Examining the influence of socio-economic status, area level deprivation and exposure to air pollution on asthma in childhood in England

Submitted by Philip James McBride to the University of Exeter as a thesis for the degree of Doctor of Philosophy in Medical Studies, October 2021.

I certify that all material in this thesis which is not my own work has been identified and that no material has previously been submitted and approved for the award of a degree by this or any other University.

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Abstract

The co-location of air pollution and socio-economic deprivation is increasingly well documented and studies have found that the socio-spatial distribution of health-related environmental characteristics, specifically air pollution, can be an important driver of geographical inequalities in health. The most deprived members of society face the highest exposures and the greatest risks due to a concept termed the triple jeopardy. Children face an increased susceptibility to air pollution exposure, and exposure can result in a range of health issues, such as asthma.

Linking longitudinal data from the Millennium Cohort Study (MCS), air pollution data available from EMEP4UK, and area level deprivation data from the Index of Multiple Deprivation, this thesis aims to explore the relationship between air pollution exposure, and both individual and area level socio-economic status to understand how these exposures interact to impact respiratory health in children. Following data linkage, cross-sectional analysis, time series analysis and multilevel modelling are employed to examine the data. Multilevel modelling is used to appropriately attribute variations in spatial health outcomes to differences between places, differences between people within places and differences over time. The use of multilevel modelling is an innovative step in understanding the relationship between socio-economic factors, air pollution and health outcomes.

Multilevel modelling found that 85% of the variation in asthma prevalence in children lies within MSOAs, whilst 14% of the variation was found to be over time. In comparison, 47% of the variation in wheezing was found to be due to differences over time. Two- and three-way interaction terms were included in the analysis to explore the impact of individual level socio-economic status, area level deprivation and air pollution exposure on asthma and wheezing prevalence in children, however no association was found. Moving forward, focussing interventions on improving both individual and area level socio-economic status, and implementing policies to lower pollution in the most deprived areas could help alleviate the health burden faced by the most deprived in society when exposed to air pollution.

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Abbreviations

BMI	Body Mass Index
CI	95% confidence interval
CLS	Centre for Longitudinal Studies
CO	Carbon monoxide
DIC	Deviance Information Criterion
EMEP	European Monitoring and Evaluation Programme
EMEP4UK	European Monitoring and Evaluation Programme for the UK
GEE	Generalised Estimating Equations
ICC	Intra-class correlation coefficient
IGLS	Iterative generalised least squares
IMD	Index of Multiple Deprivation
IOTF	International Obesity Task Force
ISAAC	International Study of Asthma and Allergies in Childhood
LSOA	Lower-layer Super Output Area
MCMC	Markov chain Monte Carlo
MCS	Millennium Cohort Study
MSOA	Middle-layer Super Output Areas
N	Nitrogen
NO	Nitrogen monoxide
NO_2	Nitrogen dioxide
NO_X	Nitrogen oxides
O_3	Ozone
OR	Odds ratio
PM	Particulate matter
PM_{10}	Coarse particulate matter
$PM_{2.5}$	Fine particulate matter
SO_2	Sulphur dioxide
UKDS	UK Data Service
VOC	Volatile organic compounds
VPC	Variance partition coefficient

Chapter 1. Introduction

Understanding persistent and increasing spatial inequalities in health is an important field of academic enquiry for epidemiologists and public health researchers. The aim of this research is to explore the impact of air pollution on health outcomes in childhood, controlling for both area and individual level deprivation across the English regions. Linking spatially disaggregated data on air pollution and area level deprivation with microdata containing demographic, socio-economic and health outcomes, this thesis uses a multilevel modelling approach to estimate the impact of air pollution exposure on respiratory health in childhood controlling for both individual and area level socio-economic profile.

The co-location of air pollution and socio-economic deprivation is increasingly well documented. The socio-economic patterning of residential opportunities means that individuals that are constrained financially face limited choices of where to live, and are more likely to reside near major sources of pollution, including roads with high traffic density, industrial facilities, waste disposal facilities, or airports (Gunier et al., 2003, Perlin et al., 1999). Recent studies have found that the socio-spatial distribution of health-related environmental characteristics, specifically measures of air pollution, can be an important driver of geographical inequalities in health status (Briggs et al., 2008, Crouse et al., 2009, Richardson et al., 2011). However, few studies have specifically looked at effect modification by individual level socio-economic status. Thus, studies have estimated the impact of air pollution on health outcomes adjusting for socio-economic position using area level deprivation as a proxy for individual socio-economic status.

This is problematic for two reasons. First, not all the people living in poor places are equally poor. Residential patterns of socio-economic status are heterogeneous even at small area levels such as the Lower-layer Super Output Area (LSOA). Thus, using an average area level indicator of socio-economic status loses information on the distribution or heterogeneity of socio-economic status within areas. Second, using only either individual or area level proxies fails to take account of both individual and area level context on health outcomes. The interaction between individual socio-economic status and area level deprivation

will be examined to ascertain if air pollution will have a more adverse impact on respiratory health for people with low socio-economic status living in the most deprived areas than people with low socio-economic status living in less deprived areas.

Currently there is no single dataset containing all the relevant data required for this research. Childhood data are available via The Millennium Cohort Study (MCS). Local level air pollution data are available from the UK Centre for Ecology & Hydrology via the EMEP4UK model, and deprivation data is available through the Index of Multiple Deprivation (IMD). Using spatially explicit data linkage techniques, data from the Millennium Cohort, the EMEP4UK model and the Index of Multiple Deprivation will be anonymously linked at the LSOA level to create a dataset with the necessary variables to examine the influence of socio-economic status, area level deprivation and exposure to air pollution on respiratory health in childhood in England.

1.1 Hypotheses

Air pollution exposure and its influence on human health is complex. This thesis sets out to examine how individual and area level socio-economic interact with air pollution to further impact childhood respiratory health, and seeks to address this with the following hypotheses:

1. The association between respiratory health and air pollution is stronger amongst individuals of lower, compared to higher, socio-economic status.
2. Area level deprivation will interact with individual socio-economic status so that the impact of pollution on respiratory health is stronger for people with low socio-economic status living in the most deprived areas than people with low socio-economic status living in less deprived areas.

The remainder of this Introduction Chapter outlines the motivations for this focus.

1.2 Respiratory health

Affecting approximately 300 million people globally (Braman, 2006), asthma is defined as "a heterogeneous disease, usually characterised by chronic airway inflammation", and is associated with a history of respiratory symptoms which includes wheezing, shortness of breath, tightness of chest and a reduced airflow

(Reddel et al., 2015). Research examining the impact of air pollution across the life course found that childhood is a particularly vulnerable time period for an individual (Schwartz, 2004). Weinmayr et al. (2010) found that exposure to air pollution had statistically significant associations with asthma symptoms in child respiratory health. A further review by Rodriguez-Villamizar et al. (2016) explored the effects of ambient air pollution on the respiratory health of children in Canada. This review confirmed the adverse effects that air pollution has on the respiratory system, lung function and health service use in children. It also found an association between traffic-related exposures and adverse respiratory outcomes.

The UK has one of the highest asthma mortality rates among young people for high-income countries worldwide and the highest rates of asthma symptoms globally in children (Gupta et al., 2018). The UK also has the highest rates of asthma related hospital admissions in Europe. A fifth of British children have been diagnosed with asthma by a doctor (Panico et al., 2007), while recent research has found that asthma affects approximately 15% of the population of England by the time they are in their early teens (Lewis et al., 2018). Asthma, and in particular, asthma in childhood is thus an important health priority within the UK.

1.3   Air Pollution

Air pollution is defined as 'the presence of substances in the atmosphere that can cause adverse effects to man and the environment' (Tiwary and Williams, 2018). Air pollution broadly incorporates any unwanted substance that contaminates the air and is detrimental to air quality, and can be anthropogenic, arising from human activities, or biogenic, arising naturally from the environment, such as animals or plants (Tiwary and Williams, 2018). Whilst natural events such as volcanic eruptions and wildfires account for some contribution to air pollution, anthropogenic activities have outweighed natural sources as the main source of air pollution for some time, to a larger degree since the Industrial Revolution (Kampa and Castanas, 2008).

Kampa and Castanas (2008) categorise pollutants into four main groups which include:

- gaseous pollutants, such as nitrogen oxides (NO_X), ozone (O_3) and sulphur dioxide (SO_2);
- persistent organic pollutants, like dioxins;

- heavy metals, which include lead and mercury; and
- particulate matter (PM).

To be classified as a health risk, a pollutant must undergo clinical, epidemiological or animal studies that show an association between pollutant exposure and detrimental health impacts. Research indicates (Chen and Kan, 2008) that the key air pollutants that impact people's health include:

- Particulate matter (PM);
    - PM$_{10}$ (particulate matter with a particle diameter of 10 μm or under);
    - PM$_{2.5}$ (particulate matter with a particle diameter of 2.5 μm or under);
- Nitrogen oxides (NO$_X$) which is a generic term for the nitrogen oxides that are most associated with air pollution;
    - nitrogen dioxide (NO$_2$);
    - nitric oxide (NO);
- Ozone (O$_3$).

Focussing on the impact of air pollution on child respiratory health, these are the pollutants of interest to this thesis. It is important to note that a full chemical analysis of air pollutants and a full physiological discussion of the impact of air pollution on human health was deemed outside the remit of this thesis. As such the aim of the remainder of this chapter is to introduce the air pollutants of interest to the rest of this thesis and briefly set the context of the mechanisms in which air pollution may impact human health.

1.3.1 Pollutants of interest

1.3.1.1   Particulate Matter (PM)

PM encompasses both natural and man-made pollutant emissions (Kelly and Fussell, 2012). Natural sources of PM include pollen, fungal spores, volcanic ash, sea salt, wind-blown dust and soil particles, among others. Man-made sources of PM include fossil fuel combustion, industrial processes, construction work, quarrying and mining activities, cigarette smoking and wood stove burning (Kelly and Fussell, 2012). In urban areas, road transportation is the main source of PM pollution with factories and power stations that burn fossil fuels also contributing a significant amount of PM in less developed countries. PM produced from road

transport includes engine emissions and wear, tyre and brake wear and dust from the surface of the road (Kelly and Fussell, 2012). Diesel vehicles produce a higher volume of PM than their unleaded counterparts, and thus are the largest single source of vehicle emitted PM. Kelly and Fussell (2012) state that due to the increase in the number of diesel cars in the industrialised world, diesel exhaust particles can account for up to 90% of airborne PM in some cities. PM can be described as being either primary or secondary particles. Primary particles are released directly from their source into the atmosphere, whilst secondary particles come about within the atmosphere following chemical reactions (Kelly and Fussell, 2012).

Depending on the diameter of the PM, PM can be classified into three size ranges; ultrafine, fine and coarse PM. $PM_{0.1}$ (ultrafine PM) has a diameter that is 0.1 µm or less and is generated directly by combustion and photochemical activity (Valavanidis et al., 2008), as well as from transportation emissions. These particles are unstable and exist for only a short period of time, however, they are capable of growing in size through coagulation and condensation and are capable of the deepest lung penetration, having the potential for passing into the blood stream.

$PM_{2.5}$ (fine PM) is made up of particles with a diameter of 2.5 µm and under. The main source of $PM_{2.5}$ is transportation emissions. $PM_{2.5}$ can also be called respirable particles due to their ability to enter the alveolar gas exchange region in the lungs, where up to 50% of the particles are retained (Valavanidis et al., 2008). Additionally, due to their porous surface, they can absorb and retain toxic substances. PM_{10} (coarse PM) is made up of particles with a diameter of 10 µm or less and come from the combustion of fossil fuels. PM_{10} can also be called thoracic particles as they can travel beyond the nose, throat and larynx and become deposited along the airways in the thorax (Kelly and Fussell, 2012). The majority of PM suspended in the air consists of around 90% to 95% coarse particles, whilst the smaller particles account for only 1% to 8% of airborne PM (Valavanidis et al., 2008). However, $PM_{2.5}$ is more prolific, resulting in a larger total surface area than coarse particles.

Figure 1.1 depicts the variation in PM levels in the UK from 1970 to 2018, using the 1970 outputs as a reference for the index line. Both PM_{10} and $PM_{2.5}$ follow the same pattern and it can clearly be seen that PM pollution has decreased

considerably since 1970. Troughs in the graph that are visible in the years 1972, 1974 and 1984 coincide with mining strikes (Spence and Stephenson, 2007, Hughes, 2012). Figures 1.2 and 1.3 illustrate the proportion of PM_{10} and $PM_{2.5}$ that are derived from each pollutant source in the UK from 1970 to 2018.
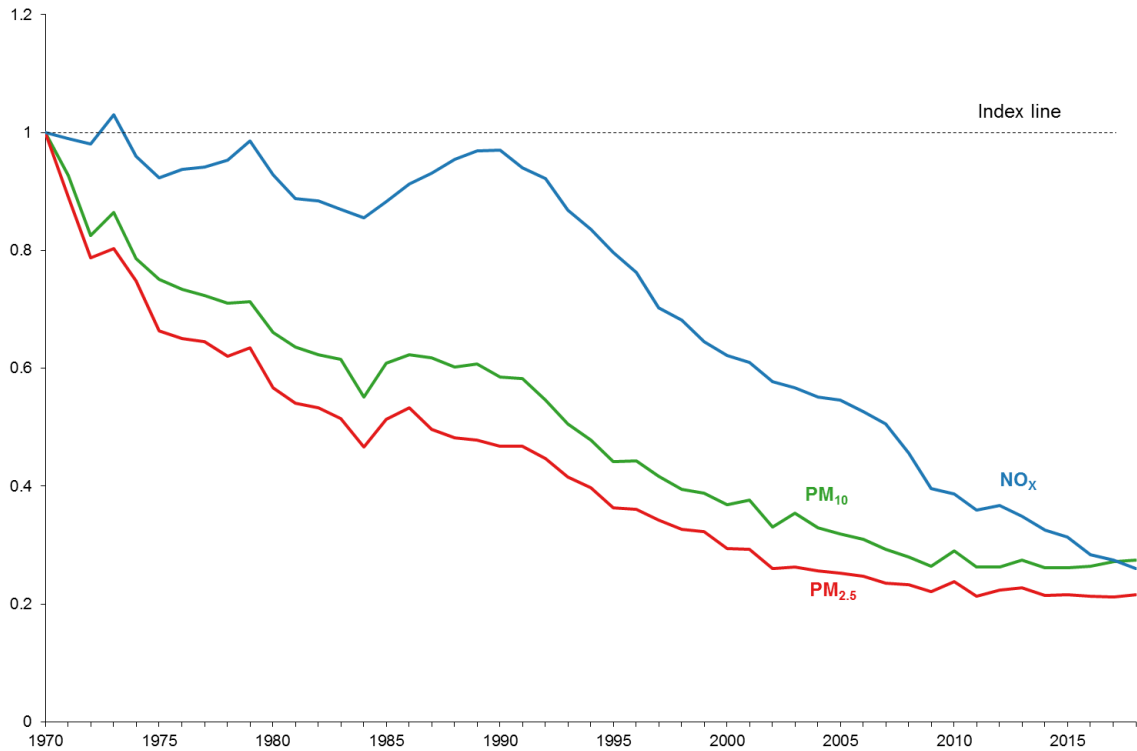
Figure 1.1   Historical trends in UK emissions of key air pollutants between 1970 and 2018 (adapted from NAEI UK, 2018)

■ Agriculture/Waste ■ Production Processes
■ Road Transport ■ Other Transport
■ Combustion in Industry/Commercial/Residential ■ Public Electricity and Heat Production
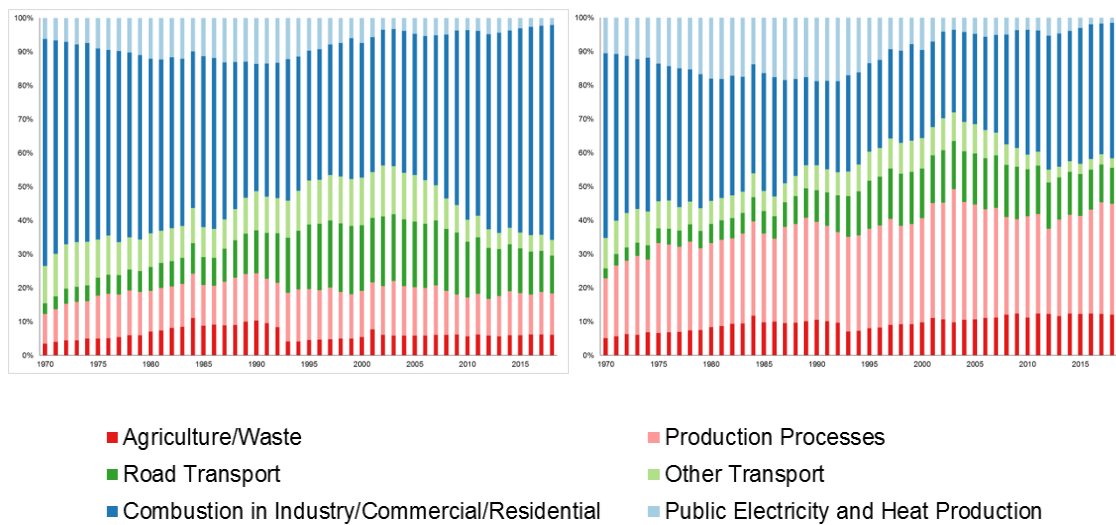
Figures 1.2 & 1.3   Graphs showing the proportion of PM_{10} (*1.2*) and $PM_{2.5}$ (*1.3*) that comes from various sources (adapted from NAEI UK, 2018)

Table 1.1 summarises the different characteristics of PM, including size, where the particles are most commonly deposited based on their size and some examples of the impacts these particles have on human health.

Table 1.1 Characteristics of particulate matter

Pollutant	Size (µm)	Common deposition area	Examples of impact on human health
$PM_{0.1}$	$PM < 0.1$	Blood vessels	Mortality, decreased lung function, lung cancer, bronchitis
$PM_{2.5}$	$0.1 < PM < 2.5$	Alveoli	Mortality, heart rate variability, cardiac arrhythmia, deep vein thrombosis
PM_{10}	$2.5 < PM < 10$	Thorax	Mortality, lung cancer, COPD, CVD, asthma

1.3.1.2   Nitrogen oxide (NO_X)

NO_X is the term given to a group of highly reactive gases. The majority of these gases are emitted in the air as nitric oxide (NO) and nitrogen dioxide (NO_2). Fossil fuel combustion is the main anthropogenic source of NO_X, particularly from transportation but also from industrial processes like power generation (Brook et al., 2004). Furthermore, a high temperature can result in combustion that oxidises atmospheric nitrogen (N), firstly to NO and then to NO_2. Urban areas with a high concentration of traffic can experience a high local NO_X concentration. A usual daily pattern of NO_X pollution follows a generally low background reading, with peaks in the morning and evening, coinciding with rush-hour traffic. N found in fossil fuels can become oxidised under oxygen-rich combustion conditions, however NO_X is also produced naturally and can be released from sources such as fires and volcanoes. NO has a low solubility in water, being able to spread to all parts of the respiratory system and diffuse through both the epithelium and the capillary vessels of the lungs, disrupting the alveolar structures and the function they play (Boningari and Smirniotis, 2016). Acid rain is an example of the environmental impact caused by NO_X pollution.

Figure 1.1 also illustrates the variation in levels of NO_X emission in the UK from 1970 to 2018. NO_X levels tended to fluctuate near to 1970 levels for 20 years before starting to decrease slowly at the beginning of the 1990s. Figure 1.4 illustrates the proportion of NO_X that is derived from various sources in the UK from 1970 to 2018.
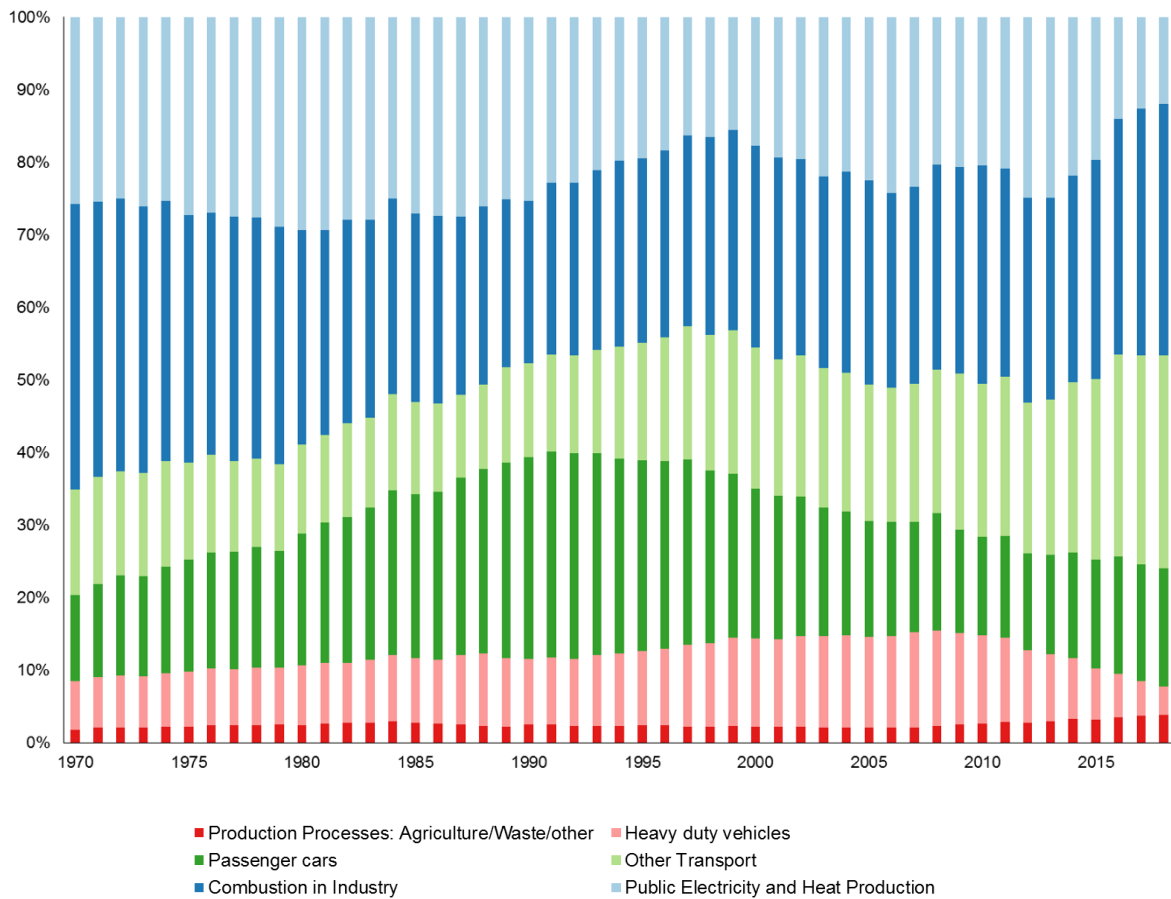
Figure 1.4　　　　　The proportion of NO$_X$ that comes from various sources (Adapted from NAEI UK, 2018)

1.3.1.3　　　　Ozone (O$_3$)

Whilst O$_3$ is important in the stratosphere for blocking the sun's harmful ultraviolet light, at ground level it is toxic to human health (Curtis et al., 2006). At the ground level O$_3$ is formed through photochemical reactions between sunlight and other pollutants, such as NO$_X$ emitted from vehicular and industrial sources (Brook et al., 2004). These reactions are more common during periods of warmer weather, thus O$_3$ production peaks with the highest summer temperatures. In terms of its day-to-day production, there tends to be a broad peak of O$_3$ formation from late morning through to late afternoon, although large-scale vehicle use can cause such an increase in O$_3$ production that the elevated concentration expands over thousands of square miles (Brook et al., 2004). However, O$_3$ concentrations tend to be lower in city centres compared to rural areas as a result of O$_3$ scavenging by NO from traffic (Brunekreef and Holgate, 2002).

1.3.2 Air pollution exposure and its health effects

In the UK, air pollution exposure is accountable for up to 50,000 premature deaths annually, as well as decreasing life expectancy by 7 to 8 months on average (Jephcote and Chen, 2012). For England and Wales, it has been estimated that a 10 µg/m3 reduction in annual $PM_{2.5}$ would result in a total population gain of over 29 million life-years (Jephcote and Chen, 2012).

At the same time, research on the relationship between individual socio-economic status and individual living environment and human health outcomes has grown (Braubach et al., 2009). People that are disadvantaged or marginalised are more likely to experience a more polluted and hazardous living environment, which in turn has impacts on their health (Briggs et al., 2008). Within this context, Jerrett et al. (2001) proposed the term 'triple jeopardy' to explain how disadvantaged groups face increased risks from social and behavioural determinants of health, higher risks from high ambient pollution exposure and an effect modification that makes exposure to ambient air pollution exert disproportionately large health effects on them when compared to more advantaged groups.

Although air pollution levels are decreasing, the focus on air pollution, socio-economic status and child health is particularly important as evidence from the UK demonstrates that childhood poverty is increasing (Wickham et al., 2016). At the same time, the UK government has abolished previous plans and policies that attempted to eradicate childhood poverty (Wickham et al., 2016).

1.4 Thesis structure

It is within this context that the thesis continues as follows:

Chapter 1 introduces the topic of the thesis, explaining the rationale behind its creation. It presents the hypotheses and structure of the thesis. Chapter 1 also introduces asthma and wheezing as proxies for respiratory health, and presents a brief overview of air pollution, specifying the pollutants of interest to this thesis. The effects of air pollution exposure are discussed, as are the health impacts of exposure.

Chapter 2 provides a review of the current literature concerning air pollution, its impact on health and how it interacts with socio-economic status to further

exacerbate health issues faced. Chapter 2 also presents the theoretical framework underpinning this thesis, the social causation theory. The complex, interacting relationship between air pollution, area level deprivation and individual level socio-economic status, referred to as the triple jeopardy is also introduced. Particular attention is given to the health impacts faced by children. The review highlights the need for future analysis to take into consideration socio-economic status at both the individual and area level when exploring the impact of air pollution on health.

Chapter 3 details the data and methods used throughout this thesis. Cohort data from the Millennium Cohort Study, air pollution data from EMEP4UK and deprivation data available through the Index of Multiple Deprivation were all used in the analyses. Data linkage was necessary to compile all data available into one working dataset before statistical analysis through cross-sectional, time series and multilevel approaches could be carried out.

Chapter 4 presents the results from cross-sectional analysis, providing a basis to understand the relationship between air pollution, area level deprivation, and individual socio-economic status on childhood respiratory health. This analysis examined each wave individually to investigate the impact of exposures at each point in time.

Chapter 5 presents the results from time series analysis, building on the previous chapter with the inclusion of time. This chapter specifically examines how respiratory health is influenced by both early life exposures (in Wave 1) to air pollution and socio-economic status, and by exposures over time. This approach allows for conclusions to be drawn about the importance of critical periods of exposure as well as the accumulative effect of the different exposures.

Chapter 6 presents the results from multilevel modelling, an approach that accounts for both the spatial and temporal aspects of the data, further building on previous analyses. Multilevel modelling considers the natural nested structure of the data, providing a robust analytical method that details how much of the variation in respiratory health can be accounted for between Middle-Layer Super Output Areas (MSOAs), within MSOAs or over time.

Chapter 7 discusses the results presented in previous chapters, positing explanations for the findings. Particular attention is given to interaction terms that

aimed to examine the relationship between air pollution exposure and individual and area level socio-economic status. Conclusions are drawn from the findings, strengths and limitations are discussed and future policy is considered.

Chapter 2. Literature Review

2.1   Introduction

Air pollution is a major global public health risk (Boogaard et al., 2019). Following the Industrial Revolution and the associated increase in air pollution levels due to the combustion of fossil fuels, several major air pollution events occurred that provided quantitative evidence of the adverse effects that short-term air pollution exposure had on health (Dockery and Pope III, 1994). As scientific knowledge developed, people began to fully understand the impact that air pollution exposure had on their health. The first published piece of literature that focussed on air pollution was published in 1911, although it wasn't until the 1950s that literature discussing air pollution was regularly published (Figure 2.1). More recently, literature has discussed how the very nature of air pollution is changing (Landrigan, 2017). Whilst air pollution in the home has been decreasing since the 1990s, there has been an increase in ambient air pollution brought about by rapid globalisation and its associated industrialisation.

The 12,000 excess deaths associated with the Great Smog of London in December of 1952 drew widespread attention to the negative impact of air pollution on human health and led to the implementation of The Clean Air Act, a policy that aimed to reduce dangerous levels of air pollution (Polivka, 2018). The Great Smog was a landmark case in environmental epidemiology due to the scale of the disaster and for providing empirical evidence of the relationship between air pollution and human health. The first recorded piece of literature that examined both air pollution and health was published in 1915, but again it was not until the 1950s that literature examining this topic was regularly published (Figure 2.1). As the literature expanded in this area, the pollutants that were most frequently examined in relation to human health, and as mention in Chapter 1, included;

- Particulate matter (PM);
  - PM_{10} (particulate matter with a particle diameter of 10 μm or under);
  - $PM_{2.5}$ (particulate matter with a particle diameter of 2.5 μm or under);

- Nitrogen oxides (NO_X) which is a generic term for the nitrogen oxides that are most associated with air pollution;
  - nitrogen dioxide (NO_2);
  - nitric oxide (NO);
- Ozone (O_3).

As research on the negative implications of air pollution on human health developed, studies in this area expanded to include the role of individual societal factors, such as socio-economic status, and their interaction with air pollution and subsequent impact on health (Jerrett et al., 1997, Briggs et al., 2008). This research demonstrated that people with lower socio-economic status were more likely to be exposed to higher levels of air pollution, while also having increased vulnerability to the impacts of air pollution due to worse baseline health. The first piece of literature that examined air pollution, health and socio-economic status was published in 1970, and interest in health inequality and environmental injustice has grown steadily since (Figure 2.2)
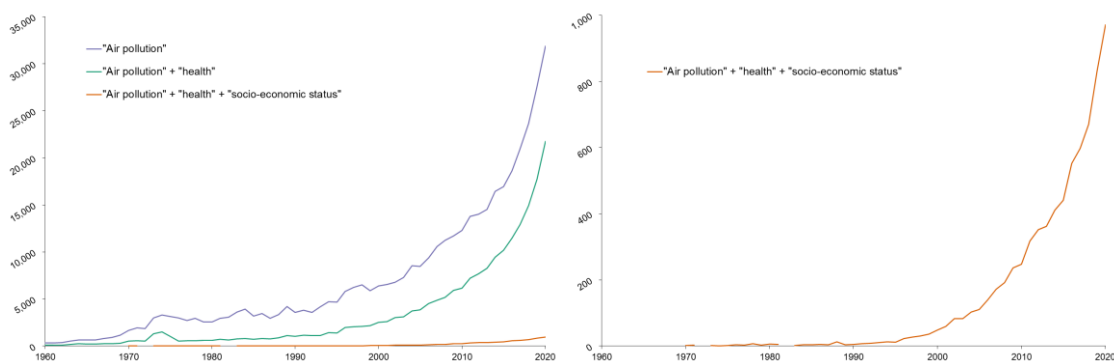
Figure 2.1 and 2.2   Number of published pieces of work with titles containing the terms "air pollution", "health" and "socio-economic status" between the years 1960 and 2020 (Review of papers available on Scopus, 2021)

This review continues by discussing the differing definitions used for asthma in epidemiology studies, and how asthma is clinically diagnosed in children. Following this, literature that explores exposure and susceptibility to air pollution is discussed, also examining how this is impacted by socio-economic status. The theoretical framework that underlies the relationship between air pollution, socio-economic status and health is then outlined before exploring literature that discusses the links between air pollution and socio-economic status, and socio-

economic status and its impacts on health. The review moves on to examine the association between air pollution, socio-economic status and health, as well as focussing specifically on the health of children.

2.2   Air Pollution: Exposure and Susceptibility to Exposure

People are exposed to air pollution in a variety of ways, the most common method being through inhalation. However, exposure may also arise through the ingestion of food and water that has become contaminated from air pollution and dermal contact (Kampa and Castanas, 2008). When examining the impact of air pollution on health, studies look at both long- and short-term exposures (day-to-day variation), with both types of exposures found to negatively impact on people's health. In turn the temporal impact of air pollution can be either acute or chronic. An acute condition is one that is severe and occurs rapidly, within a few hours to a few weeks, whilst a chronic condition develops over a longer period of time, usually a number of years (Pisano, 1996). Increases in mortality, morbidity and hospital admissions are associated with both long- and short-term exposure to pollutants (Brunekreef and Holgate, 2002).

2.2.1   Long-term exposure

Studies focussing on the impacts of long-term exposure to air pollution have found consistent associations between long-term exposures and negative health impacts, such as decreased lung function, chronic bronchitis, increased risk of lung cancer, and cardiopulmonary mortality (Valavanidis et al., 2008). Studies focussing on long-term $PM_{2.5}$ exposure found that it impacts heart rate variability, blood viscosity and coagulability, cardiac arrhythmia, deep vein thrombosis, atherogenesis, among other health impacts (Valavanidis et al., 2008). Pope III et al. (2002) conducted a study to examine the effect of long-term exposure to $PM_{2.5}$ on all-cause, cardiopulmonary and lung cancer mortality and found that a 10 µg/m3 increase in $PM_{2.5}$ was associated with a 4% increased risk of all-cause mortality, a 6% increased risk of cardiopulmonary mortality and an 8% increase in lung cancer mortality. This research also found that long-term exposure to PM has larger, more persistent and cumulative effects when compared to short-term exposures (Pope III, 2007). Similarly, long-term exposure to NO_X has also been shown to be associated with an increase in mortality rates as well as having an impact on lung function, and subsequent repercussions (Stockfelt et al., 2015,

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Mölter et al., 2013). Long-term exposure to O_3 has also been seen to be associated with an increase in mortality, specifically mortality related to respiratory and circulatory issues (Jerrett et al., 2009, Lim et al., 2019, Turner et al., 2016).

2.2.2 Short-term exposure

The impact of short-term exposure to air pollution has received considerable attention (Bell et al., 2013, Wilson et al., 2005, Chen et al., 2016, Guo et al., 2013), particularly the impact of big pollution events on mortality and hospital admissions. With regard to the impact of short-term exposure to air pollution on mortality, higher mortality rates are particularly seen among the elderly and/or those with chronic illnesses (Valavanidis et al., 2008). Indeed, the APHEA (Air Pollution and Health: a European Approach) found an increase in daily mortality associated with an increase in air pollution concentration. Brunekreef and Holgate (2002) and Dockery and Pope III (1994) had similar findings in the United States. The APHEA also found an increase in hospital admissions associated with increased air pollution concentration, and Schwartz et al. (1993) found an increase in emergency department visits for asthma related health issues. An increase in reported asthma attacks was also associated with increased air pollution concentrations (Dockery and Pope III, 1994, Laurent et al., 2008, Cai et al., 2016).

Short-term exposure to PM has been linked with increased hospital admissions, as well as both increased mortality and morbidity (Bell et al., 2013). Short-term exposure to PM specifically exacerbates certain respiratory diseases, such as asthma (Miri et al., 2017). Exposure to NO_X has been associated with an increased occurrence of acute respiratory diseases and a decrease in pulmonary function (Valavanidis et al., 2008). Respiratory morbidity, decreased immune system and lung function, lung inflammation and reduced lung growth have also all been linked to NO_X exposure (Boningari and Smirniotis, 2016). Exposure to NO_X pollution also gives rise to respiratory disease, like bronchitis and emphysema, and exacerbates issues surrounding heart disease (Boningari and Smirniotis, 2016, Valavanidis et al., 2008). Short-term exposure to O_3 can lead to increased hospital admissions for respiratory illness, as well as resulting in airway

inflammation, decreased lung function, pulmonary disease and asthma, and cardiovascular mortality (Tager et al., 2005).

Although providing important evidence on the negative impact of big weather and pollution events on human health, Valavanidis et al. (2008) argue short-term exposure studies only capture:

> (i) deaths attributable to these events in the relative short term; and

> (ii) are likely to only capture deaths of the most frail people who would most likely have died shortly afterwards regardless.

In response, Valavanidis et al. (2008) argue that, long-term studies following people facing exposure to air pollution, especially those exposed to low or very low concentrations of pollutants, consistently over a period of time are important to understand the extent to which air pollution impacts health.

2.2.3 Child susceptibility to air pollution

The susceptibility of an individual to both long- and short-term exposure to air pollution is variable and depends on different factors, such as age. Children and the elderly are more susceptible to the effects of air pollution in comparison to middle-aged adults (Jephcote and Chen, 2012, Lavigne et al., 2012, Chen et al., 2014). Children face increased risk from exposure to air pollution due to their undeveloped respiratory and immune systems (Schwartz, 2004). Compared to adults, children have a larger lung surface area per kilogram of body weight, meaning they breathe up to 50% more air per kilogram of body weight. As a child's lungs are still developing, specifically the epithelial layer, there is an increased permeability which results in an increased absorption of detrimental pollutants (Jephcote and Chen, 2012). This in turn increases the chance of tissue inflammation and agitators passing into the blood stream, which may result in asthma. Research has found that long-term exposure can stunt the development of cardiorespiratory organs which could result in long-term limiting health conditions.

2.3 Socio-economic Status

As noted previously, exposure to air pollution is influenced by socio-economic status, and at the individual level people with a lower socio-economic status may

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be exposed to increased levels of air pollution through a number of pathways (Jerrett et al., 1997, Briggs et al., 2008). The pathways leading to higher exposure rates are multiple and multifaceted. For example, certain employment opportunities and poorly maintained housing are associated with increased exposure to air pollution (Blanc et al., 2006). At the area level, people with a lower socio-economic status are more likely to live closer to polluting factories, airports, waste management facilities and main roads, consequently facing increased risk of exposure to lower quality air (Blanc et al., 2006). To fully understand how an individual's rate of exposure and susceptibility to air pollution varies depending on socio-economic status, with a view to modelling the impact of air pollution and human health it is important to explore the theoretical concepts underpinning these relationships.

2.4   Health inequalities – the theoretical framework

Since the release of the Black Report (Gray, 1982), the dominant conceptual framework underlying the analysis of poor health in the social sciences is the multifactorial model of disease causation. This model posits that most illnesses are the result of multiple causes, determinants, and risks involving a complex set of interactions between individuals, the environment, and other factors. Based on this model, social research seeks to identify characteristics that increase the likelihood an individual has of developing a particular disease. Following the Black Report and the subsequent Marmot Report (Marmot et al., 2010) research in the health and social sciences has focussed on the role of socio-economic status on health outcomes. Whilst much of this research acknowledges that socio-economic status is a multi-dimensional concept, empirically it has been modelled through a combination of individual factors including educational outcomes, occupation and income (Elo, 2009). Education is believed to impact on health through the accumulation of knowledge regarding health-promoting behaviours, as well as through problem solving and decision making skills (Elo, 2009). Those working in a higher occupational class are more likely to have jobs that are not physically challenging or that could be perceived as being dangerous. Income directly influences an individual's ability to make use of certain resources, for example high quality housing and health care access. Building upon this, education, occupation, and income are all interlinked, as education influences

subsequent occupation and therefore income, and so should be considered when exploring a person's socio-economic status (Chi et al., 2016).

2.4.1 The social causation theory

Under the umbrella of the multifactorial model of disease causation, the social causation theory of health outcomes has received much attention. The social causation theory states that health inequality is caused by the negative effect a lower socio-economic status has on health, therefore, circumstances in higher socio-economic positions are more beneficial to health than in lower socio-economic positions (Kröger et al., 2015). Although health inequality has been widely researched, the causal mechanisms underpinning the relationship between low socio-economic status and poor health are much debated (Foverskov and Holm, 2016). Socio-economic status is theorised to have a negative effect via mediating factors that are underpinned by:

- material,
- cultural-behavioural or
- psychosocial

factors (Skalická et al., 2009, Foverskov and Holm, 2016). Understanding what factors impact negatively on health outcomes is important in deciding the most effective policy measures for population health.

2.4.1.1 The materialist explanation

The materialist explanation views material conditions as the most important factor influencing an individual's health. It reflects their social position in society and focuses on income and what income enables, such as access to goods and services and exposures to material risk factors, like poor housing conditions, hazards in the workplace and environmental exposures, such as high ambient air pollution concentrations (Foverskov and Holm, 2016, Skalická et al., 2009). Expanding on this, land use restriction can explain further; people with lower income have a restricted choice when deciding where to live and are therefore more likely to live in close proximity to sources of pollution, such as industrial or waste disposal facilities, airports and busy roads due to the affordability of these locations (Crouse et al., 2009). Furthermore, polluting facilities are frequently prevented from being situated near affluent areas due to zoning restrictions, land

prices and prevailing winds. Therefore, people with financial difficulties are more likely to reside in poor quality housing in areas with higher ambient air pollution concentrations. In the UK, research by Mitchell and Dorling (2003) found that the communities with access to the fewest cars tend to experience the highest levels of air pollution due to increased risks of exposure when travelling via foot or public transport. In addition, the areas that experience the highest levels of air pollution whilst simultaneously emitting the lowest levels of air pollution are among the poorest in the country (Mitchell and Dorling, 2003).

2.4.1.2 The cultural-behavioural explanation

The cultural-behavioural explanation suggests that differences in health behaviour are a consequence of disadvantage and that unhealthy behaviour may be more culturally acceptable within groups of people with similar socio-economic status (Skalická et al., 2009). There is much evidence demonstrating that people with lower social status are more likely to partake in less healthy behaviours (Foverskov and Holm, 2016) such as smoking, physical inactivity and excessive alcohol consumption. Cigarette smoke is a major component of indoor air pollution and exposure to it is a significant risk factor for respiratory symptoms and diseases, especially in children (Seaton, 1996, Pugmire et al., 2014). Physical inactivity can result in overweight or obese individuals, which in turn could impact on their susceptibility to air pollution. Children with obesity are more likely to suffer from asthma, and children that are overweight may be more susceptible to the pulmonary effects of pollutant exposure (Matsui, 2014). Furthermore, overweight and obese children have a decreased response to inhaled steroids, increasing the health risks associated with respiratory events (Forno et al., 2011).

2.4.1.3 The psychosocial explanation

The psychosocial explanation focusses on how social inequality can make people experience feelings of subordination or inferiority, and how these feelings can have an effect on health (Skalická et al., 2009). The relationship between socio-economic status and health is explained by the unequal distribution of psychosocial risk factors, such as levels of control and work demands, a lack of social support, or imbalances in effort-reward (Skalická et al., 2009). The psychosocial determinants of health operate at the individual level, are subjective

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to the individual (Denton et al., 2004) and can be split into three main groups that are interrelated: critical life events, chronic stressors and psychological resources. Exposure to stress inducing life events increases risk of psychological distress and psychiatric disorders, poor physical health and substance abuse (Denton et al., 2004). Exposure to chronic stress, which is the ongoing and challenging conditions of daily life such as financial stress, social life stress and family health stress, is also associated with distress and chronic health conditions (McDonough and Walters, 2001). Psychological resources such as self-esteem and sense of coherence are also determinants of health (Denton et al., 2004), for example, low self-esteem is linked with an increased prevalence of depression which could in turn increase an individual's susceptibility to the negative impacts of air pollution exposure.

2.4.2 The triple jeopardy

As noted in Chapter 1, the last two decades have seen an increased interest in the relationship between the environment and health inequalities (Jephcote and Chen, 2012, Hansell et al., 2016).The relationship between socio-economic status and an individual's living environment must also be considered as one of the main influencing factors pertaining to environmental inequalities as the quality and environmental context of housing, which is dictated by socio-economic status (Braubach et al., 2009). As discussed, people that are disadvantaged or marginalised are therefore more likely to experience a more polluted and hazardous living environment, which in turn has impacts on their health (Briggs et al., 2008).

Within this context, Jerrett et al. (2001) proposed the term 'triple jeopardy' to explain how disadvantaged groups face increased risks from social and behavioural determinants of health, higher risks from high ambient pollution exposure and an effect modification that makes exposure to ambient air pollution exert disproportionately large health effects on them when compared to more advantaged groups. O'Neill et al. (2003) built on this work using the social causation framework to understand the pathways in which the effects of air pollution exposure on health are differently distributed by socio-economic status. According to O'Neill et al. (2003) air pollution exposure may impact health outcomes via:

- Materialist factors: air pollution exposure is differently distributed based on socio-economic status (Hajat et al., 2015), so the more disadvantaged are exposed to higher concentrations of air pollution both at home and in the workplace.

- Cultural-behavioural factors: some health conditions such as asthma, diabetes and cardiovascular diseases, as well as certain behavioural traits that increase vulnerability to air pollution, are linked to socio-economic status (Denton et al., 2004).

- Psychosocial factors: low socio-economic status may directly increase susceptibility to air pollution related health consequences due to raised levels of psychosocial stress (Forastiere et al., 2007).

Similarly, to the social causation theory, the triple jeopardy concept mirrors the materialist, cultural-behavioural and psychosocial explanations and should therefore be considered collectively. The explanations presented interact with each other in a multitude of pathways to have an impact upon people's health in different ways. For example, an individual suffering from poor health would have limited employment opportunities, thus also having a limited income which further impacts their mobility and access to power. This then impedes their ability to move away from or to mitigate against the hazards in their community, such as exposure to air pollution (Briggs et al., 2008). These theories provide a clear and robust example of the complex relationship between health, socio-economic status and the environment, especially air pollution. Before discussing literature that focusses on this interaction between air pollution, socio-economic status and health, literature examining the relationship between air pollution and socio-economic status will first be discussed.

2.5   The relationship between air pollution and socio-economic status

Jerrett et al. (1997) examined the relationship between air pollution and socio-economic status in Ontario, Canada. This study explored the relationship between household income, housing prices, manufacturing employment, population change and air pollution emissions. Jerrett et al. (1997) found a significant relationship between these aforementioned variables and air pollution emissions. Together, these variables explained roughly 63% of the variation in pollution emissions, however household income was shown to have a positive

association with pollution emissions which disagreed with the original hypothesis. Jerrett et al. (2001) examined whether populations that had low socio-economic status were more likely to experience high levels of air pollution, this time looking specifically in Hamilton, Ontario. Using a comprehensive intra-urban air pollution monitoring network, Jerrett et al. (2001) found that two large steel-makers based in the study area had created zones with high levels of pollution. This study estimated that sickness and death caused by PM emissions in this area cost roughly Can$537 million per annum. From the research, Jerrett et al. (2001) concluded that people with a lower socio-economic status were exposed to higher levels of ambient air pollution in Hamilton than groups with a higher socio-economic status. Pollutant exposure was significantly negatively associated with house prices, whilst unemployment and low income were also found to be significant predictors of exposure.

