian	111	8.27	76	6.93
Hawaiian/PI	11	0.82	2	0.18
AI/AN	8	0.60	8	0.73
Other	4	0.30	19	1.73
Missing	13	0.97	43	3.92
Body Mass Index of mother (BMI)				
Underweight	37	2.76	29	2.64
Normal	333	24.8	314	28.62
Overweight	310	23.08	272	24.79
Obese	456	33.95	375	34.18
Unknown	207	15.41	107	9.75
Education of mother				
Less than high school	264	19.66	233	21.24
HS/GED	439	32.69	335	30.54
Some college/Bachelor	495	36.86	384	35
Advanced degree	43	3.2	29	2.64
Unknown	102	7.59	116	10.57
Smoking status of mother				
Not a smoker	1106	82.35	1035	94.35
Smoker	106	7.89	47	4.28
Unknown	131	9.75	15	1.37
Prenatal care				

No prenatal care	-	-	59	5.38
Early prenatal care	-	-	858	78.21
Late prenatal care	-	-	102	9.3
Missing	-	-	78	7.11
Principal source of payment for prenatal care				
No insurance/no prenatal care	73	5.44	59	5.38
Public	733	54.58	715	65.18
Private	383	28.52	299	27.26
Self-pay	16	1.19	17	1.55
Other	10	0.74	-	-
Unknown	128	9.53	7	0.64
WIC				
Yes	539	40.13	667	60.8
No	595	44.3	407	37.1
Unknown/Not stated	209	15.56	23	2.1
Neighborhood household income				
Quartile 1 (<$36025.85)	312	23.23	295	26.89
Quartile 2 ($36025.85 - $46624.35)	326	24.27	277	25.25
Quartile 3 ($46624.35 - $53664.82)	373	27.77	252	22.97
Quartile 4 (=$53664.82)	322	23.98	266	24.25
Missing	10	0.74	7	0.64
Season of birth				
Warm season (May-October)	672	50.04	588	53.6
Cold season (November-April)	671	49.96	509	46.4
Season of death				
Warm season (May-October)	-	-	578	52.69
Cold season (November-April)	-	-	519	47.31

Abbreviations: PI=Pacific Islander; AI/AN= American Indian/Alaska Native;
HS/GED = High School/ General Education Diploma; WIC= Women Infants Children; SJV= San Joaquin Valley.

3.2.Exposure statistics

3.2.1. Exposures statistics for fetal death

The distribution of air pollutants ($PM_{2.5}$, $O_{3)}$ and meteorological variables (temperature, relative humidity) during the study period is presented in **Table S1**. The average levels of $PM_{2.5}$ were notably higher during the cold season, while O_3 concentrations were significantly elevated in the warm season. More specifically, the mean concentrations of $PM_{2.5}$ in cold months and O_3 in warm months were 13.1 $\mu g/m^3$ and 57.5 ppb respectively. The standards set by the United States Environmental

Protection Agency for annual mean concentration of $PM_{2.5}$ is 9mg/m^3 and O_3 is 70ppb (USEPA, 2022a). The distribution of exposures during case and control periods for both the pollutants for fetal deaths are presented in **Table S2.** During the warm season, the control period generally exhibited higher $PM_{2.5}$ concentrations across most lag days compared to the case period. In contrast, during the cold season, higher PM2.5 levels were observed in the case period for later lag days, while immediate lag days had higher concentrations in the control period. In the warm season, ozone (O3) concentrations were comparable between the case and control periods. Conversely, during the cold season, slightly elevated O3 levels were observed in the control period. **Table S3** provides the Pearson correlation coefficients between air pollutants and meteorological variables by season. A positive correlation (r = 0.4) was observed between $PM_{2.5}$ and O_3 during the warm season, whereas a negative correlation (r = -0.38) was identified during the cold season. The correlation coefficient between O_3 and temperature was strongly positive across both seasons, with a coefficient of r = 0.7. **Table S4** illustrates the Pearson correlation coefficients between the lags (lag 0 to lag 14) of air pollutants and meteorological variables. Correlation coefficients for adjacent lag periods exhibited substantially higher values than those for lag periods that were further apart.

3.2.2. Exposures statistics for infant death

Table S5 shows the distribution of exposures during case and control periods for both the pollutants for infant deaths. In the warm season, higher $PM_{2.5}$ concentrations were observed in the case period during immediate lag days (lag0–lag6), while the control period had higher levels during later lag days (lag7–lag14). In contrast, the cold season showed slightly elevated $PM_{2.5}$ concentrations in the case period compared to the control period. For ozone (O_3), the warm season had consistently higher concentrations in the case period across all lag days, while the cold season exhibited slightly higher O_3 levels in the control period. **Table S6** displays a positive correlation (r = 0.4) between $PM_{2.5}$ and O_3 during the warm season, whereas the cold season reflected a negative correlation (r = -0.4). A positive correlation was consistently found between O_3 and temperature in both warm and cold seasons (r = 0.7). Further, **Table S7** presents the Spearman correlation coefficients between various lags (from lag 0 to lag 14) of air pollutants and weather variables. The correlation coefficients between lags in closer proximity were significantly higher compared to those observed between the most distant lag days.

3.3.Main findings

Table 2 presents the results from the conditional logistic regression models estimating the odds ratios for the association between air pollutants and fetal and infant death risk in the SJV by seasons of delivery for fetal death and season of death for infant death.

3.3.1. Associations between air pollutants and fetal death

In Table 2, $PM_{2.5}$ exposures during the cold season generally appeared to be associated with higher odds of fetal death on the same day and within two weeks. However, only the odds associated with six days (lag 6), seven days (lag 7), ten days (lag

10), and fourteen days (lag 14) post-exposure were statistically significant. More specifically, a 10 mg/m^3 increase in $PM_{2.5}$ concentration was associated with 5-7% increased odds of fetal death, with evidence of higher risk estimate with larger lag days (aOR lag_6 1.05, 95% CI 1.00,1.12); (aOR lag_7 1.07, 95% CI 1.01,1.14); (aOR lag_{10} 1.06, 95% CI 1.00,1.13) and (aOR lag_{14} 1.06, 95% CI 1.00,1.13). Lag 7 had the strongest association compared to other significant lag days. The unadjusted model presented significant estimates, mirroring those in the adjusted model. Interestingly, inverse associations were observed for $PM_{2.5}$ during the warm season. For example, a 10 mg/m^3 increase in $PM_{2.5}$ concentration on the day of event (lag 0) in the warm season was associated with 23% (aOR lag_0 0.77, 95% CI: 0.70,0.83) decrease in risk of having fetal death.

A significant positive association was observed between O_3 exposure and fetal death in all lag days after adjusting for lag effect, $PM_{2.5}$, temperature, and relative humidity. More specifically, a 10-ppb increase in O_3 concentration was associated with 5-8% increased odds of fetal death. The risk estimates were strongest for exposures one to four days before the event (aOR lag_{1-4} 1.03, 95% CI 1.03,1.14) and in the later lag days (aOR $lag_{11-12,14}$ 1.08, 95% CI 1.03,1.14) as well as 6% increased odds (aOR lag_0 1.06, 95% CI 1.01,1.12) of fetal death on the day of the event. In comparison to the unadjusted model, the adjusted model showcased stronger associations.

3.3.2. Association between air pollutants and infant death

We did observe a trend of increased magnitude of association between exposure to $PM_{2.5}$ and infant death on most of the days (lag 0-6, lag 8-11, lag 13-14), preceding the event, although these findings lacked statistical significance **(Table 2).** On the other hand, there appeared to be inverse associations between $PM_{2.5}$ and infant death in all lags during the warm season..

We found a strong association between exposure to O_3 and infant death during the warm season. A 10-ppb rise in O_3 was linked to a 9-11% increase in the odds of infant death within a two-week exposure window across all lag days. Risks were more pronounced for exposures on event day (lag 0), three days before (lag 3), and fourteen days before (lag 14). More specifically, a 10-ppb increase in exposure to O_3 was associated with 1.11 (aOR $lag_{0,3,14}$ 1.11, 95% CI: 1.05,1.17) times the odds of fetal death on the same day (lag 0), three days (Lag 3) and fourteen days (lag 14) post-exposure. However, we also observed an inverse relationship with O3 exposure in the cold season. For example, a 10-ppb increase in exposure to O_3 was associated with a 7% (aOR lag_4 1.09, 95% CI: 1.04,1.15) decrease in odds of having infant death one day post-exposure.

Table 2: Association between air pollutants and fetal and infant deaths in the SJV, California 2016 - 2019

Pollutant	Season		Fetal death (20-42weeks)		Infant death (< 1 year)	
		Lag (day)	Unadjusted OR (95% CI)[a]	Adjusted OR (95% CI)[b]	Unadjusted OR (95% CI)[a]	Adjusted OR (95% CI)[b]
$PM_{2.5}$	Warm	0	0.76 (0.70,0.83)	0.77 (0.70,0.83)	0.90 (0.83,0.99)	0.90 (0.83,0.99)
		1	0.80 (0.73,0.87)	0.79 (0.73,0.87)	0.90 (0.83,0.99)	0.90 (0.83,0.99)

		2	0.78 (0.72,0.86)	0.78 (0.72,0.85)	0.90 (0.83,0.98)	0.90 (0.83,0.98)
		3	0.79 (0.72,0.86)	0.78 (0.72,0.86)	0.90 (0.83,0.99)	0.90 (0.83,0.99)
		4	0.81 (0.74,0.88)	0.80 (0.74,0.88)	0.89 (0.81,0.96)	0.89 (0.81,0.97)
		5	0.82 (0.75,0.89)	0.81 (0.75,0.88)	0.90 (0.83,0.98)	0.90 (0.83,0.98)
		6	0.82 (0.75,0.90)	0.81 (0.75,0.89)	0.88 (0.81,0.96)	0.88 (0.81,0.96)
		7	0.82 (0.75,0.90)	0.83 (0.76,0.90)	0.85 (0.78,0.93)	0.86 (0.78,0.94)
		8	0.82 (0.75,0.90)	0.81 (0.75,0.89)	0.88 (0.81,0.96)	0.88 (0.81,0.96)
		9	0.83 (0.75,0.90)	0.82 (0.75,0.89)	0.88 (0.81,0.96)	0.89 (0.81,0.96)
		10	0.83 (0.76,0.90)	0.82 (0.75,0.89)	0.89 (0.81,0.96)	0.89 (0.81,0.97)
		11	0.82 (0.75,0.89)	0.82 (0.75,0.89)	0.86 (0.78,0.94)	0.87 (0.79,0.95)
		12	0.79 (0.73,0.87)	0.79 (0.73,0.87)	0.84 (0.77,0.91)	0.85 (0.78,0.92)
		13	0.80 (0.73,0.87)	0.79 (0.73,0.86)	0.86 (0.79,0.94)	0.87 (0.79,0.95)
		14	0.83 (0.76,0.90)	0.82 (0.75,0.90)	0.87 (0.80,0.95)	0.87 (0.80,0.95)
	Cold	0	0.98 (0.92,1.05)	0.99 (0.93,1.06)	1.06 (0.99,1.13)	1.05 (0.98,1.12)
		1	1.03 (0.97,1.09)	1.03 (0.97,1.09)	1.06 (0.99,1.13)	1.05 (0.99,1.12)
		2	1.02 (0.95,1.08)	1.02 (0.96,1.08)	1.05 (0.98,1.12)	1.04 (0.98,1.12)
		3	1.02 (0.96,1.08)	1.02 (0.96,1.09)	1.06 (0.99,1.13)	1.04 (0.98,1.12)
		4	1.04 (0.98,1.11)	1.04 (0.98,1.11)	1.03 (0.96,1.11)	1.03 (0.96,1.09)
		5	1.05 (0.99,1.12)	1.05 (0.99,1.12)	1.05 (0.98,1.12)	1.04 (0.98,1.12)
		6	1.06 (1.00,1.12)	1.05 (1.00,1.12)	1.03 (0.96,1.09)	1.02 (0.95,1.08)
		7	1.06 (1.00,1.13)	1.07 (1.01,1.14)	1.00 (0.93,1.06)	0.99 (0.93,1.06)
		8	1.05 (0.99,1.12)	1.05 (0.99,1.12)	1.03 (0.96,1.09)	1.02 (0.95,1.08)
		9	1.06 (1.00,1.13)	1.06 (0.99,1.13)	1.02 (0.96,1.09)	1.02 (0.96,1.09)
		10	1.06 (1.00,1.13)	1.06 (1.00,1.13)	1.03 (0.96,1.11)	1.03 (0.96,1.11)
		11	1.05 (0.99,1.12)	1.06 (0.99,1.13)	1.00 (0.93,1.07)	1.00 (0.93,1.07)
		12	1.02 (0.97,1.08)	1.03 (0.97,1.09)	0.98 (0.91,1.05)	0.98 (0.91,1.05)
		13	1.03 (0.97,1.09)	1.03 (0.97,1.09)	1.01 (0.94,1.08)	1.00 (0.94,1.07)
		14	1.07 (1.01,1.13)	1.06 (1.00,1.13)	1.02 (0.95,1.08)	1.01 (0.94,1.08)
O_3	Warm	0	1.03 (0.99,1.08)	1.06 (1.01,1.12)	1.12 (1.06,1.17)	1.11 (1.05,1.17)
		1	1.05 (1.01,1.11)	1.08 (1.03,1.14)	1.11 (1.05,1.16)	1.09 (1.04,1.15)
		2	1.06 (1.01,1.11)	1.08 (1.03,1.14)	1.11 (1.05,1.16)	1.09 (1.04,1.16)
		3	1.05 (1.00,1.11)	1.08 (1.03,1.14)	1.12 (1.06,1.17)	1.11 (1.04,1.16)
		4	1.05 (1.01,1.11)	1.08 (1.03,1.14)	1.11 (1.05,1.16)	1.09 (1.04,1.16)
		5	1.04 (1.00,1.09)	1.07 (1.02,1.13)	1.09 (1.04,1.15)	1.09 (1.03,1.15)
		6	1.04 (1.00,1.09)	1.07 (1.02,1.13)	1.11 (1.05,1.17)	1.09 (1.04,1.16)
		7	1.03 (0.99,1.08)	1.05 (1.00,1.11)	1.09 (1.04,1.15)	1.09 (1.04,1.15)
		8	1.03 (0.98,1.07)	1.05 (1.00,1.11)	1.11 (1.05,1.16)	1.09 (1.04,1.16)
		9	1.02 (0.98,1.07)	1.05 (1.00,1.11)	1.09 (1.04,1.15)	1.09 (1.03,1.15)
		10	1.03 (0.98,1.08)	1.05 (1.01,1.11)	1.09 (1.04,1.15)	1.09 (1.04,1.15)
		11	1.05 (1.00,1.11)	1.08 (1.03,1.14)	1.09 (1.04,1.15)	1.09 (1.04,1.16)

		12	1.05 (1.01,1.11)	1.08 (1.03,1.14)	1.09 (1.04,1.15)	1.09 (1.04,1.16)
		13	1.04 (1.00,1.09)	1.07 (1.02,1.13)	1.11 (1.04,1.16)	1.09 (1.04,1.16)
		14	1.05 (1.00,1.11)	1.08 (1.03,1.14)	1.12 (1.06,1.17)	1.11 (1.04,1.16)
	Cold	0	0.91 (0.87,0.96)	0.93 (0.89,0.98)	0.95 (0.90,1.01)	0.94 (0.90,1.00)
		1	0.93 (0.89,0.98)	0.94 (0.90,0.99)	0.94 (0.9,1.00)	0.93 (0.89,0.99)
		2	0.93 (0.89,0.98)	0.95 (0.90,1.00)	0.94 (0.90,0.99)	0.93 (0.89,0.99)
		3	0.93 (0.89,0.98)	0.94 (0.90,0.99)	0.95 (0.90,1.01)	0.94 (0.89,0.99)
		4	0.93 (0.89,0.98)	0.94 (0.90,0.99)	0.94 (0.90,0.99)	0.93 (0.89,0.99)
		5	0.92 (0.88,0.97)	0.94 (0.90,0.99)	0.94 (0.89,0.99)	0.93 (0.88,0.98)
		6	0.92 (0.88,0.97)	0.94 (0.90,0.99)	0.95 (0.90,1.00)	0.93 (0.89,0.99)
		7	0.91 (0.87,0.96)	0.92 (0.88,0.97)	0.93 (0.89,0.99)	0.93 (0.89,0.99)
		8	0.90 (0.87,0.95)	0.92 (0.88,0.96)	0.94 (0.90,1.00)	0.93 (0.89,0.99)
		9	0.90 (0.86,0.95)	0.91 (0.87,0.96)	0.93 (0.89,0.98)	0.93 (0.88,0.98)
		10	0.91 (0.87,0.95)	0.92 (0.88,0.97)	0.93 (0.89,0.99)	0.93 (0.89,0.99)
		11	0.93 (0.89,0.97)	0.94 (0.90,0.99)	0.93 (0.89,0.99)	0.93 (0.89,0.99)
		12	0.93 (0.89,0.98)	0.94 (0.90,0.99)	0.93 (0.89,0.99)	0.94 (0.89,0.99)
		13	0.92 (0.88,0.97)	0.93 (0.89,0.98)	0.94 (0.89,0.99)	0.93 (0.89,0.99)
		14	0.93 (0.89,0.97)	0.94 (0.90,0.99)	0.95 (0.90,1.00)	0.94 (0.89,0.99)

[a]Models only adjusted for time invariant confounders by design; and ORs were obtained for 10 units increase in each pollutant concentration.
[b]Models adjusted for the other pollutant, temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5}$, particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.
Note: Bold numbers indicate a significant positive association.

