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DISCUSSION PAPER SERIES

IZA DP No. 15464

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ABSTRACT

Prenatal Exposure to $PM_{2.5}$ and Infant Birth Outcomes: Evidence from a Population-Wide Database

There are growing concerns about the impact of pollution on maternal and infant health. In the UK in 2018, 36% of local authorities had levels of PM2.5 where exposure exceeded the annual level recommended by the World Health Organisation at the time. Using a population database of births in Northern Ireland linked to localised geographic information on pollution in mothers' postcodes (zip codes) of residence during pregnancy, we examine whether prenatal exposure to PM2.5 is associated with a comprehensive range of birth outcomes. Overall, we find little evidence that particulate matter is related to worse infant outcomes once we implement a fixed effects approach that accounts for time-invariant factors common to mothers. While reducing pollution remains an urgent public health priority, our results imply that improvements in short-run levels of prenatal PM2.5 exposure are unlikely to be sufficient by themselves to reduce disparities in birth outcomes.

JEL Classification: I10, J10, Q53

Keywords: pollution, $PM_{2.5}$, infant outcomes, sibling fixed-effects, birth weight

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1 Introduction

Climate change, a growing literature on the associations between air pollution and adverse outcomes, and an awareness of growing health disparities associated with differential environmental exposures, have combined to generate increasing interest in empirically estimating how exposure to particulate matter and other pollutants affect population health (Currie et al. 2014). In the US alone, almost 50 million people live in counties where levels of exposure exceed national air quality standards (Sullivan and Krupnick 2018). Exposure to fine particulate matter with a diameter of 2.5μm or less ($PM_{2.5}$) has one of the biggest impacts of particulate pollution on human health; according to a Committee on the Medical Effects of Air Pollutants, a 10μg/m^3 increase in ambient $PM_{2.5}$ concentration is associated with a 6% increase in all-cause mortality (Committee on the Medical Effects of Air Pollutants 2021). Estimates of the economic impact of environmental exposure that are based on calibrated models of mortality risk imply that global health-related losses are of the order of trillions of dollars annually (Yin et al. 2021). Moreover, exposure to $PM_{2.5}$ is higher for socioeconomically-disadvantaged communities and the impact on those with underlying health conditions is likely greater (Fecht et al. 2015; Brunt et al. 2017; Milojevic et al. 2017; Bowe et al. 2019). Consequently, in addition to its overall impact, particulate matter may contribute to reinforcing health inequalities.

One population that is particularly important to study in this context is pregnant women and infants. Despite recent reductions in maternal and early-life mortality in many higher income countries, outcomes during the period around childbirth may still be among those most susceptible to environmental conditions. Biological mechanisms through which the impact of pollution is likely to operate include gestational hypertension and placental health (Abraham et al. 2018; Luyten et al. 2018; Nobles et al. 2019). Moreover, previous studies that have directly examined the association between particulate matter and early life outcomes such as birth weight and infant mortality have found a negative relationship in a variety of contexts (Currie et al. 2009; Fu et al. 2019; Goyal et al. 2019). Reflecting the importance of interactions with the healthcare system around birth, health expenditures during this stage of life for both infants and mothers are amongst the highest for pre-retirement age-groups, for example with half of all Medicaid spending in the US occurring during this life stage (Xu et al. 2015). Life-saving treatments for premature and infants experiencing poor health represent extraordinary medical progress but are expensive (Almond et al. 2005), thus from a public policy point of view addressing risk factors for these outcomes, potentially including exposure to particular matter, remains a policy priority. In addition to impacting directly on the contemporaneous health of women and infants, reducing exposure to pollution may also have medium and long-term benefits (Sanders 2012). Improvements in early life environment as reflected in measures such as birth weight, which $PM_{2.5}$ is hypothesized to affect, have been linked to better adolescent and adult outcomes such as education, health, and earnings (Almond and Currie 2011). An emerging literature has found evidence of intergenerational impacts of infant health, including for pollution (Colmer and Voorheis 2020).

Quantifying the relationship between particulate matter and early life outcomes is challenging because of the potential for omitted factors to bias estimates. Given we expect pollution exposure to be associated with other background characteristics, further evidence on how exposure to pollutants causally affects these outcomes is important for informing public policy. Air quality can be thought of as a local amenity (Chay and Greenstone 2005), therefore we expect the cost of living, for instance through wage and house prices within neighbourhoods that are differentially affected, to adjust to reflect differing exposure. Without an empirical strategy to adjust for such factors that may be difficult to measure directly, estimates

from correlational studies could be biased. Assessing the magnitude of the relationship between air pollution and early life outcomes is important, because if there is no effect of $PM_{2.5}$ exposure per se, or relevant effect sizes are small, alternative strategies such as increasing access to ante-natal services and addressing social determinants such as material hardship and education may be additionally required to improve maternal and infant health and reduce disparities. Studies that adopt an identification strategy to account for unobserved confounders in estimating the impact of $PM_{2.5}$ on infant outcomes are often focused on the US (e.g. Currie et al. 2014).

Our approach in this paper is to use administrative birth records from Northern Ireland (NI) over a 7-year period (2011-2017), with outcome information linked to prenatal pollution exposure to $PM_{2.5}$. We make a number of contributions to the literature. First, we consider a range of measures of infant health and birth outcomes beyond just mortality and birth weight, which together provide a comprehensive assessment of short-run infant morbidity. Second, the scale of the data and time horizon allow us to link infants born to the same mothers living in NI, but with different levels of exposure to particulate matter. Our empirical strategy is to implement a mother fixed effects approach, which adjusts for all time-invariant factors common to siblings born to the same parent. We identify pollution exposure using postcode of residence, allowing us to capture highly localised $PM_{2.5}$ measures for the population of mothers, while still adjusting for socioeconomic characteristics of census tracts. This is an important advantage of the data given concerns about omitted variable bias at the local level. Finally, the location in which we study the impact of pollution on infant outcomes is also important, as our analysis takes place in the context of a universal healthcare system (the UK's National Health Service) that is largely free at the point of use, which stands in contrast to much of the previous literature (which we summarise below).

The rest of this paper is structured as follows. In Section 2, we review previous literature to contextualize the paper. In Section 3 we discuss our methodological approach. Section 4 describes our data and the NI context, while results are presented in Section 5. Section 6 provides a discussion and Section 7 concludes. Further analysis is presented in the Appendix.

2 Literature

This paper examines a range of outcome measures for infant health using an approach designed to account for omitted variable bias. In what follows we first summarise findings from analyses that consider birth outcomes other than birth weight (the most commonly studied outcome). We then summarise studies, including for birth weight, which examine the impact of pollution using an empirical approach with an explicit identification strategy, with a focus on the economics literature, noting that in some cases the most relevant studies are for pollutants other than $PM_{2.5}$.

APGAR Score

Several papers have examined APGAR scores as an outcome of pollution. In an analysis of shale gas development in the US state of Pennsylvania (Hill 2018), mother's residential proximity to a gas well was associated with an increase in the probability of an APGAR score of less than 8 by 26 percent. Negative associations between APGAR scores and pollutants, including $PM_{2.5}$ have also been documented in China (Wei et al. 2020) and Poland (Wojtyla et al. 2020). In France, carbon monoxide was associated with both APRGAR score and head circumference (Gomez et al. 2005).

Head Circumference and Infant Length

In their review and meta-analysis, Fu et al. (2019) investigated the relationship between air pollutants NO_2 and $PM_{2.5}$ and birth outcomes including head circumference and length. $PM_{2.5}$ exposure ($10\mu g/m^3$) during the entire pregnancy was negatively associated with head circumference at birth (β = − 0.30 cm, 95% CI − 0.49, − 0.10), and NO_2 exposure during the entire pregnancy was significantly linked to shorter length at birth (β = − 0.03 cm, 95% CI − 0.05, − 0.02).

Placental Outcomes

The placenta plays a crucial role in pregnancy, and is one of the hypothesised biological mechanisms through which impacts of pollution are expected to operate. Initially, the placenta functions to suppress the mother's immune system, preventing rejection of the embryo. Subsequently, the placenta transfers oxygen, nutrition and other factors necessary for supporting growth from the mother to the foetus, as well as waste material from the foetus to the mother. Because the placenta comes into contact with substances that both mother and foetus are exposed to over the course of a pregnancy, the impact of particulate matter may be apparent here first. Moreover, the placenta regulates signals affecting the developmental process, including progesterone and other hormones, which play a role in determining appropriate responses to environmental exposures. Nanoparticles, such as those found in air pollution, can pass through the placental barrier, and the smallest (less than 240 nanometres) can enter the foetal bloodstream (Wick et al. 2010). In their review, Luyten et al. (2018) discuss a range of potential pathways through which pollution exposures may affect foetal growth and other outcomes via the placenta, including direct DNA damage and epigenetic expression.

A number of empirical studies support these mechanisms using data from Europe (Janssen et al. 2012; van den Hooven et al. 2012; Abraham et al. 2018; Nawrot et al. 2018; Giovannini et al. 2020), Brazil (Hettfleisch et al. 2017), and Japan (Michikawa et al. 2016). In the US, researchers used the Rhode Island Child Health Study to investigate the association between $PM_{2.5}$, black carbon, and the expression of 10 imprinted genes in the placenta, and birth weight (Kingsley et al. 2017). $PM_{2.5}$ and black carbon were associated with changes in expression of 41 and 12 of 108 placental imprinted genes, respectively, which implies possible impacts on foetal growth and development.

Literature with an identification strategy

Table 1 summarises recent papers in the economics literature that examine the relationship between pollution exposures and infant outcomes (or, in one case, later life outcomes of in utero exposure) and which adopt an explicit identification strategy. Of the 14 papers listed, six adopt a (parental or area) fixed effects (FE) approach, six use an instrumental variables (IV) approach (e.g. exploiting variation in weather conditions or the business cycle, some in combination with FE), and four adopt a difference-in-differences (DID) approach (e.g. exploiting regulatory changes). Seven of these studies take place in the US, and infant outcomes analysed in these papers only include mortality, gestational age, preterm birth and birth weight. Although not all studies find statistically significant impacts in all cases, overall these papers suggest a negative impact of pollution exposure on these outcomes. In terms of magnitude the implied relationships are generally important, for instance in a paper that used data from California, every 1% reduction in PM_{10} exposure was associated with a 1% reduction in weekly infant mortality (Knittel et al. 2016). Overall, Table 1 highlights the need for further research that considers additional contexts outside the US and examines outcomes beyond mortality and birth weight or gestational age, evidence that this paper provides.

Table 1 Summary of Literature with Identification Strategies on the Impact of Pollution on Infant Health Outcomes

Authors/Journal	Population	Identification strategy	Pollutant/Exposure duration	Outcome	Effects sizes
Chay, K., Greenstone, M., 2003, QJE	US counties from 1980 to 1982	County FE. IV using stacked first-differences, with changes in mean TSP (average total suspended particulate pollution) and per capita income instrumented by first and second lags of TSP and income levels.	TSP (average total suspended particulate pollution) Exposure: changes in TSP.	2500 fewer infants died during the recessions from 1980–1982 than would have in the absence of reductions in TSP.	A 1 $\mu g/m^3$ reduction in TSP resulting in about 4–7 fewer infant deaths per 100,000 live births (an elasticity of 0.35).
Currie, J., Neidell, M., & Schmieder, J. F. 2009, JHE	New Jersey 1990s, mothers' residential location	Mother FE, interaction effect with smoking (controlling for neighbourhood FE too, therefore using variation in pollution exposure within families between children)	CO exposure by trimester	Birth weight, infant mortality. CO has negative impacts on infant health both before and after birth.	A one unit change in mean CO during the last trimester of pregnancy increases the risk of low birth weight by 8%. A one unit change in mean CO during the first 2 weeks after birth increases the risk of infant mortality by 2.5%.
Currie J., & Walker R., 2011, AEJ:AE	Pennsylvania for 1997 to 2002, and New Jersey for the years 1994 to 2003	DID exploiting the introduction of electronic toll collection (EZ-Pass) in 98 regions	Emissions cause by traffic congestion (e.g., CO)	Policies intended to curb traffic congestion can have significant health benefits for local populations	EZ-Pass reduced the incidence of prematurity and low birth weight in the vicinity of toll plazas by 6.7–9.1 percent and 8.5–11.3 percent, respectively
Sanders, N. J. 2012, JHR	Texas, from 1981 to 1983	IV: instruments for TSP using annual employment changes in the county-level manufacturing	Annual mean average TSP weighted by the inverse distance	A statistically significant relationship between prenatal pollution exposure and educational outcomes,	A standard deviation decrease in TSP is associated with approximately a 6 percent of a within-county

		sector, exploiting a period of industrial recession in Texas from 1981 to 1983 and its impact on manufacturing production and associated ambient TSP levels.	from pollution monitors. All pollution monitors within 20 miles of a county were considered.	specifically performance on standardized high school exit exams.	standard deviation increase in test performance.
Coneus, K., & Spiess, C. K., 2012, JHE	Germany, from 2002 to 2007	Area and family FE using the German Socio Economic Panel (SOEP).	CO, SO_2, NO_2, Ozone, PM_{10} mean pollution exposures during pregnancy. Also mean pollution exposures in month before birth.	CO impacts fetal growth and birth weight. Ozone levels impact birth length, fetal growth, and the probability of toddlers having a bronchitis or some other health condition.	An increase in the average CO exposure during the month before birth lowers birth weight by, on average, 289 g. The impact on birth weight and fetal growth towards the end of pregnancy appears to be significantly higher than at earlier stages.
Luechinger, S., 2014, JHE	Germany, from 1985 to 2003	FE and IV: (Natural experiment created by the mandated desulfurization at power plants, and power plants' location and prevailing wind directions)	SO_2 Exposure: Annual mean SO_2 concentration measured at air quality monitors for 1985–2003	Desulfurization at power plants resulted in considerable benefits for infant health.	The resulting instrumental variable estimates imply a marginal effect of SO_2 on infant mortality of 0.045. Between 25 and 44 percent of the decrease in infant mortality over the sample period is estimated to be due to the improvement in air quality.
Greenstone, M., & Hanna, R., 2014, AER	India (city level panel data) 1986–2007	DID using air pollution regulation in India	PM, SO_2, NO_2	The catalytic converter policy is associated with a reduction in the infant mortality rate.	Reduction in the infant mortality rate of 0.64 per 1,000 live births. However, this estimate is imprecise and is not statistically significant.
Tanaka, S., 2015, JHE	China	DID approach, based on an the air pollution regulation "Two	Overall pollutant emissions	Air pollution regulations led to reductions in infant mortality	Infant mortality decreased by 20%

