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Born in Smog: The Short- and Long-Run Health Consequences of Acute Air Pollution Exposure in Historical London, 1892–1919*

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Abstract

There is strong evidence that exposure to atmospheric pollution is detrimental to health. However, most current and historical research has focussed on the short-run consequences of exposure to pollution on health, and historical researchers have not been able to assess the effects of pollution on a wide range of health indicators. This paper uses fog events at a daily level as a proxy for acute extreme pollution events in historical London (1892-1919). It tests whether exposure to fog at birth and at the time of sickness influenced a wide range of indicators of child health in the short and long term, including birth outcomes (birth weight, length, stillbirth, premature birth and neonatal death), mortality risk (mortality before age 15), growth outcomes (heights and weights in infancy, childhood and adolescence), and morbidity outcomes (incidence, prevalence and sickness duration from respiratory diseases and measles). Being born on a fog day did not have strong effects on birth or growth outcomes or on morbidity outcomes for upper respiratory diseases. However, being born on a fog day increased mortality risk from respiratory diseases and increased incidence, prevalence and sickness duration from measles, influenza and other lower respiratory diseases. I also find short-run effects of fog on sickness duration from influenza and measles. Overall, the mixed results suggest that atmospheric pollution caused significant ill health in historical London but only for limited dimensions of health.

Keywords: ambient air pollution, morbidity, child growth, respiratory disease, health transition

JEL Codes: N33, I12, Q53

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1 Introduction

The air of nineteenth- and early twentieth-century industrial cities swirled with smoke, soot, and sulphur, the choking byproducts of the coal that powered factories and warmed homes. The smog in London was particularly bad, sometimes so thick that people could not see across the street and struggled to find their own homes (Luckin 2003, p. 35). This paper considers the short- and long-run health costs of this pollution for children living in London and assesses whether changes in pollution affected the health transition, the vast improvements in health that have occurred since the mid-nineteenth century.

Today, the World Health Organization states that 'air pollution is the most important environmental determinant of health' (WHO 2024, p. 2). Common pollutants including particulate matter (PM), sulphur dioxide (SO_2) and nitrous oxides (NO_x) damage health by irritating the lungs and respiratory tract and exacerbating respiratory disease and other respiratory complications including asthma (Manisalidis et al. 2020). Outside the respiratory system, the smallest particulate matter less than 2.5 μm in diameter ($PM_{2.5}$) can enter the bloodstream directly via the lungs and damage other body systems, increasing the risk of cardiovascular diseases, diabetes, obesity and neurological conditions. $PM_{2.5}$ pollution can also cross the placental barrier and affect children developing *in utero*, causing low birth weight and shorter gestational ages (Pryor et al. 2022). While most of the evidence of the harms of pollution is related to the immediate costs of pollution exposure (Currie et al. 2014), there is growing evidence that exposure to pollution *in utero* and in early life may have persistent consequences for health at later ages (Bharadwaj et al. 2016; von Hinke and Sørensen 2023).

The health consequences of pollution today are becoming clear, but applying these findings to the past is challenging for several reasons. First, we do not have instrumental measures of pollution before the 1950s that would allow us to track changes in historical pollution exposure or estimate health effects of specific dosages of pollution (Clay and Troesken 2011; Hanlon 2024). Second, the mix of pollutants historically was different from today with higher levels of SO_2, lower levels of NO_2 and similar levels of PM pollution between London and current polluted megacities (Brimblecombe and Grossi

2009). Third, people are far more aware of the health consequences of pollution today and practice avoidance behaviours that were not possible in the past, e.g. staying indoors or installing air purifiers. Thus, the effects of pollution exposure on health today are net of these avoidance behaviours and may underestimate the health costs in the past in the absence of avoidance. Fourth, before the introduction of vaccinations for common childhood diseases in the 1960s, nearly all children contracted diseases like measles and whooping cough. Today very few are infected, so any specific effect of pollution on outcomes from these diseases would not be measured with current data. Finally, both the health of the average person and medical technology and care have improved vastly since the nineteenth century. These improvements may attenuate the health costs of pollution today relative to historical contexts. Given the differences between past and present, it is necessary to study the health effects of pollution in historical settings directly.

This paper tests the effect of atmospheric pollution on health by focussing on London at the turn of the twentieth century, a time when London was extremely polluted (Brimblecombe and Grossi 2009; Hanlon 2024). I use fogs in London to proxy extreme pollution events (Clay and Troesken 2011). In London, fog events occurred on cold, rainless, still days where the pollution emitted from the city was trapped in place, leading to sharp, acute increases in pollution levels. The fog events were determined by meteorological phenomena and are therefore exogenous to individuals resident in London. Hanlon (2024) has used weekly data to show that these fog events affected mortality in London from 1866 to 1965. This paper uses daily fog data to measure the consequences of exposure to extreme pollution *in utero*, at birth and in the first month on the health outcomes of individuals in two data sources covering London 1892-1919: children born in the Queen Charlotte Hospital (a maternity hospital) and living in the Foundling Hospital (an orphanage). These sources allow me to analyse the effects of pollution on child health across a number of dimensions such as birth outcomes (birth weight, birth length, stillbirth, premature birth and neonatal death), mortality outcomes (mortality before age *c.* 15), growth outcomes (heights and weights in infancy, childhood and adolescence), and

morbidity outcomes (incidence,[1] prevalence[2] and sickness duration of respiratory diseases and measles).

The results are mixed. In the short run, fog events close to birth did not affect birth outcomes. Exposure to fog at the time of sickness influenced influenza and measles sickness durations, but did not affect catarrh (the common cold) and tonsillitis sickness duration. The long-run outcomes from exposure to pollution *in utero* and at birth were also mixed. *In utero* exposure to fog events did not strongly affect any health outcomes. Fog exposure around birth also did not affect child or adolescent growth or upper respiratory tract infections in late childhood and adolescence. However, children born on fog days and exposed to greater levels of fog in the first month of their life had higher mortality risk from respiratory causes. Children born on fog days also experienced greater incidence and prevalence of lower respiratory diseases between ages 5 and 10. Finally, children born on fog days had 77.3% and 27.2% longer sickness durations from influenza and measles respectively than children not born on fog days when exposed to these diseases between ages 5 and 15.

This paper makes three key contributions to the literature. First, it provides novel historical evidence on the long-run effects of exposure to extreme pollution at and around birth. The historical literature on the health costs of pollution to date has almost exclusively focussed on the short-run effects of pollution exposure. For example, Beach and Hanlon (2018) find strong effects of pollution, proxied by industrial coal consumption, upwind of a district on infant mortality in England in 1851-60. Clay et al. (2024a) show that the opening of new coal-fired power plants in the United States from 1938 to 1962 increased infant mortality. Hanlon (2024) shows that dense fogs in London led to sharp increases in weekly mortality, particularly affecting the elderly but also children and prime-age adults suffering from measles and tuberculosis respectively. The only historical paper looking at longer-term exposure is Bailey et al. (2018), which shows that British men born in more polluted districts had significantly lower adult stature. The focus on short-run effects of pollution exposure is also common in contemporary research as well

[1]Incidence is the number of sickness events per child year exposed.
[2]Prevalence is the time sick per child year exposed.

(Currie et al. 2014). However, a growing literature suggests that pollution exposure *in utero* and around birth has negative consequences for later health for both children and adults. In children, pollution exposure *in utero* and around birth increases the risk of developing asthma (Bharadwaj et al. 2016; Sbihi et al. 2016). In adults, individuals exposed to the Great London Smog in December 1952 *in utero* were more likely to experience respiratory hospitalizations in older age (Martin-Bassols et al. 2024; von Hinke and Sørensen 2023).

This paper builds from this literature by analysing the long-run effects of exposure to extreme pollution events in early life on childhood health in a historical population. The paper presents novel evidence that children exposed to extreme pollution (fog events) on their birthday were scarred by this experience. They faced heightened mortality risk, increased morbidity and longer sickness durations from measles and influenza. Thus, the health costs of pollution were not limited simply immediate responses to acute pollution events but also contributed to ill health for individuals years after the exposure. Contrary to the previous literature, I do not find long-run effects of *in utero* exposure to extreme pollution on health outcomes. This may be in part because this paper focusses on child health and cannot speak to old-age outcomes.

Second, as nearly all historical papers on the health effects of pollution have used mortality, a rare and extreme event, as their outcome variable, this paper contributes by exploring a much richer set of health outcomes. Historical fog events did not affect birth or child growth outcomes, which is somewhat puzzling since pollution has been found to affect both types of outcomes in previous historical and modern studies (Bailey et al. 2018; Hanlon 2024; Shah and Balkhair 2011; Spears et al. 2019). However, fog events capture a particular margin of pollution exposure, moving from high to extreme levels of pollution, and do not reflect differences between unpolluted and polluted environments. Thus, fog events may not capture chronic pollution exposure well. The morbidity outcomes also provide interesting insights. Not only did children born on fog days experience longer influenza sickness durations, they were also more susceptible to contracting influenza as well. The morbidity effects highlight that pollution influenced less extreme outcomes than

mortality and therefore had a wider impact on survivors' health.

Third, the paper builds on earlier work by exploring the interaction between pollution and specific diseases. While being born on a fog day had lasting effects on influenza and measles morbidity, it did not affect morbidity from less severe, upper respiratory infections such as catarrh and tonsillitis. The strong effects of pollution on influenza and measles morbidity is in line with the existing literature showing that mortality from the 1918 influenza pandemic was worse in more polluted environments (Clay et al. 2018; Franke 2022) and that fog events increased measles deaths in the short run (Hanlon 2024).

That pollution affected some diseases but not others yields insights into the mechanisms through which pollution affected health and the wider costs of pollution to child health. If asthma were the key mechanism through which pollution affected respiratory morbidity, then we would expect to see effects for all respiratory diseases, not just influenza. The importance of pollution exposure on the birthday, a novel finding in both the historical and contemporary literature, suggests that if the first breath a child takes is full of toxic pollutants, this may permanently damage their lungs, prime their immune systems for stronger responses to infection in the future, and increase their susceptibility to respiratory morbidity later in life. The limited effect of pollution on upper respiratory morbidity is also intriguing. Upper respiratory infections made up the vast majority of respiratory morbidity in the Foundling Hospital, 79.6% and 73.4% of incidence and prevalence respectively, raising questions about whether pollution could create enough morbidity to affect child growth (Bailey et al. 2018).

Overall, this paper shows that atmospheric pollution contributed to ill health in the past in real but also limited ways. Rising ambient pollution in the nineteenth century acted as a countervailing force to the improvements in health occurring as part of the health transition, but also reductions in pollution were unlikely to be important drivers of the health transition.

2 Data

2.1 Pollution Data

While contemporary studies focus on well-specified and defined definitions of pollution, instrumental measures of atmospheric pollution do not exist for the UK before the 1950s. This has led historical climatologists, meteorologists and economic historians to use other instrumental and non-instrumental weather sources to proxy pollution. The best estimates available on the development of pollution in London suggest that SO_2 and PM pollution peaked in the 1890s and either far exceeded or were similar to pollution levels in highly polluted megacities in the twenty-first century (Figure 1). NO_2 pollution peaked later because of rising transport emissions. While there was some improvement in pollution levels in the early twentieth century, the major reduction in pollution did not occur until after the Clean Air Act was passed in 1956 (Brimblecombe and Grossi 2009). However, while these estimates give a rough trend and peak level of pollution, they are not helpful for precisely estimating the effects of pollution on health. They are very rough estimates, hide considerable year-to-year and within-year variation, and since the estimates are for 20 to 50 year periods, prevent the use of identification strategies that could provide causal estimates of pollution on health.

This paper follows Clay and Troesken (2011) and Hanlon (2024) in using fog events as a proxy for pollution in historical London. The vast majority of fogs in London are radiation fogs. These occur in specific meteorological conditions. Typically, on a clear, cold day, radiation from the sun warms the ground during the day. Afterwards, during the night, the heat from the ground is released into the air, cooling the air close to the ground to below the dew point and allowing fog to emerge. Appendix Figure A.2 compares the weather conditions on foggy vs. non-foggy days, showing that fogs occur on cold days with relatively high pressure (clear skies), but the most strong difference between foggy and non-foggy days is in the wind speed. Fog is much more likely to form on very still mornings. Thus, we can think of fogs as proxying a set of weather conditions that prevent air pollution from being dispersed across space. Pollution being emitted by factories and

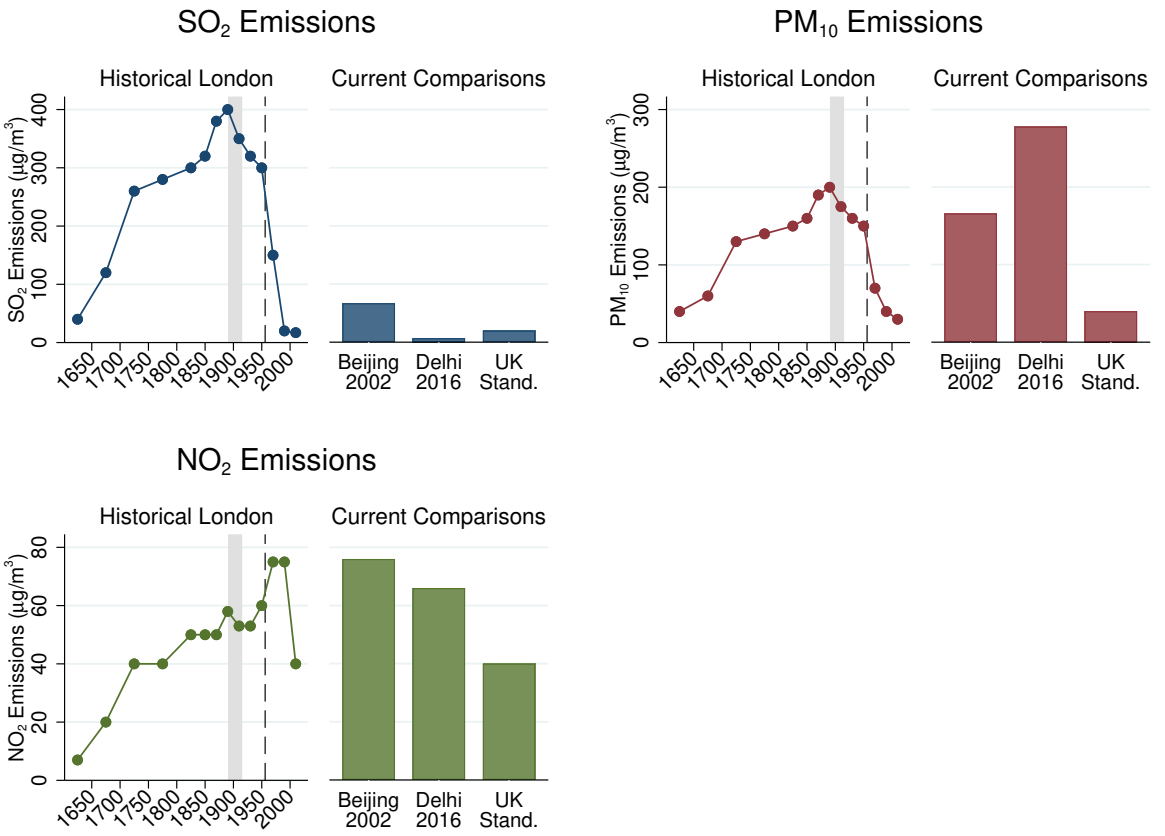

Figure 1: Estimated trends in major pollutants in London compared with current megacities and UK standards

Notes: Annual average pollution levels over time. The grey shaded area highlights the period studied in this paper and the black dashed vertical line marks the passage of the Clean Air Act in 1956. The London figures are predicted from data on coal imports to London, a good proxy for coal consumption. The estimates also take into account (through fairly rough assumptions) changes in the coal mix over time toward cleaner coal, improving combustion technology that reduced emission from factories and the expansion of the footprint of the city over this time period (Brimblecombe 1977). The PM_{10} emissions figures are predicted from the SO_2 figures before 1950, so these should not be read as independent.

Sources: Historical London - Brimblecombe and Grossi (2009, p. 1356); Beijing - Beijing Municipal Bureau of Statistics (2024, Chapter 7-23); Delhi - National Statistical Office, India (2024, p. 84); UK Standards - Department for Environment, Food and Rural Affairs (2023).

(more importantly in London) households simply stayed where it was rather than being blown down wind or purged from the air with rain. Where both fog and instrumental pollution measures overlap in the 1950s, Hanlon (2024) has shown that fog is a very good predictor of pollution levels.

The data on fogs comes from the Registrar General's Weekly Returns of Births and Deaths in London. The final page of the report includes a wide range of instrumental and non-instrumental measures of the weather as observed at the Greenwich Observatory. As part of this report, the officials provided general remarks about the weather each day (see Appendix Figure A.1 for an example). Here they noted when the weather was foggy, sometimes distinguishing levels of fog density as well. Hanlon (2024) used weekly counts of fogs to analyse short-run changes in mortality in London from 1866 to 1965. For this paper, I have recollected this data at the daily level in order to more precisely link fog exposure in precise critical windows (e.g. at birth) with subsequent health outcomes. Hanlon (2024) also focussed on dense fogs in his analysis of mortality, but I will include all fog days for a number of reasons. First, this paper analyses a wider set of health outcomes that are less extreme than mortality and therefore, less extreme pollution events may still matter. Second, while I am confident that meteorologists at the Greenwich Observatory could distinguish foggy conditions from non-foggy conditions, I am less confident that more subjective measures about the density of fog would be consistent across different observers. Finally, my datasets of health outcomes have limited sample sizes, so increasing the sample size of the treated fog group is necessary to be able to detect effects. A final concern is that the Greenwich Observatory is located on the southeastern outskirts of London rather than in central London where the hospitals were located (Appendix Figure A.3). While this may seem far, weather conditions are highly correlated across space, so it is unlikely that the low wind speeds that allowed pollution to build up would be present in Greenwich but not in central London even if fog were not present in both places.

2.2 Health Data: Queen Charlotte Hospital

The first source of health outcomes for this paper are the inpatient records of the Queen Charlotte Hospital.[3] The Queen Charlotte Hospital was a specialist maternity hospital founded in 1752. By the turn of the twentieth century, it was located on Marylebone Road in central London. The hospital had both outpatient and inpatient services. The outpatients were exclusively married women who were delivered by midwives in their homes, though complicated cases were transferred to the hospital. The inpatient service consisted of eight beds in the labour wards and fifty beds for lying-in patients: the patients remained in the hospital for the first two weeks following the birth, the lying-in period, where they recovered from childbirth. The inpatient service served both single and married pregnant women, but single women were required to be 'respectable', meaning that the child was their first illegitimate child and that they were of good standing before their 'fall'. The hospital was completely free to patients. Women mostly gained access to the hospital by receiving a sponsorship letter from a patron of the hospital but a few also applied independently. In practice, hospital patrons gave their sponsorship letters to clergymen or other social workers of the time who passed them along to deserving women, women who could not afford to be attended by a midwife and met the respectability criterion.

Once given a letter, the women attended the hospital in advance of their delivery to register and obtain an order of admission (Select Committee of the House of Lords on Metropolitan Hospitals 1891, pp. 519-25). The vast majority of women then travelled to the hospital after going into labour (Queen Charlotte Hospital 1892, p. 6). Women were admitted to the hospital throughout the day, but there were peaks in the late morning and late evening and fewer admissions between 11:00 pm and 2:00 am (see Appendix Figure C.4 for the distribution). The women gave birth at median 5.72 hours after admission and 91.7% gave birth within 36 hours of admission, suggesting that most women were not receiving prepartum care. The remaining 8.3% of women were in the hospital for longer before delivery and may have been complicated cases transferred from

[3]Records on outpatients are not available until the 1930s.

the outpatient services. While I include these women in the analysis, excluding them does not qualitatively alter the findings (results not reported).

The hospital's register of inpatients listed each mother's name, address, age, marital status, number of previous pregnancies, dates and times of admission and delivery and the midwife who attended the birth (see Appendix A.2 for more detail). There was also information about the health of the child including their presentation during delivery, sex, whether they were born alive or stillborn, whether they were premature or not, their birth weight and length, their date of discharge and whether the child died before discharge. There were 31,239 deliveries in the Queen Charlotte Hospital between 1892 and 1913, representing 1.12% of births in London over that period. I collected a random sample of 160 births from each year, which serve as the basis for the analysis in this paper. Descriptive statistics are reported in Appendix Table A.1.