One of the first studies in the UK that explored environmental inequalities regarding air pollution and socio-economic status was carried out by Friends of the Earth in 1999 (McLaren et al., 1999). Using data from their own 'Factorywatch' project as well as income data for every postcode sector in the country, Friends of the Earth discovered that there were 662 polluting factories in postcode areas with an average annual income below £15,000. In addition, only five factories were found in areas with an average annual income above £30,000. Areas with a higher number of factories were found to generally have a lower average annual income. Teesside in North East England had one area with 17 factories and an average annual income of £6,200, 64% below the national average. McLaren et al. (1999) found that the poorest families, those with an average annual income below £5,000, were twice as likely to live within the vicinity of a factory when compared to families with an average annual income over £60,000. The study also found that over 90% of factories in London were situated in areas with a below average income.

Research in Montreal, Canada, by Crouse et al. (2009) explored the association between socio-economic status and ambient air pollution, specifically NO_2 at the household and neighbourhood level. The research found a clear association between NO_2 concentration and both material and social deprivation indicators at the neighbourhood level, including household income and proportion of people that live alone. Crouse et al. (2009) also found that there were certain areas in

Montreal that experienced a 'double burden', as these areas experienced both high levels of deprivation and high concentrations of ambient NO_2. However, the highest levels of pollution were not only found in the more deprived areas, but in some wealthier areas also. Before adjusting the analysis, Crouse et al. (2009) found that neighbourhoods with a high proportion of individuals with lower education also experienced lower levels of air pollution, and vice versa. One possible explanation of this is 'student ghettos', where high concentrations of students live in areas of the city that experience a high volume of traffic.

Research in London by Goodman et al. (2011) explored the impacts of traffic based air pollution and area and individual level socio-economic status. The concentration of NO_X steadily decreased moving out from central London, and throughout the city the mean air pollution concentration was higher in areas of increased deprivation, although the magnitude of the association was overestimated in the study. In Minnesota, Pratt et al. (2015) examined the combination of traffic, air pollution, ethnicity and socio-economic status. Owning a car lowered the emissions someone faced when compared to walking or using public transport, which are the more common methods of transport for those with a lower socio-economic status. Ethnic minorities and those with low socio-economic status experienced higher exposure to pollution and were therefore at a disproportionately greater risk of health impacts. It is difficult to investigate socio-economic status without also discussing the influence it has on an individual's health. The following section discusses the findings from literature that touched on this topic.

2.6   Respiratory health: asthma and wheeze

Asthma is a complex respiratory disease and due to its complexity, over time epidemiological studies have typically employed differing definitions for asthma. For example, a recent systematic review (Islam et al., 2021) examined 190 studies published between 1995 and 2020 that focussed on asthma and wheeze in children under the age of 13 years, and found that ten different definitions for asthma and five different definitions for wheeze were used. Islam et al. (2021) found that when defining asthma, epidemiological studies have either used an evidence-based definition or an operational definition. An evidence-based definition depends on clear evidence of the type, severity and frequency of

symptoms, which is challenging to ascertain in children. Diagnosing asthma clinically can be difficult with children and is generally not done for children under five years (Moral et al., 2019). Clinical diagnosis may consist of three different tests; a spirometry test to show if airways are blocked and narrow; a FeNO (fractional exhaled nitric oxide) test measuring airway inflammation which can show allergic asthma; and a peak flow test, which measures how quick a child can exhale. The results from these tests, along with relevant information about symptoms (such as potential triggers, severity, duration, etc.) are considered by a medical professional when making an asthma diagnosis.

An operational definition of asthma was found to be generally based off of parental responses to a questionnaire developed by the International Study of Asthma and Allergies in Childhood (ISAAC), and focuses on parent reported symptoms over time. Of the 190 articles, Islam et al. (2021) found that a diagnosis of asthma was based on asking parents if 'A child having ever had asthma ('asthma ever')' in 89 articles. Reported asthma was further broken down into ten separate definitions based on whether it was from parental reporting, healthcare professional diagnosis, or medical records. Children that experienced a recent asthma attack ('current asthma') were reported in 53 articles, and this classification had 25 different definitions. Clinically diagnosed asthma ('doctor-diagnosed asthma') was seen in 76 articles with five different definitions. There were other less common categories of asthma used in a small number of articles; 'diagnosed-asthma' in two articles; 'asthma-like syndrome' in three articles; 'probable asthma' in three articles; 'past asthmatics' in two articles; 'persistent asthma' in two articles; and 'possible asthma' in one article. In studies that were interested in wheezing, 'wheeze ever' was recorded when a child had experienced wheezing at some point throughout their life, and this was seen in 95 articles. The most common definition used for wheezing was 'current wheeze' which was used in 129 articles, and recorded whether a child had experienced wheezing within the previous 12 months. This could be broken down into eight differing definitions depending on wheezing frequency and additional symptoms. 'Exercise-induced wheeze' was recorded in 49 articles when a child experienced wheezing after partaking in physical activity. 'Persistent wheeze' was recorded in two articles, and 'infant asthma' was recorded in one article.

Asthma is a nebulous term and issues relating to the definition of the disease have been a common topic of research in the epidemiological field. With the definition varying between studies, the ISAAC initiative aimed to create a universal definition of childhood asthma through the use of its accessible questionnaire, although it remains a challenge (Dharmage et al., 2019). With some definitions being more sensitive, and others more specific, there has been misclassification of asthma (Dharmage et al., 2019). However, an exact definition may never be feasible as research is indicating that asthma is in fact an umbrella term for several similar diseases (Pavord et al., 2018). Therefore it is likely that asthma will continue to be a nebulous term with an ever-evolving definition as more is understood about the disease through further research.

2.7   The relationship between socio-economic status and respiratory health

Examining the impact of socio-economic status and respiratory health, Basagaña et al. (2004) used data from the European Community Respiratory Health Survey to explore the link between socio-economic status and asthma prevalence for young adults in 32 centres in 15 countries throughout Europe, the United States, Australia and New Zealand. The study found a range in asthma prevalence from 2.8% to 15.7%, giving an overall prevalence of 8.4%, with a larger prevalence seen in people of low social class and a low educational level. There was also evidence that regardless of an individual's socio-economic status, if they lived in a centre with a generally low education level, they still faced a higher risk of asthma. Possible explanations for the increased prevalence of asthma among the less affluent vary. Basagaña et al. (2004) hypothesised that early life events are possibly influential, such as the diet of the mother or the postnatal environment. A further explanation could be that poorer patients are likely to have poorly controlled asthma, owing to lack of concern or dismissal of symptoms (Ernst et al., 1995). Other factors that could be considered are lack of accessibility to healthcare and differences in both the prescription and use of asthma medication.

Basagaña et al. (2004) had taken universal healthcare into consideration and stated that it does not always equate to equal access nor equal utilisation. Their work found that in Spain, both the employed and unemployed visited physicians

with the same frequency, however those that were unemployed were less likely to be seen by a specialist. Further research found that prescription rates of inhaled steroids (Lang et al., 1997) and the proportion of people suffering from asthma receiving anti-inflammatory drugs were lowest amongst those with a lower socio-economic status. In contrast, Rona (2000) found that the lower socio-economic groups were prescribed more medication than the higher socio-economic groups.

In the UK Violato et al. (2009) examined the effects of household income on children's respiratory health using the UK Millennium Cohort Study and found a weak positive association between low income and childhood respiratory health, controlling for parental health, socio-economic status of grandparents and health impacts from maternal behaviour. One reason for such a weak association compared to other countries could be due to differences in health services. Indeed, the findings discussed in this section can all be related back to the concept of health inequalities, and the reasoning behind the findings can easily be linked to the materialist explanation provided for the social causation theory.

2.8 The association between air pollution, socio-economic status and health

The research discussed previously consisted of studies exploring the association between air pollution exposure and its negative impacts on health, as well as studies examining how people that are more disadvantaged face an increased burden of poor health when compared to those of higher socio-economic status. Bringing this research together, Fairburn et al. (2019) conducted a systematic review examining research that discussed social inequalities and air pollution exposure. The review found that those experiencing higher levels of deprivation and lower levels of socio-economic status were more likely to also experience high levels of pollutants, specifically PM_{10}, $PM_{2.5}$, NO_2 and NO_X.

The remainder of the chapter examines the relationship between exposure to air pollution, socio-economic status and health. Whilst cross-sectional analysis provides a valuable insight into the relationship between air pollution, socio-economic status and health, it is not without limitations. These studies cannot be used to address longitudinal issues as they provide no direct indication of the causal mechanisms that lie behind environmental inequalities (Richardson et al.,

2011). The studies also cannot account for the accumulation of exposure across life course (Richardson et al., 2013). As such, for ease of comparison the literature is divided into studies using a cross sectional and a longitudinal approach.

2.8.1 The association between air pollution, socio-economic status and health: Cross-sectional analysis

2.8.1.1 England

Wheeler and Ben-Shlomo (2005) utilised the Health Survey for England to explore the relationship between air quality and respiratory health, whilst examining how socio-economic status and exposure to air pollution also tie in with this relationship to further impact on respiratory health. This research found that those of a lower social class lived in areas that typically experienced worse air quality, an example of environmental inequality. This was not found to be the case in rural areas however as the more affluent were more likely to live near good transport links, such as main roads, meaning that those of a lower socio-economic status lived in areas that were increasingly remote, further from development and consequently, less polluted. An improved quality of air was also associated with better lung function in adults, although there was no pattern with asthma (Wheeler and Ben-Shlomo, 2005). The results did not prove that social class differences in respiratory function were explained by air pollution inequity. However, there was a weak suggestion that for men, poor air quality interacted with low social class, with double the impact compared to men in high social class households (Wheeler and Ben-Shlomo, 2005).

Briggs et al. (2008) investigated how environmental inequity varies in England with regard to different air pollutants, differing socio-economic status and different scales and contexts. Briggs et al. (2008) stated how there was a complex relationship at work, where poor health can lead to lower employment opportunities, lower income, restricted mobility and less access to power, thus increasing people's risk of worsening health. This relationship can be related to the social causation theory, specifically the materialist and psychosocial explanations (Foverskov and Holm, 2016). The pollutants examined in this study were NO_X, PM_{10}, SO_2 and total volatile organic compounds (VOC). The north of England experienced higher rates of air pollution and lower socio-economic

status, whilst inner city areas were also found to experience higher levels of air pollution as well as being more deprived. This investigation returned a significant positive association between health and income, employment and education, as well as a significant positive association between health and distance to emission sources, percentage of industrial land, proximity to airports and PM_{10} emissions, among other variables. Similar to the research carried out by Wheeler and Ben-Shlomo (2005), Briggs et al. (2008) found strong associations in urban, rather than rural areas. Briggs et al. (2008) also found evidence to suggest that deprivation could exacerbate the impacts of environmental exposures in some cases through increasing susceptibility to environmental factors, which could be due to already impaired health status and poorer access to health care.

Research in both England and the Netherlands carried out by Fecht et al. (2015) looked at the associations between air pollution, both PM_{10} and NO_2, and population characteristics, which were socio-economic status, ethnicity and the age profile at the neighbourhood level. There was an association between air pollution with both deprivation and ethnicity, meaning that people that had a lower socio-economic status and were an ethnic minority, were more likely to experience higher levels of air pollution.

Similar to previous research, Fecht et al. (2015) hypothesised that people with lower socio-economic status experienced elevated levels of air pollution in urban areas due to their home location, which was normally in close proximity to busy main roads and industrial sites. As previously stated, home location can be related back to socio-economic status and therefore the materialist explanation of the social causation theory, as an individual's income dictates where they can afford to live (Foverskov and Holm, 2016). With regard to the relationship between ethnicity and higher pollution levels, it was hypothesised that people of minority ethnicities may endure low quality of air to be closer to friends and family, as they tend to live congregated in the same neighbourhood, regardless of socio-economic status (Fecht et al., 2015).

2.8.1.2 Europe

Research in Rome (Forastiere et al., 2007) found that in urban areas, those with a higher socio-economic status were more likely to be living in areas of increased traffic emissions than those of a lower socio-economic status. However,

individuals with lower socio-economic status were still more likely to experience worse health than those of higher socio-economic status, even if they lived in less polluted areas. This could be due to differential susceptibility, however as this study looked only at road traffic emissions, it may not be representative of the actual air pollution concentration that an individual would experience in a given area. Forastiere et al. (2007) further stated that individuals that are less affluent may spend a longer proportion of their time outside, working on the street for example, therefore facing air pollution exposure for longer periods of time than someone who would work indoors. The wealthiest could also own second homes, away from the busy city centre, thus spending less time in the highly polluted area. Those of a lower social class also experienced higher rates of hospital admissions.

Schikowski et al. (2008) studied women in the Ruhr region in Germany examining how the combination of occupational exposures, outdoor air pollution and smoking would impact the socio-economic status and respiratory health relationship. This research found that women with a lower level of education were more likely to experience respiratory issues, such as reduced lung function. Schikowski et al. (2008) also found that long-term exposure to high levels of PM_{10} was significantly associated with reduced lung function. However, the relationship between poor respiratory health and low levels of education was lessened when adjusted for smoking and ambient air pollution.

A Europe-wide study conducted by Richardson et al. (2013) was interested in particulate air pollution and health inequalities, and the relationship with household income. Some of the richest areas in Western Europe were also the most polluted, similar to findings by Forastiere et al. (2007), although it was suggested that income-related inequalities in exposure to ambient air pollution may contribute to Europe-wide mortality inequalities. There was also evidence that people living in lower income regions were more susceptible to the health impacts of air pollution. Morelli et al. (2016) investigated the risk related to $PM_{2.5}$ exposure in the urban areas of Grenoble and Lyon in France, and looked at the relationship with social deprivation. This study included the number of full-term low birth weight cases which were attributable to air pollution, and carried out the investigation at a small scale. Areas of high deprivation experienced a greater

burden of PM$_{2.5}$ exposure on mortality, lung cancer and full-term low birth weight when compared to less deprived neighbourhoods.

2.8.1.3 Worldwide

Jerrett et al. (2004) conducted a study in Hamilton, Canada to test the hypothesis that socio-economic characteristics modify the acute health effects of ambient air pollution exposure. In a citywide model, increased mortality was found to be associated with air pollution exposure and also in intra-urban zones that had lower socio-economic characteristics. Weighted regression analysis suggested that underlying socio-economic characteristics modify the health effects of air pollution exposure (Jerrett et al., 2004). Three possible explanations were proposed to explain these findings. First, that people working in the manufacturing industry, like steel factories, face increased exposure to air pollution at their workplace, which, when combined with ambient air pollution exposure could have a greater impact on their health. Second, people with lower education were found to move around less, therefore experiencing lower exposure measurement error, reducing the bias toward the null. Third, working in manufacturing and educational levels serve as proxies for many social variables representing material deprivation, and poor material conditions increase an individual's susceptibility to health risks from air pollution. Another study in the Hamilton-Burlington area of Ontario explored how income, mortality and air pollution were related (Finkelstein et al., 2003). It was found that those living in low income areas had higher mortality rates than those living in higher income areas. Mean pollutant levels tended to be higher in areas with increased levels of deprivation, and both pollutant and income levels were associated with mortality differences. When compared to those living in areas with higher income and lower levels of pollution, all others in the study had a higher risk of death from non-accidental causes.

Richardson et al. (2011) investigated the association between exposure to PM$_{10}$ and mortality and health inequalities in New Zealand. A positive association was found between PM$_{10}$ exposure and respiratory disease mortality, as well as a socio-economic gradient. This means that those living with a low socio-economic status experience an increased risk of respiratory disease mortality. Socio-economic inequalities were found to be greater in the most polluted areas,

however this was not always statistically significant. Richardson et al. (2011) did not find that health inequalities were heightened in areas with increased exposure and instead stated that other socio-economic aspects were likely to have more of an impact than PM_{10} pollution, such as housing quality. Housing quality as justification for the inequalities experienced is an example of the materialist explanation of the social causation theory (Foverskov and Holm, 2016).

In China, Jiao et al. (2018) examined the non-linear relationship between area level air pollution and socio-economic status in urban area. This study found that as socio-economic status increased, so did levels of air pollution, similar to what was seen in Forastiere et al. (2007). This raises the suggestion that air pollution is a 'by-product' of economic development. However, this was only true up to a certain level because as socio-economic levels increased further, air pollution levels started to decrease. The study also found that the health effects associated with air pollution on people with lower socio-economic status were significantly greater when compared to people with a higher socio-economic status (Jiao et al., 2018).

2.8.2 The association between air pollution, socio-economic status and health: Longitudinal analysis

Hill et al. (2019) examined the impact of income inequality on the relationship between air pollution and life expectancy in the United States and found that states with higher levels of $PM_{2.5}$ pollution were more likely to exhibit a lower average life expectancy. This association was stronger in states with high levels of income inequality. A similar study conducted by Jorgenson et al. (2020) also examined the impact of $PM_{2.5}$ exposure on life expectancy in the United States, including both income inequality and racial composition in the study. The results found that air pollution exposure is more detrimental to life expectancy in areas with higher levels of income inequality and larger black populations. Jorgenson et al. (2021) conducted a study that investigated the effects of $PM_{2.5}$ pollution and income inequality on life expectancy across 136 nations. Again there was a negative association found between $PM_{2.5}$ pollution and average life expectancy which was amplified by increased levels of income inequality.

Whilst there is a wide range of longitudinal studies interested in air pollution epidemiology, there are a limited number that examine the impacts of the

association of air pollution and socio-economic status on health. Longitudinal studies that are interested in this relationship tend to focus on the effects faced by children, commonly following birth cohort studies. These are discussed further in Section 2.8.2.

2.9 The association between air pollution, socio-economic status and children's health

There is a growing body of literature that is interested in the effects of air pollution on the health of children, and how these effects are associated with socio-economic status. Rodriguez-Villamizar et al. (2016) conducted a systematic review to explore the evidence of socio-economic status as an effect modifier of the association between asthma exacerbations in children and ambient air pollution (Rodriguez-Villamizar et al., 2016). The studies included in this review displayed an association between hospitalisation and air pollution exposure, where a stronger effect was seen on children living in higher levels of deprivation. However, only one study confirmed the effect modification by statistically significant interactions between air pollutants and socio-economic status, most likely due to a limited sample size of the original studies (Rodriguez-Villamizar et al., 2016). This literature review continues by discussing the evidence base exploring the association between air pollution, socio-economic status and children's health. As above, the literature is divided into cross-sectional and longitudinal analysis. As noted, whilst cross-sectional analysis provides valuable insights on human health at one point in time, longitudinal studies are particularly valuable for child epidemiology. A focus on child health is particularly important with regard to air pollution, as research has demonstrated that as children develop and grow, they are more susceptible to the impacts of air pollution (Esposito et al., 2014).

2.9.1 The association between air pollution, socio-economic status and children's health: Cross-sectional analysis

2.9.1.1 England

Jephcote and Chen (2012) examined the hospitalisation of children aged up to 15 years with respiratory issues and how this was related to socio-economic status and exposure to vehicular PM_{10} emissions in the city of Leicester from

2000 to 2009. This study found that higher levels of PM_{10} emissions were related to an increase in respiratory related hospitalisations for children. This study was a precursor to a later investigation by Jephcote et al. (2014) who built on this research, again in Leicester, to study the spatial relationships between minor and severe respiratory conditions, including to what extent socio-environmental mechanisms were responsible for the worsening of respiratory health in children. The research indicated that exposure to poor socio-environmental factors could cause upper respiratory tract infection episodes in children, with continued exposure resulting in longer periods of recovery. In addition, if a child was not sufficiently recovered before temperatures began to decrease in colder months, the child faced the risk of infection with a virus, further worsening their respiratory health.

2.9.1.2 Worldwide

Ostro et al. (2001) studied how certain pollutants, including PM_{10}, $PM_{2.5}$ and NO_X, exacerbated the impacts of asthma in African-American children in Los Angeles. The research looked at the interaction between air pollution and asthma severity, socio-economic status and respiratory infections. A considerable number of the children included in this study were from families with a relatively low socio-economic status. Ostro et al. (2001) found that daily average PM_{10} concentration was associated with the probability and incidence of coughing, wheezing and shortness of breath. Exposure to $PM_{2.5}$ was found to produce similar effects as PM_{10}, albeit to a lesser magnitude. NO_X was also found to be associated with the daily probability of wheezing and with episodes of coughing and wheezing. Furthermore, asthma severity, income and the use of medicine was not found to significantly impact the association of PM_{10} with either daily probability of symptoms or the onset of episodes of coughing or wheezing.

In Seoul, Lee et al. (2006) looked at asthma related hospital admissions for children and how this was related to socio-economic status, analysing pollution data for pollutants that included PM_{10}, CO, NO_2 and O_3. The findings suggested that children living in areas with a low average socio-economic status were exposed to higher levels of NO_2 and CO, and that more children from these areas were admitted to hospital due to asthma than children from higher socio-economic status districts. The relative risk for O_3 was found to increase

significantly from higher to lower socio-economic status. Lee et al. (2006) goes on to list reasons explaining the increased pollutant susceptibility of children in lower socio-economic status areas; the pattern of environmental exposure to the pollutant; the child's health, influenced by exercise, diet and degree of socio-psychological stress; accessibility and provision of medical services; the surrounding physical and sociological environment.

The explanations provided for these findings have all been previously discussed in relation to the social causation theory (Foverskov and Holm, 2016). Bell et al. (2007) examined the relationship between air pollution, including PM_{10}, $PM_{2.5}$ and NO_2, and low birth weight in Connecticut and Massachusetts in the US. Low birth rate is indicative of health as it is associated with a higher risk of infant and childhood mortality and this research did find that there was an association between air pollution exposure and birth weight. However $PM_{2.5}$ was found to have a greater impact on the birth weight of babies born to black mothers, indicating a relationship with socio-economic status due to previous findings related to socio-economic status and ethnicity (Bell et al., 2007).

A study by Rosenlund et al. (2009) in Rome investigated the association between traffic-related pollution and lung function in schoolchildren. The air pollution indicators used were residential levels of NO_2, self-reported traffic level and proximity to busy roads. A strong association was found between estimated NO_2 exposure and decreased lung function, with stronger associations found in female children, older children, children of high socio-economic status and those exposed to smoking through their parents. The strong association seen with children of higher socio-economic status echoes the findings of Forastiere et al. (2007). Grineski et al. (2010) explored the relationship between race, ethnicity and health insurance status with air pollution, specifically NO_2, on the hospitalisation of children for asthma in Phoenix, Arizona in the US, from 2001 until 2003. This study found that there was an increased risk of admission to hospital with asthma for children without health insurance when compared to children with private health insurance. A lack of health insurance can be indicative of having a lower socio-economic status, and it was found that black and Hispanic children without health insurance faced a greater risk from air pollution when compared to white children with health insurance.

In Windsor, Canada, Cakmak et al. (2016) explored the association of schoolchildren's respiratory health with traffic type, traffic volume and air pollution stratifying by socio-economic status, based on household income and education at the household level. Increased traffic density within 200 metres of a child's house and increased air pollution were associated with increased respiratory symptoms and this association was stronger in areas of low income and low educational levels. This evidence shows that children living in a deprived area are more at risk of certain respiratory health problems due to a higher volume of traffic and consequently increased air pollution exposure. In up to 62% of cases the differences between high and low socio-economic groups were statistically significant, indicating that socio-economic status was a significant effect modifier.

Kravitz-Wirtz et al. (2018) examined early life exposure to air pollution, area level poverty and asthma risk in children in the US. The study theorised why children living in the most deprived areas would face increased exposure and be more susceptible to air pollution for a number of reasons. Poor healthcare and a lack of nutritious foods, as well as increased exposures to psychosocial stressors such as violence were all listed. The association between pollution exposure and asthma was found to be significant for children living in areas of high poverty.

2.9.2 The association between air pollution, socio-economic status and children's health: Longitudinal analysis

Gauderman et al. (2007) studied the impact that traffic-related air pollution had on children's lung development in California over eight years. The results showed that children living within 0.5 km of a freeway had a decreased lung capacity when compared to children that lived at least 1.5 km away from a freeway. This indicated that the increased exposure faced by children living closer to a freeway had negative impacts on their health. The study found that low socio-economic status was associated with increased exposure to traffic-related emissions, with those that were more deprived more likely to live closer to a freeway. However there was no significant association between socio-economic status and forced expiratory volume or lung-function growth. Clougherty et al. (2007) studied the influence of violence exposure as a stressor that influenced asthma rates among children in Boston. As stated by Foverskov and Holm (2016), exposure to stress has the potential to be a primary pathway through which socio-economic status

impacts health as explained through the psychosocial explanation of the social causation theory. The study found an association between NO_2 exposure from traffic emissions and asthma diagnosis only among children that were exposed to a high level of continued violence.

In Stockholm, Sweden, Nordling et al. (2008) investigated the impact that air pollution from transportation had on the respiratory system of children over four years. This study found a positive association between traffic-related air pollution, in this case PM_{10} and NO_X, during the first year of the children's lives and indicators of airway disease in the same children when they were four years of age. These indicators were wheezing, a lower lung output and pollen sensitivity. The children with the highest socio-economic status were found to be exposed to the most air pollution as they tended to live in the inner city where there was a higher concentration of traffic. Chang et al. (2009) was interested in repeated hospital visits in California for children with asthma and the association with residential proximity to busy roads. There was a positive association between distance to roads and freeways and repeated hospital visits, as those living within 0.3 km of main roads were more likely to return to the hospital with respiratory issues. There was also a stronger association found for children without private health insurance, again this is an indicator of having a lower socio-economic status. Shankardass et al. (2009) explored childhood asthma incidence in relation to high parental stress or low socio-economic status and traffic-related air pollution. This study looked at children between the ages of five and nine from California that did not suffer from asthma or wheezing, and followed up over three years to discover if any participant had been newly diagnosed with asthma. The study found that there was a significantly increased risk of developing asthma for children with high parental stress when compared to those with low parental stress. Stress was also associated with effects of in utero tobacco smoke. Furthermore, there was also an increased risk of asthma for children from more deprived families, black children and underweight children. This suggests that children from households of high stress were more susceptible to the impacts of air pollution.

2.10 Conclusion

Air pollution and its negative impact on human health is well established; however, literature that also includes the impact of socio-economic status at both individual and area levels has been limited to date. The evidence outlined in this Literature Review indicate that there is an association between socio-economic status and air pollution, one which can have a lasting impact on an individual's health throughout their life. The theories that underpin this relationship have been discussed and evidence has supported this framework.

Whilst findings are inconsistent, this inconsistency demonstrates the need for future analysis to fully understand the complex relationships at play. Within this context, this thesis aims to explore the association between air pollution, socio-economic status and respiratory health in children through answering the following hypothesis;

1. Whether the association between asthma and air pollution is stronger amongst children of lower, compared to higher, socio-economic status, and;
2. Whether area level deprivation interacts with individual socio-economic status so that the impact of air pollution exposure on asthma is stronger for children with low socio-economic status living in the most deprived areas than children with similar socio-economic status living in less deprived areas.

The next chapter, Chapter 3, outlines the data and methods used in this study that seeks to understand the above hypothesis.

Chapter 3. Data and Methods

3.1   Introduction

This chapter introduces the data and research design and methods used to explore the relationships between respiratory health in children, individual and area level socio-economic status and air pollution exposure. As noted in the Introduction (Chapter 1), this thesis specifically seeks to address the following hypotheses;

1. The association between respiratory health and air pollution is stronger amongst individuals of lower, compared to higher, socio-economic status.
2. Area level deprivation will interact with individual socio-economic status so that the impact of pollution on respiratory health is stronger for people with low socio-economic status living in the most deprived areas than people with low socio-economic status living in less deprived areas.

This chapter will begin by examining the different data sources used in this thesis and the associated variables each data source provides. These data sources include; the Millennium Cohort Study (MCS) (outlined in section 3.2.1), the European Monitoring and Evaluation Programme for the UK (EMEP4UK) (outlined in section 3.2.2) and the Index of Multiple Deprivation (IMD) (outlined in section 3.2.3).

To create the necessary dataset to answer the hypotheses outlined above, data linkage was necessary to compile all relevant data into one dataset. Section 3.4 outlines the data linkage methodology used. Section 3.5 introduces the statistical methods used to analyse the research question; cross-sectional analysis is the first analytical method that will be discussed, followed by time series analysis and finally multilevel modelling. Each analytical method aims to build on the previous to better understand the relationships at play.

3.2   Secondary Data Sources

3.2.1 Millennium Cohort Study

The Millennium Cohort Study (MCS) is a longitudinal study that is conducted by the Centre for Longitudinal Studies (CLS) at the Institute of Education, University of London (Plewis et al., 2007). This study aims to follow children born in the UK around the turn of the millennium throughout their lives. The study is funded by the Economic and Social Research Council (ESRC), as well as a selection of UK government departments, the Welsh Government, the Scottish Government and the Northern Irish Executive. The study seeks to provide the basis for comparison with previous cohort studies and to facilitate international comparative research (Connelly and Platt, 2014). Furthermore, the study allows the in-depth analysis of the inequalities faced by a contemporary cohort of individuals throughout their life course. The research design for the MCS was based on the following five principles (Plewis et al., 2007);

1. "The MCS should provide data about children living and growing up in the four countries of the UK."
2. "The MCS should provide usable data for sub-groups of children, in particular those living in advantaged and disadvantaged circumstances, and for children of ethnic minorities and those living in Scotland, Wales and Northern Ireland."
3. "As well as data about children, the study should provide data about their family circumstances and the broader socio-economic context in which the children grow up."
4. "The MCS should include children born throughout a single 12-month period."
5. "All children born as members of the MCS population should have a known and non-zero probability of being included in the selected sample."

The study comprises children from England and Wales that were born between 1 September 2000 and 31 August 2001, and children from Scotland and Northern Ireland that were born between 24 November 2000 and 11 January 2002. These children had to be living in the UK when they were nine months old and their families would also have had to be eligible to receive Child Benefit (Plewis et al.,

2007). To be eligible to receive Child Benefit, one must be responsible for a child under the age of 16, or older depending on different factors, and must be living in the UK. In 2001, 6.02 million families in England were eligible for Child Benefit (Sorensen, 2002).

As per the principles, the study was designed to accurately reflect the total population whilst also being representative of key sub-groups, thus the study oversampled children from deprived backgrounds. This allowed the effects of disadvantage on children's outcomes to be better addressed. Areas that featured a relatively high ethnic minority concentration were also oversampled to reflect the increasing diversity of the UK, and to examine the different health, educational and social outcomes across ethnic groups.

Due to these demands, the population was stratified. Specifically in England, the population fit into three strata (Plewis et al., 2007);

1. An 'ethnic minority' stratum where the proportion of ethnic minorities in that ward in the 1991 Census was at least 30%.
2. A 'disadvantaged' stratum which comprised of children living in wards that were in the poorest 25% using the Child Poverty Index for England and Wales (excluding wards falling into the ethnic minority stratum).
3. An 'advantaged' stratum, capturing children living in wards other than those in the other two strata.

The sample is clustered by characteristics of electoral wards and randomly selected within each stratum which produced a disproportionately stratified cluster sample. Following this, a list of all children that would turn nine months old during the survey period, that lived in a selected ward, and that were entitled to Child Benefit were written to, with an opt out option if they did not wish to be included in the survey. Just over half (51%) of the children surveyed in the first wave were male and 82% were White. Around 2.5% were Indian, 4.8% were Pakistani, 2% were Bangladeshi, 1.3% were Black Caribbean, 2% were Black African and 3% had mixed ethnicity (Plewis et al., 2007).

The children in the MCS were first surveyed when they were aged nine months (Wave 1) and follow up surveys took place at ages 3 (Wave 2), 5 (Wave 3), 7 (Wave 4) and 11 (Wave 5) (Connelly and Platt, 2014). Further follow up occurred

at ages 14 (Wave 6) and 17 (Wave 7), but only the first five sweeps are used in this thesis.

Table 3.1 depicts the number of wards sampled, the target number of responses and the actual received number of responses in Wave 1. There were a number of families with twins and triplets, and a small amount with multiple cohort members due to two separate pregnancies during the eligibility period.

Table 3.1 Total target and achieved responses for the MCS in Wave 1 in the UK

	Wards sampled	Target responses	Achieved responses	
			Children	(Families)
England	200	13,146	11,695	(11,533)
Wales	73	3,000	2,798	(2,760)
Scotland	62	2,500	2,370	(2,336)
Northern Ireland	63	2,000	1,955	(1,923)
Total	398	20,646	18,818	(18,552)

Table 3.2 depicts the total number of children sampled at each wave, focussing on the total number of children in England that were surveyed. The study has experienced attrition, re-entry and late entrants, as well as non-response due to refusal, non-contact, emigration or death.

Table 3.2 Total number of families and children that participated in the MCS in Waves 1 through 5

Wave	Year	Age	Total no. of families	Total no. of children	No. of children in England
1	2001	9 months	18,552	18,818	11,695
2	2004	3	15,590	15,808	10,188
3	2006	5	15,246	15,460	9,884
4	2008	7	13,857	14,043	8,955
5	2012	11	13,287	13,469	8,618

As stated, the sample surveyed was clustered geographically and disproportionately stratified to over-represent areas with high proportions of ethnic minorities in England, residents of areas of high child poverty and residents of the three smaller countries of the UK respectively (Plewis et al., 2007). Due to this, sample design weights or probability weights are used to correct for MCS cases having unequal probabilities of selection that result from the stratified cluster sample design. These weights are included in the data.

The Millennium Cohort Study is multidisciplinary and records a range of information related to the experiences and lives of the children and their families, as well as information related to their surrounding environment or neighbourhood. As the study is longitudinal, there are repeated measures for a number of topics, such as health variables, and data focusses on different stages of development over the child's life course. The study examines topics such as income, housing type, parental education and employment, cognitive development, school choice and physical growth, among others (Connelly and Platt, 2014). As well as the main parent interview survey, each wave has a selection of other interviews and measurements taken, for example, second co-resident parent interview, sibling interview, teacher interview, cohort member measurements, cohort member assessments and self-completed activities. Further enhancement studies have also been carried out, such as oral fluid examination, direct measures of physical activity using accelerometers, and through the collection of baby teeth. Sub-studies of the cohort have also occurred, which included a postal survey for health workers working in the sample areas and pre-school research carried out in a small sample (Connelly and Platt, 2014). The data collected has been further enhanced through linkage with administrative records, such as hospital and educational records. This linkage further improves the usefulness of the data for research and analysis. Due to the extensive range of data available with the MCS, it has been used in many different studies, including epidemiological studies. Jayaweera and Quigley (2010) used the MCS to investigate how ethnic minorities' access and use healthcare, and Hawkins et al. (2008) explored the links between maternal employment and childhood weight gain.

3.2.1.1   UKDS Secure Lab

As the data contained in the MCS includes sensitive information regarding the area that cohort members live in, access is only granted through the UK Data Service's Secure Lab. The Secure Lab provides remote access to sensitive or confidential data, such as geographical data, in a controlled and safe environment. Data cannot be downloaded from the Secure Lab, although results can be released, following a statistical disclosure control process that ensures no potentially identifiable information is included. Training is necessary in order to access the Secure Lab.

3.2.2 EMEP4UK – European Monitoring and Evaluation Programme for the UK

Air pollution data was produced from the EMEP4UK model, made available through the UK Centre for Ecology & Hydrology. The EMEP4UK model is a nested regional atmospheric chemistry transport model (ACTM) based on the main EMEP MSC-W model (Vieno et al., 2014, Simpson et al., 2012). The EMEP4UK model is driven by the Weather Research Forecast (WRF) model and the horizontal resolution scales down from 50 km x 50 km in the main EMEP 'Greater European' domain to 5 km x 5 km for the domain covering the British Isles. The boundary conditions for the British Isles domain are derived from the results of the European domain in a one-way nested setup.

The EMEP4UK model is capable of representing the UK's hourly atmospheric conditions at a horizontal scale ranging from 100 km to 1 km. The model simulates hourly to annual average atmospheric composition and deposition of various pollutants; including PM_{10}, $PM_{2.5}$, secondary organic aerosols, elemental carbon, and secondary inorganic aerosols, SO_2, NH_3, NO_X and O_3 (Vieno et al., 2016). Additionally, dry and wet deposition of pollutants are routinely calculated by the model. The EMEP4UK model output is compared with observational data from over 180 sites from the Automatic Urban and Rural Network (AURN network) throughout the British Isles (Lin et al., 2017).

Data from the EMEP4UK model have previously been used in epidemiological research, for example Graham et al. (2020) examined the impact that weather has on particulate matter and human health. Doherty et al. (2009) used EMEP4UK to examine the impact that heatwave episodes may have on O_3 levels, and consequently mortality rates.

3.2.3 Index of Multiple Deprivation (2010)

Data describing the area level socio-economic status of cohort members is available through the Index of Multiple Deprivation (IMD), which is derived from the Indices of Deprivation (IoD) (Ministry of Housing and Government, 2011). The IoD are measures of relative deprivation at the Lower-layer Super Output Area (LSOA) level across England and are produced by the Ministry of Housing, Communities and Local Government (McLennan et al., 2011). The IoD provides

a set of relative measures of deprivation for small areas across England, based on the seven different domains of deprivation, which are; Income deprivation; Employment deprivation; Education, skills and training deprivation; Health deprivation and disability; Crime; Barriers to housing and services; Living environment deprivation.

The combination of information from the different domains produces an overall relative measure of deprivation, and this is the Index of Multiple Deprivation (IMD). The IMD is the official measure of relative deprivation for small areas in England, each with a population of roughly 1,500, and ranks every LSOA in England from the most deprived area (1) to least deprived area (32,844). LSOAs are similarly given a score, with the LSOA with the highest score being the most deprived (McLennan et al., 2011). The IMD is a combination of the seven domains that produce an overall relative measure of deprivation.

Each domain is weighted differently, which has been derived from academic literature on poverty and deprivations, as well as the levels of robustness of the indicators: Income deprivation (22.5%); Employment deprivation (22.5%); Education, skills and training deprivation (13.5%); Health deprivation and disability (13.5%); Crime (9.3%); Barriers to housing and services (9.3%); Living environment deprivation (9.3%) (McLennan et al., 2011). The IMD was first recorded in 2000, with following versions recorded in 2004, 2007, 2010, 2015 and 2019, and is available for download from the UK government's website. It was decided that IMD 2010 was suitable to be used for this study as, whist there is some temporal variation, deprivation rates remain broadly consistent. Indeed, 88% of the most deprived LSOAs in IMD 2010 were also among the most deprived LSOAs in IMD 2007. In addition, 83% of the most deprived LSOAs in IMD 2015 were among the most deprived in IMD 2010 also (Lad, 2011).

Figure 1 shows the national distribution of the IMD 2010 in England and illustrates how most city centres contain areas with high levels of deprivation. Of the 326 local authorities in England, 56% contain at least one LSOA which is among the most deprived in the country (Lad, 2011).
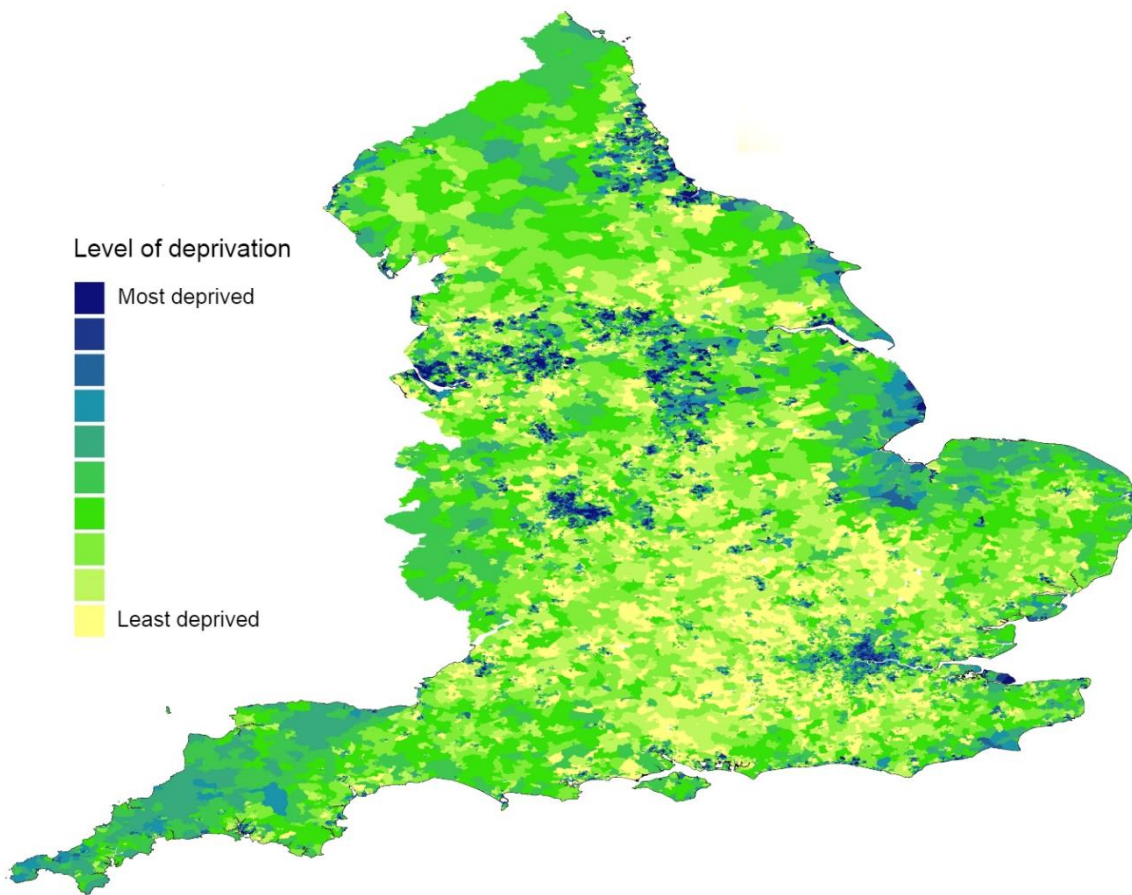
Figure 3.1   The LSOAs of England classified by level of deprivation (using IMD 2010 data from Ministry of Housing and Government (2011)

The IMD has been utilised in many epidemiological studies, and the impact of deprivation on health has been well documented (Hawley et al., 2013, Jordan et al., 2004). Deprivation at the area level has been shown to be associated with worse health, increased comorbidity levels (Morrissey et al., 2016) and issues related to healthcare access and use (Kontopantelis et al., 2018). With regard to respiratory health, Gupta et al. (2018) investigated asthma mortality, hospital admissions and prevalence and how they varied with socio-economic status in England, and found that as average IMD score increased, so too did emergency asthma admissions rate per 100,000.

3.2.4 Ethics

Ethical approval for this research was granted by Chair's Action from the University of Exeter College of Medicine and Health Research Ethics Committee (application reference number 18/02/159). A UK Data Service SURE (Safe User

of Research data Environments) training course was attended on 13/06/2018. This training course was necessary to access the MCS data through the UK Data Service Secure Lab, due to the small area location information being sensitive, secure and potentially disclosive. Access was then carried out through remote access from the data user's organisational computer and was carried out in a secure setting.

3.3 Variables

This Section outlines both the outcome and explanatory variables used throughout the thesis.

3.3.1 Outcome variables - measures of respiratory health

The Millennium Cohort Study recorded information regarding different health conditions that cohort members experienced (Plewis et al., 2007), as well as health conditions their family members experienced.

Health related questions were consistent from Wave 2 onwards, however in Wave 1 when the cohort members were nine months old, slightly different questions were used. Beginning in Wave 2, questions regarding respiratory health were taken from the International Study of Asthma and Allergies in Childhood (ISAAC) core questionnaire for asthma (Asher and Weiland, 1998), available in Appendix A. This validated questionnaire has been used in many studies to measure child respiratory health (Al Ghobain et al., 2012, Ocampo et al., 2017, Lee, 2010). The cohort member's parent or guardian were asked a series of questions regarding their respiratory health, and the outcomes of interest in this study are 'ever had asthma' (asthma) and 'had wheezing in the last 12 months' (wheeze).

3.3.1.1 The International Study of Asthma and Allergies in Children (ISAAC)

The International Study of Asthma and Allergies in Children (ISAAC) was developed with three main aims;

- To understand the prevalence and severity of asthma, rhinitis and eczema in children worldwide, allowing for comparisons between countries.

- To obtain baseline measures for assessment of future trends in the prevalence and severity of the diseases.

- Provide a framework for future aetiological research into factors affecting these diseases.

The study consisted of three separate phases, in which Phase One consisted of assessing the prevalence and severity of asthma and allergic diseases in specific populations using core questionnaires. The use of a standardised core questionnaire allowed for comparisons to be drawn about the epidemiology of asthma, among other allergic diseases, between different populations (Asher and Weiland, 1998). A sample of the ISAAC questionnaire discussed here can be found in Appendix A. The two questions featured in the ISAAC questionnaire that directly relate to this study were:

- Has your child ever had asthma?   [Yes] [No]

- Has your child had wheezing or whistling in the chest in the last 12 months?   [Yes] [No]

Responses to these questions are related to 'asthma ever' and 'current wheezing' respectively, and report the prevalence of asthma and current wheezing among the cohort of interest. The questionnaire is completed by the parent or guardian of the children, and is interested in the child's self-reported history of asthma and wheezing, therefore a clinical diagnosis was not necessary. This is one reason ISAAC has been so widely used in epidemiological studies interested in respiratory health in children, as it is readily accessible and simple to incorporate.

Based off of the above questions giving responses about 'asthma ever' and 'current wheezing', the questions used in the Millennium Cohort Study that provide the outcome variables for this study are;

- Has [^Cohort child's name] ever had asthma?   [Yes] [No]

- Has [^Cohort child's name] had wheezing or whistling in the chest in the last 12 months?   [Yes] [No]

A sample of the questionnaire used in the MCS is available in Appendix B. Wave 2 was the first year that the ISAAC questionnaire was used in the MCS, and so responses in Wave 1 are not uniform with the subsequent waves. The MCS has

been frequently used to investigate asthma and wheeze in children in the UK, for example to measure sex discordance in asthma (Arathimos et al., 2017), to examine asthma in children born following infertility treatment (Carson et al., 2013), and to explore ethnic variation in asthma and wheezing (Panico et al., 2007). Many studies have utilised the 'asthma ever' and 'current wheeze' when examining the prevalence of asthma and wheeze in children (Arathimos et al., 2017, Carson et al., 2013, Panico et al., 2007), whilst others have focussed specifically on wheezing (Taylor-Robinson et al., 2016, Griffiths et al., 2018, Quigley et al., 2018) and other allergies, such as eczema (Panico et al., 2014), or specifically on asthma (Kelly et al., 2019) and other allergies (Henderson and Quenby, 2021). Some studies, such as the one conducted by Pike et al. (2019) combined 'asthma ever' and 'current wheeze' to create the variable 'current asthma'. From the available research, 'asthma ever' and 'current wheeze' are recurrently used in epidemiological studies about asthma, therefore these terms are a good choice for this study.