3.3.3. Effect modification analysis for fetal death.

We found that the associations between air pollutants and maternal race were higher among Asian (N=111) and Hawaiian/PI (N=11). However, due to the limited sample size for some racial/ethnic groups, the estimates were unstable with wide confidence intervals (not shown in the table). We also observed effect modification by neighborhood income and maternal age. But there were no sex-specific differences.

Table 3 illustrates the associations between air pollutants and fetal death stratified by neighborhood income. $PM_{2.5}$ exposures on low-income group in the cold season demonstrated an elevated risk across all the lag days, but significant associations were only found for later lag days from nine to fourteen days after exposure. However, there were no significant association between PM2.5 and fetal death in higher income neighborhoods. The impact of O_3 exposures were observed in both low and high-income groups in the warm season. In the low-income group, we found a consistently significant positive association between O_3 exposure and odds of fetal death across all examined lag days. However, in the high-income group, these associations were observed only on specific immediate (lag 1 – lag 4) and later lag days (lag 11 – lag 14). Comparatively, the

associations between O3 exposure and fetal death were more pronounced and stronger in low- income groups.

Table 3: Association between air pollutants and fetal death by neighborhood income in the SJV, California, 2016 - 2019

Pollutant	Season	Lag (day)	Adjusted OR (95% CI)[a]	
			Low income (<50th percentile) (N=638)	High Income (>=50th percentile) (N=695)
$PM_{2.5}$	Warm	0	0.73 (0.64,0.83)	0.78 (0.69,0.89)
		1	0.75 (0.67,0.85)	0.83 (0.74,0.94)
		2	0.74 (0.65,0.83)	0.83 (0.73,0.93)
		3	0.73 (0.64,0.83)	0.84 (0.75,0.95)
		4	0.73 (0.64,0.83)	0.86 (0.76,0.97)
		5	0.74 (0.65,0.83)	0.86 (0.77,0.97)
		6	0.73 (0.64,0.83)	0.87 (0.78,0.98)
		7	0.74 (0.66,0.83)	0.89 (0.78,0.99)
		8	0.74 (0.65,0.83)	0.88 (0.78,0.99)
		9	0.78 (0.69,0.88)	0.86 (0.76,0.97)
		10	0.78 (0.69,0.88)	0.85 (0.75,0.96)
		11	0.78 (0.69,0.88)	0.84 (0.75,0.95)
		12	0.78 (0.69,0.88)	0.80 (0.71,0.90)
		13	0.76 (0.67,0.85)	0.82 (0.72,0.92)
		14	0.78 (0.69,0.88)	0.84 (0.75,0.95)
	Cold	0	1.05 (0.96,1.15)	0.95 (0.87,1.04)
		1	1.08 (0.99,1.18)	1.01 (0.92,1.09)
		2	1.05 (0.96,1.16)	1.00 (0.91,1.09)
		3	1.05 (0.95,1.15)	1.02 (0.93,1.11)
		4	1.04 (0.95,1.15)	1.04 (0.96,1.13)
		5	1.05 (0.96,1.16)	1.04 (0.97,1.13)
		6	1.04 (0.96,1.14)	1.05 (0.97,1.14)
		7	1.06 (0.97,1.15)	1.07 (0.99,1.16)
		8	1.05 (0.96,1.16)	1.06 (0.98,1.15)
		9	1.12 (1.02,1.22)	1.04 (0.95,1.13)
		10	1.12 (1.02,1.22)	1.03 (0.95,1.13)
		11	1.12 (1.02,1.22)	1.02 (0.93,1.11)
		12	1.12 (1.02,1.22)	0.97 (0.89,1.06)
		13	1.08 (1.00,1.17)	0.99 (0.90,1.08)
		14	1.12 (1.03,1.21)	1.02 (0.93,1.11)
O_3	Warm	0	1.11 (1.03,1.18)	1.03 (0.96,1.11)
		1	1.12 (1.04,1.20)	1.07 (1.00,1.15)

		2	1.11 (1.03,1.18)	1.08 (1.01,1.16)
		3	1.11(1.03,1.18)	1.07 (1.00,1.15)
		4	1.08 (1.01,1.17)	1.07 (1.00,1.15)
		5	1.07 (1.00,1.15)	1.06 (0.99,1.14)
		6	1.08 (1.01,1.16)	1.06 (0.99,1.14)
		7	1.05 (0.98,1.12)	1.05 (0.98,1.13)
		8	1.07 (1.00,1.15)	1.05 (0.98,1.13)
		9	1.08 (1.01,1.16)	1.05 (0.98,1.13)
		10	1.07 (1.00,1.15)	1.06 (0.99,1.14)
		11	1.09 (1.02,1.17)	1.07 (1.00,1.15)
		12	1.09 (1.02,1.17)	1.08 (1.01,1.16)
		13	1.08 (1.01,1.16)	1.07 (1.00,1.15)
		14	1.09 (1.03,1.17)	1.07 (1.00,1.15)
	Cold	0	0.91 (0.85,0.98)	0.93 (0.87,1.00)
		1	0.92 (0.86,0.99)	0.97 (0.90,1.04)
		2	0.92 (0.86,0.99)	0.98 (0.91,1.05)
		3	0.91 (0.85,0.98)	0.98 (0.91,1.05)
		4	0.90 (0.84,0.97)	0.97 (0.90,1.04)
		5	0.90 (0.83,0.96)	0.97 (0.90,1.04)
		6	0.90 (0.83,0.96)	0.96 (0.90,1.03)
		7	0.87 (0.81,0.93)	0.95 (0.90,1.02)
		8	0.90 (0.83,0.96)	0.95 (0.89,1.02)
		9	0.90 (0.83,0.96)	0.96 (0.90,1.03)
		10	0.89 (0.83,0.95)	0.97 (0.90,1.04)
		11	0.91 (0.85,0.98)	0.97 (0.90,1.04)
		12	0.90 (0.84,0.97)	0.98 (0.91,1.05)
		13	0.90 (0.84,0.97)	0.97 (0.90,1.04)
		14	0.91 (0.85,0.98)	0.97 (0.90,1.04)

[a]Models only adjusted for time invariant confounders by design; and ORs were obtained for 10 units increase in each pollutant concentration.
[b]Models adjusted for the other pollutant, temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5,}$ particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.
Note: Bold numbers indicate a significant positive association.

Table 4 outlines the association between air pollution and fetal death stratified by maternal age. In the colder season, a positive association between $PM_{2.5}$ exposure and fetal death were observed in only mothers aged 18-25 years. Conversely, in warmer seasons, the impacts of O_3 exposure were more pronounced in mothers between 26-35 years compared to other age groups.

Table 4: Association between air pollutants and fetal death by maternal age in the SJV, California, 2016 – 2019

Pollutant	Season	Adjusted OR (95% CI)[a]				
		Lag (day)	Less than 18 years (N=27)	Between 18-25 years (N=388)	Between 26-35 years (N=628)	Greater than 35 years (N=293)
$PM_{2.5}$	Warm	0	1.28 (0.75,2.18)	0.83 (0.70,0.98)	0.75 (0.67,0.85)	0.61 (0.50,0.74)
		1	1.23 (0.71,2.14)	0.90 (0.77,1.07)	0.77 (0.68,0.87)	0.62 (0.52,0.76)
		2	1.07 (0.57,1.99)	0.90 (0.76,1.05)	0.76 (0.67,0.86)	0.61 (0.49,0.75)
		3	1.22 (0.70,2.14)	0.90 (0.76,1.05)	0.75 (0.67,0.86)	0.63 (0.52,0.78)
		4	1.36 (0.80,2.30)	0.89 (0.76,1.04)	0.76 (0.67,0.87)	0.66 (0.54,0.80)
		5	1.27 (0.70,2.30)	0.92 (0.79,1.08)	0.76 (0.67,0.86)	0.67 (0.56,0.82)
		6	1.21 (0.68,2.14)	0.92 (0.79,1.08)	0.75 (0.67,0.85)	0.68 (0.56,0.82)
		7	1.20 (0.69,2.08)	0.94 (0.80,1.11)	0.78 (0.70,0.89)	0.68 (0.57,0.83)
		8	1.26 (0.72,2.18)	0.89 (0.75,1.05)	0.78 (0.69,0.89)	0.69 (0.57,0.83)
		9	1.40 (0.83,2.35)	0.89 (0.75,1.04)	0.81 (0.72,0.91)	0.66 (0.54,0.80)
		10	1.44 (0.88,2.35)	0.90 (0.76,1.05)	0.80 (0.71,0.90)	0.67 (0.54,0.81)
		11	1.42 (0.87,2.35)	0.83 (0.69,0.97)	0.83 (0.74,0.93)	0.67 (0.55,0.82)
		12	1.48 (0.91,2.39)	0.83 (0.70,0.97)	0.80 (0.71,0.90)	0.65 (0.54,0.78)
		13	1.36 (0.80,2.28)	0.84 (0.72,1.00)	0.79 (0.71,0.90)	0.64 (0.53,0.78)
		14	1.41 (0.86,2.32)	0.85 (0.72,1.01)	0.82 (0.73,0.92)	0.67 (0.55,0.80)
	Cold	0	0.82 (0.55,1.21)	1.08 (0.96,1.23)	0.96 (0.88,1.05)	0.96 (0.84,1.11)
		1	0.78 (0.52,1.20)	1.18 (1.06,1.33)	0.99 (0.90,1.08)	0.99 (0.87,1.14)
		2	0.69 (0.41,1.14)	1.17 (1.05,1.32)	0.97 (0.89,1.07)	0.97 (0.83,1.13)
		3	0.78 (0.52,1.18)	1.17 (1.05,1.32)	0.96 (0.87,1.06)	1.00 (0.87,1.16)
		4	0.87 (0.61,1.24)	1.17 (1.04,1.31)	0.98 (0.89,1.07)	1.05 (0.91,1.20)
		5	0.81 (0.51,1.28)	1.22 (1.09,1.36)	0.97 (0.88,1.06)	1.07 (0.94,1.22)
		6	0.77 (0.48,1.23)	1.22 (1.09,1.36)	0.97 (0.88,1.06)	1.07 (0.95,1.22)
		7	0.77 (0.49,1.18)	1.23 (1.11,1.38)	1.01 (0.92,1.09)	1.08 (0.96,1.22)
		8	0.80 (0.53,1.21)	1.16 (1.03,1.31)	1.01 (0.92,1.11)	1.08 (0.96,1.22)
		9	0.90 (0.62,1.27)	1.16 (1.03,1.31)	1.04 (0.95,1.13)	1.04 (0.91,1.20)
		10	0.92 (0.69,1.24)	1.17 (1.05,1.32)	1.02 (0.93,1.13)	1.06 (0.92,1.21)
		11	0.91 (0.67,1.23)	1.08 (0.95,1.22)	1.06 (0.97,1.16)	1.06 (0.93,1.21)
		12	0.95 (0.72,1.23)	1.08 (0.97,1.22)	1.02 (0.93,1.12)	1.03 (0.90,1.17)
		13	0.86 (0.59,1.28)	1.11 (0.99,1.24)	1.02 (0.93,1.11)	1.02 (0.90,1.16)
		14	0.90 (0.64,1.27)	1.13 (0.99,1.27)	1.05 (0.96,1.14)	1.05 (0.93,1.20)
O_3	Warm	0	0.90 (0.63,1.28)	1.02 (0.93,1.12)	1.12 (1.04,1.20)	1.02 (0.91,1.14)
		1	0.93 (0.65,1.34)	1.06 (0.97,1.17)	1.13 (1.05,1.21)	1.07 (0.97,1.20)
		2	0.97 (0.67,1.40)	1.07 (0.98,1.17)	1.13 (1.05,1.21)	1.08 (0.97,1.21)

		3	0.93 (0.64,1.36)	1.07 (0.98,1.17)	1.13 (1.05,1.21)	1.07 (0.96,1.20)
		4	0.91 (0.64,1.31)	1.07 (0.97,1.17)	1.13 (1.05,1.21)	1.06 (0.95,1.18)
		5	0.93 (0.66,1.32)	1.05 (0.96,1.15)	1.13 (1.05,1.21)	1.05 (0.94,1.17)
		6	0.92 (0.64,1.34)	1.05 (0.95,1.15)	1.13 (1.05,1.21)	1.05 (0.95,1.17)
		7	0.94 (0.66,1.33)	1.03 (0.94,1.14)	1.09 (1.02,1.18)	1.05 (0.94,1.16)
		8	0.93 (0.65,1.34)	1.04 (0.95,1.15)	1.11 (1.03,1.18)	1.05 (0.94,1.17)
		9	0.90 (0.63,1.31)	1.04 (0.95,1.15)	1.09 (1.02,1.17)	1.06 (0.95,1.18)
		10	0.90 (0.63,1.31)	1.05 (0.96,1.15)	1.09 (1.02,1.18)	1.06 (0.95,1.17)
		11	0.92 (0.64,1.32)	1.07 (0.98,1.18)	1.12 (1.04,1.20)	1.06 (0.95,1.17)
		12	0.92 (0.65,1.31)	1.07 (0.97,1.17)	1.13 (1.05,1.21)	1.06 (0.96,1.18)
		13	0.92 (0.63,1.33)	1.06 (0.97,1.16)	1.12 (1.04,1.20)	1.07 (0.96,1.18)
		14	0.92 (0.64,1.31)	1.06 (0.97,1.16)	1.12 (1.04,1.20)	1.06 (0.96,1.18)
	Cold	0	1.27 (0.90,1.77)	0.90 (0.81,0.98)	0.90 (0.84,0.98)	0.95 (0.85,1.05)
		1	1.32 (0.93,1.86)	0.93 (0.84,1.02)	0.92 (0.85,0.99)	1.00 (0.90,1.12)
		2	1.37 (0.95,1.95)	0.93 (0.85,1.03)	0.92 (0.86,0.99)	1.00 (0.90,1.12)
		3	1.32 (0.91,1.90)	0.93 (0.85,1.02)	0.92 (0.85,0.99)	0.99 (0.90,1.11)
		4	1.29 (0.90,1.83)	0.93 (0.85,1.03)	0.92 (0.85,0.99)	0.98 (0.89,1.09)
		5	1.31 (0.92,1.84)	0.92 (0.83,1.01)	0.92 (0.85,0.99)	0.98 (0.88,1.08)
		6	1.31 (0.90,1.88)	0.91 (0.83,1.00)	0.91 (0.85,0.99)	0.98 (0.88,1.08)
		7	1.32 (0.94,1.86)	0.90 (0.83,0.99)	0.90 (0.83,0.96)	0.97 (0.88,1.08)
		8	1.32 (0.93,1.86)	0.91 (0.83,1.00)	0.90 (0.83,0.97)	0.98 (0.88,1.08)
		9	1.28 (0.90,1.81)	0.91 (0.83,1.00)	0.90 (0.83,0.96)	0.98 (0.89,1.09)
		10	1.28 (0.90,1.81)	0.92 (0.83,1.01)	0.90 (0.83,0.96)	0.98 (0.89,1.09)
		11	1.29 (0.92,1.83)	0.94 (0.85,1.03)	0.90 (0.84,0.98)	0.98 (0.89,1.09)
		12	1.29 (0.91,1.83)	0.93 (0.85,1.03)	0.91 (0.85,0.99)	0.99 (0.90,1.09)
		13	1.29 (0.90,1.88)	0.93 (0.84,1.02)	0.90 (0.84,0.98)	0.99 (0.90,1.11)
		14	1.29 (0.92,1.81)	0.93 (0.84,1.02)	0.91 (0.84,0.98)	0.99 (0.90,1.09)

[a]Models only adjusted for time invariant confounders by design; and ORs were obtained for 10 units increase in each pollutant concentration.
[b]Models adjusted for the other pollutant, temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5,}$ particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.
Note: Bold numbers indicate a significant positive association.