	(145 counties from 1991 to 2000)	Control Zones" (TCZ) policy implemented in some Chinese counties.		within the TCZ cities subjected to particularly stringent regulations.	
Knittel, C. R., Miller, D. L., & Sanders, N. J., 2016, REStat	California, from 2002 to 2007	IV: Interactions between mobile source emissions and local weather conditions as a source of exogenous variation in pollution.	PM_{10} and CO Exposure: Vector of average pollution levels for the first, second, and third trimesters of gestation.	PM_{10} has a statistically and economically significant impact on weekly infant mortality. CO has a large negative effect but is not statistically significant.	A 1 unit decrease in PM_{10} saves roughly 10 lives per 100,000 live births, an elasticity of approximately 1; a standard deviation increase in traffic results in a 0.2% of a standard deviation increase in infant deaths. Effects are largest for premature and low birthweight infants.
Arceo et al., 2016, EJ	Mexico City for the years 1997–2006.	IV: number of thermal inversions in a given week to instrument for pollution levels that week.	PM_{10}, SO_2, CO, Ozone Exposure: weekly measures of pollution for each of the 56 municipalities in Mexico City	The adverse effects of pollution on infant mortality in Mexico City is statistically significant.	1 ppb increase in CO over a week leads to a 0.0046 per 100,000 births increase in the infant mortality rate. A 1µg/m3 increase in PM_{10} leads to an increase in the mortality rate of 0.23 per 100,000 births.
Cesur et al., 2017, EJ	Turkish provinces between 2001 and 2011.	IV: exploiting variation in the timing of deployment and intensity of expansion of natural gas infrastructures	PM_{10}	Expansion of natural gas services has led to a significant reduction in the rate of infant mortality.	1pp increase in the rate of subscriptions to natural gas services would lower the infant mortality rate by 4%
Hill, E. L., 2018, JHE	Pennsylvania, from 2003 to 2010.	DID model in which mothers living within 2.5km of a shale gas well or permit site before drilling are used as a control for	Shale gas development as a potential pollution source.	Extensive margin: Babies born to mothers who lived within 2.5km of at least one gas well during pregnancy	A statistically significant increase in low birth weight of 1.36 pp and a reduction in term birth weight of 49.58g,

		those exposed after drilling began – to estimate the impact of exposure to shale gas development on birth outcomes.		experienced adverse birth outcomes. Intensive margin: Each additional well drilled within 2.5km of the mother's residence increases low birth weight and premature birth by 0.3 percentage points and reduces term birth weight by 5g.	on average. No statistically significant effect for premature birth.
van den Berg, G. J., Paul, A., & Reinhold, S., 2020, LabEcon	Sweden, from 1992 to 2004	Parental FE	No direct pollution measure was used, but it is hypothesised that economic downturns are negatively associated with pollution.	Economic downturns are beneficial to the health outcomes of newborn infants in developed countries. Air pollution decreases during downturns.	A one-percentage- point increase in the unemployment rate is associated with an approximately 10% reduction in the incidence of having a birth weight below 1500 grams and of dying within 28 days after birth.
Alexander, D., Schwandt, H., 2022, Restud	US counties from 2007 to 2015	County and time FE. Exploiting the introduction of cheating diesel cars in the US by Volkswagen.	PM2.5, PM_{10}, Ozone	Birth weight, Gestational age, Preterm birth, and Infant mortality	Additional cheating diesel car per 1,000 cars increases $PM_{2.5}$, PM_{10}, and Ozone by 2, 2.2, and 1.3%, respectively, while the low birth weight rate and infant mortality rate increase by 1.9 and 1.7%, respectively.

3 Methods

In this paper, we model birth outcomes as a function of $PM_{2.5}$ exposure during pregnancy, where exposure levels are spatially matched to mothers via their home postcode at the time of birth. Postcode is a low-level geographic identifier comparable to zip code in the US, and NI has over 61,800 postcode units, with an average of 20 addresses per unit (NISRA 2021). As described in the next section, $PM_{2.5}$ is the average level in that location over the course of a calendar year. However, we also consider models adjusting for time of birth during the year. Following Currie et al. (2009) we estimate regression models of the following form:

$$Infant\ Outcome_{imp} = \beta PM2.5_p + Weather_p\delta + X_{imp}\gamma + \varepsilon_{imp}$$

Where the outcome of interest for infant i born to mother m in location p, is a function of $PM_{2.5}$ location-level exposure, location-level weather conditions, and a vector of control variables (X_{imp}) that includes a constant. ε_{imp} is an idiosyncratic error term. We begin with a model that adjusts for month and year of birth, background sociodemographic characteristics (mother's age, employment status, relationship status, smoking status, number of previous pregnancies (gravida) and child sex) and weather (temperature and rainfall in location of residence). We are unable to control for postcode fixed effects as the anonymisation procedure did not permit having access to these identifiers, but in later models we do include controls for neighbourhood characteristics at the Super Output Area (SOA) level which roughly correspond to census tracts in the US. SOAs are geographical boundaries designed to comprise local populations of similar sizes. NI is split into 890 SOAs, each with an average of 2,100 residents and 818 households (NISRA 2019). In our final set of models we include mother fixed effects that control for all factors common to infants with the same mother. The main coefficient of interest is β, which captures the adjusted association between $PM_{2.5}$ exposure and the outcome of interest. By comparing how this coefficient changes as additional model controls are added we can learn about the structure of confounding variables. For example, by including SOA-level fixed effects we can assess whether estimates of the impact of pollution are confounded by shared local-level factors such as material disadvantage. Additionally, by including mother fixed effects, we are able to test whether estimates for β are affected by unobserved factors, which are likely important for the reasons described above. In our model with mother fixed effects, parameter estimates are identified by variation in prenatal pollution exposures for siblings to the same mother.

In our main models, we use a weighted average of birth and conception year $PM_{2.5}$, where the weights reflect gestation time during each year. We measure this pollution exposure using categorical indicator variables for average $PM_{2.5}$ exposure based on the following intervals: 3-6, 6-10, and 10-16 $\mu g/m^3$. We are particularly interested in exploring how being in the category above the recommended limit for $PM_{2.5}$ set by the World Health Organization at the time (10 $\mu g/m^3$), subsequently halved in the 2021 revised guidelines, is related to infant outcomes. However, we also consider specifications where we included additional categories as well as modelling the postcode average $PM_{2.5}$ as a more continuous measure based on the median of shorter intervals. Doing so allows us to assess the functional form of potential relationships between pollution and our outcomes.

4 Data and Context

We use data on births in Northern Ireland from 2011 to 2017. Northern Ireland is a nation within the UK located on the island of Ireland. Unlike the Republic of Ireland with which it shares a land border, as part of the UK the health system in Northern Ireland is run as a component of the National Health Service (NHS) which is largely free at the point of access. Economically, living standards in Northern Ireland are broadly comparable to the rest of the UK, with median annual earnings for full time employees of GBP29,000 in 2021 (UK median GBP31,000).

The birth data used in this paper are collected as part of the NI Maternity System (NIMATS). NIMATS compiles information on mothers and infants from demographic and clinical data collected during delivery, supplemented with data from ante-natal check-ups and the post-natal period, and has been used elsewhere to study birth outcomes, e.g. Mongan et al. (2019) and Saad et al. (2021). The main source of data for NIMATS (excluding data input) is the Patient Administration System (PAS). The PAS provides mainly demographic details recorded when the mother attended for her booking appointment and also data recorded on admission to hospital for delivery. The NIMATs data are available for research purposes to accredited researchers and can be accessed through the Honest Broker Service, a Trusted Research Environment for Health and Social Care (HSC) NI which is hosted within the HSC Regional Business Services Organisation (RBSO/BSO). It is possible to merge NIMATS data with confidential identifiers provided that the ultimate analysis dataset does not permit identification disclosure. For this project, we merged NIMATS data with $PM_{2.5}$ annual pollution in each postcode in each year using mother's address at the time of birth. To preserve confidentiality, $PM_{2.5}$ levels were merged into categories that did not permit identification of individuals. Other weather variables were merged in the same way. All other variables used in this analysis are present in the original NIMATS database, including outcome and control variables for mothers. Patients within the UK's NHS, which in NI is run by the Health and Social Care (HSC) organisation, are assigned a unique Health and Care Number. This number is not observed by researchers; instead, mothers are assigned a unique anonymised ID which permits multiple pregnancies to the same mother to be identified. All data are prepared for analysis so that researchers do not have access to identifiable information, and all analysis took place onsite within the Honest Broker Service's 'Safe Haven' in Belfast. The NIMATS data include the following control variables that we adjust for in the analysis: mother's age, employment status (employed or not), relationship status (single parent or not), mother's smoking status (smoker or not), gravida, and child sex. We include additional controls for rainfall and temperature, as these factors have been shown to be predictive of health and wellbeing as well as pollution exposure (Buckley et al. 2014; Noelke et al. 2016; Kalisa et al. 2018).

The pollution and weather variables are derived from UK-wide datasets. The pollution data are published by the Department for Environment, Food and Rural Affairs (DEFRA) to provide policy support and to fulfil the UK's air quality reporting obligations. Annual average background $PM_{2.5}$ concentrations are modelled on a 1km x 1km grid and calibrated using measurements from sites within the national monitoring network. These data have been used in other studies, e.g., in the context of pregnancy outcomes in Scotland (see Dibben and Clemens (2015) and Clemens et al. (2017)). The weather data – annual mean air temperature (in degrees Celsius) and annual mean total precipitation (in millimetres) – are produced by the Met Office (2018) using interpolation techniques applied to data from a network of UK land surface observations. To transform the pollution and weather data from the grid to the postcode level (in order to match with mother's address), we carried out spatial interpolation (Kriging) using Geographic Information System (GIS) software.

Descriptive statistics for outcome, exposure and control variables are shown in Table 2. There were a similar number of births per year over the study period 2011-2017 (between 23,000 and 26,000). The modal age of mothers was in the 31-35 category (31%), with the vast majority in a relationship (94%), 75% employed, 15% who smoke, and 38% having three or more pregnancies. Just under one-fifth of the full sample of births experienced low average $PM_{2.5}$ exposure during pregnancy, while the majority (71%) experienced medium exposure; just under one-in-ten experienced the highest level of exposure. The average infant weighed 3,421 grams at birth and measured 51.5 centimetres in length and 34.7 centimetres in head circumference. Approximately 6.1% of infants were born below 2,500 grams (the classification for low birth weight). In terms of delivery, 7.6% of infants were delivered before 37 weeks, 0.4% were born still, and 14.5% were delivered through a crash, emergency or urgent caesarean. As expected, average outcomes in our data closely match comparable official statistics for NI during this period (PHA 2019). Compared to England, Wales and Scotland, a smaller proportion of babies in NI have low birth weight (PHA 2018). Around 10.5% of infants were resuscitated using either chest compression, a positive pressure (PP) tube or mask. A small minority of new-borns received an APGAR score below 7, with 7.8% receiving 1-minute score, and 1.7% receiving a 5-minute score, below this level. In terms of placenta outcomes, 14.6% were deemed to have an 'unhealthy' placenta, 8.7% had placental infarcts, 25.6% had a calcified placenta, and 1.7% had a retroplacental clot.

