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Toxic Air and Troubled Minds: The Effect of In-utero Exposure to Air Pollution on Child and Adolescent Mental Health

Christina Munkholm Andersen, Marius Opstrup Morthorst[*,1]

[1] *Department of Economics and Business Economics, Aarhus University*

Abstract

We study the impact of in-utero exposure to air pollution on infant and mental health in a low-pollution setting. Identification relies on a sibling comparison to address concerns about endogenous exposure to pollution. We leverage high-resolution pollution data on $PM_{2.5}$ linked to Danish administrative registers. In-utero exposure modestly reduces birth weight and length. It also increases the probability of an ADHD diagnosis in childhood, with effects concentrated in the third trimester and evident by age eight. We find evidence of an inflammatory response, as exposure increases the risk of pregnancy-related hypertension. Results highlight persistent impacts of early-life pollution exposure.

Keywords: air pollution, infant health, mental health
JEL: I14, I18, J13, Q53

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1 Introduction

Air pollution is a major threat to human health, and actions to reduce pollution levels are on the global policy agenda. Air pollution has been documented to have both short- and long-term negative effects on human health, as well as non-health effects by decreasing cognition and productivity (Deryugina et al., 2019; Currie and Walker, 2019; Aguilar-Gomez et al., 2022). In particular, in-utero exposure to air pollution has been shown to negatively impact infant health and longer-run economic outcomes. However, less is known about how in-utero exposure to air pollution affects mental health in childhood and adolescence.

Mental health problems are increasing in many countries, particularly among children and young people, where more than 1 in 10 live with a diagnosable mental disorder (OECD/EU, 2018; Kieling et al., 2024). Mental health disorders impose significant costs on both individuals and society, including the cost of treatment, social security programs, and indirect losses from reduced productivity, amounting to between 5-8% of GDP in most Western countries (Arias et al., 2022).[1] Understanding factors that influence mental health is of great importance to patients, policymakers, and society.

In this paper, we aim to understand how early-life stressors, such as exposure to pollution, during a critical and vulnerable stage of life can shape mental health outcomes later in life. Specifically, we estimate the impact of in-utero exposure to ambient air pollution on infant health, followed by child and adolescent mental health outcomes. The setting of our study is Denmark, a country generally characterized by low pollution levels and a publicly funded, high-quality healthcare system. This context underscores the importance of identifying adverse effects of in-utero exposure to air pollution, highlighting that even small levels of air pollution is harmful, and that all reduction in levels of air pollution could potentially improve public health.

We leverage granular pollution data and link it with population-level administrative data to obtain a dataset with rich information on exposure to air pollution, infant health, mental health care utilization, and socioeconomic information on both children and their parents. This unique data allows us to conduct detailed analysis, thereby extending current evidence of in-utero exposure to air pollution. Firstly, because of the detailed air pollution and neonatal data, we are able to map out specific (in-utero) periods of vulnerability. Secondly, because of the longitudinal nature of the administrative data, we are able to follow children from infancy to adolescence and thereby map out how exposure impacts immediate health outcomes and sets a trajectory for mental health later in life. Lastly, we explore potential mechanisms through the rich Danish population-level administrative data.

To identify the causal effect of in-utero exposure to air pollutants on the outcomes of interest, we rely on variation in air pollution levels across different pregnancies within families. Exposure to air pollution may be endogenous either due to selection into timing of pregnancy and residential sorting. Some individuals or families may choose to become pregnant at different times, and some individuals might sort into living in higher- or lower-polluted areas. By using sibling comparisons, we limit issues of selection, sorting, and all other time-invariant family characteristics that can lead to endogenous exposure to higher levels of pollution (Currie and Schwandt, 2013). A further challenge is to separate the effect of pollution from other seasonal factors—such as temperature, influenza, and broader time trends that may also impact fetal development. We address this by controlling for month of birth and year of birth, and weather conditions. We show empirically that this approach mitigates potential selection issues and balances the sample (Pei et al., 2018).

First, we show that in a setting generally characterized by low pollution levels, such as Denmark, exposure to air pollution has only modest implications for infant health. Specifically, we find that if the average

[1]Similar trends and disease burdens are found even in countries with free-of-charge publicly provided health care such as Denmark; see e.g., Plana-Ripoll et al. (2022), Christensen et al. (2022), and Danish Health Authority (2022).

exposure to $PM_{2.5}$ increases by 10 $\mu g/m^3$ in the third trimester — a change roughly equivalent to moving from the 10% least exposed to the 10% most exposed—birth weight decreases by 13 grams (a 0.4% reduction) and length at birth by 0.1 centimeters.[2] However, we do not find that in-utero exposure to $PM_{2.5}$ affects other more severe infant health outcomes including the probability of low birth weight, APGAR scores and the probability of being born preterm. These results are more modest than those reported in studies conducted in more polluted environments (e.g., Currie et al. (2009), Bharadwaj et al. (2017), and Ferro et al. (2024)) while comparable to results from Norway with similar pollution levels (Ling, 2023).[3]

Next, we show that despite the limited immediate implications for infant health, even exposure to low levels of air pollution in the prenatal period has far-reaching consequences for mental health in childhood and adolescence. Specifically, we find that an increase in the average exposure to $PM_{2.5}$ by 10 $\mu g/m^3$ in the third trimester increases the probability of being diagnosed with ADHD by age 8 by 0.4 percentage points, corresponding to a 40% increase relative to the baseline mean. Estimates for the probability of being diagnosed with ADHD by age 12 are similar in magnitude but statistically insignificant. Our results indicate that exposure to air pollution increases the probability of being diagnosed earlier, which, according to prior evidence, could be attributed to more severe symptoms of ADHD (Dalsgaard et al., 2014). Further, we find that exposure to $PM_{2.5}$ has a greater negative impact for girls than boys and are driven primarily by more affluent families (proxied by maternal education). The results on ADHD align with previous neurotoxicological evidence, which has demonstrated that laboratory-induced exposure to pollutants in animals disrupts their brain development in a manner similar to that observed in humans with ADHD, autism spectrum disorder, and schizophrenia (Cory-Slechta et al., 2023). The modest effects we find on infant health are not inconsistent with the pronounced results on ADHD, as the fetal origins literature indicates that some in-utero health shocks may remain latent, emerging prominently only in later years (Almond and Currie, 2011; Almond et al., 2018).

Lastly, we show that exposure to air pollution increases the probability of pregnancy complications, driven by an increase in the probability of being diagnosed with pregnancy-related hypertension (HDP). A common pathological feature of air pollution on the human body is an increased inflammatory response (Arias-Pérez et al., 2020) and systemic inflammation from the pulmonary or cardiovascular system can affect neurodevelopmental health (Block and Calderón-Garcidueñas, 2009; Jayaraj et al., 2017). The result on maternal HDP helps suggest inflammation as a key biological mechanism in understanding how in-utero exposure to air pollution affects child development.

A growing number of papers in economics have studied the impact of in-utero exposure to air pollution on both infant health (e.g., Chay and Greenstone (2003a,b), Currie and Neidell (2005), Currie (2009), Currie and Walker (2011), Arceo-Gomez et al. (2016)), child physical health (e.g., Lleras-Muney (2010), Alexander and Schwandt (2022), Ferro et al. (2024), and Klauber et al. (2024)), and later life economic outcomes (e.g., Sanders (2012), Bharadwaj et al. (2017), Isen et al. (2017), and Molina (2021)). These studies are part of a larger literature that highlights how in-utero and early life are critical periods that shape both long-run health and economic trajectories (e.g., Almond and Currie (2011) and Almond et al. (2018)). The study closest to ours is Ferro et al. (2024), who study in-utero exposure to air pollution for both infant health and

[2]While birth weight and LBW are commonly used proxies for infant health, the findings on length at birth reinforce those for birth weight, offering a more detailed perspective on fetal growth. In-utero exposure to air pollution has previously been associated with lower length at birth (Jedrychowski et al., 2009; Estarlich et al., 2011; van den Hooven et al., 2012; Ling, 2023) and length at birth is in itself associated with later-life cognitive ability and educational attainment (Eide et al., 2007; Silventoinen et al., 2023).

[3]It is not straight forward to directly compare the pollution levels in this studies to others since the main pollutant differs. Following European Environment Agency the average level of PM_{10} in Denmark is 25 $\mu g/m^3$. The average exposure to PM_{10} in Ferro et al. (2024) and Currie et al. (2009) is 26.7 $\mu g/m^3$ and 29.7 $\mu g/m^3$.

later child health in Tuscany, Italy. Specifically, they investigate the effects of naturally occurring variation in PM_{10} and deploy different fixed-effect strategies to account for sorting issues. They find negative impacts on birth outcomes as well as increased numbers of hospital visits and increased use of prescription medications in the first 10 years of life. The effects on worsened child health are driven by diagnoses of nervous system diseases (e.g., epilepsy) and prescription medications.

Our paper contributes to this literature in several dimensions. Foremost, we show that in-utero exposure to air pollution negatively impacts mental health outcomes, specifically neurodevelopmental disorders such as ADHD. Mental health, in particular among adolescents, is a growing concern. However, we are not aware of any prior research showing a causal impact of in-utero exposure to air pollution on later life mental health disorders.[4]

Our study also contributes in terms of the data and variation used, as well as the setting and population studied. As highlighted by Currie et al. (2011), Currie et al. (2014), and Currie and Walker (2019), the majority of the existing evidence of in-utero exposure to pollution in economics: i) exploits policy changes (e.g., the Clean Air Act of 1970 in the US) or other highly specific situations (e.g., wildfires) to induce exogenous variation in pollution; ii) relies on data from specific measuring stations; iii) studies highly polluted (and often local) geographical areas; and iv) pays less attention to effects in childhood and adolescence (often because of data availability). In this study, we contribute by relying on sibling comparison to provide plausible random variation in ambient air pollution levels, that is measured using granular satellite-based data that we link to nationwide registers in a low-polluted setting, Denmark.

Lastly, we also contribute to a growing literature in economics that investigates the causes of mental health (Currie, 2009; Layard, 2017; Currie, 2025). This literature has paid less attention to studying mental health conditions during adolescence—the so-called 'missing middle' (Currie and Stabile, 2006; Almond et al., 2018). The study closest to ours in this setting is Persson and Rossin-Slater (2018), who finds negative effects of maternal stress during pregnancy (arising from deaths of close relatives) on a several mental health-related outcomes including the probability of being diagnosed with ADHD in adolescence. We contribute to this literature by studying how in-utero exposure to air pollution affects mental health in childhood and adolescence.

The outline of the paper is as follows: In Section 2, we describe what air pollution is, how pollution levels have evolved in Denmark, the physiological implications of being exposed to air pollution, and the institutional setting surrounding health care in Denmark. In Section 3, we present the data used in the analyses and descriptive statistics. In Section 4, we describe our identifying variation and outline our empirical strategy. In Section 5, we present the findings. Finally, in Section 6, we conclude.

2 Background

2.1 Adverse health consequences of in-utero exposure to air pollution

It is well established in the economics literature that the in-utero period is a critical phase and exposure to adverse environmental insults can have long-lasting health effects (Almond and Currie, 2011; Almond et

[4] Several epidemiological studies have shown positive associations between early life exposure to air pollution and neurodevelopmental disorders (e.g., Myhre et al. (2018), Newbury et al. (2024), and Bradley et al. (2024)). In economics, several studies have examined how short-term exposure to air pollution affects adult mental health. Zhang et al. (2017) and Chen et al. (2024) find negative effects on subjective well-being, psychological distress, and depressive symptoms in China; Sarmiento et al. (2023) and Brehm et al. (2024) find that low emission zones in Germany improve subjective well-being and mental health; and Persico and Marcotte (2022) finds that air pollution increases suicide rates and suicide-related hospitalizations. All five studies differ from ours by focusing on adults and short-term exposure, and the first three rely on self-reported mental health outcomes.

al., 2018). In this paper, we focus on air pollution as an adverse environmental insult. In the following, we highlight potential physiological and pathological explanations proposed in the medical and toxicological literature on how in-utero exposure to air pollution affects both physical and mental health.

Ample epidemiological evidence has linked exposure to air pollution with various diseases affecting the respiratory, cardiovascular, and central nervous systems (Brunekreef and Holgate, 2002; Dominski et al., 2021). Although the biological mechanisms and disease specifics vary, inflammation is consistently identified as a common pathological feature in many diseases associated with air pollution exposure (Arias-Pérez et al., 2020).[5] On average, a person inhales approximately 14 liters of air each day, which comprises about 78% nitrogen, 21% oxygen, and 1% airborne pollution particles. When inhaled, these pollution particles enter the lungs where they can cause irritation and inflammatory states in various parts of the airways. This can lead to several respiratory diseases, including asthma (Guarnieri and Balmes, 2014), respiratory conditions (Jans et al., 2018; Simeonova et al., 2021), and chronic obstructive pulmonary disease (Feng et al., 2016). Sufficiently small pollution particles, including particulate matter ($PM_{2.5}$), are capable of crossing the blood-air barrier and entering the bloodstream. Once in the circulatory system, these particles can cause an inflammatory state, which may lead to a range of cardiovascular diseases including obesity (Shi et al., 2022), hypertension (including pregnancy-related hypertension) (Pedersen et al., 2014), and heart failure (Shah et al., 2013). Once in the bloodstream, pollution particles can also cross the blood-brain barrier, affecting the central nervous system (brain and spinal cord). The specific molecular mechanisms by which pollution impacts the central nervous system are still a topic of debate within the medical and toxicological literature. However, (neuro)inflammation is widely recognized as a significant mechanism (Block and Calderón-Garcidueñas, 2009; Jayaraj et al., 2017). Furthermore, inflammation is considered an etiological factor in both neurodevelopmental disorders (such as ADHD and autism spectrum disorder) and neurodegenerative disorders (such as Alzheimer's disease and Parkinson's disease) (Santos et al., 2022; Ransohoff, 2016).

The biological mechanisms outlined above depart from the perspective of a (grown) person. The mechanisms of course do not directly apply to fetal development. For fetal development in general and the impact of in-utero exposure to air pollution specifically, the placenta plays an important mediating role. The placenta is a temporary organ whose primary function is to provide the fetus with oxygen, nutrition, and relevant hormones while removing carbon dioxide and other waste. In that sense, it acts as a gatekeeper between mother and fetus. There are two complementary ways air pollution, that is floating around the maternal circulatory system, can affect the fetus (Luyten et al., 2018). First, sufficiently small particles can penetrate the placenta and even smaller particles can enter the bloodstream of the fetus. As an example, black carbon, a small pollution particle that constitutes a significant part of $PM_{2.5}$, has been found on the fetal side of the placenta (Bové et al., 2019) as well as in fetal organ tissue (Bongaerts et al., 2022). Second, maternal inflammation due to air pollution may also enter the placenta and pass to the fetus (Nachman et al., 2016; Stapleton, 2016; Luyten et al., 2018). Irrespective of the specific molecular mechanisms involved, the main pathway is that disruptions in the placenta caused by in-utero exposure to air pollution can impair fetal development. These disturbances may lead to changes in epigenetic programming, resulting in deterioration of physiological and neurological development. Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by symptoms of impulsivity, hyperactivity, and an inability to maintain concentration. The specific etiology (why ADHD occurs) and pathology (how it manifests in the brain) remain subjects of ongoing discussion in the medical literature (Posner et al., 2020; Thapar et

[5]Inflammation is the body's natural physiological response to illnesses, infections, environmental toxins, or injuries.

al., 2024). There is a large genetic component to ADHD, with a heritability of around 75–80%, but—as highlighted in this study—environmental and societal factors can also play a role. However, the specific biological mechanisms including potential fetal programming from exposure to environmental toxins during the in-utero period are still not fully mapped out but with inflammation and stress highlighted as potential channels (Thapar et al., 2024).

2.2 Air pollution

Air pollution is a mix of hazardous substances in the atmosphere originating from both natural and human-made sources.[6] The sources of human-made air pollution include vehicle emissions, fuel oils and natural gas used to heat homes, by-products of manufacturing and power generation—particularly from coal-fueled power plants—and fumes from chemical production. The composition and concentration of air pollution can vary greatly depending on geographic location, weather conditions, and the types of emitting sources.