Considering the representativeness of women giving birth in the Queen Charlotte Hospital, the women were negatively selected on socioeconomic status and health in some ways. They were poor working-class women who could not afford to hire a midwife on their own. At 45% of patients, single women were also greatly over-represented relative to the population of women giving birth in London. They likely had fewer resources and diminished support relative to their married counterparts. In addition, because the hospital only delivered single women for their first illegitimate birth, the share of primiparous mothers was higher than in the London population: over 60% of women in the Queen Charlotte Hospital were giving birth for the first time. First births tend to have more complications and also lower birth weights, so this may skew the sample relative to the London population (Schneider 2017).

However, there were a few ways that the women were positively selected. The respectability requirement likely excluded the most desperately poor women. Women suffering from infectious disease at the time of delivery were also not admitted, so none of the women were actively suffering from tuberculosis for instance. Finally, the women were required (if able) to breastfeed their children during the lying-in period in the hospital, which likely increased the breastfeeding rates among the women and may have benefited

the children's health (Queen Charlotte Hospital 1892, p. 6). Despite these issues, the Queen Charlotte sample still provides useful information about child health in historical London.

2.3 Health Data: Foundling Hospital

The second source of data for this paper is the extremely data rich cohort study of children that I have reconstructed from the records of the London Foundling Hospital. Although called a hospital, the institution was more similar to an orphanage. It was founded in 1739 to care for the children of unwed mothers from infancy until the children reached maturity. Although it operated for a short period in the eighteenth century like a typical European foundling hospital, taking in all children given up by their mothers, it shifted policy in 1760 introducing selection criteria to the admissions process, which were still in place in the late nineteenth century (Levene 2007). To be admitted, children had to be the first-born, illegitimate child of an otherwise respectable woman, and their mother had to have been abandoned by their father. The Foundling Hospital staff conducted extensive interviews and checks to ensure that the women met these criteria. In prior work, we tested for selection into the Foundling Hospital and found virtually no selection into the hospital based on observable characteristics (Arthi and Schneider 2021).

Figure 2 presents the life stages of children under Foundling Hospital care. Because mothers were not allowed to apply to give up their children until after the child was born, children spent their first few months living with their mothers (life stage 1): the median age at admission was 91 days old. 80% of the children were born in London with 20% coming from outside London (see Appendix A.3.4 for more detail). After being admitted, the children were sent to small, rural villages in Kent, Surrey and later Essex to be fostered with respectable families (life stage 2). The children remained in the countryside until they were five or six years old when they returned to the Foundling Hospital main site in Bloomsbury, central London. In the hospital's records, the return from the countryside is called re-admission. Finally, from ages 5 to 15 or 16, the children lived in the Foundling Hospital main site (life stage 3) until they were discharged from the hospital. A small

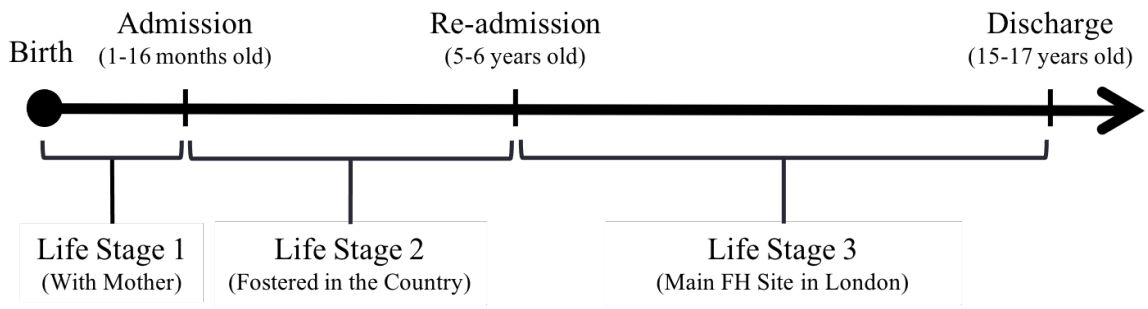

Figure 2: Life stages of children in the Foundling Hospital

number of children (2.5% of the sample) were restored to their mothers before discharge and fall out of observation, but there is no evidence of selective loss to follow up through this mechanism (Arthi and Schneider 2021).

The analysis in this paper focusses on children admitted to the Foundling Hospital between 1893 and 1914 when the hospital admitted on average 50 children per year. Starting in January 1893, the medical officer, William John Cropley Swift, kept a medical record of each child admitted, recording considerable details about the children's health at admission, re-admission and discharge. The medical record was no longer updated with re-admission and discharge information from April/May 1919, likely when Cropley Swift retired as medical officer. The full details of the information available are presented in Appendix Table A.2, but the important health outcomes for this paper are anthropometric outcomes (weight at admission; height and weight at re-admission; and height and weight at discharge) and mortality with causes of death. Descriptive statistics and further detail are provided in Appendix A.3. The medical record also includes the dates of birth, admission, re-admission, discharge and if necessary dates the child was restored to their mother or died so that the periods of exposure in each life stage are very well defined for each child.

In addition to keeping the medical register, the medical officer also managed the hospital's infirmary, which treated children living in the Foundling Hospital's main site during life stage 3 when they got sick. The medical officer kept a weekly infirmary report that listed all the children in the infirmary that week, their age, their date of admission to the infirmary and the disease for which they were being treated. The children discharged

that week and their date of discharge were also listed. This data therefore allows me to reconstruct the morbidity history of children including all the diseases they were treated for in the infirmary and their sickness duration for each sickness event. Importantly, this data is for a defined population, so it is possible to compute precise incidence and prevalence rates.

While the weekly infirmary reports begin in the eighteenth century, the focus of this paper is on sickness events between March 1897 and October 1915. During this period, there were 6,409 sickness events with 695 different disease classifications which I coded into 205 diseases using the ICD-10. Sickness duration could be reconstructed for 97.2% of sickness events. These diseases varied from mundane things like catarrh, the common cold, and minor injuries to more serious infectious diseases like measles and bronchitis and chronic diseases like epilepsy. One might worry that the medical officer had fixed rules about how long children were to remain in the infirmary for specific types of diseases, but Appendix Figure A.4 shows that there was large variation in sickness duration from all respiratory diseases, suggesting that sickness duration was actually capturing children's sickness experiences.

Because this paper studies the health effects of air pollution, I will focus on respiratory infections and measles which in severe cases has respiratory complications. Table 1 presents the number of cases, incidence, prevalence and median sickness duration for respiratory diseases with more than five cases in the data. The most common diseases were catarrh, tonsillitis and influenza, though measles' greater sickness duration made its prevalence greater than that of influenza. The table also indicates which respiratory diseases are considered upper respiratory diseases affecting the nose, throat and larynx and lower respiratory diseases affecting the bronchial tubes and lungs. This data allows me to test the effects of pollution on individual-level incidence and prevalence of disease as well as sickness duration of sickness events.

Overall, the Foundling Hospital Cohort Dataset is an unique historical source. The extent and quality of the longitudinal data is unparalleled for this time period. In particular, the quality of the morbidity data is more complete than many current surveys of

Table 1: Period respiratory morbidity in the Foundling Hospital

Disease	Upper/Lower Resp. Disease	Cases	Incidence	Prevalence	Median Sickness Duration (days)
Catarrh	Upper	1083	162.67	3.78	6
Tonsillitis	Upper	846	127.07	2.92	7
Influenza	Lower	426	63.99	1.69	7
Chronic Tonsillitis	Upper	193	28.99	.56	6
Bronchitis	Lower	50	7.51	.45	12
Bronchopneumonia	Lower	30	4.51	.37	20
Pneumonia	Lower	11	1.65	.13	24.5
Sore Throat	Upper	9	1.35	.04	6
Laryngitis	Upper	7	1.05	.02	7
Pharyngitis	Upper	6	.9	.01	5
Measles		315	47.31	1.81	13

Notes: Incidence is cases per 1,000 child years exposed. Prevalence is years sick per 1,000 child years exposed. These are period rates reflecting the population of children in the hospital between the start and end of the infirmary records. Sample sizes are smaller in the analysis below because the data is restricted to children born in London or to a subset of cases (see Appendix A.3.5 for further discussion). In the analysis below, tonsillitis and chronic tonsillitis are combined because the two diseases behave similarly.

Sources: Foundling Hospital Cohort Dataset (2025).

respiratory morbidity. The fact that the same medical officer recorded sickness events for the entire period (with a few gaps for his holidays) makes the data far more comparable than studies based on different doctors' diagnoses. However, the foundling children are a selected and unrepresentative group of children, so it is important to assess the external validity of findings from this dataset.

In some dimensions, the health of children in the Foundling Hospital was better than for a typical London child of the period. The children were relatively well nourished compared to other London children: their rate of child stunting, the share of children too short for their age, was lower than that of London children as measured by the London County Council (London County Council 1907; Schneider et al. 2024). They also received better medical care: mortality rates in the Foundling Hospital between ages 1 and 5 were lower than in London and the surrounding counties where the children were fostered (Arthi and Schneider 2021). Few children in London were cared for by a doctor and team of nurses every time they got sick. Epidemics were far less frequent in the Foundling Hospital than in surrounding London. For instance, the hospital did not have a measles case between June 1903 and February 1911 despite regular biennial epidemics outside

(Hardy 1993, p. 30). The hospital also managed to avoid water-borne diseases during the period covered here. While there was a typhoid epidemic in 1891, there were no cases of typhoid in the 18 years studied here (Cropley Swift 1911, p. 8). This was atypical for London where typhoid was still common even if typhoid mortality rates had fallen (Hardy 1993, p. 152).

On the other hand, there were ways in which the foundling children's health was inferior to typical London children. They were less likely to have been breastfed in infancy than typical children (Arthi and Schneider 2021) and as illegitimate children whose fathers did not support them likely did not have the same care in early life as legitimate children. The fact that the children lived in large wards with many other children may have increased their viral loads and hence sickness severity when an epidemic did break out. It also seems unlikely that the children received the same kind of attention and care in an institutional setting as they would have received in a family. Still, despite these caveats, the Foundling Hospital Cohort Study provides the richest detail on child health of any dataset available for this period.

3 Methods

This paper examines the effect of acute extreme pollution exposure (fog days) on children's health in both the short run and long run. Short-run outcomes, occurring within days of pollution exposure, include the effects of pollution on birth outcomes and sickness events aged c. 5-15. Long-run outcomes, occurring long after pollution exposure, include mortality (outside the neonatal period), child growth, and the incidence, prevalence and duration of sickness from respiratory diseases or diseases with respiratory complications (measles). I analyse the long-run costs of pollution exposure *in utero*, on a child's birthday, and in the first month of their life. Greater discussion of the logic behind the empirical strategy is included in Appendix B.

3.1 Empirical Equations: Short-run Effects

To understand the effects of fog exposure at birth on birth outcomes, I use regressions of the following form:

$$Y_i = \alpha + \beta Fog_b + X'\gamma + Q_b + C_b + W_b + \epsilon_i \tag{1}$$

where Y_i are birth outcomes for child i. Birth weight and length as continuous variables are estimated via OLS whereas the binary outcomes stillbirth, premature birth and neonatal death in the first two weeks of life are estimated with logistic regressions. The main variable treatment of interest is Fog_b, which is a binary variable equal to 1 if a child was born on a fog day and zero otherwise. b indexes the birthday as the day of fog exposure, but this will be allowed to vary later. Note though that these regressions are purely cross-sectional in nature. The regressions include individual level controls ($X'\gamma$) for the child's sex, mother's marital status, mother's age and its square, and child's parity, which can all affect birth outcomes. Q_b are quarter of birth fixed effects, which control for any confounding from seasonality. C_b are birth year fixed effects with the year beginning July 1st so as not to break the year during middle of winter when pollution and respiratory disease are at their worst. These control for any trends over time in birth outcomes and pollution exposure. W_b are weather condition controls in the week of birth including temperature, pressure, rainfall and humidity and their squares. Thus, the logic of this identification strategy is that assignment into treatment (being born on a fog day) is random. The other controls are included to ensure balance between the treatment and control group on other potentially important characteristics. The regressions are estimated on birth outcomes using the Queen Charlotte Hospital sample.

The estimation strategy is similar when analysing the short-run effects of pollution exposure on children's sickness duration in the Foundling Hospital main site in central London from *c.* ages 5 to 15. Here the empirical equation takes the form:

$$D_j = \alpha + \beta_1 Fog_t + X'\gamma + Q_t + Y_t + W_t + \epsilon_j \tag{2}$$

where D_j is the sickness duration for sickness event j. I focus on respiratory diseases and measles because there is strong biological grounds for thinking that these diseases were affected by pollution.[4] Fog_t is a binary variable taking one if a child was exposed to fog on the day they entered the infirmary (t) and zero if not. The regressions include individual controls ($X'\gamma$) for child's sex and age at the beginning of the sickness event. Q_t and Y_t are quarter and year fixed effects respectively related to the beginning date of the sickness event (t). W_t are again weekly weather controls with respect to time t. Because sickness duration is a count variable, I use zero-truncated negative binomial models to estimate the regressions. I use heteroskedasticity robust standard errors rather than cluster-robust standard errors because there is no clustering in sampling or treatment assignment (Abadie et al. 2023).

For the short-run sickness duration variables, I also consider lagged exposure to fog taking the following form:

$$\begin{aligned} D_j = \alpha + \phi_1 \sum_{s=7}^{9} Fog_{t-s} + \phi_2 \sum_{s=4}^{6} Fog_{t-s} + \phi_3 \sum_{s=1}^{3} Fog_{t-s} + \beta_1 Fog_t \\ + X'\gamma + Q_t + Y_t + W_t + \epsilon_j \end{aligned} \tag{3}$$

where common variables with Equation 2 are the same, but I have added counts of the number of fogs in three day intervals going back nine days before admission to the infirmary on day t.

3.2 Empirical Equations: Long-run Effects

To estimate the long-run effects of pollution exposure at or around birth, we can re-use Equation 1 substituting health outcomes later in childhood for Y_i: mortality, child growth,

[4] This helps to reduce false positives from testing the effect of fog on types of disease where we would not necessarily expect pollution to matter.

individual-level incidence and prevalence of disease, and sickness duration at the sickness event level. I employ different regression models depending on the structure of the data: Cox proportional hazard models and competing risk models for mortality; OLS models from child growth and individual-level incidence and prevalence of disease; and zero-truncated negative binomial models for sickness duration. The range of individual-level controls included depends on the models and data available. I also restrict the analysis to children born in London since only children born in London would be exposed to London pollution at birth (see Appendix A.3.4). For the Foundling Hospital cohort, this is relatively straightforward to do although there are a few children who were born in London, but moved outside London shortly after birth. Thus, they are included in the sample when analysing exposure at or before birth, but removed when analysing exposure after birth.

Building on Equations 1 and 5 above, I also explore exposure to pollution after birth in the following form:

$$Y_i = \alpha + \eta_1 \sum_{s=1}^{28} Fog_{b+s} + X'\gamma + Q_b + C_b + W_b + \epsilon_i \tag{4}$$

where $\eta_1 \sum_{s=1}^{28} Fog_{b+s}$ is the count of fogs an individual was exposed to in the first four weeks of their life. I focus on the first four weeks because this is the traditional neonatal period, and only 0.7% of Foundling Hospital children were admitted before 28 days of age.

When analysing the long-run effects of pollution exposure around birth on sickness duration for sickness events, I also include sickness quarter, year and age fixed effects. While these variables are unlikely to be confounders, I include them to ensure balance between the treatment and control groups. I cluster the standard errors at the individual level since some children were sick from the same disease more than once and their treatment assignment, in relation to their birth date, is clustered (Abadie et al. 2023). Note also that the sample of sickness events under analysis changes when analysing short-

run and long-run effects of pollution exposure because I do not have exact birthdates for children born before 1892 and the fog data ends on 24 October 1914, about a year before the final entries in the infirmary book. This issue is discussed in detail in Appendix A.3.5.

Finally, we can extend the baseline regression above to capture prenatal exposure to pollution as well:

$$Y_i = \alpha + \phi_1 \sum_{s=169}^{252} Fog_{b-s} + \phi_2 \sum_{s=85}^{168} Fog_{b-s} + \phi_3 \sum_{s=1}^{84} Fog_{b-s} + X'\gamma + Q_b + C_b + W_b + \epsilon_i \tag{5}$$

where the common variables with Equation 4 are the same, but we add counts of the number of fogs experienced in the first ($\sum_{s=169}^{252} Fog_{b-s}$), second ($\sum_{s=85}^{168} Fog_{b-s}$) and third ($\sum_{s=1}^{84} Fog_{b-s}$) trimester of pregnancy assuming all children were born at full term.[5] This measure captures pollution exposure of the mother while the child was *in utero*. The *in utero* effects are tested against all outcomes.

3.3 Identification and Selection

A key assumption of the empirical strategy is that fog events are exogenous to individuals' characteristics.[6] While the samples used in this paper are not random samples of the population as described above, because we are comparing children selected through the same process, selection would only be a threat to internal validity if the selection procedure for children exposed to fog was somehow different from those not exposed to fog. Given that fog events were determined by meteorological conditions and women have limited control over when they give birth, it is hard to imagine how selection into giving birth on a fog day would be influenced by anything other than the extreme pollution event.[7] Nor would selection be a problem for short-run sickness events in the Foundling Hospital since we observe all children at risk. However, selection into admission to the Queen Charlotte or Foundling Hospital could potentially be a problem if the selection

[5] Unfortunately, gestational age was not systematically reported for all children, so this is the only way to assign exposure.

[6] Other, less important identification concerns are discussed in Appendix C.

[7] Note that caesarian sections and inductions were extremely rare in this period.

procedure differed by fog exposure. I will consider selection into each institution in turn.

For the Queen Charlotte Hospital, we might worry that fogs affected the types of women who could travel to give birth in the hospital, especially since there is anecdotal evidence that travel was difficult on the heaviest fog days (Luckin 2003, p. 35). However, in my sample, there is no evidence that births were less likely to occur on fog days: 12.4% of births in my sample occurred on fog days and 11.9% of days in the same period were fog days. The difference in these proportions is tiny and statistically insignificant. Interestingly, the timing of admission seems to vary somewhat on fog days (Appendix Figure C.4). The typical morning and evening peaks in admissions were less pronounced on fog days, perhaps because women delayed coming to the hospital until the morning fog had lifted or had to travel more slowly due to low visibility. However, this difference in admission time does not appear to have affected the care received at the hospital: the median time between admission and delivery was 24 minutes longer for children born on fog days. There were also no meaningful or statistically significant differences in maternal age, marital status or parity between mothers giving birth on fog days or not (Appendix Table C.1). Thus, there do not appear to be systematic differences between children in the Queen Charlotte Hospital being born on fog days or not.

Turning to the Foundling Hospital, the first worry might be that children born on fog days might differ by parental characteristics. Appendix Table C.2 shows that there are no differences in their father's social status, and while children born on fog days had mothers who were 0.62 years older on average, this difference is small and unlikely to strongly influence child health (see Appendix Figure C.6). We might also worry that children born on a fog day would be less likely to be admitted to the Foundling Hospital because they had a greater risk of death before admission while they were living with their mothers. This does not appear to be the case. The share of admitted children born on a fog day (12.2%) is not meaningfully or statistically different from the expected share if we assume children at risk of admission were born uniformly across the year from the earliest child's birthday to the latest one's (12.0%). Admission ages were also not different, so the two groups did not face differences in exposure before admission. The Foundling

Hospital itself could have also potentially contributed to the selection. We can test this from 1909 to 1914 when we have information on both accepted and rejected applications. However, there is no difference in acceptance rates between children born on a fog day or not (Appendix Table C.2).

Although there are no differences in the share of children born on fog days or admission age, we might worry that children born on fog days would face greater mortality risks and that this mortality might selectively cull the weakest individuals, leading to survival bias, a form of collider bias (Schneider 2020). We can test this by comparing the birth weights and lengths of a subsample of 160 children born in the Queen Charlotte Hospital and later admitted to the Foundling Hospital (Appendix Table C.2). While the birth weights and lengths of children admitted to the Foundling Hospital born on fog days are greater than those not born on fog days, the differences are not statistically significant and they are similar in magnitude to the differences found at birth when analysing the full sample of children born in the Queen Charlotte Hospital, indicating very little survival bias in this setting.

