# PRENATAL EXPOSURE TO PM2.5 AND INFANT HEALTH : EVIDENCE FROM QUEBEC

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# Prenatal Exposure to PM2.5 and Infant health: Evidence from Quebec[*]

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**Abstract/Résumé**

This paper examines the effects of prenatal exposure to fine particulate matter (PM2.5) on birth outcomes in a low-pollution setting. Using linked administrative data on births and air quality in Quebec (2008-2015), we compare infants exposed to different pollution levels within the same neighborhoods and time periods to account for socioeconomic and seasonal differences. We find no significant effects at the population level, but exposure increases the risk of low birth weight and preterm birth among female infants and children of less-educated mothers. These findings suggest that even in low-exposure environments, current air quality standards may not sufficiently protect vulnerable populations. Strengthening pollution advisories for pregnant women and refining regional air quality policies could help mitigate these risks.

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Cet article examine les effets de l'exposition prénatale aux particules fines (PM2,5) sur les effets à la naissance dans un contexte de faible pollution. En utilisant des données administratives couplées sur les naissances et la qualité de l'air au Québec (2008-2015), nous comparons les nourrissons exposés à différents niveaux de pollution dans les mêmes quartiers et périodes afin de tenir compte des différences socioéconomiques et saisonnières. Nous ne constatons pas d'effets significatifs au niveau de la population, mais l'exposition augmente le risque de faible poids de naissance et de naissance prématurée chez les nourrissons de sexe féminin et les enfants de mères moins instruites. Ces résultats suggèrent que même dans les environnements à faible exposition, les normes actuelles de qualité de l'air ne protègent peut-être pas suffisamment les populations vulnérables. Le renforcement des avis de pollution pour les femmes enceintes et l'affinement des politiques régionales en matière de qualité de l'air pourraient contribuer à atténuer ces risques.

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# 1 Introduction

It is now widely accepted that genetics alone cannot explain health at birth. Since the formulation of the fetal origins hypothesis (Barker 1990), there has been increasing interest in the economic field in assessing the impact of in utero health shocks for two main reasons. First, given the importance of early life conditions in predicting later human capital outcomes (e.g., Almond et al. 2005; Black et al. 2007; Bharadwaj et al. 2017, 2018), prenatal health shocks could significantly impact society's future well-being. Second, understanding the impact of adverse health shocks in utero is crucial to evaluating the benefits of interventions designed to reduce them. From this perspective, a vast body of economic literature has documented the negative impact of in utero health shocks, including air pollution, on children's health at birth (e.g., Chay and Greenstone 2003; Currie et al. 2009; Currie and Walker 2011; Sanders and Stoecker 2015; Simeonova et al. 2021). However, some important pollutants have not yet received substantial attention.

This paper focuses on the impact of fine particulate matter ($PM_{2.5}$) on children's birth outcomes. $PM_{2.5}$ is particularly concerning due to its microscopic size, which allows it to penetrate deep into the human body and potentially cross the placental barrier, affecting fetal development. Furthermore, its small size allows it to infiltrate buildings more easily than other pollutants, reducing the protective effect of indoor air (e.g., Cyrys et al. 2004). Despite its hazardous nature, the causal impact of $PM_{2.5}$ on the early stages of child development, particularly at birth, is understudied in economics.[1]

This study aims to estimate the effect of $PM_{2.5}$ on children's birth outcomes using data from the province of Quebec, Canada. Our study uses the universe of births in Quebec between 2008 and 2015, combined with detailed air pollution data. Quebec provides a compelling setting for this study for several reasons. First, unlike the United States, health care in Quebec is predominantly

[1] In fact, the causal relationship between $PM_{2.5}$ and health is well established for either adult health (e.g., Deryugina et al. 2019; Ward 2015) or cognition (e.g., Archsmith et al. 2018; Ebenstein et al. 2016; Graff Zivin and Neidell 2012; Heyes et al. 2016). The only attempts to establish a causal link between $PM_{2.5}$ and child health are more or less inferential. For example, Alexander and Schwandt (2022) exploits the increase in the number of cars that do not meet emission standards (cheating diesel car scandal in 2015) in US counties and shows that an additional cheating car per 1,000 cars increases the low birth weight and infant mortality rates by 1.9 and 1.7 percent, respectively, while $PM_{2.5}$ levels among other pollutants increase by 2 percent.

publicly funded, eliminating financial barriers to access. Second, pollution levels in Canada are relatively low compared to the United States and significantly lower than in countries like China. Consequently, the marginal costs of reducing pollution may or may not exceed the associated marginal benefits, so documenting the latter is important.

Our understanding of the specific levels at which air pollution, particularly $PM_{2.5}$, affects health remains limited. To our knowledge, only Palma et al. (2022) attempt to address this question by comparing the effects of exposure to days that exceed the thresholds of the Italian and World Health Organization for coarser particulate matter (PM10) in Italy. Regarding $PM_{2.5}$, Li and Zhang (2024) provide more direct evidence of its causal effects on neonatal health. However, their findings are based on a setting with significantly higher levels of pollution than those typically experienced in the developed world. Jahanshahi et al. (2022) consider a context close to our own in terms of universal health coverage, but they say very little about the effect of exposure to the extensive margin.

Our main novelty is that we question this threshold effect. Regulatory standards for air pollution are often set based on the statistical distribution of pollution concentration instead of health effects. We ask what are the health effects of an additional day of exposure to high pollution, measured either at the yearly threshold or the daily threshold set by public health authorities. In so doing, our study could be informative about how sharp spikes in pollution could influence birth outcomes, a question we believe is important, especially since we consider a setting of low air pollution levels. Furthermore, we measure exposure at the trimester and throughout pregnancy levels, allowing us to examine the relationship between fetal development stages and $PM_{2.5}$ exposure.

Estimating the causal effect of air pollution on health presents challenges due to endogeneity concerns, particularly residential sorting. Families may self-select into neighborhoods based on amenities, which could introduce unobserved confounders. In our context, we find that pollution exposure is correlated with pre-determined family characteristics, suggesting potential bias. Prior literature (Graff Zivin and Neidell 2013) suggests that in most developed regions, this sorting bias attenuates estimated pollution effects toward zero. To address this, we employ a two-way fixed-effects approach, controlling for postal code-by-month and week-of-birth fixed effects. This strategy ensures that comparisons are made within small geographic areas over time, leveraging plausibly exogenous variation in pollution exposure. Additionally, we control for weather conditions, sociodemographic factors at the neighborhood level, and detailed socioeconomic indicators from tax records. As a robustness check, we estimate a mother fixed-effects model, which controls for time-invariant family characteristics that could influence birth outcomes.

To address this problem, we use a two-way fixed-effects estimation that controls for unobserved confounders at the postal code-by-month and week of birth levels. Our approach leverages sharp variations in pollution levels that are not correlated with a comprehensive set of family background characteristics. In addition, our preferred specification controls for weather variables and their squared terms, sociodemographic variables at the zip code level, and detailed socioeconomic variables from tax records. Importantly, we are also able to compare our main results to those using a mother fixed effects approach, which by design keeps stable families' unobserved time-invariant preferences.