In this study, we focus primarily on particulate matter ($PM_{2.5}$) as it is one of the most dangerous pollutants according to WHO. Particulate matter can take many forms and characterize a group of tiny liquid or solid particles—typically chemical components such as sulfate, nitrate, organic and black carbon, mineral dust, and sea spray—emitted or formed in the atmosphere. One of the most granular forms of PM is $PM_{2.5}$, which is 30 times thinner than a human hair and can easily penetrate lung tissue when inhaled. The most common human-made sources of $PM_{2.5}$ include coal-burning power plants, vehicle emissions, and industrial activities.

The local weather plays an important role in air quality and pollution. Air pollution particles are typically very light, and wind (depending on speed and direction) can transport and distribute pollutants over large distances. Temperatures may also affect pollution levels. Colder weather often induces increased electricity use and heating from indoor activities, as well as increased automobile use, which in turn increases human-made pollution. Lastly, heavy rain forces the heaviest pollutants to the ground and can help dissolve certain pollutants.

2.3 Air pollution in Denmark

Denmark and the rest of Scandinavia are generally characterized by low air pollution levels. In recent years, Denmark has most often been below the pollution levels recommended by the European Union and WHO.[7] Despite being a low-polluted environment, there is still substantial variation in air pollution levels across time and space. This variation plays a key role in the identification strategy used in this study.

Figure 1 shows how air pollution (exemplified by $PM_{2.5}$) has decreased substantially in the last 25 years in Denmark.[8] In 1998, the $PM_{2.5}$ level was on average 13 $\mu g/m^3$ and decreased to around 8 $\mu g/m^3$ in 2017.

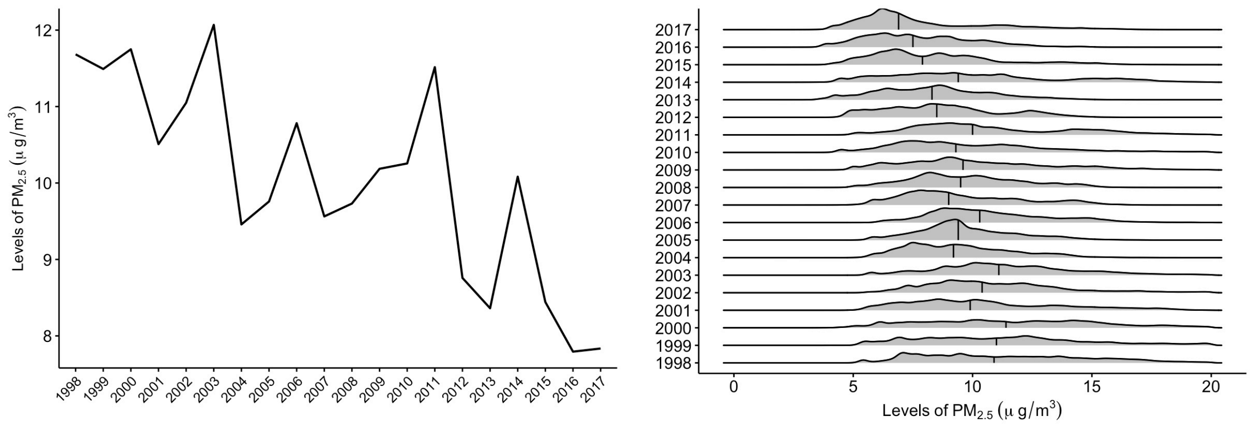
In Denmark, like most developed countries, pollutants exhibit a strong seasonal pattern. For $PM_{2.5}$, levels are higher during the winter and lower in the summer. Figure 2 shows that, on average, the level of

[6] Natural sources include volcanic eruptions, wildfires, and dust storms, which release pollutants into the atmosphere. These sources are very uncommon in a Danish setting.

[7] WHO's guidelines from 2021 state that the 24-hour average of $PM_{2.5}$ should not exceed 15 $\mu g/m^3$. EU's guidelines from 2008 state that the annual mean of $PM_{2.5}$ should not exceed 20 $\mu g/m^3$.

[8] The decrease in air pollution started early; however, Figure 1 shows the trend for the period studied in this paper. The figure focuses on $PM_{2.5}$ as our main measure of air pollution; however, different types of air pollution are highly correlated since they stem from the same sources. The correlation between $PM_{2.5}$ and carbon monoxide (CO) is estimated to be 75%, with similar trends observed for CO. The decrease is observed both in the pollution levels (air quality) and in emissions from different sources (Ellermann et al., 2023).

Figure 1: Yearly levels of $PM_{2.5}$



(a) Mean yearly variation (b) Distribution of the variation

Notes: The figure shows trends in $PM_{2.5}$ from 1998 to 2017. Panel A shows the mean levels and Panel B shows the distribution. The figures are based on all household addresses.

$PM_{2.5}$ is highest in winter, particularly in January and February, and lowest during summer, particularly in August. These seasonal fluctuations are driven by both weather conditions and seasonal human behavior, such as increased heating and car use during colder months, combined with meteorological factors that limit pollutant dispersion.

The time trend and the seasonal patterns are the main drivers of variation in pollution in Denmark. For example, the time trends and seasonal patterns explains around 52% of the variation in $PM_{2.5}$ levels, while considering geographical differences (at a municipality level) only accounts for 4% of the variation.[9] The residual variation after controlling for time and seasonality is the variation that we utilize for identification in this study.

2.4 The Danish health care context

Denmark has a universal, publicly funded healthcare system where all residents are covered by the National Health Insurance. This entity ensures both primary and secondary health care services free of charge to resident citizens. Most hospitals are publicly operated, whereas the Primary Care Providers (PCPs) operate in small practices as private entities. PCPs are reimbursed based on the services provided and act as gatekeepers to specialists and hospitals.[10][11]

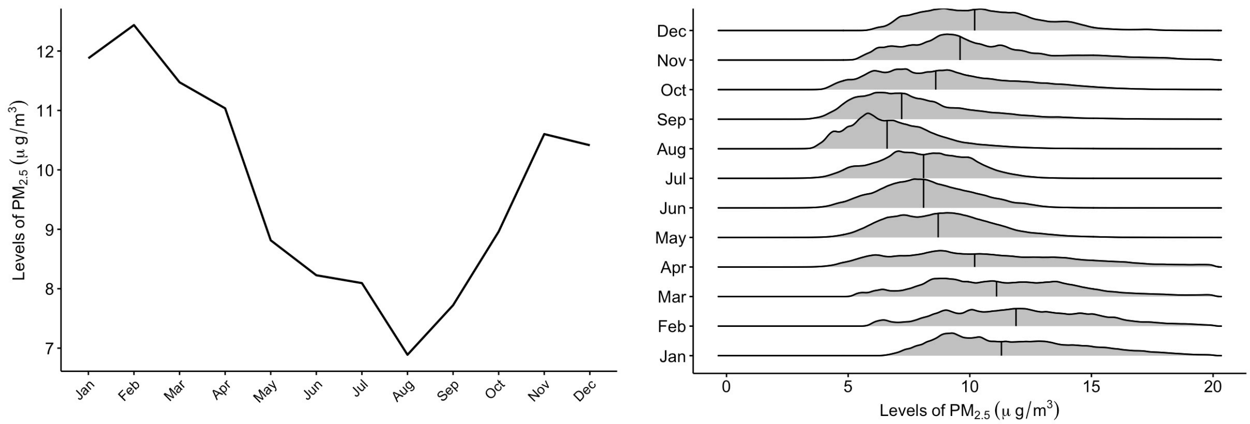
When a woman suspects or discovers a (potential) pregnancy, she can visit her PCP to have the pregnancy confirmed. Following this visit, pregnant women are offered a number of routine check-ups and tests during their pregnancy, as well as delivery at a hospital or maternity ward and home visits by a nurse in the weeks after giving birth. In Denmark, approximately 60,000 children are born each year, and 99% of deliveries take place at a hospitals. Both maternal and perinatal mortality rates are low and have been declining for

[9] The numbers for explained variation are the R^2 from i) a regression of monthly levels of $PM_{2.5}$ on year and month fixed effects, and ii) a regression on year, month and municipality fixed effects. The former results in an $R^2 = 0.52$ while the latter results in an $R^2 = 0.56$ suggesting that accounting for municipality differences only contributes to explaining an additional 4% of the variation.

[10] All Danish residents have the right to be assigned a PCP, preferably of their own choice.

[11] For a more detailed description of the Danish health care system, please see Schmidt et al. (2019).

Figure 2: Monthly levels of $PM_{2.5}$



(a) Mean monthly variation

(b) Distribution of the variation

Notes: The figure shows seasonal patterns in $PM_{2.5}$ from 1998 to 2017. Panel A shows the mean levels and Panel B shows the distribution. The figures are based on all household addresses.

the past 40 years (Wüst, 2022).[12]

Mental health treatment in Denmark is largely based around psychiatric clinics providing outpatient treatment and extensive crisis support, with designated clinics for youth and adolescent treatment.[13] Diagnoses for neurodevelopmental disorders, including ADHD, in youth (under 18 years) can only be made by a child psychiatrist. Treatment for ADHD in children consists of a combination of medication and therapy.

3 Data and descriptive statistics

3.1 Data sources

The data used in this study is derived from several administrative registers, all maintained by Statistics Denmark, complemented with pollution data from Van Donkelaar et al. (2021) and weather data from the Danish Meteorological Institute (DMI). We link the data from different registers using the unique Danish civil registration number, which allows matching a given sample of all children born in Denmark, their parents, and their siblings. Additionally, we link local measures of air pollution and weather to the administrative registers.

We identify the population used in the study through the Birth Register, which includes information about the universe of births in Denmark starting from 1970 up to 2017. For each child, the data includes information on the exact date of birth, gender, gestational age, birth weight, length at birth, plurality, APGAR score, and information about the mother. We derive information on childrens mental disorder diagnoses, from the National Patient Register, which includes all in- and outpatient hospital admissions and corresponding ICD-10 diagnoses from 1980 to 2018. We link these data to other register data that provide information on demographic characteristics, such as parental age, education, ethnicity, and marital status at birth.

Data on pollution concentrations are provided by Van Donkelaar et al. (2021) at a 1x1 km grid covering

[12] For a more detailed description of prenatal and early life care in Denmark, please see Danielsdottir and Ingudottir (2020).
[13] All major hospitals still provide inpatient care in psychiatric wards for severe mental disorders.

the whole of Denmark in the period from 1998 to 2020. The data consist of levels of $PM_{2.5}$ measured as $\mu g/m^3$ at monthly level. We assign all Danish household addresses by matching the longitude and latitude of the addresses to the 1x1 km cell.[14] The pollution data consists of estimated concentrations of $PM_{2.5}$ from state-of-the-art environmental science research that combines spatially-continuous measurements of pollution from satellites (i.e., aerosol optical depth) with other observable pollution correlates such as emissions inventories, chemical transport models, land use characteristics, and weather patterns. It is additionally combined with information from simulation- and monitor-based sources. This data source is validated against WHO monitor stations and has a correlation of 0.68 for Europe (Hammer et al., 2020; Van Donkelaar et al., 2021). Recent economic applications include Fowlie et al. (2019), Alexander and Schwandt (2022), Brehm et al. (2024), and Currie et al. (2023).

Additionally, we include meteorological data from the DMI. This data consists of daily information on precipitation, temperature, and cloud cover. We use data from 102 different monitoring stations placed throughout Denmark. For all municipalities without a weather station, the observations are computed as an average from the neighboring municipalities that reflect common tendencies. If there are no measurement stations within this region, we impute from the national daily average.

3.2 Sample(s)

In our empirical analysis, we are interested in estimating the impact of in-utero exposure to air pollutants on a range of outcomes measured from infancy to adolescence. Because all relevant outcomes are not available for all potential birth cohorts — either because registers are not available or because the later-born cohorts are simply too young for the outcomes to be observed yet — we make use of a range of different (sub)samples that we outline in the following.

We sample all live singleton births in Denmark in the period 1999–2017. We restrict the sample to observations where maternal ID and birth weight are not missing. We further restrict the sample to observations where the mother's address during pregnancy can be mapped to 1 km $\times$ 1 km grid cells of pollution data. As our empirical analysis relies on within-family comparisons, we further limit our sample to children with at least one sibling (with the same mother) born within the same period (1999 to 2017). These restrictions leave 812,504 children for whom we measure infant health. We then further restrict our sample to children who are at least 12 years old in 2022 to be able to measure mental health. This leaves us with a sample consisting of 478,280 children. The sample selection is shown in Table A1, while Figure A1 shows the cohorts included and when in time the outcome of interest is measured.

3.3 Variables

For the analysis on infant health, we focus on five outcomes of interest: i) birth weight, ii) the probability of being born with low birth weight (LBW), iii) length at birth, iv) APGAR score and v) the probability of being born preterm. Birth weight is measured in grams, LBW is defined as a birth weight below 2500 grams, length at birth is measured in centimeters and the probability of being born preterm is determined as the probability of being born before gestational week 37. While birth weight, LBW and preterm are commonly used proxies for infant health, length at birth is in itself associated with later life cognitive ability and educational attainment (Eide et al., 2007; Silventoinen et al., 2023) and in-utero exposure to air pollution

[14] The longitude and latitude of all Danish addresses are obtained through an API provided by DAWA (Danmarks Adressers Web API).

has previously been associated with lower length at birth (Jedrychowski et al., 2009; Estarlich et al., 2011; van den Hooven et al., 2012; Ling, 2023). The APGAR score summarizes children's health status across five categories: skin color, heart rate, reflexes, muscle tone, and breathing effort. Assigning each category a score of zero, one, or two results in a total score on a ten-point scale, where a higher number reflects better health.

For mental health, we focus on both in- or outpatient hospitalizations between 1999 and 2022 with i) the probability of having an ADHD diagnosis, and ii) the probability of having any psychiatric diagnosis.[15] We measure mental health outcomes in two periods: i) from age 5–8, and ii) from age 5–12. As neurodevelopmental disorders are viewed as chronic once the child is diagnosed, we include the youngest children in both periods. Figure A2 shows the distribution of ages when the first diagnosis is given.

Pollution data is constructed on a trimester level. For our main measurement of pollution, $PM_{2.5}$ from Van Donkelaar et al. (2021), we calculate an average of maternal exposure at the trimester level. Since the pollution data is on a monthly level, we use the gestational length to calculate an average to measure the exposure within each trimester and the entire pregnancy.[16]

As control variables, we include meteorological data, where we use daily weather information to derive trimester-level information. For each trimester, we calculate the average temperature, total precipitation (measured in cm), and average percentage cloud cover.[17] We also include information about maternal smoking, maternal age at birth, and the sex of the child. All information is obtained through Danish administrative data.

3.4 Descriptive statistics

Table 1 presents descriptive statistics for the sibling sample used in the analysis and the full background population, as well as the standardized difference in means (SDM). Panel A reports information for the children, while Panel B reports information on the mothers. Our sample consists of 812,793 children in 357,726 families. Half of the children included in our sample are girls, and the share of children whose mother is of Danish origin is around 85%.The average in-utero exposure to $PM_{2.5}$ is around 11 $\mu g/m^3$. The time of birth is somewhat equally distributed across the year, with slightly fewer births during winter months and slightly more births during the summer. Mothers are, on average, 29 years old when they have their first child, and has on average around 2.5 children. They, on average, earn just below 28 thousand USD a year. Around 30% of mothers are unskilled, 30% have vocational education or short-cycle higher education, and lastly, 39% have a college education (including medium-cycle higher education and university).[18] Around 15% of the mothers smoked at some point during the pregnancy, and the average BMI is around 24.5. The SDM shows that there are no systematic differences in either individual or maternal characteristics between the sibling sample used in the analysis and the full background population, except for the number of children in the family, due to our restriction to families with two or more children.

[15] ADHD diagnoses are defined as ICD-10 codes F90 and F988. Any psychiatric diagnosis is defined as any ICD-10 code within F. The variable is constructed as a dummy and equals one if the child has at least one relevant diagnosis.