Finally, one might worry that children born on fog days were visibly weaker and that this might have affected the care they received from their mothers before admission or their mother's decision to give them to the Foundling Hospital. However, this seems unlikely. As we will see, there was no effect of being born on a fog day on birth weight or length, raising doubt about whether these children did appear weaker. In addition, there were no differences in the breastfeeding rate or breastfeeding duration between children born on fog days or not. The child's age at which the mothers applied to the Foundling Hospital was also not different between the groups (see Appendix Table C.2). Thus, there do not appear to be reinforcing or compensating investments in relation to exposure to severe pollution at birth.

Given this discussion, I argue that fog events are randomly assigned with respect to individuals and sickness events and can therefore provide causal estimates of the effect of pollution on both short- and long-run health outcomes. However, the margin of pollution studied here should be interpreted as moving from high levels to extreme levels of

pollution, a particular margin that may not translate to moving from low to high levels of pollution.

4 Results: Short-Run Effects

I begin with short-run effects of pollution exposure, focussing on birth outcomes and sickness duration from respiratory diseases in late childhood and adolescence.

4.1 Birth Outcomes

Beginning with birth outcomes, I do not find strong effects of fog events on birth outcomes. There is no effect of fog exposure at birth or fog exposure in the week before birth on birth weight (Appendix Table D.1). While most gains in infant weight are made across the third trimester (Hanson et al. 2015), we might expect that a pollution spike could trigger early labour and lead children to be born at an earlier gestational age with a lower birth weight. This does not appear to be the case in this period, nor is there an effect of fog exposure on prematurity either.[8] In fact, fog exposure at birth and in the three days before birth has a small positive and statistically significant effect on birth length (Appendix Table D.2) with children born on fog days being 0.4 cm longer (10% of an sd of birth length). This is an unexpected and puzzling result, though it is compatible with the birth weight results which show small positive effects that are not statistically significant. This positive effect cannot be explained by survival bias because it remains when I include or exclude stillbirths and also there is no effect of fog events around birth on stillbirths (Appendix Table D.3). Since the fog exposures are close to birth and the fetuses are mostly also close to term, it seems unlikely that there are unobserved stillbirths biasing the results. We do have to be somewhat circumspect about the stillbirth results though because both stillbirths and fog events are rare events, so the power to estimate the effect of fog events at birth on stillbirths is low. There is also no effect of fog events on infant deaths in the first two weeks after birth (Appendix Table D.3). In summary,

[8]Note that I do not observe gestational age, only whether the child was full term or not, so there is potential measurement error in this variable.

there are not important health consequences of extreme pollution exposure close to birth on short-run birth outcomes.

4.2 Sickness Duration

The second set of short-run effects relate to exposure to fogs at the time a child contracted an illness at ages *c.* 5 to 15. I focus on sickness duration from three respiratory illnesses catarrh, tonsillitis and influenza that are among the most prevalent in the data and measles which often results in respiratory complications. Unfortunately, there were too few sickness events for bronchitis and pneumonia to analyse them separately. The regressions are estimated with zero-truncated negative binomial models to account for the count nature of sickness duration as a variable.

Table 2 presents the results. There are no statistically significant or meaningful effects of recent pollution exposure on sickness duration from catarrh or tonsillitis. There is a marginally statistically significant, positive effect of fog exposure on the day of admission to the infirmary on influenza sickness duration. This amounts to a 34.1% increase in sickness duration (3 additional days sick at the mean of 9.7 days) for those admitted on a fog day versus those admitted on a normal day. One might worry that this effect is driven by outliers, but looking at the raw sickness duration distributions presented in Figure 3A, it is clear that the distribution of influenza sickness duration for children admitted on a fog day is shifted to the right. For measles, there is no effect for the day of admission, but there is a statistically significant effect of the number of fog events 1 to 3 days before admission on measles sickness duration. Each extra fog event increases the sickness duration by 14.2% (2 additional days sick at the mean of 14.3 days). Again, these differences are not driven by outliers (Figure 3B). The difference in the timing of the effect for influenza and measles may be driven by the different incubation periods for each disease. Influenza has a very short incubation period of 1-2 days (Treanor 2014, p. 467) whereas measles has a longer incubation period of 10 days (Moss and Griffin 2014, p.542). Therefore, it makes sense that exposure closer to entry to the infirmary is more important for influenza compared to measles.

Table 2: Effect of pollution exposure at time of sickness on sickness duration from respiratory diseases and measles

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Measles
$\sum_{s=7}^{9} Fog_{t-s}$	0.062	-0.030	0.092	0.067
	(0.075)	(0.033)	(0.081)	(0.074)
$\sum_{s=4}^{6} Fog_{t-s}$	0.094	0.032	-0.102	0.055
	(0.066)	(0.035)	(0.063)	(0.084)
$\sum_{s=1}^{3} Fog_{t-s}$	0.002	0.064	-0.047	0.133**
	(0.050)	(0.042)	(0.082)	(0.063)
Fog_t (Ad Day)	0.043	-0.034	0.293*	-0.075
	(0.118)	(0.082)	(0.171)	(0.112)
Sex Dummy	Yes	Yes	Yes	Yes
Sickness Weather	Yes	Yes	Yes	Yes
Sickness Age FE	Yes	Yes	Yes	Yes
Sickness Year FE	Yes	Yes	Yes	Yes
Sickness Quarter FE	Yes	Yes	Yes	Yes
$\sum Fog_t$	123	119	49	24
N (sickness events)	1021	1010	398	269

Notes: Estimated with zero-truncated negative binomial models. Coefficients with robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively.

Sources: Foundling Hospital Cohort Dataset (2025).

While the non-significance and small effect sizes for catarrh and tonsillitis suggest that the results are not an artefact of omitted variables or other unobservable biases, I also conduct a placebo check and show that fog events at or before admission do not affect sickness durations from injuries where we would expect no effect to exist (Appendix Table D.16).

5 Results: Long-Run Effects

Having discussed short-run effects, I now turn to longer-term effects of pollution exposure around birth on health at later ages. This includes the effect of *in utero* exposure to pollution on birth and later outcomes as well as pollution exposure at and after birth on later outcomes.

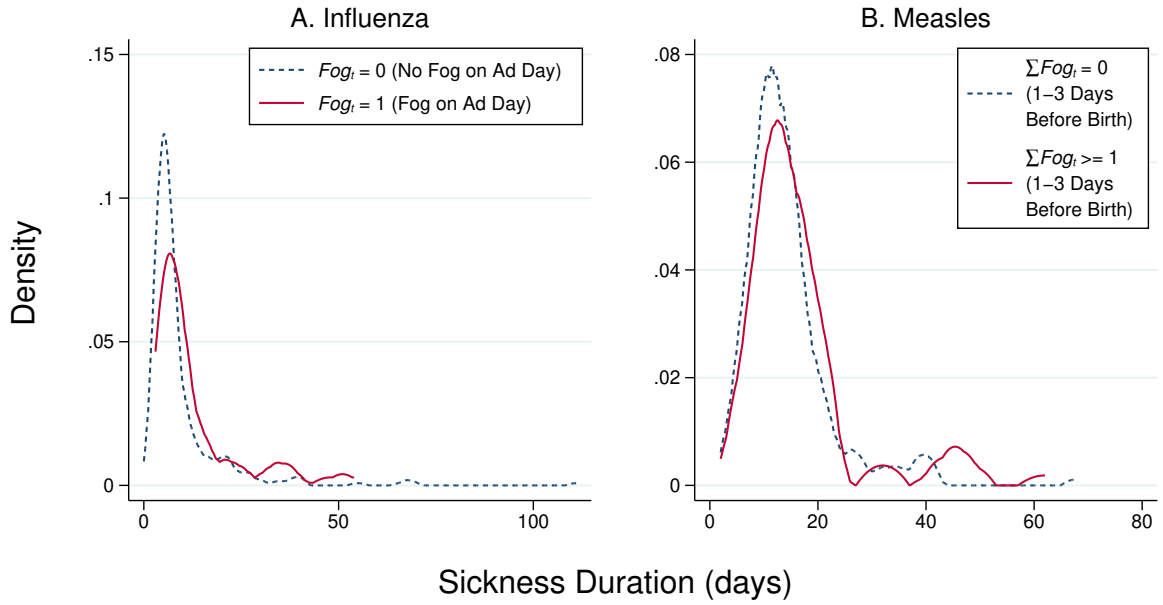

Figure 3: Raw distribution of influenza sickness duration for children admitted to the infirmary or not on fog days

Sources: Foundling Hospital Cohort Dataset (2025).

5.1 Birth Outcomes

In addition to the short-run effects described in the previous section, we can also explore the longer-run effects of exposure to pollution in the various stages of pregnancy on birth outcomes. Here the power issues of the short-run effects are less of a problem because the pollution exposure variable is the sum of fog events in each trimester of pregnancy rather than a binary comparison of those born on fog days or not. The results are presented in Appendix Tables D.5-D.7 and show no statistically significant or historically meaningful effects of *in utero* exposure to fog events on birth weight, stillbirth, neonatal death and premature birth. There is a significant effect of second trimester fog exposure on birth length with a one standard deviation increase in fog exposure leading to a very small 0.07 standard deviation decrease in birth length. Overall, though it appears that *in utero* pollution exposure did not affect birth outcomes.

5.2 Mortality

Turning next to mortality, unfortunately, because death is a relatively rare event and the total sample size in the Foundling Hospital Cohort Dataset is relatively small, there are limitations to what we can learn about the long-run effects of pollution on mortality. However, mortality is one of the few health outcomes available in this dataset that reveals how pollution affected the health of young children under age five, who were particularly prone to respiratory disease. Thus, it is worth tentatively exploring this outcome.

Table 3 shows the pollution exposure stratified by three categories: survival, deaths from respiratory causes and deaths from other causes. Respiratory causes refer to cause of death descriptions that reference influenza, bronchitis, pneumonia, tuberculosis[9] or whooping cough. The medical officer often reported multiple causes of death, so a death is counted as having a respiratory cause if one of these five diseases is mentioned. Table 3 shows that 95 out of 760 children who survived were born on a fog day (12.5%) . Children who died of a respiratory cause were more likely to have been born on a fog day (18.9%), but children dying of other causes were less likely to have been born on a fog day (5.8%). Note though that the power for these calculations is very limited: only 7 children who died of respiratory diseases were born on a fog day. The differential pollution exposure is also present when analysing the number of fogs that children experienced in the first 28 days after their birthday. Children dying of respiratory diseases experienced on average 4.6 fog events in their first 28 days, whereas survivors and children who died of other causes experienced an average of 3.5 and 3.0 fog days respectively.

These differences are intriguing, but they should not be interpreted naively since the simple averages do not account for seasonality or the censoring in the data based on the ages at which children were admitted and discharged from the hospital. In addition, the differential effect of pollution exposure on mortality from respiratory causes versus other causes suggests that competing risks may be important in understanding this effect. Children born on fog days may die from respiratory causes before becoming at risk of non-respiratory causes. When competing risks are present, traditional Cox proportional

[9]Tuberculosis includes pulmonary and general tuberculosis but excludes tuberculosis not present in the lungs.

Table 3: Differences in pollution exposure stratified by survival status

	Fog_b (Birthday)			$\sum_{s=1}^{28} Fog_{b+s}$	
Mortality Status	Share	Sum	N	Mean	SD
Survivor	0.125	95	760	3.45	3.81
Death (Respiratory Cause)	0.189	7	37	4.57	4.10
Death (Other Cause)	0.058	4	69	3.00	3.95

Notes: Fog_b is an indicator variable equal to one if a child was born on a fog day and 0 otherwise. $\sum_{s=1}^{28} Fog_{b+s}$ is the number of fog events a child is exposed to in the first 28 days after the birthday. The sample is limited to children born in London and further limited to children living in London after birth when analysing fog exposure after birth.

Sources: Foundling Hospital Cohort Dataset (2025).

hazard models can provide biased estimates of the effect of covariates on the cause-specific mortality risk (Andersen et al. 2012). Therefore, competing risk models are required to model the cause-specific mortality risk (Fine and Gray 1999).

Table 4 confirms that competing risks are an issue in this setting. Specifications 1-3 estimate the effect of being born on a fog day and the cumulative exposure to fog events in the first 28 days after the birthday on mortality risk between admission and discharge from the Foundling Hospital. The coefficients are very close to one and insignificant showing no effect. However, when using a competing risk model to account for potential bias, we see that children born on a fog day are more than twice as likely to die from respiratory causes. This effect is only statistically significant at the 10% level, but the magnitude is very large. Cumulative pollution exposure in the 28 days after the birthday also affected mortality risk with a one standard deviation increase in fog exposure (3.8 fog events) leading to a 40% increase in mortality risk, statistically significant at the 5% level. I also tested for the effect of pollution exposure *in utero* on mortality risk, but there were no significant effects (see Appendix Table D.11). Because the sample size is limited, these results should not be over-interpreted, but they do present strongly suggestive evidence that pollution exposure not only had a short-run influence on mortality, as Hanlon (2024) has shown, but also raised mortality risk from respiratory causes across an individual's childhood and adolescence.[10]

[10]Note that although the Foundling Hospital Cohort Dataset is not ideal for estimating this kind of effect, it is currently impossible to estimate it for London from other sources. The only way to do this

Table 4: Effect of pollution exposure on mortality risk

	(1) All	(2) All	(3) All	(4) Respiratory	(5) Respiratory	(6) Respiratory
Fog_b (Birthday)	0.941	0.949		2.256*	2.212*	
	(0.319)	(0.325)		(1.028)	(1.025)	
$\sum_{s=1}^{28} Fog_{b+s}$		1.047	1.047		1.092**	1.096**
		(0.033)	(0.033)		(0.046)	(0.046)
Sex Dummy	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Decade FE	Yes	Yes	Yes	Yes	Yes	Yes
$\sum Fog_b$	106	102		106	102	
N	860	829	829	860	829	829

Notes: The sample is restricted to children born in London and is restricted further to children living in London with their mothers before being admitted to the Foundling Hospital when analysing the effect of fog exposure in the first 28 days. Individuals enter observation upon admission to the Foundling Hospital and are censored at discharge, death or the date when the medical record stopped being updated. Hazard ratios with standard errors in parentheses. Models 1-3 employ the Cox Proportional Hazard model. Models 4-6 use a competing risk model where respiratory deaths are the outcome of interest and other causes of death are the competing risk. Mean and SD of $\sum_{s=1}^{28} Fog_{b+s}$ are 3.5 and 3.8 respectively. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

5.3 Individual-Level Morbidity Results

While the mortality results in the last section are intriguing, it is also important to consider how pollution affected the morbidity experience of children. I begin by analysing the morbidity experiences of individual children between ages *c.* 5 and 10 before analysing sickness events. The age range is restricted to ages 5 to 10 to increase the number of children in the sample. Analysing a longer age range substantially reduces the sample size (see Appendix Figure A.5).

We begin by analysing sickness incidence, the number of times a child experiences a given disease or category of diseases per year of exposure between a child's readmission to the Foundling Hospital and age 10. For instance, some children would never get sick from flu whereas others might experience it several times between readmission and age

would be to link individual-level birth and death records for London in order to obtain the birth date and the cause of death. Collecting data of this kind would require negotiating access with the UK government and a multi-million pound data transcription and linkage effort and therefore is out of the scope of the current project.

Table 5: Effect of pollution exposure at birth on individual-level sickness incidence

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Upper Resp	(5) Lower Resp
Fog_b (Birthday)	-0.021	0.002	0.056***	-0.013	0.054**
	(0.039)	(0.035)	(0.019)	(0.052)	(0.022)
	[-0.025]	[0.003]	[0.167]	[-0.011]	[0.135]
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
Mean Dep. Var.	0.21	0.17	0.05	0.39	0.08
$\sum Fog_b$	63	63	63	63	63
N	471	471	471	471	471

Notes: Individual-level sickness incidence is the number of cases per year of exposure between re-admission and age 10. The sample is restricted to children observed in the infirmary data to age 10 and children born in London. For definitions of upper and lower respiratory diseases, see Table 1. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses and standardised coefficients in square brackets. *** denotes statistical significance at the 1% level. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

10. Table 5 shows that being born on a fog day increases individual-level incidence of influenza and lower respiratory diseases but does not affect upper respiratory diseases such as catarrh and tonsillitis. The effects for influenza and lower respiratory diseases are both statistically significant and large in magnitude relative to upper respiratory diseases. The influenza incidence rate for children born on fog days is double the mean influenza incidence rate in the sample. Thus, children exposed to extreme pollution at birth seem to be more susceptible to influenza infections later in childhood.

We can also analyse the effect of fog exposure on individual sickness prevalence, the percentage of time exposed that an individual was sick. This captures both how often they contracted the disease but also the sickness duration of their sickness events. Table 6 shows that the effect of pollution exposure at birth on sickness prevalence is fairly similar to incidence. There are large effects of pollution exposure at birth on influenza and lower respiratory prevalence, but smaller and insignificant effects for upper respiratory diseases. The magnitude of the coefficients on upper respiratory diseases are larger than for incid-

Table 6: Effect of pollution exposure at birth on individual-level sickness prevalence

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Upper Resp	(5) Lower Resp
Fog_b (Birthday)	0.083	0.161	0.191***	0.264	0.198**
	(0.203)	(0.136)	(0.071)	(0.243)	(0.082)
	[0.026]	[0.075]	[0.178]	[0.065]	[0.116]
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
Mean Dep. Var.	0.51	0.44	0.14	0.96	0.23
$\sum Fog_b$	63	63	63	63	63
N	471	471	471	471	471

Notes: Individual-level sickness prevalence is the number of sickness days per 100 days exposed between re-admission and age 10 or the percentage of days spent sick. The sample is restricted to children observed in the infirmary data to age 10 and children born in London. For definitions of upper and lower respiratory diseases, see Table 1. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses and standardised coefficients in square brackets. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

ence but still substantially smaller than the very large effects for influenza. Again, it is clear that extreme pollution exposure on the day of birth matters for lower respiratory morbidity in later childhood.

Appendix Tables D.12 to D.15 report the results of pollution exposure *in utero* and in the 28 days after birth on incidence and prevalence, and largely show that these effects are statistically insignificant or only marginally significant (at the 10% level). Puzzlingly, these coefficients tend to be negative, suggesting that being exposed to more fog events during these periods decreases sickness incidence and prevalence. However, the effects sizes tend to be small, so we should not over-interpret these. These results confirm that extreme pollution exposure on the day of birth has a particularly strong effect on lower respiratory morbidity later in childhood.

5.4 Sickness Duration Results

I now turn from analysing individual-level morbidity data to using sickness events themselves as the unit of observation in order to study sickness duration directly. When studying sickness duration, I do not combine larger categories of respiratory diseases because the diseases have different distributions of sickness duration (see Appendix Figure A.4), which would mean that the composition of disease within these categories might drive the results. Table 7 presents the results. There is no effect of being born on a fog day for catarrh or tonsillitis, but children born on fog days have longer, statistically significant sickness durations from influenza and measles when experiencing these diseases between the ages of c. 5 and 15. These are large effects: children born on a fog day have 77.3% longer flu sickness durations (about 7 days increased sickness duration from a mean of 9.7 days) and 27.2% longer measles sickness durations (about 4 days increased sickness duration from a mean of 14.5 days).

Again, one might reasonably worry that this result is being driven by outliers, so Figure 4 plots the raw distributions of sickness duration for influenza and measles for children born on a fog day or not. For children born on a fog day, the entire distribution of influenza sickness duration is shifted upwards, so the results are not driven by outliers. For measles, the sickness duration distribution for children born on fog days appears very similar to the distribution of children not born on fog days at lower lengths of sickness duration, but there is a subset of children born on fog days who have longer sickness events (though still within the range observed in the control group). This is what we would expect since long sickness durations from measles occur when measles leads to pneumonia. Thus, it appears that children born on fog days are more likely to experience respiratory complications from measles and therefore have longer sickness durations.