3.3.4. Effect modification analysis for infant death.

Like fetal deaths, the estimates were unstable with wide confidence intervals and the observed associations between air pollutants and maternal race were meaningless for infant deaths due to the limited sample size for some racial/ethnic groups (not shown). We also found no sex-specific differences for infant deaths, but the associations were modified by neighborhood income and maternal age.

Table 5 represents the association between air pollutants and infant death by neighborhood income. $PM_{2.5}$ did not exhibit significant associations with the low-income group but was associated with increased odds of infant death in some lags in the high-income group. In the high-income group, $PM_{2.5}$ exposure in the cold season was positively associated with higher odds of infant death only five (lag 5) and nine (lag 9) days post-exposure. While O_3 exposure in the warm season demonstrated statistically significant associations across all lags in both low and high-income groups, with slightly stronger associations witnessed in the high-income group.

Table 5: Association between air pollutants and infant death by neighborhood income in the SJV, California, 2016 - 2019

Pollutant	Season	Lag (day)	Adjusted OR (95% CI)[a]	
			Low income (<50th percentile) (N=572)	High Income (>=50th percentile) (N=518)
$PM_{2.5}$	Warm	0	0.89 (0.79,0.99)	0.97 (0.85,1.11)
		1	0.88 (0.78,0.99)	0.97 (0.85,1.11)
		2	0.87 (0.78,0.98)	0.96 (0.85,1.09)
		3	0.88 (0.78,0.99)	0.96 (0.84,1.09)
		4	0.84 (0.75,0.95)	0.99 (0.86,1.14)
		5	0.85 (0.75,0.95)	1.01 (0.88,1.15)
		6	0.84 (0.75,0.95)	0.95 (0.83,1.09)
		7	0.81 (0.72,0.91)	0.94 (0.83,1.07)
		8	0.83 (0.74,0.94)	0.96 (0.85,1.09)
		9	0.82 (0.72,0.92)	0.98 (0.87,1.12)
		10	0.83 (0.74,0.95)	0.97 (0.85,1.12)
		11	0.83 (0.74,0.93)	0.94 (0.83,1.09)
		12	0.82 (0.72,0.92)	0.91 (0.79,1.05)
		13	0.84 (0.75,0.94)	0.93 (0.81,1.06)
		14	0.84 (0.75,0.95)	0.94 (0.83,1.07)
	Cold	0	1.03 (0.95,1.13)	1.08 (0.98,1.20)
		1	1.02 (0.94,1.12)	1.07 (0.98,1.18)
		2	1.01 (0.93,1.11)	1.07 (0.98,1.18)
		3	1.02 (0.93,1.13)	1.07 (0.97,1.18)
		4	0.98 (0.90,1.07)	1.09 (0.99,1.22)
		5	0.99 (0.90,1.08)	1.12 (1.01,1.24)
		6	0.98 (0.90,1.07)	1.06 (0.96,1.17)
		7	0.94 (0.85,1.04)	1.04 (0.94,1.16)
		8	0.97 (0.89,1.06)	1.07 (0.98,1.18)
		9	0.95 (0.87,1.05)	1.09 (1.00,1.20)

		10	0.97 (0.89,1.07)	1.08 (0.98,1.20)
		11	0.97 (0.88,1.06)	1.05 (0.94,1.17)
		12	0.95 (0.87,1.04)	1.02 (0.91,1.14)
		13	0.98 (0.90,1.07)	1.03 (0.93,1.15)
		14	0.98 (0.90,1.07)	1.05 (0.95,1.16)
O_3	Warm	0	1.12 (1.04,1.20)	1.13 (1.04,1.23)
		1	1.08 (1.01,1.17)	1.11 (1.02,1.20)
		2	1.08 (1.01,1.17)	1.11 (1.02,1.20)
		3	1.09 (1.01,1.17)	1.11 (1.02,1.20)
		4	1.09 (1.02,1.18)	1.09 (1.00,1.18)
		5	1.08 (1.01,1.17)	1.08 (1.00,1.17)
		6	1.09 (1.02,1.17)	1.09 (1.01,1.18)
		7	1.08 (1.01,1.17)	1.09 (1.01,1.18)
		8	1.08 (1.01,1.17)	1.09 (1.01,1.18)
		9	1.09 (1.01,1.17)	1.08 (1.01,1.17)
		10	1.08 (1.01,1.16)	1.09 (1.01,1.18)
		11	1.08 (1.01,1.16)	1.11 (1.02,1.20)
		12	1.08 (1.01,1.17)	1.11 (1.02,1.20)
		13	1.09 (1.02,1.17)	1.09 (1.01,1.20)
		14	1.09 (1.02,1.17)	1.11 (1.02,1.20)
	Cold	0	0.96 (0.90,1.04)	0.97 (0.90,1.05)
		1	0.94 (0.87,1.01)	0.94 (0.87,1.02)
		2	0.94 (0.87,1.01)	0.94 (0.87,1.02)
		3	0.94 (0.88,1.02)	0.95 (0.88,1.03)
		4	0.95 (0.88,1.02)	0.93 (0.86,1.01)
		5	0.94 (0.87,1.02)	0.93 (0.86,1.01)
		6	0.94 (0.88,1.02)	0.94 (0.87,1.02)
		7	0.94 (0.87,1.02)	0.94 (0.87,1.02)
		8	0.94 (0.87,1.01)	0.94 (0.87,1.02)
		9	0.94 (0.87,1.02)	0.93 (0.86,1.01)
		10	0.94 (0.87,1.01)	0.94 (0.87,1.02)
		11	0.93 (0.87,1.01)	0.95 (0.87,1.03)
		12	0.94 (0.87,1.01)	0.95 (0.88,1.03)
		13	0.94 (0.88,1.02)	0.94 (0.87,1.02)
		14	0.95 (0.88,1.02)	0.94 (0.87,1.02)

[a]Models only adjusted for time invariant confounders by design; and ORs were obtained for 10 units increase in each pollutant concentration.
[b]Models adjusted for the other pollutant, temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5,}$ particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.
Note: Bold numbers indicate a significant positive association.

Table 6 represents the association between air pollutants and infant death by maternal age. O_3 exposure demonstrated increased odds of infant death among mothers aged 18-25 and 26-35 years compared to those under 18 years, with particularly pronounced associations observed in the age group between 18-25 years. These associations were statistically significant across all lags in these two age groups. However, $PM_{2.5}$ exposure did not exhibit any significant associations with infant death across maternal age groups.

Table 6: Association between air pollutants and infant death by maternal age in the SJV, California, 2016 – 2019

Pollutant	Season	Lag (day)	Adjusted OR (95% CI)[a]			
			Less than 18 years (N=17)	Between 18-25 years (N=409)	Between 26-35 years (N=478)	Greater than 35 years (N=192)
$PM_{2.5}$	Warm	0	0.48 (0.16,1.42)	0.98 (0.84,1.13)	0.88 (0.77,0.99)	0.96 (0.79,1.18)
		1	0.68 (0.24,1.95)	0.94 (0.81,1.08)	0.87 (0.77,0.99)	0.99 (0.82,1.20)
		2	0.53 (0.18,1.58)	0.94 (0.81,1.11)	0.88 (0.78,0.99)	0.95 (0.79,1.16)
		3	0.54 (0.20,1.50)	0.97 (0.83,1.13)	0.84 (0.73,0.96)	0.99 (0.80,1.21)
		4	0.51 (0.18,1.41)	0.93 (0.81,1.08)	0.83 (0.72,0.94)	1.00 (0.82,1.23)
		5	0.51 (0.19,1.37)	0.94 (0.81,1.09)	0.84 (0.74,0.96)	1.02 (0.83,1.24)
		6	0.55 (0.20,1.57)	0.93 (0.81,1.08)	0.81 (0.71,0.93)	0.97 (0.79,1.20)
		7	0.57 (0.20,1.60)	0.90 (0.77,1.04)	0.83 (0.72,0.94)	0.92 (0.75,1.13)
		8	0.61 (0.21,1.74)	0.90 (0.78,1.05)	0.83 (0.73,0.96)	0.98 (0.80,1.18)
		9	0.60 (0.20,1.76)	0.87 (0.74,1.02)	0.85 (0.75,0.96)	1.00 (0.83,1.21)
		10	0.49 (0.17,1.40)	0.91 (0.78,1.06)	0.86 (0.75,0.98)	0.97 (0.78,1.20)
		11	0.40 (0.13,1.29)	0.90 (0.78,1.05)	0.83 (0.73,0.95)	0.95 (0.77,1.17)
		12	0.44 (0.15,1.24)	0.89 (0.76,1.04)	0.82 (0.72,0.94)	0.90 (0.73,1.13)
		13	0.46 (0.15,1.40)	0.93 (0.81,1.07)	0.81 (0.71,0.93)	0.93 (0.75,1.16)
		14	0.50 (0.17,1.48)	0.93 (0.80,1.08)	0.82 (0.72,0.93)	0.95 (0.78,1.16)
	Cold	0	1.02 (0.52,1.99)	1.09 (0.99,1.22)	1.03 (0.93,1.14)	1.04 (0.91,1.20)
		1	1.42 (0.76,2.67)	1.05 (0.95,1.17)	1.02 (0.92,1.14)	1.07 (0.95,1.21)
		2	1.12 (0.56,2.24)	1.06 (0.94,1.20)	1.03 (0.93,1.14)	1.03 (0.91,1.17)
		3	1.15 (0.72,1.83)	1.08 (0.97,1.22)	0.99 (0.89,1.11)	1.07 (0.92,1.23)
		4	1.07 (0.69,1.68)	1.05 (0.94,1.17)	0.97 (0.87,1.08)	1.08 (0.94,1.26)
		5	1.06 (0.69,1.63)	1.06 (0.95,1.18)	0.99 (0.90,1.09)	1.11 (0.97,1.26)
		6	1.16 (0.68,2.00)	1.05 (0.95,1.16)	0.95 (0.85,1.06)	1.05 (0.90,1.22)

		7	1.20 (0.69,2.06)	1.00 (0.90,1.13)	0.97 (0.87,1.08)	1.00 (0.87,1.15)
		8	1.27 (0.71,2.28)	1.02 (0.90,1.14)	0.98 (0.88,1.09)	1.06 (0.93,1.20)
		9	1.26 (0.66,2.43)	0.98 (0.86,1.12)	1.00 (0.90,1.11)	1.08 (0.97,1.22)
		10	1.03 (0.59,1.77)	1.02 (0.90,1.16)	1.01 (0.90,1.13)	1.05 (0.90,1.22)
		11	0.85 (0.41,1.77)	1.01 (0.90,1.14)	0.98 (0.88,1.09)	1.03 (0.88,1.20)
		12	0.92 (0.52,1.64)	1.00 (0.89,1.13)	0.96 (0.87,1.07)	0.98 (0.84,1.15)
		13	0.96 (0.48,1.95)	1.04 (0.94,1.16)	0.95 (0.85,1.07)	1.01 (0.86,1.18)
		14	1.06 (0.56,2.00)	1.04 (0.93,1.16)	0.96 (0.86,1.07)	1.03 (0.90,1.17)
O_3	Warm	0	1.38 (0.85,2.24)	1.15 (1.05,1.24)	1.12 (1.02,1.21)	1.07 (0.94,1.22)
		1	1.28 (0.78,2.08)	1.12 (1.02,1.22)	1.09 (1.00,1.18)	1.03 (0.90,1.16)
		2	1.34 (0.79,2.26)	1.12 (1.02,1.21)	1.09 (1.01,1.18)	1.03 (0.90,1.17)
		3	1.29 (0.78,2.14)	1.12 (1.02,1.21)	1.11 (1.01,1.20)	1.04 (0.91,1.18)
		4	1.32 (0.81,2.14)	1.11 (1.02,1.21)	1.11 (1.02,1.20)	1.02 (0.90,1.16)
		5	1.32 (0.80,2.18)	1.11 (1.01,1.21)	1.09 (1.01,1.18)	1.01 (0.90,1.15)
		6	1.31 (0.80,2.12)	1.11 (1.01,1.21)	1.11 (1.01,1.20)	1.03 (0.90,1.17)
		7	1.29 (0.80,2.10)	1.12 (1.02,1.22)	1.09 (1.01,1.18)	1.03 (0.90,1.16)
		8	1.28 (0.78,2.12)	1.12 (1.02,1.22)	1.09 (1.01,1.18)	1.02 (0.90,1.16)
		9	1.28 (0.75,2.18)	1.13 (1.03,1.22)	1.08 (1.00,1.17)	1.02 (0.90,1.16)
		10	1.28 (0.77,2.14)	1.12 (1.03,1.22)	1.08 (1.00,1.17)	1.03 (0.90,1.17)
		11	1.37 (0.82,2.28)	1.12 (1.02,1.22)	1.09 (1.01,1.18)	1.04 (0.91,1.18)
		12	1.38 (0.83,2.30)	1.12 (1.02,1.22)	1.09 (1.01,1.18)	1.05 (0.92,1.18)
		13	1.38 (0.84,2.24)	1.11 (1.01,1.21)	1.09 (1.01,1.20)	1.04 (0.91,1.18)
		14	1.36 (0.84,2.18)	1.12 (1.02,1.21)	1.11 (1.01,1.20)	1.04 (0.92,1.18)
	Cold	0	0.68 (0.44,1.03)	0.97 (0.88,1.06)	0.98 (0.90,1.07)	0.91 (0.80,1.04)
		1	0.62 (0.39,0.99)	0.94 (0.86,1.03)	0.96 (0.89,1.05)	0.88 (0.77,1.00)
		2	0.66 (0.43,1.02)	0.94 (0.86,1.03)	0.96 (0.89,1.05)	0.88 (0.78,1.00)
		3	0.64 (0.41,0.98)	0.94 (0.86,1.03)	0.97 (0.90,1.06)	0.89 (0.78,1.01)
		4	0.64 (0.43,0.98)	0.93 (0.85,1.02)	0.98 (0.90,1.06)	0.87 (0.76,0.99)
		5	0.65 (0.43,0.99)	0.93 (0.85,1.02)	0.97 (0.89,1.05)	0.87 (0.76,0.99)
		6	0.64 (0.42,0.98)	0.93 (0.85,1.03)	0.97 (0.90,1.06)	0.88 (0.77,1.00)
		7	0.64 (0.42,0.97)	0.94 (0.86,1.03)	0.97 (0.89,1.05)	0.88 (0.77,1.00)
		8	0.62 (0.39,1.00)	0.94 (0.86,1.03)	0.97 (0.89,1.05)	0.87 (0.77,0.99)
		9	0.62 (0.40,0.99)	0.94 (0.87,1.03)	0.96 (0.88,1.04)	0.88 (0.77,0.99)
		10	0.63 (0.40,0.99)	0.94 (0.86,1.03)	0.96 (0.89,1.04)	0.88 (0.77,1.00)
		11	0.67 (0.43,1.04)	0.94 (0.86,1.03)	0.97 (0.89,1.05)	0.89 (0.78,1.01)
		12	0.68 (0.44,1.04)	0.94 (0.86,1.03)	0.96 (0.89,1.05)	0.90 (0.78,1.02)
		13	0.68 (0.44,1.03)	0.93 (0.85,1.02)	0.97 (0.9,1.05)	0.89 (0.78,1.01)
		14	0.67 (0.44,1.00)	0.94 (0.86,1.03)	0.97 (0.9,1.06)	0.89 (0.78,1.01)

[a]Models only adjusted for time invariant confounders by design; and ORs were obtained for 10 units increase in each pollutant concentration.