[16] Based on the date of birth and gestational length we calculate the estimated day of conception and use this to divide the pregnancy into trimesters. To match trimesters to months, we weight the monthly measures by the share of the month that is part of a given trimester.

[17] In a robustness check shown in Section 5.3 we control for temperatures in a more flexible way by including the share of days in each trimester where temperatures falls into bins of $5°C$ starting from below $0°C$ and ending with above $20°C$

[18] *Unskilled* is defined as having the highest completed level of education corresponding to either primary education, general upper secondary education, or missing.

Table 1: Descriptive Statistics

	Population		Sample		SDM
	(1) Mean	(2) SD	(3) Mean	(4) SD	(5)
Panel A: Individual characteristics					
Female	0.49	0.50	0.49	0.50	-0.001
Season of birth					
Winter	0.23	0.42	0.23	0.42	0.000
Spring	0.25	0.43	0.25	0.43	0.000
Summer	0.27	0.44	0.27	0.44	0.000
Autumn	0.25	0.43	0.25	0.43	0.000
Average exposure to $PM_{2.5}$ during					
1st trimester	11.25	2.42	11.25	2.48	0.002
2nd trimester	11.25	2.48	11.27	2.50	0.005
3rd trimester	11.11	2.50	11.13	2.52	0.004
Total	11.20	1.61	11.21	1.53	0.006
Panel B: Maternal characteristics at birth					
Native	0.85	0.36	0.86	0.35	0.027
Age (at birth)	30.13	4.90	29.93	4.71	-0.031
Number of children	2.02	0.97	2.46	0.82	0.35
Income (in USD)	27,234	21,068	27,735	20,841	0.017
Level of education					
Unskilled	0.30	0.46	0.29	0.45	-0.023
Vocational education	0.29	0.45	0.28	0.45	-0.023
Short further education	0.05	0.22	0.05	0.22	0.000
College	0.37	0.48	0.39	0.49	0.022
Smoking	0.15	0.37	0.13	0.34	-0.033
BMI	24.48	7.88	24.50	7.91	0.002
Number of children	1,135,562		812,504		

Notes: The table presents descriptive statistics for the full background population and the sibling sample (used in the analysis). Each cell in Column 1 and 3 shows means of selected covariates, and column (2) and (4) show the corresponding standard deviation. Column (5) presents the standardized difference in means (SDM) (or the standardized bias); i.e., the difference in means as a share of the square root of the average sample variances of the background population (column 1) and the sibling sample (column 3). For season-of-birth variables: Winter is defined as: December, January, and February. Spring is defined as: March, April, and May. Summer is defined as: June, July, and August. Autumn is defined as: September, October, and November. Maternal income is computed as an average of the three years prior to child birth and converted from DKK to USD at a rate of 6.58. Maternal education is determined at the year of child birth. To calculate the number of children only children born in or after 1999 is used.

4 Empirical strategy

4.1 Identifying variation

To identify the causal effect of in-utero exposure to air pollution on infant health and longer-run mental health care outcomes, we rely on variation in air pollution across different pregnancies within families.

As highlighted in Section 2, pollution levels in Denmark vary substantially both across time and seasonally within the year. Since 1998, $PM_{2.5}$ has decreased by 32%. Further, $PM_{2.5}$ levels are, on average, 22% higher in December than in July. A woman who is pregnant during the winter months will be exposed to a substantially higher level of air pollution than if the majority of her pregnancy occurred during summer months. However, there may be several factors confounding the relationship between exposure to air pollution and later life health. First, variation in pollution levels may coincide with other seasonally varying factors, such as temperatures, infectious diseases, or economic conditions, which could affect the outcomes of interest. Second, the timing of when the child is conceived may be correlated with other factors such as socioeconomic background, which could also affect the outcomes of interest.

To overcome the first challenge, we control for weather, time (year of birth fixed effects), and seasonality (month of birth fixed effects).

The second major concern is that women with different preferences or characteristics (exemplified by their observed socioeconomic background) tend to conceive in different months. In particular, we worry that women from a lower socioeconomic background have a higher probability of giving birth during winter months. If such sorting exists, exposure to air pollution is not independent of other factors that also affect child outcomes. Relatedly, residential sorting into different neighborhoods is a concern. We worry that families from lower socioeconomic backgrounds tend to sort into neighborhoods that are more polluted, while families from higher socioeconomic backgrounds tend to sort into neighborhoods that are less polluted. As a consequence of residential sorting, exposure to air pollution will be endogenous.

To account for these two types of sorting, we follow Currie and Schwandt (2013), Currie et al. (2009) and Bharadwaj et al. (2017), and make a sibling comparison which effectively controls for maternal and residential selection into pollution exposure during pregnancy.

Panel A in Figure 3 presents the levels of pollution during 3rd trimester included in our analysis. The figure shows that there is variation in the levels that each fetus is exposed to during the last period of the pregnancy. Panel B in Figure 3 presents the residual distribution of pollution during the 3rd trimester after regressing $PM_{2.5}$ on temperature, month-, year- and family fixed effects.[19] The figure highlights that even after controlling for these trends and family effects, there is still substantial variation in pollution levels.[20] This is the plausibly random variation in exposure to pollution that drives the identification in our analysis.[21]

4.2 Econometric approach

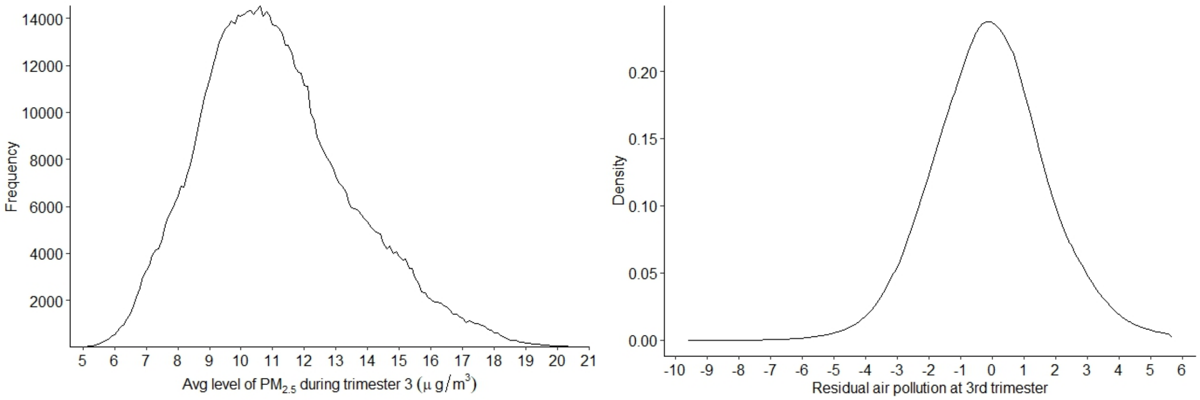
We estimate the following empirical model to analyze the impact of in-utero exposure on infant and child outcomes at a trimester level:

[19] Similar patterns the first and second trimester is presented in Figure A3 and A4.

[20] In addition we also plot the residual variation across both years and seasons in Figures A5, A6, and A7. The figures show that there is substantial variation across both months and years.

[21] As highlighted in Section 2 air pollution can move across large distances and 75% of air pollution observed in Denmark originates from continental Europe.

Figure 3: Variation in pollution levels in 3rd trimester



(a) Raw levels of pollution (b) Residual pollution

Notes: Panel A shows the individual pollution levels during the 3rd trimester. Panel B shows the residuals from a regression exposure to $PM_{2.5}$ in the 3rd on average temperatures and year-, month- and family fixed effects.

$$Y_{if} = \alpha + \sum_{t=1}^{3} \beta_t E_{ift} + \theta_{BirthYear} + \tau_{BirthMonth} + \mu_f + \delta X_{if} + \sum \mu_t W_{if} + u_{if} \tag{1}$$

where i indexes individuals, f families, and t trimesters. Y_{if} measures child health at birth and child- and adolescent mental health outcomes - all defined in Section 3. E_{ift} captures the average pollution in each trimester. We follow Schwandt (2018) and Bharadwaj et al. (2017) and compute trimesters using the date of birth and the gestational age. We assign weeks 1-13 to trimester 1, weeks 14-26 to trimester 2, and weeks 27-birth as trimester 3. $\theta_{BirthYear}$ is a birth year fixed effect, $\tau_{BirthMonth}$ is a birth month fixed effect, and μ_f is a mother fixed effect. X_{if} is a set of child and mother time-variant characteristics, including the maternal age at the time of birth, the sex of the child, and maternal smoking status. W_{if} captures the average temperature for child i in each trimester, as temperature may have a direct effect on maternal and fetal health (Deschenes et al., 2009), but also on the formation of pollution.

The parameters of interest are β_1, β_2, and β_3 which, under the relevant assumptions discussed below, capture the causal effect of exposure to air pollution during the first, second, and third trimesters, respectively.

Additionally, we estimate a specification (Equation 2) that captures average pollution during the entire pregnancy:

$$Y_{if} = \alpha + \beta_{total} E_{ifTotal} + \theta_{BirthYear} + \tau_{BirthMonth} + \mu_f + \delta X_{if} + \gamma W_{if} + u_{if} \tag{2}$$

4.3 Validity

The empirical model described above aims to analyze the causal relationship between in-utero exposure to air pollution, infant health, and later life mental health. The identifying assumption in the equation above is that, after controlling for time trends, seasonality, mother fixed effects, observable maternal characteristics, and weather controls, exposure to air pollution is uncorrelated with the error term, u_{igt}. In other words, $\beta_{trimester}$ captures the effect of exposure to air pollution if this exposure is independent of factors that may also affect child outcomes.

To test the independence assumption we implement the balancing test proposed by Pei et al. (2018). The idea behind the test is to estimate a model similar to Equation 1 but with time-varying maternal characteristics as the dependent variables. Specifically, we estimate the following three models.

$$Y_{if} = \alpha + \sum_{t=1}^{3} \beta_t E_{ift} + u_{if} \tag{3}$$

$$Y_{if} = \alpha + \sum_{t=1}^{3} \beta_t E_{ift} + \theta_{BirthYear} + \tau_{BirthMonth} + \delta X_{if} + \sum \tau W_{if} + u_{if} \tag{4}$$

$$Y_{if} = \alpha + \sum_{t=1}^{3} \beta_t E_{ift} + \theta_{BirthYear} + \tau_{BirthMonth} + \mu_f + \delta X_{if} + \sum \tau W_{if} + u_{if} \tag{5}$$

For Equations 3-5, the notation and variables are identical to Equation 1, except that now the dependent variable, Y_{if}, represents selected maternal characteristics, including earnings, education, ethnicity, smoking, and BMI. Equation 3 regresses the maternal characteristic of choice on in-utero exposure to air pollution. Equation 4 also includes birth year fixed effects, seasonality fixed effects, time-varying maternal characteristics (excluding the characteristic used as the dependent variable to avoid endogeneity concerns) and average temperature on a trimester level. Lastly, Equation 5 includes family fixed effects. These balancing regressions examine whether there is selection of mothers into exposure to different pollution levels and the extent to which sibling comparisons eliminate this selection. Pei et al. (2018) highlight that this approach to balance testing (estimating models with potential control variables as the dependent variable in the regression model) is particularly valuable when proxies for true underlying potential confounders are poorly measured. This method contrasts with the more standard coefficient comparison test, where controls are sequentially added as independent variables to observe changes in the estimate of interest.

The results for the Pei approach are shown in Table 2. Panel A shows the coefficients from a regression based on Equation 3. Here we observe large and statistically significant coefficient estimates, indicating selection into different levels of pollution. Panel B incorporates additional controls as presented in Equation 4. The coefficient estimates are smaller in magnitude compared to Panel A, but remain statistically significant. This indicates that controlling for selected time-varying characteristics does not eliminate selection into different levels of pollution. Panel C shows the same regressions as in Panel B, but where we additionally include mother fixed effects (Equation 5). Since the mother's origin is constant over time, mother fixed effects perfectly control for this characteristic, and it is not possible to estimate this regression. Unlike Panels A and B, these estimates are much smaller in magnitude and most are statistically insignificant. This indicates that maternal fixed effects effectively control for factors influencing pregnancy timing and residential sorting. Consequently, we argue that exposure to air pollution can be treated as exogenous given the inclusion of these controls and fixed effects.

5 Results

5.1 Air pollution and infant health

The first step of our empirical analysis is to establish the effect of in-utero exposure on infant health. Table 3 shows the results of the effect of air pollution on infant health outcomes. We find that an increase in exposure to air pollution affects birth weight and length at birth negatively across all trimesters, and the

Table 2: Balancing regressions of maternal characteristics on air pollution

	(1) Mother foreign	(3) Morther's age	(3) Log(income)	(4) P(college)	(5) P(smoking)	(6) Maternal BMI
Panel A: No controls						
Trimester 1 * 10	-0.031***	-0.581***	-0.193***	-0.113***	0.065***	-0.100***
	(0.002)	(0.021)	(0.006)	(0.002)	(0.002)	(0.044)
Trimester 2 * 10	-0.029***	-0.252***	-0.061***	-0.041***	0.011***	-0.171***
	(0.001)	(0.020)	(0.005)	(0.002)	(0.002)	(0.045)
Trimester 3 * 10	-0.035***	-0.612***	-0.306***	-0.114***	0.074***	0.003
	(0.001)	(0.020)	(0.005)	(0.002)	(0.001)	(0.045)
Panel B: Baseline Controls						
Trimester 1 * 10	-0.003	-0.309***	-0.052***	0.012***	-0.010***	-0.068
	(0.002)	(0.033)	(0.008)	(0.003)	(0.002)	(0.066)
Trimester 2 * 10	0.012***	-0.177***	-0.026***	0.009***	0.015***	-0.057
	(0.002)	(0.033)	(0.008)	(0.003)	(0.002)	(0.069)
Trimester 3 * 10	0.014***	-0.337***	-0.073***	0.008**	-0.018***	0.021
	(0.003)	(0.036)	(0.009)	(0.003)	(0.003)	(0.077)
Panel C: Baseline Controls + Mother FE						
Trimester 1 * 10		-0.002	-0.012	-0.004*	-0.000	0.103
		(0.003)	(0.010)	(0.002)	(0.002)	(0.087)
Trimester 2 * 10		-0.013***	0.012	-0.005**	-0.002	0.246***
		(0.003)	(0.010)	(0.002)	(0.002)	(0.090)
Trimester 3 * 10		-0.002	-0.014	-0.001	-0.003	0.019
		(0.003)	(0.011)	(0.002)	(0.003)	(0.105)
N(A)	1,131,838	1,134,959	1,012,592	1,111,674	1,134,959	792,700
N(B)	1,113,020	1,116,562	996,950	1,094,101	1,116,562	781,320
N(C)		812,793	706,342	798,032	812,504	514,595

Notes: Baseline controls include fixed effects for year and month of birth, and (unless chosen as depended variable) sex of the child, maternal age at birth, average temperature each trimester. The sample include all mothers who gave birth between 1999 and 2017. The sample size vary across outcomes due to missings in the depended variable. Standard errors are clustered on the family level.*** p<0.01, ** p<0.05, * p<0.1.

effect is strongest in the third trimester, corresponding to the fact that this is the period during which the fetus gains the most weight and undergoes the final stages of physical development (Schwandt, 2018). For the other infant health outcomes, we do not find any results indicating that an increase in exposure to air pollution affects infant health negatively.

In Panel A in Table 3, Column (1) shows the results for birth weight. We observe a clear pattern in which the negative effect of in-utero exposure to air pollution becomes more pronounced across trimesters. Specifically, for the third trimester, we find that when the average exposure to $PM_{2.5}$ increases by 10 $\mu g/m^3$ birth weight decreases by 13 grams, corresponding to a 0.4% reduction relative to the mean birth weight of 3,527 grams. An increase of 10 $\mu g/m^3$ corresponds to moving from the 10% least exposed during the third trimester to the 10% most exposed.