Interestingly, at least for influenza, these effects are specifically localised to the day of birth. Appendix Table D.17 shows that the effects do not hold for children exposed to fog the day before birth or the day after birth. For measles, there is no effect the day before birth, but a similar effect the day after birth. When analysing *in utero* exposure and exposure in the first 28 days, again there are not strong influences of *in utero* ex-

Table 7: Effect of pollution exposure at birth on sickness duration from respiratory diseases and measles

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Measles
Fog_b (Birthday)	-0.032	0.125	0.573***	0.241**
	(0.131)	(0.098)	(0.148)	(0.115)
Sex Dummy	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
Birth Year Trend	Yes	Yes	Yes	Yes
Sickness Quarter FE	Yes	Yes	Yes	Yes
Sickness Year FE	Yes	Yes	Yes	Yes
Sickness Age FE	Yes	Yes	Yes	Yes
$\sum Fog_b$	109	108	36	29
Clusters (individuals)	393	364	174	245
N (sickness events)	800	704	235	245

Notes: Sample restricted to children born in London. Estimated with zero-truncated negative binomial models. Coefficients with standard errors clustered at the individual level in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

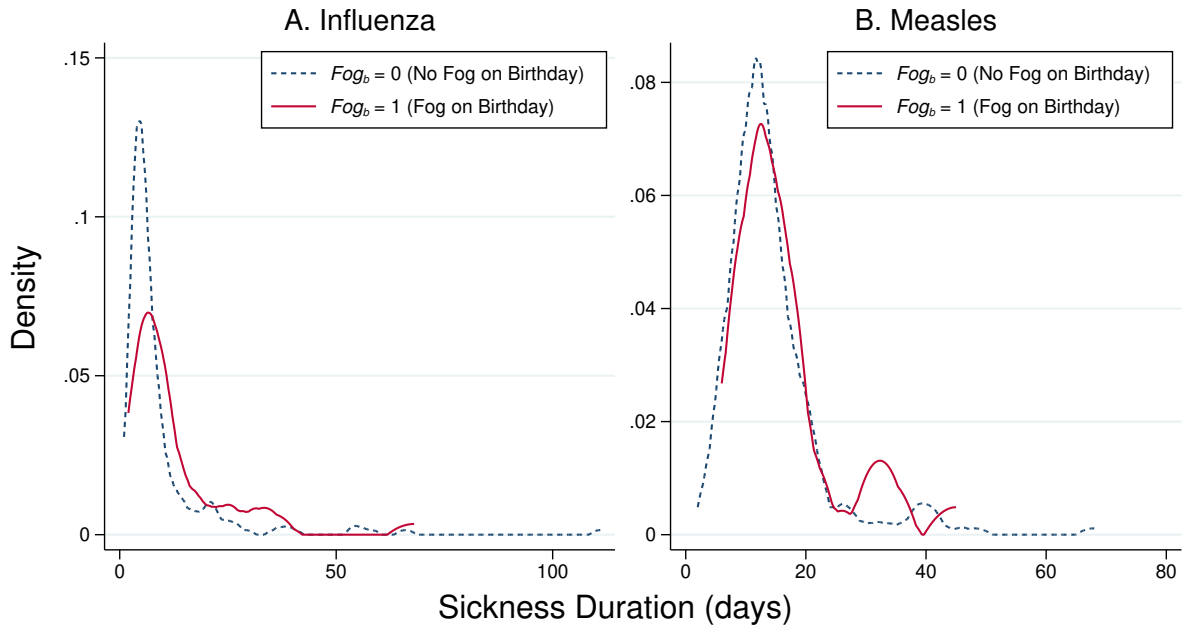

Figure 4: Raw distribution of influenza and measles sickness duration for children born and not born on fog days

Sources: Foundling Hospital Cohort Dataset (2025).

posure or exposure after birth (Appendix Tables D.18 and D.19). Thus, there is a special consequence of being exposed to extreme pollution on the day of birth.

Finally, we can test the relative magnitude of the effect of pollution exposure at birth and short-run pollution exposure at the time of sickness by analysing both forms of exposure in a subsample of sickness events (see Appendix Figure A.5C). Table 8 shows that, in this subsample, the magnitude of the effect of being born on a fog day is slightly larger than the magnitude of experiencing a fog day upon admission to the infirmary at the start of the sickness incident.[11] Thus, the long-run effects of pollution exposure at birth are large and important in influencing children's morbidity experience, at least for influenza.

5.5 Anthropometric Outcomes

The final set of long-run outcomes analysed are measures of weight, height and BMI for the children that were taken at admission, re-admission and discharge. These have been standardised using the WHO growth standard/reference to account for the fact that children were not all measured at the same age. The results are presented in Appendix Tables D.8-D.10. Exposure to fog at birth and in the 28 days after the birthday is never statistically significant. When analysing pollution exposure *in utero*, exposure in the first and second trimesters weakly increases weight-for-age Z-scores in childhood, but these coefficients are only statistically significant at the 10% level. Overall, though, there is little evidence that this margin of pollution exposure strongly affected child growth.

6 Discussion

This paper presents mixed results of the impact of pollution on child health in turn-of-the-twentieth-century London. The null results with respect to birth outcomes are incongruous with modern studies that show effects of pollution on birth outcomes (Currie

[11] The short-run effect size is larger than that reported in Table 2 above. It is difficult to fully explain this, but note that this is a separate subsample, so there may be composition effects or different influenza strains over time that are affecting the magnitude of the coefficients.

Table 8: Effect of pollution exposure at birth and sickness event on sickness duration from influenza and measles

	(1) Influenza	(2) Influenza	(3) Influenza	(4) Measles	(5) Measles	(6) Measles
Fog_b (Birthday)	0.573***		0.629***	0.241**		0.193*
	(0.148)		(0.129)	(0.115)		(0.103)
Fog_t (Ad Day)		0.520**	0.442***		-0.170	-0.152
		(0.203)	(0.168)		(0.121)	(0.128)
Sex Dummy	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	No	Yes	Yes	No	Yes
Birth Weather	Yes	No	Yes	Yes	No	Yes
Birth Quarter FE	Yes	No	Yes	Yes	No	Yes
Birth Year Trend	Yes	No	Yes	Yes	No	Yes
Sickness Weather	No	Yes	Yes	No	Yes	Yes
Sickness Quarter FE	Yes	Yes	Yes	Yes	Yes	Yes
Sickness Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Sickness Age FE	Yes	Yes	Yes	Yes	Yes	Yes
$\sum Fog_b$	36		34	29		29
$\sum Fog_t$		28	27		22	21
Clusters (individuals)	174	163	162	245	224	221
N (sickness events)	235	226	223	245	224	221

Notes: The sample is restricted to children born in London. Estimated with zero-truncated negative binomial models. Coefficients with standard errors clustered at the individual level in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

et al. 2014; Johnson et al. 2021; Siddika et al. 2016). However, it is important not to over-represent the magnitude of these effects. Taking historical stillbirths as an example, (Hanlon 2024) found that *in utero* exposure in the first trimester and exposure in the four weeks before birth to heavy fog events in historical London increased stillbirths by 0.7% and 1.55% respectively. While these effect sizes are within the confidence intervals of the effects reported in this paper, they are still small. A recent meta-analysis found that the effects of pollutants on the risk of stillbirth were of a similar order of magnitude, though the meta-analysis results were not statistically significant (Siddika et al. 2016). For reference, the stillbirth rate fell from a high of 41.4 stillbirths per 1,000 total births in 1933 to 22.6 in 1950 in England and Wales, a 45.4% decline over that period (Office for National Statistics 2022). The modern effects of pollution on birth weight are also very small (Currie et al. 2014), which makes sense from a historical perspective given that mean birth weights have not changed dramatically since the nineteenth century in polluted industrial cities despite sharp reductions in pollution (Schneider 2017). One might further doubt these small effect sizes because of the potential for publication bias or because most studies are not able to analyse exogenous variation in pollution exposure. In any case, it is hard to argue that pollution was a major determinant of poor birth outcomes in the past.[12]

This paper's mortality results present a potential way of reconciling conflicting results in the current literature on pollution and infant mortality in historical Britain. Studying fog events in London from 1866 to 1965, Hanlon (2024) finds no short-run effect of fog days on infant mortality in weekly data, but Beach and Hanlon (2018) find that plausibly exogenous spatial variation in industrial coal pollution from upwind districts affected average infant mortality rates at the registration district level in England and Wales in the 1850s. This paper confirms Hanlon (2024)'s finding that fog events at birth did not affect infant deaths associated with prematurity: there was no short-run effect of fog events on neonatal mortality, nor did fog events trigger early labour since there were no

[12]One caveat to this is that I am least certain about the location of mothers in both samples in the first trimester, so if some of them were not living in London this could attenuate the effects of the *in utero* exposures (see Appendix A.3.4).

negative effects on birth weight or length. However, I do find that children born on fog days and exposed to greater numbers of fogs in the first month of life had substantially higher risk of respiratory mortality after being admitted to the Foundling Hospital in infancy even though they were living in the countryside removed from London's horrid pollution. This suggests that the higher infant mortality in polluted places may not have arisen from short-run excess mortality from extreme pollution events but from the longer-term effect of extreme pollution exposure in early life on exacerbating serious respiratory infections. The absence of effects of pollution on prematurity and the fact that there does not appear to be selective culling of children born on fog days before admission to the Foundling Hospital (median age of 3 months) suggests that the costs of early life pollution exposure were stronger later in infancy, in the post-neonatal period (age 1 month to 12 months).

The most exciting and novel results in this paper, however, are the morbidity results. There are two surprising findings. First, acute extreme pollution events in both the short and long term only influenced lower respiratory disease morbidity (influenza, bronchitis and pneumonia) and not upper respiratory disease morbidity (catarrh and tonsillitis). This fits well with the mortality results since lower respiratory infections are much more likely to result in death and also with earlier studies that showed that pollution strongly affected mortality during the 1918 influenza pandemic (Clay et al. 2018; Franke 2022). However, when considering respiratory morbidity in the Foundling Hospital, upper respiratory infections made up 79.6% and 73.4% of incidence and prevalence of all respiratory diseases, so they were dominant in children's experience of sickness. This raises questions about the extent to which pollution exposure could create the kind of chronic respiratory morbidity that could influence child growth and may help to explain why I do not find any effect of pollution exposure on child growth contrary to existing historical literature (Bailey et al. 2018). On the other hand, median sickness durations of 6 or 7 days for common respiratory infections seems long given what children experience today and suggests that chronic pollution exposure may have exacerbated respiratory infections.

The second surprising finding is that there were long-run effects of acute extreme

pollution exposure on the day of birth on mortality risk from respiratory infections and sickness incidence, prevalence and duration from influenza and other lower respiratory diseases. Contemporary and historical studies have mostly focussed on either the short-run effects of acute pollution exposure or long-run effects of chronic pollution exposure. While there is evidence that acute pollution exposure *in utero* during the Great Smog of London in 1952 affected old age respiratory morbidity (Martin-Bassols et al. 2024; von Hinke and Sørensen 2023), this paper is the first to my knowledge to document such specific health costs of being born during an acute extreme pollution event. Being born on a fog day made children more likely to contract lower respiratory infections and increased sickness duration for lower respiratory diseases and measles. The magnitude of these long-run effects was also larger than the short-run effects. For instance, being born on a fog day increased influenza sickness duration by 77.3% whereas entering the infirmary on a fog day resulted in a 34.1% increase in sickness duration. The gap between these effects was smaller when including both in the same regression (see Table 8), but in any case, the magnitude of the long-run effect was large.

I have not found studies analysing the health costs of acute pollution exposure precisely on an individual's birthday, so the biological mechanisms that could produce this result are somewhat speculative. One mechanism might be asthma. Perinatal pollution exposure increases the risk of young children developing asthma (Sbihi et al. 2016), and asthma increases the risk of contracting respiratory diseases and exacerbates these infections (Sharma et al. 2022). However, asthma exacerbates both upper and lower respiratory infections. For instance, individuals with asthma exposed to the common cold, an upper respiratory infection, are more likely to experience lower respiratory symptoms than individuals without asthma (Corne et al. 2002). Since I do not find effects for upper respiratory infection, asthma does not seem a likely candidate mechanism. Another mechanism might be differences in gestational age since preterm children's lungs are not fully developed (Warburton 2017). However, there is no evidence that children born on fog days had lower gestational age since they did not have lower birth weight and were not more likely to be recorded as premature.

However, there are three possible mechanisms that could explain the results. First, pollution might cause damage to developing lung structures and inhibit the structural changes in the lungs that occur at birth. The lungs undergo a rapid and fundamental transformation at birth from being fluid-filled and positive pressure *in utero* to being gas-filled and lower pressure. At birth, the fluid in the lungs is rapidly absorbed, the alveoli (air sacs where gas transfer occurs) inflate with the first breath and the surfactant system (chemicals that reduce surface tension in the alveoli) springs into action to facilitate gas transfer in the alveoli (Warburton 2017). It is possible that exposure to extreme pollution in the immediate postnatal period could affect these processes, damaging alveoli and surfactant development and increasing the risk of respiratory disease (Ubags et al. 2020). Second, pollution exposure at birth may change how the immune system develops and functions in the lungs perhaps priming the immune system to more severe response to infection in the future (Ubags et al. 2020). The neonatal immune system operates differently from the immune system even later in infancy (Levy and Wynn 2014), which may explain the localised effect at birth. These more severe responses (e.g. cytokine storm) can exacerbate respiratory infections later in life (Chang et al. 2024). Finally, there is evidence that pollution exposure can lead to epigenetic changes in lung cells (Durham and Adcock 2013). While the prenatal period is seen as most sensitive for epigenetic changes, postnatal changes are possible and especially in the respiratory system which changes so dramatically at birth (Marsit 2015).

In extending these findings to the wider literature, two limitations must be considered. First, the children in the Queen Charlotte and Foundling Hospital samples were not necessarily representative of all children in London, so we need to consider the external validity of the findings (see also the discussion of representativeness in Section 2). For the Queen Charlotte Hospital, if anything, the fact that the patients tended to be poor and single might make them more susceptible to pollution as they likely lived in more polluted areas and had poorer baseline health. Thus, the fact that there is no effect even among these women suggests that the results are likely to apply to other women in London. The Queen Charlotte patients were also disproportionately drawn from less polluted

West London whereas East London tended to be more polluted because prevailing winds blew the pollution toward the north east (Heblich et al. 2021). Although this may mean that the shock of a fog event was smaller in magnitude for women in the sample than for women in East London, the shock would still be large relative to current pollution exposures that have been found to influence birth outcomes. Therefore, there is no reason to believe that the null effects for birth outcomes are related to the geographic distribution or non-representativeness of women in the Queen Charlotte Hospital.

Assessing the external validity of effects from the Foundling Hospital sample is more challenging since the children were healthier and had access to better medical care than the typical London child of the time, but also lived in an institutional setting, increasing their exposure to respiratory disease. Of course, the children were not born in the institution, so aside from being illegitimate and primiparous, the children's early life experiences would not have been so different from the typical London child. The institutional setting makes it difficult to assess the short-run effect of fog events on contracting respiratory diseases, which are not analysed in this paper. For epidemic diseases like influenza and measles, the timing of infection was determined by the introduction of the pathogen to the population rather than individuals pollution-related susceptibility. Catarrh and tonsillitis were endemic but even then the fact that children slept in wards with up to forty other children suggests that the disease transmission process was not comparable to that of the typical child. This also may have meant that average viral loads were higher for these children compared to the typical child. However, this would have affected all children equally, so it is not clear that this would bias the differential fog effect found in this paper. Finally, most of the mortality in the Foundling Hospital data occurred while the children were fostered with families in the countryside from infancy to *c.* age 5. Thus, the mortality results may capture more typical living conditions.

A final concern for external validity is the pollution proxy used in this paper. Fog events proxy an acute increase in pollution from high to extreme levels far above the annual average levels in Figure 1. However, all of the children were exposed to high levels of pollution similar to recent levels in Beijing and Delhi while they were living in London.

This makes it difficult to extrapolate for instance from the results on mortality risk to long run changes in respiratory mortality in London over time. It also means that the margin of pollution exposure in this paper may not capture the effects of persistent, chronic exposure to high pollution on health. While I do not find effects of the number of fog events individuals were exposed to *in utero* and limited effects for fog exposure in the first month of life, the number of fog events is still an imperfect proxy for chronic exposure.

7 Conclusion

Using a wide range of health outcomes from two historical datasets, this paper has tested the effects of acute exposure to extreme pollution (fog events) on individual-level health outcomes in both the short and long-run. While there are no effects of fog events on birth outcomes or child growth, there were short-run effects of fog events on influenza and measles sickness duration and there were long run effects of pollution exposure at birth on mortality from respiratory diseases and incidence, prevalence and sickness duration from lower respiratory diseases and measles. Although mixed, these effects suggest that the health costs of pollution were a countervailing force to the improvements in health that were occurring during London's health transition. The high baseline pollution levels (Figure 1) and regular fog events (Figure 5A) contributed to increased morbidity and mortality from respiratory diseases in the capital. However, as is clear from Figures 5B and 5C, childhood mortality rates from bronchitis, other respiratory diseases and measles fell dramatically between 1890s and 1920s despite the high levels of pollution whereas pneumonia mortality remained more or less stagnant. The frequency of fog events did fall from the 1890s to the 1910s, reducing some of the exposure to extreme pollution events, but it is hard to believe that this decline or the modest declines in average pollution rates presented in Figure 1 could explain the substantial fall in mortality.

Translating these pollution effects to trends in health indicators over time is challenging for a number of reasons. First, sometimes coal pollution was associated with in-

creasing incomes and other amenities that could counteract the health costs (Clay et al. 2024b). For instance, studying the twentieth-century United States, Clay et al. (2024a) find that when new coal-fired power plants were opened in locations with low baseline access to electricity, the benefits of gaining access to electricity offset the costs of increased local pollution and infant mortality was unaffected by the new plant. However, when new plants were opened in locations with high electricity access, the new plants led to significant increases in infant mortality. Thus, pollution may have heterogeneous effects depending on how it relates to these other factors. Second, the health costs of extreme pollution events may have changed over time. Hanlon (2024) finds that the short-run effect of London fog events on mortality became smaller over time, and the decline in the effect was particularly pronounced for measles. One explanation for this diminishing effect is that average pollution levels were falling, if modestly (see Figure 1), and therefore the spike in pollution from fog events became smaller over time. However, it may also indicate that new technologies or practices mitigated the health costs of pollution. Taking measles as an example, a reduction in the effect of fog events on measles mortality would not be driven by the fact that fewer children were contracting measles. Measles is the most infectious human pathogen, and all children continued to contract measles until the introduction of a vaccine in the 1960s. Instead, it suggests that other factors such as improving nutrition, better nursing care or pollution avoidance mitigated the fog effect over time (Schneider 2023). Grappling with these complex factors is difficult, and more research is needed to understand the changing relationship between pollution and health over time. However, on balance it seems more likely that increasing pollution served as a countervailing force to the health transition, slowing improvements in health over time, rather than reductions in pollution being an important cause of improving health across the health transition.