3.3.1.2   Asthma – 'asthma ever'

As discussed in Section 1.2, asthma is a chronic respiratory condition that effects around 6 to 8% of children in the UK (Bloom et al., 2019), with symptoms that include wheezing, loss of breath, coughing and chest tightness, all of which are indicative of a limitation of airflow in the respiratory system. Of the health conditions surveyed in the MCS, asthma was the condition that best represented the respiratory health of the cohort members. As stated, the questionnaire used in Wave 1 featured different questions from future waves, and the parent or guardian of the cohort member was originally asked;

- We would like to know about any health problems for which [^Cohort child name] has been taken to the GP, Health Centre or Health Visitor, or to Casualty, or you have called NHS direct.

- What ^was this problem?

    o Wheezing or asthma   [Yes] [No]

The responses to this question covered a wide array of health conditions and responses to this question were recorded as a categorical variable (yes, no, refusal, don't know, not applicable). Responses were recoded as a binary

variable to record that the child had asthma or not (1 = yes, child has had asthma, 0 = no, child has not had asthma). However as previously mentioned there are difficulties surrounding the diagnosis of asthma in young children (Bush, 2007), and proof of clinical diagnosis was not required when answering. Thus the responses to this question are self-reported and open to the interpretation of the parent respondent.

As discussed in Section 3.3.1.1, starting in Wave 2 questions were taken from the ISAAC core questionnaire for asthma, and the parent or guardian of the cohort member was asked;

- Has [^Cohort child's name] ever had asthma?   [Yes] [No]

and again responses were recorded as a categorical variable (yes, no, refusal, don't know, not applicable), which was then recoded as a categorical variable and then recoded into a binary variable (1 = yes, child has had asthma, 0 = no, child has not had asthma) during data preparation (University of London, 2021g, University of London, 2021h, University of London, 2021b, University of London, 2021a). This variable was named 'asthma' across all waves.

3.3.1.3   Wheeze – 'current wheeze'

Given the difficulties faced when attempting to diagnose asthma in children, especially those aged under five years (Moral et al., 2019), and as a confirmed clinical diagnosis was not required, a child having experienced a wheezing episode in the previous 12 months was also considered to be an appropriate indicator of the cohort member's respiratory health, in addition to the knowledge that wheezing is a symptom for asthma. Current wheeze has been used in many epidemiological studies that have examined asthma in children.

The same question seen in Section 3.3.1.2 for asthma in Wave 1 was also used for determining wheezing prevalence as there were no other suitable responses. Beginning in Wave 2, when questions were taken from the ISAAC core questionnaire, the parent or guardian of the cohort member was asked specifically about wheezing. They were first asked if their child had ever had a wheezing episode in their life. If they responded 'yes', they were then asked about wheezing episodes within the previous 12 months;

- Has [^Cohort child's name] ever had wheezing or whistling in the chest at any time in the past? [Yes] [No]

    o Has [^Cohort child's name] had wheezing or whistling in the chest in the last 12 months? [Yes] [No]

This was available as a categorical variable, and during data preparation for this study, was recoded into a binary variable (1 = yes, child has wheezed in the previous 12 months, 0 = no, child has not wheezed in the previous 12 months). This variable was named 'wheeze' across all waves.

3.3.2 Exposure of interest - measures of air pollution

The EMEP4UK model provided annual average surface concentrations at a resolution of 5 km^2 for PM_{10}, $PM_{2.5}$, NO_2, NO and O_3 (Vieno et al., 2016). As the pollutants were presented as annual average concentrations, it was decided that a resolution of 5 km^2 was sufficient as there would not be a significant variation in concentration as a smaller resolution. Additionally, the mean size of a LSOA is 4 km^2 (Mitchell and Popham, 2007), which further supports 5 km^2 as an appropriate scale of data.

The choice of pollutants was based on previous research that demonstrated these five pollutants have the greatest influence on respiratory health (Chen and Kan, 2008). Figures 3.2 – 3.6 show the annual average surface concentration of each pollutant for the five years in question. For the purpose of this research, analyses are divided into two:

(i) analysis that include all five pollutants – multiple exposure models; and

(ii) analysis that only use NO_2 as an explanatory variable – single exposure models.

Running two separate pollutant specifications was necessary as correlation between air pollutants is a well-established modelling issue in air pollutant and human health research (Koenig, 1999). Indeed, a correlation analysis using the MCS data at the individual level following linkage with pollution data found that NO_2 is heavily correlated with NO and O_3, and is also correlated with PM_{10}, and $PM_{2.5}$. $PM_{2.5}$ and PM_{10} were heavily correlated, as was NO and O_3. NO was also

correlated with PM_{10} and $PM_{2.5}$. Table 3.3 presents the results from the test for collinearity.

Thus, in the interest of overcoming modelling difficulties, especially multicollinearity, it was decided that it would be useful to focus solely on one pollutant for certain models. Therefore, as NO_2 is indicative of ambient air pollution and also road traffic levels through association, NO_2 only models were run.

As was the case for IMD, quartiles were also created for the pollutants, ranging from most to least polluted areas. Again, other quartile variables were created for each pollutant that recorded the initial concentration that the cohort members were exposed to in Wave 1.

Table 3.3 Test for multicollinearity (continued overleaf)

	Asthma	Wheezing	Child is female	Child is white British	Child is obese	Mother is employed	Mother has asthma	Mother smokes
Asthma	1							
Wheezing	0.45	1						
Child is female	-0.05	-0.05	1					
Child is white British	0.01	0.01	0.00	1				
Child is obese	0.03	0.03	0.02	-0.05	1			
Maternal employment	-0.02	-0.02	0.00	0.22	-0.02	1		
Maternal asthma	0.14	0.10	0.02	0.10	0.01	-0.01	1	
Maternal smoking	0.05	0.04	-0.02	0.14	0.03	-0.11	0.07	1
Lives below poverty line	0.05	0.02	0.00	-0.29	0.04	-0.48	0.02	0.18
Lives in social housing	0.07	0.03	0.00	-0.10	0.06	-0.29	0.06	0.27
Lives in urban area	0.03	0.03	0.00	-0.20	0.03	-0.09	0.00	0.07
IMD Score	0.05	0.03	0.00	-0.34	0.07	-0.31	0.01	0.16
NO_2 concentration	-0.02	0.01	0.00	-0.46	0.04	-0.17	-0.05	-0.03
PM_{10} concentration	-0.04	0.04	0.00	-0.26	0.02	-0.14	-0.02	-0.01
$PM_{2.5}$ concentration	-0.04	0.04	-0.01	-0.26	0.01	-0.14	-0.02	-0.01
NO concentration	-0.02	0.01	0.00	-0.41	0.04	-0.15	-0.04	-0.04
O_3 concentration	0.01	0.00	0.00	0.41	-0.03	0.13	0.04	0.04

Table 3.3 Test for multicollinearity (continued)

	Lives below poverty line	Lives in social housing	Lives in urban area	IMD score	NO$_2$ conc.	PM$_{10}$ conc.	PM$_{2.5}$ conc.	NO conc.	O$_3$ conc.
Lives below poverty line	1								
Lives in social housing	0.44	1							
Lives in urban area	0.16	0.14	1						
IMD Score	0.47	0.39	0.29	1					
NO$_2$ concentration	0.20	0.20	0.40	0.34	1				
PM$_{10}$ concentration	0.09	0.13	0.24	0.12	0.69	1			
PM$_{2.5}$ concentration	0.08	0.11	0.22	0.10	0.69	0.95	1		
NO concentration	0.16	0.19	0.29	0.26	0.93	0.69	0.69	1	
O$_3$ concentration	-0.16	-0.17	-0.32	-0.28	-0.91	-0.48	-0.56	-0.85	1
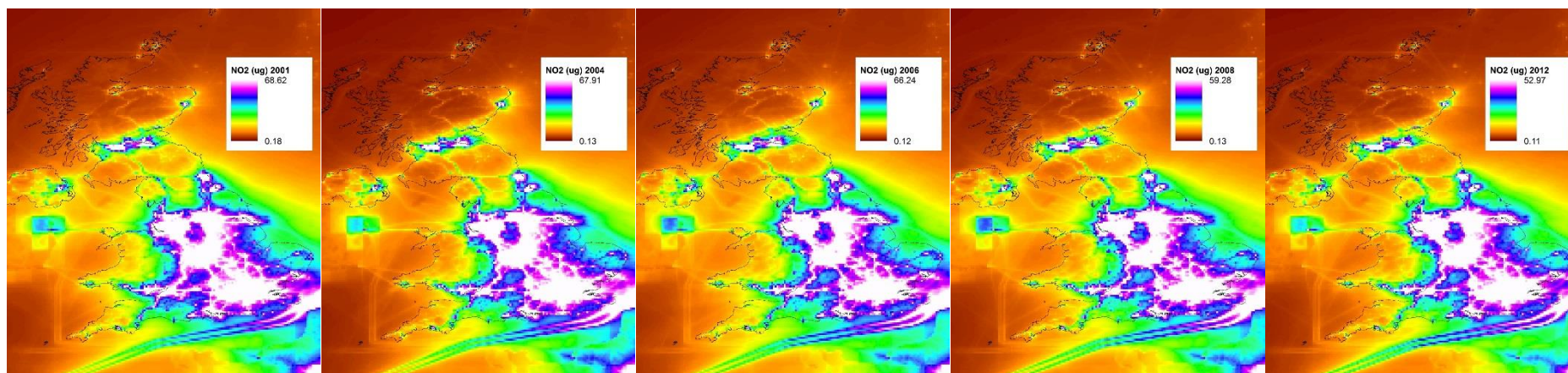
Figure 3.2 Annual average surface concentration of NO₂ (μg) in 2001, 2004, 2006, 2008 and 2012.
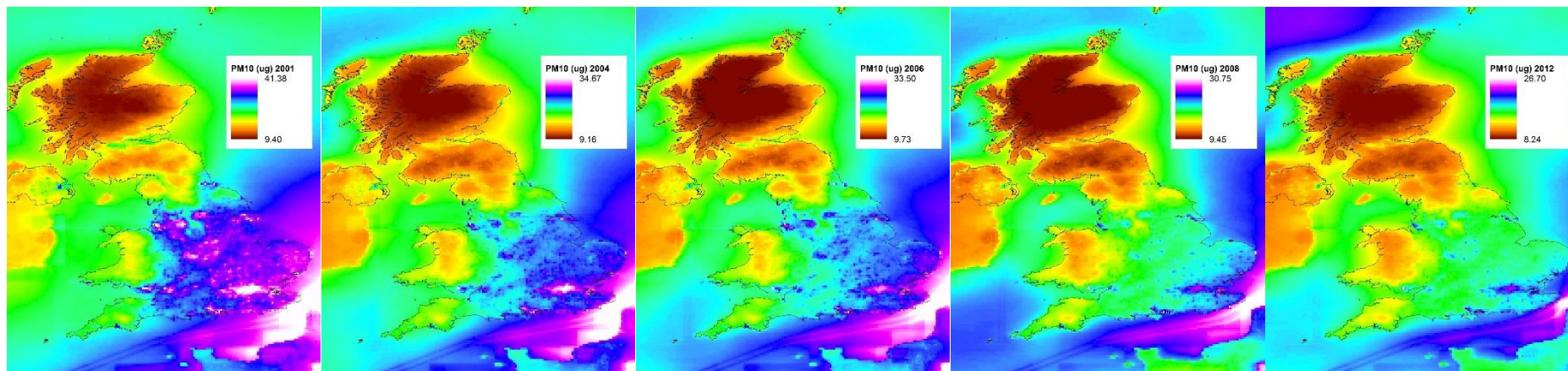
Figure 3.3 Annual average surface concentration of PM₁₀ (μg) in 2001, 2004, 2006, 2008 and 2012.
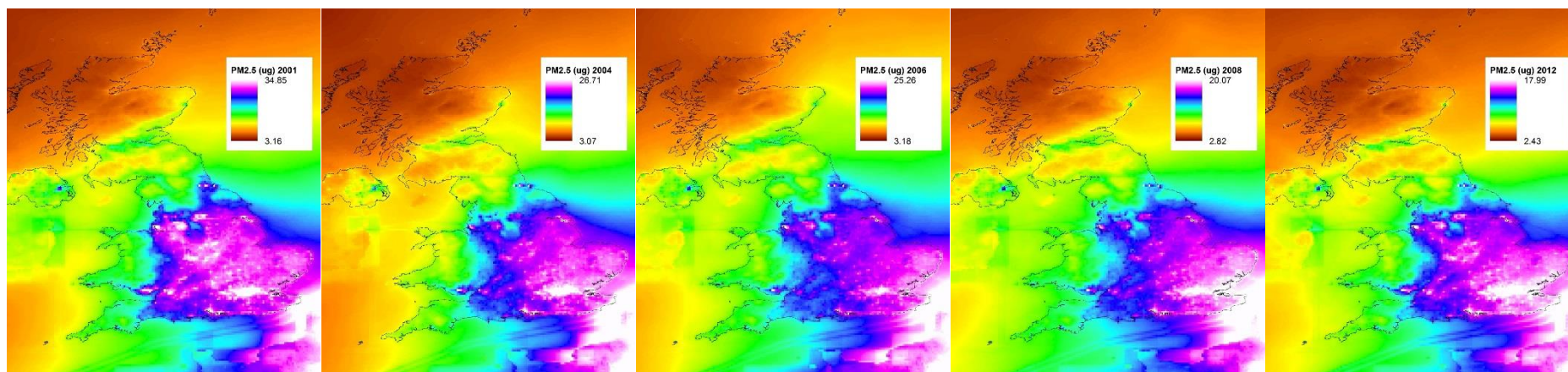
Figure 3.4   Annual average surface concentration of PM$_{2.5}$ (µg) in 2001, 2004, 2006, 2008 and 2012.
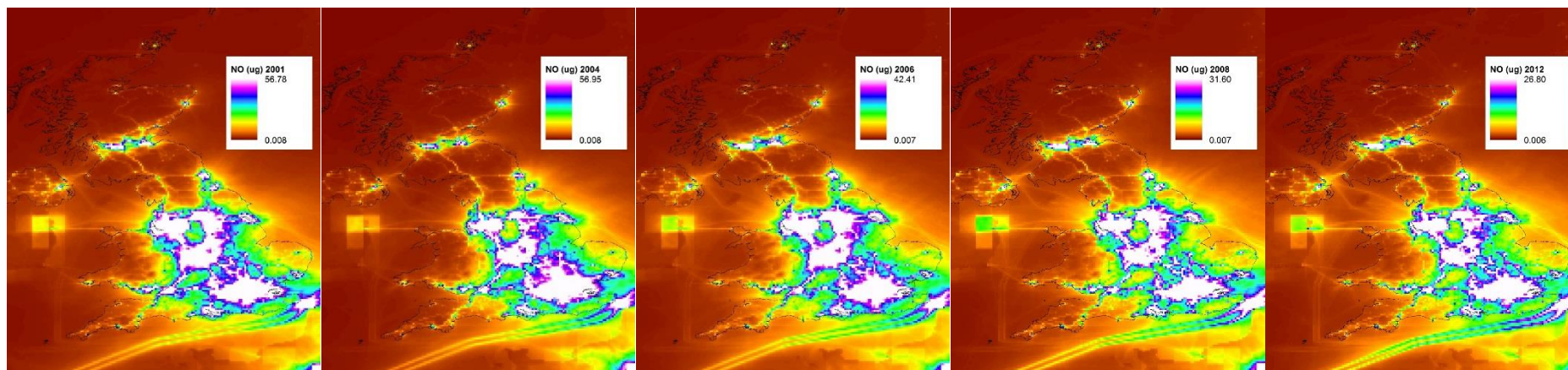
Figure 3.5   Annual average surface concentration of NO (µg) in 2001, 2004, 2006, 2008 and 2012.

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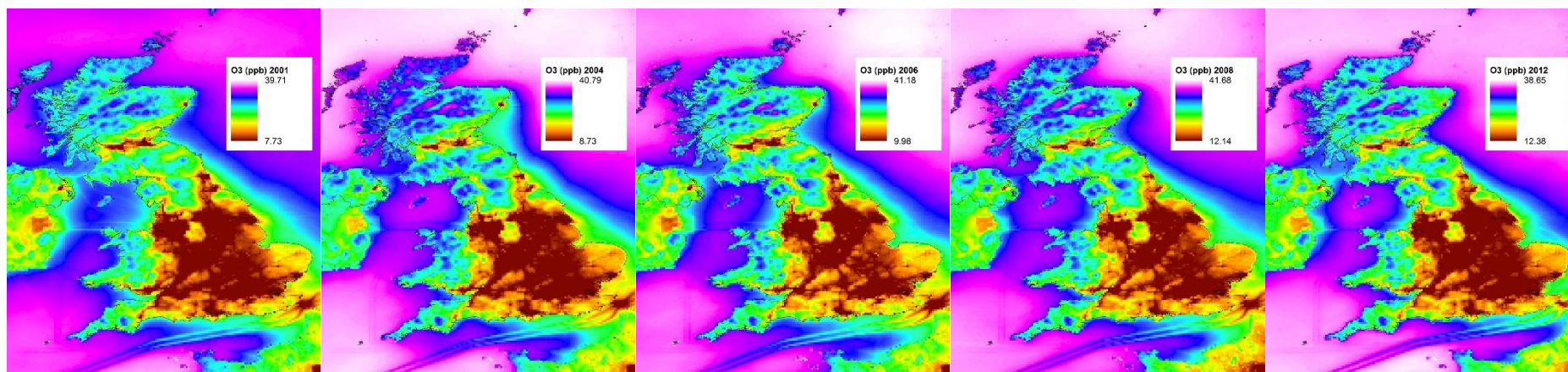
Figure 3.6   Annual average surface concentration of O₃ (μg) in 2001, 2004, 2006, 2008 and 2012.

3.3.3 Key confounders - measures of socio-economic status

Previous research has shown that socio-economic status is intrinsically linked with human health (Kontopantelis et al., 2018, Gupta et al., 2018). To address the hypotheses of this thesis, information on socio-economic status is required at both the family level, available from the MCS, and area level, available from the IMD. Familial (individual) socio-economic status is required to answer the first hypothesis; *the association between respiratory health and air pollution is stronger amongst individuals of lower, compared to higher, socio-economic status,* whilst both individual and area level socio-economic status are necessary to answer the second hypothesis; *area level deprivation will interact with individual socio-economic status so that the impact of pollution on respiratory health is stronger for children with low socio-economic status living in the most deprived areas than children with low socio-economic status living in less deprived areas.*

3.3.3.1 Individual level socio-economic status – poverty

Information regarding familial income was recorded in each wave of the MCS, and from this it was calculated whether or not a family was living below the poverty line, that is, if the family earned below 60% of the national median income before housing costs (Bradshaw and Holmes, 2010). This was then recorded as a categorical variable and was recoded into a binary variable (1 = living below the poverty line, 0 = living above the poverty line) during data preparation. This variable was named 'poverty' across all waves. A new variable was also created that recorded if the cohort member lived below the poverty line in Wave 1 to allow for analysis related to the initial environment the cohort member grew up in, and this was called 'poverty initial'. As seen in Table 3.5, in Wave 5, around 26% of participants lived below the poverty line in England, and when focussing on London alone, this increased to 30%.

3.3.3.2 Area level socio-economic status – IMD Score

The IMD provides data on both the score and rank of each LSOA in England, and as previously discussed, this study uses the 2010 IMD score. IMD is a continuous variable where a higher score corresponds to an increased level of deprivation. The IMD score was also recoded into quartiles and these quartiles were divided into LSOAs with the highest levels deprivation, LSOAs with medium to high levels

of deprivation, LSOAs of low to medium levels of deprivation and LSOAs of the lowest levels deprivation. Using quartiles allows for comparisons between differing levels of deprivation in analysis. Furthermore, quartiles also allow for nonlinearities to be highlighted in the data, for example if an area of medium-high deprivation was associated with a higher rate of asthma, as opposed to an area of high deprivation. This variable was named 'imd' across all waves, whilst the quartiles were named 'imd1' (least deprived) through to 'imd4' (most deprived). In addition to the quartiles, further quartiles were created that recorded the IMD of the area the cohort member lived in during Wave 1 in order to have a record of what level of deprivation the cohort member experienced during their first years. For the multilevel modelling analysis, IMD was aggregated to the MSOA level and recorded as a decile, ranging from least deprived (1) to most deprived (10).

3.3.4 Other potential confounders

The covariates of interest to this study were all available in the MCS.

3.3.4.1   Sex

Sex is an important indicator of respiratory health in children, and features prominently in research, as male children are more likely to have respiratory health issues when compared to female children (Osman, 2003). Sex is available as a binary variable from the MCS (1 = female, 0 = male), and in Wave 1, around 49% of children were female. This variable was named 'sex' across all waves.

3.3.4.2   Ethnicity

Ethnicity can be related to both respiratory health and socio-economic status, as well as potential air pollution exposure, and this is evidenced in the literature for example Fecht et al. (2015) found that neighbourhoods that are over 20% non-White have higher mean PM_{10} and NO_2 concentrations when compared to neighbourhoods with less than 20% non-White, and Netuveli et al. (2005) showed that individuals of ethnic minorities are at higher risk of asthma incidence when comparted with White groups. Originally available as a nominal variable that included many different ethnicities, such as White British, Indian, Pakistani and African, during data preparation this variable was recoded condensed into a binary variable for this research, with a focus on those that were White British as

they made up the vast majority of the cohort members and this would allow insight into how ethnic minorities fared (1 = White British, 0 = other). In Wave 1, 73% of the children were recorded as being White British. This variable was named 'whitebrit' across all waves. Following review, ethnicity was instead included as a categorical variable. As seen in Table 3.5, in Wave 5 around 75% of the cohort members were white. In London specifically in Wave 5, only 40% of the cohort was white. This categorical variable was named 'ethnicity' across all waves.

3.3.4.3 Obesity

Rates of obesity have been linked to socio-economic status in previous research (Stamatakis et al., 2010), and the impact that obesity has on respiratory health has also been widely researched (Beuther et al., 2006, Shore and Johnston, 2006). Obesity can also provide an insight into lifestyle determinants of health, for example, levels of physical activity. It was therefore important to include this variable to fully explore the relationship here. From Wave 2 onwards, the height and weight of all cohort members were recorded and BMI was calculated to determine the prevalence of obesity, which was defined by the International Obesity Task Force (IOTF) cut-offs for BMI, that were sex and age specific (Brophy et al., 2009). The obesity result was then used to record if the children were obese or not in a new binary variable (1 = obese, 0 = not obese). Whilst a record of obesity is unavailable for Wave 1, around 5% of children were recorded as being obese in Wave 2. This variable was named 'obese' across all waves.

3.3.4.4 Maternal employment

Previous research has explored the possible relationship between maternal employment and respiratory health in children, as well as the possible links with air pollution exposure, however results have been inconclusive (Morrill, 2011). Maternal employment was therefore included in this research to further explore these relationships, in an attempt to better understand the associations present. Available as a categorical variable that provides information on the working life of the cohort member's mother, a new variable for maternal employment was created as a binary variable during the data preparation phase, which grouped both full-time and part-time work together, irrespective of hours worked in a week (1 = mother is in employment, 0 = mother is not in employment). In Wave 1, 44%

of mothers were employed. This variable was named 'motheremployed' across all waves.

3.3.4.5 Maternal asthma

Maternal asthma has been shown to be related to increased asthma rates in children in previous literature (Lebold et al., 2020, Lim and Kobzik, 2009), and so is a valuable piece of information when further exploring the relationships at play in this study. Maternal asthma was recorded as a categorical variable and was recoded into a binary variable during the data preparation stage (1 = mother has asthma, 0 = mother does not have asthma). In Wave 1, roughly 16% of children had a mother that had asthma. This variable was named 'motherasthma' across all waves.

3.3.4.6 Maternal smoking

The relationship between exposure to tobacco smoke and its impacts on respiratory health has been studied extensively (Gonzalez-Barcala et al., 2013), as is the relationship between smoking habits and socio-economic status (Hiscock et al., 2012). Therefore, as this variable would indicate that a child is potentially exposed to second-hand tobacco smoke, it is important that it is included. Recorded as a categorical variable that detailed the types of tobacco products smoked, a new binary variable was created during the data preparation stage for maternal smoking based on whether or not they currently smoked (1 = mother smokes, 0 = mother does not smoke), and in Wave 1, around 27% of children had a mother that was currently a smoker. This variable was named 'mothersmokes' across all waves.

3.3.4.7 Housing tenure

Housing tenure, specifically families living in social housing, was of interest to this research because this type of housing may be indicative of lower socio-economic status. Furthermore, previous research has examined how living in social housing can impact on a child's respiratory health as well as how it can influence their exposure to air pollution (Pevalin et al., 2008). These relationships are complex and required further examination. Housing tenure was available as a categorical variable, encompassing many different types of housing tenures, such as owning own home, owning home with mortgage, living with parents, etc. A binary variable

was created during the data preparations stage that grouped the respondents who rented from a local authority or rented from a housing association together, as they lived in social housing (1 = lives in social housing, 0 = does not live in social housing). In Wave 1, around 27% of children lived in social housing. This variable was named 'socialhousing' across all waves.

3.3.4.8   Urban residency

Living in an urban area indicates an increased exposure to air pollution (Hulin et al., 2010), and so this was an important variable to include in the analysis. The relationship between child respiratory health and living in an urban or rural area is also worth considering. A categorical variable was available that recorded the living environment of the cohort members based on the ONS rural/urban classification. This variable contained details such as if the cohort member lived in a sparse or less sparse urban area, or a village or isolated dwelling, for example. During the data preparation stage, this was recoded as a binary variable to record those that either lived in an urban area or not (1 = lives in urban area, 0 = lives in rural area), and in Wave 1, around 89% of children lived in an urban area. This variable was named 'urban' across all waves.

Table 3.4 presents a summary table of the variables included in this research, and Table 3.5 presents the descriptive statistics of the cohort per wave.

Table 3.4 Summary table of variables included in this research

Variable	Variable description	Source	Level
Asthma	Child has ever had asthma	MCS	Individual
Wheeze	Child has wheezed in the last 12 months	MCS	Individual
Sex	Is the child female	MCS	Individual
Ethnicity	Is the child white British	MCS	Individual
Obesity	Is the child obese	MCS	Individual
Maternal employment	Is the child's mother employed	MCS	Individual
Maternal asthma	Does child's mother have asthma	MCS	Individual
Maternal smoking	Is the child's mother a smoker	MCS	Individual
Social housing	Does the child live in social housing	MCS	Individual
Urban	Does the child live in an urban area	MCS	Individual
Poverty	Does the child live below the poverty line	MCS	Individual
IMD	Index of Multiple Deprivation score	IMD 2010	Area
PM_{10}	Average annual PM_{10} conc.	EMEP4UK	Area
$PM_{2.5}$	Average annual NO_2 conc.	EMEP4UK	Area
NO_2	Average annual $PM_{2.5}$ conc.	EMEP4UK	Area
NO	Average annual NO conc.	EMEP4UK	Area
O_3	Average annual O_3 conc.	EMEP4UK	Area

Table 3.5 Summary statistics of the cohort make-up per wave

	All England (%)					Excluding London (%)					London only (%)				
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Asthma	7	12	15	16	17	7	12	15	16	17	7	10	13	14	15
Wheeze	7	19	16	12	11	7	20	16	12	11	7	15	15	11	9
Child is female	49	49	49	50	50	49	49	49	50	50	49	49	48	49	49
Ethnicity															
White	73	76	77	77	75	81	83	83	84	82	45	46	44	43	40
Mixed	4	4	4	4	4	3	3	3	3	3	8	8	8	8	8
Indian	4	4	4	4	4	3	3	2	2	3	9	10	10	11	10
Pakistani & Bangladeshi	11	10	9	9	10	11	10	9	9	10	10	10	9	10	12
Black	6	5	5	5	5	2	1	2	2	2	21	20	22	21	23
Other	2	2	2	2	2	1	1	1	1	1	7	7	6	7	7
Child is obese	N/A	5	6	6	6	N/A	5	5	5	6	N/A	7	8	9	8
Mother is employed	44	49	55	61	66	45	50	56	63	67	42	44	49	55	60
Mother has asthma	16	16	16	16	16	17	17	17	17	17	12	12	13	12	12
Mother smokes	27	26	26	24	21	29	28	27	25	22	20	18	18	17	14
Lives in urban area	89	86	85	84	84	86	83	82	81	81	100	100	100	100	100
Lives below the poverty line	37	32	34	29	26	37	32	33	29	25	38	34	37	33	30

3.4   Data Linkage

Data linkage was a key step in compiling the necessary data to address the hypotheses stated above. Indeed, data linkage is well established in environmental and human health literature, especially those including longitudinal and epidemiological studies (Christen and Churches, 2006). This section outlines the steps required to carry out the analyses presented in Chapters 4, 5 and 6.

3.4.1   Data preparation

Air pollution data was made available as NetCDF (Network Common Data Format) (.nc) datasets by colleagues working on EMEP4UK (Vieno et al., 2014), which is a widely used file format in atmospheric research (Michna and Woods, 2013) and is generally used for weather and climate models. A NetCDF dataset contains dimensions (latitude and longitude information), variables (air pollution concentrations) and attributes (used to store metadata) (Michna and Woods, 2013). Each year was available as a separate NetCDF dataset, however file conversion was necessary to get the data in a readable format for use in Stata. In ArcMap, using Multidimension Tools, a netCDF file can be opened as a raster layer. To do so, the variable of interest must be specified, which in this case would be one of the pollutants for that particular year, as well as specifying the X and Y dimensions, which are longitude and latitude respectively. This step is repeated for each pollutant in a particular year, resulting in five raster layers for each year, and 25 raster layers in total.

The next step involved opening a shapefile (.shp) of the LSOA boundaries in England. As the shapefile was using a different geographic coordinate system than the one used by the raster layers, it was necessary to convert the coordinate system of the shapefile. This ensures that all layers are correctly aligned, otherwise data could be hundreds of metres out of position. Following this, using the Conversion Toolbox the raster dataset is converted to polygon features. The input raster is vectorised during this conversion, and a non-simplified output is requested to ensure no data is lost through the polygon being smoothed into a simpler shape. As the newly created polygon and the original LSOA shapefile are both spatial datasets, it is then possible to join both datasets together through a spatial join. If more than one air pollution value was recorded for an LSOA, the mean concentration of all the values was calculated. The majority of LSOAs (over

50%) had only one value for air pollution concentration. The air pollution data for each year were joined with one LSOA shapefile, to create five merged datasets, one for each Wave. The newly merged dataset, containing variables for the LSOA code and its associated annual average concentration of NO_2, PM_{10}, $PM_{2.5}$, NO, and O_3, can then be exported from ArcMap in .csv format to Microsoft Excel. From here, the dataset can be exported in DTA (.dta) format for use in Stata. The DTA files were then sent to the UK Data Service to be uploaded to the Secure Lab for further use, along with the IMD 2010 dataset.

3.4.2 Secure Lab

In the Secure Lab, Stata (StataCorp, 2017) was used for data cleaning and to prepare the separate data files for linkage to create one overall data set.

Each wave had different files available for linkage, and each file contained different necessary variables. These files were;

- Longitudinal family file: This file forms the basis of the final dataset and contains the MCSID, an anonymised identifier for each family that appears in every file, allowing for linkage. This file also contains weights which are required for statistical analysis.
- Parent interview file: This file contains important information at each wave as the parent of the cohort member is questioned about any developments in the cohort members' lives. This file contains information on respiratory health, sex, maternal health and maternal smoking habits.
- Child measurement file: This file contains height and weight data for the cohort members, providing the necessary data to calculate the BMI.
- Derived family file: This file contains information about housing tenure, maternal employment and data about the socio-economic status of the family.
- Geographically linked file: This file contains information about the family location in terms of urban/rural.
- Secure geographical family file: This file contains the LSOA codes that the cohort members live in, allowing for spatial analysis.

Unnecessary variables were dropped to make the final data set a more manageable size. Next, as this study was focussing on children living in England, data for cohort members living in Northern Ireland, Scotland and Wales were also

dropped. Families with twins and triplets were dropped, keeping only singleton children. Finally, families where the mother of the cohort member was not the primary respondent were also dropped. Focussing on singleton children and families where the mother is the main respondent is commonly seen in research involving the Millennium Cohort Study (Russell et al., 2014, Heikkilä et al., 2011, Hindmarsh et al., 2017), and doing so avoids non-independence of siblings (Fitzsimons and Pongiglione, 2017). Additionally, this also ensures a consistent relationship between the survey respondent and cohort member.

Binary variables were then created using the data provided, as mentioned in section 3, whilst some new variables were also created, such as the obese variable using the newly calculated BMI, again mentioned in Section 3. Further to this, a wave variable was created to distinguish the separate waves once linkage had occurred. All variables were given uniform names to allow for linkage. These steps were repeated throughout all files over all five waves to achieve uniformity over each file. This would then enable all variables to successfully link together when creating the final dataset.

As stated, the longitudinal family file formed the base of the final dataset, and this file was combined with the parent interview file using the unique MCSID identifiers. This was in turn merged with the child measurement file, the family derived data file, the geographically linked file and the secure geographical data file, again using the unique family identifier MCSID. The secure geographical data file contained the 2001 LSOA code that the cohort member lived in, and this was required for merging the Millennium Cohort Study dataset with the air pollution data file and the IMD file which were previously uploaded. As a result, a dataset was created that contained all necessary variables for each wave. This process was then repeated for all subsequent waves, creating five separate datasets that were ultimately appended together to form the panel dataset. This was the data set that was then used for all analyses in this thesis. An illustration of the linkage is shown in Figure 3.7.
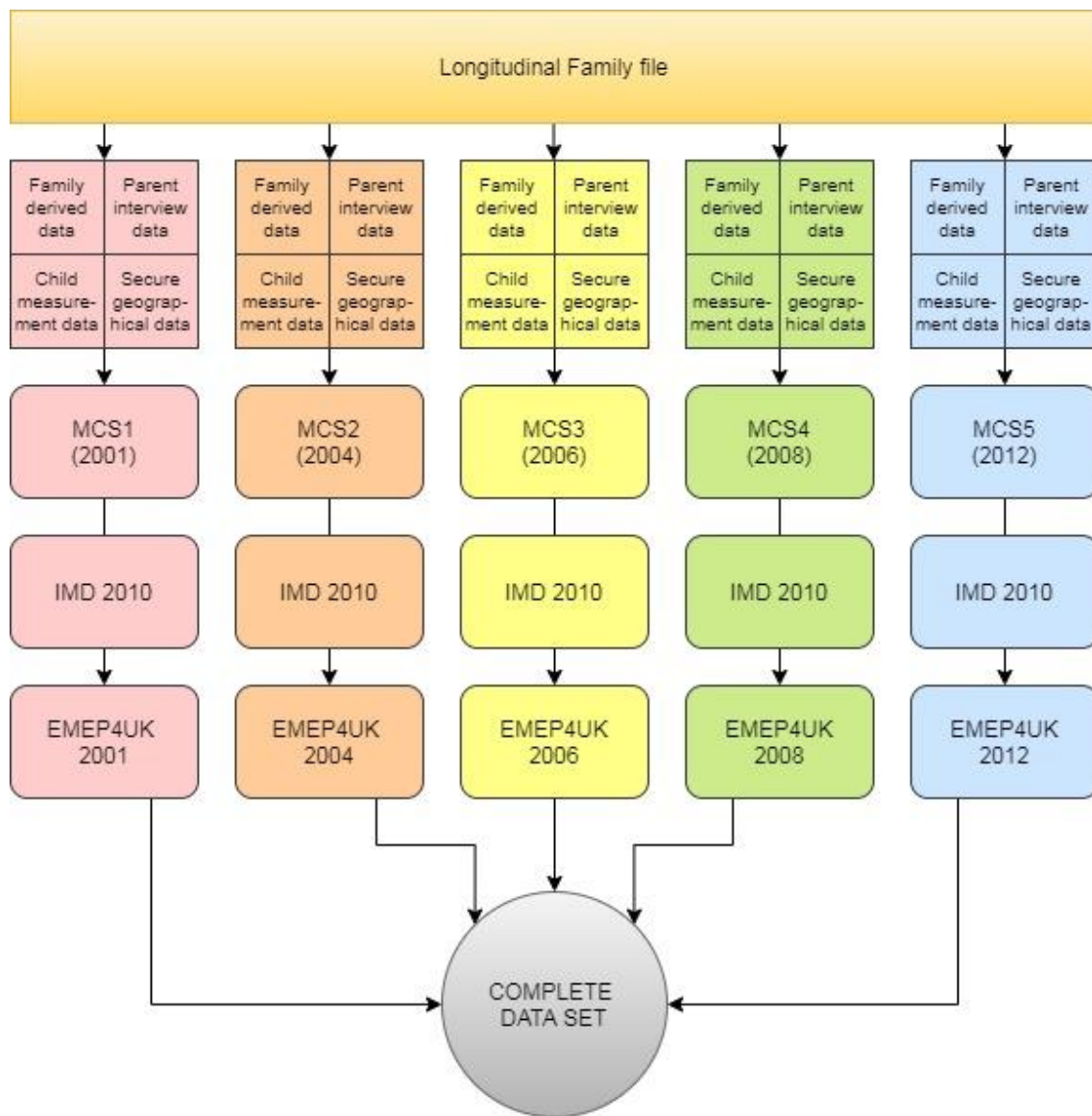
Figure 3.7 The linkage of all separate data sets, creating one complete data set used for analyses.

3.5　Statistical analysis

As the objective of this research was to understand the interlinkage between household poverty, area level deprivation and air pollution on asthma and wheeze over time, this thesis uses a longitudinal research design. However, as a first step, cross-sectional analysis is carried out, looking at each wave individually, before building on this through time-series analysis. Finally, multilevel modelling is used to fully examine the data across both time and space.

3.5.1　Cross-sectional analysis

Cross-sectional analysis is a type of observational analysis, in which the investigator measures the outcome and exposures in the study participants at the same time (Setia, 2016). Cross-sectional analysis is a common analytical method in epidemiological studies as it is relatively easy to run, as well as being inexpensive and faster than other methods of statistical analysis. It provides a snapshot of the data at a specific moment in time, allowing for initial conclusions to be drawn from the data, and so is a useful way to check research hypotheses. Cross-sectional analysis can therefore help form the basis for future analysis in a study.

The cross-sectional logistic binary regressions for each wave were carried out in Stata. The logistic models were fit using the logistic command. The logistic command fits a maximum-likelihood logistic regression model of the dependent variable on the independent variables, where the dependent variable, in this case either asthma or wheezing, is a binary variable. The logistic regression model can be written as;

$$\log \Pr(y_i = 1 | x_i) = \beta_0 + \beta_1 x_{1i} + \cdots + \beta_n x_{ni}$$

Where y is the binary response variable, and i is the individual. The predictor variables (sex, obesity, etc.) are represented by x. The intercept term is depicted by β_0 and represents the log odds when the predictor variables are 0. The coefficient β_n of a predictor variable (x_n) is the predicted change in the log of the estimated odds corresponding to a one unit change. For the purpose of this thesis, model depending, $y_i = 1$ represents each child within each wave has had asthma or a recent wheezing episode.

Estimates are presented in terms of odds ratios. In total, 20 cross-sectional logistic regressions were carried out; one for each wave focussing on asthma, including all air pollutants; one for each wave focussing on asthma, including NO_2 as the only air pollutant; one for each wave focussing on wheezing, including all air pollutants; one for each wave focussing on wheezing, including NO_2 as the only air pollutant.

However, cross-sectional analysis is not without its limitations. In terms of statistical analysis methods it is a relatively simple analytical technique, but it does not take into consideration the temporal aspect of the data, as the analysis looks only at one moment in time. Therefore, important relationships or interactions can be missed. Hence, to build upon the findings from cross-sectional analyses, longitudinal analyses were conducted. This is useful as it facilitates understanding of the temporal relationship between exposure and outcome, which cross-sectional analysis cannot do. In addition, causal inference is stronger when we can be clear that the exposure preceded the outcome in time. Thus, the next step in this statistical analysis was to include the temporal element, which is done through time-series analysis.

3.5.2 Time-series analysis

As the objective of this research was to understand the interlinkage between household poverty, area level deprivation and air pollution on asthma and wheeze over time, a population-averaged logit model using generalised estimating equations (GEE) was selected as the most appropriate way to analyse the data. A population-averaged logit model using GEE approach was chosen due to time-invariant predictors and a strong likelihood of autocorrelated residuals over time (Hubbard et al., 2010). A population-averaged approach is focused on modelling the mean response across the population of units at each time point as a function of time.

The population-averaged logistic model can be written as;

$$\log \Pr(y_{it} = 1 | x_{it}) = \beta_0^{PA} + \beta_1^{PA} x_{it} + \cdots \beta_n^{PA} x_{nit}$$

where $\text{logit p} = \log (p/1\text{-}p)$ is the usual logit 'link' function for any probability between 0 and 1 (Szmaragd et al., 2013). Similar to the logistic regression model presented in Section 3.5.1, y is the binary response variable, i represents the

individual, and t represents time, which in this case is the wave. Therefore $y_{it} = 1$ represents a child that has had asthma or a recent wheezing episode at that point in time. The predictor variables (sex, obesity, etc.) are represented by x. Here, PA signifies that this is a population-averaged model, and the intercept term is depicted by β_0^{PA}, representing the log odds when the predictor variables are 0. The coefficient β_n^{PA} of a predictor variable (x_n) is the predicted change in the log of the estimated odds corresponding to a one unit change.

Models were run in Stata using the xtlogit command, specifying the option pa to signify population-averaged model. Prior to running the model, a panel variable (LSOA) and time variable (wave) was specified using xtset. The command xtlogit fits a population-averaged logit model using GEE and produces the estimates as odds ratios. GEE is a two-stage method, in which a 'working correlation matrix' is first estimated (Szmaragd et al., 2013) before this is used to adjust the estimates of the logistic model parameters and standard errors for autocorrelation. For population-averaged logistic models, the quasi-likelihood information criterion is used to choose between the matrices.

For this analysis, the quartiles of IMD score and air pollution exposure were used. The inclusion of the different categorisations for these variables (highest levels of deprivation/pollution, mid-high levels of deprivation/pollution, mid-low levels of deprivation/pollution, and lowest levels of deprivation/pollution) would highlight any nonlinearities in the data. Time-series analysis was also used to explore the influence that a child's early environment and initial exposures had on their respiratory health over time. This was achieved by running the models using the initial variables, recorded in Wave 1, for poverty, IMD and air pollution, which were previously discussed, and comparing the estimates with the outputs from running the outputs with the regular time-varying data. This would allow comparisons to be made about how being exposed to certain characteristics in early life influence respiratory health versus being exposed to changing characteristics over a period of time.

In total, 44 logistic regressions were carried out and presented in 28 tables, 22 models (14 tables) focussing on asthma prevalence and 22 models (14 tables) focussing on rates of wheezing. The following logistic models were run four times to explore the association between respiratory health (asthma and wheezing) and different variables, examining both the impact of exposures to socio-economic

status and air pollution over time (time-varying exposures) and in Wave 1 (initial exposures);

i. Model 1 explored the association of asthma with family level socio-economic status at Wave 1, using the poverty variable.

ii. Model 2 explored the association of asthma with area level deprivation at Wave 1, as measured by IMD score.

iii. Model 3 explored the association of asthma with exposure to NO_2 at Wave 1 (Models 1 to 3 were presented in one table).

iv. Model 4 explored the association of asthma with poverty, IMD and NO_2 exposure at Wave 1.

v. Model 5 built on Model 4 by including all individual covariates, as outlined in section 3.4.

vi. Model 6 added to Model 5 with the inclusion of an interaction term that examined the relationship between poverty, IMD score and NO_2 exposure at Wave 1.

vii. Models 7 to 11 are similar to Models 1 to 5, except this time the models were run including exposure to all air pollutants at Wave 1, NO_2, PM_{10}, $PM_{2.5}$, NO and O_3, as opposed to only NO_2.

viii. Models 1 to 11 we rerun, this time using time-varying exposures to poverty, IMD and NO_2 exposure.

ix. All models would then be repeated to explore the impact on wheezing in the previous 12 months.

Whilst this time-series analysis provides a valuable insight into the relationship between these different variables and respiratory health over time, as well as allowing comparisons to be drawn between initial (Wave 1) and time-varying exposures (such as household income and different pollutants), it does not fully explain the relationship between the different variables. Specifically, it does not explain how much of the variation in respiratory health is due to variables at the individual level, or the LSOA level, or indeed the variation due to the temporal aspect of the data. Therefore, the final statistical analysis method to be examined as part of this thesis is multilevel modelling.

3.5.3 Multilevel modelling

Multilevel models (MLM) are similar to standard regression models in that they aim to model the relationship between a response variable and a set of explanatory variables, however MLM account for the nested structure of observations at different levels. For example, if a study was interested in examination scores of pupils in a school, the levels could be individual pupils (level 1), within classes (level 2), within schools (level 3). Level 1 observations within the same level 2 unit tend to be more similar to each other than level 1 observations in different level 2 units. In terms of the study presented in this thesis, this would mean that children living in the same MSOA would be more similar compared to each other than children in other MSOAs. One explanation for this would be that individuals end up in the same area through some mechanism that could be related to their specific characteristics, for example, families with limited income would be more likely to live in deprived areas. Indeed, this resonates with Tobler's First Law of Geography, "everything is related to everything else, but near things are more related than distant things" (Tobler, 1970).