Column (2) shows the results for the probability of having a low birth weight (birth weight below 2,500 grams). We find that when the average exposure to $PM_{2.5}$ increases by 10 $\mu g/m^3$ in the third trimester, the probability of low birth weight increases by 0.2 percentage points. However, this estimate is insignificant.

Column (3) shows the results for the length at birth. In line with the results in Column (1) we observe more pronounced effects in later trimesters. Specifically, we find that when the average exposure to $PM_{2.5}$ increases by 10 $\mu g/m^3$ in the third trimester, the length decreases by 0.1 cm. Column (4) show the results for APGAR score. We find that the coefficient estimates across all trimesters are negative, suggesting a decrease in the APGAR score. However, all the estimates are insignificant. Column (5) show that results on the probability of being born preterm. All the points estimates as close to 0 and insignificant. This suggests that the effects on birth weight and length are not driven by preterm births.

Panel B in Table 3 shows the effect of cumulative exposure to $PM_{2.5}$ during the entire pregnancy. The coefficient estimates are consistent with those in Panel A and reflect the cumulative effect across trimesters.

Table 3: In-utero exposure to air pollution and birth outcomes

	(1) Birth weight	(2) P(LBW)	(3) Length	(4) Apgar score	(5) P(preterm)
Panel A: Trimester					
Trimester 1 * 10	-2.27	-0.001	-0.075	0.000	-0.002
	(4.99)	(0.002)	(0.055)	(0.007)	(0.002)
Trimester 2 * 10	-9.11*	0.001	-0.106**	-0.001	-0.001
	(4.78)	(0.002)	(0.051)	(0.007)	(0.002)
Trimester 3 * 10	-13.36**	0.002	-0.111*	-0.002	-0.001
	(5.40)	(0.002)	(0.058)	(0.008)	(0.002)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.52
Panel B: Total					
Total * 10	-20.69**	0.001	-0.264***	-0.008	-0.002
	(9.21)	(0.004)	(0.099)	(0.013)	(0.003)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.52
Mean	3526	.04	51.58	9.87	.03

Notes: Pollution is measured at the mother's home address. In Panel B, the total is calculated as the average during the entire pregnancy. Number of observations varies between Columns, since not all outcomes are measured for especially the earliest cohorts. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, and the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Relative to the previous literature on air pollution and infant health, the effects we find are smaller in magnitude. However, it is straight forward to directly compare our results, since 1) the type of pollutant studied are different, 2) the measurement of the pollution is different, and 3) background air pollution levels are typically higher compared to levels in Denmark. Currie et al. (2009) find that a 1 ppm[22] increase in exposure to carbon monoxide (CO) in New Jersey decreases birth weight by 16.65 grams, corresponding to a reduction of 0.5% relative to the baseline mean birth weight. Similarly, Bharadwaj et al. (2017) find that a 1 ppm increase in exposure to CO in Chile during the third trimester decreases birth weight by 12.6 grams corresponding to a reduction of 0.54% relative to the baseline mean birth weight. CO is generally considered a more harmful pollutant due to its size and chemical nature, hence it is expected that the magnitude of the effects estimated in studies that use CO would be larger than in our study. Similarly, both studies are conducted in areas characterized by relatively high levels of background pollution. In a more related setting, Ling (2023) find that a 10 $\mu g/m^3$ increase in exposure to nitric oxide (NO) during the third trimester in Norway decreases birth weight by 13.9 grams and length at birth by 0.52 centimeter, corresponding to a reduction of 0.4% and 1% respectively relative to the baseline means. While $PM_{2.5}$ and NO are also not directly comparable pollutants the results are similar in magnitude to the results presented above.

Further, our results on birth weight also align with findings from previous studies of other mild early-life stressors. Almond and Mazumder (2011) find that when Ramadan (a religious period characterized by fasting) coincides with pregnancy, birth weight decreases by 18 grams, corresponding to a 0.5% reduction relative to baseline mean birth weight. Currie and Schwandt (2013) find that conception in June and August (relative to January) decreases birth weight by 8 grams, corresponding to a 0.23% reduction. More severely, Schwandt (2018) finds that maternal hospitalization for influenza during pregnancy decreases birth weight by 84.5 grams, corresponding to a 2.4% reduction. Lastly, Persson and Rossin-Slater (2018) find that the stress caused by the death of a close relative during pregnancy decreases birth weight by 11.5 grams, corresponding to 0.3%.

To uncover potential heterogeneity in the effects of $PM_{2.5}$ exposure on infant health, we investigate the estimated effects across sex[23] (Table A2) and socioeconomic status[24] (Table A3). For birth weight, we find that the effect in the third trimester are around one third larger for boys than girls, however the difference is not significant. Consistent with the main results, we do not observe any significant effects for the other infant health outcomes.[25] For socioeconomic status, proxied by maternal education at birth, we find a positive gradient with larger reductions in birth weight for less affluent families. Again, we do observe any major patterns for the other infant health outcomes.

5.2 Air pollution and mental health

Next, we investigate the effect of air pollution on mental health during childhood and early adolescence. Table 4 presents effects across different age groups and by trimester (Column (1)–(2)) as well as cumulative exposure during pregnancy (Column (3)–(4)). In this section, we strictly focus on ADHD and any types of mental health diagnosis.

Panel A, Column (1), shows the effects of in-utero exposure to $PM_{2.5}$ on the probability of being diagnosed

[22] Ppm (parts per million) are not directly translatable to $\mu g/m^3$, as it requires specific information on temperature and atmospheric pressure (atm). For CO, 1 $\mu g/m^3 \approx 0.000873$ ppm at 25°C and 1 atm.

[23] In this specification, we estimate heterogeneity by interacting exposure to $PM_{2.5}$ with sex. This approach allows us to utilize the full sample size and incorporate high-dimensional fixed effects simultaneously.

[24] In this specification, we proxy socioeconomic status by maternal level of education at birth. We estimate heterogeneity by splitting the sample into three groups based on maternal level of education. Sibling pairs in which the mother attains a different educational level between siblings are dropped for this analysis.

[25] This is expected as the sample is split somewhat evenly between girls and boys, and a positive effect on, for example, the probability of having low birth weight for girls would imply a negative effect for boys (i.e., that boys benefit from more exposure to air pollution).

with ADHD. We find an increased probability of being diagnosed with ADHD by age 8 for children exposed to pollution during the second and third trimesters. A 10 $\mu g/m^3$ increase during the third trimester raise the probability of an ADHD diagnosis by 0.4 percentage points, for both age groups. This corresponds to a 40% increase relative to the baseline mean probability of having an ADHD diagnosis. We find similar results in Column (3), where our results show that when the average exposure during the entire pregnancy increases by 10 $\mu g/m^3$, the probability of ADHD increases by 0.7 percentage points. While coefficient estimates are similar for the probability of being diagnosed with ADHD by age 12, effects are not statistically significant (Columns (2) and (4)).[26]

Panel B shows the effects of in-utero exposure to $PM_{2.5}$ on the probability of having a psychiatric diagnosis. For both age groups, we do not find any evidence that an increased exposure to $PM_{2.5}$ affects the probability of receiving a psychiatric diagnosis. Our coefficient estimates are close to zero and inconclusive.

Table 4: In-utero exposure to air pollution and mental health diagnosis

	(1) Diagnosed by age 8	(2) Diagnosed by age 12	(3) Diagnosed by age 8	(4) Diagnosed by age 12
Panel A: ADHD				
1st trimester*10	0.001	-0.000		
	(0.002)	(0.002)		
2nd trimester*10	0.003*	0.002		
	(0.002)	(0.002)		
3rd trimester*10	0.004**	0.004		
	(0.002)	(0.002)		
Total*10			0.007**	0.005
			(0.003)	(0.004)
Observations	478,280	478,280	478,280	478,280
R-squared	0.50	0.52	0.50	0.52
Mean	0.01	0.03	0.01	0.03
Panel B: Any psychiatric diganosis				
1st trimester*10	0.001	-0.005		
	(0.002)	(0.003)		
2nd trimester*10	0.003	-0.000		
	(0.002)	(0.003)		
3rd trimester*10	-0.001	-0.001		
	(0.002)	(0.004)		
Total*10			0.004	-0.003
			(0.005)	(0.007)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.54	0.51	0.54
Mean	0.03	0.06	0.03	0.06

Notes: Pollution is measured at the mother's home address. Total is calculated as the average during the entire pregnancy. Column (1) and (3) indicate if the child has been diagnosed by age 8. Column (2) and (4) indicate if the child has been diagnosed by age 12. The outcomes are defined as a dummy indicate if the child has a diagnosis in the relevant time spend. In all cases the child is only included once in each regression. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking and the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Consistent with our findings on birth weight and length at birth, we find that exposure to $PM_{2.5}$ during the third trimester is also the period that is most impactful for the probability of receiving an ADHD diagnosis in childhood. Previous studies on the effects of prenatal exposure to air pollution on test scores, cognition,

[26] The reported coefficient and standard errors in Column (1) and (2) in Panel A are the same but show two different significance levels. This is due to rounding.

and socio-emotional skills consistently identify the second and third trimesters as the most harmful periods for exposure to air pollution (Bharadwaj et al., 2017; Molina, 2021; Thies, 2024), and that total prenatal exposure negatively affects cognition (Sanders, 2012; Zhang et al., 2024). Furthermore, the neurotoxicological literature based on animal studies also finds that exposure to higher levels of air pollution in the final stages of pregnancy significantly causes persistent neurodevelopmental changes (Cory-Slechta et al., 2023).

We find effects on ADHD diagnoses by age 8, but to a lesser extent by age 12. This pattern can stem from two different margins. First, it may reflect new ADHD cases that would not have emerged without increased exposure to air pollution (extensive margin response). Second, since ADHD is largely heritable (children are genetically predisposed) and early-diagnosed children tend to have more severe symptoms (Dalsgaard et al., 2014), the effects could also stem from children who would otherwise have milder symptoms but experience greater severity due to pollution exposure (inframarginal response). While we cannot disentangle these dynamics, diagnosis provides access to medical treatment, and given that treatment provides better health and economic outcomes, early treatment could be viewed as welfare-improving.[27]

Comparing our results on ADHD to studies that investigate the effect of school starting age on ADHD diagnosis, we generally find smaller effect sizes. In a U.S. context, Elder (2010) find that being born after the school starting age cutoff in kindergarten decreases the probability of an ADHD diagnosis by 2.5 percentage points, corresponding to a 39% relative reduction. Similarly, Evans et al. (2010) report an negative effect of 2.1 percentage points, corresponding to a 24% relative reduction. In contrast, Dalsgaard et al. (2012) find no visible effect of school starting age on ADHD diagnosis in a Danish context. While the absolute effects in the U.S. studies are larger than those in our analysis, the relative reductions are more comparable. Importantly, these studies do not directly impose age restrictions on diagnosis timing, and being older-for-grade involves broader differences that are not directly comparable to a 10 $\mu g/m^3$ increase in ambient air pollution. In a more closely related setting, Persson and Rossin-Slater (2018) show that prenatal stress—measured as the death of a close relative during pregnancy—increases the probability of receiving an ADHD medication prescription at ages 9–11 by 0.0062 percentage points, a 25% increase relative to baseline. This magnitude is more in line with our findings.

As highlighted in Section 5.1, we find modest effects of exposure to air pollution on infant health. This raises the question: how can these findings be reconciled with the more pronounced effects on the probability of being diagnosed with ADHD? Firstly, while the impacts on birth weight and length at birth are modest, they are still non-negligible. Birth weight is a significant determinant of later life outcomes, and even minor changes can have long-lasting effects (Black et al., 2007; Oreopoulos et al., 2008; Royer, 2009; Figlio et al., 2014). The absence of pronounced effects on infant health also aligns with the 'fetal origins' hypothesis, which suggests that the persistent nature of fetal programming — biological mechanisms experienced in-utero — may manifest such that health effects remain latent for many years (Almond and Currie, 2011; Almond et al., 2018).

Next, we investigate heterogeneity across sex, socioeconomic status, and parental ADHD on mental health outcomes. Table 5 shows the effects of exposure to $PM_{2.5}$ during each trimester on mental health across sex. Results for the probability of a diagnose by age 8 are shown in Column (1) and (3), and the results for the probability of a diagnose by age 12 are shown in Column (2) and (4). Panel A shows the results of exposure to $PM_{2.5}$ on the probability of receiving an ADHD diagnosis, and Panel B shows the results of exposure to

[27] Here, we implicitly assume that medical treatment for ADHD is beneficial. In Denmark, evidence suggests that treatment reduces injuries, hospitalizations, and criminal behavior (Dalsgaard et al., 2014, 2015). However, there is a larger discussion about the benefits of treating marginally diagnosed children (see, e.g., Persson et al. (2021)), which we discuss briefly in relation to our analysis on heterogeneous effects by socioeconomic status below.

$PM_{2.5}$ on the probability of receiving any psychiatric diagnosis.

We find that girls have a significantly higher probability of receiving an ADHD diagnosis and a psychiatric diagnosis. Panel A - Column (1) shows that when the average $PM_{2.5}$ exposure increases by 10 $\mu g/m^3$ in the third trimester, the probability of girls receiving an ADHD diagnosis by age 8 increases by 0.9 percentage points. This corresponds to an 90% increase relative to the baseline mean. We do not find any evidence that prenatal exposure to air pollution affects boys' probability of receiving an ADHD diagnosis. We find stronger effects when we investigate the effects for girls being diagonsed by age 12 in Column (2). When the average $PM_{2.5}$ exposure increases by 10 $\mu g/m^3$ in the third trimester, the probability of girls receiving an ADHD diagnosis by age 12 increases by 2.8 percentage points. This corresponds to an increase of 140% relative to the baseline mean for girls. We do not find any evidence that prenatal exposure to air pollution affects boys' probability of receiving an ADHD diagnosis.

In Panel B, Column (1), we find that when the average $PM_{2.5}$ exposure increases by 10 $\mu g/m^3$ in the third trimester, the probability of girls receiving a psychiatric diagnosis by age 8 increases by 0.1 percentage points. This corresponds to a 5% increase relative to the mean. For boys by age 8, we do not find any results suggesting that prenatal exposure to air pollution affects the probability of receiving a psychiatric diagnosis. Column (2) shows the results for children by age 12, and we observe even stronger effects for girls. When the average $PM_{2.5}$ exposure increases by 10 $\mu g/m^3$ in the third trimester, the probability of girls receiving a psychiatric diagnosis by age 12 increases by 1.6 percentage points. For the total exposure in Columns (3) and (4), we do not find any significant differences in total exposure effects between boys and girls.

Overall, our results suggest that girls are more affected by in-utero exposure to air pollution. While prior research has generally found that boys are more vulnerable to early-life environmental insults (Aguilar-Gomez et al., 2022), recent evidence from Molina (2021) and Zhang et al. (2024) shows that in-utero exposure to pollution reduces cognitive ability in ways that disproportionately hinder girls' educational attainment and labor market outcomes.[28] These gendered effects arise not from biological differences, but from postnatal behavioral responses: parents allocate resources unevenly across children, and individuals adjust educational investments based on perceived returns, both of which can amplify early disadvantages for girls. ADHD diagnoses are generally more prevalent among boys, in part because they tend to exhibit more overt hyperactive symptoms (Posner et al., 2020; Thapar et al., 2024). However, the neurotoxicological literature does not provide major evidence of sex-based differences in biological vulnerability to pollution exposure (Cory-Slechta et al., 2023). Therefore, the gendered pattern we observe in ADHD diagnoses may reflect behavioral responses by parents, teachers, or healthcare professionals to more noticeable symptoms in girls, rather than differential biological effects.

Next, we investigate heterogeneity across socioeconomic status. As with infant health, we define socioeconomic status using the maternal level of education at the time of birth. The results are presented in Table 6. Panels A and B show the results for the probability of receiving an ADHD diagnosis. Panels C and D show the results for the probability of receiving psychiatric diagnosis. For ADHD, we find some evidence suggesting that an increase in exposure to air pollution among children from high socioeconomic status increases the probability of receiving an ADHD diagnosis. When the average $PM_{2.5}$ exposure increases by 10 $\mu g/m^3$, the probability of an ADHD diagnosis increases by 0.8 percentage points if the mother has a college degree or more. This corresponds to an 80% increase relative to the baseline. We do not find any evidence of heterogeneity in the probability of receiving a psychiatric diagnosis across socioeconomic status.