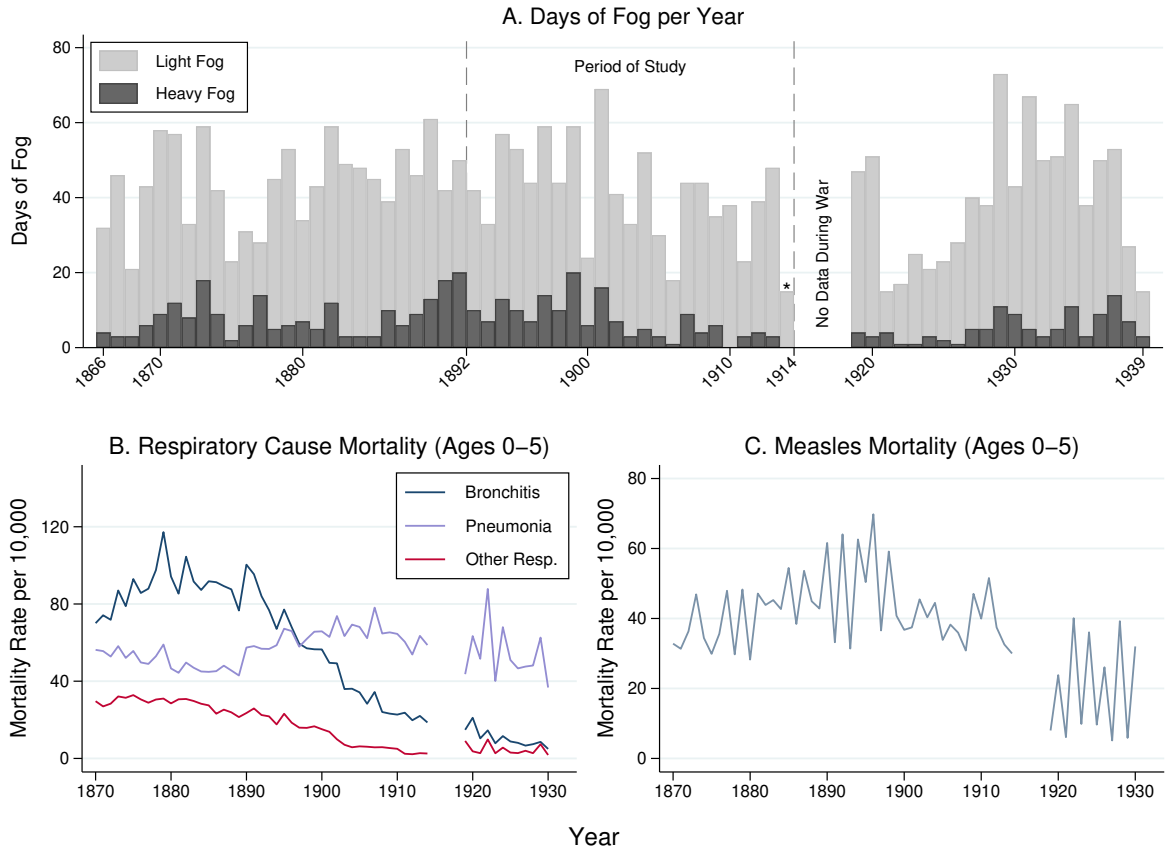

Figure 5: Fog events and respiratory and measles mortality in London, 1866-1939

Notes: For panels B and C, the population at risk was interpolated for the 0 to 5 age group using geometric interpolation between census population counts. Other resp. includes all deaths from respiratory diseases aside form bronchitis and pneumonia. *Data for 1914 is incomplete stopping on 24 October.

Sources: Mortality and fog data was transcribed from the Weekly Return of Births and Deaths in London ... (1870-1930) by Hanlon (2024). Population counts by age for London were drawn from various census publications from 1861 to 1931.

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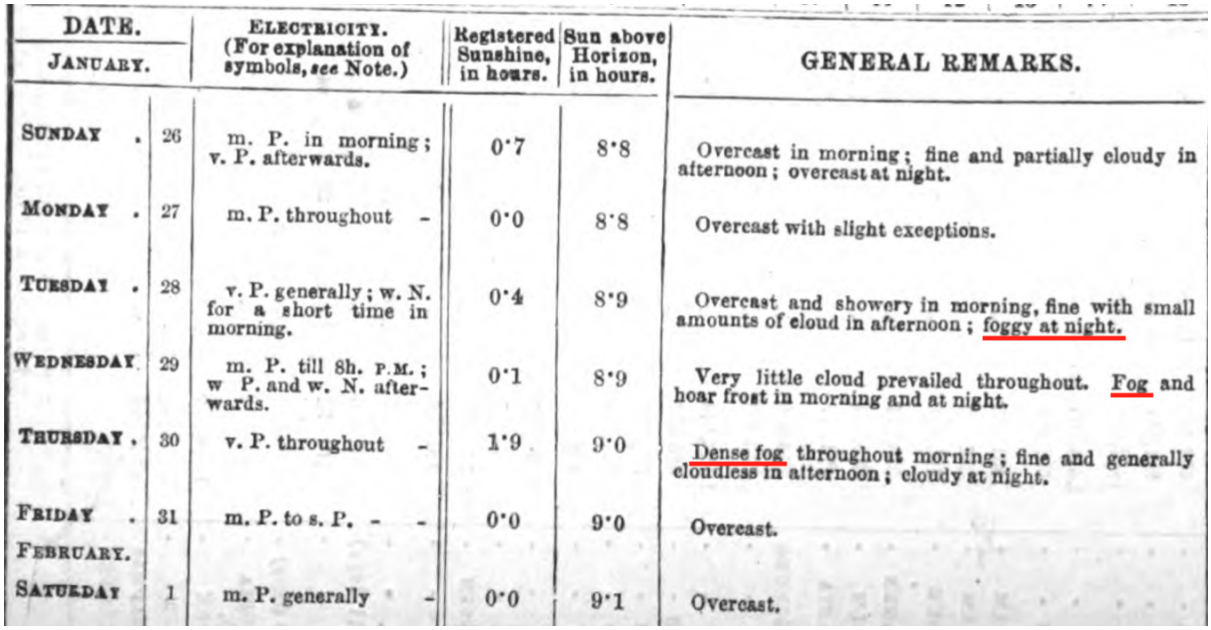
A Data Sources

A.1 Pollution Data



DATE. JANUARY.		ELECTRICITY. (For explanation of symbols, *see* Note.)	Registered Sunshine, in hours.	Sun above Horizon, in hours.	GENERAL REMARKS.
SUNDAY .	26	m. P. in morning; v. P. afterwards.	0·7	8·8	Overcast in morning; fine and partially cloudy in afternoon; overcast at night.
MONDAY .	27	m. P. throughout -	0·0	8·8	Overcast with slight exceptions.
TUESDAY .	28	v. P. generally; w. N. for a short time in morning.	0·4	8·9	Overcast and showery in morning, fine with small amounts of cloud in afternoon; foggy at night.
WEDNESDAY	29	m. P. till 8h. P.M.; w P. and w. N. afterwards.	0·1	8·9	Very little cloud prevailed throughout. Fog and hoar frost in morning and at night.
THURSDAY .	30	v. P. throughout -	1·9	9·0	Dense fog throughout morning; fine and generally cloudless in afternoon; cloudy at night.
FRIDAY .	31	m. P. to s. P. - -	0·0	9·0	Overcast.
FEBRUARY.					
SATURDAY	1	m. P. generally -	0·0	9·1	Overcast.

Figure A.1: Example page from the Weekly Return of Births and Deaths in London showing information about fogs

Source: Weekly Return of Births and Deaths in London ... (1896).

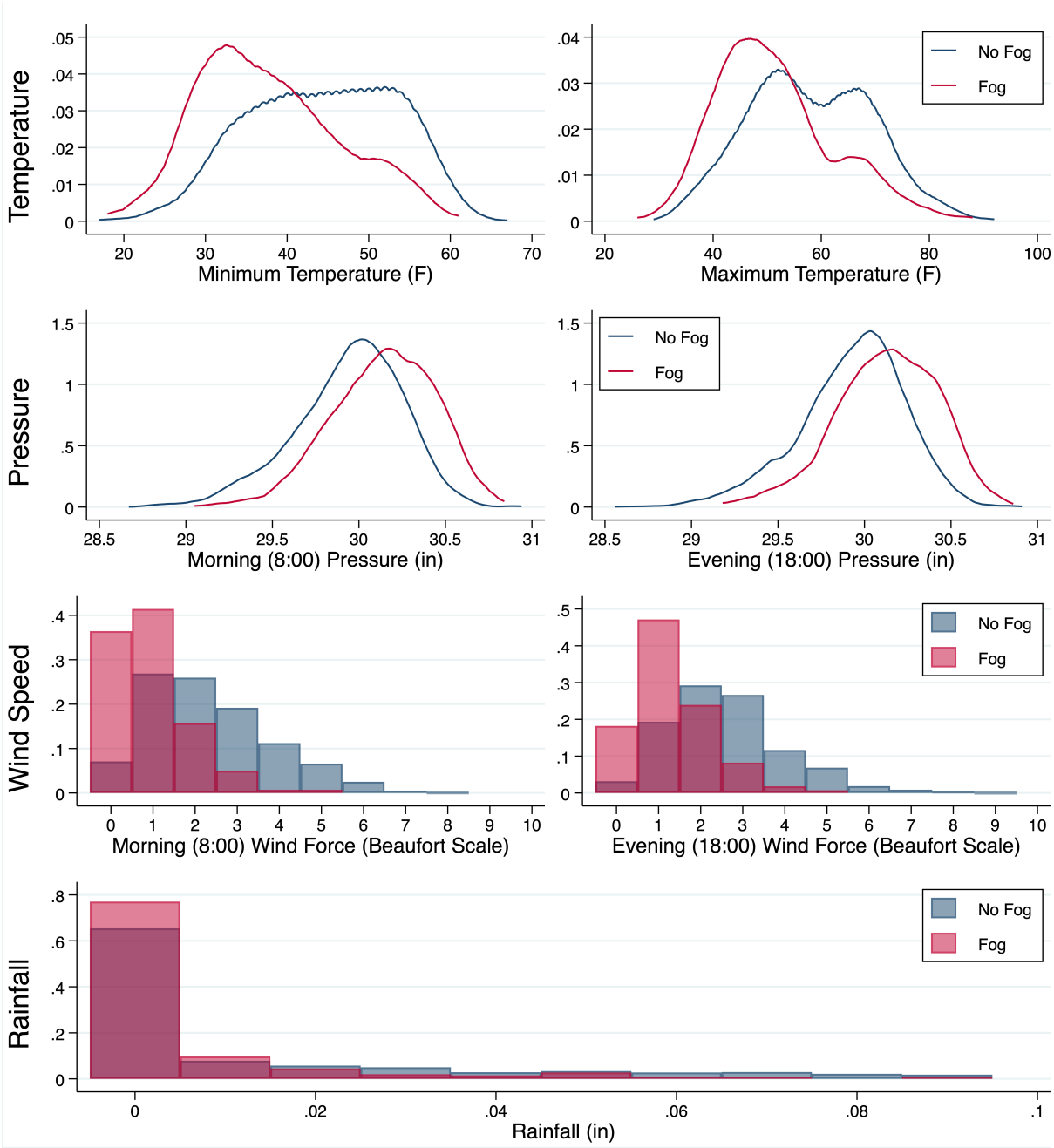


Figure A.2: Weather conditions on days with and without fog in London 1900-10

Notes: The fog days are observed at the Greenwich observatory, but the other weather data is from the Met Office Daily weather reports which were reported at Brixton. Wind speed data are only available from 1900-6.

Sources: Fog data - Weekly Return of Births and Deaths in London ... (1900-10); All weather data except windspeed - Craig and Hawkins (2020); Windspeed data - Meteorological Office (1900).

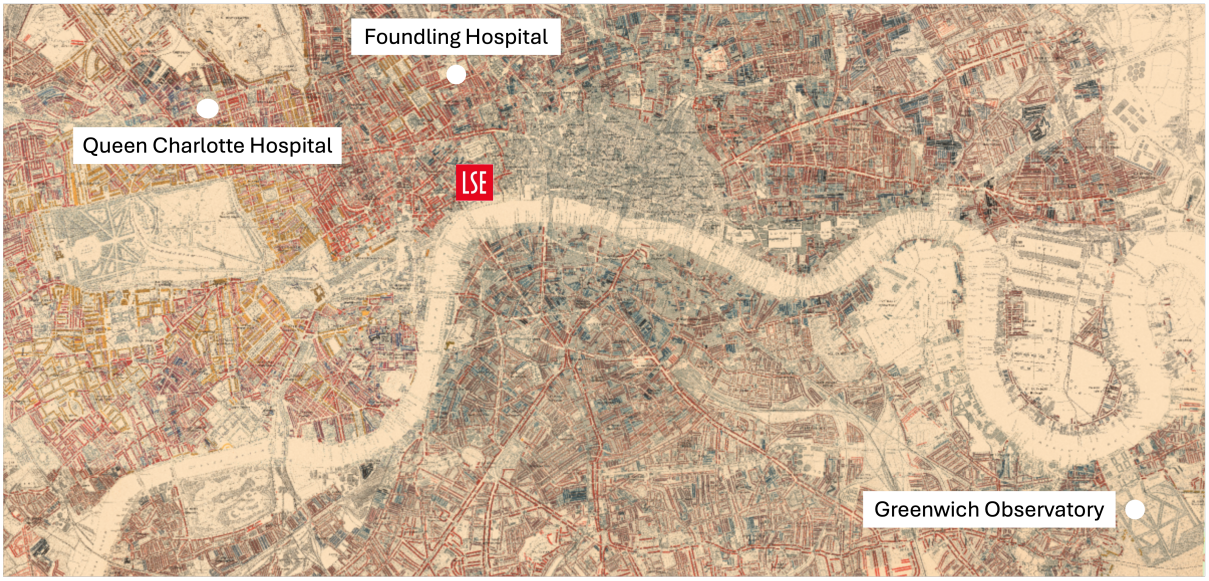


Figure A.3: Map showing locations where data was collected

Source: Booth Poverty Map 1898-99, https://booth.lse.ac.uk/.

A.2 Queen Charlotte Hospital

The Queen Charlotte Hospital Dataset was constructed from the 'Register of in-patients' held in the London Archives, references H27/QC/B/01/008 to H27/QC/B/01/015.

Table A.1 presents descriptive statistics for the dataset. The raw dataset contains 3,543 observations, but we restrict the analysis to singleton births and also exclude some observations where clerical errors in the original source prevent us from identifying the date of delivery precisely. The final sample contains 3,384 observations, though the sample size is smaller for birth weight and length because these variables were not always recorded for stillbirths.

Table A.1: Descriptive statistics for the main health outcomes for the Queen Charlotte Hospital Dataset

	N	Mean	St Dev	Min	Max	Sum
Health Outcomes						
Birth Weight (kg)	3292	3.146	0.556	0.37	5.10	
Low Birth Weight	3292	0.101	0.302	0.00	1.00	334
Birth Length (cm)	3322	52.770	3.771	10.16	76.20	
Premature Birth	3364	0.076	0.265	0.00	1.00	256
Stillbirth	3378	0.043	0.203	0.00	1.00	145
Neonatal Death	3241	0.023	0.148	0.00	1.00	73
Treatment Variables						
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	3384	10.251	8.345	0.00	36.00	
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	3384	10.312	8.294	0.00	36.00	
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	3384	10.014	8.321	0.00	36.00	
$\sum_{s=7}^{9} Fog_{b-s}$	3384	0.332	0.704	0.00	3.00	
$\sum_{s=4}^{6} Fog_{b-s}$	3384	0.347	0.714	0.00	3.00	
$\sum_{s=1}^{3} Fog_{b-s}$	3384	0.349	0.705	0.00	3.00	
Fog_b	3384	0.124	0.330	0.00	1.00	420
Controls						
Mother's Age (years)	3379	25.495	5.844	13.00	47.00	
Single (share)	3381	0.451	0.498	0.00	1.00	1524
Parity	3384	1.126	1.863	0.00	6.00	

Notes: There are 3,384 observations in the data. The sample is restricted to singleton births and excludes a small number of cases where clerical errors in the original source make the date of delivery uncertain. *Sources*: Queen Charlotte Hospital Dataset (2025).

A.3 Foundling Hospital

The Foundling Hospital Cohort Study was reconstructed from records of the Foundling Hospital held in the London Archives (TLA). Table A.2 provides the information included in the records and their respective catalog reference numbers. Note that the medical register, the petitions, the register of applications and some of the weekly infirmary reports are restricted access because they include personal information that is less than 110 years old. Coram, the charity that succeeded the Foundling Hospital, manages access to these records. Because of an earlier agreement that I signed to access the archival sources that were restricted under the 110-year rule, I am not allowed to share the data.

Table A.2: Structure and sources of the Foundling Hospital Dataset

Event/Life Stage	Years	Source (TLA Reference)	Socioeconomic/ Administrative Information	Health Information
Birth	1892-1908	FH Petitions (A/FH/A/8/1/2/102-117)	Mother's approximate address Mother's age Where child was born Father's occupation When mother last saw father What became of father	Child's birthday Child's sex
	1909-1914	Register of applications (A/FH/A/8/5/1)	Mother's approximate address Mother's age Father's occupation	Child's birthday Child's sex
	1892-1908	Registers of In-Patients Queen Charlotte Hospital (H27/QC/B/1/8-13)	Mother's marital status Mother's age	Child's birthday Child's sex Child's parity Birth weight Birth length
LS1: Pre-admission to FH (0-1 year old)	1892-1914	Medical Record (A/FH/A/18/15/1)		Infant feeding practice (breast, milk or food) Duration of breastfeeding
Admission to FH (around 0.37 years old)	1893-1914	Medical Record (A/FH/A/18/15/1)	Admission date Hospital number Admission age	Child's birthday Child's sex Weight Subjective nutritional assesment Vaccinated Diseases present at entry
LS2: Time Fostered in Country (1-6 years old)	1893-1919	Medical Record (A/FH/A/18/15/1)	County child was fostered in	Diseases child was treated for in country
Return from Country to FH (4-6 years old)	1897-1919	Medical Record (A/FH/A/18/15/1)	Re-admission date Re-admission age	Weight Height Subjective nutritional assesment Eye exam Ear exam
LS3: Time Resident in FH (6-17 years old)	1897-1919	Medical Record (A/FH/A/18/15/1)	School standard	Diseases child was treated for in hospital Re-vaccinated
	1897-1915	Weekly Infirmary Reports (A/FH/A/18/5/30-35)		All diseases child was treated for in infirmary Complications from diseases Dates of entry to and exit from the infirmary Duration of each sickness event
Discharge from FH (around 15-17 years old)	1907-1919	Medical Record (A/FH/A/18/15/1)	Discharge date Discharge age Employment after discharge	Weight Height Subjective state of health
Other Life Events				
Restored to Parents (any age)	1892-1919	Medical Record (A/FH/A/18/15/1)	Date of restoration Who child was restored to	
Deaths (any age)	1892-1919	Medical Record (A/FH/A/18/15/1)	Date of death Place of death	Cause of death

Notes: Gray shaded rows reflect information about life stages, i.e. periods of time, and white cells reflect life events, i.e. one point in time.

A.3.1 Descriptive Statistics for Anthropometric Outcomes

Table A.3: Descriptive statistics for the anthropometric health outcomes for the Foundling Hospital Cohort Dataset

	N	Mean	St Dev	Min	Max
Anthro Outcomes					
WAZ Infancy	865	-2.096	1.483	-6.38	1.80
WAZ Age 4–6	727	-1.020	0.875	-4.09	1.29
HAZ Age 4–6	729	-1.599	1.080	-7.80	3.21
BAZ Age 14–16	330	-0.340	0.952	-3.28	2.06
HAZ Age 14–16	330	-1.996	1.105	-6.00	0.55
Treatment Variables					
$\sum_{s=169}^{252} Fog_{b-s}$ (1^{st} Tri)	866	9.887	8.147	0.00	36.00
$\sum_{s=85}^{168} Fog_{b-s}$ (2^{nd} Tri)	866	9.940	8.265	0.00	36.00
$\sum_{s=1}^{84} Fog_{b-s}$ (3^{rd} Tri)	866	10.254	8.670	0.00	36.00
Fog_b	866	0.122	0.328	0.00	1.00
$\sum_{s=1}^{28} Fog_{b+s}$	866	3.477	3.826	0.00	16.00
Controls					
Male	866	0.515	0.500	0.00	1.00
Mother's Age (years)	862	21.459	3.381	13.00	36.00

Notes: WAZ is weight-for-age Z-scores; HAZ is height-for-age Z-scores; BAZ is BMI-for-age Z-scores. All Z-scores are relative to the WHO standard/reference.

Sources: Foundling Hospital Cohort Dataset (2025).

A.3.2 Descriptive Statistics for Individual-Level Morbidity Outcomes

Table A.4: Descriptive statistics for the individual-level morbidity outcomes (ages 5-10) for the Foundling Hospital Cohort Dataset

	N	Mean	St Dev	Min	Max
Incidence					
All Causes	471	1.331	0.860	0.00	11.41
All Respiratory	471	0.466	0.436	0.00	2.76
Upper Respiratory	471	0.388	0.397	0.00	2.32
Lower Respiratory	471	0.075	0.138	0.00	0.74
Catarrh	471	0.210	0.289	0.00	2.25
Tonsillitis (All)	471	0.175	0.232	0.00	1.12
Influenza	471	0.055	0.114	0.00	0.74
Prevalence					
All Causes	471	5.068	6.007	0.00	77.97
All Respiratory	471	1.205	1.567	0.00	12.95
Upper Respiratory	471	0.965	1.376	0.00	12.95
Lower Respiratory	471	0.235	0.583	0.00	7.61
Catarrh	471	0.511	1.104	0.00	12.95
Tonsillitis (All)	471	0.443	0.727	0.00	5.20
Influenza	471	0.141	0.365	0.00	2.42
Treatment Variables					
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	471	10.565	8.239	0.00	36.00
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	471	10.701	8.249	0.00	35.00
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	471	11.452	9.066	0.00	36.00
Fog_b	471	0.134	0.341	0.00	1.00
$\sum_{s=1}^{28} Fog_{b+s}$	471	3.919	3.970	0.00	16.00
Controls					
Male	471	0.503	0.501	0.00	1.00
Mother's Age (years)	471	21.450	3.223	14.00	32.00

Notes: At the individual level, incidence is measured as sickness events per year exposed. Prevalence is measured as the percentage of time exposed sick from that specific cause.