In terms of longitudinal data, as each individual is observed over time, it is possible to observe individual change that is due to either the passing of time or differences in the explanatory variables. In this study, the observations over time, or repeated measures, (level 1) are nested within individuals (level 2), who themselves are nested within MSOAs (level 3). This nested structure is illustrated in the classification diagram, Figure 8, showing the data hierarchy. Here, the arrows between nodes signify a nested relationship. One key advantage of MLM is that it allows you to partition variance, thus showing how much of the variance in asthma or wheezing occurs between individuals and LSOAs respectively.
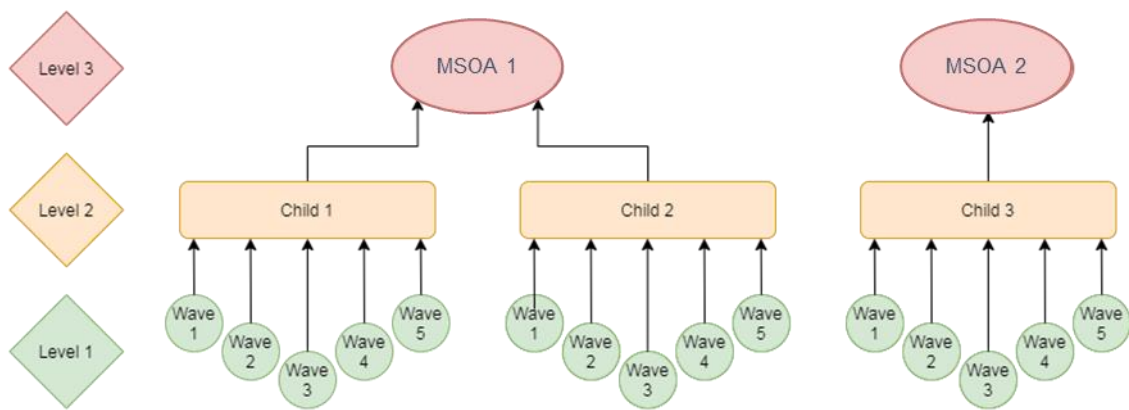
Figure 3.8   Classification diagram for the three-level model

Using the runmlwin command in Stata, the models are run through MLwiN, with the outputs produced in tables in Stata. MLwiN is a specialised software package used for fitting multilevel models, and can estimate multilevel models for continuous, binary, count, ordinal and nominal responses (Leckie and Charlton, 2013). The software provides fast estimation by both iterative generalised least squares (IGLS), resulting in maximum likelihood estimates, and by Bayesian estimation using Markov chain Monte Carlo (MCMC) methods. The command runmlwin allows users to fit models by both IGLS and MCMC algorithms and allows control over all aspects of the model specifications and estimations (Leckie and Charlton, 2013).

As asthma and wheezing are binary variables, a binary response model was most appropriate. The first step involved the estimation of discrete response multilevel models. MLwiN has two approximation methods; quasi-likelihood and Markov chain Monte Carlo (MCMC) methods. Quasi-likelihood methods approximates discrete response multilevel models as continuous response multilevel models so standard IGLS algorithm can be applied and there are four quasi-likelihood methods; first and second order marginal quasi-likelihood (MQL1, MQL2) and; first and second order penalised quasi-likelihood (PQL1, PQL2). PQL2 is the most accurate but the least stable and is the slowest to converge, whilst MQL1 is least accurate but the most stable and quickest to converge. These methods are known to be biased and are only used for model exploration, with final models fitted by MCMC (Leckie and Charlton, 2013).

This is therefore the method that was followed during data analysis. To begin with, an asthma model was fit using runmlwin, fitting the model by MQL2, before

refitting the model by MCMC. This produces a fixed part parameters and a random parts parameters table. The following models for asthma were estimated:

- Model 1: Null model with no explanatory variables included
- Model 2: Individual level variables included
- Model 3: Individual and area level variables included (NO_2 only)
- Model 4: Individual and area level variables included (all air pollutants)
- Model 5: Individual and area level variables & an interaction term for poverty*IMD*NO_2

These models were then repeated for wheezing, giving a total of ten models.

The fixed parts parameters table presents the odds ratios, as well as one-tailed p-values based on the posterior distribution. The 95% Bayesian credible intervals are also presented (the 2.5th and 97.5th quantiles of the posterior distribution). The random parts parameters table presents variance between individuals or LSOAs through the mean.

The 95% Bayesian credible intervals are also presented as are the intra-class correlation coefficient (ICC) and variance partition coefficient (VPC) results. Included in this table are the odds and probability of a child having asthma or wheezing, as well as the deviance information criterion (DIC) which is useful for comparing multilevel models, as the model with the lowest DIC tends to be the most informative model. For binary models, there is no single ICC or VPC value as the level 1 variance is a function of the mean. However, the model can be formulated in terms of a continuous latent response variable which underlies the observed binary response (Leckie and Charlton, 2013). As a result, this gives a result (the ICC) that can be interpreted as the propensity of the cohort members to have asthma or wheezing, or which can be further interpreted as a VPC that describes the variation in this propensity that lies between the levels.

The three-level model can be written as

$$y_{tij} \sim binomial\left(n_{tij}, \pi_{tij}\right)$$

$$logit\left(\pi_{tij}\right) = \beta_{0ij} x_0$$

$$\beta_{0ij} = \beta_0 + v_{0j} + u_{0ij}$$

where y_{tij} is the binary response, t represents time, i represents the child (individual level) and j represents the LSOA (area level) (Rasbash et al., 2013, Browne, 2015). The parameter n_{tij} is known as the denominator, and when dealing with binary data, is equal to 1 for all units. The mean parameter is represented with π_{tij}. The constant is represented by x_0. β_0 represents the fixed parameters, with the subscript matching the subscript of corresponding explanatory variables, v_{0j} is the effect of the LSOA j and u_{0ij} is the effect of child i within LSOA j.

3.6 Conclusion

In conclusion, this chapter has presented an overview of the data, the MCS, EMEP4UK and IMD and methods, cross-sectional, time series and multilevel modelling, used in the remainder of this thesis. The breadth of data made available for this research is a key strength going forward. Longitudinal data enables extensive analysis over time, which will highlight key relationships found in the data that would be lost in analysis that does not consider the influence of time. Indeed, the linkage used in this research also facilitates extensive analysis, bringing together two different records of socio-economic status, allowing the impacts of individual and area level socio-economic status to be examined.

Furthermore, the inclusion of air pollution data at the LSOA level further enhances this research. Section 5 outlined the methods chosen to analyse the data with the aim of testing each of the hypotheses. The methods chosen are another strength of this research. The cross-sectional analysis allows for the data to be examined wave by wave, to help understand the relationship between respiratory health, socio-economic status and air pollution at various points in the child's life. Time series analysis builds on this through the inclusion of time, and further improves the results by examining how respiratory health can be impacted by early life exposures when compared to lifetime exposures. Finally, multilevel modelling, including both temporal and spatial characteristics of the data, is the most robust analytical method used, and with the inclusion of interaction terms, this methodological approach aims to fully explore the associations present in the data and answer the hypotheses of this thesis. The next chapter will present the results from the cross-sectional analyses outlined above.

Chapter 4. Cross-sectional Analysis

4.1   Introduction

This chapter presents the results of the cross-sectional analysis. This analysis aims to answer the hypotheses that the association between respiratory health and air pollution is stronger amongst children of lower, compared to higher socio-economic status, as well as that area level deprivation will interact with individual socio-economic status so that the impact of air pollution on respiratory health is stronger for children with low socio-economic status living in the most deprived areas than children with low socio-economic status living in less deprived areas. Variables detailing if a child has had asthma or if the child has wheezed in the previous 12 months are used as a proxy for respiratory health. It is important to note that both the asthma and wheezing variables are parent reported response variables, as opposed to asking if the child had been diagnosed with a respiratory health issue by a doctor. For example, the survey question for asthma was "has [name] ever had asthma?", and the question for wheezing was "has [name] had wheezing or whistling in the chest in the last 12 months?"

This chapter begins by focussing on the impact of asthma, first examining the results from cross-sectional analyses that included all pollutant exposures, before moving on to examine the results from analyses that included NO_2 as the specific pollutant of interest. Then interaction terms are included in the analyses to further examine the association between respiratory health, socio-economic status and air pollution exposure. These models are then rerun using wheezing as the outcome variable.

4.2   Cross-sectional analysis focussing on asthma

4.2.1   All pollutants

To examine the impact of air pollution on asthma, preliminary analysis of the data included NO_2, PM_{10}, $PM_{2.5}$, NO and O_3. Table 4.2.1 presents the results of five logistic regression models, each model examining a wave of the MCS, thus showing the impact on asthma when the cohort members were aged 9 months in

Wave 1, 3 years old in Wave 2, 5 years old in Wave 3, 7 years old in Wave 4 and 11 years old in Wave 5.

Table 4.2.1 shows that in Wave 1, when cohort members were 9 months old, a female child (OR 0.66, CI 0.56 – 0.78) or a child that lives in an area with a higher concentration of PM_{10} (OR 0.32, CI 0.10 – 0.98) is less likely to have ever had asthma as reported by the parent, whilst a child that lives in a more deprived area (OR 1.42, CI 1.10 – 1.84) is statistically significantly more likely to have had asthma. Similarly, if a child has a mother that has asthma (OR 1.69, CI 1.39 – 2.05) or smokes (OR 1.38, CI 1.14 – 1.68), they are statistically significantly more likely to have had asthma also.

From the data for Wave 2, when the cohort members were 3 years old, a female child (OR 0.68, CI 0.59 – 0.79) is statistically significantly less likely to have had asthma. If a child has a mother that has asthma (OR 2.40, CI 2.04 – 2.83), smokes (OR 1.17, CI 0.99 – 1.39), lives in social housing (OR 1.59, CI 1.30- 1.94) or lives in a more deprived area (OR 1.42, CI 1.10 – 1.83), they are statistically significantly more likely to have had asthma.

When the children were 5 years old in Wave 3, the results show that a female child (OR 0.65, CI 0.57 – 0.75) is statistically significantly less likely to have had asthma. A child that has a mother who suffers from asthma (OR 2.22, CI 1.91 – 2.59), lives below the poverty line (OR 1.26, CI 1.05 – 1.51), lives in social housing (OR 1.24, CI 1.04 – 1.49) or lives in a more deprived area (OR 1.67, CI 1.33 – 2.11) is statistically significantly more likely to have had asthma.

Looking at Wave 4 when the children were 7 years old, a female child (OR 0.67, CI 0.58 – 0.77) is statistically significantly less likely to have had asthma. A child that is obese (OR 1.55, CI 1.20 – 2.02), has a mother that has asthma (OR 2.36, CI 2.02 – 2.76), lives in social housing (OR 1.34, CI 1.11 – 1.62), lives in an urban area (OR 1.33, CI 1.07 – 1.64) or lives in a more deprived area (OR 1.43, CI 1.17 – 1.76) is statistically significantly more likely to have had asthma.

The results from Wave 5, when the children were aged 11 years, show that a female child (OR 0.80, CI 0.70 – 0.92) is statistically significantly less likely to have ever has asthma. A child that is obese (OR 1.39, CI 1.06 – 1.81), has a mother that has asthma (OR 2.18, CI 1.86 – 2.56), or lives in social housing (OR 1.24, CI 1.02 – 1.51) is statistically significantly more likely to have had asthma.

Table 4.2.1 Results from cross-sectional analysis focussing on asthma including all pollutants

Asthma	Wave 1 OR		95% CI	Wave 2 OR		95% CI	Wave 3 OR		95% CI	Wave 4 OR		95% CI	Wave 5 OR		95% CI
Child is female	0.66	***	0.56 - 0.78	0.68	***	0.59 - 0.79	0.65	***	0.57 - 0.75	0.67	***	0.58 - 0.77	0.80	**	0.70 - 0.92
Child is White British	0.98		0.77 - 1.24	1.07		0.86 - 1.33	1.03		0.84 - 1.26	0.91		0.74 - 1.12	0.96		0.78 - 1.18
Child is obese	N/A			1.15		0.84 - 1.57	1.21		0.92 - 1.58	1.55	**	1.20 - 2.02	1.39	*	1.06 - 1.81
Mother is employed	1.10		0.91 - 1.34	1.06		0.91 - 1.25	1.03		0.88 - 1.2	1.10		0.93 - 1.29	1.02		0.86 - 1.21
Mother has asthma	1.69	***	1.39 - 2.05	2.40	***	2.04 - 2.83	2.22	***	1.91 - 2.59	2.36	***	2.02 - 2.76	2.18	***	1.86 - 2.56
Mother smokes	1.38	**	1.14 - 1.68	1.17	*	0.99 - 1.39	1.05		0.90 - 1.23	1.04		0.88 - 1.23	1.11		0.93 - 1.32
Lives below poverty line	1.09		0.87 - 1.37	1.06		0.87 - 1.28	1.26	*	1.05 - 1.51	1.11		0.92 - 1.34	1.02		0.82 - 1.27
Lives in social housing	1.19		0.96 - 1.47	1.59	***	1.30 - 1.94	1.24	*	1.04 - 1.49	1.34	**	1.11 - 1.62	1.24	*	1.02 - 1.51
Lives in urban area	1.18		0.87 - 1.61	1.27		0.97 - 1.65	1.20		0.95 - 1.50	1.33	*	1.07 - 1.64	1.08		0.88 - 1.32
IMD (level of deprivation)															
low	REF			REF			REF			REF			REF		
mid-low	0.98		0.75 - 1.27	1.21		0.97 - 1.51	1.41	**	1.16 - 1.72	1.30	**	1.08 - 1.57	1.19		0.99 - 1.42
mid-high	1.42	**	1.10 - 1.84	1.28	*	1.02 - 1.62	1.47	***	1.20 - 1.81	1.43	***	1.17 - 1.76	1.18		0.95 - 1.45
high	1.17		0.87 - 1.58	1.42	**	1.10 - 1.83	1.67	***	1.33 - 2.11	1.26		1.00 - 1.60	1.08		0.85 - 1.37
NO$_2$ (level of pollution)															
low	REF			REF			REF			REF			REF		
mid-low	0.78		0.48 - 1.25	0.99		0.73 - 1.33	0.89		0.67 - 1.19	0.83		0.63 - 1.08	1.02		0.79 - 1.31
mid-high	0.68		0.36 - 1.29	1.02		0.64 - 1.65	0.90		0.61 - 1.34	0.61		0.37 - 1.00	0.99		0.61 - 1.62
high	1.46		0.59 - 3.65	1.02		0.53 - 1.96	0.85		0.43 - 1.68	0.56		0.22 - 1.44	1.00		0.43 - 2.34
PM$_{10}$ (level of pollution)															
low	REF			REF			REF			REF			REF		
mid-low	0.32	*	0.11 - 0.91	1.10		0.85 - 1.41	1.13		0.86 - 1.49	1.18		0.93 - 1.50	N/A		
mid-high	0.29	*	0.10 - 0.87	1.02		0.70 - 1.48	1.18		0.83 - 1.68	0.74		0.44 - 1.23	0.73		0.49 - 1.08
high	0.32	*	0.10 - 0.98	0.95		0.53 - 1.69	0.77		0.44 - 1.33	0.68		0.25 - 1.88	N/A		
PM$_{2.5}$ (level of pollution)															

low	REF		REF		REF		REF		REF	
mid-low	0.86	0.50 - 1.47	0.74	0.53 - 1.03	0.87	0.63 - 1.21	1.03	0.83 - 1.29	N/A	
mid-high	2.63	0.86 - 8.07	0.77	0.51 - 1.17	0.97	0.66 - 1.41	1.04	0.58 - 1.86	0.99	0.78 - 1.25
high	2.22	0.70 - 7.02	0.71	0.37 - 1.36	0.88	0.46 - 1.68	N/A		1.08	0.59 - 1.98
NO (level of pollution)										
low	REF		REF		REF		REF		REF	
mid-low	1.21	0.75 - 1.95	0.99	0.72 - 1.35	0.92	0.69 - 1.23	1.02	0.78 - 1.32	1.07	0.80 - 1.43
mid-high	1.35	0.75 - 2.42	0.95	0.59 - 1.53	0.77	0.52 - 1.13	1.29	0.80 - 2.08	1.27	0.80 - 2.02
high	0.81	0.34 - 1.95	0.92	0.48 - 1.74	1.07	0.59 - 1.97	1.37	0.68 - 2.75	1.42	0.67 - 3.02
O$_3$ (level of pollution)										
low	REF		REF		REF		REF		REF	
mid-low	1.20	0.87 - 1.65	0.99	0.74 - 1.31	1.18	0.79 - 1.75	1.04	0.51 - 2.14	0.92	0.64 - 1.32
mid-high	1.33	0.83 - 2.15	0.81	0.54 - 1.21	0.96	0.62 - 1.49	0.76	0.35 - 1.65	1.12	0.72 - 1.74
high	0.63	0.31 - 1.29	0.79	0.47 - 1.34	0.91	0.55 - 1.50	0.81	0.36 - 1.82	1.38	0.85 - 2.26

4.2.2 NO$_2$ only

Due to the presence of multicollinearity between the different air pollutants, as discussed in Chapter 3 (see Table 3.3), the analysis was rerun using NO$_2$ as the only pollutant of interest. Again, five logistic regressions were carried out and the results of this analysis is presented in Table 4.2.2.

Beginning with Wave 1, a female child (OR 0.66, CI 0.56 – 0.78) is statistically significantly less likely to have ever had asthma. If a child has a mother that has asthma (OR 1.69, CI 1.39 – 2.05) or smokes (OR 1.38, CI 1.14 – 1.67), or lives in a more deprived area (OR 1.43, CI 1.10 – 1.84), they are statistically significantly more likely to have had asthma. A child living in an area with high levels of NO$_2$ pollution was less likely to have had asthma (OR 0.93, CI 0.68 – 1.26), however this was not statistically significant.

It can be seen that at Wave 2 a female child (OR 0.69, CI 0.59 – 0.79) is statistically significantly less likely to have had asthma. If a child has a mother that has asthma (OR 2.40, CI 2.04 – 2.83), lives in social housing (OR 1.57, CI 1.29- 1.91) or lives in a more deprived area (OR 1.47, CI 1.15 – 1.88), they are statistically significantly more likely to have had asthma. Again, children living in areas with the highest levels of NO$_2$ pollution were less likely to have had asthma (OR 0.96, CI 0.74 – 1.24), although this was not found to be statistically significant.

The results from Wave 3 show that a female child (OR 0.65, CI 0.57 – 0.75) is statistically significantly less likely to have had asthma. A child that has a mother who suffers from asthma (OR 2.23, CI 1.91 – 2.60), lives below the poverty line (OR 1.26, CI 1.05 – 1.50), lives in social housing (OR 1.22, CI 1.02 – 1.45) or lives in a more deprived area (OR 1.62, CI 1.29 – 2.03) is statistically significantly more likely to have had asthma. A child living in an area with high levels of NO$_2$ pollution was less likely to have had asthma (OR 0.91, CI 0.71 – 1.16) however this finding was not statistically significant.

Looking at Wave 4, a female child (OR 0.67, CI 0.58 – 0.77) is statistically significantly less likely to have had asthma. Likewise, a child that lives in an area with mid-low NO$_2$ pollution (OR 0.84, CI 0.71 – 1.00) is statistically significantly less likely to have had asthma also. A child that is obese (OR 1.54, CI 1.19 – 2.00), has a mother that has asthma (OR 2.35, CI 2.01 – 2.75), lives in social

housing (OR 1.33, CI 1.11 – 1.61), lives in an urban area (OR 1.33, CI 1.07 – 1.64) or lives in a more deprived area (OR 1.40, CI 1.14 – 1.71) is statistically significantly more likely to have had asthma throughout their life. A child living in an area of high NO_2 pollution was less likely to have had asthma (OR 0.84, CI 0.64 – 1.10) however this was not found to be statistically significant.

The results from Wave 5 show that that a female child (OR 0.81, CI 0.70 – 0.92) is statistically significantly less likely to have ever has asthma. A child that is obese (OR 1.39, CI 1.07 – 1.80), has a mother that has asthma (OR 2.18, CI 1.86 – 2.56), lives in social housing (OR 1.23, CI 1.01 – 1.50) or lives in an area of mid-low deprivation (OR 1.22, CI 1.02 – 1.45) is statistically significantly more likely to have had asthma. Again a child that lived in an area of high NO_2 pollution was found to be less likely to have had asthma (OR 0.92, CI 0.68 – 1.25) although this was not statistically significant.

Table 4.2.2 Results from cross-sectional analysis focussing on asthma including NO_2 as the only pollutant

Asthma	Wave 1 OR		95% CI	Wave 2 OR		95% CI	Wave 3 OR		95% CI	Wave 4 OR		95% CI	Wave 5 OR		95% CI
Child is female	0.66	***	0.56 - 0.78	0.69	***	0.59 - 0.79	0.65	***	0.57 - 0.75	0.67	***	0.58 - 0.77	0.81	**	0.7 - 0.92
Child is White British	1.01		0.80 - 1.27	1.09		0.88 - 1.36	1.06		0.87 - 1.29	0.92		0.75 - 1.12	0.98		0.80 - 1.20
Child is obese	N/A			1.14		0.83 - 1.55	1.21		0.93 - 1.59	1.54	**	1.19 - 2.00	1.39	*	1.07 - 1.80
Mother is employed	1.10		0.91 - 1.34	1.07		0.91 - 1.26	1.02		0.87 - 1.19	1.09		0.93 - 1.28	1.02		0.86 - 1.22
Mother has asthma	1.69	***	1.39 - 2.05	2.40	***	2.04 - 2.83	2.23	***	1.91 - 2.60	2.35	***	2.01 - 2.75	2.18	***	1.86 - 2.56
Mother smokes	1.38	**	1.14 - 1.67	1.18		0.99 - 1.40	1.06		0.91 - 1.24	1.04		0.89 - 1.23	1.10		0.93 - 1.31
Lives below poverty line	1.09		0.87 - 1.37	1.07		0.88 - 1.29	1.26	*	1.05 - 1.50	1.11		0.92 - 1.34	1.03		0.83 - 1.28
Lives in social housing	1.22		0.98 - 1.52	1.57	***	1.29 - 1.91	1.22	*	1.02 - 1.45	1.33	**	1.11 - 1.61	1.23	*	1.01 - 1.50
Lives in urban area	1.10		0.81 - 1.49	1.26		0.97 - 1.64	1.20		0.96 - 1.51	1.33	**	1.07 - 1.64	1.06		0.86 - 1.29
IMD (level of deprivation)															
low	REF			REF			REF			REF			REF		
mid-low	0.98		0.76 - 1.27	1.20		0.97 - 1.49	1.39	**	1.15 - 1.68	1.29	**	1.07 - 1.55	1.22	*	1.02 - 1.45
mid-high	1.43	**	1.10 - 1.84	1.28	*	1.02 - 1.60	1.43	**	1.16 - 1.76	1.40	**	1.14 - 1.71	1.20		0.98 - 1.48
high	1.19		0.89 - 1.59	1.47	**	1.15 - 1.88	1.62	***	1.29 - 2.03	1.24		0.98 - 1.56	1.13		0.90 - 1.43
NO_2 (level of pollution)															
low	REF			REF			REF			REF			REF		
mid-low	0.93		0.70 - 1.23	0.95		0.76 - 1.19	0.87		0.72 - 1.06	0.84	*	0.71 - 1.00	0.90		0.75 - 1.07
mid-high	0.76		0.57 - 1.02	1.06		0.84 - 1.33	0.88		0.71 - 1.08	0.83		0.67 - 1.04	0.93		0.74 - 1.17
high	0.93		0.68 - 1.26	0.96		0.74 - 1.24	0.91		0.71 - 1.16	0.84		0.64 - 1.10	0.92		0.68 - 1.25

4.2.3 Interaction terms

To fully explore the relationship between childhood respiratory health, individual level socio-economic status, area level socio-economic status and air pollution, we are interested in how the effect of one variable changes when another variable changes. This is called an interaction effect (Buis, 2010). Specifically, this thesis is interested in exploring the interactions between:

I.   individual and area level socio-economic status
   a. poverty by IMD; and
II. air pollutant exposure by individual and area level socio-economic status
   a. poverty by air pollutant;
   b. IMD by air pollutant; and
   c. poverty by IMD by air pollutant.

Table 4.2.3 includes the interaction terms within each cross-sectional analysis. The interaction between the two levels of socio-economic status was generally negative, and in Wave 3 this relationship was statistically significant (OR 0.98, CI 0.96 – 1.00). This means that a child that living below the poverty line and in an area with high deprivation was less likely to have had asthma in Wave 3. The interaction between individual level socio-economic status and NO_2, as well as the interaction between area level deprivation and NO_2 exposure were generally negative and were not statistically significant. The three-way interaction was again generally negative, which means that a child that lived below the poverty line, in an area of high deprivation and high NO_2 pollution was less likely to have had asthma. However, the three-way interaction was not statistically significant.

Although exposure to high levels of NO_2 pollution was found to not have a statistically significant impact on asthma throughout, the results give way to further discussion. Asthma is a nebulous term and the Millennium Cohort Study did not require proof of medical diagnosis before recording a child as having had asthma. Therefore asthma may be an unreliable proxy of respiratory health and this could cloud the real impact that air pollution exposure has on asthma rates in children. To strengthen the results, wheezing within the previous 12 months was also included in the analysis. Wheezing within the previous 12 months may be a more appropriate and reliable indicator of respiratory health as it is relating to a temporally recent occurrence.

Table 4.2.3 Results from cross-sectional analysis focussing on asthma including interaction terms

Asthma	Wave 1 OR		95% CI	Wave 2 OR		95% CI	Wave 3 OR		95% CI	Wave 4 OR		95% CI	Wave 5 OR		95% CI
Child is female	0.66	***	0.56 - 0.70	0.68	***	0.59 - 0.79	0.66	***	0.57 - 0.75	0.67	***	0.59 - 0.77	0.81	**	0.71 - 0.93
Child is White British	0.96		0.76 - 1.22	1.09		0.89 - 1.35	1.02		0.84 - 1.24	0.89		0.73 - 1.08	0.97		0.80 - 1.19
Child is obese	N/A			1.14		0.84 - 1.56	1.21		0.92 - 1.59	1.53	***	1.18 - 1.98	1.39	*	1.07 - 1.81
Mother is employed	1.11		0.91 - 1.34	1.07		0.91 - 1.26	1.01		0.87 - 1.18	1.09		0.93 - 1.28	1.03		0.86 - 1.22
Mother has asthma	1.69	***	1.39 - 2.05	2.40	***	2.04 - 2.83	2.22	***	1.91 - 2.59	2.34	***	1.99 - 2.74	2.18	***	1.85 - 2.55
Mother smokes	1.38	**	1.15 - 1.67	1.19	*	1.00 - 1.41	1.05		0.90 - 1.23	1.04		0.88 - 1.22	1.12		0.94 - 1.33
Lives in social housing	1.24		0.99 - 1.54	1.57	***	1.29 - 1.92	1.22	*	1.02 - 1.46	1.34	**	1.12 - 1.62	1.25	*	1.02 - 1.52
Lives in urban area	1.08		0.80 - 1.44	1.35	*	1.05 - 1.74	1.17		0.94 - 1.47	1.33	**	1.07 - 1.65	1.05		0.86 - 1.30
Lives below poverty line	1.69		0.64 - 4.44	0.79		0.34 - 1.81	2.41	*	1.18 - 4.90	2.10		0.95 - 4.64	0.69		0.28 - 1.71
IMD	1.01		0.99 - 1.03	1.00		0.99 - 1.02	1.02	**	1.01 - 1.04	1.01		0.99 - 1.03	1.00		0.99 - 1.02
NO_2	1.00		0.98 - 1.02	0.99		0.97 - 1.01	1.00		0.98 - 1.02	0.99		0.97 - 1.01	0.99		0.97 - 1.02
Poverty*IMD	0.98		0.95 - 1.01	1.00		0.98 - 1.03	0.98	*	0.96 - 1.00	0.98		0.96 - 1.00	1.01		0.98 - 1.03
Poverty*NO_2	0.99		0.96 - 1.03	1.01		0.98 - 1.05	0.98		0.95 - 1.01	0.98		0.94 - 1.02	1.03		0.98 - 1.07
IMD*NO_2	0.99		0.99 - 1.00	1.00		0.99 - 1.00	0.99		0.99 - 1.00	0.99		0.99 - 1.00	0.99		0.99 - 1.00
Poverty*IMD*NO_2	1.00		0.99 - 1.00	0.99		0.99 - 1.00	1.00		0.99 - 1.00	1.00		0.99 - 1.00	0.99		0.99 - 1.00

4.3　Cross-sectional analysis focussing on wheezing

4.3.1　All pollutants

As stated in Section 4.2.1, initial analysis of the data included all air pollutants (PM_{10}, $PM_{2.5}$, NO_2, NO and O_3) and the results of this cross-sectional analysis is presented in Table 4.3.1. This table presents the results of five logistic regression models, each model examining a wave of the MCS. As before, cohort members were aged 9 months in Wave 1, 3 years old in Wave 2, 5 years old in Wave 3, 7 years old in Wave 4 and 11 years old in Wave 5.

Table 4.3.1 shows that at Wave 1, a female child (OR 0.66, CI 0.56 – 0.78) or one who lives in an area with a higher concentration of PM_{10} (OR 0.32, CI 0.10 – 0.98) is less likely to have ever had wheezed in the last year, whilst a child that lives in a more deprived area (OR 1.42, CI 1.10 – 1.84) is statistically significantly more likely to have wheezed in the last year. Similarly, if a child has a mother that has asthma (OR 1.69, CI 1.39 – 2.05) or smokes (OR 1.38, CI 1.14 – 1.68), they are statistically significantly more likely to have wheezed in the last year also.

Looking at the results from Wave 2, a female child (OR 0.78, CI 0.69 – 0.87) is statistically significantly less likely to have wheezed in the last year. If a child has a mother that has asthma (OR 1.93, CI 1.68 – 2.22), lives in social housing (OR 1.30, CI 1.10 – 1.53), lives in an urban area (OR 1.25, CI 1.02 – 1.54) or lives in a more deprived area (OR 1.39, CI 1.16 – 1.67), they are statistically significantly more likely to have wheezed in the last year.

Moving on to examine the results from Wave 3, the analysis shows that a female child (OR 0.74, CI 0.65 – 0.84) is statistically significantly less likely to have wheezed in the last year. A child that has a mother who suffers from asthma (OR 1.82, CI 1.56 – 2.12), lives below the poverty line (OR 1.20, CI 1.01 – 1.43), or lives in an area with mid-high deprivation (OR 1.26, CI 1.04 – 1.53) is statistically significantly more likely to have wheezed in the last year.

Looking at Wave 4, a female child (OR 0.70, CI 0.60 – 0.81) is statistically significantly less likely to have wheezed in the last year. A child that is obese (OR 1.71, CI 1.29 – 2.27), has a mother that has asthma (OR 2.03, CI 1.70 – 2.43), lives in an urban area (OR 1.30, CI 1.02 – 1.65), lives in an area of mid-high deprivation (OR 1.26, CI 1.01 – 1.58), or lives in an area of mid-low $PM_{2.5}$

concentration (OR 1.38, CI 1.08 – 1.76) is statistically significantly more likely to have wheezed in the last year.

The results from Wave 5 show that a female child (OR 0.70, CI 0.59 – 0.82) is statistically significantly less likely to have wheezed in the last year. A child that is obese (OR 1.85, CI 1.38 – 2.47) or has a mother that has asthma (OR 1.85, CI 1.52 – 2.23) is statistically significantly more likely to have wheezed in the last year.

Table 4.3.1 Results from cross-sectional analysis focussing on wheezing including all pollutants

Wheeze	Wave 1 OR		95% CI	Wave 2 OR		95% CI	Wave 3 OR		95% CI	Wave 4 OR		95% CI	Wave 5 OR		95% CI
Child is female	0.66	***	0.56 - 0.78	0.78	***	0.69 - 0.87	0.74	***	0.65 - 0.84	0.70	***	0.60 - 0.81	0.70	***	0.59 - 0.82
Child is White British	0.98		0.77 - 1.24	0.98		0.82 - 1.18	0.91		0.75 - 1.09	0.86		0.69 - 1.08	0.92		0.73 - 1.17
Child is obese	N/A			1.03		0.79 - 1.34	1.28		0.99 - 1.67	1.71	***	1.29 - 2.27	1.85	***	1.38 - 2.47
Mother is employed	1.10		0.91 - 1.34	1.10		0.97 - 1.25	1.04		0.90 - 1.21	1.04		0.87 - 1.24	0.89		0.72 - 1.09
Mother has asthma	1.69	***	1.39 - 2.05	1.93	***	1.68 - 2.22	1.82	***	1.56 - 2.12	2.03	***	1.70 - 2.43	1.85	***	1.52 - 2.23
Mother smokes	1.38	**	1.14 - 1.68	1.15		1.00 - 1.32	1.12		0.96 - 1.30	0.99		0.82 - 1.19	1.02		0.83 - 1.26
Lives below the poverty line	1.09		0.87 - 1.37	0.94		0.80 - 1.11	1.20	*	1.01 - 1.43	0.98		0.79 - 1.21	1.19		0.91 - 1.56
Lives in social housing	1.19		0.96 - 1.47	1.30	**	1.10 - 1.53	1.13		0.95 - 1.35	1.16		0.94 - 1.44	0.86		0.67 - 1.10
Lives in urban area	1.18		0.87 - 1.61	1.25	*	1.02 - 1.54	1.08		0.88 - 1.33	1.30	*	1.02 - 1.65	1.10		0.87 - 1.40
IMD (level of deprivation)															
low	REF			REF			REF			REF			REF		
mid-low	0.98		0.75 - 1.27	1.24	*	1.04 - 1.46	1.16		0.97 - 1.39	1.15		0.94 - 1.41	1.06		0.86 - 1.32
mid-high	1.42	**	1.10 - 1.84	1.39	***	1.16 - 1.67	1.26	*	1.04 - 1.53	1.26	*	1.01 - 1.58	1.03		0.80 - 1.32
high	1.17		0.87 - 1.58	1.22		1.00 - 1.50	1.18		0.94 - 1.47	1.11		0.85 - 1.45	0.99		0.75 - 1.31
NO$_2$ (level of pollution)															
low	REF			REF			REF			REF			REF		
mid-low	0.78		0.48 - 1.25	0.97		0.76 - 1.25	0.96		0.73 - 1.25	0.98		0.72 - 1.34	0.95		0.71 - 1.28
mid-high	0.68		0.36 - 1.29	0.87		0.59 - 1.29	0.99		0.68 - 1.45	0.73		0.42 - 1.27	0.83		0.46 - 1.50
high	1.46		0.59 - 3.65	0.86		0.50 - 1.49	0.74		0.38 - 1.46	0.78		0.35 - 1.75	0.61		0.19 - 1.96
PM$_{10}$ (level of pollution)															
low	REF			REF			REF			REF			REF		
mid-low	0.32	*	0.11 - 0.91	1.09		0.88 - 1.34	1.05		0.82 - 1.34	1.00		0.77 - 1.30	0.77		0.49 - 1.23
mid-high	0.29	*	0.10 - 0.87	1.04		0.77 - 1.4	0.96		0.69 - 1.34	0.76		0.46 - 1.25	N/A		
high	0.32	*	0.10 - 0.98	0.83		0.51 - 1.34	0.76		0.45 - 1.26	0.25		0.06 - 1.12	N/A		
PM$_{2.5}$ (level of pollution)															

	OR	95% CI	OR	95% CI	OR	95% CI	OR		95% CI	OR	95% CI
low	REF		REF		REF		REF			REF	
mid-low	0.86	0.5 - 1.47	0.89	0.68 - 1.17	1.11	0.82 - 1.51	1.38	*	1.08 - 1.76	1.06	0.81 - 1.40
mid-high	2.63	0.86 - 8.07	0.89	0.63 - 1.24	1.21	0.85 - 1.73	1.11		0.62 - 2.00	1.28	0.61 - 2.68
high	2.22	0.7 - 7.02	0.81	0.48 - 1.38	1.23	0.68 - 2.24	N/A			N/A	
NO (level of pollution)											
low	REF		REF		REF		REF			REF	
mid-low	1.21	0.75 - 1.95	1.12	0.88 - 1.45	0.88	0.66 - 1.17	0.99		0.74 - 1.33	0.86	0.61 - 1.21
mid-high	1.35	0.75 - 2.42	1.14	0.78 - 1.65	0.86	0.59 - 1.26	1.18		0.69 - 2.01	1.00	0.57 - 1.76
high	0.81	0.34 - 1.95	1.08	0.64 - 1.82	1.20	0.67 - 2.16	1.20		0.53 - 2.69	2.13	0.72 - 6.32
O₃ (level of pollution)											
low	REF		REF		REF		REF			REF	
mid-low	1.20	0.87 - 1.65	0.97	0.77 - 1.21	0.88	0.58 - 1.33	1.08		0.69 - 1.68	1.06	0.69 - 1.64
mid-high	1.33	0.83 - 2.15	0.84	0.61 - 1.15	0.99	0.64 - 1.54	0.91		0.53 - 1.54	0.98	0.59 - 1.65
high	0.63	0.31 - 1.29	0.90	0.60 - 1.36	0.93	0.57 - 1.53	1.05		0.57 - 1.92	1.42	0.81 - 2.50

4.3.2 NO₂ only

Again, because of the issue of covariance among the air pollutants, the analysis was also conducted using NO_2 as the only pollutant of interest. Again, five logistic regressions were carried out and the results of this analysis is presented in Table 4.3.2.

Table 4.3.2 shows that at Wave 1, a female child (OR 0.66, CI 0.56 – 0.78) is statistically significantly less likely to have ever had wheezed in the last year, whilst a child that lives in a more deprived area (OR 1.43, CI 1.10 – 1.84) is statistically significantly more likely to have wheezed in the last year. Furthermore, if a child has a mother that has asthma (OR 1.69, CI 1.39 – 2.05) or smokes (OR 1.38, CI 1.14 – 1.67), they are statistically significantly more likely to have wheezed in the last year also.

Looking at the results from Wave 2, a female child (OR 0.78, CI 0.69 – 0.87) is statistically significantly less likely to have wheezed in the last year. If a child has a mother that has asthma (OR 1.93, CI 1.68 – 2.23), has a mother that smokes (OR 1.15, CI 1.00 – 1.32), lives in social housing (OR 1.27, CI 1.08 – 1.50), lives in an urban area (OR 1.24, CI 1.01 – 1.52) or lives in a more deprived area (OR 1.26, CI 1.04 – 1.54), they are statistically significantly more likely to have wheezed in the last year.

Examining the results from Wave 3, the analysis shows that a female child (OR 0.74, CI 0.65 – 0.84) is statistically significantly less likely to have wheezed in the last year. A child that has a mother who suffers from asthma (OR 1.82, CI 1.57 – 2.13), lives below the poverty line (OR 1.20, CI 1.01 – 1.43), or lives in an area with mid-high deprivation (OR 1.23, CI 1.02 – 1.49) is statistically significantly more likely to have wheezed in the last year.

Moving on to look at Wave 4, a female child (OR 0.70, CI 0.60 – 0.81) is statistically significantly less likely to have wheezed in the last year. A child that is obese (OR 1.70, CI 1.29 – 2.25), has a mother that has asthma (OR 2.03, CI 1.70 – 2.42), or lives in an urban area (OR 1.30, CI 1.02 – 1.65) is statistically significantly more likely to have wheezed in the last year.

The results from Wave 5 show that a female child (OR 0.70, CI 0.59 – 0.82), or a child that lives in an area of mid-high NO_2 concentration (OR 0.73, CI 0.56 – 0.96)

is statistically significantly less likely to have wheezed in the last year. A child that is obese (OR 1.84, CI 1.38 – 2.46) or has a mother that has asthma (OR 1.84, CI 1.52 – 2.23) is statistically significantly more likely to have wheezed in the last year. To further examine the associations between exposure variables, interaction terms can be included in the models.

Table 4.3.2 Results from cross-sectional analysis focussing on wheezing including NO_2 as the only pollutant

Wheeze	Wave 1 OR		95% CI	Wave 2 OR		95% CI	Wave 3 OR		95% CI	Wave 4 OR		95% CI	Wave 5 OR		95% CI
Child is female	0.66	***	0.56 - 0.78	0.78	***	0.69 - 0.87	0.74	***	0.65 - 0.84	0.70	***	0.60 - 0.81	0.70	***	0.59 - 0.82
Child is White British	1.01		0.80 - 1.27	1.01		0.84 - 1.21	0.91		0.76 - 1.10	0.85		0.68 - 1.06	0.94		0.74 - 1.19
Child is obese	N/A			1.02		0.78 - 1.32	1.27		0.98 - 1.66	1.70	***	1.29 - 2.25	1.84	***	1.38 - 2.46
Mother is employed	1.10		0.91 - 1.34	1.11		0.98 - 1.26	1.04		0.90 - 1.21	1.02		0.85 - 1.22	0.89		0.73 - 1.10
Mother has asthma	1.69	***	1.39 - 2.05	1.93	***	1.68 - 2.23	1.82	***	1.57 - 2.13	2.03	***	1.70 - 2.42	1.84	***	1.52 - 2.23
Mother smokes	1.38	**	1.14 - 1.67	1.15	*	1.00 - 1.32	1.12		0.96 - 1.30	0.99		0.82 - 1.19	1.03		0.84 - 1.26
Lives below the poverty line	1.09		0.87 - 1.37	0.96		0.81 - 1.12	1.20	*	1.01 - 1.43	0.97		0.78 - 1.20	1.17		0.90 - 1.52
Lives in social housing	1.22		0.98 - 1.52	1.27	**	1.08 - 1.50	1.12		0.94 - 1.34	1.18		0.95 - 1.46	0.87		0.69 - 1.11
Lives in urban area	1.10		0.81 - 1.49	1.24	*	1.01 - 1.52	1.07		0.87 - 1.31	1.30	*	1.02 - 1.65	1.08		0.85 - 1.36
IMD (level of deprivation)															
low	REF			REF			REF			REF			REF		
mid-low	0.98		0.76 - 1.27	1.22	*	1.03 - 1.43	1.13		0.94 - 1.35	1.15		0.94 - 1.40	1.10		0.89 - 1.36
mid-high	1.43	**	1.10 - 1.84	1.35	**	1.14 - 1.61	1.23	*	1.02 - 1.49	1.22		0.98 - 1.52	1.06		0.82 - 1.35
high	1.19		0.89 - 1.59	1.26	*	1.04 - 1.54	1.14		0.92 - 1.42	1.03		0.79 - 1.33	1.02		0.78 - 1.35
NO_2 (level of pollution)															
low	REF			REF			REF			REF			REF		
mid-low	0.93		0.70 - 1.23	1.04		0.86 - 1.25	0.94		0.78 - 1.13	0.95		0.78 - 1.15	0.84		0.68 - 1.03
mid-high	0.76		0.57 - 1.02	1.05		0.87 - 1.28	0.93		0.77 - 1.13	0.83		0.65 - 1.06	0.73	*	0.56 - 0.96
high	0.93		0.68 - 1.26	0.86		0.69 - 1.07	0.87		0.70 - 1.10	0.85		0.62 - 1.16	0.94		0.66 - 1.35

4.3.3 Interaction terms

Table 4.3.3 includes the interaction terms of interest. As before, the interactions of interest are between individual and area level socio-economic status (poverty * IMD) and NO_2 exposures (poverty * NO_2, IMD * NO_2, poverty * IMD * NO_2).

The interaction between the two levels of socio-economic status was generally mixed, as in Wave 2, a child living below the poverty line and in an area of high deprivation was less likely to have experienced wheezing in the previous 12 months (OR 0.99, CI 0.98 – 1.02) whilst in Wave 4 a child living in a similar situation was more likely to have experienced wheezing in the previous 12 months (OR 1.01, CI 0.98 – 1.03) although these interactions were not statistically significant. The interactions between individual level socio-economic status and NO_2, as well as the interactions between area level deprivation and NO_2 exposure were also mixed and not statistically significant. The three-way interaction generally did not show any association, for example in Wave 2 (OR 1.00, CI 0.99 – 1.00), and these interactions were not statistically significant.

Table 4.3.3 Results from cross-sectional analysis focussing on asthma including NO_2 as the only pollutant

Wheeze	Wave 1 OR		95% CI	Wave 2 OR		95% CI	Wave 3 OR		95% CI	Wave 4 OR		95% CI	Wave 5 OR		95% CI
Child is female	0.66	***	0.56 - 0.70	0.77	***	0.67 - 0.87	0.74	***	0.65 - 0.84	0.70	***	0.60 - 0.81	0.70	***	0.59 - 0.82
Child is White British	0.96		0.76 - 1.22	0.99		0.83 - 1.20	0.93		0.77 - 1.12	0.86		0.69 - 1.07	0.97		0.77 - 1.24
Child is obese	N/A			1.02		0.78 - 1.33	1.27		0.98 - 1.65	1.70	***	1.28 - 2.24	1.84	***	1.39 - 2.46
Mother is employed	1.11		0.91 - 1.34	1.11		0.98 - 1.27	1.03		0.89 - 1.20	1.02		0.85 - 1.22	0.89		0.73 - 1.09
Mother has asthma	1.69	***	1.39 - 2.05	1.96	***	1.69 - 2.24	1.82	***	1.56 - 2.12	2.03	***	1.70 - 2.43	1.84	***	1.52 - 2.22
Mother smokes	1.38	***	1.15 - 1.67	1.17	*	1.02 - 1.34	1.12		0.96 - 1.30	1.00		0.83 - 1.20	1.04		0.84 - 1.27
Lives in social housing	1.24		0.99 - 1.54	1.30	**	1.10 - 1.53	1.12		0.94 - 1.34	1.18		0.96 - 1.47	0.89		0.70 - 1.14
Lives in urban area	1.08		0.80 - 1.44	1.30	**	1.07 - 1.59	1.09		0.89 - 1.34	1.32	*	1.03 - 1.68	1.10		0.86 - 1.40
Lives below the poverty line	1.69		0.64 - 4.44	1.10		0.55 - 2.19	1.53		0.77 - 3.03	0.95		0.38 - 2.36	0.66		0.22 - 2.02
IMD	1.01		0.99 - 1.03	1.01		0.99 - 1.02	1.01		0.99 - 1.02	0.99		0.98 - 1.01	0.99		0.98 - 1.02
NO_2	1.00		0.98 - 1.02	1.00		0.99 - 1.02	0.99		0.98 - 1.01	0.99		0.97 - 1.01	0.98		0.95 - 1.00
Poverty*IMD	0.98		0.95 - 1.01	0.99		0.98 - 1.02	0.99		0.96 - 1.01	1.01		0.98 - 1.03	1.00		0.97 - 1.04
Poverty*NO_2	0.99		0.96 - 1.03	0.99		0.97 - 1.02	1.00		0.97 - 1.03	1.01		0.96 - 1.05	1.04		0.98 - 1.11
IMD*NO_2	0.99		0.99 - 1.00	0.99		0.99 - 1.00	1.00		0.99 - 1.00	1.00		0.99 - 1.00	1.00		0.99 - 1.00
Poverty*IMD*NO_2	1.00		0.99 - 1.00	1.00		0.99 - 1.00	1.00		0.99 - 1.00	0.99		0.99 - 1.00	0.99		0.99 - 1.00

4.4   Conclusion of Cross-sectional Analysis of Asthma and Wheezing

The results show that female children are consistently less likely to have had asthma or to have had a recent episode of wheezing. The results also indicate that maternal asthma is also consistently related to higher rates of asthma and wheezing among cohort members throughout the five waves. Furthermore, higher rates of IMD as well as living below the poverty line, are also shown to be related to higher rates of asthma and wheezing at different stages in the lives of the cohort members. Maternal smoking patterns were also linked to increased rates of asthma and wheezing during early childhood and living in social housing and being obese were also related to increased rates of both asthma and wheezing. The impact of pollution varied throughout, with few statistically significant relationships found. Living in an area that experienced mid-low levels of $PM_{2.5}$ pollution was found to have a statistically significant impact on wheezing rates in Wave 4, when compared to areas of low $PM_{2.5}$ pollution. Whilst this statistically significant relationship was seen only in one wave, it is worthwhile exploring this relationship in further analysis with a different methodological approach.