The more pronounced effect on ADHD diagnosis for more affluent families raises the question of whether

[28]Similarly, Andersen et al. (2024), observe that girls' cognition is more affected by daily variations in air pollution levels.

Table 5: In-utero exposure to air pollution and mental health diagnosis - Gender

	(1) Diagnosed by age 8	(2) Diagnosed by age 12	(3) Diagnosed by age 8	(4) Diagnosed by age 12
Panel A: ADHD				
1st trimester*10	-0.002	-0.009		
	(0.003)	(0.007)		
2nd trimester*10	0.004	0.005		
	(0.003)	(0.007)		
3rd trimester*10	0.001	-0.002		
	(0.003)	(0.007)		
1st trimester*Female*10	0.003	0.019***		
	(0.003)	(0.006)		
2nd trimester*Female*10	-0.002	0.003		
	(0.004)	(0.006)		
3rd trimester*Female*10	0.008***	0.030***		
	(0.003)	(0.006)		
Total*10			0.002	-0.001
			(0.005)	(0.012)
Total*Female*10			0.005	0.042***
			(0.004)	(0.009)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.52	0.52	0.54
Mean – Boys	0.02	0.08	0.02	0.08
Mean – Girls	0.01	0.02	0.01	0.02
Panel B: Any psychiatric diagnosis				
1st trimester*10	-0.002	-0.011		
	(0.004)	(0.009)		
2nd trimester*10	0.006	0.012		
	(0.004)	(0.009)		
3rd trimester*10	-0.007	-0.010		
	(0.004)	(0.009)		
1st trimester*Female*10	-0.001	0.009		
	(0.004)	(0.008)		
2nd trimester*Female*10	-0.006	-0.007		
	(0.004)	(0.008)		
3rd trimester*Female*10	0.008**	0.026***		
	(0.004)	(0.008)		
Total*10			0.000	0.000
			(0.007)	(0.016)
Total*Female*10			-0.003	0.019
			(0.006)	(0.012)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.54	0.51	0.54
Mean – Boys	0.05	0.15	0.05	0.15
Mean – Girls	0.02	0.07	0.02	0.07

Notes: Pollution is measured at the mother's home address. Total is calculated as the average during the entire pregnancy. Column (1) and (3) indicate if the child has been diagnosed by age 8. Column (2) and (4) if the child has been diagnosed by age 12. The outcomes are defined as a dummy indicate if the child has a diagnosis in the relevant time spend. In all cases the child is only included once in each regression. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, and the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

higher socioeconomic status families are more affected by pollution or simply more proactive in seeking medical attention for their children. Some evidence suggests that the latter explanation may be at play as parents in high socioeconomic status families are more prone to act on potential symptoms and obtain an ADHD diagnosis, which creates a social gradient (Madsen et al., 2017). Our findings align with this pattern, which is of particular importance when a diagnosis provides access to treatment, other therapeutic resources, and, in Denmark, easier access to special education (Tegtmejer et al., 2022; Tegtmejer and Säljö, 2023). The benefits of diagnosing the marginal child remain debated among academics and policymakers, with mixed evidence. Marginal diagnoses driven by physician leniency show positive shorter-run effects on education and reduced risky behavior (Dalsgaard et al., 2014; Chorniy and Kitashima, 2016), while those stemming from family spillovers appear to have no impact on longer-run labor market outcomes (Persson et al., 2021). While our analysis does not directly assess the benefits of a marginal diagnosis, a positive selection into diagnosis by more affluent families — combined with its shorter-run benefits — may exacerbate health and educational inequalities further.

As highlighted above, ADHD is largely heritable, as children of parents with ADHD are genetically predisposed (Posner et al., 2020). In our sample, around 1 percent have at least one parent diagnosed with ADHD. Table 7 presents the effects of in-utero exposure to air pollution on the probability of being diagnosed with ADHD, split by whether a parent has an ADHD diagnosis. The general pattern suggests that in-utero exposure to air pollution increases the probability of an ADHD diagnosis for children both with and without a parental ADHD diagnosis. Although the effects for children with a parental ADHD diagnosis are not statistically significant, they are around 20 times larger than the effects for children without a diagnosed parent.[29] It is important to note that a parental ADHD diagnosis is not directly equivalent to a purely genetic predisposition, as parental awareness of their own ADHD diagnosis may make them more aware of symptoms in their children, leading to a higher probability of seeking a diagnosis. Therefore, the results should be interpreted with caution. Nevertheless, this exercise illustrates that while in-utero exposure to air pollution negatively affects most children, there may be subgroups, such as those with a potential genetic predisposition, for whom the effects are particularly pronounced.

5.3 Robustness

In this section, we examine the robustness of our baseline results for both infant and mental health. Despite that the validity test in section 4 shows that controlling for family invariant characteristics effectively eliminates potential sorting and selection issues, we might still worry that there is avoidance behavior. As it is infeasible to directly measure avoidance behavior, we restrict the sample to families who do not move between the birth of their children. If the decision to move is correlated with air pollution, and families tend to move to less polluted places, then exposure to air pollution is not exogenous. Table A4 and A5 show that this restriction does not change the results for both infant and mental health. Hence, we argue that avoidance behavior is not an issue in our study.

As an additional approach to check whether geography-specific pollution impacts the results, we investigate whether our results change with the inclusion of municipality fixed effects. The municipality fixed effect captures potential systematic geographical differences in exposure to air pollution for parents that moves between births of their children. The results are shown in Table A8 and A9. We find similar effects as in

[29] As only 1 percent of children in the sample have a parental ADHD diagnosis, this estimate is likely underpowered. Around 2.5–3% of the adult population in Denmark is treated for ADHD. We are therefore not able to identify all children with parents who have ADHD.

Table 6: In-utero exposure to air pollution and mental health by maternal education

	Unskilled		Vocational education		College	
	(1) Diagnosed by age 8	(2) Diagnosed by age 12	(3) Diagnosed by age 8	(4) Diagnosed by age 12	(5) Diagnosed by age 8	(6) Diagnosed by age 12
Panel A: ADHD - Trimester						
1st trimester*10	0.007*	0.009*	0.000	0.001	-0.003	-0.005
	(0.004)	(0.006)	(0.003)	(0.004)	(0.002)	(0.003)
2nd trimester*10	0.004	0.005	0.002	0.002	0.003*	0.001
	(0.004)	(0.005)	(0.003)	(0.004)	(0.002)	(0.003)
3rd trimester*10	0.002	-0.003	0.002	0.004	0.007***	0.004
	(0.004)	(0.006)	(0.003)	(0.005)	(0.002)	(0.003)
Observations	120,999	120,999	159,843	159,843	152,244	152,244
R-squared	0.51	0.52	0.51	0.54	0.51	0.53
Mean	.02	.04	.01	.03	.01	.02
Panel B: ADHD - Total						
Total*10	0.010	0.012	0.004	0.004	0.008**	0.000
	(0.007)	(0.010)	(0.006)	(0.008)	(0.004)	(0.006)
Observations	120,999	120,999	159,843	159,843	152,244	152,244
R-squared	0.51	0.52	0.51	0.54	0.51	0.53
Mean	.02	.04	.01	.03	.01	.02
Panel C: Any psychiatric - Trimester						
1st trimester*10	0.010*	0.007	-0.004	-0.016**	-0.002	-0.002
	(0.005)	(0.008)	(0.005)	(0.007)	(0.004)	(0.006)
2nd trimester*10	0.005	0.009	0.001	-0.001	0.006*	0.002
	(0.005)	(0.008)	(0.005)	(0.007)	(0.003)	(0.005)
3rd trimester*10	-0.006	-0.011	0.002	0.003	-0.003	0.001
	(0.006)	(0.008)	(0.005)	(0.007)	(0.004)	(0.006)
Observations	121,121	121,121	159,843	159,843	152,244	152,244
R-squared	0.51	0.54	0.52	0.55	0.51	0.53
Mean	.03	.08	.03	.06	.02	.05
Panel D: Any psychiatric - Total						
Total*10	0.014	0.014	-0.001	-0.013	-0.000	-0.000
	(0.010)	(0.014)	(0.008)	(0.012)	(0.007)	(0.010)
Observations	121,121	120,999	159,843	159,843	152,244	152,244
R-squared	0.51	0.54	0.52	0.55	0.51	0.53
Mean	.03	.08	.03	.06	.02	.05

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Vocational education defines all mothers who have vocational education and training or short further education. Column (1), (3) and (5) indicate if the child has been diagnosed by age 8. Column (2), (4) and (6) indicate if the child has been diagnosed by age 12. The outcomes are defined as a dummy indicate if the child has a diagnosis in the relevant time spend. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 7: In-utero exposure to air pollution and mental health by parental ADHD diagnosis

	Parental ADHD		No parental ADHD	
	(1) Diagnosed by age 8	(2) Diagnosed by age 12	(3) Diagnosed by age 8	(4) Diagnosed by age 12
Panel A: Trimester				
1st trimester*10	0.145**	0.418***	-0.001	-0.004
	(0.070)	(0.150)	(0.002)	(0.005)
2nd trimester*10	-0.062	-0.201	0.003	0.008
	(0.071)	(0.153)	(0.002)	(0.005)
3rd trimester*10	0.085	0.253*	0.004	0.011*
	(0.070)	(0.145)	(0.002)	(0.006)
Observations	4,431	4,431	471,631	363,550
R-squared	0.56	0.56	0.50	0.52
Panel B: Total				
Total*10	0.010	0.170	0.005	0.016
	(0.128)	(0.264)	(0.004)	(0.010)
Observations	4,431	4,431	471,631	471,631
R-squared	0.55	0.56	0.50	0.52
Mean	.13	.37	.01	.05

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Vocational education defines all mothers who have vocational education and training or short further education. Parental ADHD is determined as a dummy that indicates if either the father or the mothers have an ADHD diagnosis. Column (1) and (3) indicate if the child has been diagnosed by age 8. Column (2) and (4) indicate if the child has been diagnosed by age 12. The outcomes are defined as a dummy indicate if the child has a diagnosis in the relevant time spend. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

the main specification, suggesting that geographical specific pollution is a limited issue.

As highlighted in the section above, ADHD is largely heritable with a strong genetic component. While ADHD diagnosis follows standardized clinical guidelines, it ultimately relies on the professional judgment at the margin.[30] These facts warrant another concern: In families where an older child is first diagnosed with ADHD, this diagnosis may potentially spill over to affect the probability of a diagnosis for younger children (Persson et al., 2021). To mitigate such concerns, we investigate whether our results change when we control for birth order in the baseline specification.[31] The results are shown in Table A6 and A7. For infant health, we find that the effect size for birth weight and length at birth is slightly reduced relative to the main specification, but the overall pattern remains. More importantly, for mental health the inclusion of birth order does not change the results, and the estimated effect sizes are of the same magnitude.

As weather can affect pollution levels in various ways, we show that our results are robust to the inclusion of precipitation and cloud cover. We include precipitation, as heavy precipitation forces the heaviest pollutants to the ground and can help dissolve certain pollutants. Cloud cover, meanwhile, can be used as a proxy for exposure to sunlight and, consequently, vitamin D, which is essential for fetal development. Table A10 and A11 show that the inclusion of total precipitation, average percentage cloud cover each trimester, along with including a more flexible specification of temperatures[32] does not change our main results. Hence, we argue that different weather conditions during pregnancy do not affect our results.

[30] As highlighted in Section 2 diagnosis for a neurodevelopmental disorder in Denmark, such as ADHD, can only be made by a child psychiatrist.

[31] Information on birth order is constructed using all siblings and not only siblings within the sample. If a family is comprised of child A born 1996, child B born 1999, and child C born 2022, then only child B and C are included in the sample, categorized as birth order 2 and 3 respectively.

[32] We include the share of days in each trimester, where temperatures falls into bins of 5°C starting from below 0°C and ending with above 20°C

Lastly, to further strengthen the validity of our results, we perform a placebo test in which we estimate the effect of exposure to air pollution during the first month after birth on infant health (Table A12). As expected, we find small and insignificant coefficient estimates highlighting that postnatal exposure to air pollution does not affect infant health.[33]

In the baseline specification (Equation 1), we implicitly assume additive separability, which implies that the effects of exposure to average pollution levels in each trimester on the outcome of interest (e.g., infant health and mental health) simply add up to the total effect of pollution exposure throughout the entire pregnancy (i.e., the effect in Equation 2). In Equation 1, each coefficient estimate β_1, β_2, and β_3 represents the marginal effect of increasing exposure to $PM_{2.5}$ by 10 $\mu g/m^3$ and thereby reflects the effect of increasing average exposure to pollution in the specific trimester combined with the effect of increasing the overall average level of pollution. However, if the overall level of average pollution that a pregnant woman is exposed to has a separate effect, then the marginal effect for each trimester would instead represent a reallocation of pollution levels between trimesters given a constant level of overall average exposure. To investigate whether that is the case, we consider the following specification:

$$Y_{if} = \alpha + \beta_{trim2} E_{if2} + \beta_{trim3} E_{if3} + \beta_{Total} E_{ifTotal} + \theta_{BirthYear} + \tau_{BirthMonth} + \mu_f + \delta X_{if} + \sum \mu W_{if} + u_{if} \quad (6)$$

Equation 6 is a modified specification of Equation 1 that includes separate effects for exposure to air pollution in the second trimester (β_{trim2}), the third trimester (β_{trim3}), and the overall level (β_{Total}) (as $E_{ifTotal} = E_{if1} + E_{if2} + E_{if3}$, with exposure to the first trimester E_{if1} omitted due to collinearity).[34] β_{trim3} in this specification represents the marginal effect of increasing exposure to $PM_{2.5}$ in the third trimester ($Eif3$) by 10 $\mu g/m^3$ while simultaneously decreasing exposure to $PM_{2.5}$ in the first trimester ($Eif1$), as $E_{ifTotal} = E_{if1} + E_{if2} + E_{if3}$. In other words, the coefficient β_{trim3} reflects the marginal effect of 'redistributing' pollution from the first trimester to the third trimester, with the total level $E_{ifTotal}$ held constant. Similarly, Equation 7 and 8 each consider the case where air pollution in the second (E_{if2}) and third (E_{if3}) trimester is omitted:

$$Y_{if} = \alpha + \beta_{trim1} E_{if1} + \beta_{trim3} E_{if3} + \beta_{Total} E_{ifTotal} + \theta_{BirthYear} + \tau_{BirthMonth} + \mu_f + \delta X_{if} + \sum \mu W_{if} + u_{if} \quad (7)$$

$$Y_{if} = \alpha + \beta_{trim1} E_{if1} + \beta_{trim2} E_{if2} + \beta_{Total} E_{ifTotal} + \theta_{BirthYear} + \tau_{BirthMonth} + \mu_f + \delta X_{if} + \sum \mu W_{if} + u_{if} \quad (8)$$

Table A13 shows the results for these altered specifications for birth weight (Panel A) and the probability of being diagnosed with ADHD at different ages (Panel B and C). Column (1) corresponds to Equation 6, Column (2) to Equation 7, and Column (3) to Equation 8. For birth weight, the first specification highlights that if 10 $\mu g/m^3$ of $PM_{2.5}$ is reallocated from the mean exposure in the first trimester to the third trimester,

[33] In the placebo test, we abstract from considering exposure to air pollution prior to conception, as air pollution may affect a woman's body and physical health before pregnancy, potentially causing an increased inflammatory state (cf. Section 2.1). Similarly, we abstract from considering the effects of postnatal exposure to air pollution on later-life mental health, as there may be independent effects of air pollution exposure during infancy on long-term mental health outcomes.