Our findings indicate that, on average, $PM_{2.5}$ exposure in utero does not significantly affect birth outcomes at the population level. However, we uncover important heterogeneous effects: female infants and those born to less-educated mothers experience significantly worse outcomes when exposed to high pollution levels. Specifically, a 10-unit increase in $PM_{2.5}$ concentration during pregnancy raises the likelihood of low birth weight and preterm birth more for female infants than for males. Additionally, infants born to mothers without a university education are 29% more likely to have low birth weight and 26% more likely to be born prematurely under similar exposure conditions. Notably, short-term exposure to pollution spikes (exceeding the daily threshold) has a stronger adverse effect than long-term exposure above the yearly threshold. This suggests that interventions aimed at limiting short-term pollution surges could yield disproportionate benefits for vulnerable populations.

Our paper extends the analysis by examining the effects on particularly vulnerable infants. We replicate our analysis using medical data from children born in or admitted to a Neonatal Intensive

Care Unit (NICU) in Quebec. Although average levels of pollution concentration do not show significant effects, exposure to days of high pollution has a pronounced impact. An additional day of exposure to pollution levels that exceed the annual threshold increases the risk of very low birth weight by 60% and low 5-minute Apgar scores by an average of 31%. These large effects are potentially due to the fact that children in NICUs are sicker on average. This paper contributes to the debate on the potential benefits of further investment in efforts to reduce pollution even at relatively low levels.

The rest of the paper proceeds as follows. Section 2 provides a medical background on the mechanisms. Section 3 presents the data. Section 4 presents our estimation approach. Section 5 presents the results. Section 6 discusses some threats to the validity of our conclusions. Section 7 puts the size of the effects in perspective. Section 8 concludes the paper.

## 2 Biological pathways

$PM_{2.5}$ refers to tiny particles in the air that are 2.5 micrometers in diameter or smaller (for comparison, a human hair has a diameter of 70 micrometers). These particles come from various sources, including vehicle exhaust, industrial emissions, construction sites, and even wildfires. $PM_{2.5}$ is particularly concerning because it can penetrate deep into the lungs and enter the bloodstream. The particles are made up of different substances, some of which are more toxic than others, such as heavy metals and organic compounds.[2]

Exposure to $PM_{2.5}$ during pregnancy can significantly affect birth outcomes, either directly or indirectly through mother's health. When pregnant women breathe in $PM_{2.5}$, it can cause inflammation in their bodies. This inflammation can cross the placenta and reach the fetus, potentially damaging its development. Inflammation and oxidative stress caused by $PM_{2.5}$ can damage cells and disrupt normal growth, which could in theory lead to issues such as low birth weight and preterm birth (e.g., Brook et al. 2010; Glinianaia et al. 2004).

[2] For instance, Jedrychowski et al. (2017) show that Polycyclic Aromatic Hydrocarbons (PAH), a component of $PM_{2.5}$, mediate all the effects of $PM_{2.5}$ on birth outcomes in an experimental setting in Poland.

$PM_{2.5}$ also contains harmful substances that can disrupt hormones necessary for a healthy pregnancy. This can affect how the placenta develops and functions, which is critical for providing nutrients and oxygen to the fetus. Poor placental function can result in growth restrictions and other birth complications (e.g., Janssen et al., 2017).[3]

Exposure to $PM_{2.5}$ can affect fetal health and growth in different ways, depending on its timing. In the first trimester, the fetus organs form, so exposure can cause congenital problems and affect overall growth. In the second trimester, the fetus grows rapidly and develops essential functions. Exposure during this phase can harm lung development and restrict growth. In the third trimester, the fetus gains weight and matures. Exposure at this later stage can lead to preterm birth and low birth weight as the placenta can struggle to provide adequate nutrition and oxygen (e.g., Veras et al., 2008).

Studies have shown that exposure to $PM_{2.5}$ can also lead to maternal health problems such as hypertension, which can further affect fetal development and increase the risk of complications such as preeclampsia (e.g., van den Hooven et al., 2011).

While studies like ours are necessary to inform the causal relationship between in utero exposure and birth outcomes, they cannot disentangle the mechanisms. In addition, very little is known about the specific levels of $PM_{2.5}$ at which these effects become significant. If the relationship between $PM_{2.5}$ exposure and birth outcomes is highly non-linear, the effects could differ significantly in low-pollution versus high-pollution settings.

# 3 Data

Our birth outcomes are derived from birth certificates, which contain detailed demographic data on the parents and the exact location of residence of the mothers during pregnancy. We are able to match mothers in our sample to tax files and education records, allowing us to include precise socioeconomic status as a control in our regressions. We determine exposure to pollution during

[3] This pathway is actually similar to one of the expected effect of smoking.

pregnancy on the basis of average readings from air pollution monitors located near the mother's home during pregnancy. We augment our set of controls by including meteorological variables that come from monitor readings, as well as neighborhood characteristics derived from the 2006 and 2016 Canadian Censuses of Population.

## 3.1 Birth record data

Our analysis uses the Canadian Vital Statistics Database (CVSD) for births between 2008 and 2015. This dataset contains valuable information on parental demographic characteristics (such as residence, age, place of birth, and marital status) and childbirth outcomes (including exact date of birth, sex, birth weight, gestational age, and parity). Consistent with most previous studies, our primary health variables are birth weight, gestational age, low birth weight (an indicator of birth weight below 2.5 kg), and preterm birth (an indicator of birth before 37 weeks of gestation).

In addition to these health variables, we include "small for gestational age" (SGA), which is defined as birth weight below the 10th percentile by sex and gestational age for all births between 2008 and 2015. To limit the influence of congenital and birth defects, we restrict the sample to children weighing at least 500 grams and with a gestational age of at least 26 weeks (i.e., having completed at least two trimesters).[4] This restriction leaves us with approximately 702,000 births, or about 87,750 births per year.[5]

We use a confidential version of the CVSD that allows us to link each mother in our sample to her tax and postsecondary education records for the year of maternity, using her social insurance number.[6] Therefore, we are able to include in our regressions a series of variables related to socioeconomic status, both at the individual and family level, such as income and unemployment benefits, which have been shown to be associated with poor neonatal health (see Kramer (1987)).

[4] Information on congenital abnormalities or birth defects is not included in the birth certificates.

[5] This is similar to the 88,436 births in Quebec in 2010. See https://statistique.quebec.ca/en/document/births-quebec.

[6] This confidential version is part of a data integration project called "The Impact of Preterm Birth on Socioeconomic and Educational Outcomes of Children and Families (IPB)" and is available at the Canadian Research Data Centre Network.

We are able to link over 97% of our observations.

Our main regression sample consists of births from mothers who reside within a 10 km radius of an air pollution station monitored by the Quebec Ministry of Environment during their pregnancy. We match mothers to the nearest pollution monitor using the geographic coordinates of the centroids of their postal codes and those of the pollution monitors. This proximity-based approach results in a 60% reduction in our sample size.

Table 1 presents the means of key variables for both the original sample and the regression sample. The majority of pollution monitors are located in urban settings, resulting in a selected sample of births. However, the results are still representative of more densely populated areas, where changes in pollution levels are likely to impact a greater number of births.

Panel A shows the means of the birth outcomes. We observe that all birth outcomes are better for the children in the regression sample. For example, the incidences of low birth weight and prematurity are 1% lower on average for children born to mothers living closer to an air pollution monitor.