The individual and area level socio-economic status of children and their families were represented by poverty (individual) and IMD score (area) in this analysis. For both asthma and wheezing, children living below the poverty line or living in an area of higher deprivation were more likely to have had asthma or wheezing during different waves. These relationships were, at times, found to be statistically significant. A full discussion examining these results is offered in Chapter 7.

The inclusion of interaction terms provides some support of the hypothesis that there is a relationship between air pollution and poor respiratory health as measured by wheezing, although further research is needed. Cross-sectional analysis ignores the temporal aspect that is available with this longitudinal dataset and it is not possible to determine whether the exposures, in this instance air pollution, socio-economic status and area level deprivation or outcome, poor respiratory health came first. The fact that the data is available in a longitudinal format is one strength of this research, thus it is important to use a methodological approach that allows for time to be included to fully explore the relationships present.

Chapter 5. Time Series Analysis

5.1　Introduction

This chapter presents the results of the time series analysis, as outlined in Chapter 3. Building on cross-sectional analysis presented in Chapter 4, the next step is to fully incorporate the temporal aspect of the data into the analysis, and examine how the respiratory health of cohort members is impacted by time-based exposure to different levels of air pollution and socio-economic status. Given the temporal nature of air pollution concentration, as well as changing socio-economic status, it is important to consider this data throughout time as information can be overlooked when examining only one specific moment in time.

As discussed in Chapter 3, time-series analysis examines both initial and time-varying exposures to poverty, IMD and air pollution concentrations. This allows for comparisons to be drawn between the impact that the exposures a cohort member faced during their earliest years (recorded in Wave 1) and the impact that exposures that change over the subsequent years have on their respiratory health. As with the cross-sectional analysis, both asthma and wheezing are considered as the indicator for respiratory health, and separate analyses are run to examine the impacts of all the air pollutants as well as analyses including NO_2 as the only air pollutant of interest.

5.2　Time series analysis of asthma rates

5.2.1　Time series analysis of asthma rates with initial exposures from Wave 1

To begin, analysis focussed on the initial exposures of poverty, IMD and pollution concentration that cohort members would have faced in Wave 1 when they were nine months old. Models were run that specifically looked at Wave 1 exposures to NO_2 pollution, and later Wave 1 exposures to all pollutants.

Table 5.1 depicts the results of running three separate regression models for poverty, IMD and NO_2 exposure in Wave 1. There is a statistically significant increase in asthma occurrence over time for a child that lived below the poverty line in Wave 1 (OR 1.49, CI 1.33 – 1.67), as well for as a child that lived in an

area of higher deprivation in Wave 1 (OR 1.71, CI 1.48 – 1.99). In terms of air pollution, there is a statistically significant decrease in asthma for a child that lived in an area of low-medium NO_2 pollution in Wave 1 (OR 0.86, CI 0.72 – 1.02), and whilst the odds ratio increases for higher levels of pollution, it loses significance (OR 0.94, CI 0.80 – 1.11).

Table 5.1 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and NO_2 in Wave 1 on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line (initial)		1.49	***	1.33 - 1.67
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.25	***	1.07 - 1.47
	medium-high	1.63	***	1.40 - 1.89
	high	1.71	***	1.48 - 1.99
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	0.86	*	0.72 - 1.02
	medium-high	0.92		0.79 - 1.09
	high	0.94		0.80 - 1.11

Table 5.2 runs the model with poverty, IMD and NO_2 pollution at Wave 1 in one regression. Similarly, a child that lived below the poverty line at Wave 1 is statistically significantly more likely to have had asthma over time (OR 1.27, CI 1.12 – 1.45), and this is also the case for a child that lived in an area of high deprivation at Wave 1 (OR 1.55, CI 1.30 – 1.85). On the other hand, those that lived in areas of higher NO_2 pollution are statistically significantly less likely to have had asthma over time (OR 0.80, CI 0.67 – 0.94).

Table 5.2 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO_2 in Wave 1 on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line (initial)		1.27	***	1.12 - 1.45
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.21	**	1.03 - 1.42
	medium-high	1.56	***	1.33 - 1.83
	high	1.55	***	1.30 - 1.87
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	0.82	**	0.69 - 0.97
	medium-high	0.84	**	0.71 - 0.99
	high	0.80	***	0.67 - 0.94

Table 5.3 builds on Table 5.2 with the inclusion of individual level variables. Similarly, a child that lived below the poverty line in Wave 1 is statistically

significantly more likely to have had asthma over time (OR 1.13, CI 0.98 – 1.31). Likewise, a child living in an area of higher deprivation is statistically significantly more likely to have had asthma over time (OR 1.41, CI 1.18 – 1.69) (excluding low-medium levels of deprivation). A child that lived in an area of higher NO_2 pollution in Wave 1 is statistically significantly less likely to have had asthma over time (OR 0.78, CI 0.64 – 0.95), as is a female child (OR 0.70, CI 0.63 – 0.78). If a child is obese (OR 1.32, CI 1.10 – 1.58), has a mother that is employed (OR 1.10, CI 0.99 – 1.22), has a mother that suffers asthma (OR 2.28, CI 2.01 – 2.59), lives in social housing (OR 1.32, CI 1.15 – 1.51) or lives in an urban area (OR 1.23, CI 1.04 – 1.45), they are statistically significantly more likely to have had asthma over time.

Table 5.3 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO_2 in Wave 1, and individual level variables, on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line (initial)		1.13	*	0.98 - 1.31
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.14		0.97 - 1.34
	medium-high	1.41	***	1.19 - 1.67
	high	1.41	***	1.18 - 1.69
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	0.79	***	0.66 - 0.94
	medium-high	0.77	***	0.65 - 0.92
	high	0.78	**	0.64 - 0.95
Child is female		0.70	***	0.63 - 0.78
Child is White British		1.02		0.87 - 1.20
Child is obese		1.32	***	1.10 - 1.58
Mother is employed		1.10	*	1.00 - 1.22
Mother has asthma		2.28	***	2.01 - 2.59
Mother smokes		1.08		0.96 - 1.21
Lives in social housing		1.32	***	1.15 - 1.51
Lives in urban area		1.23	**	1.04 - 1.45

Table 5.4 explores the interactions between poverty, IMD and NO_2 pollution at Wave 1 and how they interact to influence asthma rates among the cohort members. The interaction terms were included in the analysis to fully explore the associations present in the data between the exposure variables of interest. Interacting individual and area level socio-economic status (poverty and deprivation) and air pollution exposure within a three-way interaction, aims to answer the second hypothesis; whether area level deprivation interacts with

individual level socio-economic status so that the impact of air pollution exposure on respiratory health is stronger for children with low socio-economic status living in the most deprived areas than children with similar socio-economic status living in less deprived areas. Four interaction terms are presented. Although none are statistically significant, the three-way interaction, poverty*IMD*NO_2 is positive, which indicates that those living in poverty, in a more deprived and polluted area are more likely to have had asthma.

Table 5.4 Time series analysis looking at the impact of different exposures on asthma rates in children, including interactions

Asthma	OR		CI 95%
Child is female	0.70	***	0.62 - 0.78
Child is White British	1.00		0.85 - 1.17
Child is obese	1.33	**	1.11 - 1.59
Mother is employed	1.11	*	1.00 - 1.22
Mother has asthma	2.29	***	2.02 - 2.60
Mother smokes	1.08		0.96 - 1.22
Lives in social housing	1.33	***	1.16 - 1.53
Lives in urban area	1.21	*	1.03 - 1.43
Lives below the poverty line (initial)	1.40		0.34 - 5.71
IMD score (initial)	0.84		0.59 - 1.21
NO_2 (initial)	0.97		0.70 - 1.34
Poverty*IMD (initial)	0.85		0.47 - 1.52
Poverty*NO_2 (initial)	0.90		0.57 - 1.43
IMD*NO_2 (initial)	0.98		0.87 - 1.11
Poverty*IMD*NO_2 (initial)	1.08		0.88 - 1.31

Table 5.5 expands the NO_2 exposure analysis presented in Table 5.1 by also including the exposures of PM_{10}, $PM_{2.5}$, NO and O_3 at Wave 1. Again, a child that lived below the poverty line in Wave 1 is statistically significantly more likely to have had asthma over time (OR 1.49, CI 1.33 – 1.67), as is a child that lived in an area of higher IMD (OR 1.71, CI 1.48 – 1.99). Whilst there were positive results for children that lived in areas with the highest levels of $PM_{2.5}$ pollution in Wave 1 (OR 1.05, CI 0.59 – 1.86), areas with high NO_2 pollution (OR 1.33, CI 0.80 – 2.22) and areas with low-medium (OR 1.19, CI 0.96 – 1.47) and medium-high levels (OR 1.14, CI 0.84 – 1.56) of O_3 pollution, these were not statistically significant. A child that lived in an area of low-medium NO pollution at Wave 1 is statistically significantly less likely to have had asthma over time (OR 0.68, CI 0.52 – 0.90) as is a child living in area of high PM_{10} pollution (OR0.75, CI 0.44 – 1.28) although this was not statistically significant.

Table 5.5 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and all air pollutants in Wave 1 on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line (initial)		1.49	***	1.33 - 1.67
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.25	***	1.07 - 1.47
	medium-high	1.63	***	1.40 - 1.89
	high	1.71	***	1.48 - 1.99
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	1.11		0.85 - 1.44
	medium-high	1.07		0.72 - 1.58
	high	1.33		0.80 - 2.22
PM_{10} (level of pollution) (initial)	low	REF		
	low-medium	0.75		0.47 - 1.18
	medium-high	0.77		0.47 - 1.26
	high	0.75		0.44 - 1.28
$PM_{2.5}$ (level of pollution) (initial)	low	REF		
	low-medium	0.79		0.55 - 1.12
	medium-high	0.98		0.57 - 1.69
	high	1.05		0.59 - 1.86
NO (level of pollution) (initial)	low	REF		
	low-medium	0.68	***	0.52 - 0.90
	medium-high	0.95		0.65 - 1.40
	high	0.76		0.46 - 1.24
O_3 (level of pollution) (initial)	low	REF		
	low-medium	1.19		0.96 - 1.47
	medium-high	1.14		0.84 - 1.56
	high	0.95		0.63 - 1.44

Table 5.6 re-runs the regression presented in 5.5, this time including all pollutants, poverty and IMD exposures at Wave 1 in one model. A child that lived below the poverty line in Wave 1 is statistically significantly more likely to have had asthma over time (OR 1.27, CI 1.12 – 1.44), as is a child that lived in an area of higher deprivation (OR 1.56, CI 1.31 – 1.86). A child that lived in an area of high $PM_{2.5}$ pollution (OR 1.05, CI 0.58 – 1.88), an area of medium-high NO pollution (OR 1.01, CI 0.69 – 1.49) or an area of low-medium (OR 1.23, CI 0.99 – 1.53) or medium-high O_3 pollution (OR 1.13, CI 0.82 – 1.55) is more likely to have had asthma over time, however only the area of low-medium O_3 pollution was statistically significant. A child that lived in an area of low-medium NO pollution in Wave 1 is statistically significantly less likely to have had asthma over time (OR 0.72, CI 0.54 – 0.95).

Table 5.6 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all air pollutants in Wave 1 on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line (initial)		1.27	***	1.12 - 1.44
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.20	**	1.02 - 1.41
	medium-high	1.56	***	1.33 - 1.84
	high	1.56	***	1.31 - 1.86
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	0.97		0.74 - 1.28
	medium-high	0.86		0.58 - 1.28
	high	0.89		0.52 - 1.52
PM_{10} (level of pollution) (initial)	low	REF		
	low-medium	0.85		0.54 - 1.35
	medium-high	0.85		0.52 - 1.40
	high	0.80		0.47 - 1.37
$PM_{2.5}$ (level of pollution) (initial)	low	REF		
	low-medium	0.78		0.55 - 1.11
	medium-high	0.95		0.54 - 1.65
	high	1.05		0.58 - 1.88
NO (level of pollution) (initial)	low	REF		
	low-medium	0.72	**	0.54 - 0.95
	medium-high	1.01		0.69 - 1.49
	high	0.93		0.56 - 1.56
O_3 (level of pollution) (initial)	low	REF		
	low-medium	1.23	*	0.99 - 1.53
	medium-high	1.13		0.82 - 1.55
	high	0.93		0.61 - 1.41

Table 5.7 builds on the model used in Table 5.6 with the inclusion of individual level variables. A child that, in Wave 1, lived in an area of medium-high (OR 1.42, CI 1.20 – 1.68) or high deprivation (OR 1.41, CI 1.18 – 1.70), or an area of low-medium O_3 pollution (OR 1.22, CI 0.98 – 1.52) is statistically significantly more likely to have had asthma over time, whilst a child that lived in an area of low-medium NO pollution (OR 0.71, CI 0.53 – 0.95) is statistically significantly less likely to have had asthma. Furthermore, a female child is statistically significantly less likely to have had asthma over time (OR 0.70, CI 0.63 – 0.78), whilst a child that is obese (OR 1.32, CI 1.11 – 1.58), has a mother that is employed (OR 1.10, CI 0.99 – 1.21), has a mother that has asthma (OR 2.30, CI 2.02 – 2.61), lives in social housing (OR 1.31, CI 1.14 – 1.51) or lives in an urban area (OR 1.23, CI 1.04 – 1.46) is statistically significantly more likely to have had asthma over time.

Table 5.7 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all pollutants in Wave 1, and individual level variables, on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line (initial)		1.12		0.97 - 1.29
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.14		0.97 - 1.34
	medium-high	1.42	***	1.20 - 1.68
	high	1.41	***	1.18 - 1.70
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	0.93		0.71 - 1.23
	medium-high	0.79		0.52 - 1.18
	high	0.85		0.50 - 1.46
PM_{10} (level of pollution) (initial)	low	REF		
	low-medium	0.80		0.51 - 1.28
	medium-high	0.78		0.47 - 1.29
	high	0.72		0.42 - 1.25
$PM_{2.5}$ (level of pollution) (initial)	low	REF		
	low-medium	0.80		0.56 - 1.14
	medium-high	1.02		0.58 - 1.79
	high	1.15		0.63 - 2.08
NO (level of pollution) (initial)	low	REF		
	low-medium	0.71	**	0.53 - 0.95
	medium-high	0.99		0.67 - 1.46
	high	0.94		0.56 - 1.57
O_3 (level of pollution) (initial)	low	REF		
	low-medium	1.22	*	0.98 - 1.52
	medium-high	1.09		0.79 - 1.50
	high	0.91		0.60 - 1.40
Child is female		0.70	***	0.63 - 0.78
Child is White British		1.01		0.86 - 1.19
Child is obese		1.32	***	1.11 - 1.58
Mother is employed		1.10	*	0.99 - 1.21
Mother has asthma		2.30	***	2.02 - 2.61
Mother smokes		1.08		0.96 - 1.21
Lives in social housing		1.31	***	1.14 - 1.51
Lives in urban area		1.23	**	1.04 - 1.46

5.2.2 Time series analysis of asthma with time varying exposures throughout the Waves

Moving on from Wave 1 exposures, analysis then focussed on time varying exposures of poverty, IMD and air pollution concentration. Time varying analysis allows variables to vary for each wave instead of using only the initial recording

from Wave 1. It is important to take this into consideration as the exposure variables change over time. For example, a family's socio-economic status may change so they no longer live below the poverty line. Additionally, as evident in Figure 3.2, air pollution concentrations vary over time, as such it is important to consider the effects of exposure to varying concentrations over time in comparison to initial early life exposure.

Table 5.8 shows the results of three separate regressions for poverty, IMD and NO_2 pollution. A child that lives below the poverty line (OR 1.44, CI 1.31 – 1.59) is statistically significantly more likely to have had asthma over time. Similarly, a child living in an area of high deprivation (OR 1.73, CI 1.50 – 2.00) is also more likely to have had asthma. Table 5.8 also shows that a child living in an area of high NO_2 pollution is less likely to have had asthma (OR 0.97, CI 0.84 – 1.13); however these results were not statistically significant.

Table 5.8 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and NO_2 on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line		1.44	***	1.31 - 1.59
Level of deprivation (IMD)	low	REF		
	low-medium	1.36	***	1.18 - 1.57
	medium-high	1.57	***	1.36 - 1.80
	high	1.73	***	1.50 - 2.00
NO_2 (level of pollution)	low	REF		
	low-medium	0.95		0.85 - 1.05
	medium-high	0.95		0.84 - 1.08
	high	0.97		0.84 - 1.13

Table 5.9 included all variables in a single model. From this, it can be seen that a child living below the poverty line (OR 1.25, CI 1.12 – 1.38), or in an area of higher deprivation (OR 1.63, CI 1.39 – 1.91) is statistically significantly more likely to have had asthma throughout their life, whilst a child living in an area of higher NO_2 pollution is statistically significantly less likely to have had asthma over time (OR 0.80, CI 0.68 – 0.93).

Table 5.9 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO_2 on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line		1.25	***	1.12 - 1.38
Level of deprivation (IMD)	low	REF		
	low-medium	1.34	***	1.16 - 1.54

		OR		CI 95%
	medium-high	1.53	***	1.32 - 1.78
	high	1.63	***	1.39 - 1.91
NO$_2$ (level of pollution)	low	REF		
	low-medium	0.89	**	0.80 - 1.00
	medium-high	0.87	**	0.76 - 0.99
	high	0.80	***	0.68 - 0.93

Table 5.10 further builds on this with the addition of individual level variables. A child living below the poverty line (OR 1.12, CI 1.00 – 1.24), as well as living in an area of higher deprivation (OR 1.38, CI 1.16 – 1.64) is statistically significantly more likely to have had asthma throughout their life. On the other hand, a child living in an area of higher NO$_2$ pollution (OR 0.77, CI 0.64 – 0.92) is statistically significantly less likely to have had asthma. Furthermore, a female child is also statistically significantly less likely to have had asthma (OR 0.70, CI 0.63 – 0.79). A child that is obese (OR 1.33, CI 1.11 – 1.59), has a mother that is employed (OR 1.10, CI 1.00 – 1.22), has a mother that has asthma (OR 2.28, CI 2.01 – 2.59), lives in social housing (OR 1.35, CI 1.18 – 1.55) or an urban area (OR 1.25, CI 1.06 – 1.47) is statistically significantly more likely to have had asthma throughout their life.

Table 5.10 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO$_2$, and individual level variables, on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line		1.12	**	1.00 - 1.25
Level of deprivation (IMD)	low	REF		
	low-medium	1.27	***	1.10 - 1.46
	medium-high	1.33	***	1.14 - 1.56
	high	1.38	***	1.16 - 1.64
NO$_2$ (level of pollution)	low	REF		
	low-medium	0.83	***	0.74 - 0.94
	medium-high	0.83	***	0.72 - 0.95
	high	0.77	***	0.64 - 0.92
Child is female		0.70	***	0.63 - 0.79
Child is White British		0.96		0.82 - 1.13
Child is obese		1.33	***	1.11 - 1.59
Mother is employed		1.10	*	1.00 - 1.22
Mother has asthma		2.28	***	2.01 - 2.59
Mother smokes		1.08		0.96 - 1.21
Lives in social housing		1.35	***	1.18 - 1.55
Lives in urban area		1.25	***	1.06 - 1.47

Table 5.11 explores the interactions between time varying poverty, IMD and NO$_2$

pollution. Four interaction terms are shown, and these are not statistically significant.

Table 5.11 Time series analysis looking at the impact of different exposures on asthma rates in children, including interactions

Asthma	OR		CI 95%
Child is female	0.71	***	0.63 - 0.79
Child is White British	0.95		0.81 - 1.11
Child is obese	1.33	**	1.12 - 1.60
Mother is employed	1.10		0.99 - 1.22
Mother has asthma	2.28	***	2.00 - 2.58
Mother smokes	1.09		0.97 - 1.22
Lives in social housing	1.36	***	1.18 - 1.56
Lives in urban area	1.28	**	1.08 - 1.51
Lives below the poverty line	1.23		0.76 - 2.01
IMD score	1.01		1.00 - 1.02
NO_2	0.98	*	0.97 - 1.00
Poverty*IMD	0.99		0.98 - 1.01
Poverty*NO_2	1.00		0.98 - 1.03
IMD*NO_2	1.00		1.00 - 1.00
Poverty*IMD*NO_2	1.00		1.00 - 1.00

Table 5.12 is similar to Table 5.8, but includes all pollutants (PM_{10}, $PM_{2.5}$, NO and O_3, as well as NO_2). A child living below the poverty line (OR 1.44, CI 1.31 – 1.59) is statistically significantly more likely to have had asthma, as is a child living in areas of higher deprivation (OR 1.73, CI 1.50 – 2.00). Regarding the pollution variable, a child living in areas of higher PM_{10} (OR 1.03, CI 0.94 – 1.81) and NO_2 pollution (OR 1.30, CI 0.94 – 1.81) also has an increased likelihood of having had asthma throughout their life, however these results were not statistically significant. Furthermore, a child living in an area of high O_3 (OR 0.99, CI 0.81 – 1.23) or NO (OR 0.94, CI 0.68 – 1.31) pollution is less likely to have had asthma, although this also was not statistically significant. A child living in an area of higher $PM_{2.5}$ pollution (OR 0.55, CI 0.39 – 0.76), however, was statistically significantly less likely to have had asthma.

Table 5.12 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and all air pollutants on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line		1.44	***	1.31 - 1.59
Level of deprivation (IMD)	low	REF		
	low-medium	1.36	***	1.18 - 1.57
	medium-high	1.57	***	1.36 - 1.80

	high	1.73	***	1.50 - 2.00
NO$_2$ (level of pollution)	low	REF		
	low-medium	1.06		0.93 - 1.22
	medium-high	1.12		0.90 - 1.40
	high	1.30		0.94 - 1.81
PM$_{10}$ (level of pollution)	low	REF		
	low-medium	1.08		0.96 - 1.22
	medium-high	1.09		0.88 - 1.35
	high	1.03		0.73 - 1.46
PM$_{2.5}$ (level of pollution)	low	REF		
	low-medium	0.81	***	0.72 - 0.91
	medium-high	0.73	***	0.61 - 0.88
	high	0.55	***	0.39 - 0.79
NO (level of pollution)	low	REF		
	low-medium	0.94		0.82 - 1.08
	medium-high	0.95		0.76 - 1.19
	high	0.94		0.68 - 1.31
O$_3$ (level of pollution)	low	REF		
	low-medium	0.98		0.84 - 1.13
	medium-high	0.92		0.77 - 1.10
	high	1.00		0.81 - 1.23

Table 5.13 presents the results of a single model that includes all the variables examined in Table 5.12. A child living below the poverty line (OR 1.26, CI 1.13 – 1.39) is statistically significantly more likely to have had asthma throughout their life, as is a child living in areas of higher deprivation (OR 1.57, CI 1.33 – 1.84). A child living in areas of higher PM$_{2.5}$ deprivation is statistically significantly less likely to have had asthma (OR 0.66, CI 0.46 – 0.94).

Table 5.13 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all air pollutants on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line		1.26	***	1.13 - 1.39
Level of deprivation (IMD)	low	REF		
	low-medium	1.34	***	1.16 - 1.54
	medium-high	1.53	***	1.32 - 1.77
	high	1.57	***	1.33 - 1.84
NO$_2$ (level of pollution)	low	REF		
	low-medium	0.95		0.83 - 1.10
	medium-high	0.94		0.75 - 1.18
	high	0.95		0.68 - 1.32
PM$_{10}$ (level of pollution)	low	REF		
	low-medium	1.07		0.95 - 1.21
	medium-high	1.08		0.87 - 1.33

		OR		CI 95%
	high	0.90		0.63 - 1.29
PM2.5 (level of pollution)	low	REF		
	low-medium	0.85	***	0.76 - 0.96
	medium-high	0.78	***	0.65 - 0.93
	high	0.66	**	0.46 - 0.94
NO (level of pollution)	low	REF		
	low-medium	0.96		0.83 - 1.09
	medium-high	0.96		0.77 - 1.20
	high	1.00		0.71 - 1.40
O3 (level of pollution)	low	REF		
	low-medium	0.98		0.84 - 1.14
	medium-high	0.91		0.75 - 1.09
	high	0.93		0.75 - 1.15

Table 5.14 includes the individual level variables. The analysis shows that a child living below the poverty line (OR 1.11, CI 0.99 – 1.24) or living in areas of higher deprivation (OR 1.31, CI 1.10 – 1.56) is statistically significantly more likely to have had asthma throughout their life. Furthermore, a child living in areas of higher PM2.5 pollution (OR 0.65, CI 0.45 – 0.95) is statistically significantly less likely to have had asthma. A female child (OR 0.70, CI 0.63 – 0.78) is also statistically significantly less likely to have ever had asthma, whilst a child that is obese (OR 1.34, CI 1.12 – 1.60), has a mother that has asthma (OR 2.29, CI 2.01 – 2.60), lives in social housing (OR 1.37, CI 1.19 – 1.57) or an urban area (OR 1.25, CI 1.05 – 1.47) is statistically significantly more likely to have had asthma.

Table 5.14 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all pollutants, and individual level variables, on asthma rates in children

Asthma		OR		CI 95%
Lives below the poverty line		1.11	*	0.99 - 1.24
Level of deprivation (IMD)	low	REF		
	low-medium	1.26	***	1.09 - 1.46
	medium-high	1.32	***	1.13 - 1.55
	high	1.31	***	1.10 - 1.56
NO2 (level of pollution)	low			
	low-medium	0.90		0.78 - 1.04
	medium-high	0.91		0.72 - 1.15
	high	0.96		0.68 - 1.35
PM10 (level of pollution)	low	REF		
	low-medium	1.07		0.95 - 1.21
	medium-high	1.03		0.84 - 1.28
	high	0.86		0.60 - 1.25
PM2.5 (level of pollution)	low	REF		

	low-medium	0.85	***	0.75 - 0.95
	medium-high	0.78	***	0.65 - 0.94
	high	0.65	**	0.45 - 0.95
NO (level of pollution)	low	REF		
	low-medium	0.97		0.84 - 1.11
	medium-high	0.98		0.78 - 1.23
	high	1.00		0.71 - 1.40
O_3 (level of pollution)	low	REF		
	low-medium	0.98		0.84 - 1.15
	medium-high	0.92		0.76 - 1.11
	high	0.97		0.78 - 1.20
Child is female		0.70	***	0.63 - 0.78
Child is White British		0.96		0.82 - 1.12
Child is obese		1.34	***	1.12 - 1.60
Mother is employed		1.08		0.97 - 1.20
Mother has asthma		2.29	***	2.01 - 2.56
Mother smokes		1.08		0.96 - 1.22
Lives in social housing		1.37	***	1.19 - 1.57
Lives in urban area		1.25	***	1.06 - 1.47

5.2.3 Comparing the effects of initial and time-varying exposures on asthma

To illustrate the difference between impacts on asthma rates based on exposures in Wave 1 and over time, Figures 1a and 1b show the log odds of a child having had asthma based on the IMD score of the LSOA that they lived in in Wave 1 (5.1) or that they lived in in each wave (5.2). These graphs show that someone living in the least deprived quartile, either in Wave 1 or over time, is consistently less likely to have had asthma than someone living in a more deprived area.

Figures 5.3 and 5.4 show the log odds of a child having had asthma based on the annual average NO_2 concentration of the LSOA that they lived in in Wave 1 (5.3) or that they lived in in each wave (5.4). These graphs show that there is not much variability over the quartiles.

Figures 5.5 and 5.6 show the log odds of a child having had asthma based on whether they lived below the poverty line in Wave 1 (5.5) or in each wave (5.6). These graphs show that someone living above the poverty line, either initially or over time, is consistently less likely to have had asthma than someone living below the poverty line.

Whilst asthma is a good indicator of respiratory health, it may not be a consistently reliable predictor of poor respiratory health, as discussed previously. Indeed, wheezing within the previous 12 months may be a more reliable indicator of the current respiratory health of the cohort member. The next Section of this Chapter reruns the analysis with wheezing as the outcome variable.
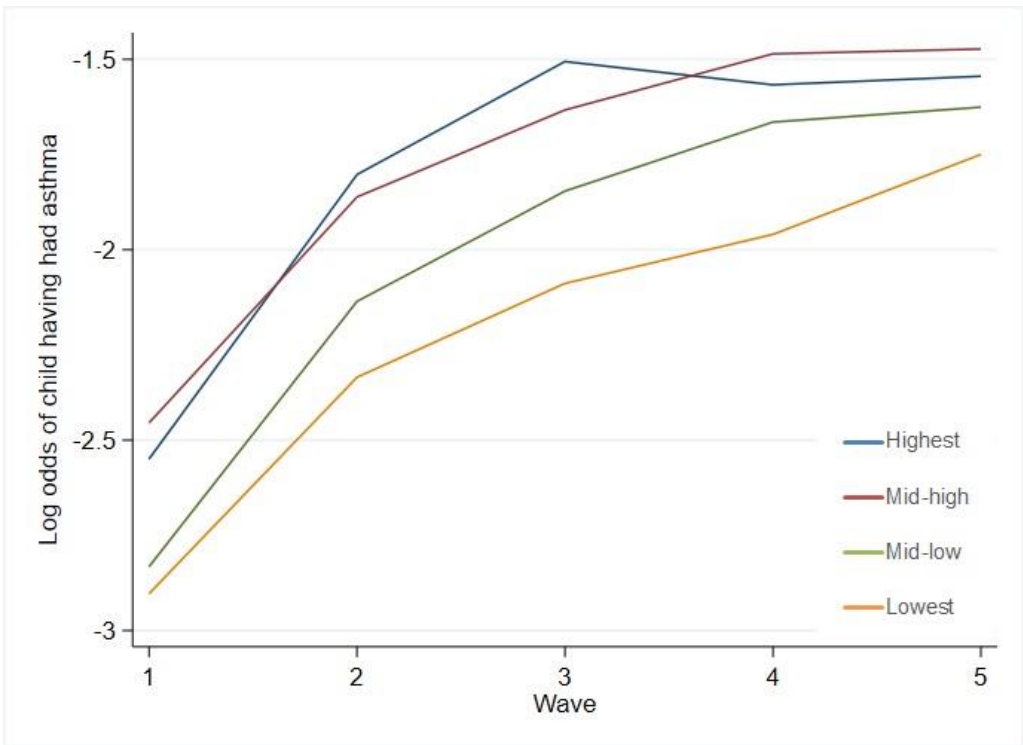
Figure 5.1 The log odds of a child having had asthma throughout the five waves of the MCS based on the IMD score (separated into quartiles) of the LSOA they lived in during Wave 1.
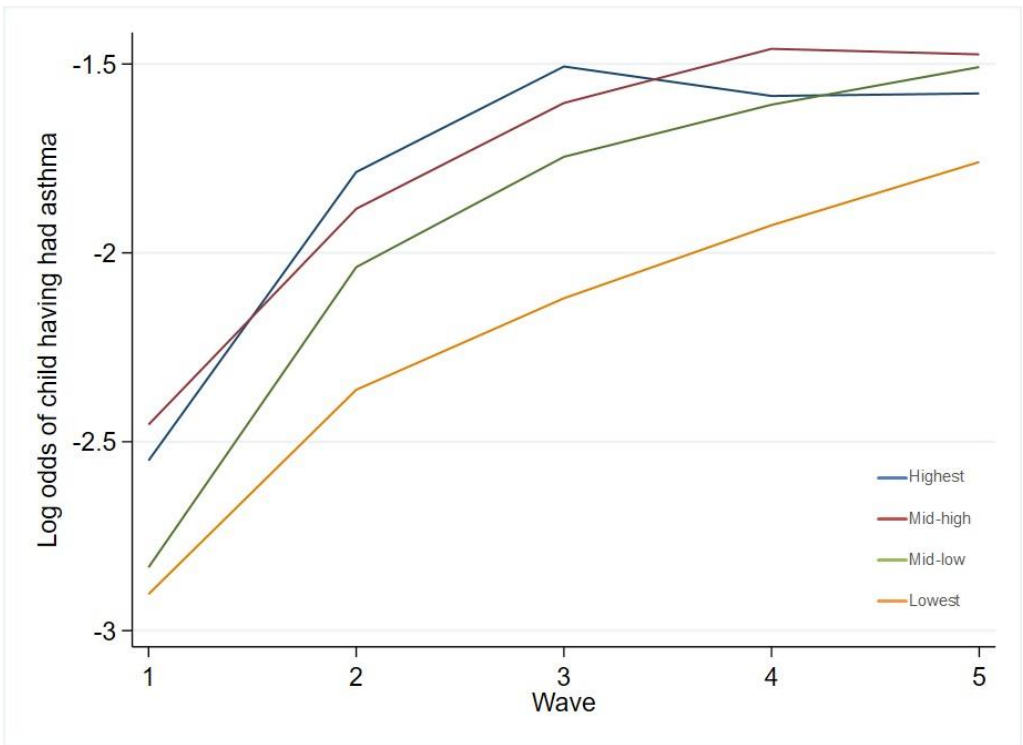
Figure 5.2 The log odds of a child having had asthma throughout the five waves of the MCS based on the IMD score (separated into quartiles) of the LSOA they lived in during each wave.
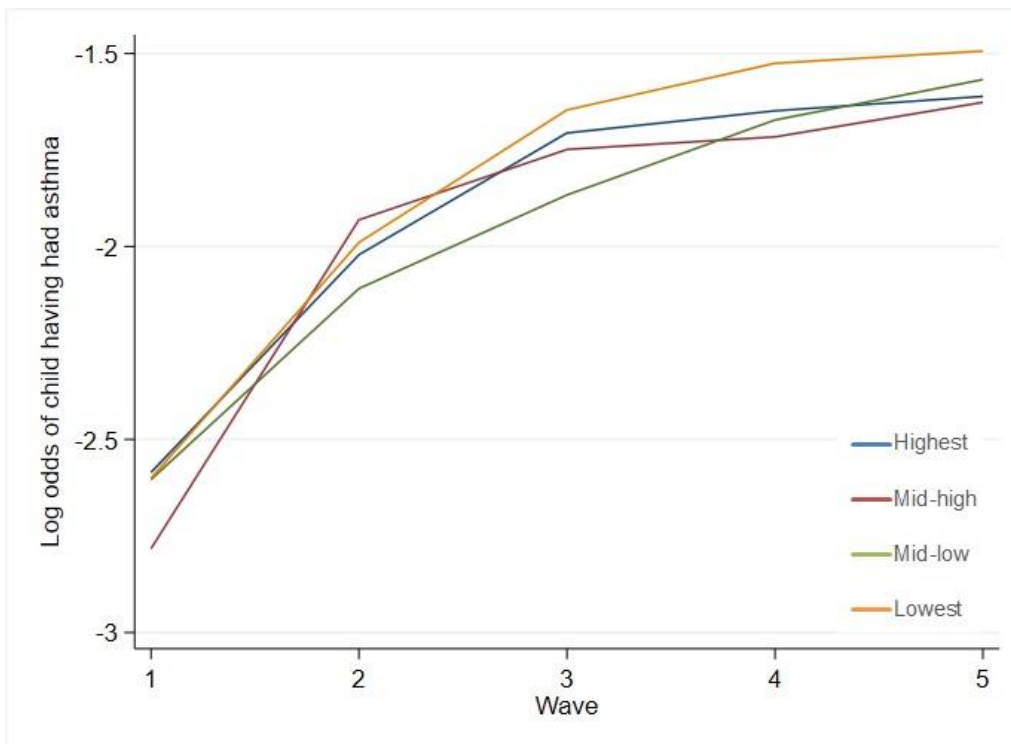
Figure 5.3   The log odds of a child having had asthma throughout the five waves of the MCS based on the annual average NO_2 concentration (separated into quartiles) of the LSOA they lived in during Wave 1.
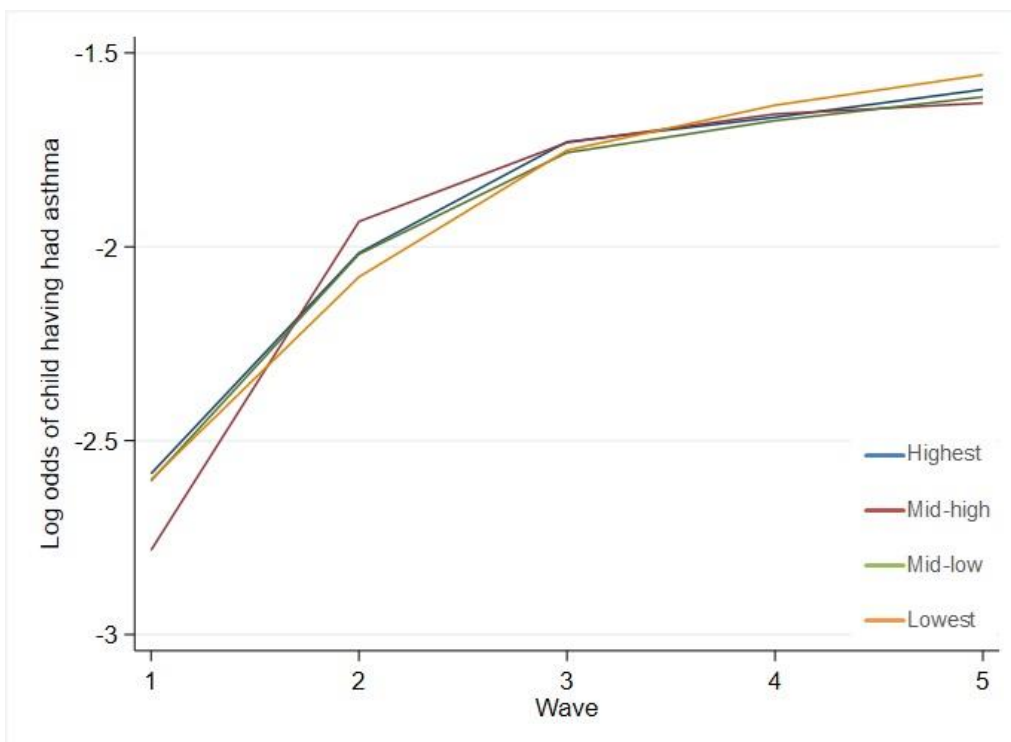
Figure 5.4   The log odds of a child having had asthma throughout the five waves of the MCS based on the annual average NO_2 concentration (separated into quartiles) of the LSOA they lived in during each wave.
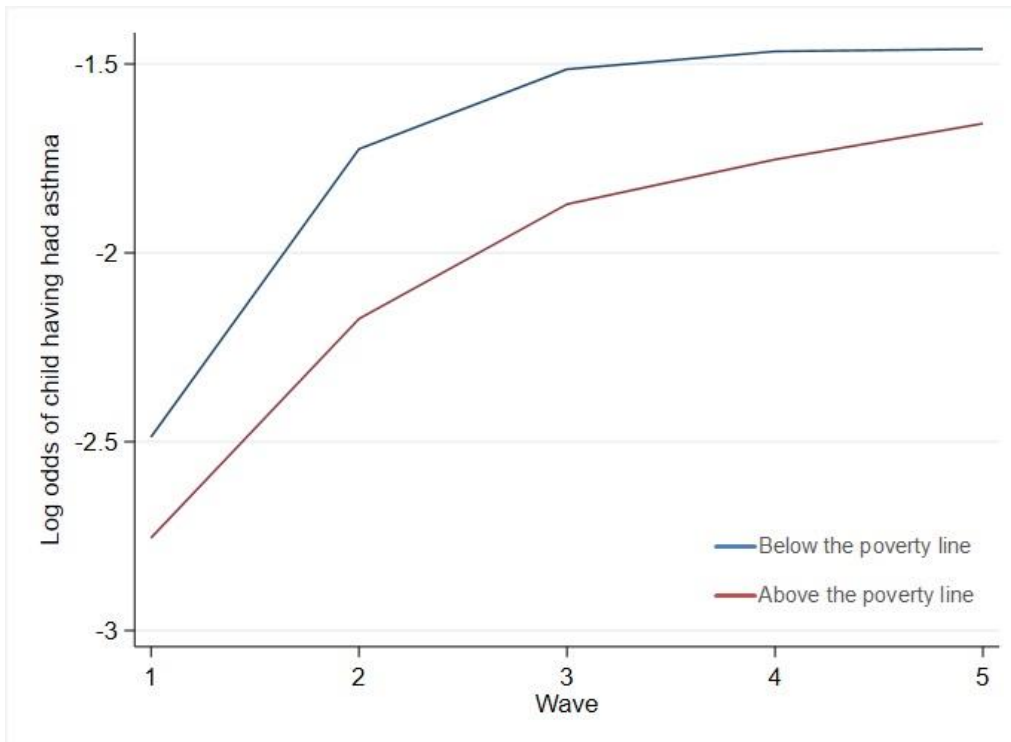
Figure 5.5   The log odds of a child having had asthma throughout the five waves of the MCS based on their socio-economic situation (living below or above the poverty line) of the LSOA they lived in during Wave 1.
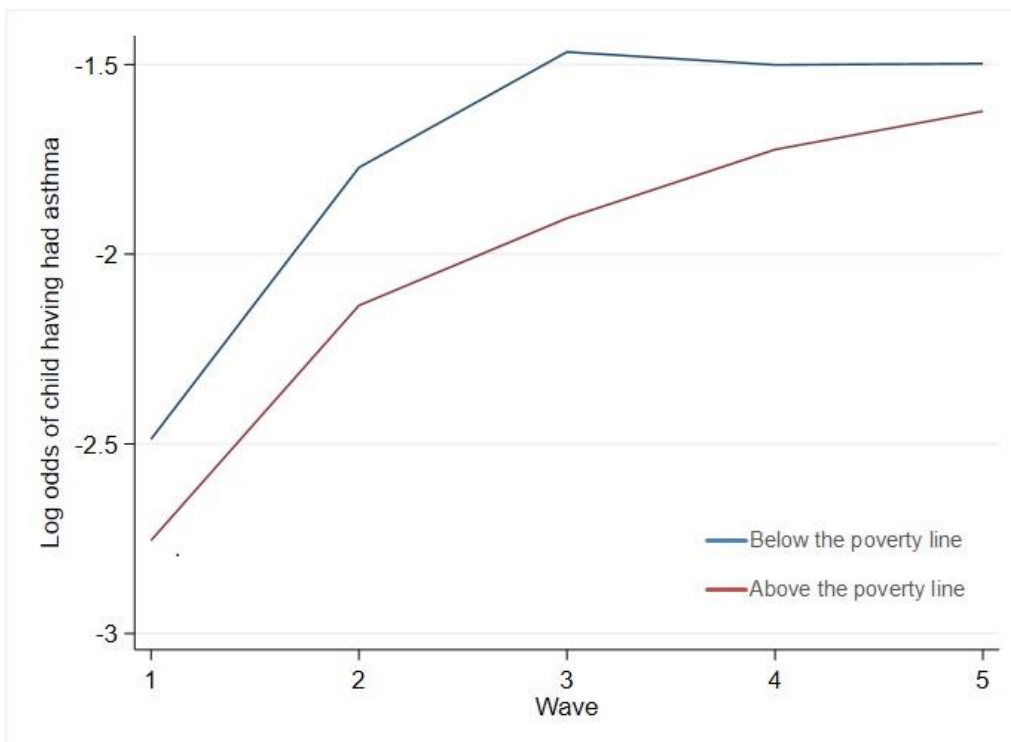
Figure 5.6   The log odds of a child having had asthma throughout the five waves of the MCS based on their socio-economic situation (living below or above the poverty line) of the LSOA they lived in during each wave.

5.3 Time series analysis of wheezing rates

5.3.1 Time series analysis of wheezing rates with initial exposures from Wave 1

Analysis began by examining initial recordings of poverty, IMD and air pollution levels from Wave 1. Table 5.15 presents the results from three separate regression analyses for poverty, IMD and NO_2 pollution at Wave 1. A child that lived below the poverty line at Wave 1 (OR 1.31, CI 1.19 – 1.44) is statistically significantly more likely to have wheezed in the previous 12 months, as is a child that initially lived in an area of higher deprivation (OR 1.32, CI 1.17 – 1.50). A child that lived in an area with a high level of NO_2 pollution (OR 0.95, CI 0.82 – 1.01) is less likely to have wheezed in the previous 12 months, however this relationship was not statistically significant.

Table 5.15 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and NO_2 in Wave 1 on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line (initial)		1.31	***	1.19 - 1.44
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.20	***	1.05 - 1.38
	medium-high	1.43	***	1.27 - 1.62
	high	1.32	***	1.17 - 1.50
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	0.94		0.81 - 1.09
	medium-high	0.98		0.85 - 1.13
	high	0.95		0.82 - 1.09

Table 5.16 shows the results when these variables were included in a single model. Here it can be seen that a child that lived below the poverty line in Wave 1 (OR 1.21, CI 1.09 – 1.35) is statistically significantly more likely to have wheezed in the previous 12 months, as is a child that lived in an area with higher levels of deprivation (OR 1.22, CI 1.05 – 1.41). A child that lived in an area of high NO_2 pollution is statistically significantly less likely to have wheezed in the previous 12 months (OR 0.86, CI 0.74 – 1.00).

Table 5.16 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO_2 in Wave 1 on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line (initial)		1.21	***	1.09 - 1.35
Level of deprivation (IMD) (initial)	low	REF		

		OR		CI 95%
	low-medium	1.17	**	1.03 - 1.34
	medium-high	1.38	***	1.22 - 1.57
	high	1.22	***	1.05 - 1.41
NO$_2$ (level of pollution) (initial)	low	REF		
	low-medium	0.92		0.79 - 1.07
	medium-high	0.94		0.82 - 1.08
	high	0.86	**	0.74 - 1.00

Table 5.17 includes individual variables in the regression. From this analysis, a child that lived in an area of low-medium (OR 1.12, CI 0.98 – 1.28) or medium-high levels of deprivation in Wave 1 (OR 1.28, CI 1.12 – 1.46) is statistically significantly more likely to have wheezed in the previous 12 months. A child that lived in an area of high NO$_2$ pollution in Wave 1 (OR 0.82, CI 0.70 – 0.97) is statistically significantly less likely to have wheezed in the previous 12 months. Furthermore, a female child (OR 0.73, CI 0.67 – 0.80) is also statistically significantly less likely to have wheezed in the previous 12 months, whilst a child that is obese (OR 1.33, CI 1.13 – 1.57), has a mother with asthma (OR 1.86, CI 1.70 – 2.11), has a mother that smokes (OR 1.11, CI 1.01 – 1.23) or lives in an urban area (OR 1.21, CI 1.05 – 1.39) is statistically significantly more likely to have wheezed in the previous 12 months.

Table 5.17 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO$_2$ in Wave 1, and individual level variables, on wheezing rates in children.