[34] Throughout the whole analysis we use the average pollution in each trimester. This also implies that total exposure to average air pollution throughout the pregnancy, $E_{ifTotal} = E_{if1} + E_{if2} + E_{if3}$, is a sum of trimester averages, each weighted equally.

then birth weight decreases by 12.34 grams (holding the mean exposure in the second trimester and the mean overall exposure constant). The magnitude of the coefficient estimates is rather close to baseline estimates (Column 1 in Table 3), which suggests that there is limited effect of the overall mean level of air pollution and that an increase in pollution levels in the third trimester matter (irrespective of whether it stems from a reallocation from another trimester or additional pollution). For the probability of an ADHD diagnosis by age 8, we see similar patterns. The magnitude for the coefficient estimates is close to the baseline estimates (Column 1 in Table 4), which again suggests that it is the increase during the third trimester that driving the effect and to a less extend the overall pollution levels.

5.4 Further analysis on maternal health

Next, we investigate how exposure to air pollution affects maternal health throughout the pregnancy. As highlighted in Section 2.1, exposure to air pollution may lead to an increased inflammatory response in mothers manifesting as a respiratory infection, cardiovascular issues, or mental health challenges (Arias-Péres et al., 2020; Cory-Slechta et al., 2023). Therefore, we investigate how exposure to air pollution during pregnancy affects the probability of being diagnosed with influenza like-illness, pregnancy complications (including pregnancy-related hypertension), birth complications, mental health, depression, and anxiety during pregnancy.[35]

Table 8 shows the effects of exposure to air pollution on maternal health during pregnancy. Column (1)-(5) shows the results for conditions related to physical health, while Column (6)-(7) shows the results related to mental health. Column (1) shows the results for influenza like-illness, which serves as a proxy for respiratory illnesses. Air pollution has previously been associated with increased risk of respiratory influenza (Horne et al., 2018; Darrow et al., 2014). However, we find a coefficient estimate that is small in magnitude and not statistically significant, suggesting that exposure to air pollution during the pregnancy does not seem to affect the probability of being diagnosed with influenza like-illness. Next, Column (2) shows the effect on the probability of being diagnosed with any pregnancy complications, which primarily includes diagnosis for hypertension during pregnancy, gestational diabetes, and liver disorders related to pregnancy. We find that when $PM_{2.5}$ increases by 10 $\mu g/m^3$ during the pregnancy, the probability of being diagnosed with pregnancy complications increases by approximately 0.8 percentage points. This increase corresponds to a 43% increase relative to the baseline mean, and the effect is statistically significant. To investigate this result in more detail, Column (3) shows that the results on pregnancy complications are mainly driven by diagnosis related to hypertensive disorders during pregnancy (HDP).[36] When $PM_{2.5}$ increases by 10 $\mu g/m^3$, the probability of HDP increases by approximately 0.8 percentage points. This increase corresponds to a 54% increase relative to the baseline mean, and the effect is also statistically significant.

In Column (4) and (5) we do not find any evidence to suggest that the probability of complications or during birth a C-section increases due to exposure during the pregnancy.

Lastly, Column (6) shows that the probability of being diagnosed with a mental health disorder increases slightly during pregnancy due to exposure to air pollution. When $PM_{2.5}$ increases by 10 $\mu g/m^3$ during the pregnancy, the probability of mental health disorders increases by 0.4 percentage points. However, the

[35] In this section we focus on the average exposure during the entire pregnancy, and not the trimester-wise exposure, as we are interested in the whole pregnancy period, and not the effect on specific trimesters. We follow Schwandt (2018) and define influenza like-illness as ICD10: J00, J01, J04, J10-J11, J12-18, R06-R07, B34. We follow Hamann et al. (2019) and define pregnancy complications as ICD10: O10–O15, O244, O268G, O268H, O264. We follow Maibom et al. (2021) and define birth complications as O60-O69, O71-O75. Hypertensive disorders are defined as ICD10: O10-O15. Depression during pregnancy is defined as F32-33, and anxiety during pregnancy as F4. All diagnosis codes include both in- and outpatient hospitalizations.

[36] Defined as ICD10: O10–O15

coefficient estimate is only significant at a 10% level. Column (7) and (8) show that the slight increase in overall mental health disorders is not driven by the probability of being diagnosed with either anxiety or pregnancy-related depression.

Table 8: In-utero exposure to air pollution and maternal health during pregnancy

	Physiological diagnosis					Mental health diagnosis		
	(1) Influenza like-illness	(2) Pregnancy complications	(3) Hypertensive disorder	(4) Birth complications	(5) C-section	(6) Mental health	(7) Depression	(8) Anxiety
Total*10	0.0005	0.0077***	0.0082***	-0.0102	-0.0021	00040*	-0.0005	-0.0010
	(0.0004)	(0.0026)	(0.0024)	(0.0092)	(0.0056)	(0.0023)	(0.0007)	(0.0008)
Observations	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504
R-squared	0.43	0.51	0.51	0.56	0.75	0.54	0.48	0.47
Mean	0.0004	0.018	0.015	0.41	0.19	0.013	0.001	0.001

Notes: Pollution is measured at the home address for the mother. Across all regressions we control for the mothers age when she gives birth, the sex of the child, maternal smoking, and the average temperature during pregnancy. We include year of birth, month of birth, and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

The results on hypertension align with previous epidemiological evidence on the association between exposure to air pollution and HDP (Pedersen et al., 2014). As highlighted in Section 2.1, a common pathological feature of air pollution on the human body is an increased inflammatory response, and the result on maternal HDP helps suggest inflammation as a key biological mechanism in understanding how in-utero exposure to air pollution affects child development.

5.5 Implications for high pollution areas

As described previously, Denmark is characterized by low pollution levels, and the is on average below the guidelines from WHO and EU. However, most other industrialist and developing countries do not meet these guidelines. With some assumptions, we can use our results to create a back-of-the-envelope estimate of the extent to which a reduction in pollution to WHO recommended levels (15 $\mu g/m^3$) in other countries would reduce ADHD incidence. To do so, we use the following three cities as examples: Los Angeles (US), Milan (Italy), and New Delhi (India). We assume that the effect of in-utero exposure to air pollution is linear, and that other factors such as the underlying distribution of ADHD and socioeconomic variables are constant.[37] The various calculations are shown in Table 9.

The average yearly exposure to $PM_{2.5}$ in Los Angeles was 21.1 $\mu g/m^3$ in 2022, and hence it would require a reduction of 6.2 $\mu g/m^3$ to reach the recommendations from WHO. When we estimate an effect of 0.4 percentage points decrease in the probability of an ADHD diagnosis by age 8, when $PM_{2.5}$ increases by 1 $\mu g/m^3$, this would correspond to a decrease of 248 cases of ADHD per 100,000 births in Los Angeles, if $PM_{2.5}$ levels were reduced to 15 $\mu g/m^3$.

Milan is considered one of the most polluted cities within Europe due to its geographical location. The average yearly exposure to $PM_{2.5}$ in Milan was 23 $\mu g/m^3$ in 2022, and hence it requires a reduction of 8 $\mu g/m^3$ to reach the guidelines from WHO. Such a reduction would result in a reduction of 320 cases of ADHD per 100,000 births.

Even though the pollution levels in Los Angeles and Milan are higher compared to Denmark, they are still relatively low in a global setting, especially compared to cities in China, Bangladesh, and India. New

[37]In reality, the effect could differ if there are nonlinearities or substantial contextual differences between Denmark and each of these cities. Nevertheless, this back-of-the-envelope calculation provides a rough magnitude of how reducing pollution to the WHO guideline may decrease the probability of ADHD.

Delhi is considered one of the most polluted cities in the world. In 2022, the yearly average of $PM_{2.5}$ was 98 $\mu g/m^3$. Hence, it would require a reduction of 83 $\mu g/m^3$ to reach the guideline from WHO. Such a reduction could potentially lead to 3,220 fewer cases of ADHD per 100,000 births.

Table 9: Extrapolations

City	$PM_{2.5}$ levels	Reduction to reach WHO	Calculation	Cases per 100,000 births
Los Angeles	21.2	6.2	6,2*0.004*100	248
Milan	23	8	8*0.004*100	320
New Delhi	98	83	83*0.004*100	3320

Notes: Data from Los Angeles is from The California Air Resources Board Pollution. Data from Milan is from Statista.com. Data from New Delhi is from Ministry of Environment, India. We use the estimated effect size of a 1 $\mu g/m^3$ increase in $PM_{2.5}$ on the probability of an ADHD diagnosis by age 8 from Table 4.

6 Conclusion

In this paper, we investigate the effects of in-utero exposure to air pollution, specifically $PM_{2.5}$, on both infant health and child and adolescent mental health disorders. Our study exploits variation in $PM_{2.5}$ exposure levels across pregnancies within families.

A child whose mother was pregnant during the winter months is, on average, exposed to higher levels of air pollution than if the mother had been pregnant during the summer months. To address potential selection into the timing of pregnancy or sorting into areas with differing pollution levels, we employ a sibling fixed effects strategy. Our analysis utilizes rich, population-wide administrative data linked with granular pollution and weather data.

We find that in a setting characterized by generally low pollution levels, such as Denmark, exposure to $PM_{2.5}$ during pregnancy—particularly in the third trimester—reduces birth weight and length but does not appear to significantly impact other severe infant health outcomes, such as the probability of being low birth weight, reduced APGAR scores, or the probability of being born preterm.

Importantly, we find that in-utero exposure to air pollution has detrimental effects on mental health later in life. Specifically, exposure to $PM_{2.5}$ increases the probability of being diagnosed with attention deficit hyperactivity disorder (ADHD). This effect is most pronounced for exposure during the third trimester and is particularly evident among children by age 8. Additionally, the effects are stronger for girls than for boys and are driven by more affluent families (proxied by maternal education).

Lastly, we show that an increase in exposure to $PM_{2.5}$ during pregnancy increases the probability of pregnancy complication, mainly driven by the probability of hypertensive disorders, which suggests an elevated inflammatory response in the mother.

From a policy perspective, our findings are highly relevant as worsening mental health—particularly among adolescents—is a growing concern worldwide. Despite this, causal evidence on the impact of environmental factors on mental health disorders remains limited. Understanding how early-life exposure to pollution influences mental health outcomes is critical for designing effective interventions and policies to safeguard future generations.

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ONLINE APPENDIX

Toxic Air and Troubled Minds: The Effect of In-utero Exposure to Air Pollution on Child and Adolescent Mental Health

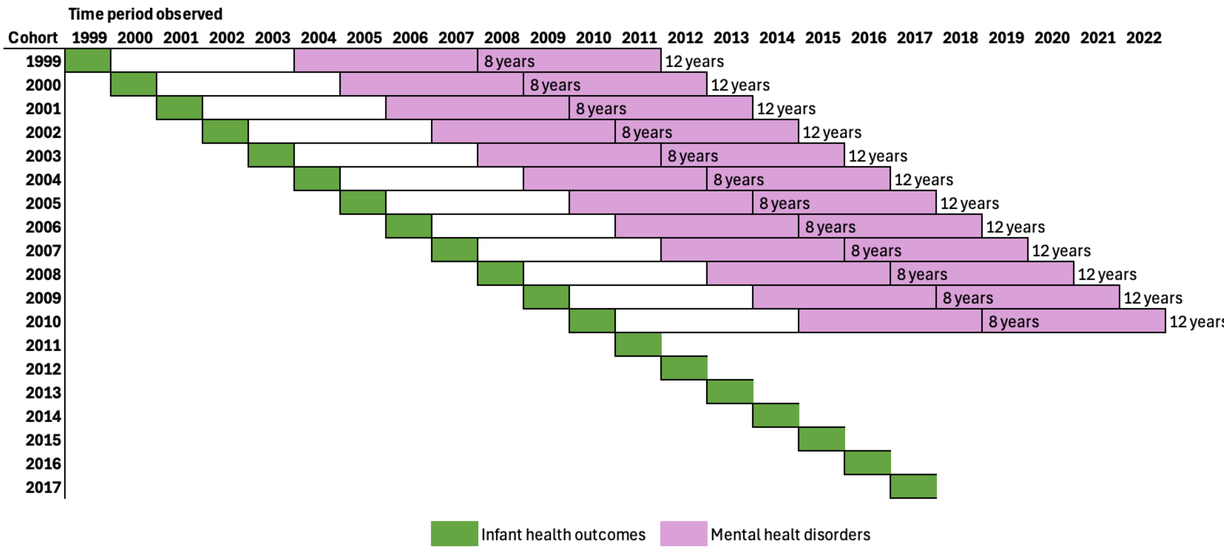
Christina Munkholm Andersen and Marius Opstrup Morthorst

September 2025

A Appendix Figures and Tables

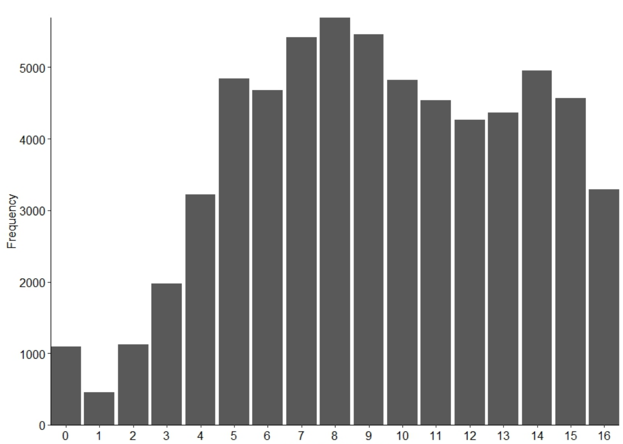
A Figures

Figure A1: Sample selection and timing of outcomes

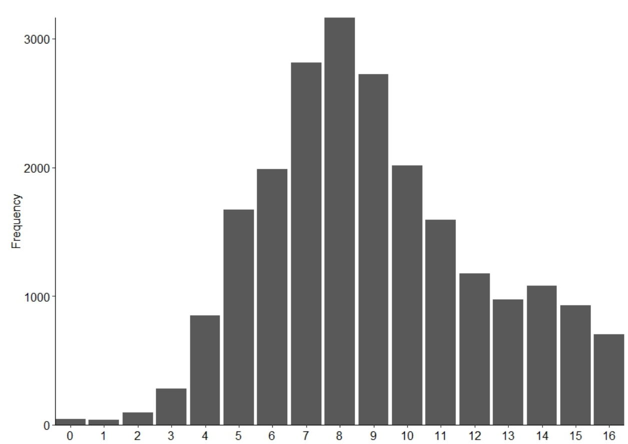


Notes: This figure illustrates the sample selection of cohorts and timing of when outcomes are measured.

Figure A2: Age at first mental health diagnosis



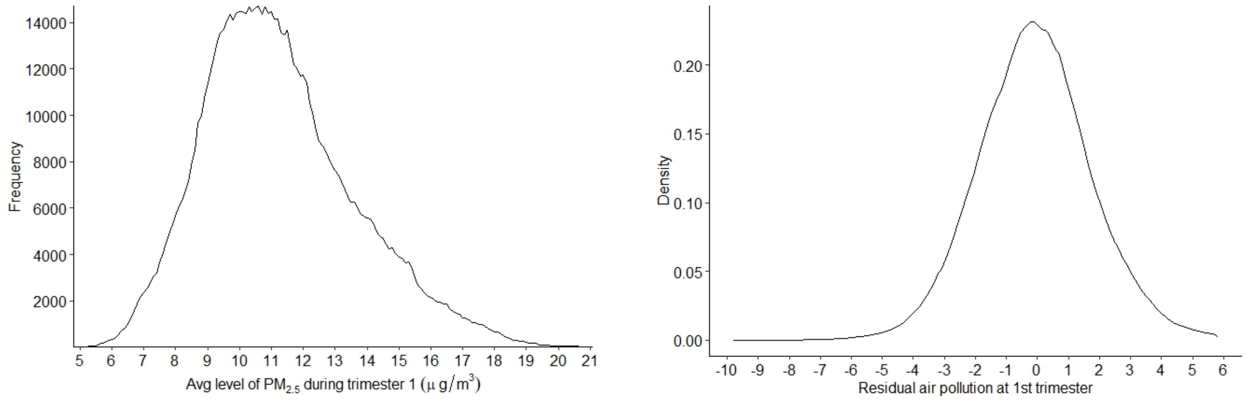
(a) Any psychiatric diagnosis



(b) ADHD

Notes: Included are children in the main sample. The graph indicate when the child for the first time receives a diagnosis.