[b]Models adjusted for the other pollutant, temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5,}$ particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.
Note: Bold numbers indicate a significant positive association.

3.4.Additional analysis

3.4.1. Whole-year associations between air pollutants and fetal and infant death

Table S8 represents a whole- year analysis that generally suggests a slight but insignificant increase in odds of fetal and infant death. However, there is no evidence of association in the whole- year analysis, confirming that seasonal difference is pronounced.

3.4.2. Single pollutant model for fetal death and infant death

In **Table S9**, the single pollutant model depicts the relationship between air pollutants and fetal death. The findings indicate that there is no significant association between fetal death and O_3 exposure. However, a positive association was observed with $PM_{2.5}$ exposure, particularly at lag 7 ($aORlag_7$: 1.06 (95% CI: 1.01, 1.13) and lag 14 ($aORlag_{14}$: 1.05 (95% CI: 1.00, 1.12).

Table S10 represents the association between air pollutants and infant death in a single pollutant model. The findings suggest no evidence of significant association with $PM_{2.5}$ exposure. However, a notable significant association was observed with O_3 exposure across all lags except lag 9 and lag 12.

4. Discussion

4.1.Principal findings

This case-crossover study investigated the acute associations between exposure to air pollutants, specifically $PM_{2.5}$ and O_3, and the risk of fetal and infant death in the SJV. Our findings indicate that higher $PM_{2.5}$ levels in the cold season and higher O_3 levels in the warm season were associated with increased odds of both fetal and infant deaths. The associations with O_3 were notably more consistent and pronounced for both outcomes compared to those of $PM_{2.5}$. Our study also found that individuals in lower-income neighborhoods might be more susceptible to the detrimental effects of air pollution on fetal and infant death compared to those in higher-income neighborhoods.

Given the SJV has not achieved compliance with the National Ambient Air Quality Standards (NAAQS) for $PM_{2.5}$ and O_3 and the diverse population in this region, these findings support that air pollution may be contributing to the perinatal health burden in the region and efforts to reduce exposures are critical.

4.1.1. Interpretation for fetal death

Our findings, aligning with existing research, confirm that $PM_{2.5}$ exposure during pregnancy is linked to an increased risk of fetal death (DeFranco et al., 2015; Rochelle

Green et al., 2015; Liang et al., 2021; Siddika et al., 2016; Tan et al., 2023; Tong et al., 2022; Wainstock et al., 2021; Wang et al., 2023; Shaoping Yang et al., 2018). In a retrospective cohort study spanning from 1999 to 2009 in California, Green et al. observed a positive association between $PM_{2.5}$ exposure and increased risk of stillbirth throughout the pregnancy (R. Green et al., 2015). The study by Green et al., involved comparing the risk of fetal death between two distinct groups of women, one with high exposure levels and the other with low exposure levels. As such, this design seeks to explain why some women experienced fetal death while others did not. Despite our different approach using a case-crossover analysis to address the question of why death occurs at one time but not another, it is reassuring to observe the consistency of findings.

Additional research conducted in China further confirms the positive relationship between $PM_{2.5}$ exposure and fetal death. For instance, a recent cohort study carried out in Wuhan, China, between 2011 and 2017, identified a positive link between exposure to $PM_{2.5}$ and fetal death (Tan et al., 2023), a prospective cohort study conducted in Wuhan, China, from June 10, 2011, to June 9, 2013, and analyzed using logistic regression, found a steady increase in stillbirth risk associated with high $PM_{2.5}$ exposure as pregnancies advanced (S. Yang et al., 2018), a population-based prospective cohort study conducted in China revealed that for every increase of 10 $\mu g/m^3$ in $PM_{2.5}$ levels, there was a corresponding rise in the risk of stillbirths (Zang et al., 2019), and another retrospective cohort study in seven Guangdong cities in China conducted using a cox proportional hazards model also found a positive link between $PM_{2.5}$ exposure and stillbirths (Liang et al., 2021). This convergence of results despite using different study designs and statistical methods of analysis reinforces the validity of our findings and the broader implications of air pollution on fetal health.

Meanwhile, two US studies, both employed a time-stratified case-crossover study design, showed mixed results. The California study (1999-2009) found no significant link (Sarovar et al., 2020) between $PM_{2.5}$ and fetal death, while the New Jersey study (1998-2004) noted increased risk, although the estimates were not statistically significant (Faiz et al., 2013). In contrast, our study looked at a broader span of lag days (0-14) and identified increased risk across most lags and notable significant associations particularly in the later lag periods. Despite using similar study designs as ours, the discrepancies in findings could be attributed to different ranges of lag days, measurement units for $PM_{2.5}$ (IQR vs. $\mu g/m^3$), season-specific stratification for the pollutants and exposure assessments limited to 10 km from maternal residences in the other studies. Our study measured the mean concentration of $PM_{2.5}$ in mg/m^3, as opposed to the Interquartile Range (IQR) used in these two studies. Furthermore, our study is based on more recent data (2016-2019), unlike the earlier timeframes of the other two studies. In addition, it is important to pinpoint that our whole year analysis (without season-specific stratification) did not find any significant associations like these studies. Hence, given the limited research on acute exposure, further studies focusing on short-term air pollution exposure are essential which would enhance our understanding of the impact of short-term exposure and help pinpoint the critical periods of exposure just before death. Three comprehensive systematic reviews and meta-analyses, incorporating a total of 67 cohort studies from 20 nations (Wang et al., 2023), 15 epidemiological studies across 6 nations

(Zhang et al., 2021), and 13 studies assessing the short- and long-term impacts of air pollution (Siddika et al., 2016) including $PM_{2.5}$ exposure, collectively indicate an increased risk of fetal death associated with exposure to $PM_{2.5}$.

Contrary to our hypothesis, we also observed a protective effect of $PM_{2.5}$ during the warmer seasons, a period typically characterized by lower atmospheric $PM_{2.5}$ concentrations. This finding contrasts with a study conducted in China, which reported a protective effect against intrauterine fetal death at higher $PM_{2.5}$ levels, particularly when the exposure period extended beyond eight months. This discrepancy highlights the complexity of the impact of $PM_{2.5}$ impact on fetal deaths across different environmental conditions and time frames (Yan et al., 2023). However, the current literature underscores the necessity for further research investigating the relationship between $PM_{2.5}$ exposure and fetal death, given the limited scope of existing studies and the observed inconsistencies, particularly concerning the identification of critical exposure windows.

Together, findings suggest $PM_{2.5}$ might increase fetal death burden. Although the biological mechanisms are not clear, studies have shown that fine particles can potentially cross the uteroplacental barrier, cause irreversible, hypoxic, or immune-related damage, potentially affecting the growth and development of the fetus and may also cause fetal death. The potential health implications of air pollution may be attributed to several biological mechanisms, such as systemic inflammation (Lanki et al., 2015; Viehmann et al., 2015), oxidative stress (W. Li et al., 2016; Patel, 2016; Patel et al., 2013), spontaneous premature rupture of membrane and endocrine disruption (Jiao et al., 2023; Slama et al., 2008).

Our findings regarding the association between O_3 and fetal death are also consistent with the literature. A nationwide US study by Mendola et al. reported a significant positive association for both chronic (i.e. first trimester and whole pregnancy) and acute exposures (Mendola et al., 2017). For acute exposures, Mendola et al., reported an 8% increased risk of stillbirth ≥23 weeks of gestation corresponding to a 10 ppb rise in O_3 concentrations, two days before the event (Mendola et al., 2017). For the other days preceding the event, the increment in risk tied to a 10-ppb increment O_3 concentrations ranged between 7% and 12%. Similarly, our study found the strongest association with O_3 exposure occurring two days before the event with similar effect estimates. We note that the prior study did not estimate season specific effects of O_3 while we only observed O_3 effects during warm season when O3 levels are high in the SJV. A case-crossover study in California by Sarovar et al., also found a significant association between exposure to maximum 1-hour O_3 and the risk of fetal death at lag 4 (Sarovar et al., 2020). More specifically, they found that an estimate similar to ours. Earlier studies have shown that there is typically a 48-hour interval between the occurrence of fetal death and the subsequent delivery. However, we did not implement this approach in our study and used the reported fetal death day as the case day. Although both our study and that of Sarovar et al. employed the same study design and were conducted in the same geographical region, the above listed variations might have yielded a more consistent insight into the effects of O_3 in our study compared to the study by Sarovar et al. Similarly, a retrospective cohort study conducted (January, 2008- December, 2013) in Harris County, Texas, an urban area with nonattainment status, reported a 9% rise in the risk of fetal

death for every 3.6 ppb IQR increase in O_3 exposure using a survival analysis model (Rammah et al., 2019). This study involved comparing the risk of fetal death (N=1,874) between two distinct groups of women, one with high exposure levels and the other with low exposure levels while our case-crossover study examined when the event occurred with case and controls being the same individual. It is crucial to recognize that Harris County in Texas, much like our study area, the SJV in California, is classified as a nonattainment area. However, a notable distinction exists between the two: Harris County is an urban area, whereas SJV is predominantly rural and underserved. The consistency of our results with Rammah et al., despite the variations in the study design, statistical analysis model, sample size and geographical location provides reassurance in the validity of our findings. Another retrospective cohort study in California, employing logistic regression models, identified a significant association between O_3 exposure and stillbirth during the third trimester. However, it found null associations in the first two trimesters or across the entire pregnancy (Rochelle Green et al., 2015).

Despite consistency with prior literature, we also note that our findings are appreciably different from a few existing studies (Hwang et al., 2011; Rammah et al., 2019; Shaoping Yang et al., 2018). A case-crossover study involving 1,599 cases of stillbirth in Harris County, Texas, found no evidence of association between short-term exposure to O_3 and the risk of fetal death within one week (Rammah et al., 2019). The discrepancy could be due to the fact that the Texas study did not consider season specific O_3 effects. Year-round analyses could show null effects similar to what we observed but may ignore important effects when O3 levels are higher. In a comprehensive, population-based case-control study conducted in Taiwan, Hwang et al. also discovered no evidence of a link between ozone exposure and stillbirth across all pregnancy trimesters, as well as the entire duration of gestation (Hwang et al., 2011).

The plausibility for the inverse associations observed between ozone and fetal death in the cold season is unclear. It has also been observed in the literature. In a time series analysis conducted by Dastoorpoor et al. in Iran, the impact of O_3 exposure on stillbirth risk was investigated using a Poisson regression model (Dastoorpoor et al., 2018). The study revealed a protective relationship between O_3 exposure and the risk of stillbirth while accounting for seasonality, ambient temperature and relative humidity. We suspect that the inverse associations may be because of the lack of data for other pollutants, but this remains a speculation.

These discrepancies in findings may exist due to variations in exposure assessment, level of pollutant concentration, study design, and methodologies used in prior studies. For instance, studies conducted by Faiz et al., and Rammah et al., restricted exposure assessments to readings from the closest monitor based on maternal residence (Faiz et al., 2012; Rammah et al., 2019).

Susceptible population

Our results indicate that neighborhood income level and maternal age may modify the effects of $PM_{2.5}$ and O_3 exposure. We observed a notable increase in odds of fetal death among both low- and high-income neighborhoods exposed to both pollutants, but the associations were stronger for low-income neighborhoods. This disparity is likely due

to mothers from lower socioeconomic backgrounds facing not only a higher number of pollution sources but also having fewer resources to mitigate these effects and experiencing additional stress factors (American Lung Association, 2023a). Of note, we also observed that the associations were higher among Asian and Hawaiian/PI, although confidence intervals were wide due to low sample size. In existing studies exploring birth outcomes in relation to air pollution, researchers have highlighted variations in associations with air pollution across demographic indicators (Green et al., 2009; Tong et al., 2022; Wainstock et al., 2021). This finding indicates that race may play a significant role as a modifying factor in the effects of air pollution on pregnant women. Our data also suggests a similar pattern with racial minorities, but the limited sample size does not allow a comprehensive interpretation of these observed relationships among different races/ethnicities considered in our study. Nevertheless, it is essential to acknowledge that socially disadvantaged racial minorities often bear elevated risk of adverse birth outcomes (Basu et al., 2016; Lorch et al., 2012), which presents a pressing need for more in-depth, robust research to elucidate these associations effectively.

While, some studies indicated no significant effect modification by maternal age (Faiz et al., 2013), some suggest that both younger (<25 years) and older mothers (>35 years) exposed to air pollution have an elevated risk of fetal death (R. Green et al., 2015) compared to those in the middle of reproductive age years. However, our study found higher risk of fetal death with exposure to the air pollutants among mothers between 18 – 35 years of age compared to mothers below 18 years. These age disparities may warrant further investigation. But it is important to highlight that the sample size for mothers under 18 years was too low (N=27) compared to other age-groups in our study.