However, panel C reveals worse outcomes in the positive determinants of health within the population of mothers who reside within 10 km of an air pollution monitor. This suggests that the observed improvements in neonatal health in the regression sample are more likely due to the overrepresentation of singleton births. Mothers living near a pollution monitor represent a subsample of older mothers who gave birth between 2008 and 2015. These mothers are also much more likely to be immigrants (born outside of Canada) and to live in low-income neighborhoods. This aligns with the fact that immigrants tend to reside in urban areas where pollution is more likely to be both monitored and high. In addition, these mothers are more likely to be legally married, reflecting the higher prevalence of common law unions among native populations.

Table 1: Sample means

| | All births | Regression sample |
|---|---|---|
| Number of observations | 679,800 | 250,930 |
| **Panel A: Outcomes**: | | |
| Birth weight in grams | 3,348.14 | 3,369.46 |
| | (547.03) | (516.07) |
| Gestation age in weeks | 38.85 | 39.01 |
| | (1.84) | (1.65) |
| Low birth weight (LBW) | 0.05 | 0.04 |
| | (0.23) | (0.20) |
| Very LBW | 0.008 | 0.005 |
| | (0.09) | (0.07) |
| Preterm | 0.07 | 0.06 |
| | (0.26) | (0.23) |
| Small for gestation age (SGA) | 0.10 | 0.10 |
| | (0.30) | (0.31) |
| **Panel B: Air pollution exposure**: | | |
| Average $PM_{2.5}$ in utero | - | 9.54 |
| | - | (1.71) |
| Number of days above $10\mu g/m^3$ in utero | - | 33.12 |
| | - | (10.55) |
| Number of days above $27\mu g/m^3$ in utero | - | 1.71 |
| | - | (1.43) |
| **Panel C: Demographics**: | | |
| Child Male | 0.513 | 0.513 |
| | (0.50) | (0.50) |
| Mother age | 29.74 | 30.70 |
| | (5.10) | (5.19) |
| Mother married | 0.39 | 0.54 |
| | (0.49) | (0.5) |
| Mother is born in Canada | 0.78 | 0.58 |
| | (0.42) | (0.49) |
| Mother is university educated | 0.23 | 0.23 |
| | (0.42) | (0.42) |
| Family income in 1st quartile | 0.25 | 0.22 |
| | (0.43) | (0.42) |
| Low income neighborhood | 0.21 | 0.29 |
| | (0.40) | (0.45) |

*Notes:* The regression sample consists of all singleton births during 2008 and 2015 which have no missing values for pollution, weather and SES variables. Standard errors are in parentheses.

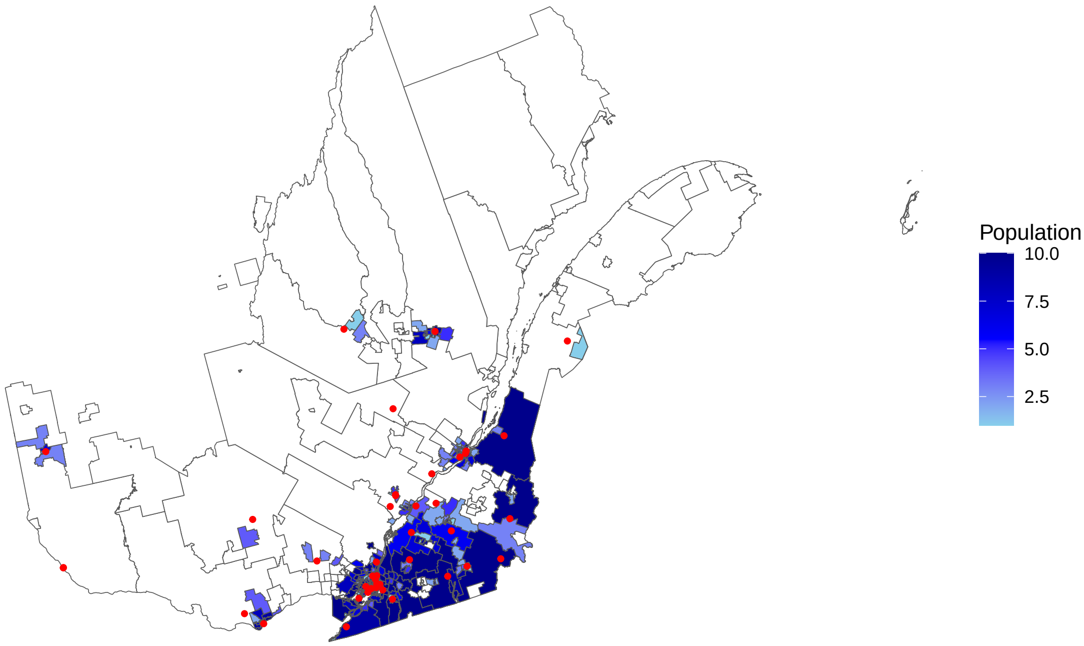


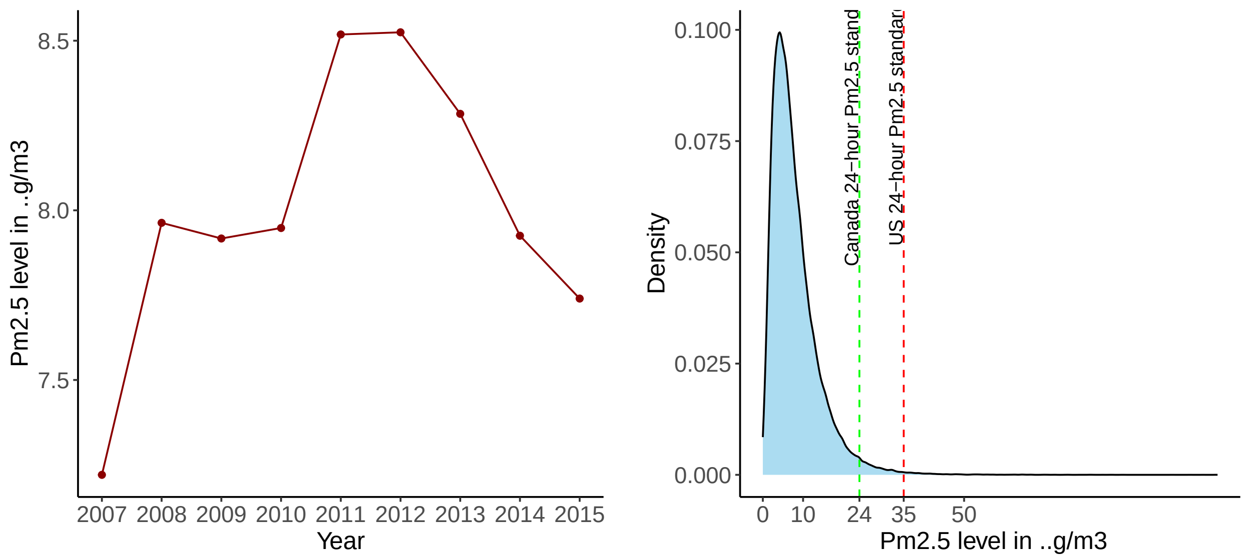

Figure 1: Location of $PM_{2.5}$ monitors in Québec

*Notes*: The figure shows the distribution of $PM_{2.5}$ monitors in the province of Quebec. It shows that monitors are concentrated in populated areas.