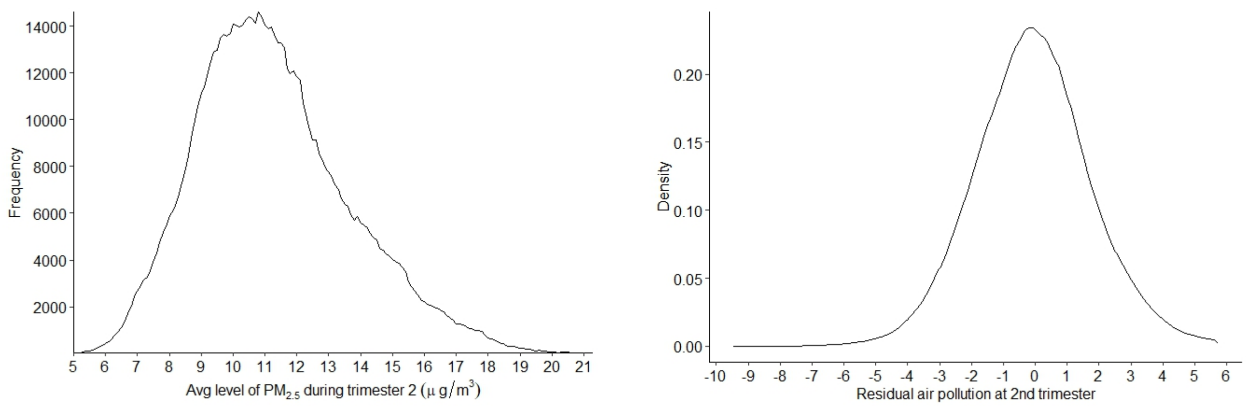
Figure A3: Variation in pollution levels in the 1st trimester



(a) Raw levels of pollution in the 1st trimester (b) Residual pollution in the 1st trimester

Notes: Panel A shows the individual pollution levels during the 1st trimester. Panel B shows the residuals from a regression exposure to $PM_{2.5}$ in the 1st on average temperatures and year-, month- and family fixed effects.

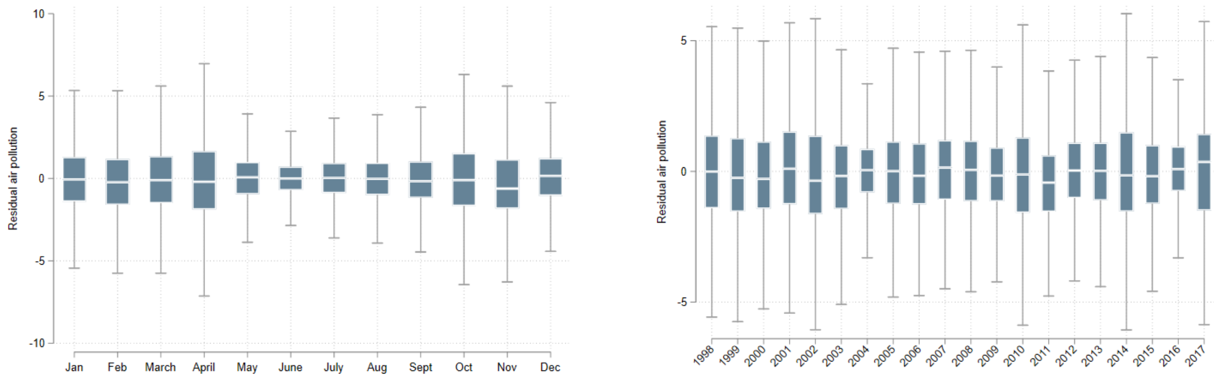
Figure A4: Variation in pollution levels in the 2nd trimester



(a) Raw levels of pollution in the 2nd trimester (b) Residual pollution in the 2nd trimester

Notes: Panel A shows the individual pollution levels during the 2nd trimester. Panel B shows the residuals from a regression exposure to $PM_{2.5}$ in the 2nd on average temperatures and year-, month- and family fixed effects.

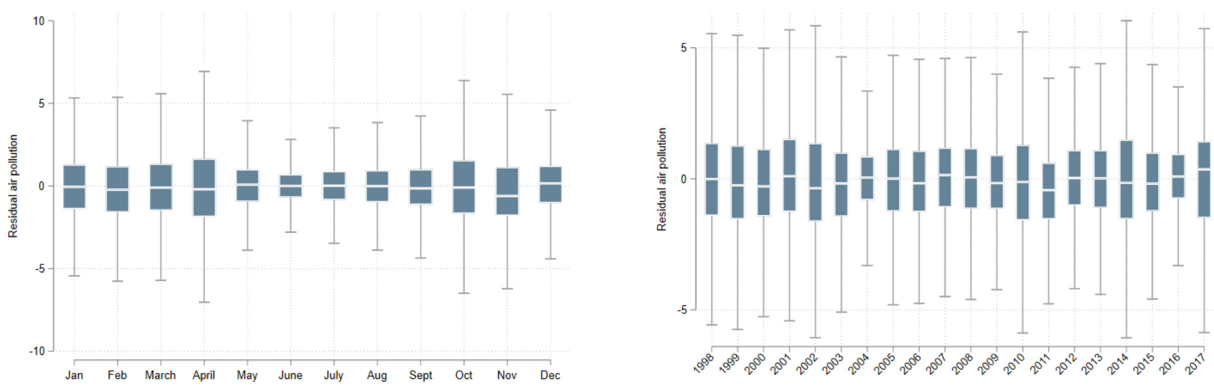
Figure A5: Variation in residual pollution in the 1st trimester



(a) Birth month (b) Birth year

Notes: Each plot shows the residuals from a regression of exposure to $PM_{2.5}$ in the 1st trimester on average temperatures and year-, month-, and family fixed effects. Panel A shows the results conditional on birth month. Panel B shows the results conditional on birth year. For each month or year, the box-whisker plots display the median, the 25th and 75th percentiles, and the lower and upper adjacent values (25th and 75th percentiles $\pm$ 1.5 times the interquartile range). For data privacy reasons, all the relevant statistics are computed as the mean of the 10 closest observations to the real value.

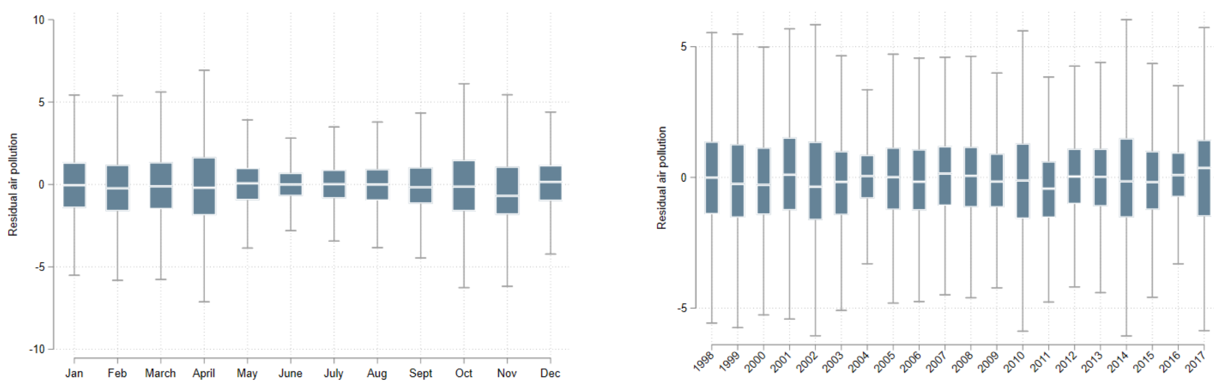
Figure A6: Variation in residual pollution in the 2nd trimester



(a) Birth month (b) Birth year

Notes: Each plot shows the residuals from a regression of exposure to $PM_{2.5}$ in the 2nd trimester on average temperatures and year-, month-, and family fixed effects. Panel A shows the results conditional on birth month. Panel B shows the results conditional on birth year. For each month or year, the box-whisker plots display the median, the 25th and 75th percentiles, and the lower and upper adjacent values (25th and 75th percentiles $\pm$ 1.5 times the interquartile range). For data privacy reasons, all the relevant statistics are computed as the mean of the 10 closest observations to the real value.

Figure A7: Variation in residual pollution in the 3rd trimester



(a) Birth month (b) Birth year

Notes: Each plot shows the residuals from a regression of exposure to $PM_{2.5}$ in the 3rd trimester on average temperatures and year-, month-, and family fixed effects. Panel A shows the results conditional on birth month. Panel B shows the results conditional on birth year. For each month or year, the box-whisker plots display the median, the 25th and 75th percentiles, and the lower and upper adjacent values (25th and 75th percentiles $\pm$ 1.5 times the interquartile range). For data privacy reasons, all the relevant statistics are computed as the mean of the 10 closest observations to the real value.

B Tables

Table A1: Sample selection

Description	Number of individuals	Percent total
Panel A: Sampling births		
All children born in Denmark in 1999-2017 + information on gestational length	1,174,358	1
Able to match to maternal address during the entire pregnancy	1,152,303	0.98
Able to match to pollution data	1,150,158	0.98
Has at least one sibling in the sample	812,504	0.69
Panel B: Mental health		
Observer mental health at age 12	478,280	0.41

Notes: To be included in the mental health sample it requires that we are able to observe mental health of the child and at least one sibling at the specific age.

Table A2: In utero exposure to air pollution and birth outcomes - Sex

	(1) Birth weight	(2) P(LBW)	(3) Length	(4) Apgar score	(5) P(Preterm)
Panel A: Trimester					
1st trimester * 10	-6.79	0.000	-0.047	-0.003	-0.002
	(5.75)	(0.002)	(0.063)	(0.008)	(0.002)
Trimester 2 * 10	-3.29	-0.001	-0.085	0.000	-0.002
	(5.59)	(0.002)	(0.060)	(0.008)	(0.002)
Trimester 3 * 10	-15.68**	0.002	-0.106	-0.005	0.000
	(6.09)	(0.003)	(0.066)	(0.009)	(0.002)
Trimester 1 * Female * 10	9.29	-0.003	-0.057	0.007	-0.000
	(5.77)	(0.002)	(0.063)	(0.008)	(0.002)
Trimester 2 * Female * 10	-11.98**	0.005*	-0.042	-0.001	0.002
	(5.74)	(0.002)	(0.061)	(0.008)	(0.002)
Trimester 3 * Female * 10	4.79	-0.001	-0.010	0.008	-0.002
	(5.66)	(0.002)	(0.062)	(0.008)	(0.002)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.52
Panel B: Total					
Total * 10	-19.45*	-0.000	-0.206*	-0.013	-0.003
	(10.12)	(0.004)	(0.109)	(0.014)	(0.004)
Total * Female * 10	-2.55	0.002	-0.120	0.010	0.002
	(8.50)	(0.003)	(0.093)	(0.012)	(0.003)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.52
Mean – Boys	3586.4	.03	51.92	9.86	.03
Mean – Girls	3461.5	.04	51.22	9.89	.03

Notes: Pollution is measured at the home address for the mother. Number of observations varies between Columns, since not all outcomes are measured for especially the earliest cohorts. In Panel B the total is calculated as the average during the entire pregnancy. In all regressions we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors clustered by mother identifier and municipality level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A3: In-utero exposure to air pollution and birth outcomes by maternal education

	(1) Birth weight	(2) P(LBW)	(3) Length	(4) Apgar score	(5) P(Preterm)	(6) Birth weight	(7) P(LBW)	(8) Length	(9) Apgar score	(10) P(Preterm)
Panel A: Unskill										
1st trimester * 10	3.53	-0.004	-0.01	0.011	-0.005					
	(10.33)	(0.005)	(0.11)	(0.015)	(0.004)					
2nd trimester * 10	-10.08	0.000	-0.06	-0.012	-0.004					
	(9.86)	(0.004)	(0.11)	(0.014)	(0.004)					
3rd trimester * 10	-24.58**	0.004	-0.10	0.006	-0.003					
	(11.15)	(0.005)	(0.12)	(0.016)	(0.005)					
Total * 10						-34.20*	0.008	-0.20	-0.009	-0.004
						(18.80)	(0.008)	(0.20)	(0.027)	(0.007)
Observations	190,348	190,348	189,707	188,881	190,348	190,348	190,348	189,707	188,881	190,348
R-squared	0.68	0.52	0.50	0.44	0.51	0.68	0.52	0.50	0.44	0.51
Mean	3471.6	.04	51.33	9.87	.04	3471.6	.04	51.33	9.87	.04
Panel B: VET										
1st trimester * 10	-10.53	0.005	-0.06	-0.004	-0.002					
	(9.20)	(0.004)	(0.10)	(0.013)	(0.004)					
2nd trimester * 10	-12.53	0.000	-0.11	-0.005	0.001					
	(8.87)	(0.004)	(0.09)	(0.013)	(0.003)					
3rd trimester * 10	-4.13	0.004	-0.06	-0.005	0.004					
	(9.83)	(0.004)	(0.10)	(0.014)	(0.004)					
Total * 10						-21.52	0.007	-0.19	-0.010	0.002
						(17.29)	(0.007)	(0.18)	(0.025)	(0.007)
Observations	250,881	250,881	250,091	249,004	250,881	250,881	250,881	250,091	249,004	250,881
R-squared	0.69	0.54	0.53	0.46	0.53	0.69	0.54	0.53	0.46	0.53
Mean	3529.87	.04	51.6	9.87	.03	3529.87	.04	51.6	9.87	.03
Panel C: College										
1st trimester * 10	-4.00	-0.004	-0.17*	0.001	0.002					
	(8.35)	(0.003)	(0.09)	(0.012)	(0.003)					
2nd trimester * 10	-5.12	0.002	-0.14	0.014	-0.001					
	(7.93)	(0.003)	(0.090)	(0.011)	(0.003)					
3rd trimester * 10	-8.70	-0.002	-0.15	-0.004	-0.002					
	(9.16)	(0.004)	(0.10)	(0.013)	(0.003)					
Total * 10						-13.62	-0.006	-0.41**	0.004	-0.001
						(15.46)	(0.006)	(0.17)	(0.022)	(0.006)
Observations	287,192	287,192	286,378	285,340	287,192	287,192	287,192	286,378	285,340	287,192
R-squared	0.68	0.53	0.52	0.47	0.52	0.68	0.53	0.52	0.47	0.52
Mean	3565.28	.03	51.76	9.88	.03	3565.28	.03	51.76	9.88	.03

Notes: Pollution is measured as outcome pollution at the mothers home address. P(LBW) is defined as the probability of a birth weight below 2500g. Mean refers to the sample mean of the outcome. Vocational defines all mothers who have vocational education and training or short further education. Number of observations varies between Columns, since not all outcomes are measured for especially the earliest cohorts. Total is calculated as the average during the entire pregnancy. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A4: In-utero exposure to air pollution and infant health - Non-mover

	(1) Birth weight	(2) P(LBW)	(3) Length	(4) Apgar score	(5) P(Preterm)
Panel A: Trimester					
1st trimester * 10	-8.65	-0.001	-0.063	0.005	-0.002
	(5.89)	(0.002)	(0.065)	(0.008)	(0.002)
2nd trimester * 10	-10.48*	-0.001	-0.096	0.006	0.000
	(5.62)	(0.002)	(0.061)	(0.008)	(0.002)
3rd trimester * 10	-14.10**	0.001	-0.117*	0.006	0.001
	(6.36)	(0.003)	(0.068)	(0.009)	(0.003)
Observations	606,110	606,110	604,261	601,679	606,344
R-squared	0.69	0.53	0.52	0.46	0.53
Panel B: Total					
Total * 10	-26.34**	-0.001	-0.232*	0.012	0.000
	(11.09)	(0.005)	(0.119)	(0.016)	(0.004)
Observations	606,110	606,110	604,261	601,679	606,344
R-squared	0.69	0.53	0.52	0.46	0.53
Mean	3524.6	.04	51.58	9.87	.03
Sample	Non movers	Non movers	Non movers	Non movers	Non movers

Notes: Pollution is measured at the home address for the mother. Non-movers are defined as families who do not move between municipalities. Number of observations varies between Columns, since not all outcomes are measured for especially the earliest cohorts. In Panel B the total is calculated as the average during the entire pregnancy. Across all regressions we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A5: In-utero exposure to air pollution and mental health - Non-mover

	(1) Age 5-8	(2) Age 5-12	(3) Age 5-8	(4) Age 5-12
Panel A: ADHD				
1st trimester	0.001	-0.002		
	(0.002)	(0.003)		
2nd trimester	0.003	0.001		
	(0.002)	(0.003)		
3rd trimester	0.003*	0.003		
	(0.002)	(0.003)		
Total			0.006	0.002
			(0.004)	(0.005)
Observations	366,233	366,233	366,233	366,233
R-squared	0.51	0.53	0.51	0.53
Mean	0.01	0.02	0.01	0.02
Panel B: Any psychiatric diagnosis				
1st trimester	0.001	-0.005		
	(0.003)	0.004)		
2nd trimester	0.002	-0.002		
	(0.003)	(0.004)		
3rd trimester	0.001	0.000		
	(0.003)	(0.004)		
Total			0.004	-0.003
			(0.005)	(0.008)
Observations	366,233	366,233	366,233	366,233
R-squared	0.52	0.54	0.52	0.54
Mean	0.02	0.06	0.02	0.06

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Non-movers are defined as families who do not move between municipalities. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.