4.1.2. Interpretation for infant death

Our study revealed an increased odds of infant death associated with higher O_3 levels, with both immediate and delayed effects. These findings are consistent with a few other studies which demonstrated a strong positive association with O_3 exposure (Gouveia et al., 2018; Hajat et al., 2007; Woodruff et al., 2008a; Yang et al., 2006). A retrospective cohort study conducted by Woodruff et al., reported that O_3 exposure during the first two months of a child's life was associated with a 20% (OR 1.20; 95% CI, 1.09-1.32) increased risk of sudden infant death syndrome. These findings align with our observation and further substantiate that the critical period for exposure to O_3 is indeed during early life. Meanwhile, in Taiwan, a case-crossover study reported an increased risk of post-neonatal deaths (i.e., infants older than 27 days but less than 365 days) with O_3 exposure but no evidence of significant association (Yang et al., 2006). The lack of significant impact of O_3 exposure on infant deaths in Taipei could be due to the low average O_3 concentration of 18.14 ppb, potentially too low for a measurable effect. Moreover, Taipei's moderate subtropical climate, with an average temperature around 22.92°C (73.5°F), may further mitigate the potential effects of O_3 exposure. This is supported by research indicating that milder temperatures in subtropical regions can reduce the respiratory stress and health risks associated with O_3 pollution, unlike in hotter climates where these effects may be amplified. Additionally, the limited number of

postneonatal deaths (N=471) might have resulted in inadequate statistical power to identify an association in this study. In contrast, our study, undertaken in the SJV, a rural setting characterized by elevated O_3 levels, particularly in the warmer season, recorded an average O_3 concentration of 57.5 ppb during the study period (2016-2019). The variation of concentration of O_3 in the atmosphere due to differences in geographical region and climatic conditions may have contributed to a more pronounced and significant influence of O_3 exposure on infant's health in our study. It is also important to note that in Taipei, the main cause of air pollution is emissions from vehicles, while in the SJV, air quality is influenced by a combination of factors including agricultural activities, wildfires, industrial emissions, and vehicle exhaust (Veloz et al., 2020). In addition, a review conducted among 27 articles did not provide any evidence of an association between O_3 exposure and the risk of infant death (Karimi & Shokrinezhad, 2020). A recent systematic review and meta-analysis, incorporating 22 studies focused on the short-term impacts of air pollution on infant death, identified a negative association between infant deaths and O_3 exposure. Out of these, 13 studies provided effect estimates, resulting in a combined OR of 0.99 (95% CI: 0.97, 1.01) for infant death with O_3 exposure (Luben et al., 2023). This study also indicated a lack of a significant connection between O_3 exposure and infant death, with variations in the strength of association and critical period of exposure which is likely due to limited body of research in this domain. Further investigation into the relationship between O_3 exposure and infant death is essential to clarify the true nature of this association. The current research presents conflicting results regarding the relationship between O_3 exposure and infant death. Such inconsistencies might result from differences in study designs, methodologies, demographics, geographical locations, and periods studied. Another factor contributing to the differences observed in studies might be consideration of how respiratory infections affect infants, with higher deaths seen in infants with these infections. Notably, a significant link was found between lower respiratory infections and infant death (Gouveia et al., 2018). This study revealed that the impact of O_3 varies seasonally, being more pronounced in the warm season for respiratory issues in infants under one year, as opposed to children over one year old. These findings reinforce the idea that infants are particularly vulnerable to the effects of O_3 in warmer seasons, when O_3 levels are typically higher in the atmosphere (Gallacher et al., 2016). In summary, the inconsistent findings highlight the need for further in-depth research to elucidate the link between O_3 exposure and infant death, considering these varied factors since infants are a vulnerable population with unique health risks. In addition, research on infant death and O_3 exposure is also limited as most studies have focused on adults (Anderson et al., 1996; Chen et al., 2023; Chen et al., 2018; C. Li et al., 2016; Yap et al., 2019). This disparity may stem from factors such as the greater availability of data on adult populations, or the perception that adults are more likely to experience adverse health effects from air pollution.

We did not find any significant association between exposure to $PM_{2.5}$ and infant deaths. This finding aligns with other research that explored the impact of total suspended particulates on infant death (Chay & Greenstone, 2003) and PM (Chen et al., 2021) on SIDS. It is also important to note that many studies have reported positive associations with particulate matter and infant death (Bobak & Leon, 1992; Goyal et al., 2019; Ha et al., 2003; Heft-Neal et al., 2018; Loomis et al., 1999; Woodruff et al., 1997; Yorifuji et

al., 2016). Studies examining the connection between exposure to PM and infant death have varied, not just in terms of the strength of the association but also regarding the critical periods of exposure. While studies by Loomis and others suggested a correlation between increased odds of infant death with $PM_{2.5}$ exposure shortly before death i.e. on the same day and four days post-exposure (Loomis et al., 1999; Yorifuji et al., 2016), Carbajal-Arroyo et al. indicated higher postneonatal death to immediate (i.e. same day) and brief exposures (i.e. cumulative lags for lag 0 – lag 2) to larger PM_{10} particles (Carbajal-Arroyo et al., 2011). Contrarily, findings from Belgium and the United Kingdom showed that while some pollutants like sulfur dioxide were associated with infant death, PM_{10} was not (Hajat et al., 2007; Scheers et al., 2011). Additional research from Korea supported the notion of a risk increase with same-day PM_{10} exposure (Ha et al., 2003). A study in Beijing, China, investigating monthly $PM_{2.5}$ exposure through generalized additive Poisson regression, indicated increased odds of infant death (aOR 1.548, 95% CI 1.06, 2.25), after accounting for concurrent PM_{10} exposure (Wang et al., 2019). Similarly, another research in China, employing a time-stratified case-crossover design, identified a higher risk of neonatal deaths associated with $PM_{2.5}$ exposure, particularly during the warmer seasons (April- September), although these findings did not reach statistical significance (He et al., 2022). Despite biologic plausibility, our study does not support a positive link between $PM_{2.5}$ exposure and infant mortality. In the meantime, a systematic review encompassing 14 studies also found little to no evidence supporting a link between exposure to PM and infant death (Glinianaia et al., 2004).

Susceptible population

We observed that O_3 exposure significantly affected infants in both low and high-income neighborhoods, with marginally stronger effects in high-income areas during immediate lag periods. Conversely, $PM_{2.5}$ had no significant association with infant death in low-income neighborhoods but was linked to higher odds of infant deaths from high-income areas at lag 5 and lag 10. Predominantly research suggests that individuals residing in low-income neighborhoods bear the greatest burden of the impact of air pollution (Gwynn & Thurston, 2001; Jbaily et al., 2022; Woodruff et al., 2003; Yazdi et al., 2021). In addition, socially disadvantaged individuals often face higher risks of experiencing adverse birth outcomes due to air pollution or other chemical toxins (Giscombé & Lobel, 2005; O'Campo et al., 2007; Yazdi et al., 2021). However, our findings underscore the pervasive impact of air pollution on infant health, irrespective of the socioeconomic status of their neighborhood. We also note that because the SJV generally has lower SES compared to the rest of the state, a high-SES area in our study may still have low SES based on state standards. Nevertheless, the fact that air pollution-related fetal deaths predominantly occur in low SES populations suggests that socio-economic conditions heighten the risks to pregnant women and unborn children, potentially due to factors like nutrition, stress, healthcare access, and exposure to additional environmental risks. We note that these factors are not likely confounders in our study due to the self-match nature of the study design.

Our results also emphasize that the link between O_3 exposure and infant death risk is significantly affected by the age of the mother, showing a more marked impact among mothers aged 18 to 35 years compared to those younger than 18 years. However, due to a

limited number of samples from mothers under 18 years old (N=17), we cannot draw definitive conclusions, making this finding uncertain in our study.

Our study also observed a pronounced effect of air pollution on racial minority groups, specifically Asian and Hawaiian/Pacific Islander populations. However, the limited sample size and broad confidence intervals preclude definitive conclusions. Despite these constraints, our findings underscore substantial health equity concerns, as they suggest a stronger positive association between exposure to air pollution and an increased risk of infant death within these communities. While Asian is the fastest-growing population in the U.S. (Budiman & Runiz, 2021), the multitude of health issues faced by both Asian and Hawaiian/Pacific Islander (ADDA; Renehan, 2022) communities are frequently overlooked. In fact, these populations face higher disparities in exposure to air pollution (Jbaily et al., 2022). The adverse health implications of these exposures contribute to a range of birth outcomes including preterm birth, low birth weight (Fleischer et al., 2014), term low-birth weight (Slama et al., 2007), birth defects (Padula et al., 2013), fetal distress (H. Liu et al., 2019) and pregnancy loss (Ha, Ghimire, et al., 2022). The findings from our study can also shed light on environmental justice issues, as underprivileged communities frequently experience disproportionate exposure to elevated air pollution levels (Liu et al., 2018).

Moreover, the exact ways in which air pollution impacts the deaths of infants remain partly unknown; however, the combination of environmental factors and epigenetic changes plays a crucial role. During the early years of life, an infant's immune system and lungs are not fully developed, making them more susceptible to the harmful effects of air pollution than adults (Gouveia et al., 2018), due to their higher risk of respiratory diseases, underdeveloped metabolic processes, and weaker natural defenses (Glinianaia et al., 2004; Ha et al., 2003). Furthermore, the detrimental effects of air pollution on health, particularly through oxidative stress and inflammation in the cardiovascular and respiratory systems, play a major role in contributing to infant death (Kannan et al., 2006). Further investigations into the association between air pollution, and fetal and infant death can deepen our knowledge of the involved biological processes, aiding healthcare practitioners in improving prenatal and postnatal care and advising pregnant mothers to reduce exposure to harmful air pollutants. Identification of the most detrimental air pollutants to pregnant women, fetuses, and infants is crucial for devising effective mitigation strategies to protect these populations.

4.1.3. Inverse association

Despite positive associations between air pollutants and perinatal deaths, we also found some unexpected protective associations. Notably, these protective effects were observed during the warm season for $PM_{2.5}$ exposure and during the cold season for O_3 exposure. These are windows during which the pollutants had the lowest concentrations. The underlying mechanisms for these observations remain elusive but a few other studies reported similar findings (DeFranco et al., 2015; Smith et al., 2020). For example, DeFranco et al. found a protective effect against fetal death from high air pollution in the first trimester, in contrast to the increased risks in later pregnancy stages (DeFranco et al., 2015). DeFranco et al. suggest this could be due to their study's focus on pregnancies

beyond 20 weeks, potentially missing early losses to pollution, thus inaccurately indicating a protective effect. Another study by Smith et al. also found similar protective effects between exposure to air pollution and stillbirth risk at different trimesters of pregnancies (Smith et al., 2020). Smith et al. noted that due to the wide range of tests conducted on different pollutants and stages of pregnancy, some findings might be coincidental in their study. They also suggested that higher pollution areas might have enhanced healthcare strategies to mitigate risks and that their method of assessing pollution exposure may not accurately reflect personal behaviors or mobility.

4.2.Strength and limitations

This research holds significance as it is conducted in the underserved SJV region, which is well-known for its non-attainment status concerning $PM_{2.5}$ and O_3. It represents the first study in this area that specifically investigates the connection between short-term exposure to air pollution and fetal and infant death. This study differs from previous research conducted in the region, which primarily focused on exploring the relationship between air pollution and the likelihood of preterm birth, low birth weight, and birth defects (Ha, Martinez, et al., 2022; Padula et al., 2014; Padula et al., 2015). There are some other important strengths of this study. Firstly, the self-matching aspect provides full control over time in variant confounders, such as genetics, demographics, and underlying health conditions. Secondly, the time-stratified control selection approach effectively minimizes seasonal and time-trend biases. In addition, the case-crossover analysis aims to determine why an event occurs at a particular moment rather than another time for the same individual. Moreover, numerous studies have concentrated on investigating the lag effects of air pollution within a one-week timeframe due to the observable immediate impacts. However, this narrow focus may have inadvertently neglected any prolonged lag effects that could be present. Our study broadened the scope of lag effects analysis to encompass two weeks. This expanded evaluation allowed us to identify and examine extended lag effects.

On the other hand, our research had certain constraints that should be considered. First, exposures were inferred from modeled data and connected to mothers' zip codes, which may not provide the most accurate representation of residential locations, particularly in areas where zip codes cover vast areas. Nevertheless, studies have demonstrated that using zip codes for exposure estimation can be adequate (Soret et al., 2006). Second of the lack of data on some co-pollutants such as NO_2 and SO_2 hinders a comprehensive understanding of air pollution dynamics in each area. It is essential to consider the intricate combination of pollutants and their potential synergistic or antagonistic interactions to ensure an accurate analysis. Without such data, our understanding of the complex nature of air pollution in these areas remains incomplete. While we did not notice a significant impact of confounding from pollutants in our study, it remains possible that an unexplored pollutant, which is strongly linked to both the pollutants and these adverse birth outcomes, could still introduce confounding. Third, our study lacks personal monitoring data, residential history, migration records, as well as insights into daily activity patterns. This lack of data could potentially lead to misclassification of exposure because it depends on multiple factors, such as the time

spent indoors and outdoors, and the time spent away from her registered living address. Fourth, we excluded miscarriage in our study because of data quality as California does not legally require the documentation of pregnancy loss occurring before 20 weeks of gestation in birth certificates. Such information could not be considered reliable in these datasets. Consequently, our analysis was confined to fetal deaths occurring within a gestational range of 20 to 42 weeks. The exclusion of deaths after 20 weeks means we systematically exclude those who died earlier with potentially higher exposures. Such selection bias would bias our results towards the null. Moreover, we did not have specific causes of infant deaths, making it challenging to ascertain whether the effects of air pollution are linked to deaths related to respiratory issues after birth, as it is known that air pollutants can adversely affect the respiratory system in infants and children due to their immature lungs. Incorporating these aspects when determining exposure could be advantageous for future research.

In addition, although most people spend the majority of their time indoors, our study focuses on outdoor air. Studies on the connection between indoor and outdoor air show that much of the pollution outside can make its way inside (John D. Spengler, 2000). Even though the direct link between outdoor air quality and a person's exposure is not very strong (with values between .2 and .6 in many PM studies), this connection gets stronger over time. This happens because an individual's indoor pollution sources usually do not change much and do not depend on outdoor air quality. Consequently, for an individual, variations in air quality are primarily influenced by changes in outdoor pollution levels (John D. Spengler, 2000). As such the self-matching study design addressed some of these issues by eliminating the influence of factors that remained consistent over an extended period of weeks. Finally, $PM_{2.5}$ is a complex combination of particles, which can differ from one region to another based on their origins. Therefore, identifying the specific components and origins of $PM_{2.5}$ that have a substantial effect on fetal and infant death is essential. For instance, $PM_{2.5}$ originating from wildfire smoke could be more harmful than general ambient $PM_{2.5}$ exposure. However, we lacked the necessary data to investigate this vital aspect, which could potentially account for the observed inverse and protective effects of air pollution on fetal and infant deaths.

5. Conclusions

This study highlights a significant increase in prenatal and postnatal fatalities linked to short-term exposure to $PM_{2.5}$ and ozone, specifically within two weeks. These effects varied by season where the impacts of $PM_{2.5}$ were pronounced in the cold season whereas the effects of O_3 were significant in the warm season. These findings substantiate the urgency for policies promoting reproductive justice, targeted at reducing the detrimental impacts of air pollution, especially within disadvantaged and financially strained communities that suffer from these adverse birth outcomes. Considering climate change elements such as escalating temperatures, increased frequency of heatwaves, and natural calamities, the importance of intensifying investigations into the effect of climate change on birth outcomes cannot be overstated. As advancing climate change threatens to exacerbate air pollution, understanding this connection becomes increasingly vital for

improving public health readiness and formulating effective adaptation strategies, especially among vulnerable populations.