Sources: Foundling Hospital Cohort Dataset (2025).

A.3.3 Descriptive Statistics for Sickness Duration Outcomes

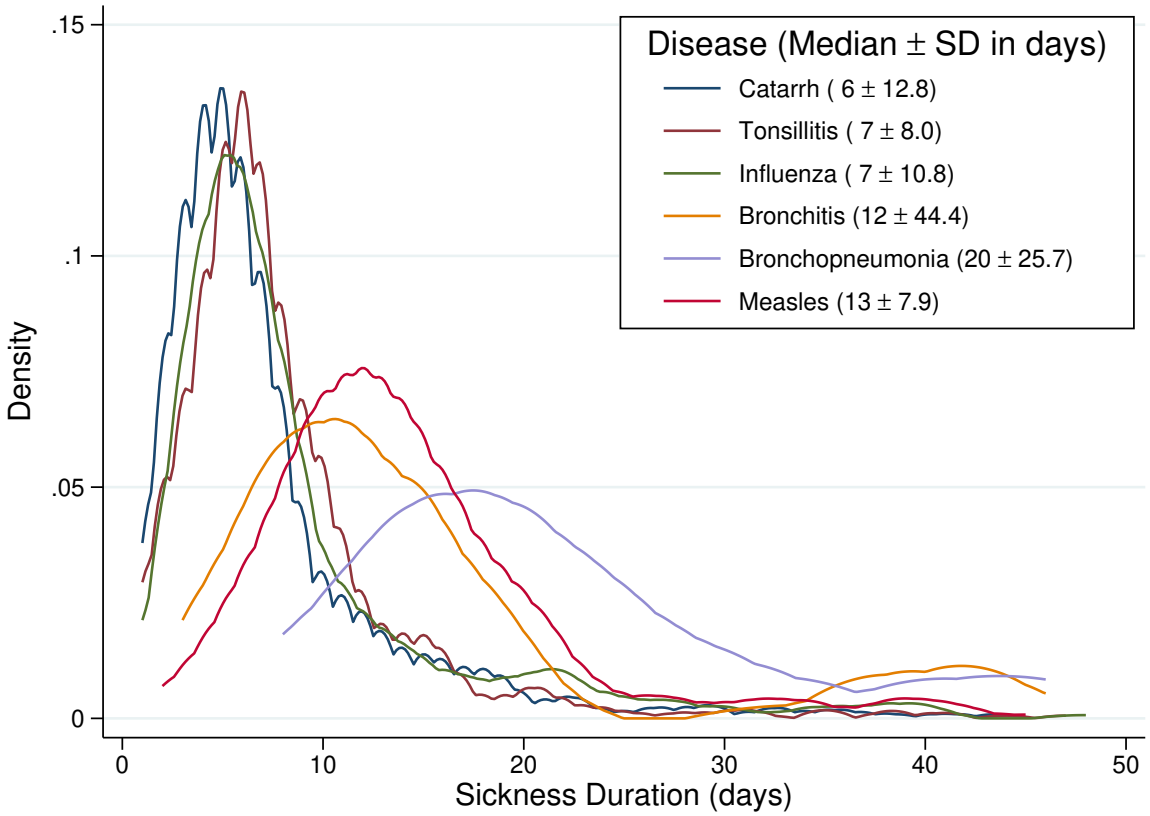


Figure A.4: Sickness duration distributions for respiratory diseases observed in the Foundling Hospital Infirmary, 1897-1915

Sources: Foundling Hospital Cohort Study (2025).

A.3.4 Determining the Children's Location of Birth

Because I am interested in understanding how pollution exposure at birth affected long-run health outcomes, it is important understand what information is available about the mother's location at and around each child's birth in the Foundling Hospital. This information is not provided in the medical record, so I had to link the medical record to two sources: the petitions mothers completed when applying to the hospital (1892-1908) and the register of applications kept by the hospital staff (1909-14). These sources have different strengths and weaknesses. The petitions list both the birth place and the mother's residence when she applied to the hospital. The register of applications, on the other hand, only lists the mother's residential address when she applied, but it provided information on both accepted and rejected applications, allowing me to study selection into the hospital.[13] Birth and/or residence locations were not available for c. 2.5% of the sample, often because the petitions were missing or the locations were illegible.

I use the following rules to decide whether a child was born in London where the birth location is unclear. If a child is resident in London at the time the mother applies to the hospital, I assume that they were born in London. This is a reasonable assumption because where both birth and residence location are present, it was never the case that a child born outside London moved with their mother to London before being admitted to the Foundling Hospital. There are, however, 27 children who are born in London and later move outside London with their mothers during life stage 1, often to live with the mother's family. This means that we cannot be entirely certain that children living outside London in life stage 1 were not born in London where the birth location is missing.[14] Thus, I exclude children who were not born in or were not resident in life stage 1 in London, roughly 20% of the sample, when analysing the effects of pollution exposure at birth on long-term health outcomes. Of course, the fact that a mother gave birth in London does not mean that she was present in London for her entire pregnancy, so there may be

[13]For a subset of 160 children, I can also link them to the Queen Charlotte Hospital and get their mother's residential address before giving birth. However, this was invariably in London because women had to be able to easily reach the hospital.

[14]Where birth and residence place are known, 19% of children are born in London and then move outside in life stage 1.

measurement error in the pollution exposure variables, especially for the first and second trimester of pregnancy.

A.3.5 Foundling Hospital Data Structure

The structure of the Foundling Hospital data and Greenwich Observatory weather data affects how the data can be analysed. Essentially, the Foundling Hospital data is the combination of two datasets: the medical records and infirmary book. The medical records provide longitudinal information for children based on their admission in infancy, their readmission at ages 4-6 and their discharge at ages 15-16. The medical record begins recording information for admission cohorts in January 1893 and the records stopped being updated at readmission and discharge in April/May 1919, likely when a new medical officer took post and stopped systematically recording the data. The infirmary books contain period information on children's sickness events during their time living at the Foundling Hospital main site in central London from approximate ages 5 to 15. These records begin in 1759 and continue until 1923, but I collected the weekly information from 27 March 1897 to 5 October 1915. The start of this interval corresponds with the first children in the medical records returning the Foundling Hospital main site, and the end date is determined by a missing infirmary book: there is no data available from October 1915 to June 1919. The infirmary book only lists a child's name, age, admission and discharge dates from the infirmary and the cause of infirmity. Thus, we can only study the effects of pollution exposure at birth on long-run health outcomes by linking the infirmary books to the medical record because we need information about the child's birth date and where they were living at and after birth.

The Greenwich Observatory weather data also presents some minor restrictions on the data as well. While the data was kept religiously across the second half of the nineteenth century, the weather data was not kept during the First World War. Thus, there are no weather or fog observations after 24 October 1914.

Figure A.5 shows how the data structure and constraints affect the sample of influenza cases that can be analysed. For instance, when analysing the effect of pollution exposure

at birth on later sickness duration, we must exclude some cases of flu that occur to children who are too old to appear in the medical record (Figure A.5A). We do not have precise birthdates for these children,[15] nor do we know whether or not they were born in London. While it would be fairly straightforward to find birthdates for these children in the archive, figuring out whether they were born in London is far more time consuming and beyond the scope of the analysis here. Thus, these influenza cases are excluded leaving 223 influenza cases within the bolded line polygon that are in our analytical sample. However, changing the research question affects the data that can be analysed. When studying the short-term effects of pollution exposure on children, we no longer need information on birthdates or where the children were born. This means that nearly all influenza cases can be included in the analytical sample, barring cases that occur after the weather data is no longer available (Figure A.5B). This leaves 398 cases in the analytical sample. Finally, in Figure A.5C, we want to test both the long-run effects and short-run effects at the same time, which limits the sample to children with birthdates, children with birth locations in London and sickness events occurring before the end of the weather data, leaving only 211 cases in the analytical sample. Thus, it is clear why the sample size changes across the various specifications in the paper.

Figures A.5D and E similarly show what cases of flu are counted when analysing incidence and prevalence of flu at the individual level. Because we want to measure incidence and prevalence for consistent age groups, there is a trade-off in capturing a wide age range and increasing the number of individuals that can be observed for the full age range. This is why the group from age *c.* 5 to 10 is reported in the paper, but the results for the age group from *c.* 5 to 15 are qualitatively similar.

[15] They are placed on the lexis diagram for illustrative purposes only using a predicted birthdate based on their ages at admission to the infirmary and some random noise.

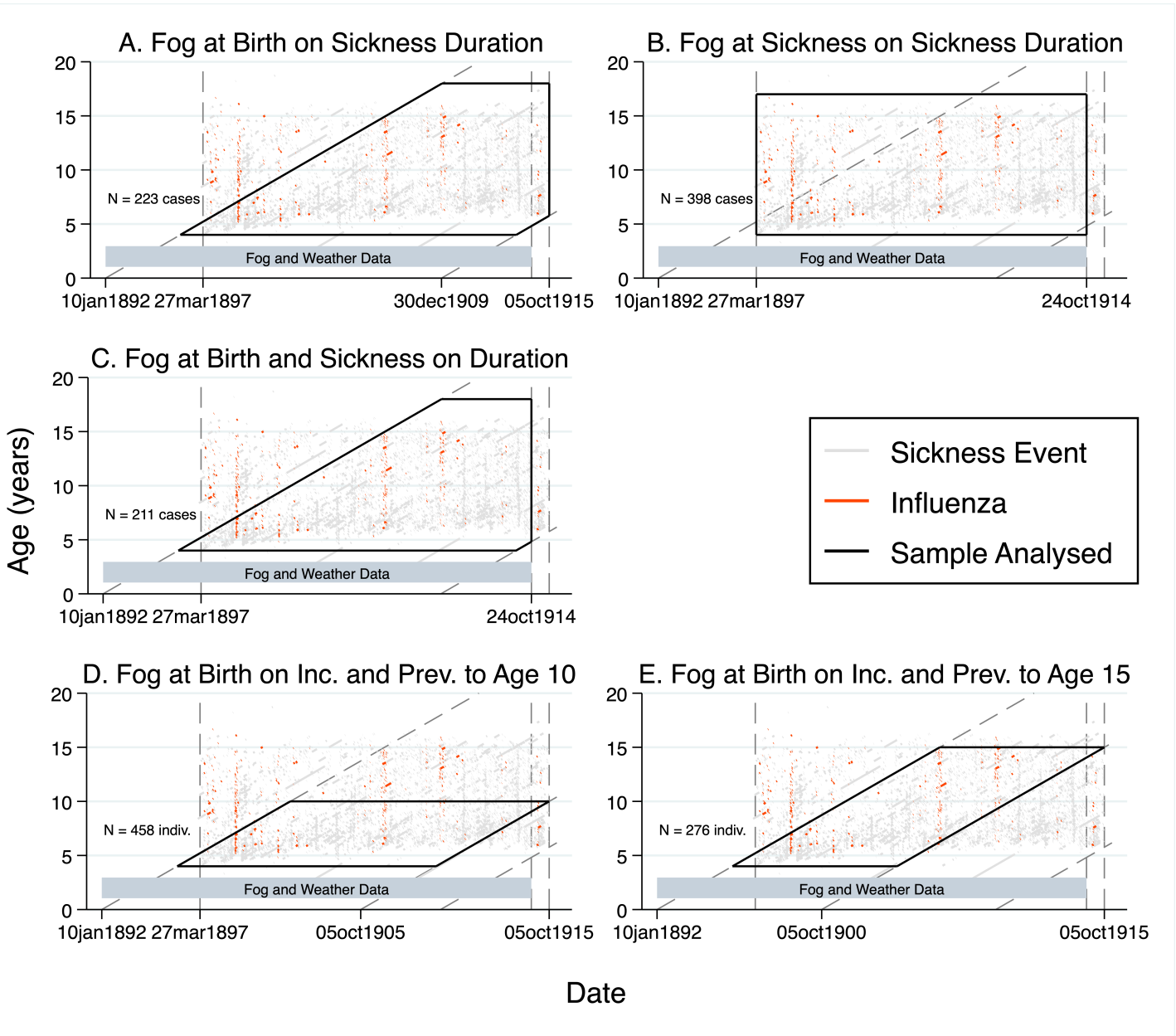


Figure A.5: Different sets of sickness events included in the sample depending on empirical strategy

Notes: Each graph reflects a different empirical strategy in the paper. The analytical sample includes influenza cases within the bolded black lines. Analysis of fog at birth is also restricted to children experiencing influenza who were born in London (see Appendix A.3.4). Inc. is incidence and Prev. is prevalence.

Sources: Foundling Hospital Cohort Dataset (2025).

B More Detail on Empirical Strategy

Figure B.1 explains how exposure to fog in relation to the birth day or date of admission to the infirmary would affect birth outcomes and sickness duration in the short run and other outcomes in the long run.

Starting with short-run effects on birth outcomes (Figure B.1A), if a child is born the day before a fog event, we would not expect pollution to affect their birth outcomes. Therefore, I do not include leads of fog exposure when analysing birth outcomes. If a child were born on a fog day, then the pollution may have directly affected their birth outcomes either by triggering early labour or exacerbating the difficulties of labour for the mother. If a child were born after fog days, this could affect their birth outcomes by influencing the health of the mother. Therefore, I test for fog exposure in each trimester to check whether this affected birth outcomes.

When considering the short-run effect of pollution on sickness duration (Figure B.1C), again looking a leads of fog exposure with respect to a child's admission date is not possible. In this case, the number of fog events a child is exposed to while in the infirmary would be positively correlated with the severity of their illness irrespective of their pollution exposure, leading to reverse causality in the relationship. However, we can still explore exposure to fogs on the day of admission and in the period preceding admission to see whether pollution exposure in these periods affected sickness duration. Looking at lagged exposure is important because we can assume that children were only admitted to the infirmary when clinical symptoms appeared for their disease. The incubation period between infection and the appearance of symptoms varies by disease: 1-2 days for influenza (Treanor 2014); 4-6 days for other respiratory diseases such as rhinoviruses and parainfluenza viruses that cause upper and lower respiratory tract infections respectively (Englund and Moscona 2014; Mackay and Arden 2014); and 10 days for measles (Moss and Griffin 2014). Therefore, differing exposure windows may be necessary to capture the effects of pollution on sickness duration from different diseases.

Exploring pollution exposure windows around birth for long-run health outcomes is also important (Figure B.1B). In this, case it is possible to study cumulative pollution

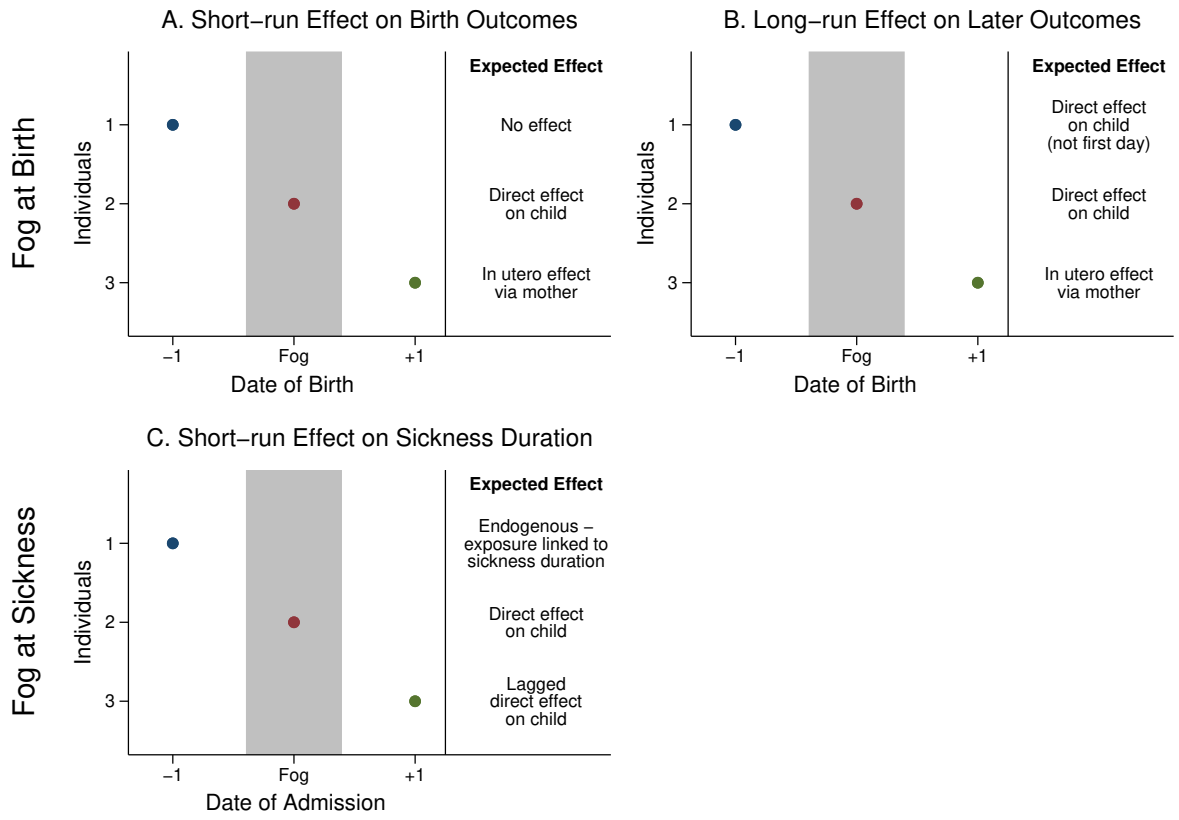


Figure B.1: Timing of pollution exposure (fogs) and the interpretation of effects on short- and long-run outcomes

Notes:

exposure after birth (leads of fog exposure) because pollution exposure in the first month of life could have long-run consequences for health, and all long-run health outcomes were measured after the first month. We can also test whether experiencing a fog event on one's birthday has negative effects for health. Finally, we can study the pollution exposure of children *in utero* by studying whether lagged pollution exposure relative to birth affected children's health status.

C Identification and Selection Issues

C.1 Sources of Collider Bias in Pollution Research

This paper measures the short and long-run health effects of exposure to acute extreme pollution events, proxied by fog events. Focussing on acute pollution events helps to address the main endogeneity concerns in analysing the health effects of pollution. The first concern is that pollution is an observable disamenity that individuals consider when making residence location choices (Currie et al. 2014). Because residence location choices are based on a host of factors including income, education, ability to commute, etc., spatial variation in atmospheric pollution is rarely randomly assigned. This sorting is a form of pretreatment collider bias (see Figure C.1) and was important in historical cities, shaping the socioeconomic character of neighbourhoods over long periods of time (Banzhaf et al. 2024; Heblich et al. 2021; Schneider 2020). Using short-run spikes in pollution as the treatment mitigates bias from sorting because these temporary shocks affected all areas in a city and would not induce cross-neighbourhood migration. In any case, the locations of the Queen Charlotte Hospital and Foundling Hospital were fixed long before the period analysed here so that sorting responses could not have affected the children exposed in these institutions: the Foundling Hospital opened its Bloomsbury site in 1745 (Pugh 2007, p. 39) and the Queen Charlotte Hospital on Marylebone Road opened in 1856 (Select Committee of the House of Lords on Metropolitan Hospitals 1891, p. 519).