## 3.2 Air pollution data

We obtain our pollution data from the Info-Air program of the Quebec government. This program collects air quality data from stations in Quebec. Because the raw data consists of hourly monitor readings, we aggregate them to daily levels. The air quality monitoring network consists of 42 monitors measuring $PM_{2.5}$ concentrations across the province. Figure 1 shows their locations and shows that they are concentrated in populated areas that include the major cities of Montreal, Laval, and Quebec City.

Figure 2 shows the annual and daily concentration of $PM_{2.5}$ in the province for the period 2007-2015. It first shows an upward trend in $PM_{2.5}$ levels from 2007 to 2011, followed by a downward trend thereafter. Panel b shows the daily concentration of $PM_{2.5}$. It shows that the average daily concentration is around 10 $\mu g/m^3$, far from the 35 $\mu g/m^3$ 24-hour US standard for $PM_{2.5}$. In addition, this threshold is almost never reached. Quebec is therefore an ideal context in which to study whether low levels of pollution still have an effect on health.




(a) Trend in $PM_{2.5}$ in Quebec over years

(b) Distribution of the daily $PM_{2.5}$ concentration

Figure 2: Annually and daily $PM_{2.5}$ in Québec

*Notes*: Panel (a) shows the annual evolution of $PM_{2.5}$ concentration in the province of Québec throughout our study period, while Panel (b) illustrates the daily distribution of $PM_{2.5}$ during the same period.

We also plot the aggregated $PM_{2.5}$ in the province per trimester during our study period. In Figure 3, we present this for the monitor readings in the cities of Montreal and Quebec. We observe a similar seasonal pattern in both cities. Specifically, each increase is followed by a decrease, and almost every first quarter is marked by an increase in pollution levels. This is not surprising, given that winter heating and fireplaces are a major source of $PM_{2.5}$ emissions.


Figure 3: Seasonality of $PM_{2.5}$ level

*Notes*: The figure plots the mean of $PM_{2.5}$ concentrations over the monitors in the province by quarter of year for the period 2007-2015.

*Construction of Exposure Measure.*—In the main analysis, we approximate the locations of the mothers by the centroid of the Forward Sortation Area (FSA), which is defined by the first three characters of the postal code and is equivalent to a census tract in population (but not boundaries). We then construct a 10-km buffer around each FSA centroid. Finally, we average all the monitor readings within this buffer, weighting them by the distance of each monitor from the centroid, to assign pollution levels to each location (and implicitly to the mothers in these locations). However, this approach introduces measurement error in the attribution of pollution (Graff Zivin and Neidell 2013). This measurement error may arise from the fact that the centroid-based approximation does not perfectly capture the actual residential location of mothers, especially in larger or more diverse FSAs. In addition, pollution levels can vary significantly within the same FSA, depending on proximity to pollution sources such as highways or industrial areas. As a result, the exposure measure might incorrectly reflect the true levels of pollution experienced by each mother, potentially leading to attenuation bias in estimating the effects of pollution on birth outcomes. Despite these limitations, this method represents the best balance between accuracy and feasibility given the available data. Alternative approaches to reducing measurement error, such as reducing the buffer size around the FSA centroid, would likely improve the precision of pollution exposure estimates but would come at the cost of a significantly smaller sample size. Such a trade-off could limit the power of the analysis, making the current approach the most practical and effective option under the circumstances.

Our data also allow us to use the exact geographical coordinates of the mothers (longitude and latitude) recorded on birth certificates.[7] Unfortunately, calculating the average exposure for each individual is computationally intensive and challenging due to the limitations of the computer capacity of the data center. Instead, we draw a 5% random sample and compare the results using both the location approximation and the exact location in Section 6.

## 3.3 Weather data

Our weather data are extracted from Environment Canada's weather information gateway.[8] Various weather stations across the country monitor weather conditions. We limit our search to stations in the province of Quebec for the period 2007-2015. We then use the longitude and latitude of each station to match them to the nearest air pollution monitor within a 30 km radius.[9]

The monitors report data on hourly or daily wind direction, wind speed, temperature (in Celsius), humidity, precipitation, and dew point. However, in our analysis, we only include weather variables that are systematically reported across all stations. These variables are wind direction and speed, temperature, and humidity. Although we retain only observations that do not have missing values, our findings remain invariant even when controlling for missing values.

[7] One advantage of birth certificates in Canada is that they report exact postal codes as well as the associated geographic coordinates.

[8] It can be accessed from `https://climate.weather.gc.ca/historical_data/search_historic_data_e.html`.

[9] A finer proximity would result in a significant loss of observations, while the gain in accuracy would be negligible since weather conditions are much the same in finer proximity.

# 4 Econometric strategy

Identifying the causal relationship between pollution exposure and health is challenged by the nonrandom assignment of pollution exposure. An important source of this phenomenon is the residential sorting of people based on unobserved preferences for attributes or characteristics of neighborhoods.[10] For example, wealthier families may want (and can afford) to live in greener neighborhoods with lower crime rates and better schools. They are therefore systematically exposed to different levels of pollution. To check whether this sorting is present in our sample, we run different regressions of pollution levels during pregnancy against different mothers' characteristics. Table 2 shows that older mothers, mothers living in low-income neighborhoods, and unemployed mothers are exposed to higher levels of $PM_{2.5}$ during pregnancy, suggesting an important role for residential sorting in our sample.

[10] See Graff Zivin and Neidell (2013) for an in-depth discussion of this issue.

Table 2: Evidence of residential sorting.

| | **Mother's age** | **Father absent** | **University** | **Child Male** |
|---|---|---|---|---|
| in utero $PM_{2.5}$ ($\mu g/m^3$) | 1.844$^{***}$ | 0.0153$^{***}$ | 0.0303 | 0.0178$^{***}$ |
| | (0.4667) | (0.0058) | (0.0029) | (0.0059) |
| Number of days above year stand. | 0.1219$^{***}$ | 0.0012$^{**}$ | −0.0024 | 0.0005 |
| | (0.0453) | (0.0005) | (0.0020) | (0.0008) |
| Number of days above the day stand. | 0.0288$^{***}$ | 0.0002$^{**}$ | 0.0005 | 0.0003$^{***}$ |
| | (0.0070) | (0.0001) | (0.004) | (0.0001) |
| Observations | 251000 | 251000 | 251000 | 251000 |
| Mean dep. | 31 | 0.030 | 0.23 | 0.51 |
| | **Low inc. area** | **Working** | **Low income** | **High income** |
| In utero $PM_{2.5}$ ($\mu g/m^3$) | 0.4116$^{***}$ | −0.2095$^{***}$ | 0.3038$^{***}$ | −0.1319$^{***}$ |
| | (0.0870) | (0.0305) | (0.0065) | (0.0424) |
| Number of days above year stand. | 0.0209$^{***}$ | −0.0130$^{***}$ | 0.0176$^{***}$ | −0.0085$^{***}$ |
| | (0.0057) | (0.0023) | (0.0036) | (0.0029) |
| Number of days above the day stand. | 0.0062$^{***}$ | −0.0032$^{***}$ | 0.0047$^{***}$ | −0.0020$^{***}$ |
| | (0.0014) | (0.0005) | (0.0008) | (0.0007) |
| Observations | 251000 | 251000 | 251000 | 251000 |
| Mean dep. | 0.29 | 0.80 | 0.28 | 0.22 |

*Notes:* The regressions do not include any controls. The errors are clustered at week-by-year and FSA levels.