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7. Supplementary tables

Table S7. Distribution of air pollutants and weather variables during the study period (2016 - 2019)

Season	Parameter	Mean	SD	Min	P25	P50	P75	Max	QR
Warm	$PM_{2.5}$ ($\mu g/m^3$)	9.8	7.9	1.0	5.7	8.3	11.7	214.9	6.0
	O_3 (ppb)	57.5	14.1	5.0	47.2	57.3	67.2	141.6	20.0
	Temperature (^0C)	20.6	5.1	-8.7	17.3	20.6	23.9	42.2	6.7
	Humidity (%)	53.8	16.9	1.3	40.7	56.0	67.6	100.0	26.9
Cold	$PM_{2.5}$ ($\mu g/m^3$)	13.1	11.8	1.0	5.2	9.3	17.2	253.4	11.9
	O_3 (ppb)	35.8	12.4	1.0	27.4	35.6	43.7	92.5	16.3
	Temperature (^0C)	11.7	5.0	-19.3	8.9	12.1	15.1	32.2	6.2
	Humidity (%)	60.7	17.1	2.7	50.2	64.0	73.4	100.0	23.2

Abbreviations: O_3, ozone; $PM_{2.5}$, particulate matter <2.5 microns; C, Celsius; SD, standard deviation; P, percentile; QR, quartile range

Table S8. Distribution of exposures among study participants by the season of delivery

Pollutant	Season	Lag day	Case period			Control period		
			IQR	Mean	SD	IQR	Mean	SD
$PM_{2.5}$ ($\mu g/m^3$)	Warm	0	5.9	9.1	5.4	5.6	9.5	5.8
		1	5.5	9.4	5.6	5.5	9.4	5.7
		2	5.1	9.2	5.6	5.3	9.4	5.5
		3	5.2	9.2	5.7	5.5	9.4	5.7
		4	5.4	9.3	6.0	5.7	9.4	5.7
		5	5.9	9.2	5.6	5.7	9.5	5.7
		6	6.1	9.4	6.1	5.7	9.5	5.7
		7	5.9	9.4	6.0	5.6	9.4	5.6
		8	5.7	9.2	5.8	5.5	9.4	5.6
		9	5.0	9.1	5.2	5.4	9.4	5.6
		10	4.8	8.8	5.1	5.5	9.4	5.8
		11	5.4	8.8	4.8	5.7	9.5	5.9
		12	5.2	8.8	4.9	6.0	9.5	5.9
		13	5.2	8.9	5.0	6.0	9.6	6.0
		14	5.3	9.1	5.5	5.8	9.5	5.8
	Cold	0	11.7	12.8	12.3	12.1	13.4	13.9
		1	11.4	12.8	12.6	11.7	13.1	13.4
		2	11.3	12.5	12.1	10.4	12.8	13.3
		3	10.1	12.5	12.5	10.2	12.7	13.0
		4	10.0	12.6	12.6	10.8	12.5	13.3
		5	11.6	13.2	14.6	11.0	12.7	13.3
		6	11.5	13.5	13.9	11.7	13.1	13.5
		7	12.4	13.9	14.2	11.8	13.1	13.2
		8	11.6	13.3	14.2	11.3	12.8	12.7
		9	10.6	13.3	14.9	10.5	12.6	12.3
		10	10.5	13.5	14.4	10.0	12.5	12.3
		11	11.5	13.2	14.5	10.4	12.3	12.5
		12	12.3	13.2	14.4	10.9	12.6	13.5
		13	12.6	13.5	15.1	11.2	13.0	13.3
		14	12.2	14.0	14.6	11.9	13.1	13.1
O_3 (ppb)	Warm	0	19.5	56.1	13.4	19.9	56.4	13.2
		1	18.3	56.3	13.3	19.7	56.5	13.1
		2	18.8	56.5	12.8	19.9	56.4	13.5
		3	18.7	56.2	12.5	19.8	56.3	13.3
		4	18.2	56.3	12.6	19.7	56.2	13.3
		5	19.4	56.1	13.0	20.2	56.2	13.4
		6	19.9	56.7	13.0	20.5	56.5	13.4
		7	18.7	56.1	12.7	19.8	56.4	13.3
		8	18.4	56.2	12.6	19.3	56.6	13.2
		9	18.9	56.2	12.8	19.6	56.5	13.3
		10	19.7	55.9	12.9	19.5	56.3	13.1
		11	20.7	56.4	12.7	19.1	56.2	13.2
		12	20.5	56.5	13.0	20.0	56.2	13.4
		13	19.5	56.8	12.7	20.9	56.6	13.4
		14	19.1	56.6	12.7	19.9	56.4	13.3
	Cold	0	15.6	35.6	11.5	15.8	35.7	11.5

		1	14.9	35.9	11.4	15.5	35.5	11.5
		2	14.9	36.2	11.4	14.5	35.7	11.3
		3	14.8	36.0	11.7	14.9	35.8	11.6
		4	15.2	35.8	11.3	15.0	35.7	11.1
		5	13.8	35.6	11.3	15.4	35.8	11.4
		6	14.9	35.7	11.2	15.2	36.0	11.5
		7	15.6	34.8	11.4	15.5	35.7	11.5
		8	14.4	34.8	11.1	15.3	35.8	11.5
		9	13.4	34.7	10.9	14.7	36.0	11.5
		10	13.8	35.2	11.1	15.1	35.9	11.7
		11	13.7	35.5	11.1	15.1	35.7	11.2
		12	13.9	35.6	11.5	15.1	35.7	11.3
		13	14.6	35.4	11.3	15.2	36.1	11.4
		14	15.2	35.5	11.4	15.6	35.7	11.5

Abbreviations: O_3, Ozone; $PM_{2.5}$, particulate matter <2.5 microns; IQR, interquartile range; SD, standard deviation

Table S9. The Spearman correlation coefficient between air pollutants and weather variables throughout the whole two weeks of exposure windows by the season of delivery

Season	Parameter	$PM_{2.5}$	O_3	Temperature	Humidity
Cold	$PM_{2.5}$ ($\mu g/m^3$)	1.0	-0.4	-0.2	0.0
	O_3 (ppb)	-0.4	1.0	0.7	-0.6
	Temperature (C)	-0.2	0.7	1.0	-0.3
	Humidity (%)	0.0	-0.6	-0.3	1.0
Warm	$PM_{2.5}$ ($\mu g/m^3$)	1.0	0.4	0.2	-0.3
	O_3 (ppb)	0.4	1.0	0.7	-0.6
	Temperature (C)	0.2	0.7	1.0	-0.4
	Humidity (%)	-0.3	-0.6	-0.4	1.0

Abbreviations: O_3, ozone; $PM_{2.5}$, particulate matter <2.5 microns; C, Celsius

Table S10. The Spearman correlation coefficient between the lags (lag 0 to lag 14) of air pollutants and weather variables throughout the whole two weeks of exposure windows by the season of delivery

Season	**Spearman Correlation Coefficients, N = 3228**															
Cold	lag(day)	pm0	pm1	pm2	pm3	pm4	pm5	pm6	pm7	pm8	pm9	pm10	pm11	pm12	pm13	pm14
	pm0	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.4	0.4	0.5
	pm1	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.4	0.4
	pm2	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.6	0.5	0.4	0.4	0.4	0.4
	pm3	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.6	0.5	0.4	0.4	0.3
	pm4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4
	pm5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.6	0.5	0.4
	pm6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5
	pm7	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7
	pm8	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5
	pm9	0.4	0.5	0.6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4
	pm10	0.4	0.4	0.5	0.6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4
	pm11	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4
	pm12	0.4	0.4	0.4	0.4	0.5	0.6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5
	pm13	0.4	0.4	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7
	pm14	0.5	0.4	0.4	0.3	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0
	Spearman Correlation Coefficients, N = 3228															
Cold	lag(day)	ozone0	ozone1	ozone2	ozone3	ozone4	ozone5	ozone6	ozone7	ozone8	ozone9	ozone10	ozone11	ozone12	ozone13	ozone14
	ozone0	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7
	ozone1	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.6	0.7
	ozone2	0.7	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.6	0.6
	ozone3	0.7	0.7	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.6
	ozone4	0.7	0.7	0.7	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7
	ozone5	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7
	ozone6	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8	0.7
	ozone7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.8
	ozone8	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8	0.7	0.7	0.7	0.7	0.7
	ozone9	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8	0.8	0.7	0.7	0.7

	ozone10	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8	0.7	0.7	0.7
	ozone11	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.7	0.7
	ozone12	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8	0.7
	ozone13	0.7	0.6	0.6	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0	0.8
	ozone14	0.7	0.7	0.6	0.6	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.8	1.0
	Spearman Correlation Coefficients, N = 3327															
Cold	lag(day)	temp0	temp1	temp2	temp3	temp4	temp5	temp6	temp7	temp8	temp9	temp10	temp11	temp12	temp13	temp14
	temp0	1.0	0.9	0.7	0.6	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.6	0.6	0.6	0.6
	temp1	0.9	1.0	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.6	0.6	0.6	0.6	0.6
	temp2	0.7	0.8	1.0	0.9	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.6	0.6	0.6	0.6
	temp3	0.6	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.6
	temp4	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.7	0.7	0.6	0.6
	temp5	0.6	0.7	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.7	0.7	0.6
	temp6	0.7	0.7	0.7	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.8	0.7
	temp7	0.7	0.7	0.7	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.7	0.7	0.8
	temp8	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.8	0.7	0.7	0.6	0.7	0.7
	temp9	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.8	1.0	0.9	0.7	0.7	0.7	0.6
	temp10	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6
	temp11	0.6	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7
	temp12	0.6	0.6	0.6	0.6	0.7	0.7	0.7	0.7	0.6	0.7	0.7	0.9	1.0	0.9	0.7
	temp13	0.6	0.6	0.6	0.6	0.6	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.9	1.0	0.9
	temp14	0.6	0.6	0.6	0.6	0.6	0.6	0.7	0.8	0.7	0.6	0.6	0.7	0.7	0.9	1.0
	Spearman Correlation Coefficients, N = 3327															
Cold	lag(day)	rh0	rh1	rh2	rh3	rh4	rh5	rh6	rh7	rh8	rh9	rh10	rh11	rh12	rh13	rh14
	rh0	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.4	0.4	0.5	0.5	0.6
	rh1	0.7	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.5	0.5

	rh2	0.6	0.7	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.4	0.4	0.5
	rh3	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.5	0.5	0.4	0.5
	rh4	0.5	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.5	0.5	0.4
	rh5	0.5	0.5	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5
	rh6	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6
	rh7	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.8	0.6	0.5	0.5	0.5	0.6	0.7
	rh8	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.8	1.0	0.7	0.6	0.5	0.5	0.5	0.6
	rh9	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.5	0.5
	rh10	0.4	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.5
	rh11	0.4	0.4	0.5	0.5	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.7	0.6	0.5
	rh12	0.5	0.5	0.4	0.5	0.5	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.7	0.6
	rh13	0.5	0.5	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.7
	rh14	0.6	0.5	0.5	0.5	0.4	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0
	Spearman Correlation Coefficients, N = 3285															
Warm	lag(day)	pm0	pm1	pm2	pm3	pm4	pm5	pm6	pm7	pm8	pm9	pm10	pm11	pm12	pm13	pm14
	pm0	1.0	0.7	0.5	0.4	0.4	0.5	0.5	0.7	0.5	0.5	0.4	0.4	0.4	0.5	0.5
	pm1	0.7	1.0	0.7	0.5	0.5	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.4	0.4
	pm2	0.5	0.7	1.0	0.7	0.5	0.5	0.4	0.4	0.5	0.7	0.5	0.5	0.4	0.4	0.4
	pm3	0.4	0.5	0.7	1.0	0.7	0.5	0.5	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4
	pm4	0.4	0.5	0.5	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.5	0.7	0.6	0.5	0.4
	pm5	0.5	0.4	0.5	0.5	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.5	0.7	0.6	0.5
	pm6	0.5	0.4	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.6	0.7	0.6
	pm7	0.7	0.5	0.4	0.4	0.5	0.6	0.7	1.0	0.7	0.5	0.4	0.4	0.5	0.6	0.7
	pm8	0.5	0.7	0.5	0.4	0.4	0.5	0.6	0.7	1.0	0.7	0.5	0.5	0.4	0.5	0.5
	pm9	0.5	0.5	0.7	0.5	0.5	0.4	0.5	0.5	0.7	1.0	0.7	0.5	0.5	0.4	0.4
	pm10	0.4	0.4	0.5	0.7	0.5	0.5	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.5	0.4
	pm11	0.4	0.4	0.5	0.6	0.7	0.5	0.5	0.4	0.5	0.5	0.7	1.0	0.7	0.6	0.5
	pm12	0.4	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.5	0.7	1.0	0.7	0.6
	pm13	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.6	0.7	1.0	0.8
	pm14	0.5	0.4	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.5	0.6	0.8	1.0
	Spearman Correlation Coefficients, N = 3285															
Warm	lag(day)	ozone0	ozone1	ozone2	ozone3	ozone4	ozone5	ozone6	ozone7	ozone8	ozone9	ozone10	ozone11	ozone12	ozone13	ozone14
	ozone0	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4	0.4	0.5	0.6
	ozone1	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4	0.4	0.5
	ozone2	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4	0.4
	ozone3	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4

	ozone4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.6	0.7	0.6	0.5	0.4
	ozone5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5
	ozone6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6
	ozone7	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7
	ozone8	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5
	ozone9	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4
	ozone10	0.4	0.5	0.6	0.7	0.6	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4
	ozone11	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4
	ozone12	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5
	ozone13	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7
	ozone14	0.6	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0
	Spearman Correlation Coefficients, N = 3360															
Warm	lag(day)	temp0	temp1	temp2	temp3	temp4	temp5	temp6	temp7	temp8	temp9	temp10	temp11	temp12	temp13	temp14
	temp0	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7
	temp1	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7
	temp2	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.7
	temp3	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7
	temp4	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7
	temp5	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7
	temp6	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8
	temp7	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8
	temp8	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8
	temp9	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7
	temp10	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7
	temp11	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7

	temp12	0.7	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8
	temp13	0.7	0.7	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9
	temp14	0.7	0.7	0.7	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0
	Spearman Correlation Coefficients, N = 3360															
Warm	lag(day)	rh0	rh1	rh2	rh3	rh4	rh5	rh6	rh7	rh8	rh9	rh10	rh11	rh12	rh13	rh14
	rh0	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.6	0.6	0.5	0.5	0.5	0.5	0.6
	rh1	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.6	0.6	0.5	0.5	0.5	0.5
	rh2	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7	0.7	0.6	0.5	0.5	0.5
	rh3	0.6	0.7	0.8	1.0	0.9	0.7	0.6	0.5	0.6	0.6	0.7	0.7	0.6	0.5	0.5
	rh4	0.5	0.6	0.7	0.9	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7	0.7	0.6	0.5
	rh5	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7	0.7	0.6
	rh6	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.7	0.7	0.7
	rh7	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7
	rh8	0.6	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.7
	rh9	0.6	0.6	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.9	0.7	0.6	0.5	0.6
	rh10	0.5	0.6	0.7	0.7	0.6	0.6	0.5	0.6	0.7	0.9	1.0	0.8	0.7	0.6	0.6
	rh11	0.5	0.5	0.6	0.7	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6
	rh12	0.5	0.5	0.5	0.6	0.7	0.7	0.7	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7
	rh13	0.5	0.5	0.5	0.5	0.6	0.7	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.8
	rh14	0.6	0.5	0.5	0.5	0.5	0.6	0.7	0.7	0.7	0.6	0.6	0.6	0.7	0.8	1.0

Abbreviations: pm, particulate matter <2.5 microns; temp, temperature; rh, relative humidity

Table S11. Distribution of exposures among study participants by the season of death