Appendix Figure A1 shows the distribution of $PM_{2.5}$ by SOA in 2017. In NI, the majority of the geographic area is rural with low population density, with only 36% of people in NI living in a rural area in 2018 (2020). The majority of the population is concentrated within the main cities of Belfast (the capital) and Derry/Londonderry and their metro areas. Rates of exposure to $PM_{2.5}$ are typically low outside of the cities, but Figure A1 shows considerable variation within urban areas. In Belfast, for example, pollution was concentrated in the downtown area and near major roadways. This, together with Figure A2 which shows that the highest-pollution areas also tend to be the most deprived according to NI's Multiple Deprivation Measure (MDM) (see NISRA 2010), highlights the need to adjust for location of residence at a disaggregated level, and motivates our inclusion of SOA fixed effects in our model estimates. Average population weighted $PM_{2.5}$ exposure has declined from almost 8$\mu g/m^3$ in 2011 to around 6$\mu g/m^3$ in 2017 (Figure A3). Figure A4 illustrates how the outcome variables have changed through time, with average outcomes in 2017 similar to their 2011 levels in most cases. However, mean birth weight decreased slightly during this period, while the proportion of babies with a 5-minute APGAR score of less than 7 increased. Mean head circumference and length both increased.

Table 2: Descriptive Statistics for Infant Outcomes, Exposure and Control Variables

	Mean	Frequency	N obs.
Outcome Variables			
Birth Weight (grams)	3421.4	*N/A*	168999
Low Birth Weight (<2,500g)	0.061	10302	168999
Resuscitated	0.105	17752	168781
1-minute APGAR Score <7/10	0.078	13213	168474
5-minute APGAR Score <7/10	0.017	2792	168486
Unhealthy Placenta	0.146	19012	130665
Placenta Infarcts	0.087	11318	130665
Placenta Calcified	0.256	33471	130665
Retroplacental Clot	0.017	2179	130665
Stillborn	0.004	634	169178
Preterm: Gestation <37 weeks	0.076	12828	169192
Crash/Urgent C-Section	0.145	24614	169198
Length (cm)	51.5	*N/A*	163366
Head Circumference (cm)	34.7	*N/A*	165593
$PM_{2.5}$ Category			
[3-6]	0.195	31348	160544
(6-10]	0.713	114439	160544
(10-16]	0.092	14757	160544
Mother/Infant Control Variables			
Mother aged <21 at birth	0.057	9582	169198
21-25	0.164	27829	169198
26-30	0.302	51026	169198
31-35	0.312	52860	169198
36 or above	0.165	27901	169198
Mother not single parent	0.943	157295	166719
Mother single parent	0.057	9424	166719
Mother not employed	0.255	42124	165232
Mother employed	0.745	123108	165232
Mother does not smoke	0.848	143261	169006
Mother smokes	0.152	25745	169006
1 Pregnancy	0.316	53516	169198
2 Pregnancies	0.302	51063	169198
3 or more pregnancies	0.382	64619	169198
Infant was female	0.487	82340	169166
Infant was male	0.513	86826	169166
Birth Month			
January	0.082	13913	169198
February	0.074	12581	169198
March	0.081	13767	169198
April	0.079	13435	169198
May	0.084	14144	169198

June	0.080	13519	169198
July	0.088	14882	169198
August	0.088	14827	169198
September	0.090	15160	169198
October	0.087	14638	169198
November	0.084	14211	169198
December	0.083	14121	169198
Birth Year			
2011	0.138	23320	169198
2012	0.149	25276	169198
2013	0.143	24158	169198
2014	0.144	24438	169198
2015	0.144	24387	169198
2016	0.143	24258	169198
2017	0.138	23361	169198
Temperature Decile			
1 (Coolest)	0.065	10951	167814
2	0.114	19078	167814
3	0.126	21161	167814
4	0.116	19408	167814
5	0.119	20000	167814
6	0.107	17976	167814
7	0.104	17494	167814
8	0.095	16019	167814
9	0.084	14016	167814
10 (Warmest)	0.070	11711	167814
Rainfall Decile			
1 (Driest)	0.033	5535	167814
2	0.081	13659	167814
3	0.089	15002	167814
4	0.092	15369	167814
5	0.114	19205	167814
6	0.119	19960	167814
7	0.124	20869	167814
8	0.131	21933	167814
9	0.123	20662	167814
10 (Wettest)	0.093	15620	167814

Notes: Table reports means and frequencies of analysis variables and the number of non-missing observations. Sample is all births between 2011 and 2017, and calculations are performed over all available data. Data on retroplacental clot and placenta outcomes are not available from June 2016 onwards, resulting in a smaller number of observations. *N/A* denotes frequency not applicable to continuous variable.

We examine the bivariate association between our outcomes of interest and $PM_{2.5}$ to determine whether a relationship exists between pollution and infant health in our data before adjusting for other factors. We focus on measuring mean outcomes by pollution category (those used in our main regression model: 3-6, 6-10, and 10-16 $\mu g/m^3$). We expect to see a correlation in this analysis based on the findings from the existing literature described above (e.g. Fu et al. 2019). Table 3 shows these bivariate means. Broadly speaking, we see the expected relationship with pollution. For instance, babies exposed to pollution in the 3-6 $\mu g/m^3$ category had a low birth rate of 5.6%, compared to 7.7% for those born into the 10-16 $\mu g/m^3$ category. Similarly, 7.5% of babies in the first category had a 1-minute APGAR score of less than 7, compared to 9.0% in the 10-16 $\mu g/m^3$ category. The continuous outcomes of birth weight, length and head circumference also show the negative expected relationship. For instance, the mean birth weight for infants in the lowest $PM_{2.5}$ category is 3,466g, compared to 3,334g in the highest $PM_{2.5}$ category. However, the pattern for preterm births and crash/urgent c-sections is less clear, with some being less common at higher pollution levels in the bivariate analysis. The magnitude of some of these differences is large, for example a birth weight difference of 132g is similar to the gradient in birth weight previously documented between infants born to households in the lowest income quintile compared to the highest income quintile (McGovern 2013). However, for the reasons described above, it is important to adjust for other factors, both observed and unobserved, that may bias estimates of the relationship between $PM_{2.5}$ and infant outcomes. The following section presents these results from our regression analysis.

In terms of how we construct the analysis sample for the regression analysis, there were a total of 169,889 births over the study period. There were a small number of infants without an identification variable (420), who were excluded. We also excluded infants who appeared multiple times and had multiple values on the same variables (271). 8,654 infants had missing values on control variables and the $PM_{2.5}$ and weather variables, with the most missing information from the following three variables: family status (2,479 missing values), maternal employment status (3,966), and SOA (2,014). This resulted in potential analysis sample of n = 160,544. We use slightly different samples depending on the outcome. For post-natal outcomes, we restrict the sample to all live births where babies are born at term (37 or more weeks). For stillbirths, we consider all pregnancies, and for preterm and crash/urgent c-sections, we restrict the sample to live births. However, as described in the robustness check section, we verified that results were not sensitive to estimation using alternative samples.

Given that we implement mother fixed effects models, an important issue is whether there is sufficient sample size and variation in pollution exposure for siblings born to the same mothers. Appendix Table A1 presents additional summary statistics documenting this information. This table highlights the importance of using population-level data such as those we use in this paper because it provides a large enough sample size to justify within family comparisons of pollution exposure. We find that there are a substantial number of infants with differing pollution categories even though they were born to the same mother. For example, among the 8,488 mothers in the lowest pollution category, 4,910 (57%) fall into a higher category during their next pregnancy. Among the 3,209 in the highest category, 60.3% subsequently fall into a lower pollution category.

Table 3: Summary Statistics for Outcome Variables by $PM_{2.5}$ Category

	$PM_{2.5}$ Category								
	3-6			6-10			10-16		
	Mean	Freq.	N obs.	Mean	Freq.	N obs.	Mean	Freq.	N obs.
Birth Weight (grams)	3466.2	*N/A*	33159	3419.4	*N/A*	119210	3334.6	*N/A*	15242
Low Birth Weight (<2500g)	0.056	1873	33159	0.060	7184	119210	0.077	1175	15242
Resuscitated	0.101	3335	33096	0.106	12570	119083	0.114	1730	15215
1-minute APGAR Score <7/10	0.075	2471	33042	0.078	9293	118853	0.090	1365	15194
5-minute APGAR Score <7/10	0.016	519	33044	0.017	1973	118859	0.018	275	15198
Unhealthy Placenta	0.101	1612	15899	0.146	14597	99903	0.198	2713	13682
Placenta Infarcts	0.059	938	15899	0.088	8813	99903	0.111	1520	13682
Calcified Placenta	0.207	3289	15899	0.260	25937	99903	0.298	4077	13682
Retroplacental Clot	0.016	250	15899	0.017	1654	99903	0.019	255	13682
Stillborn	0.003	116	33195	0.004	452	119336	0.004	64	15259
Preterm: Gestation <37 weeks	0.076	2526	33199	0.075	8953	119350	0.082	1246	15259
Crash/Urgent C-Section	0.146	4841	33199	0.147	17491	119350	0.134	2052	15259
Length (cm)	51.6	*N/A*	32406	51.4	*N/A*	115118	51.4	*N/A*	14474
Head Circumference (cm)	35.0	*N/A*	32746	34.7	*N/A*	116733	34.5	*N/A*	14735

Notes: Sample is all births 2011-2017. *N/A* denotes frequency not applicable to continuous variable.

5 Results

We begin by showing results for birth weight in Table 4. As described above, we sequentially add control variables to assess how coefficient estimates change when accounting for potential confounders. The final model in column 4 includes mother fixed effects so parameter values are based on comparing siblings of the same mothers who faced different levels of $PM_{2.5}$. All models are linear regressions (OLS) for continuous birth weight as the outcome, and the sample includes all singleton live births of 37+ weeks gestation. $PM_{2.5}$ is included as a categorical variable with the reference category being 3-6 $\mu g/m^3$. Comparing the first two columns, adding a standard set of control variables for infant and mother variables and weather slightly reduces the magnitude of estimates of the association between $PM_{2.5}$ and birth weight. Experiencing $PM_{2.5}$ during pregnancy of 10-16 $\mu g/m^3$ compared to 3-6 $\mu g/m^3$ is associated with a reduction in birth weight of around 130g in the unconditional model. After controlling for gender and observable mother characteristics, this effect falls to 86g. Adding fixed effects for mother's SOA of residence further reduces the magnitude of the coefficients, although estimates remain statistically significant. Now being in the top $PM_{2.5}$ category is associated with a reduction in birth weight of around 32g. However, in the mother fixed effects models, the coefficient is attenuated still further and is no longer statistically significant. Full model coefficients are shown in Appendix Table A2. We conduct formal tests of changes in coefficients between mother fixed effects and other models to assess whether differences are statistically significant. Because computation was not feasible otherwise, we omitted the SOA fixed effects from this test. The test statistic was 1323.3 with a p-value of <0.001 suggesting that adjusting for mother fixed effects is important for our understanding of the relationships under consideration.

Table 4: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight

	(1)	(2)	(3)	(4)
$PM_{2.5}$ (ref. [3-6])				
(6-10]	-53.0***	-40.5***	-12.2**	1.1
	(3.2)	(3.7)	(4.4)	(5.9)
(10-16]	-129.9***	-86.3***	-31.6***	-3.0
	(5.3)	(6.0)	(7.4)	(10.4)
Observations	146662	146662	146662	146662
Outcome Mean	3525.5	3525.5	3525.5	3525.5
Controls				
Year and Month of birth	No	Yes	Yes	Yes
Gender	No	Yes	Yes	Yes
Mother variables	No	Yes	Yes	Yes
Weather variables	No	Yes	Yes	Yes
SOA FE	No	No	Yes	Yes
Mother FE	No	No	No	Yes

Note: Linear model with standard errors clustered by mother in parentheses. FE denotes fixed effects. The outcome is a continuous measure of an infant's birth weight in grams. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile; Super Output Area. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 5 presents results for alternative outcomes using the same empirical approach. For head circumference, we find a similar pattern as for birth weight. In the unadjusted model, being in the highest $PM_{2.5}$ category is associated with a reduction in head circumference of 0.53 cm, however this is substantially attenuated in the mother fixed effects models. In contrast, the coefficient for length (-0.20) remains statistically significant even after adjusting for mother fixed effects. The pattern of attenuated estimates is similar for the binary outcomes, although associations are not statistically significant in all cases even in unadjusted or minimally adjusted models. For APGAR score at 1 minute, resuscitation, low birth weight, and preterm birth (gestation less than 37 weeks), none of the coefficients in the mother fixed effect models is statistically significant and generally negligible in magnitude despite being relatively large in the unadjusted model. For instance, in the model for low birth weight (<2,500g) as a dichotomous indicator as the outcome, the unadjusted coefficient for the highest pollution category is 1.5 percentage points, which is large given the overall mean in the estimation sample of 1.5%. In contrast the mother fixed effect coefficient is close to 0 and not statistically significant. Only the coefficient on the middle pollution category in Table 5 for crash/urgent c-Section is significant, which is not consistent with a smaller coefficient in the higher pollution category for this outcome and the dose response relationship expected for pollution.