A second potential source of endogeneity is related to avoidance behaviour: i.e. individual behaviours to reduce exposure to exogenous ambient pollution levels (Currie et al. 2014). Today, this could mean staying indoors on high pollution days or using air purifiers. Avoidance behaviours are mediators between short-run pollution spikes and health outcomes. This means that without controlling for these behaviours the effect estimated is the biological effect of pollution on health net of any avoidance behaviours. In actuality, this is what we are curious to estimate anyway since we want to understand the health effects of pollution in a particular context. Note that avoidance behaviours as mediators

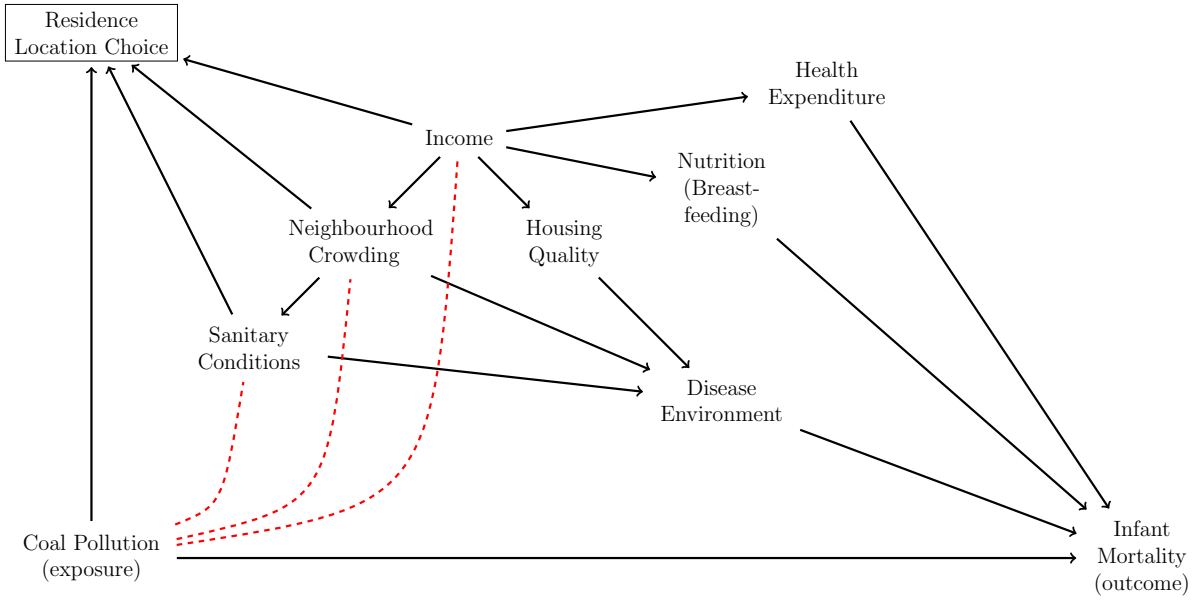


Figure C.1: DAG showing collider bias arising from endogenous residence location choices when considering the long-run health effects spatial variation in pollution exposure

Source: Schneider (2020).

are bad controls and controlling for them would induce collider bias if we assume that the rich and educated are more likely to practice avoidance behaviours (see Figure C.1) (Angrist and Pischke 2009, pp. 64-66). It is not clear to what extent avoidance behaviours were possible or followed in the past. On the foggiest days in London in the 1890s, people may have stayed indoors because poor visibility made it difficult to travel (Luckin 2003, p. 35). However, the emphasis on ventilation in the past, lack of insulation, poorly sealed windows and indoor coal fires may have limited the benefits of staying indoors when ambient pollution was bad. The focus on people in institutions in this paper also limits the heterogeneity in avoidance behaviours across individuals because the institutions were unlikely to have different avoidance policies for different groups of people within the institution.

Finally, Figure C.3 shows the classic example of collider bias, survival bias, which is discussed at length in the text and in Schneider (2020). Acute pollution events affect whether an individual survives to adulthood as does their latent health. Because we can only observe health outcomes for those who survive, we are implicitly conditioning on the collider variable Survive to Measurement. Simply put, if children born on fog days are more likely to die and those with the weakest latent health are more likely

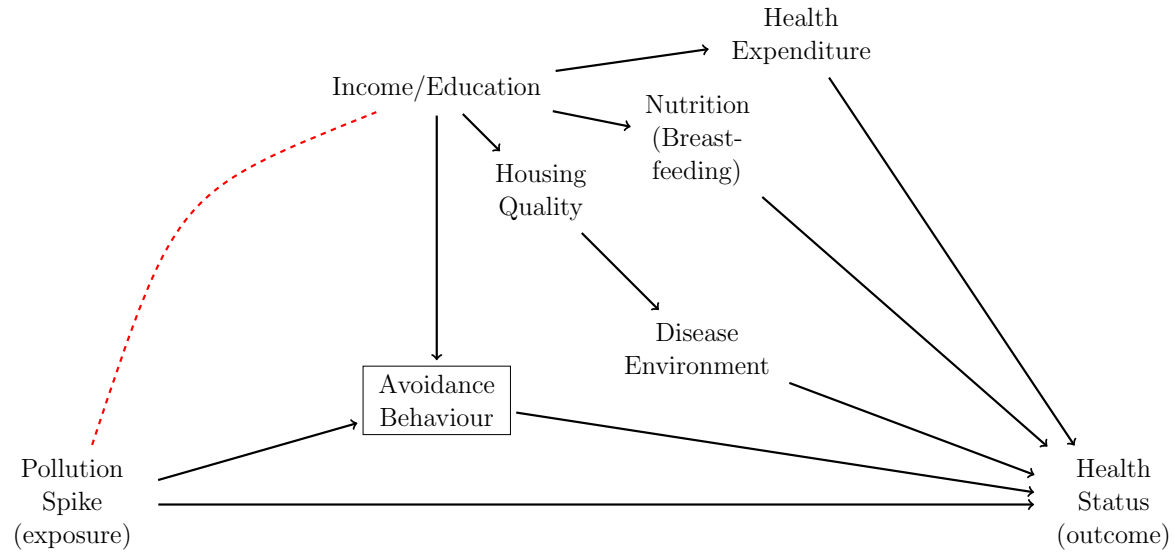


Figure C.2: DAG showing collider bias arising from controlling for avoidance behaviour as a mediator when considering the long-run health effects of short-run extreme pollution exposure

to die, the the average latent health of survivors born on fog days will be higher than the average latent health of children who were not born on fog days and do not suffer from this additional mortality risk. However, there does not appear to be much survival bias in this setting because acute pollution exposure did not affect stillbirths or neonatal deaths. While pollution exposure did affect subsequent mortality, it is not clear whether this mortality was high enough, or sufficiently selective with respect to latent health to produce survival bias. For instance, when comparing birth weights and lengths for a subsample of 160 children admitted to the Foundling Hospital who were born in the Queen Charlotte Hospital, there are no statistically significant differences in birth weight and length between those born on a fog day or not. Thus, survival bias does not appear to be a major problem in this setting.

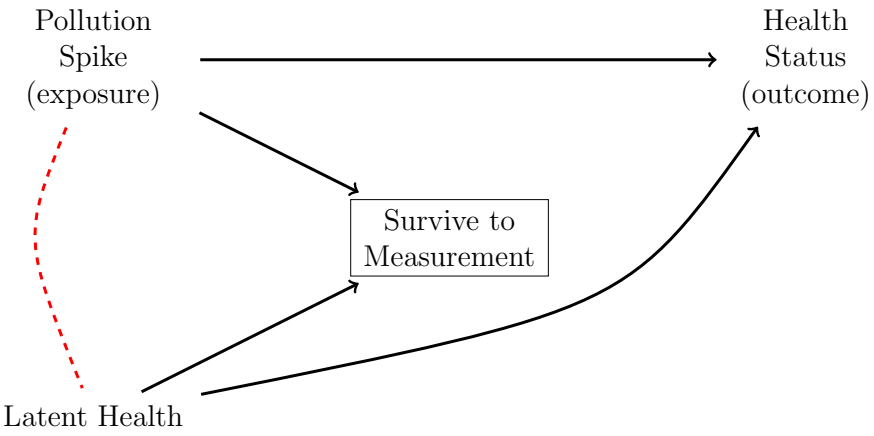


Figure C.3: DAG showing survival bias as collider bias when considering the long-run health effects of short-run extreme pollution exposure

C.2 Selection into the Queen Charlotte Hospital

There are not strong signs that selection into the Queen Charlotte Hospital varied based on whether a woman delivered her child on a fog day or not. There are slight differences in the timing of admission on fog and non-fog days (Figure C.4), but this does not translate to any meaningful differences in characteristics or care. Table C.1 shows that maternal characteristics are nearly identical between mothers giving birth on a fog day or not and that the time between admission and delivery of the child is also not meaningfully different. Maternal age and parity are not normally distributed, but their distributions are nearly identical between the two groups (Figure C.5).

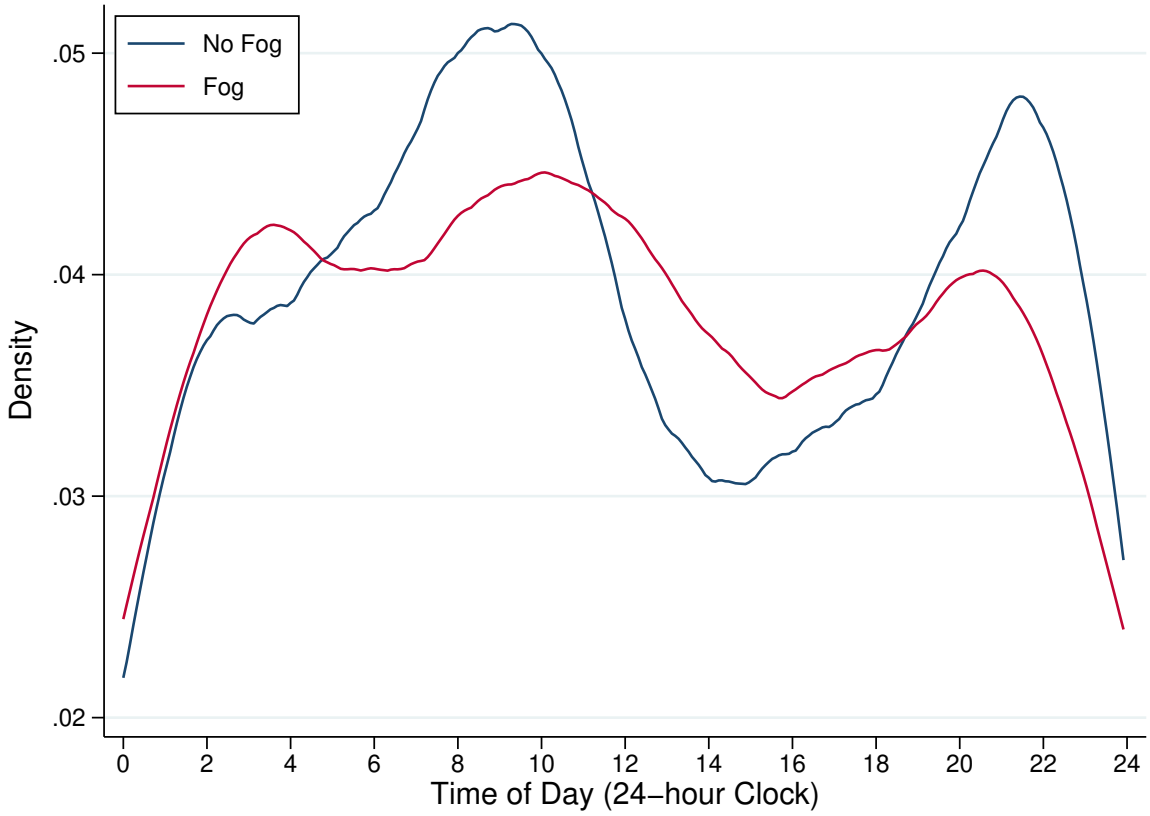


Figure C.4: Admission times of mothers delivering their children in the Queen Charlotte Hospital on days with and without fog in London 1892-1908

Sources: Queen Charlotte Hospital Dataset (2025).

Table C.1: Selection into the Queen Charlotte Hospital comparing children born on a fog day or not

	Central Tendency	$Fog_b = 0$	$Fog_b = 1$	Statistical Test	Difference/ Coefficient	p-value
Maternal Age (Years)	Median	24 (2961)	24 (418)	OLS	0.28 [0.31]	0.36
Single (Share)	Mean	0.46 (2962)	0.42 (419)	T-test	0.04	0.18
Parity	Median	0 (2964)	0 (420)	Poisson	-0.02 [0.05]	0.63
Admission to Delivery Time Gap (Hours)	Median	5.64 (2887)	6.04 (415)	OLS	0.56 [0.76]	0.46

Notes: Fog_b is an indicator variable equal to one if a child is born on a fog day and zero otherwise. Sample size in parentheses and standard errors in square brackets. For the admission to delivery time gap, women whose admission time was after their delivery time were excluded as probable clerical errors. I also exclude women who gave birth more than 96 hours after admission as these are likely to be problematic births that were transferred from the outpatient department.

Sources: Queen Charlotte Hospital Dataset (2025).

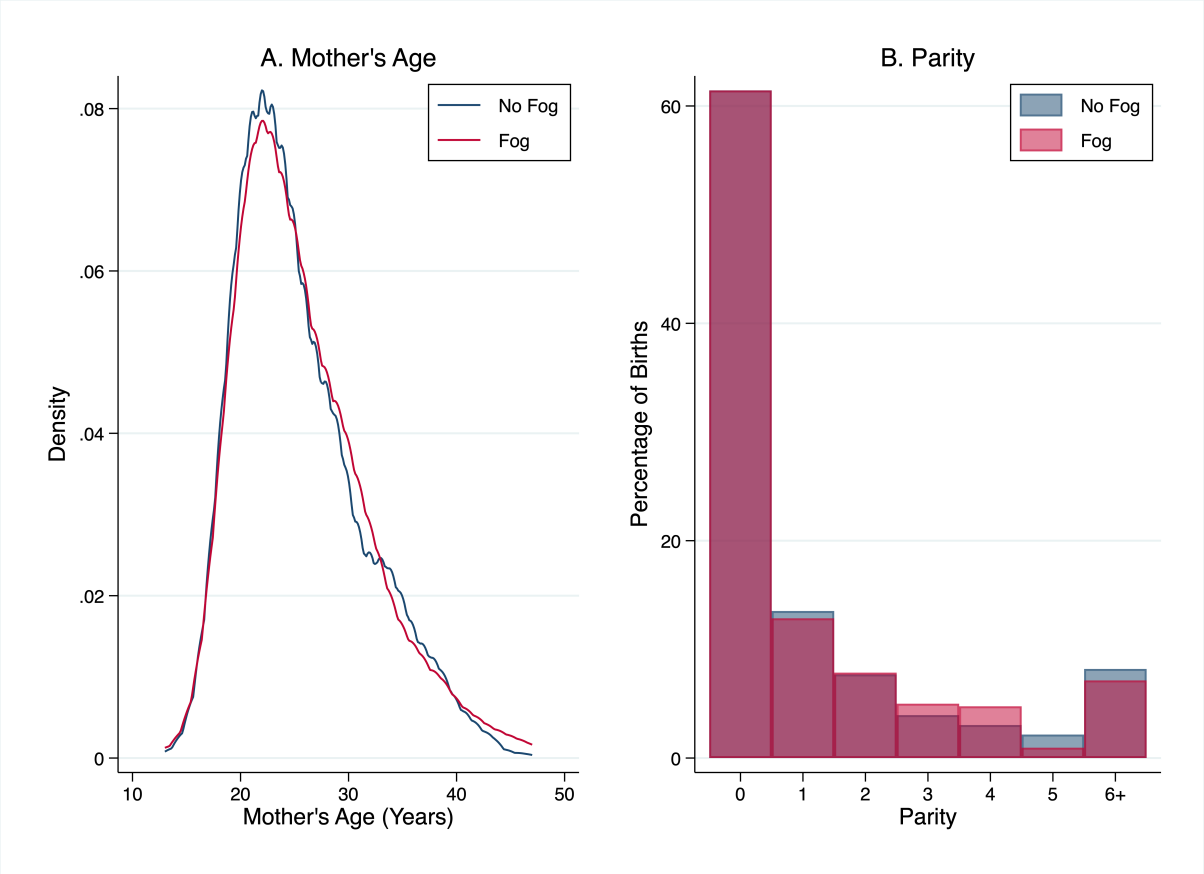


Figure C.5: Maternal age and parity distributions of mothers delivering their children in the Queen Charlotte Hospital on days with and without fog in London 1892-1908

Sources: Queen Charlotte Hospital Dataset (2025).

C.3 Selection into the Foundling Hospital

There are also not signs that selection into the Foundling Hospital either on paternal or child characteristics or through the hospital's selection procedure varied for children born on fog days or not. Table C.2 shows that the share of children with high status fathers was similar between the treatment and control. Children born on fog days had slightly older mothers, but looking at the distribution of maternal age presented in Figure C.6, these differences are unlikely to have affected health outcomes. There are limited signs of survival bias since the birth weights and lengths of children born in the Queen Charlotte Hospital and later admitted to the Foundling Hospital are similar. Likewise, there is no sign that maternal investment was different between the two groups: the breastfed share and breastfeeding duration were similar and the date on which the mother applied to give up the child to the Foundling Hospital was also similar. Finally, there were no differences in treatment and control on selection into the Foundling Hospital with the admission age and acceptance rate similar for children born on fog days or not. Thus, there is no indication that anything other than acute pollution exposure can explain the health differences between individuals born on fog days or not.

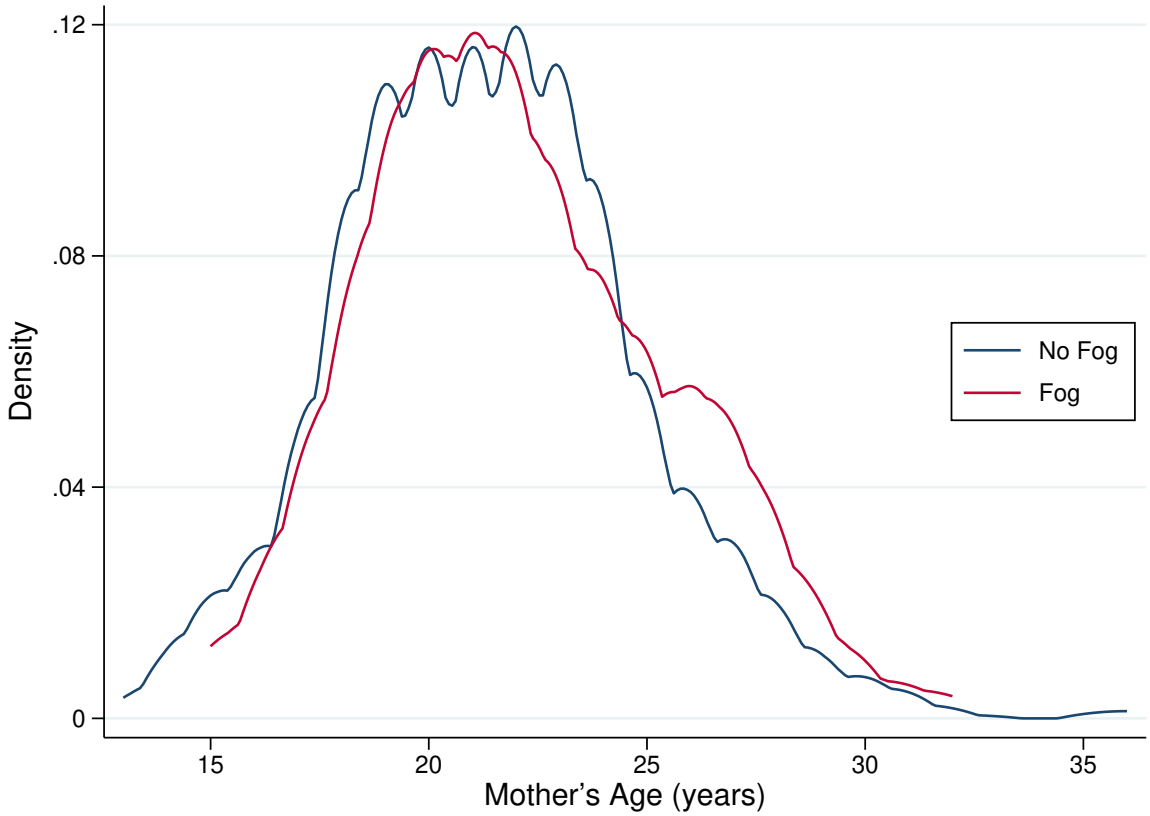


Figure C.6: Maternal age distributions of children admitted to the Foundling Hospital born on days with and without fog in London 1892-1908

Sources: Foundling Hospital Cohort Study (2025).