Pollutant	Season	Lag day	Case period			Control period		
			IQR	Mean	SD	IQR	Mean	SD
$PM_{2.5}$ ($\mu g/m^3$)	Warm	0	5.5	9.8	6.3	5.6	9.1	6.4
		1	6.3	9.6	6.6	5.8	9.3	6.3
		2	6.2	9.7	6.5	6.1	9.5	6.4
		3	6.7	9.7	6.6	5.7	9.3	5.7
		4	5.7	9.6	6.1	5.7	9.3	5.7
		5	6.2	9.6	6.2	5.6	9.3	6.1
		6	5.8	9.4	6.2	5.8	9.4	6.3
		7	5.4	9.1	5.3	5.7	9.4	6.6
		8	5.7	9.0	5.2	6.0	9.5	6.6
		9	6.0	9.1	5.5	6.0	9.7	6.7
		10	5.8	9.2	5.8	5.7	9.5	6.0
		11	6.0	9.0	5.6	5.6	9.4	5.8
		12	6.0	9.1	5.7	5.6	9.4	6.3
		13	5.7	9.0	5.7	5.7	9.3	6.4
		14	5.5	9.1	5.5	5.6	9.2	6.5
	Cold	0	11.3	12.9	13.8	10.7	12.7	14.1
		1	11.1	13.0	15.3	10.0	12.4	13.6
		2	11.9	13.1	14.3	10.6	12.6	13.9
		3	10.8	12.8	13.6	10.7	12.4	12.8
		4	11.1	12.7	13.6	10.7	12.6	13.5
		5	10.1	13.0	13.6	10.4	12.6	13.7
		6	11.2	12.9	14.1	10.8	12.7	13.5
		7	11.9	12.6	13.2	10.8	12.8	14.1
		8	10.7	13.0	14.0	10.2	12.3	13.8
		9	11.8	13.1	14.9	10.0	12.4	13.4
		10	11.5	12.7	12.6	10.3	12.2	12.8
		11	11.2	12.7	13.8	10.5	12.5	13.2
		12	10.9	12.2	11.0	10.3	12.7	14.2
		13	11.0	12.8	11.6	10.9	12.8	14.1
		14	11.0	12.9	11.5	11.0	12.8	14.5
O_3 (ppb)	Warm	0	19.4	56.8	13.0	20.2	55.8	13.3
		1	20.4	56.3	13.3	20.0	55.8	13.3
		2	20.6	56.7	13.4	18.7	55.9	13.4
		3	20.1	57.2	13.1	20.2	55.8	13.7
		4	19.6	56.9	13.1	19.7	55.9	13.2
		5	20.5	56.7	13.0	19.7	56.1	13.4
		6	18.1	56.7	12.6	20.3	55.9	13.3
		7	18.0	56.3	12.4	20.3	56.3	13.4
		8	17.5	56.7	12.2	20.2	55.9	13.4
		9	18.2	56.5	12.5	19.4	56.3	13.5
		10	18.7	56.7	12.8	20.3	56.3	13.6
		11	19.0	56.5	12.8	19.5	56.1	13.2
		12	18.1	56.5	12.7	20.2	56.1	13.3
		13	19.5	56.6	12.8	19.6	56.0	13.0
		14	18.4	57.0	12.9	19.6	56.1	13.1
	Cold	0	15.1	36.8	11.7	16.0	36.7	11.4

		1	14.9	36.8	11.3	15.9	36.8	11.7
		2	14.9	36.5	11.7	16.2	36.9	12.0
		3	15.5	36.8	11.7	15.8	36.7	11.7
		4	15.6	36.1	11.6	16.0	36.8	11.8
		5	14.9	36.3	11.7	16.8	36.9	11.7
		6	16.5	36.9	11.8	15.6	36.8	11.6
		7	13.8	36.7	11.5	15.8	36.7	11.4
		8	13.6	36.8	11.6	15.5	36.8	11.6
		9	16.0	36.6	12.1	15.4	36.9	12.0
		10	15.3	36.7	12.1	15.7	36.9	11.7
		11	14.3	36.6	12.0	16.0	36.7	11.7
		12	14.7	36.2	11.7	16.6	36.9	11.8
		13	15.6	36.4	12.1	15.6	36.8	11.6
		14	15.1	36.6	12.2	15.6	36.6	11.3

Abbreviations: O_3, ozone, $PM_{2.5}$, particulate matter <2.5 microns; IQR, interquartile range; SD, standard deviation

Table S12. The Spearman correlation coefficient between air pollutants and weather variables throughout the whole two weeks of exposure windows by the season of death

Season	Parameter	$PM_{2.5}$	O_3	Temperature	Humidity
Cod	$PM_{2.5}$ ($\mu g/m^3$)	1.0	-0.4	-0.3	0.0
	O_3 (ppb)	-0.4	1.0	0.7	-0.6
	Temperature (C)	-0.3	0.7	1.0	-0.3
	Humidity (%)	0.0	-0.6	-0.3	1.0
Warm	$PM_{2.5}$ ($\mu g/m^3$)	1.0	0.4	0.3	-0.4
	O_3 (ppb)	0.4	1.0	0.7	-0.6
	Temperature (C)	0.3	0.7	1.0	-0.4
	Humidity (%)	-0.4	-0.6	-0.4	1.0

Abbreviations: O_3, ozone; $PM_{2.5}$, particulate matter <2.5 microns; C, Celsius

Table S13. The Spearman correlation coefficient between the lags (lag 0 to lag 14) of air pollutants and weather variables throughout the whole two weeks of exposure windows by the season of death

Season	**Spearman Correlation Coefficients, N = 2504**															
Cold	lag(day)	pm0	pm1	pm2	pm3	pm4	pm5	pm6	pm7	pm8	pm9	pm10	pm11	pm12	pm13	pm14
	pm0	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.4	0.4	0.5
	pm1	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.4	0.5
	pm2	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.5	0.5	0.7	0.5	0.4	0.4	0.4	0.4
	pm3	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4
	pm4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4
	pm5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5
	pm6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6
	pm7	0.7	0.5	0.5	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7
	pm8	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.6
	pm9	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.5
	pm10	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4
	pm11	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4
	pm12	0.4	0.4	0.4	0.4	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5
	pm13	0.4	0.4	0.4	0.4	0.4	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7
	pm14	0.5	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.4	0.5	0.7	1.0
	Spearman Correlation Coefficients, N = 2504															
Cold	lag(day)	ozone0	ozone1	ozone2	ozone3	ozone4	ozone5	ozone6	ozone7	ozone8	ozone9	ozone10	ozone11	ozone12	ozone13	ozone14
	ozone0	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7
	ozone1	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.7
	ozone2	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.7
	ozone3	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7
	ozone4	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7
	ozone5	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7	0.7
	ozone6	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8	0.7
	ozone7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.7	0.8
	ozone8	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7	0.8
	ozone9	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7	0.7

	ozone10	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7	0.7
	ozone11	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8	0.7
	ozone12	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8	0.8
	ozone13	0.7	0.7	0.7	0.7	0.7	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.8	1.0	0.8
	ozone14	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.8	0.8	0.7	0.7	0.7	0.8	0.8	1.0
	Spearman Correlation Coefficients, N = 2574															
Cold	lag(day)	temp0	temp1	temp2	temp3	temp4	temp5	temp6	temp7	temp8	temp9	temp10	temp11	temp12	temp13	temp14
	temp0	1.0	0.8	0.7	0.6	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.6	0.6	0.6	0.6
	temp1	0.8	1.0	0.8	0.7	0.6	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.6	0.5	0.6
	temp2	0.7	0.8	1.0	0.8	0.7	0.7	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.5	0.5
	temp3	0.6	0.7	0.8	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.6
	temp4	0.6	0.6	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.7	0.7	0.7	0.7	0.6	0.6
	temp5	0.6	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.8	0.7	0.6
	temp6	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6	0.7	0.7	0.7
	temp7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.8	0.7	0.7	0.6	0.7	0.7	0.7
	temp8	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.8	1.0	0.9	0.7	0.7	0.7	0.6	0.7
	temp9	0.6	0.7	0.7	0.7	0.7	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7	0.6	0.6
	temp10	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.9	0.8	0.7	0.6
	temp11	0.6	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.9	0.7	0.7
	temp12	0.6	0.6	0.6	0.6	0.7	0.8	0.7	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.7
	temp13	0.6	0.5	0.5	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0	0.9
	temp14	0.6	0.6	0.5	0.6	0.6	0.6	0.7	0.7	0.7	0.6	0.6	0.7	0.7	0.9	1.0
	Spearman Correlation Coefficients, N = 2574															
Cold	lag(day)	rh0	rh1	rh2	rh3	rh4	rh5	rh6	rh7	rh8	rh9	rh10	rh11	rh12	rh13	rh14
	rh0	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.5	0.5	0.5
	rh1	0.7	1.0	0.8	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.5	0.5

	rh2	0.6	0.8	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.5
	rh3	0.5	0.6	0.7	1.0	0.8	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5	0.5
	rh4	0.5	0.5	0.6	0.8	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5
	rh5	0.5	0.5	0.5	0.6	0.7	1.0	0.8	0.6	0.5	0.5	0.5	0.6	0.7	0.6	0.5
	rh6	0.6	0.5	0.5	0.5	0.6	0.8	1.0	0.7	0.6	0.5	0.5	0.5	0.6	0.7	0.6
	rh7	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.8	0.6	0.5	0.5	0.5	0.6	0.7
	rh8	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.8	1.0	0.8	0.6	0.5	0.5	0.6	0.6
	rh9	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.8	1.0	0.7	0.6	0.5	0.5	0.5
	rh10	0.4	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.8	0.6	0.5	0.5
	rh11	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.8	1.0	0.7	0.6	0.5
	rh12	0.5	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.7	1.0	0.8	0.6
	rh13	0.5	0.5	0.5	0.5	0.5	0.6	0.7	0.6	0.6	0.5	0.5	0.6	0.8	1.0	0.8
	rh14	0.5	0.5	0.5	0.5	0.5	0.5	0.6	0.7	0.6	0.5	0.5	0.5	0.6	0.8	1.0
	Spearman Correlation Coefficients, N = 2830															
Warm	lag(day)	pm0	pm1	pm2	pm3	pm4	pm5	pm6	pm7	pm8	pm9	pm10	pm11	pm12	pm13	pm14
	pm0	1.0	0.7	0.5	0.5	0.4	0.5	0.5	0.7	0.6	0.5	0.5	0.4	0.4	0.5	0.5
	pm1	0.7	1.0	0.7	0.6	0.5	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4	0.4	0.5
	pm2	0.5	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.4	0.4
	pm3	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.4
	pm4	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.5	0.7	0.6	0.5	0.4
	pm5	0.5	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.5	0.7	0.6	0.5
	pm6	0.5	0.4	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.4	0.6	0.7	0.6
	pm7	0.7	0.5	0.5	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.6	0.7
	pm8	0.6	0.7	0.6	0.5	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5	0.6
	pm9	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.6	0.7	1.0	0.7	0.6	0.5	0.4	0.5
	pm10	0.5	0.5	0.6	0.7	0.5	0.5	0.4	0.5	0.6	0.7	1.0	0.8	0.6	0.5	0.4
	pm11	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.5	0.6	0.8	1.0	0.7	0.6	0.5
	pm12	0.4	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.6	0.7	1.0	0.8	0.6
	pm13	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.6	0.8	1.0	0.7
	pm14	0.5	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.6	0.5	0.4	0.5	0.6	0.7	1.0
	Spearman Correlation Coefficients, N = 2830															
Warm	lag(day)	ozone0	ozone1	ozone2	ozone3	ozone4	ozone5	ozone6	ozone7	ozone8	ozone9	ozone10	ozone11	ozone12	ozone13	ozone14
	ozone0	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4	0.4	0.4	0.5
	ozone1	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.5	0.5	0.4	0.4	0.4	0.5
	ozone2	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4	0.4
	ozone3	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4	0.4

	ozone4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5	0.4
	ozone5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6	0.5
	ozone6	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7	0.6
	ozone7	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5	0.7
	ozone8	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4	0.5
	ozone9	0.5	0.5	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5	0.4	0.4	0.4
	ozone10	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.8	0.5	0.4	0.4
	ozone11	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.8	1.0	0.7	0.5	0.4
	ozone12	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7	0.5
	ozone13	0.4	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0	0.7
	ozone14	0.5	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.5	0.4	0.4	0.4	0.5	0.7	1.0
	Spearman Correlation Coefficients, N = 2890															
	lag(day)	temp0	temp1	temp2	temp3	temp4	temp5	temp6	temp7	temp8	temp9	temp10	temp11	temp12	temp13	temp14
	temp0	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7	0.7
	temp1	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.7	0.7
	temp2	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.7
	temp3	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7
	temp4	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7
Warm	temp5	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8	0.7
	temp6	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8	0.8
	temp7	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8	0.8
	temp8	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7	0.8
	temp9	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7	0.7
	temp10	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7	0.7
	temp11	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8	0.7

	temp12	0.7	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9	0.8
	temp13	0.7	0.7	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0	0.9
	temp14	0.7	0.7	0.7	0.7	0.7	0.7	0.8	0.8	0.8	0.7	0.7	0.7	0.8	0.9	1.0
	Spearman Correlation Coefficients, N = 2890															
Warm	lag(day)	rh0	rh1	rh2	rh3	rh4	rh5	rh6	rh7	rh8	rh9	rh10	rh11	rh12	rh13	rh14
	rh0	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7	0.6	0.5	0.5	0.5	0.5	0.5	0.5
	rh1	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.6	0.6	0.5	0.5	0.5	0.5
	rh2	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.6	0.6	0.5	0.5	0.5
	rh3	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.6	0.6	0.5	0.5
	rh4	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.7	0.6	0.5
	rh5	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7	0.6	0.6
	rh6	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6	0.7	0.6
	rh7	0.7	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.6	0.6	0.7
	rh8	0.6	0.7	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.5	0.6
	rh9	0.5	0.6	0.7	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5	0.5
	rh10	0.5	0.6	0.6	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6	0.5
	rh11	0.5	0.5	0.6	0.6	0.7	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8	0.7	0.6
	rh12	0.5	0.5	0.5	0.6	0.7	0.7	0.6	0.6	0.5	0.6	0.7	0.8	1.0	0.8	0.7
	rh13	0.5	0.5	0.5	0.5	0.6	0.6	0.7	0.6	0.5	0.5	0.6	0.7	0.8	1.0	0.8
	rh14	0.5	0.5	0.5	0.5	0.5	0.6	0.6	0.7	0.6	0.5	0.5	0.6	0.7	0.8	1.0

Abbreviations: pm, particulate matter <2.5 microns; temp, temperature; rh, relative humidity

Table S14: Whole-year association between air pollutants and fetal and infant deaths in the SJV, California, 2016-2019