Table 5: Impact of Prenatal $PM_{2.5}$ Exposure on Other Birth Outcomes

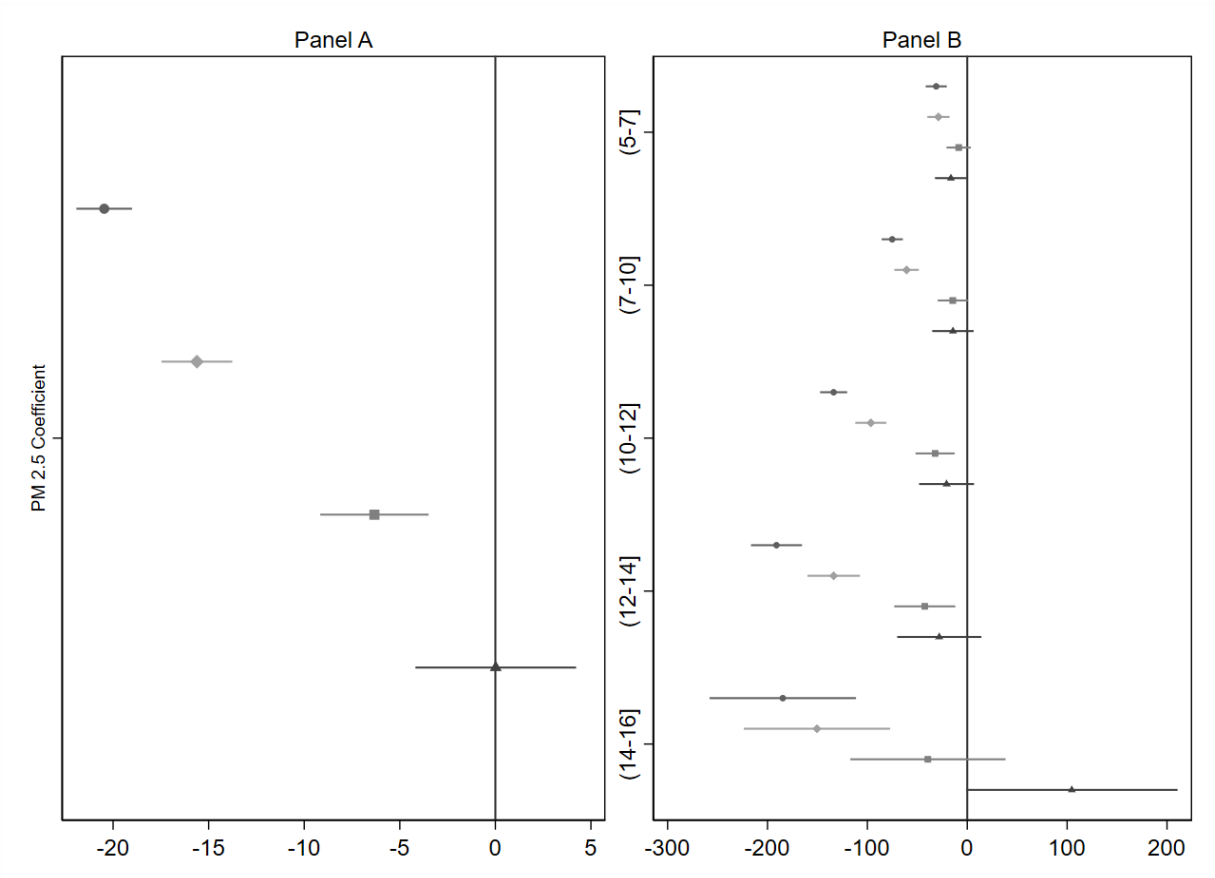
	(1)		(2)		(3)		(4)	
	Low Birth Weight: Weight <2500g (Sample A, N = 146662, mean = .015)							
(6-10]	0.005***	(0.001)	0.003***	(0.001)	0.001	(0.001)	-0.002	(0.002)
(10-16]	0.015***	(0.001)	0.009***	(0.002)	0.004	(0.002)	-0.002	(0.004)
	Resuscitated (Sample A, N = 146465, mean = .086)							
(6-10]	0.008***	(0.002)	0.006**	(0.002)	0.001	(0.003)	0.002	(0.005)
(10-16]	0.012***	(0.003)	0.009*	(0.004)	0.004	(0.005)	0.011	(0.009)
	1-minute APGAR Score <7/10 (Sample A, N = 146374, mean = .064)							
(6-10]	0.005**	(0.002)	0.002	(0.002)	-0.002	(0.002)	0.005	(0.004)
(10-16]	0.013***	(0.003)	0.010**	(0.003)	0.004	(0.004)	0.014	(0.008)
	5-minute APGAR Score <7/10 (Sample A, N = 146388, mean = .009)							
(6-10]	0.000	(0.001)	0.002*	(0.001)	0.002*	(0.001)	0.003	(0.002)
(10-16]	0.001	(0.001)	0.002	(0.001)	0.002	(0.002)	0.002	(0.003)
	Stillborn (Sample B, N = 156053, mean = .004)							
(6-10]	0.0004	(0.0004)	0.0003	(0.0005)	0.0003	(0.0006)	-0.0005	(0.0011)
(10-16]	0.0006	(0.0006)	-0.0001	(0.0007)	0.0001	(0.0009)	-0.0019	(0.0023)
	Preterm: Gestation <37 weeks (Sample C, N = 155495, mean = .056)							
(6-10]	0.000	(0.002)	-0.004*	(0.002)	-0.004	(0.002)	-0.002	(0.003)
(10-16]	0.007**	(0.002)	-0.002	(0.003)	0.000	(0.004)	-0.004	(0.006)
	Crash/Urgent C-Section (Sample C, N = 155495, mean = .14)							
(6-10]	0.003	(0.002)	0.004	(0.003)	0.010**	(0.003)	0.013**	(0.005)
(10-16]	-0.009*	(0.004)	-0.003	(0.004)	0.008	(0.005)	0.008	(0.009)
	Length in cm (Sample A, N = 144484, mean = 51.8)							
(6-10]	-0.237***	(0.021)	0.093***	(0.025)	-0.150***	(0.029)	-0.163***	(0.047)
(10-16]	-0.300***	(0.034)	0.041	(0.040)	-0.205***	(0.049)	-0.199*	(0.087)
	Head Circumference in cm (Sample A, N = 145030, mean = 35.0)							
(6-10]	-0.286***	(0.012)	-0.090***	(0.014)	-0.009	(0.017)	0.000	(0.027)
(10-16]	-0.533***	(0.020)	-0.197***	(0.024)	-0.070*	(0.029)	-0.026	(0.048)
Controls								
Year and Month of birth	No		Yes		Yes		Yes	
Gender	No		Yes		Yes		Yes	
Mother variables	No		Yes		Yes		Yes	
Weather variables	No		Yes		Yes		Yes	
SOA FE	No		No		Yes		Yes	
Mother FE	No		No		No		Yes	

Note: The models are linear with standard errors clustered by mother in parentheses. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre, and the reference category is [3-6] micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile; Super Output Area. Sample A is live singleton births of 37+ weeks gestation; Sample B is all singleton births (live or still, any gestation). Sample C is all live singleton births (any gestation). Estimation uses all available outcome data. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Functional form of the relationship between $PM_{2.5}$ and Infant Outcomes

Although our specification of exposure to $PM_{2.5}$ in three categories allows for non-linear impacts, this model may not appropriately capture the functional form of the relationship of interest with infant outcomes. We are somewhat limited because of the need to ensure confidentiality with the linked data. For this reason, for example, we are unable to include $PM_{2.5}$ as a continuous variable as this would potentially identify postcode of residence for mothers in the dataset. In addition, at the postcode level we are restricted to annual data in NI. Nevertheless, we are able to include additional categories for $PM_{2.5}$ to further explore potential non-linearity, as well as a potentially more efficient and parsimonious model with a "continuous" version of pollution exposure that uses the median values of categories in each postcode. These results are shown, for birth weight, in Figure 1. The first panel shows the additional categories, comparing the same models as in our main results, as well as the continuous version in the second panel. In each case, we find results are consistent with our main estimates, with essentially no evidence of negative effects of $PM_{2.5}$. For example, in our continuous model, every unit increase in $PM_{2.5}$ exposure is associated with a reduction in birth weight of 20g, however once we adjust for mother fixed effects the coefficient is negligible and indistinguishable from 0.

Figure 1: Impact of $PM_{2.5}$ on Birth Weight in Alternative $PM_{2.5}$ Specifications



Notes: Panel A shows the coefficient and 95% confidence interval on the $PM_{2.5}$ variable treated as a continuous variable in the following models: M1: Unadjusted (Circle); M2: M1 + Controls (Diamond); M3: M2 + SOA Fixed Effects (Square); and M4: M3 + Mother Fixed Effects (Triangle). Panel B shows the coefficients and 95% confidence intervals on the $PM_{2.5}$ variable with additional categories, with a base category of [3-5] micrograms per cubic metre.

Test of potential mechanisms

In this paper, we focus on short-run infant outcomes, and thus far find little evidence that infants are impacted by $PM_{2.5}$ during pregnancy. Nevertheless, it is possible that other outcomes beyond the time period we consider could still be affected by pollution, even if a short-run association is not evident because the impact is latent. For example, some studies have documented a relationship between $PM_{2.5}$ and child and adolescent outcomes (e.g. Anderson 2020; Colmer and Voorheis 2020). Another advantage of the data used in this paper is that we have, rarely for this literature, measures that allow us to assess one of the hypothesized biological mechanisms linking pollution to infant and child outcomes. As explained above, the placenta is the organ responsible for transmission of substances between mother and child and regulation of responses to environmental exposures (Wick et al. 2010). In our data, we have information on four measures of placental health (retroplacental clot, calcified placenta, placental infarcts, and unhealthy placenta). A retroplacental clot is an indicator of bleeding behind the placenta, which can occur due to placental abruption (premature separation of the placenta). Placental abruption is associated with a higher of risk of adverse birth outcomes, including caesarean delivery, preterm birth, and low birth weight (Downes et al. 2017). Placenta calcification refers to deposition of calcium-phosphate minerals in placenta tissue, and while there is some indication that placenta calcification is associated with adverse pregnancy outcomes, the evidence is inconclusive (Wallingford et al. 2018). A placental infarction results from the interruption of blood supply to a part of the placenta. While small infarcts are considered normal, larger and multiple infarcts are observed in association with severe early-onset pre-eclampsia and/or intra-uterine growth retardation in pregnancies that are unlikely to reach term (see Becroft et al. (2002)). The final outcome indicates whether a placenta is deemed to be not healthy. If there were to be longer-run impacts of pollution that were not apparent in our short-term outcomes, it is reasonable to expect an impact through these intermediate outcomes and the placental pathway. Therefore, we implement regression models similar to those described above for our main outcomes, with these four measures of placental health as the dependent variables as a function of $PM_{2.5}$ exposure. When we estimate associations with pollution for these measures (see Table 6), we observe the same pattern as we do for our infant outcomes (except for retroplacental clotting); a negative relationship between pollution exposure and placental health in unadjusted models, but no evidence once we compare siblings born to the same mother. Overall, this mechanism test supports our results above that do not indicate a negative impact of $PM_{2.5}$ in this population.

Given these findings, another issue we can explore in our data is the structure of omitted variable bias, specifically, what are the characteristics that attenuate the relationship between pollution and our outcomes. While we do not have information beyond the variables on parental characteristics available in NIMATS, we do know about the characteristics of SOAs where mothers live. Thus, we can replace the SOA fixed effects with a measure of SOA deprivation to determine whether this is a characteristic that affects coefficient estimates. While adjusting for mother fixed effects rendered results not statistically significant, much of the attenuation in the models comes from controlling for SOA. Including area-level averages is akin to the Mundlak approach with panel data (Mundlak 1978). Appendix Table A3 shows results where we adjust for SOA MDM decile, which sorts SOAs into deprivation deciles across seven domains as of 2010, instead of SOA fixed effects, for birth weight. In our data, these deciles are assigned to each mother's SOA at each pregnancy, and included as a categorical variable in the model. Comparing coefficient estimates for birth weight in this Table (and focusing on the (10-16] category for pollution exposure), area level socioeconomic status as measured by SOA fixed effects (column 3, where the

coefficient is -31.6) accounts for around 75% of the coefficient attenuation from the minimally adjusted model (column 2, where the coefficient is -86.3) to the mother fixed effect results (column 4, where the coefficient is -3.0). In contrast, MDM decile (column 7, where the coefficient is -66.9) accounts for around 50% of the coefficient attenuation from the minimally adjusted model to the mother fixed effect results. Therefore, MDM decile accounts for some of the omitted SOA-level factors, but the remaining attenuation still comes from SOA unobserved factors and fixed mother characteristics.