Table C.2: Selection into the Foundling Hospital comparing children born on a fog day or not

	Central Tendency	$Fog_b = 0$	$Fog_b = 1$	Statistical Test	Difference/ Coefficient	p-value
Parental Characteristics						
Maternal Age (Years)	Mean	21.41	22.03	OLS	0.62	0.08
		(756)	(106)		[0.35]	
Father High Class (Share)	Mean	0.50	0.48	T-test	-0.02	0.70
		(760)	(106)			
Child Characteristics						
Birth Weight (kg)†	Mean	3.20	3.24	T-test	0.04	0.65
		(136)	(24)			
Birth Length (cm)†	Mean	53.06	53.87	T-test	0.81	0.15
		(136)	(24)			
Maternal Investment						
Ever Breastfed (Share)	Mean	0.59	0.63	T-test	0.04	0.44
		(756)	(106)			
Breastfeeding Dur. (Days)	Median	60	64	OLS	10.61	0.31
		(263)	(35)		[10.34]	
Application Age (Days)‡	Median	58	52.5	OLS	6.49	0.72
		(370)	(36)		[17.91]	
FH Selection						
Admission Age (Days)	Median	91	87.5	OLS	4.28	0.68
		(760)	(106)		[10.26]	
Acceptance Rate (Share)‡	Mean	0.48	0.53	T-test	0.04	0.61
		(370)	(36)			

Notes: Fog_b is an indicator variable equal to one if a child is born on a fog day and zero otherwise. Means with sample size in parentheses and standard errors in square brackets. P-values are from a two-tailed T-test with equal variances or OLS. † denotes data for 1892-1908, and ‡ denotes data for 1909-14. The sample is slightly different across indicators because information about rejected applications is only available from 1909 to 1914 and information on birth outcomes for foundling hospital children born in the Queen Charlotte Hospital is only available from 1892 to 1908.

Sources: Foundling Hospital Cohort Study (2025).

D Additional Results

D.1 Birth Outcome Results

Table D.1: Effects of pollution exposure at and shortly before birth on birth weight in the Queen Charlotte Hospital, 1892-1913

	(1)	(2)	(3)	(4)
	Birth Weight	Birth Weight	Birth Weight	Birth Weight
$\sum_{s=7}^{9} Fog_{b-s}$			0.003	0.000
			(0.015)	(0.014)
$\sum_{s=4}^{6} Fog_{b-s}$			0.014	0.020
			(0.014)	(0.013)
$\sum_{s=1}^{3} Fog_{b-s}$			0.021	0.026*
			(0.016)	(0.015)
Fog_b (Birthday)	0.033	0.027	0.024	0.016
	(0.031)	(0.030)	(0.031)	(0.030)
Individual Controls	Yes	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
Stillbirths Included	Yes	No	Yes	No
$\sum Fog_b$	407	395	407	395
N	3278	3164	3278	3164
r2	0.04	0.05	0.04	0.05

Notes: Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

Table D.2: Effects of pollution exposure at and shortly before birth on birth length in the Queen Charlotte Hospital, 1892-1913

	(1)	(2)	(3)	(4)
	Birth Length	Birth Length	Birth Length	Birth Length
$\sum_{s=7}^{9} Fog_{b-s}$			0.101	0.060
			(0.093)	(0.087)
$\sum_{s=4}^{6} Fog_{b-s}$			-0.005	0.025
			(0.098)	(0.092)
$\sum_{s=1}^{3} Fog_{b-s}$			0.215**	0.296***
			(0.108)	(0.096)
Fog_b (Birthday)	0.399*	0.400**	0.315	0.285
	(0.212)	(0.189)	(0.212)	(0.191)
Individual Controls	Yes	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
Stillbirths Included	Yes	No	Yes	No
$\sum Fog_b$	409	399	409	399
N	3308	3204	3308	3204
r2	0.08	0.09	0.08	0.09

Notes: Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

Table D.3: Effects of pollution exposure at and shortly before birth on stillbirths and neonatal deaths (first two weeks) in the Queen Charlotte Hospital, 1892-1913

	(1)	(2)	(3)	(4)
	Still-birth	Still-birth	Neonatal Death	Neonatal Death
$\sum_{s=7}^{9} Fog_{b-s}$		0.122		0.275
		(0.125)		(0.181)
$\sum_{s=4}^{6} Fog_{b-s}$		-0.026		-0.132
		(0.142)		(0.208)
$\sum_{s=1}^{3} Fog_{b-s}$		-0.105		0.056
		(0.154)		(0.207)
Fog_b (Birthday)	-0.108	-0.088	0.167	0.135
	(0.300)	(0.305)	(0.363)	(0.352)
Individual Controls	Yes	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes	Yes
Birth Year Trend	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
$\sum Fog_b$	421	421	411	411
$\sum$ Stillbirth or Neonatal Death	146	146	66	66
$\sum Fog_b \times$ (SB or ND)	15	15	9	9
N	3450	3450	3322	3322

Notes: Estimated with logistic regression. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. A birth year trend rather than birth year fixed effects is used to avoid loss of observations from variables that perfectly predict success or failure. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

Table D.4: Effects of pollution exposure at or shortly before birth on premature birth in the Queen Charlotte Hospital, 1892-1913

	(1)	(2)
	Premature Birth	Premature Birth
$\sum_{s=7}^{9} Fog_{b-s}$		-0.057
		(0.112)
$\sum_{s=4}^{6} Fog_{b-s}$		-0.257**
		(0.129)
$\sum_{s=1}^{3} Fog_{b-s}$		-0.085
		(0.135)
Fog_b (Birthday)	-0.065	-0.013
	(0.237)	(0.247)
Individual Controls	Yes	Yes
Weather Controls	Yes	Yes
Birth Year Trend	Yes	Yes
Birth Quarter FE	Yes	Yes
$\sum Fog_b$	426	426
$\sum$ Premature	262	262
$\sum Fog_b \times$ (Premature)	26	26
N	3455	3455

Notes: Estimated with logistic regression. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. A birth year trend rather than birth year fixed effects is used to avoid loss of observations from variables that perfectly predict success or failure. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

Table D.5: Effects of pollution exposure *in utero* on birth weight and length in the Queen Charlotte Hospital, 1892-1913

	(1)	(2)	(3)	(4)
	Birth Weight	Birth Weight	Birth Length	Birth Length
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	-0.000	-0.001	0.002	-0.004
	(0.002)	(0.002)	(0.014)	(0.013)
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	-0.001	-0.001	-0.034***	-0.034***
	(0.002)	(0.002)	(0.013)	(0.013)
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	0.002	0.002	0.003	0.006
	(0.002)	(0.002)	(0.014)	(0.012)
Sex Dummy	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
Stillbirths Included	Yes	No	Yes	No
N	3278	3164	3308	3204
r2	0.04	0.05	0.08	0.09

Notes: Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

Table D.6: Effects of pollution exposure *in utero* on stillbirths and neonatal deaths (first two weeks) in the Queen Charlotte Hospital, 1892-1913

	(1)	(2)
	Still-birth	Neonatal Death
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	-0.003	-0.006
	(0.016)	(0.024)
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	0.010	0.017
	(0.014)	(0.021)
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	0.021	0.019
	(0.015)	(0.021)
Sex Dummy	Yes	Yes
Individual Controls	Yes	Yes
Birth Weather	Yes	Yes
Birth Year Trend	Yes	Yes
Birth Quarter FE	Yes	Yes
$\sum$ Stillbirth or Neonatal Death	146	66
N	3450	3322

Notes: Estimated with logistic regression. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. A birth year trend rather than birth year fixed effects is used to avoid loss of observations from variables that perfectly predict success or failure. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

Table D.7: Effects of pollution exposure *in utero* on premature birth in the Queen Charlotte Hospital, 1892-1913

	(1)
	Premature Birth
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	-0.000
	(0.012)
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	0.014
	(0.011)
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	-0.010
	(0.013)
Sex Dummy	Yes
Individual Controls	Yes
Birth Weather	Yes
Birth Year Trend	Yes
Birth Quarter FE	Yes
$\sum$ Premature	262
N	3455

Notes: Estimated with logistic regression. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. A birth year trend rather than birth year fixed effects is used to avoid loss of observations from variables that perfectly predict success or failure. Individual controls include a sex dummy, mother's age and its square, parity dummies and mother's civil state dummies.

Sources: Queen Charlotte Hospital Dataset (2025).

D.2 Anthropometric Results

Table D.8: Effects of pollution exposure at birth on child growth outcomes in the Foundling Hospital, 1893-1919

	(1)	(2)	(3)	(4)	(5)
	WAZ Infancy	WAZ Age 4-6	HAZ Age 4-6	BAZ Age 14-16	HAZ Age 14-16
Fog_b (Birthday)	0.209	0.097	0.192	0.065	0.191
	(0.157)	(0.096)	(0.123)	(0.131)	(0.164)
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
$\sum Fog_b$	106	90	89	44	44
N	861	723	725	326	326

Notes: WAZ is weight-for-age Z-score; HAZ is height-for-age Z-score; and BAZ is BMI-for-age Z-score all relative to the WHO child growth standard/reference. The sample is restricted to children born in London. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include a sex dummy, mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.9: Effects of pollution exposure *in utero* on child growth outcomes in the Foundling Hospital, 1893-1919

	(1)	(2)	(3)	(4)	(5)
	WAZ Infancy	WAZ Age 4-6	HAZ Age 4-6	BAZ Age 14-16	HAZ Age 14-16
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	0.007	0.016*	0.012	0.014	0.002
	(0.012)	(0.008)	(0.009)	(0.010)	(0.015)
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	0.008	0.011*	-0.001	0.011	0.003
	(0.010)	(0.007)	(0.008)	(0.010)	(0.013)
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	0.016	0.005	0.003	0.005	-0.003
	(0.010)	(0.007)	(0.008)	(0.009)	(0.012)
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
$\sum Fog_b$	106	90	89	44	44
N	861	723	725	326	326

Notes: The sample is restricted to children born in London. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include a sex dummy, mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.10: Effects of pollution exposure after birth on child growth outcomes in the Foundling Hospital, 1892-1919

	(1)	(2)	(3)	(4)	(5)
	WAZ Infancy	WAZ Age 4-6	HAZ Age 4-6	BAZ Age 14-16	HAZ Age 14-16
$\sum_{s=1}^{28} Fog_{b+s}$	0.019	-0.002	0.006	0.020	0.014
	(0.017)	(0.011)	(0.014)	(0.015)	(0.016)
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
$\sum Fog_b$	102	87	86	43	43
N	830	698	700	312	312

Notes: The sample is restricted to children born and living in London with their mothers before admission to the Foundling Hospital. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include a sex dummy, mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

D.3 Mortality Results

Table D.11: Effect of pollution exposure *in utero* on mortality risk

	(1) Respiratory	(2) Respiratory
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	1.025	1.021
	(0.026)	(0.026)
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	1.006	1.006
	(0.033)	(0.033)
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	1.023	1.021
	(0.031)	(0.032)
Fog_b (Birthday)		2.168
		(1.032)
Sex Dummy	Yes	Yes
Individual Controls	Yes	Yes
Birth Weather	Yes	Yes
Birth Quarter FE	Yes	Yes
Birth Decade FE	Yes	Yes
$\sum Fog_b$		106
N	860	860

Notes: The sample is restricted to children born in London. Hazard ratios with standard errors in parentheses. The regressions are estimated using a competing risk model where respiratory deaths are the outcome of interest and other causes of death are the competing risk. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

D.4 Individual-level Morbidity Results

Table D.12: Effect of pollution exposure *in utero* on individual-level sickness incidence

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Upper Resp	(5) Lower Resp
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	0.000	0.000	-0.001	0.000	-0.002
	(0.003)	(0.002)	(0.001)	(0.004)	(0.001)
	[0.004]	[0.012]	[-0.050]	[0.010]	[-0.111]
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	0.001	0.001	-0.000	0.002	0.001
	(0.003)	(0.002)	(0.001)	(0.003)	(0.001)
	[0.024]	[0.044]	[-0.002]	[0.037]	[0.043]
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	-0.001	-0.002	-0.001	-0.003	-0.002
	(0.002)	(0.002)	(0.001)	(0.003)	(0.001)
	[-0.043]	[-0.069]	[-0.118]	[-0.070]	[-0.114]
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
Mean Dep. Var.	0.21	0.17	0.05	0.39	0.08
$\sum Fog_b$	63	63	63	63	63
N	471	471	471	471	471

Notes: Individual-level sickness incidence is the number of cases per year of exposure between re-admission and age 10. The sample is restricted to children observed in the infirmary data to age 10 and children born in London. For definitions of upper and lower respiratory diseases, see Table 1. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses and standardised coefficients in square brackets. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.13: Effect of pollution exposure after birth on individual-level sickness incidence

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Upper Resp	(5) Lower Resp
$\sum_{s=1}^{28} Fog_{b+s}$	-0.002	-0.006*	-0.001	-0.008	-0.002
	(0.004)	(0.003)	(0.001)	(0.006)	(0.002)
	[-0.028]	[-0.104]	[-0.049]	[-0.077]	[-0.060]
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
Mean Dep. Var.	0.21	0.17	0.05	0.39	0.07
$\sum Fog_b$	62	62	62	62	62
N	452	452	452	452	452

Notes: Individual-level sickness incidence is the number of cases per year of exposure between re-admission and age 10. The sample is restricted to children observed in the infirmary data to age 10 and children born and living in London with their mothers before admission to the Foundling Hospital. For definitions of upper and lower respiratory diseases, see Table 1. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses and standardised coefficients in square brackets. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.14: Effect of pollution exposure *in utero* on individual-level sickness prevalence

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Upper Resp	(5) Lower Resp
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	-0.012	-0.003	-0.003	-0.014	-0.008*
	(0.009)	(0.008)	(0.003)	(0.013)	(0.005)
	[-0.087]	[-0.032]	[-0.061]	[-0.081]	[-0.109]
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	0.009	-0.000	-0.001	0.009	0.005
	(0.011)	(0.006)	(0.003)	(0.013)	(0.005)
	[0.068]	[-0.004]	[-0.012]	[0.055]	[0.070]
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	-0.024*	-0.003	-0.005*	-0.026*	-0.004
	(0.013)	(0.006)	(0.003)	(0.014)	(0.005)
	[-0.196]	[-0.033]	[-0.122]	[-0.169]	[-0.062]
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
Mean Dep. Var.	0.51	0.44	0.14	0.96	0.23
$\sum Fog_b$	63	63	63	63	63
N	471	471	471	471	471

Notes: Individual-level sickness prevalence is the number of sickness days per 100 days exposed between re-admission and age 10 or the percentage of days spent sick. The sample is restricted to children observed in the infirmary data to age 10 and children born in London. For definitions of upper and lower respiratory diseases, see Table 1. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses and standardised coefficients in square brackets. ** and *** denote statistical significance at the 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.15: Effect of pollution exposure after birth on individual-level sickness prevalence

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Upper Resp	(5) Lower Resp
$\sum_{s=1}^{28} Fog_{b+s}$	-0.007	-0.016	-0.007	-0.024	-0.012
	(0.014)	(0.010)	(0.005)	(0.018)	(0.009)
	[-0.026]	[-0.085]	[-0.077]	[-0.068]	[-0.085]
Sex Dummy	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
Mean Dep. Var.	0.52	0.44	0.14	0.97	0.23
$\sum Fog_b$	62	62	62	62	62
N	452	452	452	452	452

Notes: Individual-level sickness prevalence is the number of sickness days per 100 days exposed between re-admission and age 10 or the percentage of days spent sick. The sample is restricted to children observed in the infirmary data to age 10 and children born and living in London with their mothers before admission to the Foundling Hospital. For definitions of upper and lower respiratory diseases, see Table 1. Estimated with OLS. Unstandardised coefficients with heteroskedasticity robust standard errors in parentheses and standardised coefficients in square brackets. ** and *** denote statistical significance at the 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

D.5 Short-run Sickness Duration Results

Table D.16: Placebo effect of pollution exposure at time of sickness on sickness duration from injuries

	(1) Injuries
$\sum_{s=7}^{9} Fog_{t-s}$	0.028
	(0.074)
$\sum_{s=4}^{6} Fog_{t-s}$	-0.026
	(0.086)
$\sum_{s=1}^{3} Fog_{t-s}$	0.069
	(0.071)
Fog_t (Ad Day)	0.194
	(0.208)
Sex Dummy	Yes
Sickness Weather	Yes
Sickness Age FE	Yes
Sickness Year FE	Yes
Sickness Quarter FE	Yes
$\sum Fog_t$	22
N (sickness events)	309

Notes: Estimated with zero-truncated negative binomial models. Coefficients with robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively.

Sources: Foundling Hospital Cohort Dataset (2025).

D.6 Long-run Sickness Duration Results

Table D.17: Effect of pollution exposure at and around birth on sickness duration from respiratory diseases and measles

	(1) Influenza	(2) Influenza	(3) Influenza	(4) Measles	(5) Measles	(6) Measles
Fog_{b-1}	-0.075			0.109		
	(0.129)			(0.109)		
Fog_b (Birthday)		0.569***			0.214*	
		(0.156)			(0.117)	
Fog_{b+1}			0.085			0.212*
			(0.142)			(0.127)
Sex Dummy	Yes	Yes	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
Sickness Quarter FE	Yes	Yes	Yes	Yes	Yes	Yes
Sickness Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Sickness Age FE	Yes	Yes	Yes	Yes	Yes	Yes
$\sum Fog$	34	36	38	37	29	29
Clusters (individuals)	150	148	153	220	212	212
N (sickness events)	203	205	207	220	212	212

Notes: Sample restricted to children born in London. The reference group is held constant in the three specifications for each disease to make the comparisons straightforward to interpret. The reference group is children not born the day before, the day of, or the day after a fog event. Estimated with zero-truncated negative binomial models. Coefficients with standard errors clustered at the individual level in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.18: Effect of pollution exposure *in utero* on sickness duration from respiratory diseases and measles

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Measles
$\sum_{s=169}^{252} Fog_{b-s}$ (1st Tri)	0.001	0.008	-0.002	-0.002
	(0.006)	(0.006)	(0.012)	(0.008)
$\sum_{s=85}^{168} Fog_{b-s}$ (2nd Tri)	0.001	0.008*	-0.010	-0.009*
	(0.007)	(0.005)	(0.009)	(0.005)
$\sum_{s=1}^{84} Fog_{b-s}$ (3rd Tri)	-0.007	0.011**	-0.021**	-0.007
	(0.007)	(0.005)	(0.010)	(0.005)
Sex Dummy	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
Birth Year Trend	Yes	Yes	Yes	Yes
Sickness Quarter FE	Yes	Yes	Yes	Yes
Sickness Year FE	Yes	Yes	Yes	Yes
Sickness Age FE	Yes	Yes	Yes	Yes
$\sum Fog_b$	109	108	36	29
Clusters (individuals)	393	364	174	245
N (sickness events)	800	704	235	245

Notes: Sample restricted to children born in London. Estimated with zero-truncated negative binomial models. Coefficients with standard errors clustered at the individual level in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).

Table D.19: Effect of pollution exposure after birth on sickness duration from respiratory diseases and measles

	(1) Catarrh	(2) Tonsillitis	(3) Influenza	(4) Measles
$\sum_{s=1}^{28} Fog_{b+s}$	-0.019*	0.022*	0.010	0.002
	(0.010)	(0.013)	(0.023)	(0.008)
Sex Dummy	Yes	Yes	Yes	Yes
Individual Controls	Yes	Yes	Yes	Yes
Birth Weather	Yes	Yes	Yes	Yes
Birth Quarter FE	Yes	Yes	Yes	Yes
Birth Year Trend	Yes	Yes	Yes	Yes
Sickness Quarter FE	Yes	Yes	Yes	Yes
Sickness Year FE	Yes	Yes	Yes	Yes
Sickness Age FE	Yes	Yes	Yes	Yes
$\sum Fog_b$	104	106	35	28
Clusters (individuals)	374	349	161	236
N (sickness events)	756	670	218	236

Notes: Sample restricted to children born and living in London with their mothers before admission to the Foundling Hospital. Estimated with zero-truncated negative binomial models. Coefficients with standard errors clustered at the individual level in parentheses. *, ** and *** denote statistical significance at the 10%, 5% and 1% level respectively. Individual controls include mother's age and its square, father's occupation dummies and birth location dummies.

Sources: Foundling Hospital Cohort Dataset (2025).