Table A6: In-utero exposure to air pollution and infant health - Birth order

	(1) Birth weight	(2) P(LBW)	(3) Length	(4) APGAR	(5) Preterm
Panel A: Trimester					
1st trimester*10	-1.00	-0.001	-0.068	0.001	-0.002
	(5.01)	(0.002)	(0.055)	(0.007)	(0.002)
2nd trimester*10	-6.26	0.000	-0.099*	0.000	-0.001
	(4.79)	(0.002)	(0.052)	(0.007)	(0.002)
3rd trimester*10	-11.24**	0.002	-0.099*	-0.001	-0.000
	(5.42)	(0.002)	(0.059)	(0.008)	(0.002)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.53
Panel B: Total					
Total * 10	-15.67*	0.000	-0.246**	-0.006	-0.002
	(9.25)	(0.004)	(0.100)	(0.013)	(0.004)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.53	0.51	0.45	0.52
Mean	3525.63	.04	51.58	9.87	0.03

Notes: Pollution is measured at the home address for the mother. Number of observations varies between Columns, since not all outcomes are measured for especially the earliest cohorts. In Panel B the total is calculated as the average during the entire pregnancy. Across all regressions we control for the mothers age when she give birth, the sex of the child, maternal smoking, the average temperature each trimester and birth order. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A7: In-utero exposure to air pollution and mental health - Birth order

	(1) Diagnosed by age 8	(2) Diagnosed by age 12	(3) Diagnosed by age 8	(4) Diagnosed by age 12
Panel A: ADHD				
1st trimester*10	0.001	-0.000		
	(0.002)	(0.002)		
2nd trimester*10	0.003*	0.002		
	(0.002)	(0.002)		
3rd trimester*10	0.004**	0.004		
	(0.002)	(0.002)		
Total * 10			0.007**	0.005
			(0.003)	(0.004)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.52	0.51	0.52
Mean	.01	.03	.01	.03
Panel B: Any psychiatric				
1st trimester*10	0.001	-0.005		
	(0.002)	(0.003)		
2nd trimester*10	0.003	0.000		
	(0.002)	(0.003)		
3rd trimester*10	-0.001	-0.001		
	(0.002)	(0.004)		
Total * 10			0.004	-0.003
			(0.005)	(0.007)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.54	0.51	0.54
Mean	.03	.06	.03	.061
Fixed effects	Family	Family	Family	Family

Notes: Pollution is measured at the home address for the mother. Number of observations varies between Columns, since not all outcomes are measured for especially the earliest cohorts. In Panel B the total is calculated as the average during the entire pregnancy. Across all regressions we control for the mothers age when she give birth, the sex of the child, maternal smoking, the average temperature each trimester and birth order. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A8: In-utero exposure to air pollution and infant health - Municipality Fixed Effects

	(1) Birth weight	(2) P(LBW)	(3) Length	(4) Apgar score	(5) P(Preterm)
Panel A: Trimester					
1st trimester * 10	-1.597	-0.001	-0.044	0.001	-0.002
	(5.049)	(0.002)	(0.055)	(0.007)	(0.002)
2nd trimester * 10	-8.171*	0.001	-0.079	0.000	-0.001
	(4.821)	(0.002)	(0.052)	(0.007)	(0.002)
3rd trimester * 10	-11.946**	0.002	-0.077	-0.000	-0.000
	(5.474)	(0.002)	(0.059)	(0.008)	(0.002)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.52
Panel B: Total					
Total * 10	-17.761*	0.001	-0.182*	-0.006	-0.002
	(9.443)	(0.004)	(0.102)	(0.013)	(0.004)
Observations	812,504	812,504	810,049	806,843	812,504
R-squared	0.68	0.52	0.51	0.45	0.52
Mean	3525.63	.04	51.58	9.87	.03

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Across all regressions we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A9: In-utero exposure to air pollution and mental health - Municipality Fixed Effects

	(1) Diagnosed by age 8	(2) Diagnosed by age 12	(3) Diagnosed by age 8	(4) Diagnosed by age 12
Panel A: ADHD				
1st trimester	-0.000	-0.001		
	(0.002)	(0.002)		
2nd trimester	0.002	0.001		
	(0.002)	(0.002)		
3rd trimester	0.004**	0.003		
	(0.002)	(0.002)		
Total			0.005	0.003
			(0.003)	(0.005)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.53	0.51	0.53
Mean	0.01	0.02	0.01	0.02
Panel B: Any psychiatric diagnosis				
1st trimester	0.000	-0.005		
	(0.002)	(0.004)		
2nd trimester	0.003	0.000		
	(0.002)	(0.003)		
3rd trimester	-0.001	-0.001		
	(0.002)	(0.004)		
Total			0.003	-0.002
			(0.005)	(0.007)
Observations	478,280	478,280	478,280	478,280
R-squared	0.51	0.53	0.51	0.53
Mean	0.03	0.06	0.03	0.06

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.

Table A10: In-utero exposure to air pollution and infant health - Weather

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Panel A: Birth weight										
1st trimester*10	-8.26*	-2.27	-0.16	-4.70	-2.43					
	(4.80)	(4.99)	(5.08)	(4.99)	(5.09)					
2nd trimester*10	-13.05***	-9.11*	-6.07	-4.37	-1.92					
	(4.71)	(4.78)	(4.89)	(4.79)	(4.90)					
3rd trimester*10	-8.32	-13.36**	-11.54**	-12.11**	-10.82*					
	(5.15)	(5.40)	(5.53)	(5.42)	(5.55)					
Total*10						-26.36***	-20.69**	-14.30	-17.00*	-11.63
						(8.92)	(9.21)	(9.36)	(9.22)	(9.38)
Observations	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504
R-squared	0.68	0.68	0.68	0.68	0.68	0.68	0.68	0.68	0.68	0.68
Mean	3525.6	3525.6	3525.6	3525.6	3525.6	3525.6	3525.6	3525.6	3525.6	3525.6
Panel B: P(LBW)										
1st trimester*10	-0.000	-0.001	-0.001	-0.000	-0.000					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
2nd trimester*10	0.002	0.001	-0.000	-0.001	-0.001					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
3rd trimester*10	0.000	0.002	0.000	0.001	-0.000					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
Total*10						0.002	0.001	-0.001	-0.000	-0.002
						(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Observations	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504
R-squared	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52
Mean	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
Panel C: Length										
1st trimester*10	-0.105**	-0.075	-0.047	-0.079	-0.049					
	(0.053)	(0.055)	(0.056)	(0.055)	(0.056)					
2nd trimester*10	-0.132***	-0.106**	-0.087*	-0.072	-0.058					
	(0.051)	(0.051)	(0.053)	(0.052)	(0.053)					
3rd trimester*10	-0.062	-0.111*	-0.111*	-0.098*	-0.107*					
	(0.055)	(0.058)	(0.060)	(0.058)	(0.060)					
Total*10						-0.281***	-0.264***	-0.218**	-0.223**	-0.186*
						(0.095)	(0.099)	(0.100)	(0.099)	(0.100)
Observations	810,049	810,049	810,049	810,049	810,049	810,049	810,049	810,049	810,049	810,049
R-squared	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51
Mean	51.58	51.58	51.58	51.58	51.58	51.58	51.58	51.58	51.58	51.58
Panel D: APGAR										
1st trimester*10	-0.000	0.000	0.001	-0.000	0.001					
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)					
2nd trimester*10	-0.003	-0.001	0.001	0.001	0.002					
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)					
3rd trimester*10	0.001	-0.002	0.000	-0.002	0.000					
	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)					
Total*10						-0.008	-0.008	-0.005	-0.007	-0.004
						(0.013)	(0.013)	(0.013)	(0.013)	(0.013)
Observations	806,843	806,843	806,843	806,843	806,843	806,843	806,843	806,843	806,843	806,843
R-squared	0.45	0.45	0.45	0.45	0.45	0.45	0.45	0.45	0.45	0.45
Mean	9.87	9.87	9.87	9.87	9.87	9.87	9.87	9.87	9.87	9.87
Panel D: P(Preterm)										
1st trimester*10	-0.001	-0.002	-0.027***	-0.001	-0.0027***					
	(0.002)	(002)	(0.002)	(0.002)	(0.002)					
2nd trimester*10	0.000	-0.001	0.005***	-0.003	0.001					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
3rd trimester*10	-0.002	-0.001	0.011***	-0.001	0.008***					
	(0.002)	(002)	(0.002)	(0.002)	(0.002)					
Total*10						-0.001	-0.002	-0.008**	-0.004	-0.016***
						(0.003)	(0.003)	(0.003)	(003)	(0.003)
Observations	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504	812,504
R-squared	0.52	0.52	0.61	0.52	0.61	0.52	0.52	0.61	0.52	0.61
Mean	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03
Female	X	X	X	X	X	X	X	X	X	X
Maternal age	X	X	X	X	X	X	X	X	X	X
Smoking	X	X	X	X	X	X	X	X	X	X
Temperature	-	Linear	Bins	Linear	Bins	-	Linear	Bins	Linear	Bins
Precipitation	-	-	-	X	X	-	-	-	X	X
Cloud cover	-	-	-	X	X	-	-	-	X	X

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Across all regressions we control for the following covariates: the mothers age when she give birth, the sex of the child, maternal smoking. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A11: In-utero exposure to air pollution and mental health - Weather

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Panel A: ADHD age 5-8										
1st trimester*10	0.001	0.001	0.001	0.001	0.001					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
2nd trimester*10	0.003**	0.003*	0.003*	0.003**	0.003*					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
3rd trimester*10	0.004**	0.004**	0.004**	0.004**	0.004**					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
Total*10						0.007**	0.007**	0.007**	0.007**	0.007**
						(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Observations	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280
R-squared	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51
Mean	.01	.01	.01	.01	.01	.01	.01	.01	.01	.01
Panel B: ADHD age 5-12										
1st trimester*10	0.000	-0.000	-0.000	-0.000	-0.000					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
2nd trimester*10	0.002	0.002	0.002	0.002	0.002					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)					
3rd trimester*10	0.003	0.004	0.004	0.004	0.004					
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)					
Total*10						0.005	0.005	0.006	0.005	0.005
						(0.004)	(0.004)	(0.005)	(0.004)	(0.005)
Observations	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280
R-squared	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52
Mean	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
Panel C: Psychiatric diagnosis age 5-8										
1st trimester*10	0.00	0.00	0.00	0.00	0.00					
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)					
2nd trimester*10	0.00	0.00	0.00	0.00	0.00					
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)					
3rd trimester*10	-0.00	-0.00	-0.00	-0.00	-0.00					
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)					
Total*10						0.00	0.00	0.00	0.00	0.00
						(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Observations	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280
R-squared	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51	0.51
Mean	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
Panel D: Psychiatric diagnosis age 5-12										
1st trimester*10	-0.005	-0.005	-0.004	-0.005	-0.005					
	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)					
2nd trimester*10	-0.000	-0.000	-0.000	0.000	-0.000					
	(0.003)	(0.003)	(0.004)	(0.003)	(0.004)					
3rd trimester*10	-0.002	-0.001	-0.002	-0.001	-0.002					
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)					
Total*10						-0.004	-0.003	-0.004	-0.003	-0.004
						(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
Observations	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280	478,280
R-squared	0.54	0.54	0.54	0.54	0.54	0.54	0.54	0.54	0.54	0.54
Mean	.06	.06	.06	.06	.06	.06	.06	.06	.06	.06
Female	X	X	X	X	X	X	X	X	X	X
Maternal age	X	X	X	X	X	X	X	X	X	X
Smoking	X	X	X	X	X	X	X	X	X	X
Temperature	-	Linear	Bins	Linear	Bins	-	Linear	Bins	Linear	Bins
Precipitation	-	-	-	X	X	-	-	-	X	X
Cloud cover	-	-	-	X	X	-	-	-	X	X

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.

Table A12: Placebo exposure and infant health

	(1) Birth weight	(2) P(Low birth weight)	(3) Length	(4) Apgar score	(5) P(preterm)
One month after birth	1.63	-0.0006	-0.0565*	0.0022	-0.0014
	(2.86)	(0.0012)	(0.0308)	(0.0038)	(0.0011)
Observations	808,530	808,530	806,174	803,037	808,530
R-squared	0.68	0.53	0.51	0.46	0.52
Mean	3525.6	.04	51.58	9.87	.03

Notes: Note: Pollution is measured at the mother's home address. In all case is pollution is measured as the first full month after birth. The number of observations varies since, all outcomes are not measured for all children. In all regressions we control for the mothers age at birth, the sex of the child, indicator for maternal smoking, average temperature, total precipitation, average percentages cloud cover at the trimester level. Fixed effects for family, month and year of birth are included. Robust standard errors clustered at family in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A13: In-utero exposure to air pollution - Separability

	(1)	(2)	(3)
Panel A: Birth weight			
1st trimester*10		4.46	7.67
		(6.29)	(6.36)
2nd trimester*10	-8.20		0.57
	(6.28)		(6.58)
3rd trimester*10	-12.34*	-6.63	
	(6.51)	(6.75)	
Total*10	-2.23	-19.47	-28.19*
	(13.61)	(13.06)	(14.39)
Observations	812,504	812,504	812,504
R-squared	0.68	0.68	0.68
Panel B: ADHD - Age 8			
1st trimester*10		-0.002	-0.003
		(0.00)	(0.002)
2nd trimester*10	0.003		-0.000
	(0.002)		(0.003)
3rd trimester*10	0.004**	0.002	
	(0.002)	(0.002)	
Total*10	0.000	0.006	0.010
	(0.005)	(0.005)	(0.007)
Observations	478,280	478,280	478,280
R-squared	0.51	0.51	0.51
Panel C: ADHD - Age 12			
1st trimester*10		-0.002	-0.003
		(0.003)	(0.003)
2nd trimester*10	0.002		-0.001
	(0.003)		(0.003)
3rd trimester*10	0.004	0.002	
	(0.003)	(0.003)	
Total*10	0.001	0.001	0.010
	(0.007)	(0.007)	(0.007)
Observations	478,280	478,280	478,280
R-squared	0.52	0.52	0.52

Notes: Pollution is measured at the home address for the mother. Total is calculated as the average during the entire pregnancy. Across all specifications we control for the mothers age when she give birth, the sex of the child, maternal smoking, at the average temperature each trimester. We include year of birth, month of birth and family fixed effects. Robust standard errors are clustered by family level in parentheses. *** p<0.01, ** p<0.05, * p<0.