Table 6: Impact of Prenatal $PM_{2.5}$ Exposure on Placenta Outcomes

	(1)		(2)		(3)		(4)	
			Placenta Unhealthy (mean = .146)					
(6-10]	0.045***	(0.003)	0.033***	(0.003)	0.007	(0.004)	0.008	(0.008)
(10-16]	0.098***	(0.004)	0.065***	(0.005)	0.005	(0.007)	0.016	(0.014)
			Placenta Infarcts (mean = .086)					
(6-10]	0.030***	(0.002)	0.018***	(0.003)	0.000	(0.003)	0.001	(0.007)
(10-16]	0.051***	(0.003)	0.029***	(0.004)	-0.002	(0.005)	0.004	(0.012)
			Placenta Calcified (mean = .264)					
(6-10]	0.055***	(0.004)	0.034***	(0.004)	0.022***	(0.005)	0.010	(0.010)
(10-16]	0.094***	(0.006)	0.051***	(0.006)	0.022**	(0.008)	0.015	(0.018)
			Retroplacental Clot (mean = .015)					
(6-10]	0.001	(0.001)	0.000	(0.001)	-0.000	(0.001)	0.004	(0.003)
(10-16]	0.002	(0.001)	-0.000	(0.002)	-0.001	(0.002)	0.003	(0.005)
Observations	114506		114506		114506		114506	
Controls								
Year and Month of birth	No		Yes		Yes		Yes	
Gender	No		Yes		Yes		Yes	
Mother variables	No		Yes		Yes		Yes	
Weather variables	No		Yes		Yes		Yes	
SOA FE	No		No		Yes		Yes	
Mother FE	No		No		No		Yes	

Note: The models are linear with standard errors clustered by mother in parentheses. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre, and the reference category is [3-6] micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile; Super Output Area. The sample is live singleton births of 37+ weeks gestation. Note that observations from June 2016 onwards have been dropped because data on these outcomes are not available after this time. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Robustness checks

We conduct a number of robustness checks to assess sensitivity of results for birth weight to alternative modelling and measurement assumptions. First, we control for additional variables included in NIMATS that were excluded from our main estimates because they represent potential outcomes of pollution. However, we have verified that adjusting for hospital, maternal hypertension, and maternal BMI do not

affect results (Table A4). Second, because we have annual data we need to assign a value for $PM_{2.5}$ exposure to infants born at different times of the year. We have verified that using $PM_{2.5}$ exposure during conception year or birth year, for example, also has similarly little impact (Table A5). Some analyses of infant outcomes restrict their analysis to infants born at term. This arguably has the advantage of assessing impacts among infants of standard gestational length. However, given that gestational age is a potential outcome of some of these exposures it may not be advisable to perform this sample restriction. We have verified that the results for birth weight are consistent across multiple possible sample restrictions (Table A6). Our conclusions are also robust to estimating all models on the sample of mothers with two or more births (Table A7), including additional controls (Table A8) for partner's employment status and smoking behaviour (this last step reduces sample size given missing values), and omitting weather controls (Table A9).

Trimester-specific Exposure

Some studies suggest that exposure later during pregnancy is more strongly associated with birth outcomes than exposure earlier during pregnancy (e.g. see Currie et al., 2009). If so, it is possible that a zero effect from exposure averaged throughout pregnancy could obscure one or more trimester-specific effects. The nature of our $PM_{2.5}$ data, which vary only by calendar year, limit the extent to which we can examine this question here. Nevertheless, we are able to conduct an exploratory analysis of this issue, for birth weight, by selecting two sub-samples of mothers, the first having a pregnancy whose first month of gestation was October, and the second having a pregnancy whose first month was July. For each sub-sample, we separate the gestation period into two parts: trimester 1 (part 1) and trimester 2/3 (part 2) for October mothers, and trimester 1/2 (part 1) and trimester 3 (part 2) for July mothers. In both cases, the first part was assigned the $PM_{2.5}$ level in the conception year, and the second part was assigned the $PM_{2.5}$ level in the birth year, generating two separate $PM_{2.5}$ exposure variables. Given the reduction in sample size we estimate only models 1-3 and not the mother fixed effects model in this analysis. The results are presented in Table A10. Despite the limitations of this analysis we find no evidence of trimester-specific effects once individual, household and neighbourhood characteristics are controlled for. Our zero estimated overall effect does not appear to obscure one or more non-zero trimester-specific effects.

6 Discussion

This paper analyses records from a population-wide database of births in Northern Ireland from 2011-2017 to examine whether infants with higher levels of prenatal $PM_{2.5}$ exposure exhibit worse birth outcomes. We leverage a unique administrative dataset that allows us to link information from pollution data to mother's residential location in low-level geographic areas (postcode). Moreover, because we are able to identify births occurring to the same mother within the time period, we can adopt a mother fixed effects approach. In these models, our pollution coefficient estimates are identified by comparing outcomes for infants born to the same mother but with different pollution exposures. Our main contributions are the use of large-scale data linked to localised measures of $PM_{2.5}$ with an empirical approach that accounts for all time-invariant factors common to mothers, even those that are unobserved. In addition, we consider a wide range of outcomes, allowing us to examine a comprehensive assessment of infant health along with hypothesised biological pathways (placental health).

Overall, we find little evidence of a negative relationship between $PM_{2.5}$ in the postcode of the mother's residence during pregnancy and infant health. In unadjusted or minimally adjusted models, there is a

strong relationship for the majority of outcomes. However, once we control for mother fixed effects, almost all coefficients are small in magnitude for comparing infants born in postcodes with $PM_{2.5}$ levels of 6-9 μg/m^3 or 10-15 μg/m^3 with infants born in postcodes with $PM_{2.5}$ levels of 3-6 μg/m^3. Coefficient attenuation in the mother FE models is consistent with pollution exposure being more concentrated in communities most affected by social determinants of health. In our data, and most data that examine pollution impacts, we do not have individual-level measures of factors such as permanent income, housing quality, or social support, all of which have been linked to pregnancy outcomes and are therefore potential (unobserved) confounders. As is the case in Table 4, we would expect models that do not account for these factors to have larger coefficients. Investigating how these omitted variables affect estimates of pollution exposure on health outcomes is an ongoing challenge for the literature (Deryugina et al. 2019).

The descriptive findings are generally consistent with the previous literature and studies that examine minimally adjusted models or models that adjust for observed factors in estimating relationships between particulate matter and infant outcomes, including studies using UK data (e.g. Dibben and Clemens, 2015; Smith et al. 2017). However, previous literature that has adopted identification strategies to account for omitted variable bias has generally found that the harmful impact of particulate matter remains even after adjusting for unmeasured confounders (Currie et al. 2014). There are a number of potential explanations for why results from this analysis differ from these other papers. First, context may be important, especially for infant outcomes. Many previous papers based in higher-income countries focus on the US which has relatively high rates of infant mortality (Bairoliya and Fink 2018) and a fragmented healthcare system with disparities in access to medical care and prenatal support services. In contrast, Northern Ireland, like the rest of the UK, has a universal healthcare system that is free at point of access. It may be that the impact of pollution is lessened by greater availability of affordable care. Moreover, while Northern Ireland is diverse in terms of its socioeconomic composition, it is very homogenous in terms of race and ethnicity, with the vast majority of residents identifying as white (98.21% in the 2011 Census (NISRA 2014)). In the US, $PM_{2.5}$ related mortality disproportionately affects non-Hispanic black and African American individuals (Bowe et al. 2019).

Second, it is possible that functional forms for pollution exposures other than those we consider in this paper, such as maximum $PM_{2.5}$ levels or number of days above a certain value, might impact infant outcomes more than averages throughout pregnancy. For example, Deryugina et al. (2019) and Currie et al. (2009) consider how changes in contemporaneous exposure to $PM_{2.5}$ and CO impact older-age mortality and birth outcomes respectively. Similarly, exposure effects may be limited to a particular stage of pregnancy (e.g. the third trimester) rather than evident throughout (Currie et al. 2009). While our data have a number of advantages, we are restricted to annual averages for pollution at the postcode level from which we derive our measures of exposure during pregnancy. Having said this, the range of robustness checks that we are able to implement to assess functional form and other potential sources of sensitivity do not provide any evidence that our results are affected by using a more continuous measure of exposure, the presence of threshold effects, the inclusion/exclusion of particular individual-level control variables, changing the way we construct average prenatal exposure or define the estimation sample, or assigning different exposures to earlier and later trimesters during pregnancy. Our individual-level control variables and weather variables have relatively little impact on our conclusions, almost all of the coefficient differences between unadjusted and fully adjusted models are due to SOA and mother fixed effects, indicating results are unlikely to be due to over-controlling for potential outcomes of pollution. 9% of our sample had $PM_{2.5}$ exposure above 10μg/m^3, so based on the previous literature we

should have sufficiently high exposure levels and sufficient sample size and variation to detect adverse impacts in our data. In this regard, it is also important to note that estimates are very consistent across infant health measures and intermediary mechanisms through which pollution impacts are expected to operate, indicating that our findings are not due to a lack of a relationship with one specific variable but rather form a pattern across the range of outcomes we consider.

Third, our identification strategy is based on comparing siblings born to the same mother. If there are infant-specific factors that are correlated with pollution exposure, these within-family confounders could still bias our estimates. Generally we would expect these other factors, specifically other types of disadvantage, to be positively associated with $PM_{2.5}$ exposure, meaning that we would expect them to bias our estimates upwards if they are present. The expected direction of confounder impact is apparent in our comparison of how we enter area-level SOA controls into the model (Table A3). The results using MDM decile indicate that area-level deprivation accounts for around half of the $PM_{2.5}$ coefficient (relative to if SOA characteristics were unobserved). Given that we find a lack of impact of pollution exposure in our mother fixed effect models, however, a more relevant source of potential bias is misclassification bias or measurement error, which can attenuate estimates even when the error is random. Moreover, its consequences are exacerbated in fixed effects models (Ashenfelter and Krueger 1994; McGovern 2019). When mismeasurement or misclassification is random, the degree of attenuation depends on the correlation in pollution between siblings, the variance of the idiosyncratic error affecting pollution, and the variance in true underlying pollution exposure (the ratio of the latter two being referred to as the signal to noise ratio). The more highly correlated is the true pollution exposure within siblings, the greater the degree of attenuation bias. In their analysis of measurement error in twin fixed effect models, Kohler et al. (2011) report a range for the signal to noise ratio of 0.04–0.12 among analyses that are able to include an external validation measure for their particular exposure of interest. This would imply fixed effects analyses would be up to 14% lower than non fixed effect analyses due to measurement error alone. These figures are for non-identical dizygotic twins, who if anything would be expected to exhibit greater bias than in our sample because we would expect the correlation in pollution exposure to be higher for twins than siblings. On the basis of these figures, measurement error is unlikely to be able to fully explain why our FE estimates are close to 0. In our preferred specification in Table 4, without FE the coefficient on being in the highest $PM_{2.5}$ category is -31.6, whereas once FE are added the coefficient is -3.0. This is a far larger attenuation (99%) than the 14% implied by measurement error. Indeed, this degree of attenuation is far larger than any of the scenarios considered by Kohler et al. (2011), even those for monozygotic twins and a signal to noise ratio of up to 0.20. Even if our FE coefficient was 14% less attenuated (-4.5), this magnitude is still negligible and also half that of the estimated standard error meaning it would not be close to reaching statistical significance at conventional levels. Nevertheless, our context is different from Kohler et al. (2011) as we consider pollution exposure instead of years of schooling as the independent variable. Without having access to additional external validation data for localised $PM_{2.5}$ exposure in Northern Ireland, it is hard to assess how much a role measurement error is playing in our estimates. For this reason, future research should adopt additional identification strategies using alternative data sources to investigate this issue further. Higher frequency data linked to nationally representative samples would also be useful for exploring non-linearities in $PM_{2.5}$ exposure, and for quantifying the impact of more long-run effects on mothers and children.

Fourth, we measure pollution at the residential postcode. While this has the advantage of capturing outdoor exposure in the local neighbourhood, it may be that a mother's total exposure to $PM_{2.5}$ is more

accurately captured by a combination of sources including indoor pollution and commuting and workplace environment. Traffic-related $PM_{2.5}$ has been highlighted in the literature as being particularly harmful (Smith et al. 2017), and there is also a growing focus on indoor pollution as a determinant of health (Sharpe 2004). However, we would expect these sources to be positively correlated with our measure of neighbourhood exposure given that indoor pollution also disproportionately affects disadvantaged communities in the UK (Ferguson et al. 2021), therefore it is unclear how their omission could fully explain that we find a negligible relationship with birth outcomes. Moreover, urban-rural differences in the indoor/outdoor pollution relationship should be accounted for as in the model with SOA fixed effects we are essentially comparing exposures within small geographic areas. Further, Dibben and Clemens (2015) demonstrate robust associations between particulate pollution and birth outcomes whether pollution is measured at residential location or some combination of residential and workplace location. While this issue of indoor, commuting and workplace pollution is not one we can address in our study, future research should aim to measure such exposure for the same individuals. Advances in technology may facilitate data collection, for example through the use of wearables, air sampling or biomarkers.

This paper contributes to a number of policy-relevant debates. The infant outcomes we consider here are important in and of themselves because they measure an important dimension of population health. Policy interventions to improve maternal and infant health are notable because early life conditions have long-run impacts on child and adult outcomes, with potential intergenerational consequences (Currie 2011). Medical care around pregnancy comprises a substantial proportion of healthcare resources (Almond et al. 2005; Xu et al. 2015) and more broadly, improving indicators such as birth weight may have long-term economic benefits (Black et al. 2007). Recent evidence on private valuations of birth weight have suggested that individuals are willing to pay up to $2.40 for each additional gram (Clarke et al. 2021). Quantifying the impact of $PM_{2.5}$ exposure on these outcomes is therefore important for assessing the full range of benefits that may be associated with reducing levels of particulate matter. Moreover, there are substantial disparities in pregnancy outcomes according to parental characteristics, including but not limited to mortality and birth weight. Considering the long run consequences of these inequalities, evidence on how to best address differences in maternal and child health is an urgent priority. Given that we observe parallel gradients in pollution exposure to gradients in these outcomes, quantifying the extent to which reducing exposure to $PM_{2.5}$ is expected to reduce these disparities is important for understanding which interventions are likely to be most effective. The findings in this paper imply that further improvements in maternal and infant outcomes will likely require addressing underlying social determinants of health, such as material disadvantage, or behaviours that are known to have strong effects such as in utero exposure to smoking, rather than focusing solely on environmental pollution.