Pollutant	Lag (day)	Fetal death (20-42 weeks)		Infant death (< 1 year)	
		Unadjusted OR (95% CI)[a]	Adjusted OR (95% CI)[b]	Unadjusted OR (95% CI)[a]	Adjusted OR (95% CI)[b]
$PM_{2.5}$	0	0.97(0.90,1.03)	0.97(0.90,1.03)	1.03(0.97,1.11)	1.03(0.97,1.09)
	1	1.00(0.94,1.06)	1.00(0.94,1.06)	1.04(0.97,1.11)	1.03(0.97,1.09)
	2	0.99(0.93,1.05)	0.99(0.93,1.05)	1.03(0.96,1.09)	1.02(0.96,1.09)
	3	0.99(0.93,1.06)	0.99(0.93,1.06)	1.04(0.97,1.11)	1.03(0.97,1.11)
	4	1.01(0.95,1.07)	1.01(0.95,1.07)	1.01(0.95,1.08)	1.01(0.94,1.08)
	5	1.03(0.97,1.08)	1.02(0.97,1.08)	1.03(0.97,1.09)	1.03(0.96,1.09)
	6	1.03(0.97,1.09)	1.02(0.97,1.08)	1.01(0.94,1.07)	1.00(0.94,1.07)
	7	1.04(0.99,1.11)	1.04(0.99,1.11)	0.97(0.91,1.04)	0.97(0.90,1.04)
	8	1.02(0.96,1.08)	1.02(0.96,1.08)	1.01(0.94,1.07)	1.00(0.94,1.07)
	9	1.03(0.97,1.09)	1.03(0.97,1.09)	1.00(0.94,1.07)	1.00(0.93,1.06)
	10	1.03(0.97,1.09)	1.03(0.97,1.09)	1.01(0.94,1.08)	1.00(0.93,1.07)
	11	1.03(0.96,1.09)	1.02(0.96,1.08)	0.99(0.92,1.05)	0.98(0.91,1.05)
	12	1.00(0.95,1.06)	1.00(0.94,1.06)	0.96(0.9,1.03)	0.95(0.89,1.02)
	13	1.00(0.94,1.06)	1.00(0.95,1.06)	0.99(0.92,1.05)	0.98(0.91,1.05)
	14	1.04(0.98,1.09)	1.03(0.97,1.09)	1.00(0.93,1.06)	0.99(0.92,1.06)
O_3	0	0.96(0.92,1.00)	0.98(0.93,1.02)	1.04(0.99,1.08)	1.03(0.98,1.08)
	1	0.97(0.93,1.01)	0.98(0.94,1.03)	1.03(0.98,1.07)	1.02(0.97,1.06)
	2	0.98(0.94,1.02)	0.99(0.95,1.04)	1.02(0.98,1.07)	1.01(0.96,1.06)
	3	0.97(0.93,1.02)	0.99(0.94,1.03)	1.05(1.00,1.09)	1.03(0.99,1.08)
	4	0.98(0.94,1.02)	0.99(0.94,1.03)	1.02(0.98,1.07)	1.01(0.96,1.06)
	5	0.97(0.93,1.01)	0.98(0.94,1.02)	1.02(0.97,1.07)	1.01(0.96,1.06)
	6	0.97(0.93,1.01)	0.98(0.94,1.02)	1.03(0.99,1.08)	1.02(0.97,1.07)
	7	0.95(0.91,0.99)	0.96(0.92,1.01)	1.02(0.97,1.06)	1.01(0.96,1.05)
	8	0.95(0.91,0.99)	0.96(0.92,1.01)	1.03(0.98,1.07)	1.02(0.97,1.07)
	9	0.94(0.90,0.98)	0.96(0.92,1.00)	1.01(0.97,1.06)	1.00(0.95,1.05)
	10	0.95(0.91,0.99)	0.97(0.92,1.01)	1.02(0.97,1.06)	1.01(0.96,1.06)
	11	0.97(0.93,1.02)	0.98(0.94,1.03)	1.02(0.97,1.07)	1.01(0.96,1.06)
	12	0.98(0.93,1.02)	0.99(0.94,1.03)	1.01(0.97,1.06)	1.00(0.95,1.05)
	13	0.96(0.92,1.00)	0.98(0.93,1.02)	1.02(0.97,1.07)	1.01(0.96,1.06)
	14	0.97(0.93,1.01)	0.98(0.94,1.03)	1.03(0.99,1.08)	1.02(0.97,1.07)

[a]Models only adjusted for time invariant confounders by design; and ORs were obtained for 10 units increase in each pollutant concentration.

[b]Models adjusted for the other pollutant, temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.

Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5}$, particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley

Table S15: Association between air pollutants and fetal death in the single pollutant model in the SJV, California 2016-2019

Season	Lag (day)	$PM_{2.5}$		O_3	
		Unadjusted OR (95% CI) [a]	Adjusted OR (95% CI) [b]	Unadjusted OR (95% CI) [a]	Adjusted OR (95% CI) [b]
Warm	0	0.82(0.75,0.88)	0.83(0.77,0.90)	0.92(0.88,0.97)	1.00(0.96,1.05)
	1	0.84(0.78,0.91)	0.86(0.79,0.92)	0.93(0.90,0.98)	1.02(0.97,1.06)
	2	0.83(0.77,0.90)	0.84(0.78,0.91)	0.94(0.90,0.99)	1.03(0.98,1.07)
	3	0.83(0.77,0.90)	0.85(0.78,0.92)	0.94(0.90,0.98)	1.02(0.97,1.06)
	4	0.85(0.78,0.92)	0.87(0.80,0.93)	0.94(0.90,0.99)	1.02(0.97,1.07)
	5	0.86(0.80,0.92)	0.88(0.81,0.94)	0.93(0.89,0.98)	1.01(0.97,1.06)
	6	0.86(0.80,0.93)	0.88(0.82,0.95)	0.93(0.89,0.98)	1.01(0.97,1.06)
	7	0.88(0.82,0.95)	0.90(0.83,0.96)	0.91(0.87,0.96)	0.99(0.95,1.04)
	8	0.86(0.80,0.93)	0.88(0.81,0.95)	0.91(0.87,0.96)	0.99(0.95,1.04)
	9	0.87(0.80,0.93)	0.89(0.82,0.95)	0.90(0.87,0.95)	0.99(0.94,1.03)
	10	0.87(0.80,0.94)	0.89(0.82,0.95)	0.91(0.87,0.96)	0.99(0.95,1.04)
	11	0.86(0.80,0.93)	0.88(0.81,0.95)	0.93(0.90,0.98)	1.02(0.97,1.06)
	12	0.84(0.78,0.91)	0.86(0.79,0.92)	0.94(0.90,0.98)	1.02(0.97,1.07)
	13	0.84(0.78,0.91)	0.86(0.79,0.92)	0.92(0.89,0.97)	1.01(0.96,1.05)
	14	0.87(0.81,0.93)	0.89(0.82,0.95)	0.93(0.90,0.98)	1.02(0.97,1.06)
Cold	0	0.99(0.93,1.05)	0.99(0.93,1.05)	0.99(0.94,1.03)	0.93(0.89,0.98)
	1	1.02(0.96,1.08)	1.02(0.96,1.08)	1.00(0.96,1.04)	0.94(0.90,0.99)
	2	1.01(0.95,1.07)	1.01(0.95,1.07)	1.01(0.96,1.05)	0.95(0.90,1.00)
	3	1.01(0.95,1.08)	1.01(0.95,1.08)	1.00(0.96,1.04)	0.94(0.90,0.99)
	4	1.03(0.97,1.09)	1.03(0.97,1.09)	1.00(0.96,1.05)	0.95(0.90,1.00)
	5	1.04(0.99,1.11)	1.04(0.99,1.11)	0.99(0.95,1.04)	0.94(0.90,0.99)
	6	1.05(0.99,1.11)	1.05(0.99,1.11)	0.99(0.95,1.04)	0.94(0.90,0.99)
	7	1.06(1.01,1.13)	1.06(1.01,1.13)	0.97(0.93,1.02)	0.92(0.88,0.97)
	8	1.04(0.98,1.12)	1.05(0.99,1.12)	0.97(0.93,1.02)	0.92(0.88,0.97)
	9	1.05(0.99,1.12)	1.05(0.99,1.12)	0.97(0.93,1.01)	0.91(0.87,0.96)
	10	1.05(0.99,1.12)	1.05(0.99,1.12)	0.98(0.93,1.02)	0.92(0.88,0.97)
	11	1.05(0.98,1.12)	1.05(0.99,1.12)	1.00(0.96,1.04)	0.94(0.90,0.99)
	12	1.02(0.96,1.08)	1.02(0.97,1.08)	1.00(0.96,1.04)	0.95(0.90,0.99)
	13	1.02(0.96,1.08)	1.02(0.97,1.08)	0.99(0.95,1.03)	0.93(0.89,0.98)
	14	1.05(0.99,1.12)	1.05(1.00,1.12)	1.00(0.95,1.04)	0.94(0.90,0.99)

[a]Models not adjusted for temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.

[b]Models adjusted for temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.

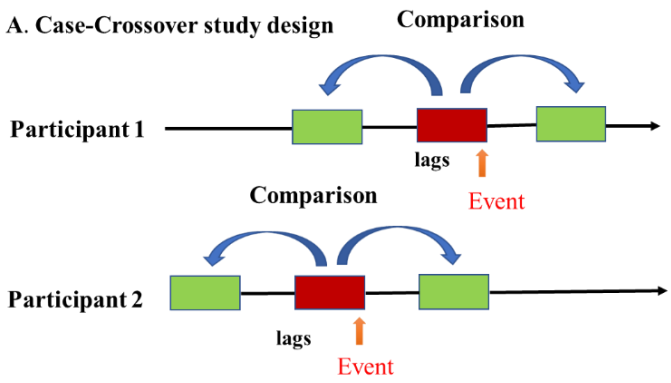
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5}$, particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.

Table S16: Association between air pollutants and infant death in the single pollutant model in the SJV, California, 2016-2019

Season	Lag (day)	$PM_{2.5}$		O_3	
		Unadjusted OR (95% CI)[a]	Adjusted OR (95% CI)[b]	Unadjusted OR (95% CI)[a]	Adjusted OR (95% CI)[b]
Warm	0	1.01(0.93,1.08)	0.99(0.91,1.07)	1.08(1.03,1.13)	1.07(1.02,1.13)
	1	1.01(0.93,1.09)	0.99(0.91,1.07)	1.07(1.02,1.12)	1.06(1.01,1.12)
	2	1.00(0.93,1.08)	0.98(0.91,1.06)	1.06(1.02,1.12)	1.06(1.01,1.12)
	3	1.01(0.93,1.09)	0.99(0.91,1.08)	1.08(1.04,1.14)	1.08(1.03,1.14)
	4	0.99(0.91,1.07)	0.97(0.90,1.05)	1.06(1.02,1.12)	1.06(1.01,1.12)
	5	1.00(0.93,1.08)	0.99(0.91,1.07)	1.06(1.01,1.12)	1.05(1.00,1.11)
	6	0.98(0.90,1.06)	0.96(0.89,1.04)	1.07(1.03,1.13)	1.07(1.02,1.13)
	7	0.95(0.88,1.03)	0.93(0.86,1.01)	1.06(1.01,1.11)	1.05(1.00,1.11)
	8	0.98(0.90,1.06)	0.96(0.89,1.04)	1.07(1.02,1.13)	1.06(1.01,1.12)
	9	0.98(0.90,1.06)	0.96(0.89,1.04)	1.05(1.00,1.11)	1.04(0.99,1.09)
	10	0.98(0.90,1.07)	0.97(0.89,1.05)	1.06(1.01,1.12)	1.05(1.00,1.11)
	11	0.96(0.89,1.04)	0.94(0.87,1.03)	1.06(1.01,1.12)	1.05(1.00,1.11)
	12	0.93(0.86,1.02)	0.91(0.84,1.00)	1.05(1.01,1.11)	1.04(0.99,1.11)
	13	0.96(0.89,1.04)	0.94(0.87,1.02)	1.06(1.01,1.12)	1.05(1.00,1.11)
	14	0.97(0.90,1.05)	0.95(0.88,1.03)	1.07(1.03,1.13)	1.07(1.02,1.13)
Cold	0	1.05(0.98,1.12)	1.04(0.98,1.11)	0.96(0.90,1.01)	0.95(0.90,1.00)
	1	1.05(0.99,1.12)	1.04(0.98,1.11)	0.95(0.90,1.00)	0.94(0.89,0.99)
	2	1.04(0.98,1.11)	1.03(0.97,1.11)	0.95(0.90,1.00)	0.94(0.89,0.99)
	3	1.05(0.98,1.13)	1.04(0.98,1.12)	0.97(0.91,1.02)	0.95(0.90,1.01)
	4	1.03(0.96,1.09)	1.02(0.95,1.09)	0.95(0.90,1.00)	0.94(0.89,0.99)
	5	1.04(0.98,1.12)	1.04(0.97,1.11)	0.94(0.90,0.99)	0.93(0.88,0.98)
	6	1.02(0.96,1.09)	1.01(0.95,1.08)	0.96(0.90,1.01)	0.94(0.90,1.00)
	7	0.99(0.92,1.06)	0.98(0.91,1.05)	0.94(0.90,0.99)	0.93(0.88,0.98)
	8	1.02(0.95,1.08)	1.01(0.95,1.08)	0.95(0.90,1.00)	0.94(0.89,0.99)
	9	1.02(0.95,1.08)	1.01(0.94,1.07)	0.93(0.89,0.99)	0.92(0.88,0.98)
	10	1.02(0.95,1.09)	1.01(0.94,1.08)	0.94(0.90,0.99)	0.93(0.89,0.98)
	11	1.00(0.93,1.07)	0.99(0.92,1.06)	0.94(0.90,1.00)	0.93(0.89,0.99)
	12	0.97(0.90,1.04)	0.96(0.90,1.03)	0.93(0.89,0.99)	0.92(0.88,0.98)
	13	1.00(0.93,1.07)	0.99(0.92,1.06)	0.94(0.90,1.00)	0.93(0.89,0.99)
	14	1.01(0.94,1.08)	1.00(0.93,1.07)	0.96(0.90,1.01)	0.94(0.09,1.00)

[a]Models not adjusted for temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
[b]Models adjusted for temperature, and relative humidity; and ORs were obtained for 10 units increase in each pollutant concentration.
Abbreviations: OR, odds ratio; CI, confidence intervals; $PM_{2.5,}$ particulate matter <2.5 microns; O_3, ozone; SJV, San Joaquin Valley.

8. Supplementary figures



B. Sample control selection scheme for August 2019

Sun	Mon	Tue	Wed	Thu	Fri	Sat
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

1. **Case periods**
2. **Control periods**

Figure S4. Study design scheme

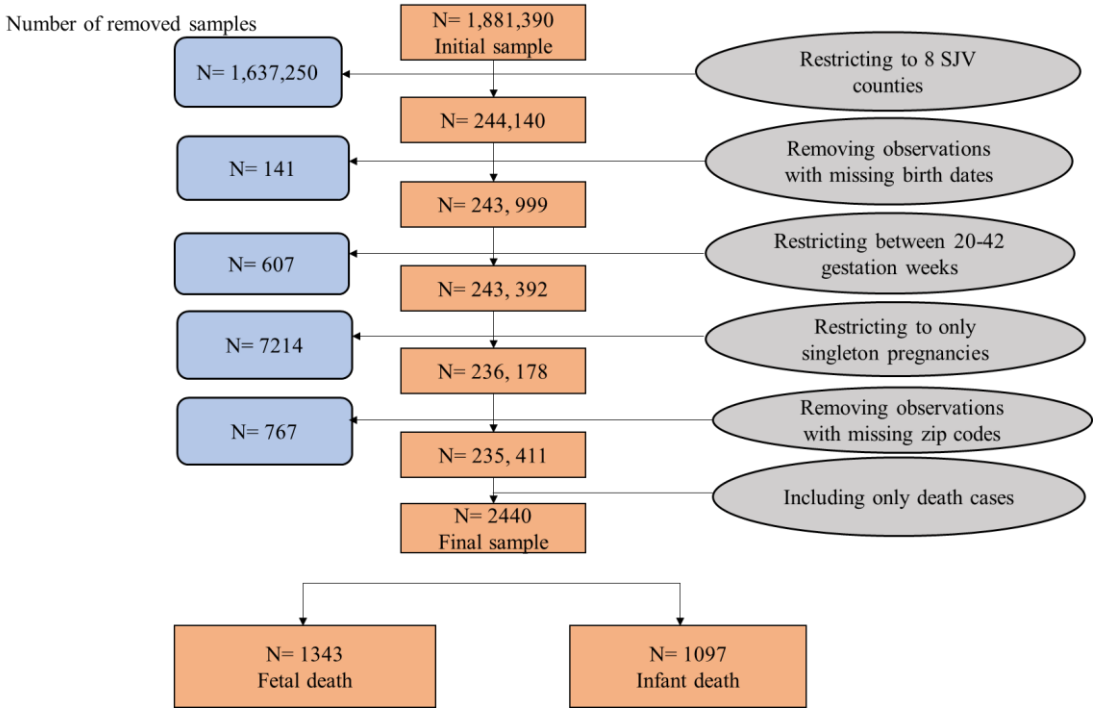


Figure S5: Flowchart representing sample selection scheme