Wheeze		OR		CI 95%
Lives below the poverty line (initial)		1.09		0.97 - 1.23
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.12	*	0.98 - 1.28
	medium-high	1.28	***	1.12 - 1.46
	high	1.11		0.96 - 1.29
NO$_2$ (level of pollution) (initial)	low	REF		
	low-medium	0.89		0.76 - 1.03
	medium-high	0.87	*	0.74 - 1.01
	high	0.82	**	0.70 - 0.97
Child is female		0.73	***	0.67 - 0.80
Child is White British		0.96		0.84 - 1.10
Child is obese		1.33	***	1.13 - 1.57
Mother is employed		0.96		0.88 - 1.05
Mother has asthma		1.89	***	1.69 - 2.11
Mother smokes		1.11	**	1.01 - 1.23
Lives in social housing		1.10		0.98 - 1.24
Lives in urban area		1.21	***	1.05 - 1.39

Table 5.18 explores the interactions between poverty, IMD and NO_2 pollution at Wave 1 and how they interact with each other over time to influence wheezing rates among the cohort members. Four interaction terms are analysed, however these are not statistically significant. However, the relationship in the three-way interaction (poverty*IMD*NO_2) is positive, which means that those living in poverty, in a more deprived and polluted area are more likely to have wheezed over time.

Table 5.18 Time series analysis looking at the impact of different exposures on wheezing rates in children, including interactions

Wheeze	OR		CI 95%
Child is female	0.73	***	0.67 - 0.80
Child is White British	0.96		0.84 - 1.09
Child is obese	1.34	***	1.14 - 1.58
Mother is employed	0.96		0.88 - 1.05
Mother has asthma	1.89	***	1.69 - 2.11
Mother smokes	1.12	*	1.01 - 1.23
Lives in social housing	1.11		0.98 - 1.24
Lives in urban area	1.21	**	1.05 - 1.39
Lives below the poverty line (initial)	1.53		0.46 - 5.06
IMD (initial)	0.97		0.73 - 1.31
NO_2 (initial)	1.03		0.80 - 1.35
Poverty*IMD (initial)	0.86		0.53 - 1.40
Poverty*NO_2 (initial)	0.84		0.57 - 1.24
IMD*NO_2 (initial)	0.97		0.87 - 1.07
Poverty*IMD*NO_2 (initial)	1.08		0.92 - 1.28

Table 5.19 builds on Table 5.15 by running a regression that includes all pollutant variables, PM_{10}, $PM_{2.5}$, NO and O_3, in addition to NO_2, as well as regressions looking at IMD and poverty exposure at Wave 1. A child living below the poverty line in Wave 1 (OR 1.31, CI 1.19 – 1.44) is statistically significantly more likely to have wheezed in the previous year, as is a child living in an area of higher deprivation (OR 1.32, CI 1.17 – 1.50). A child that resided in an area of low-medium levels of O_3 pollution in Wave 1 (OR 1.20, CI 1.01 – 1.43) is also statistically significantly more likely to have wheezed in the previous year, whilst a child living in an area of low-medium NO pollution in Wave 1 (OR 0.70, CI 0.56 – 0.88) is statistically significantly less likely to have wheezed in the previous year.

Table 5.19 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and all air pollutants in Wave 1 on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line (initial)		1.31	***	1.19 - 1.44
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.20	***	1.05 - 1.37
	medium-high	1.43	***	1.27 - 1.62
	high	1.32	***	1.17 - 1.50
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	1.18		0.96 - 1.46
	medium-high	1.14		0.83 - 1.58
	high	1.36		0.85 - 2.18
PM_{10} (level of pollution) (initial)	low	REF		
	low-medium	0.91		0.63 - 1.32
	medium-high	1.05		0.70 - 1.57
	high	1.09		0.70 - 1.69
$PM_{2.5}$ (level of pollution) (initial)	low	REF		
	low-medium	0.87		0.64 - 1.18
	medium-high	0.90		0.58 - 1.40
	high	0.86		0.54 - 1.37
NO (level of pollution) (initial)	low	REF		
	low-medium	0.70	***	0.56 - 0.88
	medium-high	0.92		0.67 - 1.27
	high	0.75		0.47 - 1.18
O_3 (level of pollution) (initial)	low	REF		
	low-medium	1.20	**	1.01 - 1.43
	medium-high	1.17		0.90 - 1.52
	high	1.00		0.71 - 1.43

Table 5.20 shows the results of a single regression including all the variables seen in Table 5.19. A child living below the poverty line in Wave 1 (OR 1.21, CI 1.09 – 1.35) is statistically significantly more likely to have wheezed over time, as is a child that lived in an area of higher deprivation in Wave 1 (OR 1.22, CI 1.06 – 1.42), and also a child that lived in an area of low-medium O_3 pollution (OR 1.22, CI 1.02 – 1.46). A child living in an area of low-medium NO pollution in Wave 1 (OR 0.72, CI 0.58 – 0.91) is statistically significantly less likely to have wheezed in the previous year. A child living in an area of high NO_2 (OR 1.05, CI 0.64 – 1.72) or high PM_{10} (OR 1.11, CI 0.71 – 1.74) pollution is more likely to have experienced wheezing over time, although this was not statistically significant. A child living in area of high $PM_{2.5}$ (OR 0.87, CI 0.54 – 1.39) is less likely to have wheezed, although this was again not statistically significant.

Table 5.20 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all air pollutants in Wave 1 on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line (initial)		1.21	***	1.08 - 1.35
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.15	**	1.01 - 1.32
	medium-high	1.37	***	1.20 - 1.56
	high	1.22	***	1.06 - 1.42
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	1.09		0.88 - 1.35
	medium-high	1.00		0.72 - 1.39
	high	1.05		0.64 - 1.72
PM_{10} (level of pollution) (initial)	low	REF		
	low-medium	0.98		0.67 - 1.42
	medium-high	1.10		0.74 - 1.66
	high	1.11		0.71 - 1.74
$PM_{2.5}$ (level of pollution) (initial)	low	REF		
	low-medium	0.86		0.63 - 1.17
	medium-high	0.89		0.57 - 1.39
	high	0.87		0.54 - 1.39
NO (level of pollution) (initial)	low	REF		
	low-medium	0.72	***	0.58 - 0.91
	medium-high	0.96		0.70 - 1.32
	high	0.86		0.54 - 1.38
O_3 (level of pollution) (initial)	low	REF		
	low-medium	1.22	**	1.02 - 1.46
	medium-high	1.15		0.88 - 1.49
	high	0.98		0.69 - 1.39

Table 5.21 includes individual level variables in the regression, and indicates that a child that lived in an area of medium-high deprivation in Wave 1 (OR 1.27, CI 1.11 – 1.45) or an area with low-medium O_3 pollution in Wave 1 (OR 1.22, CI 1.10 – 1.45) is statistically significantly more likely to have wheezed in the previous year, whilst a child that lived in an area of low-medium NO pollution in Wave 1 (OR 0.72, CI 0.57 – 0.91) is statistically significantly less likely to have wheezed in the previous year. Furthermore, a female child (OR 0.74, CI 0.67 – 0.81) is statistically significantly less likely to have wheezed in the previous year, whilst a child that is obese (OR 1.34, CI 1.14 – 1.58), has a mother that has asthma (OR 1.89, CI 1.69 – 2.11), has a mother that smokes (OR 1.11, CI 1.01 – 1.23) or lives in an urban area (OR 1.20, CI 1.05 – 1.39) is statistically significantly more likely to have wheezed in the previous year.

Table 5.21 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all pollutants in Wave 1, and individual level variables, on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line (initial)		1.09		0.97 - 1.22
Level of deprivation (IMD) (initial)	low	REF		
	low-medium	1.10		0.97 - 1.26
	medium-high	1.27	***	1.11 - 1.45
	high	1.12		0.96 - 1.30
NO_2 (level of pollution) (initial)	low	REF		
	low-medium	1.05		0.84 - 1.31
	medium-high	0.92		0.66 - 1.29
	high	0.99		0.60 - 1.63
PM_{10} (level of pollution) (initial)	low	REF		
	low-medium	0.93		0.64 - 1.34
	medium-high	1.02		0.68 - 1.53
	high	1.02		0.65 - 1.59
$PM_{2.5}$ (level of pollution) (initial)	low	REF		
	low-medium	0.89		0.65 - 1.21
	medium-high	0.95		0.61 - 1.48
	high	0.93		0.58 - 1.50
NO (level of pollution) (initial)	low	REF		
	low-medium	0.72	***	0.57 - 0.91
	medium-high	0.95		0.69 - 1.31
	high	0.87		0.54 - 1.39
O_3 (level of pollution) (initial)	low	REF		
	low-medium	1.22	**	1.02 - 1.45
	medium-high	1.11		0.85 - 1.45
	high	0.97		0.68 - 1.40
Child is female		0.74	***	0.67 - 0.81
Child is White British		0.96		0.84 - 1.10
Child is obese		1.34	***	1.14 - 1.58
Mother is employed		0.96		0.88 - 1.04
Mother has asthma		1.89	***	1.69 - 2.11
Mother smokes		1.11	**	1.01 - 1.23
Lives in social housing		1.10		0.98 - 1.23
Lives in urban area		1.20	**	1.04 - 1.39

5.3.2 Time series analysis of wheezing rates with time varying exposures

Building on the analysis presented above, the analysis moves on to explore the impact of poverty, IMD and air pollution concentration as time varying variables on the respiratory health for our cohort, using wheezing as the outcome variable.

Table 5.22 shows the results of three separate regressions, similar to table 5.15. From this, it can be seen that a child living below the poverty line (OR 1.30, CI 1.19 – 1.41) is statistically significantly more likely to have wheezed in the previous year. A child living in an area with higher levels of deprivation (OR 1.37, CI 1.21 – 1.55) is also more likely to have wheezed in the previous year, as is a child living in an increasingly polluted area (OR 1.19, CI 1.05 – 1.36) as measured by NO_2.

Table 5.22 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and NO_2 on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line		1.30	***	1.19 - 1.41
Level of deprivation (IMD)	low	REF		
	low-medium	1.22	***	1.08 - 1.38
	medium-high	1.42	***	1.26 - 1.60
	high	1.37	***	1.21 - 1.55
NO_2 (level of pollution)	low	REF		
	low-medium	1.10	*	0.99 - 1.21
	medium-high	1.18	***	1.05 - 1.32
	high	1.19	***	1.05 - 1.36

Table 5.23 includes all variables of interest in one single regression. A child living below the poverty line (OR 1.18, CI 1.07 – 1.30) is statistically significantly more likely to have wheezed in the previous year. A child living in areas of higher deprivation (OR 1.23, CI 1.07 – 1.41) is also statistically significantly more likely to have wheezed in the previous year, whilst a child living in an area of medium-high NO_2 pollution (OR 1.12, CI 1.00 – 1.26) is also statistically significantly more likely to have wheezed in the previous year.

Table 5.23 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO_2 in Wave 1 on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line		1.18	***	1.07 - 1.30
Level of deprivation (IMD)	low	REF		
	low-medium	1.20	***	1.06 - 1.35
	medium-high	1.33	***	1.18 - 1.51
	high	1.23	***	1.07 - 1.41
NO_2 (level of pollution)	low	REF		
	low-medium	1.07		0.96 - 1.18
	medium-high	1.12	**	1.00 - 1.26
	high	1.07		0.94 - 1.22

Table 5.24 includes individual level variables in the analysis. A child living in an area of low-medium (OR 1.15, CI 1.02 – 1.30) or medium-high levels of deprivation (OR 1.23, CI 1.08 – 1.40) is statistically significantly more likely to have wheezed in the previous 12 months, as is a child that lived in an area of medium-high NO_2 pollution (OR 1.12, CI 0.99 – 1.27). A female child (OR 0.74, CI 0.67 – 0.81) is statistically significantly less likely to have wheezed in the previous 12 months. A child that is obese (OR 1.33, CI 1.13 – 1.57), has a mother that has asthma (OR 1.90, CI 1.70 – 2.12), has a mother that smokes (OR 1.12, CI 1.02 – 1.24) or lives in social housing (OR 1.11, CI 0.99 – 1.25) is statistically significantly more likely to have wheezed in the previous 12 months.

Table 5.24 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and NO_2, and individual level variables, on asthma rates in children

Wheeze		OR		CI 95%
Lives below the poverty line		1.08		0.97 - 1.20
Level of deprivation (IMD)	low	REF		
	low-medium	1.15	**	1.02 - 1.30
	medium-high	1.23	***	1.08 - 1.40
	high	1.11		0.96 - 1.29
NO_2 (level of pollution)	low	REF		
	low-medium	1.04		0.93 - 1.16
	medium-high	1.12	*	0.99 - 1.27
	high	1.10		0.94 - 1.27
Child is female		0.74	***	0.67 - 0.81
Child is White British		1.02		0.90 - 1.17
Child is obese		1.33	***	1.13 - 1.57
Mother is employed		0.97		0.89 - 1.06
Mother has asthma		1.90	***	1.70 - 2.12
Mother smokes		1.12	**	1.02 - 1.24
Lives in social housing		1.11	*	0.99 - 1.25
Lives in urban area		1.09		0.94 - 1.25

Table 5.25 explores the interactions between poverty, IMD and NO_2 pollution over time and how they interact to influence wheezing rates among the cohort members. Four interaction terms are shown, and these are not statistically significant.

Table 5.25 Time series analysis looking at the impact of different exposures on wheezing rates in children, including interactions

Wheeze	OR		CI 95%
Child is female	0.74	***	0.67 - 0.81

Child is White British		1.03		0.90 - 1.17
Child is obese		1.34	***	1.14 - 1.57
Mother is employed		0.97		0.89 - 1.06
Mother has asthma		1.90	***	1.70 - 2.12
Mother smokes		1.13	*	1.02 - 1.25
Lives in social housing		1.11		0.99 - 1.25
Lives in urban area		1.11		0.96 - 1.27
Lives below the poverty line		1.25		0.82 - 1.92
IMD score		1.01		1.00 - 1.01
NO_2		1.01		1.00 - 1.02
Poverty*IMD		0.99		0.98 - 1.01
Poverty*NO_2		1.00		0.98 - 1.02
IMD*NO_2		1.00		1.00 - 1.00
Poverty*IMD*NO_2		1.00		1.00 - 1.00

Table 5.26 includes all pollutants of interest, once again run in separate models from both poverty and IMD. A child living below the poverty line (OR1.30, CI 1.19 – 1.41) is statistically significantly more likely to have wheezed in the previous 12 months throughout their life, as is a child living in an area with higher deprivation (OR 1.37, CI 1.21 – 1.55). A child living in areas of higher $PM_{2.5}$ pollution (OR 1.68, CI 1.22 – 2.32) is also statistically significantly more likely to have wheezed in the previous 12 months. A child living in areas of high NO_2 (OR 0.87, CI 0.63 – 1.20) or PM_{10} (OR 0.81, CI 0.60 – 1.11) pollution is less likely to have had wheezing, whilst a child living in area of high NO pollution (OR 1.13, CI 0.82 – 1.57) is more likely to have experienced recent wheezing over time.

Table 5.26 Time series analysis of three separate models looking at the impact of exposures to poverty, IMD and all air pollutants on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line		1.30	***	1.19 - 1.41
Level of deprivation (IMD)	low	REF		
	low-medium	1.22	***	1.08 - 1.38
	medium-high	1.42	***	1.26 - 1.60
	high	1.37	***	1.21 - 1.55
NO_2 (level of pollution)	low	REF		
	low-medium	1.04		0.90 - 1.19
	medium-high	1.00		0.81 - 1.24
	high	0.87		0.63 - 1.20
PM_{10} (level of pollution)	low	REF		
	low-medium	1.06		0.94 - 1.19
	medium-high	0.95		0.78 - 1.15
	high	0.81		0.60 - 1.11
$PM_{2.5}$ (level of pollution)	low	REF		

		OR		CI 95%
	low-medium	1.31	***	1.16 - 1.47
	medium-high	1.55	***	1.30 - 1.84
	high	1.68	***	1.22 - 2.32
NO (level of pollution)	low	REF		
	low-medium	1.00		0.87 - 1.15
	medium-high	1.02		0.82 - 1.26
	high	1.13		0.82 - 1.57
O_3 (level of pollution)	low	REF		
	low-medium	0.97		0.84 - 1.12
	medium-high	0.93		0.78 - 1.10
	high	1.03		0.84 - 1.26

Table 5.27 runs all these variables in one model. A child living below the poverty line (OR 1.17, CI 1.06 – 1.29) or in an area of higher deprivation (OR 1.33, CI 1.15 – 1.53) is statistically significantly more likely to have wheezed in the previous 12 months. A child living in areas of high PM_{10} pollution (OR 0.73, CI 0.53 – 1.01) or high NO_2 pollution (OR 0.70, CI 0.50 – 0.97) is statistically significantly less likely to have wheezed in the previous 12 months. A child living in areas of higher $PM_{2.5}$ pollution (OR 1.89, CI 1.35 – 2.63) is statistically significantly more likely to have wheezed in the previous 12 months.

Table 5.27 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all air pollutants on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line		1.17	***	1.06 - 1.29
Level of deprivation (IMD)	low	REF		
	low-medium	1.22	***	1.08 - 1.38
	medium-high	1.38	***	1.22 - 1.56
	high	1.33	***	1.15 - 1.53
NO_2 (level of pollution)	low	REF		
	low-medium	0.97		0.84 - 1.11
	medium-high	0.89		0.72 - 1.11
	high	0.70	**	0.50 - 0.97
PM_{10} (level of pollution)	low	REF		
	low-medium	1.05		0.93 - 1.19
	medium-high	0.93		0.77 - 1.13
	high	0.73	*	0.53 - 1.01
$PM_{2.5}$ (level of pollution)	low	REF		
	low-medium	1.35	***	1.20 - 1.52
	medium-high	1.61	***	1.36 - 1.92
	high	1.89	***	1.35 - 2.63
NO (level of pollution)	low	REF		
	low-medium	1.02		0.88 - 1.17
	medium-high	1.04		0.83 - 1.29

	high	1.20		0.86 - 1.66
O_3 (level of pollution)	low	REF		
	low-medium	0.97		0.84 - 1.13
	medium-high	0.92		0.78 - 1.09
	high	0.99		0.81 - 1.21

Table 5.28 builds on this by including the individual level variables. A child living in an area of high deprivation (OR 1.20, CI 1.03 – 1.39) is statistically significantly more likely to have wheezed in the previous 12 months, as is a child living in areas of higher $PM_{2.5}$ pollution (OR 1.89, CI 1.35 – 2.65). A child living in an area of high PM_{10} pollution (OR 0.72, CI 0.52 – 0.99) is statistically significantly less likely to have wheezed in the previous 12 months, as is a child living in an area of high NO_2 pollution (OR 0.72, CI 0.51 – 0.99). A female child (OR 0.74, CI 0.67 – 0.81) is also statistically significantly less likely to have wheezed in the previous 12 months, whilst a child that is obese (OR 1.35, CI 1.14 – 1.59), has a mother that has asthma (OR 1.90, CI 1.70 – 2.12) or a mother that smokes (OR 1.11, CI 1.00 – 1.23) is statistically significantly more likely to have wheezed in the previous 12 months.

Table 5.28 Time series analysis looking at the impact of exposure to different levels of poverty, IMD and all pollutants, and individual level variables, on wheezing rates in children

Wheeze		OR		CI 95%
Lives below the poverty line		1.08		0.97 - 1.20
Level of deprivation (IMD)	low	REF		
	low-medium	1.17	**	1.04 - 1.32
	medium-high	1.26	***	1.11 - 1.44
	high	1.20	**	1.03 - 1.39
NO_2 (level of pollution)	low	REF		
	low-medium	0.94		0.82 - 1.08
	medium-high	0.88		0.70 - 1.09
	high	0.72	**	0.51 - 1.00
PM_{10} (level of pollution)	low	REF		
	low-medium	1.05		0.93 - 1.18
	medium-high	0.91		0.75 - 1.11
	high	0.72	**	0.52 - 0.99
$PM_{2.5}$ (level of pollution)	low	REF		
	low-medium	1.35	***	1.20 - 1.52
	medium-high	1.61	***	1.35 - 1.93
	high	1.89	***	1.35 - 2.65
NO (level of pollution)	low	REF		
	low-medium	1.03		0.89 - 1.19

	medium-high	1.06		0.86 - 1.32
	high	1.22		0.88 - 1.69
O_3 (level of pollution)	low	REF		
	low-medium	0.98		0.84 - 1.13
	medium-high	0.93		0.79 - 1.11
	high	1.02		0.83 - 1.25
Child is female		0.74	***	0.67 - 0.81
Child is White British		1.02		0.89 - 1.16
Child is obese		1.35	***	1.14 - 1.59
Mother is employed		1.00		0.92 - 1.10
Mother has asthma		1.90	***	1.70 - 2.12
Mother smokes		1.11	**	1.00 - 1.23
Lives in social housing		1.10		0.98 - 1.24
Lives in urban area		1.12		0.97 - 1.30

5.3.3 Comparing the effects of initial and time-varying exposures on wheezing

To illustrate the difference between impacts on wheezing rates based on exposures in Wave 1 and over time, Figures 5.7 and 5.8 show the log odds of a parent reporting that a child had wheezing based on the IMD score of the LSOA that they lived in in Wave 1 (5.7) or that they lived in in each wave (5.8). These graphs show that someone living in the least deprived quartile, either initially or over time, is consistently less likely to have had wheezing than someone living in a more deprived area.

Figures 5.9 and 5.10 show the log odds of a child having had wheezing based on the annual average NO_2 concentration of the LSOA that they lived in in Wave 1 (5.9) or that they lived in in each wave (5.10). As with asthma as the outcome variable, these graphs show that there is not much variability over the quartiles, however, someone living in an area with lower levels of NO_2 pollution are shown to be more likely to have had wheezing.

Figures 5.11 and 5.12 show the log odds of a child having had wheezing based on if they lived below the poverty line in Wave 1 (5.11) or in each Wave (5.12). These graphs show that someone living above the poverty line, either initially or over time, is consistently less likely to have had wheezing than someone living below the poverty line.
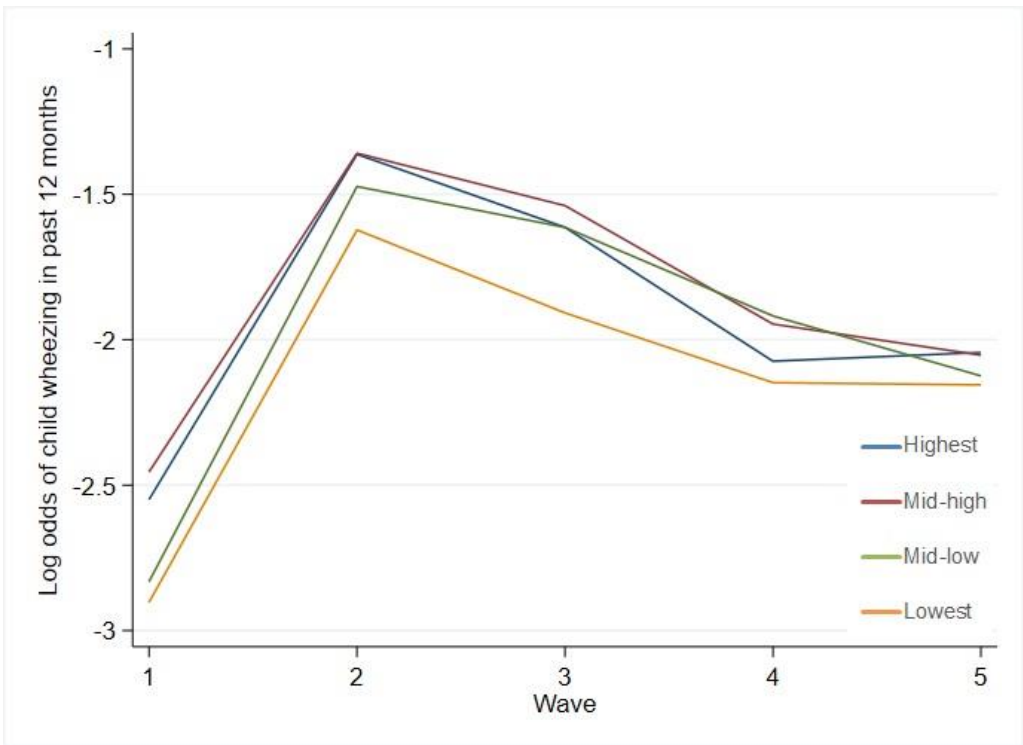
Figure 5.7   The log odds of a child having wheezed in the previous 12 months throughout the five waves of the MCS based on the IMD score (separated into quartiles) of the LSOA they lived in during Wave 1.
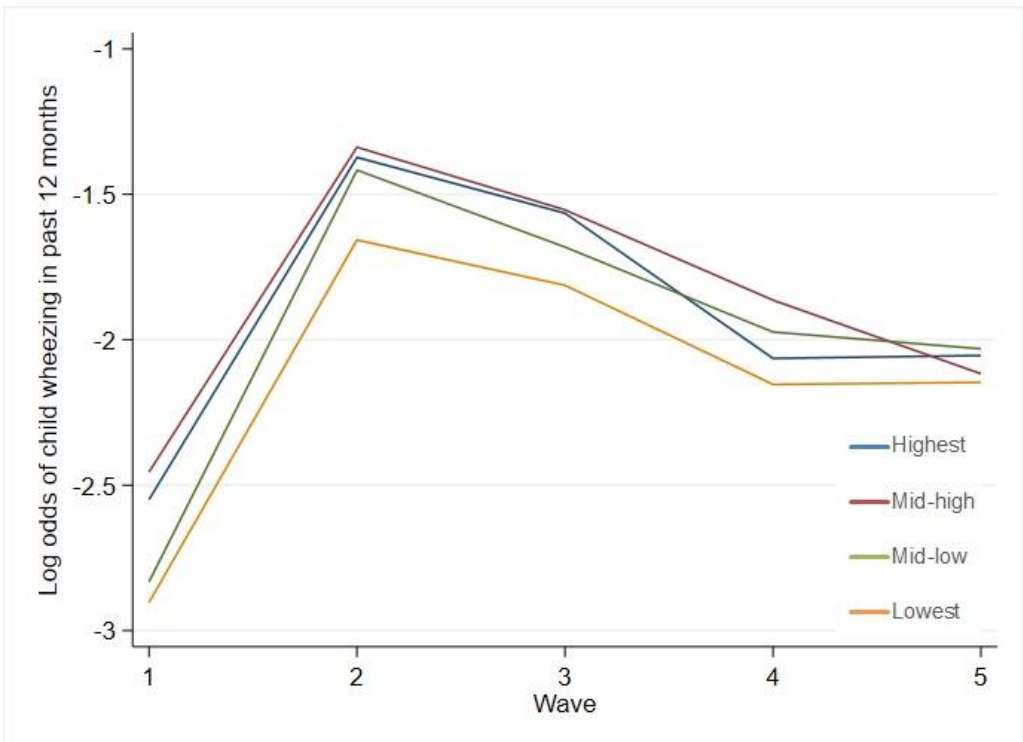
Figure 5.8   The log odds of a child having wheezed in the previous 12 months throughout the five waves of the MCS based on the IMD score (separated into quartiles) of the LSOA they lived in during each wave.
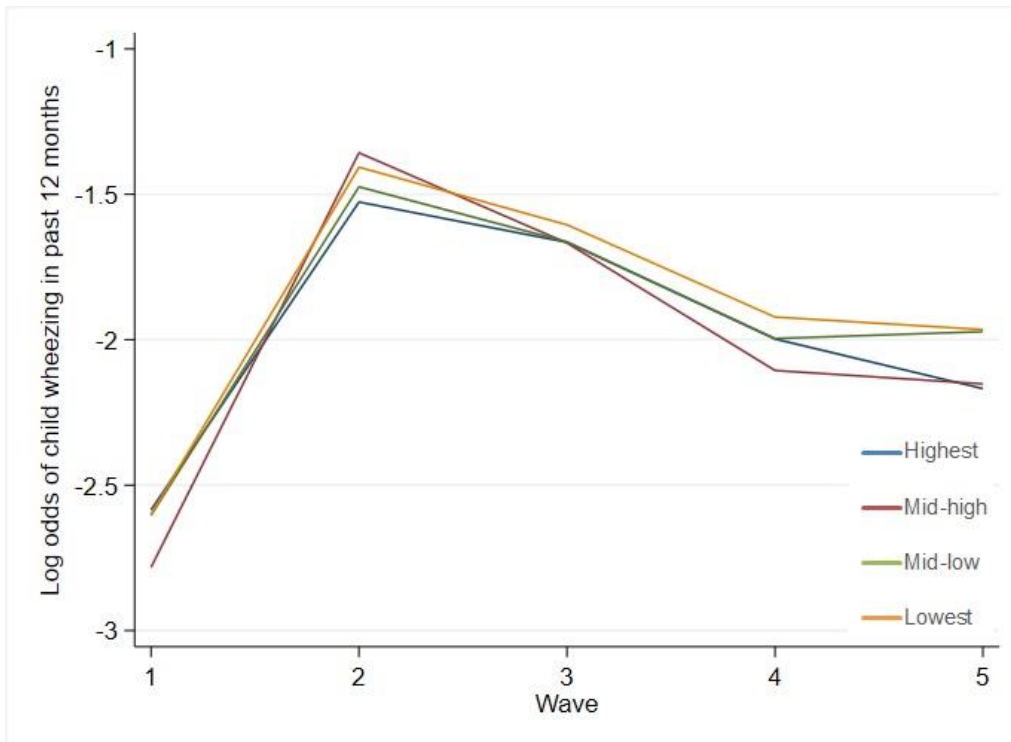
Figure 5.9   The log odds of a child having wheezed in the previous 12 months throughout the five waves of the MCS based on the annual average NO_2 concentration (separated into quartiles) of the LSOA they lived in during Wave 1.
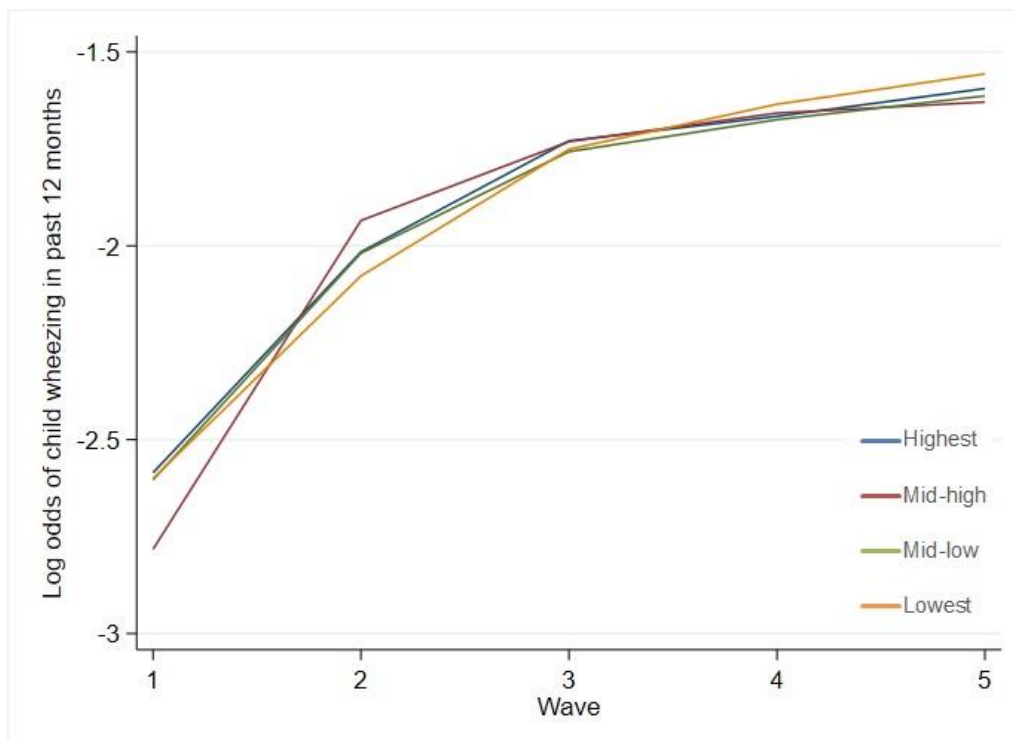
Figure 5.10   The log odds of a child having wheezed in the previous 12 months throughout the five waves of the MCS based on the annual average NO_2 concentration (separated into quartiles) of the LSOA they lived in during each wave.
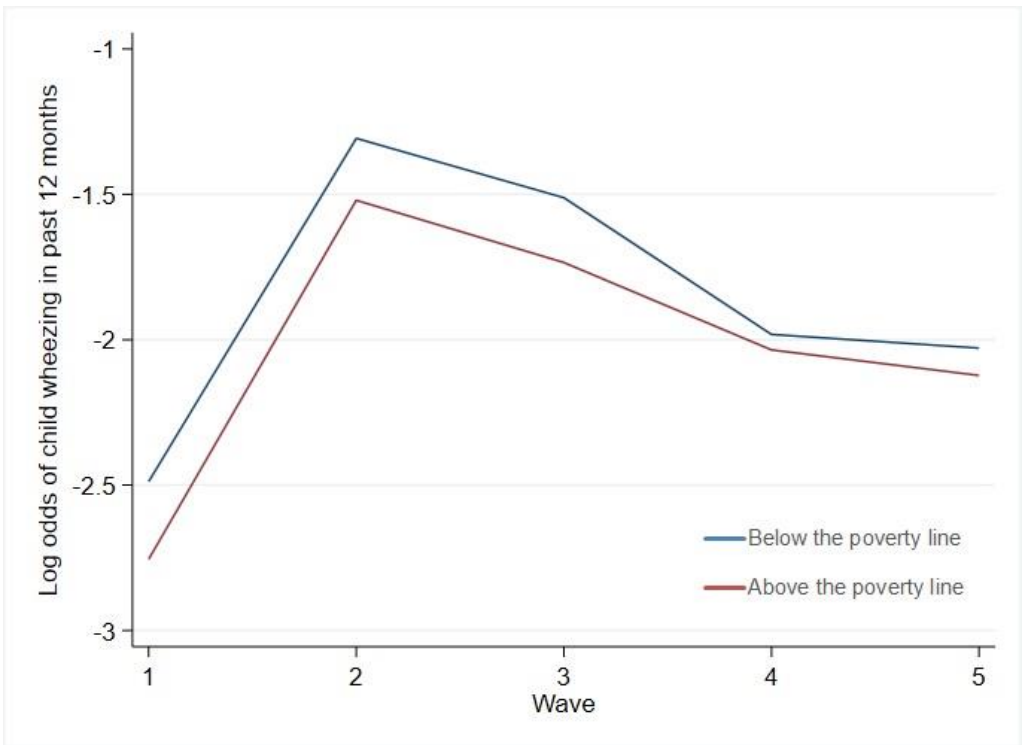
Figure 5.11   The log odds of a child having wheezed in the previous 12 months throughout the five waves of the MCS based on their individual level socio-economic status during Wave 1.
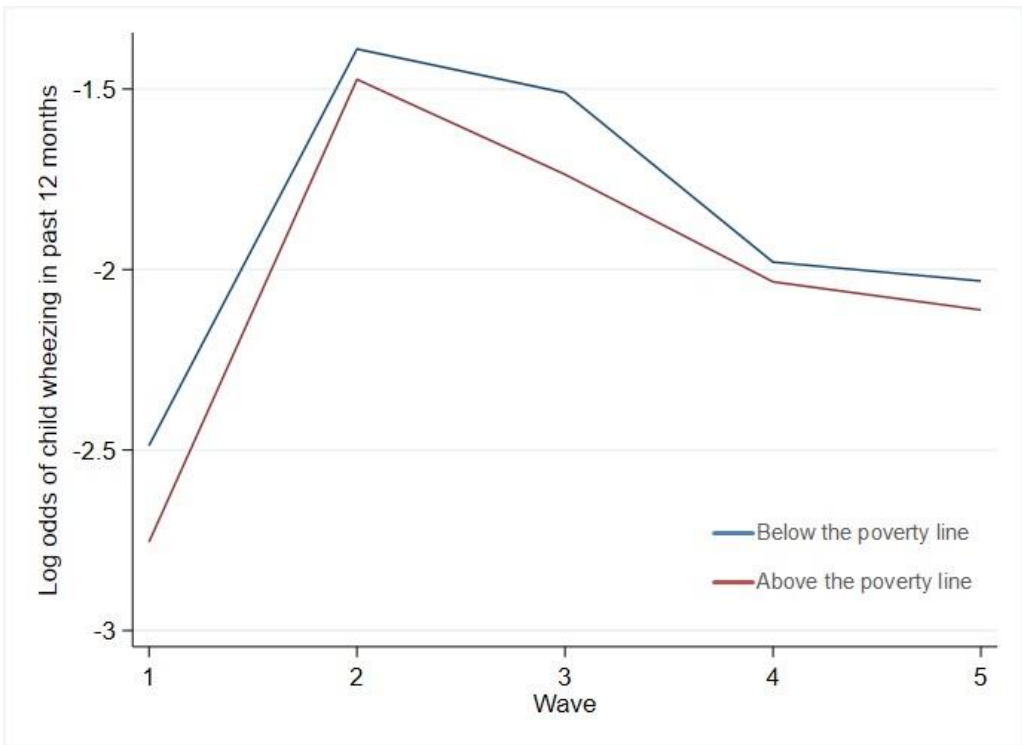
Figure 5.12   The log odds of a child having wheezed in the previous 12 months throughout the five waves of the MCS based on their individual level socio-economic status during each wave.

5.4 Conclusion

Using a time series analysis and the inclusion of initial and time-varying variants of poverty, IMD and pollution variables allowed for an in-depth analysis of their impacts on respiratory health amongst cohort members over time. The analysis presented here, allows for the exploration of the impact that each variable has on respiratory health separately, whilst the inclusion of the interaction terms further builds on these findings and allows assumptions to be made about how said socio-economic status variables interact with each other, as well as exposure to NO_2.

The results show that socio-economic status at both the individual and area level impacted the respiratory health of cohort members and increased their likelihood of both having had asthma and having wheezed in the previous 12 months, regardless of whether or not they were initially exposed to higher levels of poverty and deprivation, or experienced increased poverty and deprivation at later stages in their childhood. However, the findings show that initial exposure to higher levels of poverty in Wave 1 has a greater impact on respiratory health when compared to exposure over time. Conversely, exposure to higher levels of deprivation over time has a greater effect on respiratory health than exposure in Wave 1. The results show that it is important to consider both the critical period of a child's early development whilst also taking into consideration the accumulative effect that said exposures have on their health.

Other findings were similar to those seen in Chapter 5. Female children were less likely to have experienced asthma or wheezing, whilst children that were obese, had a mother with asthma, had a mother that was in employment, lived in social housing or lived in an urban area were all more likely to have experienced asthma or wheezing.

When examining the impacts of air pollution on asthma and wheezing, exposure to higher levels of NO_2 in Wave 1 resulted in decreased rates of asthma and wheezing over time, whilst time-varying exposures to NO_2 results in decreased rates of asthma only. This means that children that have lived in the most polluted areas during their early years (Wave 1) would be less likely to experience asthma or wheezing over time, and children living in the most polluted areas throughout their life would also be less likely to experience respiratory health problems.

Conversely, time-varying exposures to NO_2 resulted in increased rates of wheezing over time. A full discussion examining these results is offered in Chapter 7. Whilst negative associations between respiratory health and air pollution exposure contradict the hypothesis, this Chapter highlights the importance of including a temporal aspect in environmental health research. In an attempt to further explore the impacts on respiratory health and the interactions between variables, multilevel modelling will be used to analyse the data whilst taking both the temporal and spatial aspect of the data into consideration, and this is discussed in Chapter 6.

Chapter 6. Multilevel Modelling

6.1   Introduction

This chapter presents the results of the multilevel modelling analysis. As outlined in Chapter 3, multilevel models account for the nested structure of observations at different levels. Multilevel modelling is an important component of this thesis as it is a robust analytical method allowing for both spatial and temporal data to be taken into consideration during analysis. As with Chapter 4 and 5, the main predicator of interest is air pollution across time. A 3-level logistic multilevel model was used to predict the difference between both parental reported asthma and parental reported wheezing structured by year (level 1), participant (level 2) and MSOA (level 3). Individual covariates are the same as those outlined in Chapter 3 and reported in Chapter 4 and 5. Area level covariates include area level deprivation as measured by the Index of Multiple Deprivation.

In specifying the multilevel model, the LSOA level was originally considered as the spatial scale for level 3. Descriptive statistics indicated that while data was available at the LSOA level for all of MCS waves, there was a large proportion of LSOAs in which there was only one cohort member. For example, 38% of LSOAs had only one cohort member residing in them in Wave 5 (Table 6.1 and Table 6.2). However, moving up one administrative boundary to MSOA level, only 18% of MSOAs had only one cohort number. Similarly, only 3% of LSOAs had 11 or more respondents at this wave, compared to 39% of MSOAs. Although the issue of small numbers remains even using the MSOA administrative boundary, aggregating the IMD to Local Authority or regional level risked introducing ecological fallacy into the analysis (Morrissey et al., 2021b). Based on these considerations, air pollution data and IMD was aggregated up to the MSOA level. Furthermore, there is the potential that London, a wealthy yet polluted city (Font et al., 2019), could skew the results of the analyses. To counteract this, analyses were also run for England with London excluded, as well as for London only, the results of which are available in Section 6.5.

Table 6.1 Number of respondents per LSOA (%)

No. of respondents	Wave				
	1	2	3	4	5
1	2.1	20.9	28.0	32.8	38.0
2-10	36.0	60.4	62.3	61.1	58.9
11+	61.8	18.7	9.7	6.1	3.1

Table 6.2 Number of respondents per MSOA (%)

No. of respondents	Wave				
	1	2	3	4	5
1	1.2	11.4	13.7	15.2	18.3
2-10	5.7	22.3	30.9	36.6	42.6
11+	93.1	66.3	55.5	48.2	39.1

Results are presented for both the random part parameters and the fixed part parameters. As before, both asthma and wheezing are included as the indicators of respiratory health, and analyses are run for models focussing only on NO_2. Interaction terms are also included in the analysis with the intent of answering the hypothesis posed in this thesis:

- The association between respiratory health and air pollution is stronger amongst individuals of lower, compared to higher, socio-economic status.

- Area level deprivation will interact with individual socio-economic status so that the impact of pollution on respiratory health is stronger for people with low socio-economic status living in the most deprived areas than people with low socio-economic status living in less deprived areas.

Following the presentation of the results examining England as a whole, the results from the analyses focussing on England excluding London, and London only are reported. Again, baseline, individual level, area level and interaction models are presented. Separate multilevel models were also conducted for England, England excluding London, and London only for all other pollutants, as were multilevel models including interactions. These are not discussed here but are presented in Tables D.1 to D.16 in Appendix D.

6.2   Multilevel modelling analysis of asthma prevalence

This section will examine the outputs for the asthma model, first presenting the baseline model before building on this by including individual and area level variables as well as interaction terms. Using the Deviance Information Criterion

(DIC) presented during modelling, the models presented in this chapter are estimated to be the best fit for the analyses.

6.2.1 Baseline model

Table 6.3 depicts the baseline model (null model) for asthma. Table 6.3 shows the between-MSOA variance is estimated to be 0.002, whilst the within-MSOA (between-individual) variance is estimated to be 19.93. These results allow for the intraclass correlation coefficient (ICC) to be calculated, which gives an ICC 0.001 for level 3 (MSOA level) and 0.86 for the level 2 (individual level). From this, the ICC can be extrapolated for level 1 (time level) as the total variance must add up to 1, giving an ICC of 0.14.

Using the ICC, the variance partition coefficient (VPC) can be calculated, giving level 3, 2 and 1 a VPC of 0.05, 85.83 and 14.12 respectively. This means that less than 1% (0.05%) of the variation in asthma lies between MSOAs, 86% of the variation in asthma lies between individuals, and 14% of the variation in asthma outcomes lies between waves.

Table 6.3 Asthma baseline multilevel model

Random part parameters	Mean	95% CI		VPC
Level 3: MSOA	0.002	0.001	0.003	0.05
Level 2: Individual	19.93	17.04	22.16	85.83
Level 1: Wave				14.12

6.2.2 Including individual level variables

Table 6.4.1 builds on the preliminary null model by including variables at the individual level. This table shows that a female child (OR 0.46, CI 0.36 – 0.58) is less likely to have had asthma over time, and is statistically significant. This finding is in line with results from the cross-sectional and time series analysis. A child that is mixed-race (OR 1.74, CI 0.98 – 2.75), is obese (OR 1.71, CI 1.23 – 2.27), has a mother that is employed (OR 1.21, CI 1.00 – 1.41), has a mother with asthma (OR 9.61, CI 7.27 – 12.45), has a mother that smokes (OR 1.41, CI 1.14 – 1.71), lives in an urban area (OR1.82, CI 1.33 – 2.59) or lives below the poverty line (OR 1.41, CI 1.18 – 1.64) is statistically significantly more likely to have had asthma throughout their life. Again, these findings are in line with findings from previous results from cross-sectional and time series analyses.

The random parts parameters table in Table 6.4.1 shows that the between-MSOA variance (mean) is 0.05 and the within-MSOA variance is 18.75. The VPC was calculated to show that around 1% of the variation in asthma lies between MSOAs and 85% of the variation in asthma lies within MSOAs. The variation in asthma between waves is around 13%.

6.2.3 Including area level variables (IMD and NO_2)

Table 6.4.2 includes the area level variables of IMD and annual average NO_2 concentrations within the analysis. To understand if non-linearities exist, IMD is included as a decile variable, and NO_2 is included as a quartile variable, ranging from the least polluted or deprived to the most polluted or deprived. These results show that a female child (OR 0.46, CI 0.35 – 0.56) or a child that is Pakistani or Bangladeshi (OR 0.70, CI 0.43 – 1.05) is statistically significantly less likely to have had asthma over time.

A child that is mixed-race (OR 1.74, CI 0.98 – 2.75), is obese (OR 1.68, CI 1.23 – 2.24), has a mother that is employed (OR 1.23, CI 1.04 – 1.45), has a mother with asthma (OR 9.26, CI 7.05 – 12.01), has a mother that smokes (OR 1.27, CI 1.03 – 1.56) or lives below the poverty line (OR 1.27, CI 1.03 – 1.53) is statistically significantly more likely to have had asthma over time. A child that lives in an increasingly deprived area is also more likely to have had asthma (OR 3.50, CI 2.18 – 5.56) which is significantly significant. A child that lives in an area of high NO_2 pollution is less likely to have had asthma (OR 0.91, CI 0.61 – 1.33), however this is not statistically significant. These findings are again similar to the results found in the cross-sectional and time series analysis.