Overall, it is important to note that we do not view these results as undermining the imperative to reduce population exposures. There is a substantial literature examining a range of population health outcomes, with many quasi-experimental studies supporting policy intervention to address $PM_{2.5}$ and other pollutants. In our analysis, we considered short-run prenatal postcode-level exposure as a determinant of short-run (birth) outcomes. Even if reducing exposure as defined by this specific measure is not sufficient to reduce disparities in infant health at birth in Northern Ireland, there remains a strong evidence base to continue taking action to lower rates of $PM_{2.5}$ and other pollutants in other contexts and to improve other population health outcomes.

7 Conclusion

In a nationally representative dataset linking infant outcomes with ambient $PM_{2.5}$ data in Northern Ireland, a context with universal healthcare access that is largely free at the point of use, we find little evidence that higher levels of exposure have a negative impact after we adjust for characteristics common to siblings with the same mother. This finding highlights the importance of controlling for unobserved confounders in the wider literature on pollution. While research on other health outcomes emphasises that reducing exposure to $PM_{2.5}$ remains a policy priority, our results imply that addressing existing disparities in maternal and infant health in the UK is likely to require improving underlying social determinants of health in addition to environmental exposures.

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Declaration of interest

The authors have no conflicts of interest to declare.

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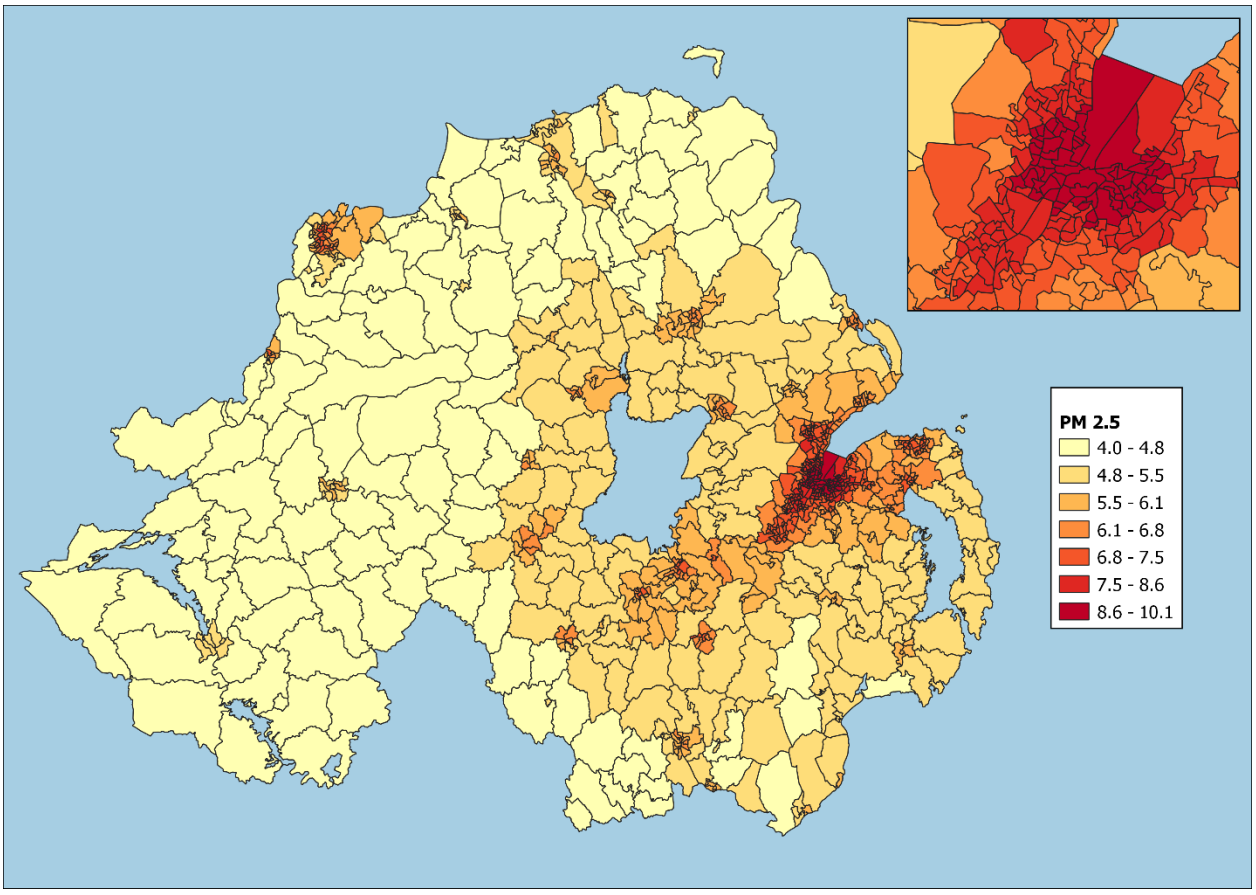
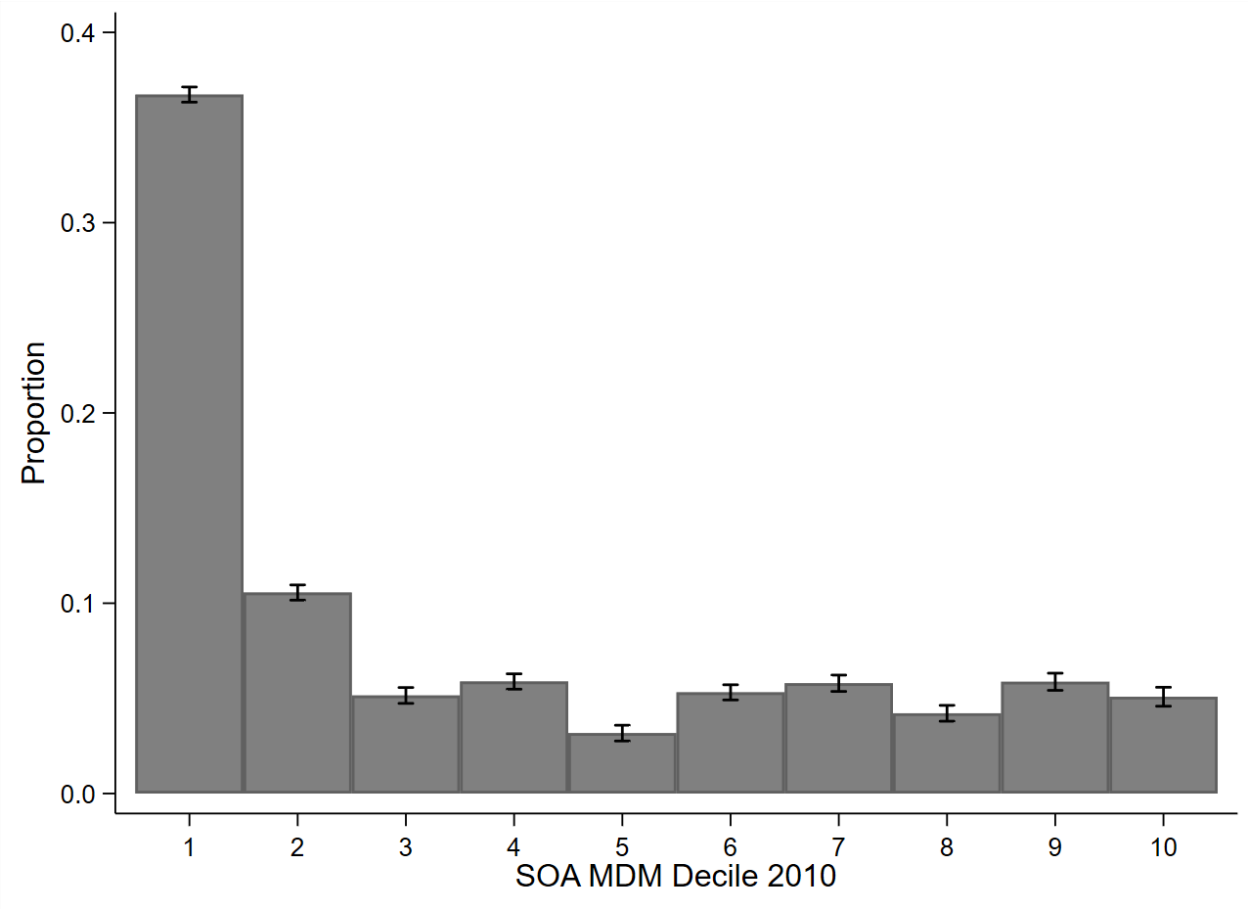
Appendix

Figure A1: Distribution of $PM_{2.5}$ by Super Output Area in Northern Ireland 2017



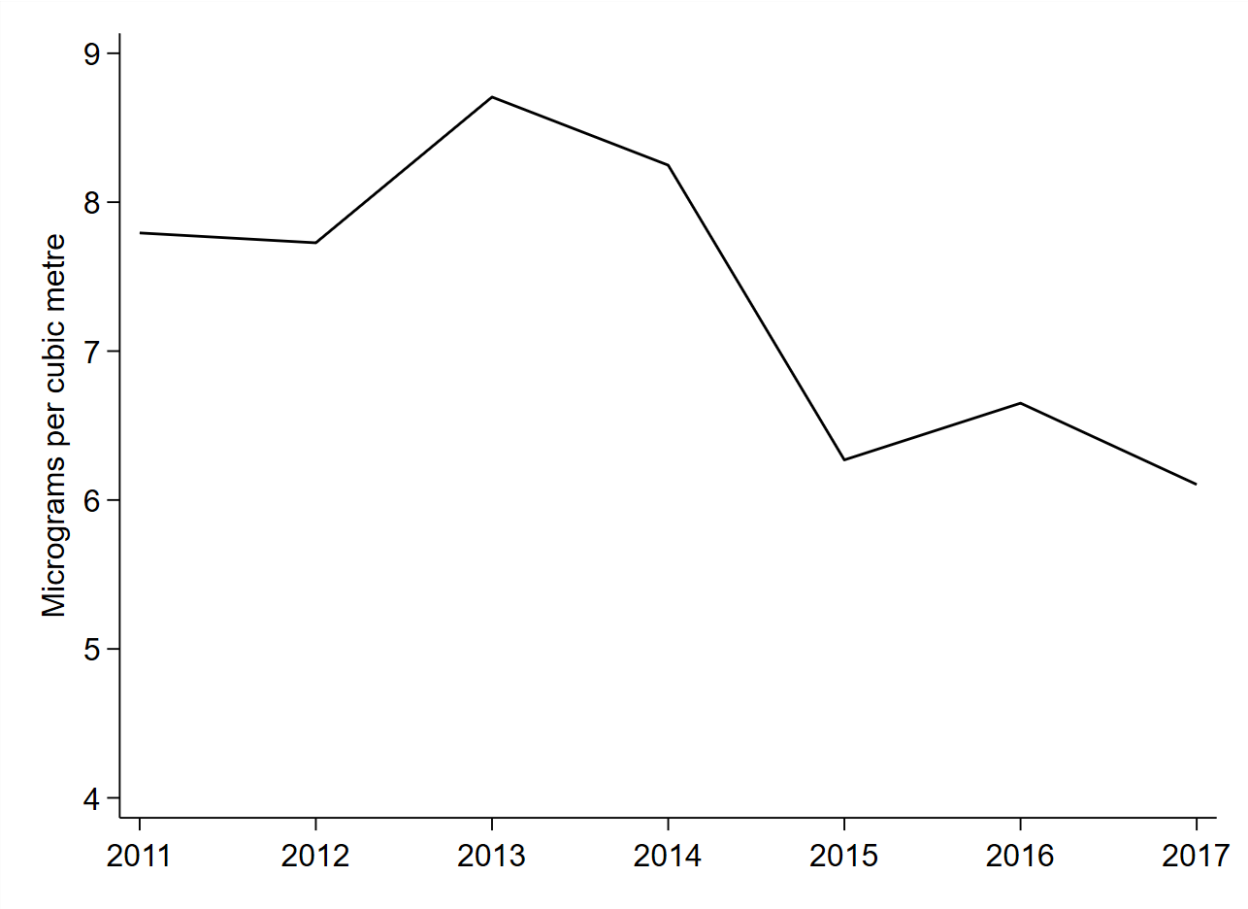
Notes: Map shows population-weighted $PM_{2.5}$ concentrations (micrograms per cubic metre) by Super Output Area, with inset showing Belfast and surrounding areas. Authors' calculations.