Statistical significance: $^{*}$p<0.1; $^{**}$p<0.05; $^{***}$p<0.01.

To estimate the effect of exposure to $PM_{2.5}$ in utero on health at birth net of residential sorting, we employ a two-way fixed effects model where we control for weather conditions and precise family SES. Our model takes the following form:

$$y_{int} = \beta PM2.5_{nt} + X_i^{'}\delta + \gamma Weather_{nt} + Z_{nt}^{'}\theta + \lambda_t + \phi_{nt} + \varepsilon_{int}, \tag{1}$$

where $i$ indexes the child, $n$ indexes the neighborhood which consist of the geographical area formed by the first three characters of the postal code, $t$ indexes the time period (week of birth or month of birth), $\phi$ denotes an interaction between month-year and neighborhood fixed effects, and $\varepsilon$ is an error term. Our outcomes of interest, denoted by $y$, are the health of the child $i$ at birth (birth weight, gestational age, low birth weight, very low birth weight, small for gestational age and prematurity). $PM_{2.5}$ represents the average level of $\mathrm{PM}_{2.5}$ during pregnancy, or the number of days during pregnancy when $\mathrm{PM}_{2.5}$ exceeded the daily threshold ($27\mu g/m^3$) or the annual threshold ($10\mu g/m^3$). To avoid endogeneity of gestation duration, we take the average of the last 38 weeks before the child's birth.[11]

Our regressors of interest, $PM2.5_{nt}$, are indexed by FSA and week of birth, meaning that children born in the same FSA and during the same week will be assigned the same exposure level. We do so for ease of computation, which is a relevant concern when working in a secure environment where computers capabilities are limited.We control for weather conditions, $Weather_{nt}$, during pregnancy in our regressions. These variables include temperature, dew point, wind speed, wind direction, and average humidity during pregnancy. Given the potential non-linearity of the relationship between weather and health, our weather controls include quadratic terms in these variables.

The vector $X_i^{'}$ contains characteristics specific to the child and his family. We improve on previous works that restrict their controls to characteristics only present in birth certificates such as child gender, mother age, mother's education, immigration status by adding family total income derived from tax files.[12] We provide a complete list of our controls in Table A1.

The level of health and pollution in a specific area can be determined by economic activity and the general characteristics of the area's inhabitants. For example, poorly educated families may have poor health outcomes and live in an area with high pollution. This is why $Z_{nt}^{'}$ contains characteristics of people living in a specific FSA. These include the population size, the proportion of

[11]Taking the average over the median (or modal) gestation length in the sample is quite common in the literature (e.g., Palma et al. 2022).

[12]For example, most previous work proxy income variables with the median income in a particular census tract block (e.g., Currie et al. 2009).

immigrants, the proportion of homeowners, and the median property value. All of this information comes from the 2006 and 2016 Canadian censuses.[13]

We control for seasonal correlation between infant health outcomes and air pollution level by including fixed effects for the week of birth, $\lambda_t$. We allow this correlation to be different between neighborhoods by including an interaction between the month of birth and FSA fixed effects, $\phi_{nt}$.

To explore the effect of quarter-of-birth exposure, we estimate a version of equation (1) in which the pollution and weather variables are measured at the quarterly level. This specification is defined as:

$$y_{int} = \sum_{s=1}^{3} \beta^s PM2.5_{nt} + X_i^{'} \delta + \sum_{s=1}^{3} \gamma^s Weather_{nt} + Z_{nt}^{'} \theta + \lambda_t + \phi_{nt} + \varepsilon_{int}, \tag{2}$$

where $s$ indexes each pregnancy trimester. Weather variables are also included at the quarterly level to account for the correlation between weather patterns and fine particle formation in each quarter. Therefore, $\beta^s$ captures the estimated effect of air pollution exposure during the $s$-th quarter on birth outcomes.

*Inference.*— We cluster the standard error at the FSA level to take into account spacial correlation in the error term. However, since exposure varies at the FSA-by-week level, we also report standard errors clustered at the same level.

*Source of variation and identification assumptions*.— Our approach is similar to a difference-in-difference's estimation. With fixed effects for the week of the year, we compare children born at the same time, but in different FSA. The identifying variation could come from the fact that a specific location experiences a random shock to the level of pollution, such as road closures, different patterns in residential heating, and the closure (or opening) of a factory. Similarly, with month-by-FSA fixed effects, we look at sharp variations coming from before and after a random shock to the level of pollution.

In Figure 4, we repeat the same exercise as in Figure 3 except that we also plot the residual vari-

[13] For births between 2006 and 2010, we use the 2006 census and the 2016 census for births between 2011 and 2015. We do not use the 2011 census due to an important change in sampling methodology.

ation after eliminating the effects of weather, week, and FSA. We see that the level and variation of pollution between quarters are considerably reduced. Interestingly, when we compare the residual variation between a more polluted city (Montreal) and a less polluted city (Quebec City), we find that the residual variation is lower in Montreal. This could suggest that the driving forces behind pollution in a highly polluted environment are more dependent on season, weather conditions, and economic activities. We argue that the variation left is as good as random. Table 3 shows that the inclusion of the weather and FSA controls and fixed effects take care of most of the differences in the characteristics of the mother's background.

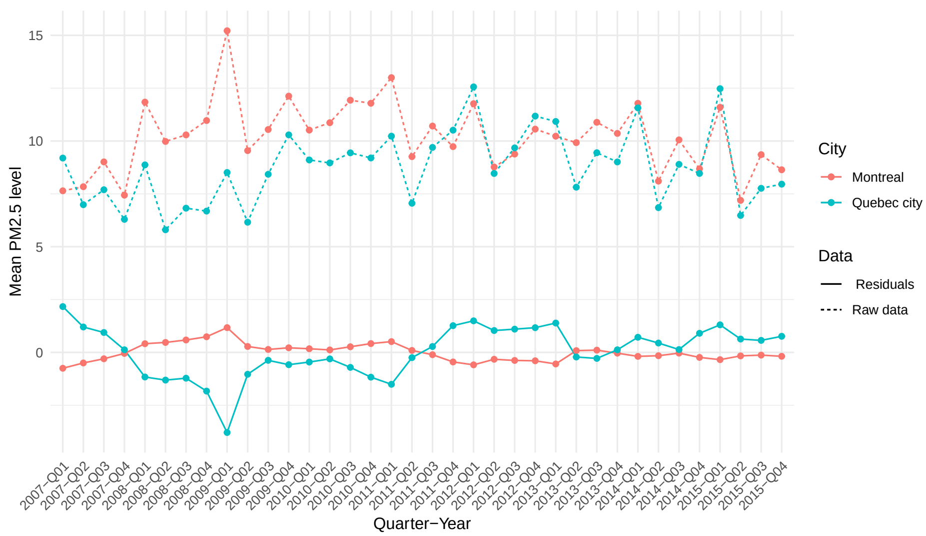



Figure 4: Raw vs. Residual Pollution

*Notes*: The figure plots the mean of $PM_{2.5}$ concentrations over the monitors in the province by quarter of year for the period 2007-2015.