The random parts parameters table in Table 6.4.2 shows that the between-MSOA variance is 0.001 and the within-MSOA variance is 19.12. The VPC shows that less than 1% (0.04%) of the variation in asthma lies between MSOAs and 85.32% of the variation in asthma lies within MSOAs. As a result, 14.64% of the variation in asthma lies between waves.

Table 6.4 Asthma individual and area level multilevel models

Fixed part parameters		6.4.1 OR		95% CI		6.4.2 OR		95% CI	
Child is female		0.46	***	0.36	0.58	0.46	***	0.35	0.56
Ethnicity	White	REF				REF			
	Mixed	1.74	*	0.98	2.75	1.67	*	0.95	2.70
	Indian	1.02		0.55	1.77	0.86		0.44	1.45
	Pakistani & Bangladeshi	0.93		0.61	1.35	0.70	*	0.43	1.05
	Black	0.82		0.45	1.33	0.66		0.37	1.08
	Other	0.91		0.37	1.87	0.83		0.32	1.86
Child is obese		1.71	**	1.23	2.27	1.68	**	1.23	2.24
Mother is employed		1.21	*	1.00	1.41	1.23	*	1.04	1.45
Mother has asthma		9.61	***	7.27	12.45	9.26	***	7.05	12.01
Mother smokes		1.41	***	1.14	1.71	1.27	*	1.03	1.56
Lives in urban area		1.82	***	1.33	2.59	1.35		0.82	1.91
Lives below the poverty line		1.41	***	1.18	1.64	1.27	*	1.03	1.53
IMD	1					REF			
	2					1.33		0.86	2.01
	3					1.27		0.82	1.91
	4					1.60	*	1.02	2.36
	5					2.49	***	1.60	3.82
	6					2.91	***	1.85	4.42
	7					2.55	***	1.68	4.03
	8					3.00	***	1.89	4.72
	9					3.12	***	1.93	5.25
	10					3.50	***	2.18	5.56

		Mean	95% CI		VPC	Mean	95% CI		VPC
NO$_2$	low					REF			
	mid-low					0.98	0.74	1.33	
	mid-high					1.01	0.75	1.34	
	high					0.91	0.61	1.33	
Random part parameters		Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA		0.05	0.01	0.16	1.44	0.001	0.001	0.002	0.04
Level 2: Individual		18.75	17.17	20.88	85.07	19.12	17.47	20.77	85.32
Level 3: Wave					13.49				14.64

6.3 Multilevel modelling analysis of wheezing prevalence

This section details the multilevel models that examine the impact on wheezing as the proxy for respiratory health. This section will begin with examining the results from the baseline null model before building on this by including individual and area level variables as well as interaction terms.

6.3.1 Baseline model

Table 6.5 presents the null model for wheezing. This table shows that the between-MSOA variance is estimated to be around 0.02, whilst the within-MSOA variance is estimated to be around 4.30. The ICC is calculated as 0.01 for the MSOA level and 0.57 for the individual level. These results can be interpreted as a VPC of 0.51 and 56.64 respectively. This means that less than 1% (0.51%) of the variation in wheezing lies between MSOAs, 56.64% of the variation in wheezing lies between individuals and 42.85% of the variation lies between waves.

Comparing these results to the null model for asthma, presented in Table 6.3, whilst variation between MSOAs is still under 1%, there is a higher variation seen in wheezing. Conversely variation for wheezing within-MSOAs, or between individuals, is much lower compared to variation for asthma. Furthermore, variation between waves is much higher for wheezing rates when compared to temporal variation in asthma rates. This could mean that wheezing rates are more variable over time, as an individual could experience wheezing temporarily, when compared to asthma rates, which may be more fixed.

Table 6.5 Wheeze baseline multilevel model

Random part parameters	Mean	95% CI		VPC
Level 3: MSOA	0.02	0.003	0.04	0.51
Level 2: Individual	4.30	3.91	4.67	56.64
Level 1: Wave				42.85

6.3.2 Including individual variables

Table 6.6.1 builds on the null model by including individual level variables. This table shows that a female child (OR 0.64, CI 0.57 – 0.72) is less likely to have wheezed in the previous 12 months, as is a child that is Black (OR 0.77, CI 0.58 – 0.99) or has a mother that is employed (OR 0.88, CI 0.79 – 0.97). A child that

is obese (OR 1.44, CI 1.19 – 1.72), has a mother with asthma (OR 2.70, CI 2.34 – 3.10), has a mother that smokes (OR 1.28, CI 1.14 – 1.45) or lives in an urban area (OR 1.34, CI 1.15 – 1.60), is statistically significantly more likely to have wheezed in the previous 12 months.

The random parts parameters table in Table 6.6.1 shows that including individual variables the between-MSOA variance is 0.001 and the within-MSOA variance is 4.08. Less than 1% (0.04%) of the variation in wheezing lies between MSOAs and 55.35% of the variation lies between individuals. The between wave variation was calculated to be 44.61%.

6.3.3 Including area level variables (IMD and NO$_2$)

Table 6.6.2 shows further development of the multilevel model to also include the area level variables of IMD and annual average NO$_2$ concentrations. This table shows that a female child (OR 0.65, CI 0.58 – 0.72) or a child with a mother in employment (OR 0.87, CI 0.78 – 0.97) is statistically significantly less likely to have wheezed in the last year. A child that is obese (OR 1.44, CI 1.18 – 1.76), has a mother with asthma (OR 2.70, CI 2.32 – 3.12), has a mother that smokes (OR 1.25, CI 1.10 – 1.41) or lives in an urban area (OR 1.49, CI 1.24 – 1.79) is statistically significantly more likely to have wheezed in the last year. A child that lives in the most deprived area (OR 1.42, CI 1.04 – 1.84) is statistically significantly more likely to have had wheezing in the previous year, whilst a child living in an area with high NO2 pollution (OR 0.64, CI 0.51 – 0.77) is statistically significantly less likely to have wheezed in the previous year.

The random parts parameters table in Table 6.6.2 shows that the between-MSOA variance is 0.001 and the within-MSOA variance is 4.11. Less than 1% (0.02%) of the variation in wheezing lies between MSOAs and 55.52% of the variation in wheezing lies between individuals. The between wave variation was calculated to be 44.46%.

Table 6.6 Wheeze individual and area level multilevel models

Fixed part parameters		6.6.1 OR		95% CI		6.6.2 OR		95% CI	
Child is female		0.64	***	0.57	0.72	0.65	***	0.58	0.72
Ethnicity	White	REF				REF			
	Mixed	1.09		0.80	1.43	1.20		0.91	1.55
	Indian	1.05		0.76	1.41	1.22		0.87	1.64
	Pakistani & Bangladeshi	0.89		0.73	1.08	0.97		0.77	1.19
	Black	0.77	*	0.58	0.99	0.96		0.69	1.24
	Other	0.82		0.51	1.24	0.98		0.59	1.49
Child is obese		1.44	***	1.19	1.72	1.44	***	1.18	1.76
Mother is employed		0.88	**	0.79	0.97	0.87	**	0.78	0.97
Mother has asthma		2.70	***	2.34	3.10	2.70	***	2.32	3.12
Mother smokes		1.28	***	1.14	1.45	1.25	**	1.10	1.41
Lives in urban area		1.34	***	1.15	1.60	1.49	***	1.24	1.79
Lives below the poverty line		1.05		0.94	1.18	1.04		0.91	1.16
IMD	1					REF			
	2					1.04		0.81	1.31
	3					1.14		0.89	1.41
	4					1.26	*	0.98	1.58
	5					1.37	**	1.06	1.73
	6					1.36	**	1.06	1.71
	7					1.24		0.96	1.59
	8					1.10		0.83	1.39
	9					1.13		0.84	1.46
	10					1.42	*	1.04	1.84

		Mean	95% CI		VPC	Mean	95% CI		VPC
NO$_2$	low				REF				
	mid-low				0.88		0.74	1.04	
	mid-high				0.84 *		0.69	0.99	
	high				0.64 ***		0.51	0.77	
Random part parameters		Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA		0.001	0.001	0.003	0.04	0.001	0.0004	0.001	0.02
Level 2: Individual		4.08	3.72	4.45	55.35	4.11	3.70	4.56	55.52
Level 3: Wave					44.61				44.46

6.4 Including interactions in multilevel models

The models previously presented in this Chapter assume that the effects of the different covariates are additive, however this is not always the case. Indeed, the hypotheses of this thesis is specifically interested in the interaction between socio-economic status, both at the individual and area level, and air pollution. An interaction between two or more variables suggests that the effect of each variable depends on the value of the other variable. For example, that the effect of NO_2 exposure on asthma or wheezing prevalence depends on the level of deprivation of the area. This section will first examine the effects of the interactions on asthma before moving onto examine wheezing.

6.4.1 Interactions in asthma multilevel models

Table 6.7 presents the results from multilevel model that included interactions between individual level socio-economic status (poverty), area level socio-economic status (IMD) and NO_2 pollution. The interaction between poverty and IMD, representing a child that lives below the poverty line and in an area of increased deprivation (OR 1.01, CI 0.99 – 1.02), shows that they are more likely to have had asthma, however this was not statistically significant. The results show no clear association between a child living below the poverty line in an area of higher NO_2 pollution (OR 1.00, CI 0.98 – 1.02), or a child living in a more deprived area that has a higher level of NO_2 pollution (OR 1.00, CI 1.00 – 1.00), or a child that lives below the poverty line, in a more deprived area that also experiences high levels of NO_2 (OR 1.00, CI 1.00 – 1.00). The random parts parameters table in Table 6.7 shows that the between-MSOA variance is 0.001 and the within-MSOA variance is 18.63. Less than 1% (0.03%) of the variation in asthma lies between MSOAs, 84.99% of the variation in asthma lies between individuals and 14.98% of the variation lies between waves.

Table 6.7 Asthma multilevel models with interaction terms

Fixed part parameters		OR		95% CI	
Child is female		0.45	***	0.36	0.56
Ethnicity	White	REF			
	Mixed	1.83	*	1.01	2.85
	Indian	1.08		0.56	1.91
	Pakistani & Bangladeshi	0.80		0.51	1.24
	Black	0.89		0.51	1.49
	Other	0.99		0.40	2.06
Child is obese		1.68	***	1.22	2.22
Mother is employed		1.24	*	1.04	1.48
Mother has asthma		9.43	***	7.19	12.46
Mother smokes		1.30	**	1.04	1.58
Lives in urban area		1.79	***	1.20	2.46
Lives below poverty line		1.32	**	1.07	1.63
IMD		1.02	***	1.01	1.03
NO_2		0.98	**	0.97	1.00
Poverty*IMD		1.01		0.99	1.02
Poverty*NO_2		1.00		0.98	1.02
IMD*NO_2		1.00		1.00	1.00
Poverty*IMD*NO_2		1.00		1.00	1.00

Random part parameters	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0004	0.002	0.03
Level 2: Individual	18.63	17.15	20.37	84.99
Level 3: Wave				14.98

6.4.2 Interactions in wheezing multilevel models

The interaction results presented in Table 6.8 show that a child that lives below the poverty line and in an area of increased deprivation (OR 0.99, CI 0.99 – 1.00) is less likely to have wheezed in the previous year, however this was not statistically significant. The results show no association between a child living below the poverty line in an area of higher NO_2 pollution (OR 1.00, CI 0.98 – 1.01), or a child living in a more deprived area that has a higher level of NO_2 pollution (OR 1.00, CI 1.00 – 1.00), or a child that lives below the poverty line, in a more deprived area that also experiences high levels of NO_2 (OR 1.00, CI 1.00 – 1.00), and these results are also not statistically significant.

The random parts parameters table show that the between-MSOA variance presented in this model is 0.001 and the within-MSOA variance is 4.09. Less than 1% (0.03%) of the variation in wheezing lies between MSOAs, 55.41% of the variation in asthma lies between individuals and 44.56% of the variation lies between waves.

Table 6.8 Wheeze multilevel models with interaction terms

	6.6.1			
Fixed part parameters	OR		95% CI	
Child is female	0.65	***	0.57	0.73
Ethnicity White	REF			
Mixed	1.13		0.81	1.51
Indian	1.08		0.78	1.44
Pakistani & Bangladeshi	0.89		0.71	1.09
Black	0.84		0.61	1.11
Other	0.88		0.53	1.34
Child is obese	1.45	***	1.18	1.76
Mother is employed	0.87	**	0.78	0.98
Mother has asthma	2.66	***	2.29	3.08
Mother smokes	1.23	**	1.08	1.39
Lives in urban area	1.30	**	1.05	1.60
Lives below poverty line	1.04		0.92	1.20
IMD	1.01	***	1.00	1.01
NO_2	0.99		0.99	1.00
Poverty*IMD	0.99		0.99	1.00
Poverty*NO_2	1.00		0.98	1.01
IMD*NO_2	1.00		1.00	1.00
Poverty*IMD*NO_2	1.00		1.00	1.00

Random part parameters	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0003	0.002	0.03
Level 2: Individual	4.09	3.67	4.47	55.41
Level 3: Wave				44.56

6.5 Multilevel model analysis focussing on England without London and London only

The multilevel models presented previously were also run examining England excluding London, and for London only. This allows for comparisons to be drawn between the three different models and to account for any potential 'London effect' in the data, whereby wealthier individuals living in the city would also be more likely to live in area with higher pollution levels, for example due to congestion. This could potentially skew data in the models examining England as a whole that have already been presented in this Chapter. Table E.1 in Appendix E details the different IMD scores and air pollution concentrations seen in the three different geographies. It can be seen that whilst London has a smaller range in IMD score, the mean is higher, so people in London would be generally wealthier compared to the other two geographies. Looking at NO_2 it can be seen that London again has a smaller range, yet a much higher mean.

6.5.1 Multilevel modelling analysis of asthma

This section will examine the outputs for the asthma models for England excluding London, and London only. First the baseline models will be presented before including individual and area level variables, and finally interaction terms. As before, using the DIC presented during modelling, the models presented in this chapter are estimated to be the best fit for the analyses.

6.5.1.1 Baseline model

Table 6.9 presents the baseline models for England excluding London (Table 6.9.1), and London only (Table 6.9.2). Beginning with the England excluding London model, Table 6.9.1 shows that the between-MSOA variance is estimated to be around 0.05, whilst the within-MSOA variance is estimated to be around 19.40. The VPC was calculated to show that 1.49% of the variation in asthma lies between MSOAs, whilst 85.50% of the variation in asthma lies between individuals and 13.01% of the variation lies between waves.

Looking at London only, Table 6.9.2 shows that the between-MSOA variance is estimated to be around 0.003, whilst the within-MSOA variance is estimated to be around 21.34. The VPC shows that less than 1% (0.08%) of the variation in

asthma in London lies between MSOAs, 86.64% of the variation in asthma lies between individuals and 13.28% of the variation lies between waves.

Table 6.9 Asthma baseline multilevel models

Random part parameters	*6.9.1 Excluding London*				*6.9.2 London only*			
	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.05	0.01	0.14	1.49	0.003	0.001	0.01	0.08
Level 2: Individual	19.4	17.03	21.32	85.50	21.34	16.81	27.54	86.64
Level 1: Wave				13.01				13.28

6.5.1.2   Including individual variables

Table 6.10.1 and Table 6.11.1 builds on the previous null models by including individual level variables. Beginning with England excluding London, Table 6.10.1 shows that a female child (OR 0.46, CI 0.36 – 0.57) is statistically significantly less likely to have had asthma. A child that is mixed-race (OR 2.27, CI 1.20 – 3.99), is obese (OR 1.90, CI 1.34 – 2.62), has a mother in employment (OR 1.18, CI 0.98 – 1.41), has a mother that has asthma (OR 8.37, CI 6.03 – 11.39), has a mother that smokes (OR 1.39, CI 1.11 – 1.71), lives in an urban area (OR 1.97, CI 1.36 – 2.79) or lives below the poverty line (OR 1.40, CI 1.15 – 1.68) is statistically significantly more likely to have had asthma. The random parts parameters table in Table 6.10.1 also shows that the between-MSOA variance is 0.003 and the within-MSOA variance is 18.71. Less than 1% (0.08%) of the variation in asthma lies between MSOAs, 85.04% of the variation in asthma lies between individuals and 14.88% of the variation lies between waves.

For London only, Table 6.11.1 shows that a female child (OR 0.46, CI 0.22 – 0.79) is statistically significantly less likely to have had asthma, whilst a child that has a mother in employment (OR 1.51, CI 0.96 – 2.28), or has a mother with asthma (OR 24.42, CI 10.27 – 52.15) is statistically significantly more likely to have had asthma. The random parts parameters table here shows that the between-MSOA variance is 0.03 and the within-MSOA variance is 21.34. Roughly around 1% (0.98%) of the variation in asthma lies between MSOAs, 86.64% of the variation in asthma lies between individuals and 12.38% of the variation lies between waves.

6.5.1.3 Including area level variables (IMD and NO_2)

Adding area level deprivation (IMD) and annual average NO_2 concentrations the models, Table 6.10.2 and Table 6.11.2 presents the results from these multilevel models. Again IMD is included as a decile variable, and NO_2 is included as a quartile variable, ranging from the least polluted or deprived to the most polluted or deprived.

In Table 6.10.2, in England excluding London, a female child (OR 0.45, CI 0.35 – 0.56) or a child that lives in an area of mid-high NO_2 pollution (OR 0.72, CI 0.50 – 1.06) is statistically significantly less likely to have had asthma. A child that is mixed-race (OR 2.15, CI 1.08 – 3.81), is obese (OR 1.84, CI 1.30 – 2.51), has a mother with asthma (OR 8.01, CI 5.92 – 10.54), has a mother that smokes (OR 1.25, CI 0.98 – 1.54), lives below the poverty line (OR 1.23, CI 1.01 – 1.52), lives in a highly deprived area (OR 4.30, CI 2.11 – 6.87) is statistically significantly more likely to have had asthma. The random parts parameters table in Table 6.10.2 shows that the between-MSOA variance is 0.002 and the within-MSOA variance is 19.05. Less than 1% (0.05%) of the variation in asthma lies between MSOAs, 85.27% of the variation in asthma lies between individuals and 14.68% of the variation lies between waves.

For London, Table 6.11.2 shows that a female child (OR 0.44, CI 0.22 – 0.78) or a child that lives in a highly deprived area (OR 0.40, CI 0.08 – 1.18) is statistically significantly less likely to have had asthma. A child that has a mother in employment (OR 1.55, CI 0.96 – 2.45), has a mother with asthma (OR 40.84, CI 15.77 – 97.63) or lives in an urban area (OR 6.95, CI 1.07 – 24.90) is statistically significantly more likely to have had asthma. The random parts parameters table in Table 6.11.2 shows that the between-MSOA variance is 0.002 and the within-MSOA variance is 26.40. Less than 1% (0.06%) of the variation in asthma lies between MSOAs, 88.92% of the variation in asthma lies between individuals and 11.02% of the variation lies between waves.

Table 6.10 Asthma individual and area level multilevel models for England excluding London

Fixed part parameters		6.10.1 OR		95% CI		6.10.2 OR		95% CI	
Child is female		0.46	***	0.36	0.57	0.45	***	0.35	0.56
Ethnicity	White	REF				REF			
	Mixed	2.27	**	1.20	3.99	2.15	*	1.08	3.81
	Indian	1.25		0.52	2.45	1.09		0.44	2.31
	Pakistani & Bangladeshi	1.09		0.70	1.56	0.77		0.47	1.15
	Black	0.98		0.33	2.27	0.74		0.24	1.72
	Other	2.06		0.49	6.30	1.60		0.37	5.03
Child is obese		1.90	***	1.34	2.62	1.84	***	1.30	2.51
Mother is employed		1.18	*	0.98	1.41	1.17		0.97	1.41
Mother has asthma		8.37	***	6.03	11.39	8.01	***	5.92	10.54
Mother smokes		1.39	**	1.11	1.71	1.25	*	0.98	1.54
Lives in urban area		1.97	***	1.36	2.79	1.40		0.92	2.00
Lives below the poverty line		1.40	**	1.15	1.68	1.23	*	1.01	1.52
IMD	1					REF			
	2					1.71	*	0.91	2.74
	3					1.48		0.79	2.40
	4					1.99	*	1.05	3.20
	5					3.29	***	1.61	5.31
	6					3.40	***	1.65	5.45
	7					3.44	***	1.66	5.36
	8					4.68	***	2.30	7.30
	9					4.97	***	2.47	7.55
	10					4.30	***	2.11	6.87

NO$_2$				OR		95% CI	
	low			REF			
	mid-low			0.91		0.65	1.26
	mid-low			0.72	*	0.50	1.26
	high			1.00		0.64	1.55

Random part parameters	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.003	0.001	0.01	0.08	0.002	0.0004	0.003	0.05
Level 2: Individual	18.71	16.91	20.99	85.04	19.05	17.31	21.29	85.27
Level 3: Wave				14.88				14.68

Table 6.11 Asthma individual and area level multilevel models for London only

		6.11.1				6.11.2			
Fixed part parameters		OR		95% CI		OR		95% CI	
Child is female		0.46	**	0.22	0.79	0.44	**	0.22	0.78
Ethnicity	White	REF				REF			
	Mixed	1.54		0.45	4.26	1.31		0.29	3.55
	Indian	0.88		0.31	2.15	0.81		0.18	2.32
	Pakistani & Bangladeshi	0.54		0.16	1.39	0.60		0.12	1.71
	Black	1.11		0.47	2.13	1.12		0.36	2.79
	Other	0.71		0.17	2.00	0.57		0.11	1.76
Child is obese		1.15		0.51	2.17	1.20		0.51	2.35
Mother is employed		1.51	*	0.96	2.28	1.55	*	0.96	2.45
Mother has asthma		24.42	***	10.27	52.15	40.84	***	15.77	97.63
Mother smokes		1.42		0.72	2.50	1.55		0.74	2.71
Lives in urban area		0.48		0.10	1.13	6.95	*	1.07	24.9
Lives below the poverty line		1.40		0.83	2.24	1.45		0.86	2.39

					Mean			VPC	Mean			VPC
IMD	1				REF							
	2				1.07		0.27	2.89				
	3				0.66		0.17	1.91				
	4				3.35		0.72	9.38				
	5				1.10		0.26	2.95				
	6				0.58		0.12	1.55				
	7				1.05		0.17	2.58				
	8				1.22		0.24	3.76				
	9				0.40	*	0.08	1.18				
	10				0.63		0.08	2.15				
NO_2	low				REF							
	mid-low				1.47		0.68	2.66				
	mid-high				1.47		0.68	2.66				
	high				2.13		0.84	5.12				

Random part parameters	Mean	95% CI		VPC	Mean	95% CI		VPC	
Level 3: MSOA	0.03	0.002	0.11	0.96	0.002	0.001	0.01	0.06	
Level 2: Individual	21.34	15.25	29.17	86.64	26.40	19.45	35.15	88.92	
Level 3: Wave				12.40				11.02	

6.5.1.4   Including interactions

To answer the hypotheses of this thesis, it is necessary to include interaction terms in the multilevel model to fully explore the interaction between individual and area level socio-economic status and air pollution exposure. As stated previously, an interaction suggests that the effect of variables within the interaction term are dependent on one another. This section will first explore the impact of interactions focussing on NO_2 as the pollutant of interest.

Table 6.12 presents the results from the multilevel models for both England excluding London, and London only. Table 6.12.1 shows that there was no association found for a child that lives below the poverty line in an area of high deprivation (OR 1.00, CI 0.99 – 1.02), lives in an area with high deprivation and high NO_2 pollution (OR 1.00, CI 1.00 – 1.00) or lives below the poverty line in an area with both high deprivation and high NO_2 pollution (OR 1.00, CI 1.00 – 1.00), however these findings are not statistically significant. The random parts parameters table in Table 6.10.1 shows that the between-MSOA variance is 0.001 and the within-MSOA variance is 19.03. Less than 1% (0.02%) of the variation in asthma lies between MSOAs, 85.26% of the variation in asthma lies between individuals and 14.72% of the variation lies between waves.

For London specifically, Table 6.12.2 shows that a child that lives below the poverty line in an area of high deprivation and high NO_2 pollution (OR 0.99, CI 0.98 – 1.00) is statistically significantly less likely to have had asthma. Here the random parts parameters table shows that the between-MSOA variance is 0.01 and the within-MSOA variance is 24.90. Less than 1% (0.31%) of the variation in asthma lies between MSOAs, 88.33% of the variation in asthma lies between individuals and 11.36% of the variation lies between waves.

Table 6.12 Asthma multilevel models with interaction terms – NO$_2$ only

Fixed part parameters		6.12.1 England exc. London				6.12.2 London only			
		OR		95% CI		OR		95% CI	
Child is female		0.45	***	0.36	0.57	0.41	***	0.20	0.68
Ethnicity	White	REF				REF			
	Mixed	2.05	*	1.07	3.83	1.96		0.47	5.07
	Indian	1.13		0.47	2.23	1.05		0.26	2.58
	Pakistani & Bangladeshi	0.80		0.47	1.30	1.09		0.28	2.93
	Black	0.79		0.26	1.72	1.53		0.49	3.30
	Other	1.61		0.32	5.18	0.97		0.20	3.14
Child is obese		1.89	***	1.34	2.61	1.04		0.49	2.03
Mother is employed		1.22	*	1.02	1.43	1.47		0.95	2.17
Mother has asthma		8.48	***	6.35	11.34	35.13	***	10.86	93.88
Mother smokes		1.30	*	1.05	1.59	1.49		0.77	2.44
Lives in urban area		1.79	***	1.18	2.76	0.07	***	0.01	0.16
Lives below poverty line		1.32	*	1.04	1.61	1.96	*	1.03	3.34
IMD		1.02	***	1.01	1.03	0.98		0.94	1.01
NO$_2$		0.99		0.98	1.01	1.02		0.96	1.06
Poverty*IMD		1.00		0.99	1.02	1.03		0.98	1.08
Poverty*NO$_2$		1.01		0.98	1.03	0.98		0.90	1.06
IMD*NO$_2$		1.00		1.00	1.00	1.00		1.00	1.01
Poverty*IMD*NO$_2$		1.00		1.00	1.00	0.99	***	0.98	1.00

Random part parameters	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0003	0.001	0.02	0.01	0.001	0.09	0.31
Level 2: Individual	19.03	17.22	20.58	85.26	24.90	17.72	33.16	88.33
Level 3: Wave				14.72				11.36

6.5.2 Multilevel modelling analysis of wheezing

This section details the multilevel models that focus on wheezing For England excluding London, and London only. This section first presents the results from the baseline null model before moving on to include individual and area level variables, and interaction terms.

6.5.2.1 Baseline model

Table 6.13 presents the results from the baseline model for England excluding London, and London only. For England excluding London, Table 6.13.1 shows that the between-MSOA variance is estimated to be around 0.01, whilst the within-MSOA variance is estimated to be around 4.34. Less than 1% (0.24%) of the variation in asthma lies between MSOAs, 56.90% of the variation in asthma lies between individuals and 42.86% of the variation lies between waves. Table 6.13.2 reports that for London only, the between-MSOA variance is estimated to be around 0.15, whilst the within-MSOA variance is estimated to be around 4.29. 4.37% of the variation in asthma lies between MSOAs, 56.58% of the variation in asthma lies between individuals and 39.05% of the variation lies between waves.

Table 6.13 Wheeze baseline multilevel models

	6.13.1 England exc. London				6.13.2 London only			
Random part parameters	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.01	0.004	0.01	0.24	0.15	0.01	0.33	4.37
Level 2: Individual	4.34	3.94	4.79	56.90	4.29	3.33	5.51	56.58
Level 1: Wave				42.86				39.05

6.5.2.2 Including individual variables

Building on the baseline model, individual level variables were then included in the analyses, and the results are presented in Table 6.14 and 6.15. For England excluding London, Table 6.14.1 reports that a female child (OR 0.65, CI 0.58 – 0.74) or a child that has a mother in employment (OR 0.88, CI 0.78 – 1.00) is statistically significantly less likely to have had wheezing in the previous 12 months. A child that is obese (OR 1.44, CI 1.13 – 1.79), has a mother with asthma (OR 2.64, CI 2.24 – 3.08), has a mother that smokes (OR 1.21, CI 1.06 – 1.37) or lives in an urban area (OR 1.39, CI 1.14 – 1.63) is statistically significantly more likely to have had wheezing in the previous 12 months. The random parts

parameters table in Table 6.14.1 shows that the between-MSOA variance is 0.003 and the within-MSOA variance is 4.15. Less than 1% (0.10%) of the variation in wheezing lies between MSOAs, 55.79% of the variation in wheezing lies between individuals and 44.11% of the variation lies between waves.

From Table 6.15.1, examining London only, a female child (OR 0.61, CI 0.45 – 0.81), a child that is Pakistani or Bangladeshi (OR 0.57, CI 0.31 – 0.96) or has a mother in employment (OR 0.77, CI 0.59 – 1.02) is statistically significantly less likely to have had wheezing in the previous 12 months. A child that is obese (OR 1.59, CI 0.99 – 2.41), has a mother with asthma (OR 3.35, CI 2.21 – 4.82), has a mother that smokes (OR 1.79, CI 1.24 – 2.46) or lives in an urban area (OR 13.12, CI 2.30 – 31.23) is statistically significantly more likely to have had wheezing in the previous 12 months. The random parts parameters table shows that the between-MSOA variance is 0.09 and the within-MSOA variance is 4.37. 2.79% of the variation in wheezing lies between MSOAs, 57.06% of the variation in wheezing lies between individuals and 40.15% of the variation lies between waves.

6.5.2.3 Including area level variables (IMD and NO$_2$)

Building on the multilevel models that examined the individual level variables, the area level variables of IMD and annual average NO$_2$ concentration were also added, the results of which are presented in Table 6.14.2 and 6.15.2.

Beginning with England excluding London in Table 6.14.2, a female child (OR 0.65, CI 0.57 – 0.73), a child that has a mother in employment (OR 0.89, CI 0.79 – 1.01) or lives in an area of high NO$_2$ pollution (OR 0.80, CI 0.65 – 0.96) is statistically significantly less likely to have had wheezing in the previous 12 months. A child that is obese (OR 1.42, CI 1.13 – 1.78), has a mother with asthma (OR 2.62, CI 2.24 – 3.05), has a mother that smokes (OR 1.18, CI 1.03 – 1.35), lives in an urban area (OR 1.43, CI 1.16 – 1.69) or lives in a highly deprived area (OR 1.57, CI 1.15 – 2.12) is statistically significantly more likely to have had wheezing in the previous 12 months. The random parts parameters table shows that the between-MSOA variance is 0.004 and the within-MSOA variance is 4.15. Less than 1% (0.13%) of the variation in wheezing lies between MSOAs, 55.81% of the variation in wheezing lies between individuals and 44.06% of the variation lies between waves.

For London only from Table 6.15.2, a female child (OR 0.61, CI 0.45 – 0.81), a child that has a mother in employment (OR 0.77, CI 0.57 – 1.03) or lives in a highly deprived area (OR 0.35, CI 0.16 – 0.79) is statistically significantly less likely to have had wheezing in the previous 12 months. A child that is obese (OR 1.64, CI 1.03 – 2.47), has a mother with asthma (OR 3.34, CI 2.22 – 4.99) or has a mother that smokes (OR 1.86, CI 1.32 – 2.61) is statistically significantly more likely to have had wheezing in the previous 12 months. The random parts parameters table in Table 6.15.2 shows that the between-MSOA variance is 0.08 and the within-MSOA variance is 4.37. 2.36% of the variation in wheezing lies between MSOAs, 57.04% of the variation in wheezing lies between individuals and 40.60% of the variation lies between waves.

Table 6.14 Wheeze individual and area level multilevel models for England excluding London

Fixed part parameters		6.14.1 OR		95% CI		6.14.2 OR		95% CI	
Child is female		0.65	***	0.58	0.74	0.65	***	0.57	0.73
Ethnicity	White	REF				REF			
	Mixed	1.16		0.81	1.60	1.17		0.80	1.64
	Indian	1.00		0.64	1.47	1.07		0.68	1.59
	Pakistani & Bangladeshi	0.99		0.78	1.21	1.01		0.78	1.29
	Black	0.68		0.39	1.11	0.7		0.40	1.09
	Other	1.06		0.50	2.05	1.08		0.50	1.92
Child is obese		1.44	**	1.13	1.79	1.42	**	1.13	1.78
Mother is employed		0.88	*	0.78	1.00	0.89	*	0.79	1.01
Mother has asthma		2.64	***	2.24	3.08	2.62	***	2.24	3.05
Mother smokes		1.21	**	1.06	1.37	1.18	*	1.03	1.35
Lives in urban area		1.39	**	1.14	1.63	1.43	***	1.16	1.69
Lives below the poverty line		1.07		0.93	1.23	1.04		0.9	1.2
IMD	1					REF			
	2					1.26		0.91	1.67
	3					1.19		0.85	1.57
	4					1.26		0.94	1.66
	5					1.47	**	1.09	1.94
	6					1.36	*	1.01	1.78
	7					1.46	**	1.07	1.91
	8					1.29		0.94	1.69
	9					1.35	*	0.99	1.8
	10					1.57	**	1.15	2.12

NO$_2$		OR		95% CI		OR		95% CI	
NO$_2$	low					REF			
	mid-low					0.91		0.76	1.06
	mid-high					0.82	*	0.69	0.99
	high					0.80	**	0.65	0.96

Random part parameters	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.003	0.001	0.01	0.10	0.004	0.001	0.01	0.13
Level 2: Individual	4.15	3.79	4.54	55.79	4.15	3.69	4.63	55.81
Level 3: Wave				44.11				44.06

Table 6.15 Wheeze individual and area level multilevel models for London only

		6.15.1				6.15.2			
Fixed part parameters		OR		95% CI		OR		95% CI	
Child is female		0.61	**	0.45	0.81	0.61	**	0.45	0.81
Ethnicity	White	REF				REF			
	Mixed	1.25		0.75	2.08	1.35		0.72	2.27
	Indian	1.40		0.78	2.33	1.40		0.77	2.18
	Pakistani & Bangladeshi	0.57	*	0.31	0.96	0.70		0.35	1.22
	Black	1.17		0.76	1.76	1.32		0.86	2.00
	Other	0.98		0.50	1.70	1.03		0.53	1.80
Child is obese		1.59	*	0.99	2.41	1.64	*	1.03	2.47
Mother is employed		0.77	*	0.59	1.02	0.77	*	0.57	1.03
Mother has asthma		3.35	***	2.21	4.82	3.34	***	2.22	4.99
Mother smokes		1.79	**	1.24	2.46	1.86	***	1.32	2.61
Lives in urban area		13.12	***	2.30	31.23	2.42		0.33	7.52
Lives below the poverty line		0.88		0.64	1.19	0.94		0.66	1.27

		Mean	95% CI		VPC	Mean	95% CI		VPC
IMD	1					REF			
	2					0.49 **	0.25	0.88	
	3					0.89	0.48	1.57	
	4					0.92	0.47	1.69	
	5					0.64	0.32	1.17	
	6					0.50 *	0.24	0.95	
	7					0.58	0.28	1.15	
	8					0.62	0.30	1.21	
	9					0.64	0.33	1.16	
	10					0.35 **	0.16	0.79	
NO_2	low					REF			
	mid-low					0.91	0.57	1.34	
	mid-high					0.98	0.56	1.60	
	high					1.01	0.58	1.54	
Random part parameters		Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA		0.09	0.01	0.33	2.79	0.08	0.01	0.24	2.36
Level 2: Individual		4.37	3.23	5.52	57.06	4.37	3.29	5.61	57.04
Level 3: Wave					40.15				40.6

6.5.2.4   **Including interactions**

As mentioned, multilevel models with interaction terms included were also run to answer the hypotheses of this thesis, and the results from this analysis is presented in Table 6.16. Table 6.16.1 shows that for England excluding London, no associations were found for a child that lives in an area of high deprivation and high NO_2 pollution (OR 1.00, CI 1.00 – 1.00) or a child living below the poverty line in an area with high deprivation and high NO_2 pollution (OR 1.00, CI 1.00 – 1.00), however these findings are not statistically significant. A child living below the poverty line in an area with high deprivation (OR 0.99, CI 0.99 – 1.00) is statistically significantly less likely to have wheezed in the previous year. A child living below the poverty line in an area with high NO_2 pollution (OR 0.99, CI 0.97 – 1.01) is also less likely to have wheezed in the previous 12 months, however this interaction is not statistically significant. The random parts parameters table in Table 6.16.1 shows that the between-MSOA variance is 0.001 and the within-MSOA variance is 4.10. Less than 1% (0.04%) of the variation in wheezing lies between MSOAs, 55.50% of the variation in wheezing lies between individuals and 44.46% of the variation lies between waves.

For London only, from Table 6.16.2, no association was found for a child that lives in an area of high deprivation and high NO_2 pollution (OR 1.00, CI 1.00 – 1.01), although this is not statistically significant. Similarly, no association was found for a child living below the poverty line in an area with both high deprivation and high NO_2 pollution (OR 1.00, CI 0.99 – 1.00) and this is significant. A child living below the poverty line in an area with high deprivation (OR 0.98, CI 0.96 – 1.01) is less likely to have had wheezing in the previous 12 months but again this was not statistically significant. A child living below the poverty line in an area of high NO_2 pollution (OR 1.01, CI 0.97 – 1.05) is more likely to have had wheezing in the previous 12 months, however this is not statistically significant. The random parts parameters table in Table 6.16.2 shows that the between-MSOA variance is 0.005 and the within-MSOA variance is 4.72. Less than 1% (0.14%) of the variation in wheezing lies between MSOAs, 56.48% of the variation in wheezing lies between individuals and 43.38% of the variation lies between waves.

Table 6.16 Wheeze multilevel modes with interaction terms – NO_2 only

Fixed part parameters		6.16.1 England exc. London				6.16.2 London only			
		OR		95% CI		OR		95% CI	
Child is female		0.65	***	0.57	0.73	0.61	**	0.44	0.81
Ethnicity	White	REF				REF			
	Mixed	1.10		0.77	1.53	1.38		0.77	2.23
	Indian	0.97		0.62	1.46	1.47		0.87	2.38
	Pakistani & Bangladeshi	0.92		0.71	1.17	0.83		0.43	1.36
	Black	0.63	*	0.36	1.00	1.31		0.87	1.91
	Other	0.96		0.44	1.79	1.10		0.56	1.96
Child is obese		1.43	**	1.14	1.76	1.59	*	0.98	2.47
Mother is employed		0.89	*	0.79	0.99	0.77	*	0.57	1.00
Mother has asthma		2.62	***	2.26	3.05	3.34	***	2.22	4.91
Mother smokes		1.17	**	1.03	1.33	1.78	***	1.24	2.45
Lives in urban area		1.28	**	1.07	1.52	5.08	*	1.25	9.45
Lives below the poverty line		1.05		0.91	1.19	1.15		0.81	1.60
IMD (level of deprivation)		1.01	***	1.00	1.01	0.99		0.97	1.01
NO_2 (level of pollution)		1.00		0.99	1.01	1.01		0.99	1.03
Poverty*IMD		0.99	*	0.99	1.00	0.98		0.96	1.01
Poverty*NO_2		0.99		0.97	1.01	1.01		0.97	1.05
IMD*NO_2		1.00		1.00	1.00	1.00		1.00	1.00
Poverty*IMD*NO_2		1.00		1.00	1.00	1.00	**	0.99	1.00

Random part parameters	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.001	0.002	0.04	0.005	0.001	0.01	0.14
Level 2: Individual	4.10	3.72	4.51	55.50	4.27	3.40	5.39	56.48
Level 3: Wave				44.46				43.38

6.6   Conclusion

Accounting for the nested structure of data available at different spatial levels, multilevel modelling is a valuable analytical technique. The analysis presented in this Chapter has built upon the findings from previous Chapters in this thesis and has highlighted the relationship between individual and area level socio-economic status, air pollution exposure and respiratory health.

Regarding the multilevel model results including all respondents in the MCS, individual level socio-economic status (living below the poverty line) was found to have a consistently statistically significant effect on asthma prevalence, with a child that lives below the poverty line more likely to have had asthma throughout their life. However, no statistically significant association was found for wheezing and living below the poverty line. Examining area level socio-economic status (IMD), a child living in an MSOA with greater deprivation was consistently more likely to have had asthma or to have experienced wheezing within the previous 12 months. This was found to be statistically significant for the most deprived deciles. Similar to results from cross-sectional and time series analyses presented previously, a child living in an MSOA with the highest levels of NO_2 was found to be less likely to have had asthma or to have wheezed in the previous 12 months. The effect of NO_2 exposure on wheezing was found to be statistically significant. Two- and three-way interaction terms were included in the analysis to account for the complex connections underlying these variables to answer the hypothesis of this thesis. It was hypothesised that a child with low individual socio-economic status living in a more deprived area would face greater health risks from exposure to higher levels of pollution, however the models showed generally weak and not statistically significant results.

In order to account for both the high levels of wealth and NO_2 in London, the models were also run without London, as well as only looking specifically at London. When excluding London, findings were similar to what was seen when examining England as a whole. A lower individual and area level socio-economic status were found to increase the likelihood of a child having asthma or wheezing, whilst exposure to higher levels of NO_2 was still found to decrease the likelihood. Conversely, when examining only London, children living in areas of the highest deprivation were found to be statistically significantly less likely to have had asthma ever or wheezing in the previous 12 months. These findings support the

decision to rerun these models to both include and exclude London, to fully comprehend the underlying relationship between these variables of interest and child respiratory health.

As seen in previous Chapters, female children were less likely to have had asthma or wheezing, and children of mothers who were employed were less likely to have had wheezing in the previous 12 months. With the inclusion of ethnicity as a categorical variable, further understanding of the relationship between ethnicity and respiratory health could be gained, such as mixed-race children being more likely to have ever had asthma. As before, a child that was obese, had a mother that smokes, or lived in an urban area were more likely to have had asthma and wheezing. The following Chapter will discuss the results of the multilevel models presented here in more detail.

Chapter 7. Discussion

7.1 Introduction

The negative impact of health, socio-economic status and air pollution on human health is what Jerrett refers to as triple jeopardy (Jerrett et al., 2001). Within this context, this thesis used a data linkage methodology and series of regression analysis to investigate:

1. Whether the association between asthma and air pollution is stronger amongst children of lower, compared to higher, socio-economic status, and;
2. Whether area level deprivation interacts with individual socio-economic status so that the impact of air pollution exposure on asthma is stronger for children with low socio-economic status living in the most deprived areas than children with similar socio-economic status living in less deprived areas.

This Chapter discusses the cross-sectional analysis results, the time series analysis results and the multilevel model results presented in Chapters 4, 5 and 6 within the context of previous research and the concept of triple jeopardy. This Chapter also discusses the strengths and potential limitations of this research, and what implications the findings could have for future research.

As noted in Chapter 1, asthma is a chronic respiratory condition that develops in around 15% of the population of England by the time they are in their early teens (Lewis et al., 2018), affecting approximately 300 million people globally (Braman, 2006). Asthma is defined as "a heterogeneous disease, usually characterised by chronic airway inflammation" and is associated with a history of respiratory symptoms which includes wheezing, shortness of breath, tightness of chest and a reduced airflow (Reddel et al., 2015). The UK has one of the highest asthma mortality rates among young people for high-income countries worldwide and the highest rates of asthma symptoms globally in children (Gupta et al., 2018). The UK also has the highest rates of asthma related hospital admissions in Europe. A fifth of British children have been diagnosed with asthma by a doctor (Panico

et al., 2007), however it is noted that it is difficult to define asthma in children under the age of five as the clinical symptoms of asthma are variable (Pedersen et al., 2011).

The Millennium Cohort Study (MCS) is an invaluable source of data for this research for several reasons. As a longitudinal study, the MCS provides a wide breadth of data over a period of time, allowing for extensive analysis to be conducted which provides a greater insight into the data, as opposed to using a dataset that collected data from only one point in time. Additionally, the MCS dataset is multidisciplinary, covering an array of topics including economic, social and demographic information (Connelly and Platt, 2014). The dataset is also intergenerational, not only providing data on the child cohort member, but also on their parents, siblings and other family members, which allows a better understanding for how inequalities are inherited through families (Connelly and Platt, 2014).

A series of cross-sectional logistic models for each wave of the MCS exploring the association between air pollution, area level deprivation and individual/familial characteristics and asthma and wheezing were presented (Chapter 4). Logistic regression models were run including all air pollutant data available (PM_{10}, $PM_{2.5}$, NO_2, NO and O_3), and including NO_2 as the only pollutant of interest. As noted in Chapter 1, much of the UK's air pollution is attributed to transport emissions, and NO_2 pollution is strongly associated with traffic density (Salonen et al., 2019), and is therefore a good indicator of ambient air pollution. To test both hypotheses, socio-economic status is examined through poverty (individual/familial level) and IMD (area level) variables. The results were presented as odds ratios. Data were then examined using time series analysis as presented in Chapter 5. First, data were analysed to explore the impact of socio-economic and air pollution exposures at Wave 1, before examining the impact of these exposures over time. Finally, data were analysed using a multilevel modelling approach (Chapter 6), allowing the data to be separated onto their separate spatial or temporal levels.

This Chapter is structured as follows. Section 7.2 will explore the main effects of air pollution and socio-economic status on the respiratory health of children in England, as well as discussing how these exposures interact with each other and the complexity of understanding this relationship. Moving on, other covariates of

interest will be discussed, and further complex interactions will also be examined. Finally, the strengths and limitations of this study will be considered.

7.2   Respiratory health and air pollution: Main effects

Concerning the impacts of air pollution on respiratory health, it is widely understood that children living in areas with higher air pollution concentrations would experience higher asthma and wheezing prevalence. This study expected to discover that children living in the most polluted LSOAs or MSOAs were more likely to have ever had asthma or to have wheezed in the previous 12 months, however outputs across all modelling approaches provided mixed and inconclusive results.

Beginning with analysis that focused on NO_2 pollution as the sole exposure and examining the data through a cross-sectional approach (Chapter 4), the results generally indicated that those living in areas with the highest levels of NO_2 pollution were less likely to have had asthma or wheezing in the previous 12 months. The time series approach (Chapter 5) showed that early life exposures (in Wave 1) to higher levels of NO_2 also resulted in a decreased likelihood of developing respiratory problems as a child. Indeed, the same result was found when examining NO_2 concentration that varied over each wave. Finally, examining the results from the multilevel modelling approach (Chapter 6) again found that children in England that were living in areas with greater NO_2 pollution were less likely to have ever had asthma or to have wheezed in the previous 12 months. In contrast, children living in London were more likely to have had asthma if they lived in an area with high NO_2 pollution.