Figure A2: Proportion of Infants with High $PM_{2.5}$ Exposure by Super Output Area Deprivation Decile



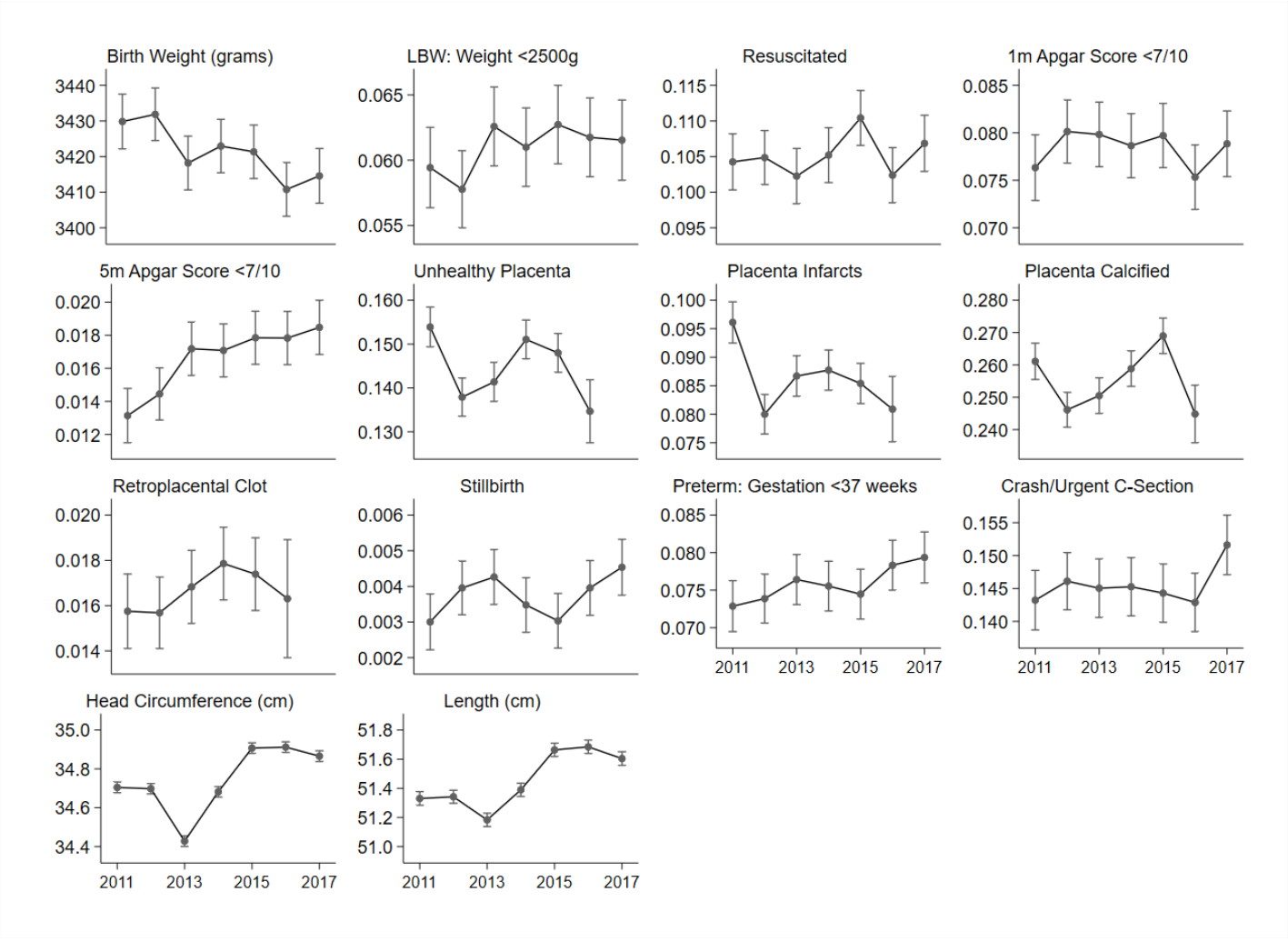
Notes: Figure shows the proportion of infants with an $PM_{2.5}$ exposure level above 10 micrograms per cubic metre by decile of deprivation. For example, decile 1 contains infants in the 10% most deprived SOAs according to the NI Multiple Deprivation Measure (2010). 95% confidence intervals are also displayed.

Figure A3: Population-Weighted $PM_{2.5}$ Levels 2011-2017



Notes: Figure shows the annual population-weighted mean $PM_{2.5}$ level (in micrograms per cubic metre) across Northern Ireland between 2011 and 2017. The unit of the underlying data is the postcode (not infant).

Figure A4: Mean Birth Outcomes by Year



Notes: Figures shows mean birth outcomes by year for all outcomes. Mean birth weight is reported in grams, while mean head circumference and length are reported in centimetres; the remainder are proportions. The sample is unrestricted, contains all available cases, and differs slightly by outcome depending on missing values. 95% confidence intervals are also reported.

Table A1: Within-Mother Variation in Prenatal $PM_{2.5}$ Exposure

$PM_{2.5}$ category at previous pregnancy	$PM_{2.5}$ category at current pregnancy			
	[3-6]	(6-10]	(10-16]	Total
[3-6]	3,578	4,821	89	8,488
	42.2	*56.8*	*1.1*	*100*
(6-10]	4,743	20,499	1,927	27,169
	17.5	*75.5*	*7.1*	*100*
(10-16]	80	1,856	1,273	3,209
	2.5	*57.8*	*39.7*	*100*
Total	8,401	27,176	3,289	38,866
	21.6	*69.9*	*8.5*	*100*

Notes: Table reports a crosstabulation of $PM_{2.5}$ at previous pregnancy with $PM_{2.5}$ at current pregnancy, for mothers with more than one pregnancy. Figures in italics are row percentages.

Table A2: Full Table for the Impact of Prenatal $PM_{2.5}$ Exposure and Control Variables on Birth Weight

	(1)		(2)		(3)		(4)	
$PM_{2.5}$ (ref. [3-6])								
(6-10]	-53.0***	(3.2)	-40.5***	(3.7)	-12.2**	(4.4)	1.1	(5.9)
(10-16]	-129.9***	(5.3)	-86.3***	(6.0)	-31.6***	(7.4)	-3.0	(10.4)
Gender (ref: Girl)								
Boy			127.1***	(2.4)	127.0***	(2.4)	138.1***	(3.3)
Age at Birth (ref: < 21)								
21-25			-11.4	(6.0)	-14.7*	(6.0)	-19.3	(11.0)
26-30			-3.4	(6.1)	-12.3*	(6.1)	-33.6*	(14.5)
31-35			1.6	(6.3)	-11.7	(6.4)	-23.2	(17.7)
36 or above			-23.9***	(6.8)	-38.9***	(6.9)	-10.6	(21.6)
Single Parent (ref: No)								
Yes			-24.2***	(5.6)	-22.6***	(5.6)	3.1	(9.0)
Employed (ref: No)								
Yes			54.0***	(3.5)	48.7***	(3.5)	-5.3	(23.4)
Smokes (Ref: No)								
Yes			-252.7***	(3.9)	-244.2***	(3.9)	-85.5***	(9.5)
No. of Pregnancies (ref: 1)								
2 Pregnancies			88.5***	(2.9)	90.2***	(2.9)	107.1***	(4.6)
3 or more Pregnancies			105.4***	(3.3)	107.4***	(3.3)	131.5***	(7.8)
Temperature Decile (Ref: 1 (Coolest))								
2			0.9	(5.2)	-1.8	(5.3)	4.9	(7.3)
3			-2.8	(5.2)	-6.4	(5.5)	-6.7	(7.6)
4			4.5	(5.5)	4.5	(5.8)	4.2	(7.9)
5			-2.9	(5.5)	-4.1	(5.9)	-3.4	(8.3)
6			-6.2	(5.6)	-6.6	(6.1)	-0.9	(8.6)
7			-8.9	(5.7)	-6.2	(6.3)	-8.3	(9.0)
8			-5.0	(6.0)	-0.4	(6.7)	-3.6	(9.5)
9			-12.1	(6.3)	-7.9	(7.1)	-6.7	(10.1)
10 (Warmest)			-5.1	(7.0)	0.8	(8.1)	5.0	(11.6)
Rainfall Decile (Ref: 1 (Driest))								
2			17.8*	(7.8)	12.8	(8.0)	15.1	(11.0)
3			13.7	(7.8)	14.3	(8.1)	11.7	(11.2)
4			29.9***	(7.9)	29.8***	(8.3)	25.5*	(11.3)
5			26.9***	(7.7)	26.7**	(8.3)	23.6*	(11.3)
6			25.4**	(7.8)	23.3**	(8.7)	21.6	(12.0)
7			27.0***	(7.8)	23.4**	(8.7)	25.1*	(12.0)
8			13.2	(7.8)	13.1	(9.0)	12.6	(12.5)
9			27.8***	(8.0)	26.0**	(9.4)	24.7	(13.1)
10 (Wettest)			39.3***	(8.2)	31.0**	(10.1)	30.9*	(14.1)
Birth Month (ref: January)								
February			2.7	(5.9)	2.6	(5.9)	15.6	(8.2)
March			-0.2	(5.8)	0.9	(5.8)	-7.4	(8.0)

April			6.8	(5.9)	7.4	(5.9)	7.9	(8.2)
May			0.0	(5.8)	1.1	(5.8)	9.8	(8.1)
June			14.0*	(6.0)	14.6*	(6.0)	28.1***	(8.2)
July			6.3	(5.8)	8.6	(5.8)	20.1*	(8.0)
August			15.9**	(5.8)	18.4**	(5.8)	29.4***	(8.1)
September			8.8	(5.7)	11.0	(5.7)	24.7**	(7.9)
October			13.5*	(5.8)	15.4**	(5.8)	26.5**	(8.2)
November			13.5*	(5.9)	15.6**	(5.9)	15.8	(8.2)
December			-7.4	(6.0)	-5.4	(6.0)	-0.1	(8.4)
Birth Year (ref: 2011)								
2012			-5.3	(4.6)	-2.5	(4.6)	4.2	(6.6)
2013			-14.5**	(4.5)	-13.8**	(4.6)	-7.2	(6.6)
2014			-15.8***	(4.7)	-17.5***	(4.8)	-5.5	(7.6)
2015			-40.4***	(4.8)	-31.6***	(4.9)	-7.5	(8.9)
2016			-48.0***	(4.8)	-34.2***	(4.9)	-10.3	(10.0)
2017			-39.4***	(5.3)	-25.1***	(5.5)	-3.6	(11.7)
Constant	3575.1***	(2.9)	3430.8***	(11.1)	3304.4***	(46.8)	3423.7***	(117.3)
Observations	146662		146662		146662		146662	
Outcome Mean	3525.48		3525.48		3525.48		3525.48	
SOA FE	No		No		Yes		Yes	
Mother FE	No		No		No		Yes	

Note: Linear model with standard errors clustered by mother in parentheses. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grams. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. SOA coefficients are not reported. There are 75,965 infants belonging to a mother with more than one birth. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table A1: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, by Type of Geographical Control

	SOA FE				SOA MDM			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
$PM_{2.5}$ (ref. [3-6])								
(6-10]	-53.0***	-40.5***	-12.2**	1.1	-53.0***	-40.5***	-34.3***	1.6
	(3.2)	(3.7)	(4.4)	(5.9)	(3.2)	(3.7)	(3.8)	(5.8)
(10-16]	-129.9***	-86.3***	-31.6***	-3.0	-129.9***	-86.3***	-66.9***	-2.4
	(5.3)	(6.0)	(7.4)	(10.4)	(5.3)	(6.0)	(6.3)	(10.3)
Observations	146662	146662	146662	146662	146662	146662	146662	146662
Outcome Mean	3525.5	3525.5	3525.5	3525.5	3525.5	3525.5	3525.5	3525.5
Controls								
Year and Month of birth	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Gender	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Mother variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Weather variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
SOA FE	No	No	Yes	Yes	No	No	No	No
SOA MDM	No	No	No	No	No	No	Yes	Yes
Mother FE	No	No	No	Yes	No	No	No	Yes

Note: Linear model with standard errors clustered by mother in parentheses. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grams. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile; SOA FE or SOA MDM decile. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table A2: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, Including Control Variables Potentially Affected by Pollution Exposure

	Without Bad Controls				With Bad Controls			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
$PM_{2.5}$ (ref. [3-6])								
(6-10]	-52.7***	-40.9***	-11.9**	2.8	-52.7***	-34.5***	-12.8**	2.2
	(3.3)	(3.8)	(4.4)	(6.0)	(3.3)	(4.0)	(4.4)	(6.0)
(10-16]	-131.2***	-88.9***	-32.7***	-6.4	-131.2***	-76.0***	-32.6***	-6.7
	(5.3)	(6.1)	(7.6)	(10.8)	(5.3)	(6.4)	(7.5)	(10.8)
Observations	142433	142433	142433	142433	142433	142433	142433	142433
Outcome Mean	3525.4	3525.4	3525.4	3525.4	3525.4	3525.4	3525.4	3525.4
Controls								
Year and Month of birth	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Gender	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Mother variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Weather variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
SOA FE	No	No	Yes	Yes	No	No	Yes	Yes
Mother FE	No	No	No	Yes	No	No	No	Yes
Bad Controls	No	No	No	No	No	Yes	Yes	Yes