Table 3: Balancing checks.

| | **Mother's age** | **Father absent** | **University** | **Child Male** |
|---|---|---|---|---|
| in utero $PM_{2.5}$ ($\mu g/m^3$) | $-1.186^{*}$ | $-0.0018$ | 0.0303 | 0.0882 |
| | (0.6878) | (0.0186) | (0.0475) | (0.0545) |
| Number of days above year stand. | $-0.0074$ | $-0.0001$ | $-0.0001$ | 0.0005 |
| | (0.0076) | (0.00035) | (0.0006) | (0.0008) |
| Number of days above day stand. | 0.0378 | $-0.0006$ | $-0.0035$ | $-0.0004$ |
| | (0.0338) | (0.0009) | (0.0022) | (0.0029) |
| Observations | 251000 | 251000 | 251000 | 251000 |
| Mean dep. | 31 | 0.030 | 0.23 | 0.51 |
| | **Low inc. area** | **Working** | **Low income** | **High income** |
| in utero $PM_{2.5}$ ($\mu g/m^3$) | 0.0109 | $-0.0093$ | $0.1601^{**}$ | $-0.0188$ |
| | (0.0459) | (0.0406) | (0.0631) | (0.0585) |
| Number of days above year stand. | 0.0005 | 0.0001 | 0.0002 | 0.0001 |
| | (0.0006) | (0.0006) | (0.0007) | (0.0007) |
| Number of days above the day stand. | 0.0008 | $-0.0019$ | 0.0037 | $-0.0015$ |
| | (0.0019) | (0.0017) | (0.0028) | (0.0024) |
| Observations | 251000 | 251000 | 251000 | 251000 |
| Mean dep. | 0.29 | 0.80 | 0.28 | 0.22 |

*Notes:* We include weather controls, as well as FSA levels controls. We also include week of birth and month-by-FSA fixed effects. Errors are clustered at week-by-year and FSA levels.

Statistical significance: $^{*}$p<0.1; $^{**}$p<0.05; $^{***}$p<0.01.

# 5 Results

## 5.1 Baseline Results

Table A2 (see Appendix) presents estimates of the effects of air pollution on neonatal health, using both average $PM_{2.5}$ concentration during pregnancy and the number of days exceeding regulatory thresholds. Across different specifications, we find mostly null and imprecise effects, except for the likelihood of being small for gestational age (SGA). This suggests that in utero exposure to $PM_{2.5}$ may restrict fetal growth. However, since this effect is not significant when using the alternative exposure measure (number of days exceeding thresholds), we remain cautious about which regulatory standard is more relevant. Although we do not find significant effects on other birth outcomes, sensitivity analyses suggest that our econometric strategy corrects for attenuation bias. Notably, prior to including our fixed effects, $PM_{2.5}$ appears to improve birth outcomes (particularly for gestational age and very low birth weight). However, after including our full set of fixed effects, these effects disappear, demonstrating the importance of accounting for neighborhood sorting. Further evidence from Table 2 supports this, showing that higher-SES mothers tend to reside in areas with better air quality, potentially biasing estimates downward.

*Trimester specific analysis*.— When considering trimester analysis, the results are similar. We find no detectable effects of air pollution whatever the way we measure it.[14]

## 5.2 Subgroups Analysis

The imprecision of the null effects presented above may mask effects within specific subgroups. To explore this, we interact our exposure measures with whether the child is male, whether the mother is university-educated, and the family income quartile (first quartile, second quartile, third quartile). We run those regressions separately including our full sets of controls and fixed effects.

These subgroups will shed light on two important empirical questions.

1. Are male fetuses more vulnerable to in utero exposure to $PM_{2.5}$ as suggested by the 'fragile

[14] Table not included, but available upon request.

male' hypothesis (e.g., Kraemer 2000)?

2. To what extent can health behaviors potentially correlated with SES explain the sensitivity to exposure to in utero air pollution?

We present the interaction of group indicators with each of our regressors of interest in Figure 5. Each panel corresponds to one of the subgroup categories (Female infants and mothers who are not university-educated), while each row represents one of the three exposure measures: average exposure during pregnancy, number of days above the annual threshold, and number of days above the daily threshold. The x-axis represents the estimated coefficients, with confidence intervals displayed as horizontal bars.

*Male vs female.*—Our findings suggest that male infants exposed to high levels of $PM_{2.5}$ have better outcomes than female infants. This appears to contradict the "fragile male" hypothesis. Although we cannot distinguish between various explanations, such as a lower intrinsic vulnerability of males to $PM_{2.5}$ or differences in maternal health behaviors for those carrying female children, our findings point towards a natural selection of stronger male fetuses as a potential driver.[15] In the model of gestation length (column 2), we find that male infants have shorter gestation periods despite being born heavier (column 1). Furthermore, the trimester-specific analysis indicates that the sex difference is primarily driven by exposure during the third trimester. Together, this could suggest that male fetuses that reach the third trimester (or simply with a longer gestation age) may be less vulnerable to air pollution. More strikingly, we find that exposure during the second trimester increases the likelihood of very low birth weight for males relative to females, which is more in line with the fragile male literature.

*Less educated vs high educated mothers.*—We find that the effects of in utero exposure to $PM_{2.5}$ are concentrated among children whose mothers did not attend university. Specifically, an additional 10-unit increase in average $PM_{2.5}$ during the entire pregnancy is associated with shorter gestation duration and higher probabilities of preterm births and low birth weight for children of less educated mothers. This heterogeneity along the education margin could be driven by differ-

[15] Li and Zhang (2024) find similar results in rural China.

ences in health behavior between highly educated and less educated mothers which could amplify the effect of air pollution (Currie, 2011). For example, highly educated mothers might be more aware of behaviors that promote a healthy fetal environment, such as quitting smoking or seeking prenatal care early in the pregnancy. This is suggested by the trimester-specific analysis. When considering the effect of the number of days with pollution levels above the daily standard (in Table A7), we find that the effect is mostly driven by exposure during the second trimester (and in the first trimester in some cases).

*Wealthier vs less wealthy.*— Whether we consider income at the individual level or at the census tract level, we do not find heterogeneity in the effect along that margin (we do not include it in the table).

We intentionally left the effects of the number of days above the standard out of the above discussion, since Table A7 depicts the same pattern as Table A6. Recall that our aim in introducing these measures of exposure is to compare the response of birth outcomes to the yearly threshold vs. the daily threshold. That is why we report the estimates of the coefficient on the interaction between days above both standards and the group indicator in Table A7 (first row vs. second row of each panel). Interestingly, we find that being exposed to an additional day above ($27\mu g/m^3$) has a greater effect than being exposed to days above lower levels ($10\mu g/m^3$) in the two groups.

## 5.3 Additional Birth Outcomes

Our analysis so far has omitted some critical birth outcomes, such as the Apgar score, due to their inaccessibility in vital statistics on births. To provide additional evidence on these outcomes, we use medical data from infants admitted to the Neonatal Intensive Care Unit (NICU) in Quebec City (namely the CHU de Québec). It is important to note that this sample overrepresents very premature infants (gestation less than 33 weeks).[16] Thus, our regressions include controls for the severity of the newborn's condition, such as delivery mode (i.e., C-section or vaginal), the DRG

[16] An important limitation of this data is that it does not contain mother-level characteristics except for the postal code. For interested readers, we recommend the paper by Beltempo et al. (2023), which offers more details about the data.