Note: Linear model with standard errors clustered by mother in parentheses. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grams. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile; Bad controls: hypertension, BMI, and hospital of birth. The sample in both cases excludes infants with missing values on any of the above control variables. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table A3: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, by Period of Exposure

	(1)		(2)		(3)		(4)	
Weighted								
(6-10]	-53.0***	(3.2)	-40.5***	(3.7)	-12.2**	(4.4)	1.1	(5.9)
(10-16]	-129.9***	(5.3)	-86.3***	(6.0)	-31.6***	(7.4)	-3.0	(10.4)
Modal								
(6-10]	-49.2***	(3.2)	-36.7***	(3.7)	-8.4*	(4.2)	-0.5	(5.7)
(10-16]	-125.1***	(5.1)	-79.7***	(5.9)	-25.3***	(7.2)	-5.1	(10.1)
Birth Year								
(6-10]	-47.9***	(3.1)	-38.8***	(3.6)	-9.1*	(4.2)	-5.1	(5.7)
(10-16]	-120.9***	(5.1)	-77.1***	(6.1)	-20.0**	(7.5)	-7.1	(10.5)
Conception Year								
(6-10]	-50.1***	(3.3)	-37.3***	(3.8)	-8.6	(4.4)	0.1	(5.9)
(10-16]	-120.8***	(5.1)	-75.6***	(5.9)	-17.0*	(7.3)	-1.2	(10.2)
Maximum								
(6-10]	-46.7***	(3.4)	-36.0***	(4.0)	-8.8	(4.6)	-1.5	(6.2)
(10-16]	-119.4***	(4.9)	-73.1***	(5.9)	-19.7**	(7.3)	-4.3	(10.4)
Observations	146662		146662		146662		146662	
Outcome Mean	3525.5		3525.5		3525.5		3525.5	
Controls								
Year and Month of birth	No		Yes		Yes		Yes	
Gender	No		Yes		Yes		Yes	
Mother variables	No		Yes		Yes		Yes	
Weather variables	No		Yes		Yes		Yes	
SOA FE	No		No		Yes		Yes	
Mother FE	No		No		No		Yes	

Note: Linear model with standard errors clustered by mother in parentheses. $PM_{2.5}$ Reference Category: [3-6] micrograms per cubic metre. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grams. Sample is all live singleton births of 37+ weeks gestation. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile; Super Output Area. $PM_{2.5}$ is specified in the following ways: Weighted (main estimates): weighted average of $PM_{2.5}$ in birth and conception year, where the weights reflect gestation time spent in each year, respectively; Modal: modal $PM_{2.5}$ category, namely birth year (conception year) if they spent most gestation time in birth year (conception year); Birth Year: $PM_{2.5}$ category in birth year; Conception Year: $PM_{2.5}$ category in conception year (may differ from or coincide with birth year); Maximum: maximum of birth and conception year $PM_{2.5}$ categories (may coincide or may differ if conception year and birth year differ). Weather controls are also adjusted in the same way as the corresponding $PM_{2.5}$ variable, except in the weighted model, where modal (rather than weighted) weather is used.

Table A4: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, by Estimation Sample

	A	B	C	D	E	F	G	H
M1								
(6-10]	-53.0***	-52.6***	-52.1***	-53.0***	-53.2***	-51.3***	-50.9***	-53.3***
	(3.2)	(3.7)	(3.7)	(3.2)	(3.3)	(4.1)	(4.0)	(3.3)
(10-16]	-129.9***	-139.0***	-138.4***	-129.9***	-128.3***	-137.0***	-136.7***	-128.3***
	(5.3)	(6.1)	(6.0)	(5.3)	(5.3)	(6.6)	(6.6)	(5.3)
M2								
(6-10]	-40.5***	-33.9***	-33.9***	-40.5***	-40.1***	-33.4***	-33.3***	-40.1***
	(3.7)	(4.4)	(4.3)	(3.7)	(3.7)	(4.4)	(4.3)	(3.7)
(10-16]	-86.3***	-83.2***	-83.9***	-86.4***	-85.7***	-85.1***	-85.7***	-85.8***
	(6.0)	(7.1)	(7.0)	(6.0)	(6.0)	(7.1)	(7.0)	(6.0)
M3								
(6-10]	-12.2**	-4.9	-4.9	-12.3**	-12.1**	-4.5	-4.2	-12.2**
	(4.4)	(5.1)	(5.0)	(4.4)	(4.3)	(5.1)	(5.0)	(4.3)
(10-16]	-31.6***	-29.1***	-29.4***	-31.9***	-31.4***	-31.5***	-31.2***	-31.7***
	(7.4)	(8.7)	(8.6)	(7.4)	(7.4)	(8.8)	(8.6)	(7.4)
M4								
(6-10]	1.1	5.2	2.7	1.3	1.8	5.3	3.5	2.0
	(5.9)	(6.9)	(6.7)	(5.9)	(5.8)	(6.9)	(6.7)	(5.8)
(10-16]	-3.0	6.1	-1.2	-2.8	-3.0	3.1	-2.4	-2.8
	(10.4)	(12.4)	(12.0)	(10.4)	(10.4)	(12.4)	(12.0)	(10.4)
Observations	146662	155893	155344	146859	148422	160378	159792	148620
Outcome Mean	3525.5	3457.5	3462.7	3524.9	3516.2	3426.5	3432.0	3515.6

Note: Linear model with standard errors clustered by mother in parentheses. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grams. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. The models are as follows: M1 is unadjusted. M2 is adjusted for birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smokes or not, gravida; temperature decile, rainfall decile. M3 is also adjusted for Super Output Area. M4 also includes mother fixed effects. Samples are denoted A, B, C, etc. For samples that include both singleton and multiple births (E-H), models 2-4 also adjust for singleton versus multiple birth. Sample Definitions: Live, Singletons, 37+ weeks gestation (A); Live and Still, Singletons, Any gestation (B); Live, Singletons, Any gestation (C); Live and Still, Singletons, 37+ weeks gestation (D); Live, Singletons and Multiples, 37+ weeks gestation (E); Live and Still, Singletons and Multiples, Any gestation (F); Live, Singletons and Multiples, Any gestation (G); Live and Still, Singletons and Multiples, 37+ weeks gestation (H). * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table A7: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, by Births Sample

	All Mothers				Mothers with 2 or more Births			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
$PM_{2.5}$ (ref. [3-6])								
(6-10]	-53.0***	-40.5***	-12.2**	1.1	-65.9***	-32.8***	-4.5	1.1
	(3.2)	(3.7)	(4.4)	(5.9)	(4.4)	(5.2)	(5.9)	(5.9)
(10-16]	-129.9***	-86.3***	-31.6***	-3.0	-146.3***	-68.7***	-14.1	-3.0
	(5.3)	(6.0)	(7.4)	(10.4)	(7.7)	(8.8)	(10.3)	(10.5)
Observations	146662	146662	146662	146662	75965	75965	75965	75965
Outcome Mean	3525.5	3525.5	3525.5	3525.5	3548.6	3548.6	3548.6	3548.6
Controls								
Year and Month of birth	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Gender	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Mother variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Weather variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
SOA FE	No	No	Yes	Yes	No	No	Yes	Yes
Mother FE	No	No	No	Yes	No	No	No	Yes

Note: Linear model with standard errors clustered by mother. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grammes. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smoker or not, gravida; temperature decile, rainfall decile; Super Output Area. The 'Mothers with 2 or more Births' (mother FE) sample is restricted to mothers with two or more birth records; the 'All Mothers' sample includes mothers with any number of births (main sample). * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table A8: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, with Additional Partner Covariates

	No Partner Covariates				With Partner Covariates			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
$PM_{2.5}$ (ref. [3-6])								
(6-10]	-53.0***	-40.5***	-12.2**	1.1	-51.0***	-36.7***	-8.9	4.0
	(3.2)	(3.7)	(4.4)	(5.9)	(3.3)	(3.9)	(4.5)	(6.1)
(10-16]	-129.9***	-86.3***	-31.6***	-3.0	-122.8***	-75.5***	-23.1**	-5.9
	(5.3)	(6.0)	(7.4)	(10.4)	(5.5)	(6.4)	(7.8)	(10.9)
Observations	146662	146662	146662	146662	133459	133459	133459	133459
Outcome Mean	3525.5	3525.5	3525.5	3525.5	3530.1	3530.1	3530.1	3530.1
Controls								
Year and Month of birth	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Gender	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Mother variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Weather variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
SOA FE	No	No	Yes	Yes	No	No	Yes	Yes
Mother FE	No	No	No	Yes	No	No	No	Yes
Partner Covariates	No	No	No	No	No	Yes	Yes	Yes

Note: Linear model with standard errors clustered by mother. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grammes. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smoker or not, gravida; temperature decile, rainfall decile; Super Output Area; Partner covariates: employed or not, smoker or not. The 'With Partner Covariates' sample is restricted to cases with complete information on all controls, including partner's smoking and employment status. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Figure A9: Impact of Prenatal $PM_{2.5}$ Exposure on Birth Weight, Excluding Weather Controls

	With Weather				Without Weather			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
$PM_{2.5}$ (ref. [3-6])								
(6-10]	-53.0***	-40.5***	-12.2**	1.1	-53.0***	-47.0***	-13.8**	-0.3
	(3.2)	(3.7)	(4.4)	(5.9)	(3.2)	(3.5)	(4.3)	(5.7)
(10-16]	-129.9***	-86.3***	-31.6***	-3.0	-129.9***	-94.7***	-31.6***	-3.3
	(5.3)	(6.0)	(7.4)	(10.4)	(5.3)	(5.5)	(7.3)	(10.2)
Observations	146662	146662	146662	146662	146662	146662	146662	146662
Outcome Mean	3525.5	3525.5	3525.5	3525.5	3525.5	3525.5	3525.5	3525.5
Controls								
Year and Month of birth	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Gender	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Mother variables	No	Yes	Yes	Yes	No	Yes	Yes	Yes
SOA FE	No	No	Yes	Yes	No	No	Yes	Yes
Mother FE	No	No	No	Yes	No	No	No	Yes
Weather variables	No	Yes	Yes	Yes	No	No	No	No

Note: Linear model with standard errors clustered by mother. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grammes. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth month, birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smoker or not, gravida; Super Output Area. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table A10: Impact of Prenatal $PM_{2.5}$ Exposure in Different Trimesters on Birth Weight

	(1)	(2)	(3)
Panel A: Births whose first month of gestation is October			
Trimester 1 $PM_{2.5}$ (ref. [3-6])			
(6-10]	-27.7	-19.2	8.3
	(14.6)	(15.7)	(17.4)
(10-16]	-73.7***	-34.5	7.1
	(21.6)	(23.6)	(26.6)
Trimester 2/3 $PM_{2.5}$ (ref. [3-6])			
(6-10]	-7.9	-22.7	-6.4
	(12.8)	(14.0)	(15.4)
(10-16]	-47.3*	-47.2*	-22.0
	(21.0)	(23.4)	(26.8)
Observations	12923	12923	12923
Outcome Mean	3524.3	3524.3	3524.3
Panel B: Births whose first month of gestation is July			
Trimester 1/2 $PM_{2.5}$ (ref. [3-6])			
(6-10]	-18.4	-9.4	3.0
	(15.8)	(17.0)	(18.5)
(10-16]	-93.5***	-65.1*	-39.1
	(23.1)	(25.8)	(29.1)
Trimester 3 $PM_{2.5}$ (ref. [3-6])			
(6-10]	-35.1*	-34.1*	-10.5
	(14.1)	(15.3)	(17.2)
(10-16]	-55.7*	-53.4*	-1.0
	(23.0)	(25.7)	(29.2)
Observations	11757	11757	11757
Outcome Mean	3523.5	3523.5	3523.5
Controls			
Year and month of birth	No	Yes	Yes
Gender	No	Yes	Yes
Mother variables	No	Yes	Yes
Weather variables	No	Yes	Yes
SOA FE	No	No	Yes
Mother FE	No	No	No

Note: Linear model with standard errors clustered by mother. FE denotes Fixed Effects. The outcome is a continuous measure of an infant's birth weight in grammes. Sample is all live singleton births of 37+ weeks gestation. The $PM_{2.5}$ exposure variable is a categorical, weighted average of $PM_{2.5}$ in birth year and $PM_{2.5}$ in conception year, where weights reflect gestation time spent in each year, respectively. The underlying units are micrograms per cubic metre. Control variables: birth year; infant: gender; mother: age group at birth, one parent family or not, employed or not, smoker or not, gravida; temperature decile, rainfall decile; Super Output Area. Model 4 (mother FE) not estimated. In Panel A, the estimation sample is restricted to mothers whose first gestation month is October; trimester 1 exposure is assigned the conception year $PM_{2.5}$ level, and Trimester 2 and 3 exposure is assigned the birth year $PM_{2.5}$ level. In Panel B, the estimation sample is restricted to mothers whose first gestation month is July; trimester 1 and 2 exposure is assigned the conception year $PM_{2.5}$ level, and Trimester 3 exposure is assigned the birth year $PM_{2.5}$ level. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.