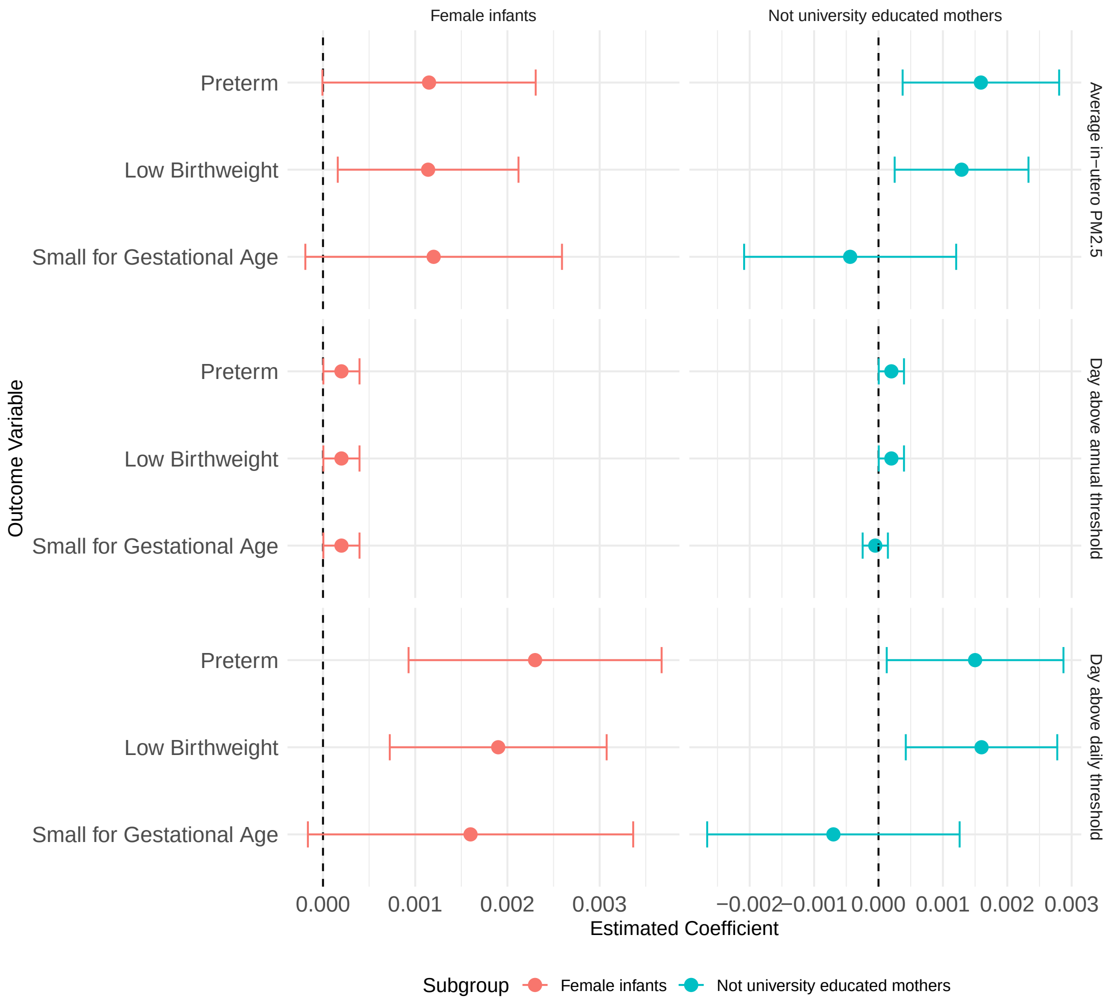



Figure 5: Effects of air pollution within subgroups

*Notes*: This figure plots the coefficients on the interaction term between our exposure measures and indicators of subgroups (Female and mothers who are not university-educated).

severity index, and the length of gestation.[17]

Table 4 provides the estimated impact of in utero exposure to $PM_{2.5}$ on birth weight and the likelihood of a 5-minute Apgar score below 7. We do not find any detectable effect of average air pollution on any of the birth outcomes. Interestingly, the standard errors are of similar magnitude to the point estimates, suggesting a precise null effect. However, regarding the effects of exposure to high pollution days, we find significant effects of days above the yearly $10\mu g/m^3$ threshold. Specifically, we find a strong effect of an additional day of exposure on the likelihoods of very low birth weight (61%) and a low 5-minute Apgar score (34%).[18] While these effects may initially appear large, the lack of an effect on the probability of low birth weight suggests that air pollution exposure may predominantly affect the lighter newborns (the 25% of babies born weighing less than 2.5 kg)[19], i.e., the sickest infants.

[17] Thus, we are indirectly examining whether in utero exposure influences intra-uterine growth restriction.

[18] Although we only report results from our preferred specification, we also ran regressions without fixed effects. Table A10 reports the results. We find that our fixed effects correct an attenuation bias, confirming the patterns in Table A2.

[19] See Appendix Table A9 for summary statistics.

Table 4: In utero exposure and child at birth : NICU sample

| Dependent Variables: | Birth Weight | LBW | VLBW | Apgar< 7 |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| *Variables* | | | | |
| in utero $PM_{2.5}$ | -476.4 | -0.0267 | 0.1614 | 0.4440 |
| | (502.2) | (0.1438) | (0.1508) | (0.2690) |
| Days above yearly threshold | -20.94 | 0.0055 | $0.0355^{**}$ | $0.0343^{*}$ |
| | (24.19) | (0.0099) | (0.0146) | (0.0175) |
| Days above daily threshold | 7.372 | -0.0010 | -0.0005 | 0.0031 |
| | (5.482) | (0.0065) | (0.0028) | (0.0056) |
| mean dep. | 2,996.416 | 0.252 | 0.058 | 0.102 |
| Controls | Yes | Yes | Yes | Yes |
| Zip X Month | Yes | Yes | Yes | Yes |
| week-by-year | Yes | Yes | Yes | Yes |
| Observations | 2,417 | 2,417 | 2,417 | 2,417 |

Notes: Controls include child sex, delivery mode, DRG severity index, weather and zip code characteristics variables as in Table A2. Standard errors are clustered at zip code and week-by-year level.

Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

# 6 Threats to validity

Measurement error is often cited in the literature along with residential sorting as a threat to the identification of the effect of air pollution on health. As we discussed above, our econometric strategy allows us to control for residential sorting. However, measurement error could arise either because mothers engage in avoidance behavior or because assignment of exposure based on approximation of people's FSA does not reflect individual exposure, either because of lack of precision or (unobserved) moves during pregnancy, or both.

### 6.1 Avoidance behavior

Avoidance behavior, if it exists, is not problematic in our context since we find no effect on average.[20] However, because we find an effect among low-educated mothers, we should interpret avoidance behavior as indicating that less educated mothers may not afford such preventive measures, possibly because they lack the necessary information. This is an interesting point, especially in the context of a universal health system where people are theoretically equal in access to health irrespective of their income.[21]

### 6.2 Measurement error in pollution

Mothers may have moved during their pregnancy introducing an error in their residence during pregnancy. In Quebec, residential leases expire at the end of June, so most people move on July 1 of each year if they have to. Consequently, the residential information of mothers whose pregnancy does not overlap with July 1 is more likely to be accurate. We check the robustness of our results using this sample of mothers.

Because we approximate locations based on the centroid of the census tract, air pollution exposure could still be measured with error. As mentioned in Section 3, we have precise geographic coordinates of mothers.[22] We draw a 5% random sample of mothers and assign exposure based on readings of monitors close to their exact locations.[23] The results are generally invariant to these different exercises.[24]

[20] It could also be that avoidance behavior in our context biased the results towards zero.

[21] As we have shown, there is no effect in subgroups based on income.

[22] Using this as the main analysis would be the best-case scenario. However, as mentioned above, we were unable to do this due to technical problems in the data center.

[23] We follow the same steps as for the centroid analysis. That is, we match the home address to the nearest air pollution monitoring stations within a 10-km radius. In the event of a tie, we use the average readings of the monitors.

[24] The corresponding tables are not included in this draft but are available upon request.

### 6.3 Residual unobserved preferences

Another source of endogeneity is the residual unobserved preferences of the mothers. A more convincing way to address this challenge is to compare the birth outcomes of children born to the same mothers but exposed to different levels of in utero air pollution. Our data contain mother identifiers, enabling us to introduce mother fixed effects in our regression analysis. Including both week-by-year and mother fixed effects in regression 1 would eliminate all variation in exposure, so we follow the approach of Currie and Neidell (2005). We estimate equation 1 with mother fixed effects but without week-by-year fixed effects.[25] Consistent with our earlier findings, we observe a null and highly imprecise effect of exposure in utero to $PM_{2.5}$.

## 7 Discussion of the magnitude of the effects

In this section, we compare the effects of in utero exposure to $PM_{2.5}$ among less educated mothers in the context of the existing literature on the effects of $PM_{2.5}$. We also discuss the policy implications of these findings.

Table A6 shows that a 10-unit increase in the average levels of $PM_{2.5}$ during pregnancy leads to a 1.29 percentage point increase in the probability of low birth weight (LBW) for children born to less educated mothers. This represents a 29% increase in the mean probability of LBW. The same increase in $PM_{2.5}$ corresponds to a 26% increase in the mean probability of preterm births. Furthermore, we find that an extra day with $PM_{2.5}$ levels above the daily threshold during pregnancy shortens gestation by 0.02 weeks, corresponding to a reduction of 0.05% in the mean gestational age. Although we do not find a detectable effect on continuous measures of birth weight, the same increase in exposure increases the likelihood of low birth weight by 0.16 percentage points (3.56%) and preterm birth by 0.15 percentage points (2.57%). The most pronounced effect is on the likelihood of very low birth weight, which increases by 0.4 percentage points (80%).[26] These

[25] The regression equation takes the following: $y_{int} = \beta PM2.5_{nt} + X_i^{'}\delta + \gamma Weather_{nt} + Z_{nt}^{'}\theta + MothersFEs + \phi_{nt} + \varepsilon_{int}$. For identification, we restrict the sample to mothers who gave birth at least twice during the study period.

[26] Significant at the 10% level.

findings suggest that efforts to maintain levels of $PM_{2.5}$ at reasonable levels could have significant positive effects on newborn health and that it could be important to focus on avoiding extreme concentration levels, even when average exposure remains low.

To provide context, we compare our findings with existing studies that estimate the causal relationship between in utero exposure to $PM_{2.5}$ and neonatal health. Although the literature is limited, two notable papers consider one of our key outcomes: low birth weight. For ease of comparison, we calculate the elasticity of $PM_{2.5}$ levels and low birth weight, representing the percentage increase in LBW due to a 1% increase in $PM_{2.5}$ levels. Our results suggest that a 1% increase in in utero exposure yields a 0.27% increase in LBW. In contrast, Alexander and Schwandt (2022) document an elasticity of 0.95 in the United States and Li and Zhang (2024) find an elasticity of 3.03 in rural China.[27]

Our findings differ significantly from those reported in these studies. A possible explanation could be the difference in baseline exposure levels. For example, in the study from China, the mean exposure level during a nine-month pregnancy is 2.5 times higher than the mean exposure in our setting. A naive calculation suggests that this exposure yields an almost eleven-fold higher response in neonatal health.[28] However, these calculations should be interpreted with caution, as they do not account for differences in healthcare systems, socioeconomic status between settings, or the nature of the particles that make up $PM_{2.5}$.

Furthermore, the significant impact of an additional day above the daily $PM_{2.5}$ threshold highlights the importance of daily monitoring. Short-term increases in $PM_{2.5}$ levels could be less predictable and could disproportionately affect vulnerable groups, making consistent monitoring and regulation crucial to protecting neonatal health.

[27] Another relevant study is Jahanshahi et al. (2022), which finds no effect of $PM_{2.5}$ exposure on low birth weight in Northern Ireland.

[28] This suggests non-linearity in the dose-response function.

# 8 Conclusion

This paper provides new evidence on the role of air pollution in shaping neonatal health. Specifically, we examine the case of fine particulate matter ($PM_{2.5}$), which, despite being a significant health risk due to its ability to penetrate the bloodstream, is relatively understudied. Using comprehensive data on all births in the province of Quebec, Canada, from 2008 to 2015, and leveraging a two-way fixed effects model, we estimate effects that are less likely to be biased by residential sorting and measurement errors.

In our main analysis, we find null and imprecise average effects of in utero exposure to $PM_{2.5}$ on neonatal health. However, significant results emerge when we examine the data by infant sex and maternal education. First, we find that female infants exposed to high levels of $PM_{2.5}$ in utero are more susceptible to adverse birth outcomes than male infants exposed to the same levels. This finding challenges the "fragile male hypothesis." We attribute this discrepancy to a screening effect, where stronger male fetuses are more likely to survive, particularly since the effect is driven by exposure during the third trimester. Second, we observe that infants born to less educated mothers are more vulnerable to high pollution levels. Specifically, exposure to days of high pollution significantly increases the likelihood of adverse birth outcomes such as low birth weight, shorter gestational age, and preterm births among these infants. These findings suggest a mediating role for health behaviors among more educated mothers, especially during the early stages of pregnancy, since exposure during the second trimester predominantly drives the results. In addition, we investigate the effects among infants born in or admitted to a Neonatal Intensive Care Unit (NICU) in Quebec. We find no detectable effect when measuring exposure as the average concentration of $PM_{2.5}$ throughout pregnancy. However, the results change when we consider exposure as the number of days with pollution levels exceeding the yearly standard. In this case, we find that exposure to in utero significantly increases the likelihood of very low birth weight and low 5-minute Apgar scores. It is important to note a caveat in relation to our interpretation: Because we do not control for other pollutants, we should interpret the results as the effects of overall air pollution rather than the specific effects of $PM_{2.5}$.

From these results, we draw two main policy conclusions. First, while pollution in a low-exposure setting like ours may not pose a significant threat to the general population, policies should target disadvantaged groups who are more vulnerable to its effects. Second, short-term increases in pollution are particularly harmful, especially for vulnerable groups. These spikes are of greater concern because poor health at birth is a significant predictor of poor future outcomes. In general, our study highlights the importance of monitoring air quality and implementing targeted policies to protect at-risk populations, thus improving newborn health outcomes and promoting long-term well-being.

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