Exploring the impacts of exposure to PM_{10} pollution, the results from all modelling approaches show that a child living in an area with a high concentration of PM_{10} pollution is generally less likely to have had asthma or wheezing when compared to a child living in an area with lower levels of PM_{10} pollution. In regards to exposure to $PM_{2.5}$, the results throughout show that exposure to high levels of $PM_{2.5}$ pollution is generally associated with a decreased likelihood of a child having had asthma. However, the model results show that living in areas with higher concentrations of $PM_{2.5}$ pollution was generally a consistent statistically significant risk factor for wheezing prevalence. When looking at the impact of NO pollution, the cross-sectional approach indicates that living in an area of high NO

pollution is a consistent risk factor for a child having had asthma or wheezing in the previous 12 months whilst results from the time series model contradict these findings and report the opposite.

The results examining exposure to O_3 pollution were again mixed. Using the cross-sectional approach found that living in areas of higher O_3 pollution decreased the risk of a child having asthma or wheezing, and similar findings were seen with the time series approach when examining exposure to O_3 pollution over the five waves. However, the time series approach that examined the impact of a child's early life exposure to O_3 pollution in Wave 1 found that higher levels of exposure increased their likelihood of having asthma or wheezing throughout their life.

The inconsistencies in results across the different modelling approaches for all pollutants could be due to how the different modelling approaches handle the data. The cross-sectional approach could only examine one wave at a time, whilst the time series approach examined the data as a whole. The incorporation of the temporal aspect of the data could account for different outputs seen between these approaches. In addition, the multilevel modelling approach further builds on this and also takes the spatial aspect of the data into account.

As established in Chapter 2, there has been a large amount of research on the relationship between air pollution and respiratory health. However as with the analyses provided in this thesis, the results from these studies have been mixed. Several studies have found statistically significant relationships between air pollution exposure and asthma occurrence in children, however, there has been great variability in said relationships.

One study found a strong association between increased exposure to PM_{10} and NO_2 pollution, among others, and increased occurrence of wheezing in children (Andersen et al., 2008). McConnell et al. (2010) also found that children that were exposed to higher levels of traffic-related air pollution at both their home and school environments were more likely to develop asthma. In addition, they found the impacts of air pollution exposure at school to be independent to exposure at home, potentially due to compulsory periods of exercise during school hours that would increase a child's inhalation rates, thereby increasing their intake of air pollution (McConnell et al., 2010). This relationship may also be due to the child

being present in school during peak traffic hours in the morning. Bowatte et al. (2015) conducted a systematic review and meta-analysis of birth cohort studies to examine the relationship between exposure to traffic-related air pollution and childhood asthma. There was a modest association between exposure to NO_2 and asthma incidence, however the association varied greatly across studies. There was an association found between early life exposure (under the age of six) to NO_2 and the incidence of asthma, although there was no pattern found in later years (Bowatte et al., 2015).

A further time series study (Oftedal et al., 2009) examining long-term exposure to traffic-related air pollution and the onset of asthma in children aged 9 to 10 years old failed to find a positive association. An explanation provided for the lack of association was exposure levels potentially being too low, and this reasoning could be applicable to the results seen in Chapters 4, 5 and 6. Further research by Heinrich and Wichmann (2004) also explored the relationship between traffic-related air pollution and asthma, however only a weak association was found. Kravitz-Wirtz et al. (2018) discussed the challenges with research in this subject, namely the heterogeneity in the definition and measurement of asthma and the different assessment methods for quantifying air pollution exposure.

Another possible explanation for the results discussed in this section could be due to the different microenvironments in which a child spends their time. In this study, a child's location was derived from the LSOA or MSOA their home was situated in, however children spend a third of their waking day in school (Driscoll et al., 2015). Here, they typically have set times for playing outside as well as timetabled exercise, and as mentioned this causes increased breathing rates and consequently increased inhalation of air pollutants (McConnell et al., 2010). Therefore, exposure at school may be just as important, if not more important than exposures in the home environment. On the other hand, Martins et al. (2012) discussed how a child spends most of their life inside, offering further explanation as to why readings of atmospheric air pollution concentration could appear to not have an impact on the prevalence of asthma.

7.3 Respiratory health and individual and area level socio-economic status (poverty and deprivation)

As noted throughout this thesis, exposure to air pollution and low income are spatially correlated. Living below the poverty line and living in an area with a higher IMD score are both indications of a low individual and area level socio-economic status respectively. In terms of individual level socio-economic status, a child is described as living below the poverty line if their family earned below 60% of the national median income before housing costs (Longford et al., 2012).

The results from Chapters 4, 5 and 6 show that living below the poverty line and living in areas of high deprivation were consistently found to be statistically significant risk factors for both asthma and wheezing amongst children, regardless of which modelling approach was used. However, the multilevel models show that a child living in an area of high deprivation in London is less likely to have had asthma or wheeze, indicating that children living in the wealthiest areas are more likely to have had asthma or a recent wheezing event.

It is well established that lower socio-economic status, both at the individual and area level, has a negative impact on individual health, including respiratory health. A study by Bacon et al. (2009) found that individual level socio-economic status is associated with worse asthma control, an increase in emergency hospital use for asthma related issues and ultimately, worse asthma morbidity. Another study (Cesaroni et al., 2003) focussed on both individual and area level indicators of socio-economic status and found that those that were more disadvantaged faced an increased rate of asthma occurrence, which was also more severe when compared to less disadvantaged individuals. Research in England found that asthma related hospital admissions were strongly associated with deprivation in the community (Gupta et al., 2018).

The association between respiratory health and socio-economic status could be explained through a number of pathways. For example, someone living below the poverty line can face challenges when trying to access healthcare, and consequently they could then be under-medicated (Rona, 2000). Furthermore, they may become reliant on crisis management to deal with their asthma, which would result in increased hospital admissions.

7.4   The association between respiratory health, air pollution and socio-economic status

The first hypothesis this thesis set out to answer was whether the association between respiratory health and air pollution is stronger amongst children of lower, compared to higher, socio-economic status or the theory of triple jeopardy. The triple jeopardy theory states that individuals that are amongst the most deprived in society face higher rates of exposure to air pollution as well as higher risks related to said exposure (Jerrett et al., 2001). Furthermore, their disadvantaged nature results in increased risks from social and cultural-behavioural determinants of health, and as a result they experience disproportionately worse health impacts when compared to individuals that are less deprived (Jerrett et al., 2001).

Studies exploring this relationship have found that families in low-income areas have been found to face increased rates of exposure to detrimental environmental pollutants, such as industrial pollution, diesel emissions, indoor allergens and second-hand smoke (Stronks et al., 1998). Furthermore, they are more likely to be exposed to damaging psychosocial stressors (Stronks et al., 1998, Beck et al., 2017) which can include poor quality or unhealthier foods, housing issues, financial insecurity, social marginalisation and violence, both in the home and the community.

Simplistically, it can be stated that health inequalities are caused by the negative effect that living in deprivation has on health, and whilst the justification behind this is complex, research is ongoing to help better understand the pathways. To date, three causal mechanisms have been put forward based on the social causation theory; neomaterial, cultural-behavioural and psychosocial explanations (Skalická et al., 2009).

First, the neo-material explanation focusses on material wealth, and how income enables behaviours that benefit health, such as providing access to goods and services (Skalická et al., 2009). Conversely, lack of income therefore limits benefits to health, through issues such as causing barriers to healthcare and poor quality housing. This could mean living in a house with a mould problem due to poor circulation, which could increase a child's likelihood of having asthma (Caillaud et al., 2018). Assari and Moghani Lankarani (2018) found that a high

familial socio-economic status, for example a substantial family income and high level of parental education, is protective against asthma, whilst a low socio-economic status, financial strain and poverty could exacerbate asthma and wheeze in children. With regard to the neo-material mechanisms, this analysis found that variables such as social housing and urban residency, both neo-material factors, were generally found to increase a child's likelihood of having respiratory health problems, and this is discussed in Section 7.5 and 7.6.

Second, the cultural-behavioural explanation relates to how people act and how their actions influence their health (Skalická et al., 2009). This includes how some behaviours may be more common and deemed more socially acceptable in less affluent areas, such as smoking, excessive alcohol consumption and increased levels of physical inactivity (Ellen et al., 2001). With regard to the cultural-behavioural mechanisms, exposures to second-hand smoke in the home can increase a child's probability of having asthma, and this is discussed in Section 7.5.

Third, the psychosocial explanation suggests that social inequality makes people feel a sense of domination or subordination, and superiority and inferiority, which impacts on people's mental and physical health (Skalická et al., 2009). For example, a mother may not feel comfortable or confident in booking an appointment to see a doctor to discuss potential health issues her child may be facing, resulting in the child's health issues remaining undiagnosed and untreated, which could then exacerbate their health problems. Another psychosocial trigger is exposure to violence which has been shown to further increase the likelihood of a child having asthma or wheezing (Wright et al., 2004). From an epigenetics perspective, maternal exposure to chronic stressors could also explain higher rates of asthma in children, and areas with higher rates of poverty are more likely to feature higher exposure to chronic stressors, such as violence (Flanigan et al., 2018). A combination of both individual level and area level psychosocial stressors throughout the life course, beginning in the prenatal stage, can explain the influence of poverty on health inequalities. The magnitude of these stressors can impact the immune system and increase a child's susceptibility to the effects of air pollution and is highest in the most deprived communities (Kravitz-Wirtz et al., 2018).

To test the theory of triple jeopardy for the MCS cohort a series of interaction terms were created. To test *Hypothesis 1,* an interaction term was created to examine the impact of living below the poverty line and average LSOA or MSOA levels of pollutants (familial poverty * NO_2). To test *Hypothesis 2* a three-way interaction term was created that examined the relationship between individual level socio-economic status (measured using the poverty variable), area level socio-economic status (measured using the IMD rank) and exposure to NO_2 pollution (familial poverty * IMD * NO_2).

With regard to *Hypothesis 1,* examining the results of the cross-sectional analysis including the interactions terms (Chapter 4), the results show that children that live below the poverty line and in an area of high NO_2 pollution were less likely to have had asthma or to have experienced wheezing in the previous 12 months.

With regard to *Hypothesis 2,* the cross-sectional analysis presented in Chapter 4, the three-way interaction term did not find a relationship between familial socio-economic status, area level deprivation and air pollution exposure and asthma and wheezing in children. When using a time series approach, the interaction term that was concerned with exposures at Wave 1 found that a child living below the poverty line, as well as in an area of high deprivation and high NO_2 pollution at this time point, was more likely to have had asthma or to have experienced wheezing in the previous 12 months. However, when the interaction term was explored across all waves, it failed to find a relationship between the three exposures and asthma or wheezing.

From the results presented from the multilevel modelling approach in Chapter 6, the interaction term shows that there was no association between the variables for a child living below the poverty line, in an area of high deprivation and high NO_2 concentration in terms of asthma or wheeze prevalence. Likewise, no association was found when examining the two-way interaction between individual level socio-economic status and NO_2 pollution. When controlling for all other pollutants, the three-way interaction was found to suggest that a child living below the poverty line, in an area of high deprivation and high NO_2 concentration would be more likely to have had asthma. When excluding London from the multilevel model, again no association was found for the three-way interaction. Regarding the two-way interaction, the results did suggest a child living below the

poverty line in an area of NO_2 pollution was more likely to have had asthma, but not wheezing, however these results were not found to be statistically significant. No association was found for the three-way interaction on wheezing prevalence when examining London only, however the results suggest that a child living below the poverty line, in an area of high deprivation and high NO_2 concentration would be less likely to have had asthma.

With some exceptions, such as the time series analysis presented in Chapter 5, the three-way interaction term did not find a relationship between familial or area level socio-economic status, air pollution exposure and respiratory health. However, in these models it appears that poverty and deprivation have stronger associations with respiratory health, and it is difficult to then disentangle the tree-way interaction following the inclusion of air pollution exposure. There is evidence that in England, the most deprived places have also been the most polluted (Milojevic et al., 2017), and these areas have continued to develop in this way over time. As a result, it is difficult to separate the effects from each other and poverty and deprivation prove to be more dominant. Nevertheless, the results still provide a valuable insight into the data through the interpretation of these results and the findings highlight the complexity of the relationship in question.

Multilevel modelling provided data regarding the variation in asthma and wheezing prevalence that lay between MSOAs, within MSOAs and between waves. Approximately 85% of variation in asthma lay within MSOAs, also termed between individuals. In comparison, only 56% of the variation in wheezing lay between individuals. Furthermore, 14% of asthma variation lay between waves, compared to approximately 43% of wheezing variation. An explanation for this contrast between asthma and wheezing prevalence could be due to the fact that wheezing is more variable over time when compared to asthma. Asthma is an established, yet hard to define chronic illness, whilst current wheeze is related to recent occurrence within the previous 12 months, allowing for more variability over time. In addition, a child can experience wheezing without having had asthma, and as a standalone symptom, it is easier to record episodes of wheezing in comparison to obtaining an asthma diagnosis.

There may be confounding taking place due to the number of variables included in the models. In population-based time-series studies of the relationship

between air pollution events and hospital admissions, various risk factors, such as diet, smoking, or socio-demographic factors, are not likely to be confounders because they do not co-vary with pollution over relatively short time periods of interest (i.e., days) when averaged over large populations (Burnett et al., 2003, Sheppard et al., 2012). However, these risk factors clearly have spatial patterns and thus must be accounted for in the analysis of cohort studies when considering the effects of longer-term pollution exposure.

Even with rich model data used for this analysis, models are needed to predict individual exposures. Special data collection and modelling efforts are required for some components of individual exposure, specifically non-ambient source exposures, individual time-activity, and building- and season-specific infiltration. Due to the nature of cohort studies, our air pollution variables were averaged to the LSOA or MSOA level and related to a yearly average and will therefore not include the full pollution distribution for an area.

7.5   Associations with other covariates

This section reviews the observed associations between the covariates and outcomes in order to explore how consistent the data and models are with the established relationships found in previous literature.

7.5.1 Biological sex

Results from Chapters 5, 6 and 7 have shown that female children were consistently less likely to have had asthma or to have experienced wheezing in the previous 12 months when compared to male children, however this differential begins to decrease in Wave 5. The results displayed significant statistical evidence for a relationship between biological sex and respiratory health.

Osman (2003) observed that epidemiological studies that looked at both the incidence and prevalence of asthma found that male children had an increased likelihood of having asthma and atopic conditions before puberty. However, this reversed following the onset of puberty, where female children then experienced an increased likelihood. Given that Wave 5 of the MCS was carried out when the children were aged around 11, which would generally coincide with the onset of puberty, this could explain the decrease seen in the differential. Skobeloff et al.

(1992) found that male children aged under 10 were twice as likely to be hospitalised with asthma compared to female children of the same age.

The biological factors behind these sex differences have been thoroughly examined, and research has found that male children have increased non-specific bronchial hyperresponsiveness in comparison to female children (Osman, 2003). Fluctuation levels of hormones over the course of puberty and atopy explain why females become more susceptible to asthma in adulthood (Osman, 2003). In addition, airway development differs between sexes, with male children experiencing a slower pace of airway development when compared to their lung volume growth (Osman, 2003). In contrast, female children experience a proportionate growth of both their airways and lung volume, and this in turn causes increased air flow rates at fixed proportions of total lung capacity (Osman, 2003). Male children therefore have a lower expiratory air flow rate at all comparable lung volumes (Osman, 2003).

7.5.2 Ethnicity

Results from Chapters 4 and 5 were inconsistent and not statistically significant. The results show that in some Waves, children that were White British were more likely to have had asthma or wheezing in the previous 12 months, whilst in other Waves the reverse was seen. Following the inclusion of ethnicity as a categorical variable, the multilevel modelling approach showed that, when compared to a child that was white, a child that was mixed-race was more likely to have had asthma whilst a child that was Pakistani or Bangladeshi was found to be less likely to have had asthma, and statistical evidence supported this relationship. Black children were also found to be less likely to have had wheezing in the previous 12 months.

Health inequalities related to ethnicity and health in England have been previously researched (Morris et al., 2005). Reports from the Health Survey of England (Whitrow and Harding, 2010) showed that Black Caribbean children reported higher rates of wheezing in the previous year compared to all other groups, whilst both Bangladeshi and Black African children reported low rates of wheezing, supporting the results seen in the multilevel model. In both England and Wales, ethnic minorities had significantly higher asthma incidence rates when compared to White groups (Netuveli et al., 2005). Furthermore, those from

ethnic minorities that were born in the UK had a higher incidence than those born elsewhere (Netuveli et al., 2005). Another study examined the differences in asthma related hospital admissions and deaths in Scotland and found substantial ethnic variation (Sheikh et al., 2016) where South Asian groups had a 20 to 50% higher rate of hospital admissions compared to White people. Conversely, Chinese groups had 30 to 40% lower admission rates compared to White people (Sheikh et al., 2016). The impact of ethnicity on respiratory health is complex and cannot be fully explored without considering the other variables, therefore this relationship will be further explored in Section 7.6.

7.5.3 Obesity

As stated in Chapter 3, obesity is a chronic condition that is generally defined as excess body fat. The MCS recorded the height and weight of cohort members and calculated the prevalence of obesity, which was defined by the International Obesity Task Force (IOTF) cut-offs for BMI, that were sex and age specific (Brophy et al., 2009). The results from all modelling approaches show that obesity was a consistent risk factor for both asthma and wheezing prevalence. The results generally provided statistically significant evidence to support this relationship, however the results were not statistically significant throughout.

Epidemiological studies have highlighted the relationship between respiratory health and obesity. Indeed, obesity is a known risk factor for incident asthma and it can impact the management of the disease (Forno and Celedón, 2017). Whilst obesity and asthma can co-exist in children, there is evidence of an "obese asthma" phenotype, where a higher body weight impacts and modifies asthma characteristics (Forno and Celedón, 2017). This phenotype is theorised to be associated with an increased number of symptoms, worse control, more frequent and more severe acute episodes, a lower response to treatment, and a lower quality of life. Studies found that higher BMI was associated with an increased prevalence of asthma among children aged around 8 years old (Bibi et al., 2004, Scholtens et al., 2009). Furthermore, obese male children were found to experience more chest symptoms than obese female children (Bibi et al., 2004), which can be related back to the sex differences in child asthma rates that was previously discussed.

7.5.4 Maternal employment

The findings from Chapters 4, 5 and 6 have shown that a child of a mother that is employed is generally more likely to have had asthma when compared to a child of an unemployed mother, whilst the reverse is true for wheezing in the previous 12 months. The results from the multilevel modelling approach provided statistical evidence for this relationship.

Maternal employment and the way in which it can impact on child health, including the impact it may have on the respiratory health of children can be complex. Morrill (2011) suggests that the theories underpinning this relationship indicate that the relationship is not causal, but rather a mother being in employment could signify inherent skills, abilities or preferences, thus making an employed individual intrinsically different from an unemployed individual. An employed mother could have a higher level of education, and through obtaining their own income, a strong sense of agency which then influences different decisions being made in relation to their child's health and wellbeing. In addition, there is a possibility that having an employed mother increases the likelihood of a child experiencing a short-term health event, but in the long run actually having better health through the development of higher cognitive abilities (Morrill, 2011). Another study specifically looked at actions taken by parents in the home to limit allergen exposure in children with asthma (Ungar et al., 2010). Women who remained at home and looked after their children instead of being in employment were found to better limit exposures their asthmatic children faced, when compared to mothers in employment and those receiving welfare (Ungar et al., 2010).

Maternal employment is linked with socio-economic status and could have a positive impact on child health through increased income and what this facilitates, such as better access to health care, and improved self-esteem and empowerment of the child's mother, enabling her to seek out appropriate diagnosis and treatment for illnesses the child may experience (Pratley, 2016). In contrast to this, a mother living in deprivation may have a decreased sense of self-esteem, and would face challenges when trying to access health care, as well as the potential of not being taken as seriously as someone who would be more confident and assertive when dealing with healthcare professionals

(Macintyre et al., 1998). However, it has been argued that maternal employment can also have a negative impact on the health of the child (Morrill, 2011). If a mother is working, especially during the early years of a child's life, the child may experience less diligent supervision during the period where the mother is at work (Morrill, 2011). This could result in the child being exposed to certain environments that could result in poorer health, for example, playing outdoors during times of increased air pollution from nearby traffic. Another argument is that the mother would not be involved in encouraging their child to partake in health promoting activities (Morrill, 2011). An asthmatic child may also not take appropriate asthma medication as and when needed if their mother is absent, which could result in a more serious asthma episode if it remains untreated for a length of time (Morrill, 2011). However, results tended to be small and insignificant, similar to the results produced in this research.

7.5.5 Maternal asthma

The results presented across all modelling approaches show that maternal asthma is a major and statistically significant risk factor for asthma and wheezing prevalence in children. However, when compared with the impacts on wheezing, maternal asthma was found to be a greater risk factor for asthma.

These findings are supported by the literature, with previous research indicating that maternal asthma is associated with an increased prevalence of asthma in children (Litonjua et al., 1998). Research suggests genetic inheritance influences asthma prevalence in children, which appears to have a stronger maternal relationship (Litonjua et al., 1998), suggesting a preferential inheritance of childhood asthma among maternal lines. Indeed, Lim and Kobzik (2009), found that children of asthmatic mothers are more likely to have asthma when compared to children of non-asthmatic mothers. This suggests that non-genetic in utero and/or post-natal factors may influence asthma susceptibility in children (Litonjua et al., 1998). Burke et al. (2003) stated that having one parent with asthma increases a child's risk of also having asthma by two to four times.

Further explanation for the impact of maternal asthma could be due to a lack of maternal asthma control during pregnancy, as this was found to heighten the risk of child asthma and recurrent wheeze (Mirzakhani et al., 2019). Mirzakhani et al. (2019) also found that the risk of child asthma and wheeze increases if both

parents have asthma. The predisposition to developing asthma appears to be established in utero, and this is genetic susceptibility. Furthermore, maternal environmental exposure could impact early life airway hyperresponsiveness and asthma risk (Mirzakhani et al., 2019).

Another possible explanation put forth has been that mothers that suffer from asthma themselves are quick to identify when similar symptoms are present in their child, and are therefore more knowledgeable about what their child is experiencing and are more likely to seek diagnosis and treatment for them, when compared to non-asthmatic mothers who may not pick up on indicative behaviours (Davidson et al., 2010).

7.5.6 Maternal smoking

The results in Chapters 4, 5 and 6 show that maternal smoking habits was generally a consistent risk factor for both asthma and wheezing prevalence throughout all different modelling approaches. The multilevel modelling approach provides evidence that maternal smoking has a statistically significant relationship with both asthma and wheezing amongst the children in the study.

Exposure to second-hand smoke is an environmental factor that has been shown to impact respiratory health. Previous research has examined the impacts of second-hand smoke on respiratory health in children, especially in relation to parental smoking habits, and has shown that exposure to second-hand smoke is related to an increased prevalence of asthma in children and may result in an increased severity of asthma (Zuraimi et al., 2008). Gonzalez-Barcala et al. (2013) found that asthma symptoms were more prominent with an increasing exposure to parental smoking, suggesting a clear detrimental impact of second-hand smoke on respiratory health. Gonzalez-Barcala et al. (2013) studied children aged 6 to 7 years old, and children aged 13 to 14 years old and found that the effect of second-hand smoke was stronger among the younger children. Palmieri et al. (1990) also discovered a stronger relationship between parental smoking and asthma in younger children, aged under 6, when compared to older children. The impact of second-hand smoke was stronger when both parents smoked, however when only one parent smoked, a stronger association was found between asthma and maternal smoking compared to paternal smoking (Gonzalez-Barcala et al., 2013).

Maternal smoking could have such an impact for a number of reasons. Typically, a child has a closer relationship with their mother, and so spends more time in their presence as opposed to with their father, especially in their younger years (Gonzalez-Barcala et al., 2013). This could result in an increased exposure to second-hand smoke from maternal smoking and may explain why the results in Wave 1 are significant. Further to this, if a child's mother is a smoker, there is a possibility that the mother continued to smoke throughout the pregnancy, exposing the foetus directly to the second-hand smoke, which in turn would have impacted development and lung maturation, as well as limiting immune system growth, increasing the chances of prematurity and, in turn, making their airways smaller (Gonzalez-Barcala et al., 2013). Once born, second-hand smoke causes damage to the respiratory system through inflammation, increasing epithelial permeability, disposition to respiratory infections and allergic sensitisation, as well as limiting response to medical treatment (Gonzalez-Barcala et al., 2013).

7.5.7 Housing

The results show across all modelling approaches that living in social housing is generally linked with an increased risk of both asthma and wheezing, and indeed this relationship was found to be statistically significant at times.

Housing type and quality have both been examined to identify how they could promote or inhibit a child's health, and housing has been linked to health through three pathways; internal housing conditions; area characteristics and; housing tenure (Gibson et al., 2011). Research has shown that social housing is characterised by extremes of poverty and environmental factors that worsen asthma, and therefore has been associated with increasing the risk of developing asthma through individual risk factors as well as community-level risk factors (Northridge et al., 2010). As stated, living in social housing is indicative of a family's socio-economic status, and can also be linked to deprivation, therefore this relationship will be further explored in Section 7.6.

7.5.8 Urban residency

The results presented in Chapters 4, 5 and 6 show that residing in an urban area is also generally a consistent risk factor for increasing the likelihood of a child having had asthma or wheezing, and this relationship was found across all

modelling approaches. There is evidence to suggest that this relationship is statistically significant in some instances. It was important to run the multilevel model for England excluding London to investigate the impact of urban residency on asthma and wheeze prevalence in children when London, a major urban area, has been removed from the analysis. Indeed, urban residency was still seen to increase the likelihood of a child having had asthma or having wheezed in the previous 12 months.

Research shows that living in urban areas increases exposure to ambient air pollution for longer durations and at greater volume than living in rural areas (Briggs et al., 2008). As previously mentioned, those living in urban areas are more likely to reside in areas with increased traffic emissions, thereby facing increased exposure to pollutants like NO_2. This in turn results in increased prevalence of asthma (Forastiere et al., 2007). The most deprived communities living in urban areas are more likely to be situated close to polluting factories, airports, waste management facilities and busier main roads, thus experiencing even higher exposure to pollutants (Achakulwisut et al., 2019). Another factor that may explain the relationship found is healthcare access. Whilst deprived communities face issues accessing healthcare, rural communities may also face barriers to accessing appropriate healthcare due to their remoteness (Estrada and Ownby, 2017). However, the protective factors that a rural area could provide are complex, but could include early exposure to microbes that could bolster the developing immune system of a child, reducing their risk of asthma (Estrada and Ownby, 2017).

7.6   Interactions between air pollution, socio-economic status and other covariates

As noted by Sheppard et al. (2012), one cannot examine each variable independently whilst ignoring other variables as they are intrinsically linked together. As stated, no singular exposure is responsible for the development of asthma or the occurrence of wheezing in children, and no singular exposure can be considered by itself (Dick et al., 2014). The following section takes into account all covariates used in the analyses (sex, ethnicity, obesity, maternal employment, maternal asthma, maternal smoking, housing type and urban residency) and considers the relationships between the covariates, socio-economic status, air

pollution exposure and respiratory health. Certain covariates have been shown to be predictors for asthma and wheezing in children. If the interactions discussed in Section 7.4 between socio-economic status and air pollution have a direct impact on the respiratory health of children, it is possible that these interactions indirectly impact respiratory health through these covariates also.

Relationships between biological sex, deprivation and air pollution exposure are complex and unclear, and more research needs to be carried out on the subject. Environmental exposures are believed to play a bigger role in regard to sex differences in asthma prevalence later in life, as children spend the majority of their time either in the home or in school (Lueke, 2011). However, sex is intrinsically linked to gender and cultural norms, roles and behaviours (Clougherty, 2010). Gender and gender roles therefore impact where individuals spend time as well as what activities they partake in, and this in turn influences the exposures they face. Indeed, gender differences in time spent outdoors and physical activity participation could explain the differences seen in the results, however evidence for this is minimal.

Previous literature has discussed the link between ethnicity and socio-economic status (Grineski et al., 2010), and the relationship with housing, parental employment and general health. Forno and Celedón (2009) discusses how certain ethnicities are disproportionately represented among people living in poverty. As poverty has been associated with an increased prevalence of asthma, poverty may explain the relationship between ethnicity and asthma or wheeze, however it is important to consider other factors, including environmental exposures, health care access, and cultural or physiological factors. Given the association with socio-economic status, ethnic minorities are more likely to face increased air pollution exposure, however this was not found to be an issue in this study. Assari and Moghani Lankarani (2018) found that living above the poverty line was associated with a decreased likelihood of having asthma, and this relationship was stronger for white children compared to black children. Children from ethnic minorities may face under-diagnosis of asthma (Panico et al., 2007) which can be a consequence of limited health care access, lack of knowledge about available services and lower levels of health literacy (Panico et al., 2007). Data suggests that Bangladeshi mothers under-report asthma and wheezing in children when compared to white mothers (Panico et al., 2007).

Another theory put forward to explain health inequalities faced by ethnic minorities is the Minorities' Diminished Return theory (Assari and Moghani Lankarani, 2018). This theory states that the socially dominant and privileged groups gain the most health benefits, whilst the socially oppressed and deprived groups gain the least health benefits from the same socio-economic resources (Assari and Moghani Lankarani, 2018).

High levels of deprivation have been associated with higher rates of obesity (Stamatakis et al., 2010). Naeem and Silveyra (2019) found that a higher BMI increased the risk of asthma for male children more so than female children. Physiologically, the relationship between asthma and obesity is complex. Obesity causes a multitude of health issues, such as a reduced lung volume and a lower response to inhaled corticosteroids (Forno et al., 2011). Di Genova et al. (2018) posited that a bidirectional relationship could be present between asthma and obesity, where an individual with asthma may be less likely to partake in physical activity, resulting in weight gain. Longitudinal studies have examined the relationship between air pollution exposure and obesity, and found that exposure to high levels of traffic-related air pollution and second-hand smoke were associated with childhood obesity (Jerrett et al., 2014).

The relationship between maternal asthma, socioeconomic status and air pollution is straightforward and can be explained through the triple jeopardy theory. Living in an area of increased deprivation would result in exposure to higher levels of air pollution, a mother that already has asthma as a pre-existing health burden would therefore have an increased susceptibility to air pollution because of this (Jerrett et al., 2001). Therefore it can be assumed that a higher proportion of mothers living in the more deprived areas would experience asthma, just as a higher proportion of children living in the more deprived areas would also experience asthma. Consequently, a more deprived area would have higher proportions of mothers with asthma and children with asthma. As deprived areas are generally more polluted, this would exacerbate issues related to both mother and child asthma. Additionally, mothers living in the more deprived areas may not seek treatment for their own asthma, or for their child's asthma due to barriers restricting health care access, or indeed a low self-esteem (Macintyre et al., 1998).

Smoking patterns are associated with deprivation in England, with an increased prevalence in more deprived areas (Hiscock et al., 2012). This variation in smoking prevalence over different socioeconomic groups contributes to pre-existing health inequalities in society (Hiscock et al., 2012). The interaction between concurrent exposures, such as ambient air pollution and second-hand smoke could exacerbate asthma in children (Norbäck et al., 2019). Whilst some research has been conducted into this interaction, the results are still unclear (Norbäck et al., 2019). Indeed, one study found that exposure to second-hand smoke limits inflammation in the airways following exposure to low-level ambient $PM_{2.5}$ exposure (Rabinovitch et al., 2011).

Social housing is rented at lower rates to those that need it most, typically those with a lower income, and social housing developments are commonly situated in lower income areas (Hills, 2007). In addition, poor quality housing can impact on health in a number of ways (Hood, 2005). High humidity and mould has been associated with increased asthma morbidity and asthma related hospital admissions, as well as more frequent wheezing. Many social housing properties have been draft-proofed to prevent heat loss, thus reducing ventilation in the home which can result in increased indoor air pollution levels, specifically levels of NO_2, which can then exacerbate asthma (Sharpe et al., 2019). Having a familial income low enough to qualify for social housing could suggest other difficulties that could be faced when accessing healthcare or other amenities. The social aspect of this type of housing can also impact a child's health. Deprived areas can experience increased level of crime and social disorder, resulting in increased levels of stress for residents (Denton et al., 2004). In regards to second-hand smoke exposure, 33% of people living in social housing smoke compared to 10% of people living in houses they own themselves (Jackson et al., 2019). Furthermore, as social housing tends to be situated in more deprived areas, people living in social housing are more likely to be exposed to higher levels of air pollution.

Whilst literature has presented evidence suggesting a relationship between urban residency and socio-economic status, positing that rural communities are at a disadvantage, results are conflicting (Wheeler and Ben-Shlomo, 2005, Briggs et al., 2008). Individuals with a lower socio-economic status that live in an urban area are more likely to face increased air pollution exposure (Briggs et al., 2008)

as the more deprived tend to be situated closer to sources of pollution. However the reverse is true in rural areas (Wheeler and Ben-Shlomo, 2005) as individuals with a higher socio-economic status are more likely to live near major transport links.

This section has illustrated the complexities that lie in understanding the impact of different variables and exposures on asthma and wheezing prevalencein children. The triple jeopardy can help understand the pathways in which respiratory health is affected. Indeed, it could be said that the triple jeopardy can apply to the complex system of interactions and direct and indirect pathways between respiratory health and air pollution, deprivation, sex, ethnicity, and other variables previously mentioned.

7.7   Strengths and limitations

This thesis draws its strength from its data and methods. The Millennium Cohort Study provided panel data that followed children from birth throughout their childhood. A multidisciplinary and intergenerational study, the data provided contained important information regarding details such as familial health information, as well as social, economic and demographic information. In total, 44,219 observations were used in the analyses, which is a substantial size of a dataset.

Through a data linkage process, the inclusion of socio-economic indicators at both the individual and area level is another strength of this thesis. As stated in Chapter 1, using only one socio-economic status proxy fails to take into account the impact of both individual and area level socio-economic status on health. As few studies have examined this relationship, there is a clear knowledge gap here. Coupled with the modelled annual average air pollution data, which was available at a high resolution of $5km^2$, the dataset used in this thesis has proven to be a real strength. Extensive analysis through both cross-sectional, time series and multilevel modelling approaches has provided a valuable insight into the relationship between air pollution, health and individual and area level socio-economic status. The interaction terms that have been included in different analyses have shown that the relationships discussed are complex, yet it is vital that they are taken into consideration. The inclusion of the different geographies of England, England excluding London, and London only in the multilevel model

analysis is another strength of this thesis. Running the multilevel models for the three different geographies allows for a better understanding of the data. There was the potential for data to be skewed when examining England as a whole due to the inclusion of London as typically there would be a concentration of wealthier individuals living in areas of high pollution. By modelling England without London, the potential 'London effect' was removed from the analysis.

However, there are certain limitations to this thesis. As stated in Section 3.3.1.1, the responses in the Millennium Cohort Study relating to the health of the cohort member are reliant on parental response, and because of this certain questions are open to scrutiny. The survey questions chosen as the focus of this thesis, which were *"has [^Cohort child's name] ever had asthma?"* providing the variable for 'asthma ever' and *"has [^Cohort child name] had wheezing or whistling in the chest in the last 12 months?"* giving the variable for 'current wheeze', are posited to parents only. Therefore clinical diagnosis was not necessary when recording whether or not a child had asthma or wheezing issues. This could result in either underrepresentation or overrepresentation of asthma and wheezing prevalence among cohort members. Indeed, other variables from the questionnaire could have been utilised when examining asthma prevalence. For example, frequency of wheezing attacks, instances of severe wheezing or hospitalisation due to wheezing or asthma could have been chosen as the outcome variables of interest (Islam et al., 2021). Such a limitation is not unique to this work, and it is common in literature interested in childhood asthma.

Asthma is indeed nebulous and issues relate to a lack of a 'gold standard definition of asthma' (Dick et al., 2014) as touched upon in Section 2.6. However, the ISAAC questionnaire which the MCS questions were based off of is a validated questionnaire which has been used in many studies investigating childhood asthma (Lee, 2010, Al Ghobain et al., 2012, Ocampo et al., 2017). Additionally there are difficulties surrounding the clinical diagnosis of a child with asthma when they are in adolescence (Caudri et al., 2009, Moral et al., 2019). Due to these difficulties, the inclusion of current wheeze in addition to ever having asthma adds to the robustness to the data. Cases where a cohort member has wheezed in the previous 12 months may provide a clearer indication of current levels asthma prevalence amongst the children in the study.

Another limitation of this study is the use of binary variables and how this can result in important data being excluded. Ethnicity, for example, uses White British as the binary variable, grouping all other variables together as 'not White British'. Information about specific ethnicities is lost and this limits both the results and interpretation of said results, as different ethnicities can be diverse in terms of their general socio-economic status, their culture and health behaviours. The data becomes generalised and interpretations cannot be made about specific groups of people. This limitation was addressed in Chapter 6, where ethnicity was instead recorded as a categorical variable comprised of six different ethnicities or groups of ethnicities. This inclusion of ethnicity as a categorical variable allowed for further interpretation of the data. For example, the results show that a child that is mixed-race has a greater likelihood of having asthma compared to a child that is white.

The use of a binary variable also limits the information learned from a mother's employment status, as it does not specify if the employment is full- or part-time or give any indication of wage. In terms of obesity, it excludes information related to children who may be recorded as being overweight or underweight. In addition, obesity had to be calculated through BMI using the recorded height and weight of all children. Whilst BMI is more of an indicator of obesity rather than a true measure (Rothman, 2008), obesity in the MCS was defined by the International Obesity Task Force and the BMI cut-offs were age and sex specific, making the variable more reliable (Brophy et al., 2009). Making social housing a binary variable also loses valuable information about other housing tenures, for example the interpretation could then exclude someone who is registered as being homeless, or assumptions could not be made about people that own their own home outright.

Another potential limitation is the way in which air pollution data was recorded. As the air pollution data are available for the same year each wave was studied, important air pollution measurements in the years preceding and following each wave are ignored. These missing data may play an important role in terms of lagged exposure and its impacts on a child's respiratory health. Furthermore, as air pollution exposure is recorded as a yearly average, all variability throughout the year is effectively smoothed over. This could exclude major air pollution events, where there may have been a peak in emissions for a period of time that

then exacerbated asthma in children. Additionally, indoor air pollution data would have been beneficial for this study, although this information was not available. In terms of the modelling approaches, correlation was shown to be a problem amongst the air pollution variables (Table 3.3 and Table C.1 to C.3 in Appendix C). Correlation was also a potential issue among the social variables, although the correlation seen here was generally weak and not a concern. It was because of correlation amongst the air pollutants that analysis was focussed on one pollutant, NO_2. Whilst modelling interactions with all pollutants included in one model would have been interesting, the high correlation would have influenced the outputs. Much like the IMD, an index of pollution or air quality, which has been seen in other countries (Cromar et al., 2020, Morrissey et al., 2021a), could be beneficial to analysis such as the multilevel models conducted here.

7.8   Conclusion

This thesis aimed to address whether:

1. The association between respiratory health and air pollution is stronger amongst individuals of lower, compared to higher, socio-economic status.
2. Area level deprivation will interact with individual socio-economic status so that the impact of pollution on respiratory health is stronger for people with low socio-economic status living in the most deprived areas than people with low socio-economic status living in less deprived areas.

The literature presented in Chapter 2 highlighted previous findings on the impact of air pollution exposure, area level deprivation and individual level socio-economic status on health. Using a data linkage approach, data from the Millennium Cohort Study, EMEP4UK and IMD, facilitated cross-sectional, time series and multilevel analysis to examine the role of air pollution, area level deprivation and familial socio-economic status on childhood respiratory health.

Whilst cross-sectional analysis was useful to quickly interpret the data at specific time points, time series analysis built on this further by examining how different exposures impacts on health. Initial exposures to air pollution and deprivation in Wave 1 were compared against exposures over time to estimate if a critical exposure period in early life or if exposures over time had a greater influence over respiratory health in children. Multilevel modelling developed this analysis one step further through the inclusion of the spatial aspect of the data in addition to

the temporal aspect. Due to this, multilevel modelling provided the most robust results and reported that both individual and area level socio-economic status had a statistically significant impact on asthma prevalence in children, thereby playing a pivot role in a child's health. Although this was not the case in London. In comparison, it was found that for wheezing, area level socio-economic status had a greater, and more statistically significant impact that individual level socio-economic status. In terms of air pollution, when looking at England the results were mixed, however in London, NO_2 was found to have a statistically significant impact on asthma, increasing the likelihood of a child ever having asthma.

The multilevel analysis also presented the variation in asthma and wheezing rates that were due to between MSOA variation, within MSOA variation and over time variation. Over time, wheezing was found to vary considerably more when compared to asthma (47% compared to 14%), whilst the majority of asthma variation was found to be within MSOAs (85%). Very little variation in asthma or wheeze occurred between MSOAs, highlighting the importance of the individual, as well as the importance of time. As stated previously, asthma is an established chronic illness, and so has the potential to be more fixed over time, whilst current wheeze allows for more temporal variability. Interaction terms were useful to fully explore the two- and three-way relationships of interest in the analysis, in order to better understand how individual level socio-economic status, area level deprivation and air pollution interact with one another to impact on childhood respiratory health.

Rates of childhood asthma are increasing in the UK and at the same time, as noted in Chapter 1, child poverty rates in the UK are rising (Wickham et al., 2016). Further insight into the role of childhood familial socio-economic status is now more crucial than ever. This research helps to understand the complex relationships between asthma, air pollution and socio-economic status. The results presented in this thesis provide a valuable insight in terms of how to approach certain issues, and how policy makers should react in order to lessen the health burden faced by children suffering from respiratory health issues. It is important to consider the individual, and how socio-economic status can exacerbate the impact of air pollution exposure on their health.

This research has also shown the need for policy and interventions to target alleviating deprivation at both the individual and area level. Deprivation underpins

all aspects of the theoretical framework presented in this thesis, and so interventions and policy should be considered with both air pollution and deprivation in mind. However, policy recommendations can only do so much, and restrictions and regulations would be more beneficial to those who would benefit most. As those living in the most deprived areas are at most risk of exacerbated health issues, enforcing low emission zones in residential areas, in particular residential areas in deprived areas, would be advantageous. In addition, stricter land use regulations could have a positive impact on people's respiratory health, as those living in deprived areas are more likely to be situated close to polluting facilities. With a specific focus on childhood respiratory health, low emission zones surrounding schools could be encouraged during the hours that school would be in session. A further suggestion would be that an air quality index, much like the one seen in the U.S. or Malaysia (Cromar et al., 2020, Morrissey et al., 2021a), be created for England, as a composite indicator of air quality. Such an index could help deal with correlation seen in this thesis.

Current evidence points towards a complex relationship between genetic susceptibility, host factors (such as obesity), and environmental exposures influencing asthma prevalence (Dharmage et al., 2019). However, socio-economic status must also be considered. The results presented here show how important individual and area level socio-economic status are when examining asthma and wheezing prevalence in children. Whilst this thesis presents a good starting point, further analysis is required to properly understand the complex interactions underpinning the relationship between individual socio-economic status, area level deprivation and pollution exposure.

Appendices

Appendix A: The International Study of Asthma and Allergies in Childhood questionnaire – relevant questions

8.2 Core questionnaire for asthma

8.2.1 Questionnaire for 6/7 year olds (strongly recommended)

1 Has your child <u>ever</u> had wheezing
   or whistling in the chest
   at any time in the past?

   Yes ☐
   No ☐

   IF YOU HAVE ANSWERED "NO" PLEASE SKIP TO QUESTION 6

2 Has your child had wheezing or
   whistling in the chest
   <u>in the past 12 months?</u>

   Yes ☐
   No ☐

   IF YOU HAVE ANSWERED "NO" PLEASE SKIP TO QUESTION 6

3 How many attacks of wheezing
   has your child had
   <u>in the past 12 months?</u>

   None ☐
   1 to 3 ☐
   4 to 12 ☐
   More than 12 ☐

4 <u>In the past 12 months</u>, how often, on average, has
   your child's sleep been disturbed due to wheezing?

   Never woken with wheezing ☐
   Less than one night per week ☐
   One or more nights per week ☐

5 In the past 12 months, has wheezing
   ever been severe enough to limit your
   child's speech to only one or two
   words at a time between breaths?

   Yes ☐
   No ☐

6 Has your child <u>ever</u> had asthma?

   Yes ☐
   No ☐

7 In the past 12 months, has your
   child's chest sounded wheezy
   during or after exercise?

   Yes ☐
   No ☐

8 In the past 12 months, has your
   child had a dry cough at night,
   apart from a cough associated with
   a cold or chest infection?

   Yes ☐
   No ☐

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Appendix B: The Millennium Cohort Study – relevant questions

ASMA
Has [^Cohort child's name] ever had asthma?
1 Yes
2 No

WHEX
In the last 12 months has [^Cohort child's name]'s chest sounded wheezy during or after exercise?
1 Yes
2 No

WHCL
In the last 12 months has [^Cohort child's name] had a dry cough at night apart from a cough associated with a cold or chest infection?
1 Yes
2 No

ECZM
Has [^Cohort child's name] ever had eczema?
1 Yes
2 No

HAFV
Has [^Cohort child's name] ever had hayfever?
1 Yes
2 No

IF not reported having MEASLES at prior interview [FF. Meas <> 1]
|
| **MEAS**
| Has [^Cohort child's name] ever had any of the following illnesses?
|
| Measles
| 1 Yes
| 2 No
|
END OF FILTER

IF not reported having CHICKENPOX at prior interview [FF.Chic <> 1]
|
| **CHIC**
| (Has [^Cohort child's name] ever had any of the following illnesses?)
|
| Chickenpox
| 1 Yes
| 2 No
|
END OF FILTER

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Appendix C: **Correlation matrices for England, England excluding London and London only**

Table C.1 Correlation matrix for variables for England (1 of 2)

	Asthma	Wheezing	Child is female	Ethnicity	Child is obese	Mother is employed	Mother has asthma	Mother smokes
Asthma	1							
Wheezing	0.455	1						
Child is female	-0.054	-0.050	1					
Ethnicity	-0.009	-0.014	-0.003	1				
Child is obese	0.030	0.028	0.016	0.060	1			
Maternal employment	-0.022	-0.021	-0.001	-0.226	-0.018	1		
Maternal asthma	0.136	0.102	0.016	-0.095	0.006	-0.009	1	
Maternal smoking	0.051	0.042	-0.024	-0.159	0.031	-0.107	0.072	1
Lives in urban area	0.028	0.025	0.000	0.199	0.033	-0.094	0.000	0.067
Lives below poverty line	0.047	0.025	0.004	0.307	0.040	-0.481	0.016	0.181
IMD Score	0.042	0.026	-0.004	0.370	0.060	-0.293	0.004	0.139
NO_2 concentration	-0.006	-0.006	0.000	0.480	0.050	-0.152	-0.048	-0.043
PM_{10} concentration	-0.020	-0.005	-0.002	0.335	0.036	-0.102	-0.028	-0.041
$PM_{2.5}$ concentration	-0.024	-0.006	-0.005	0.324	0.033	-0.097	-0.034	-0.049
NO concentration	-0.012	-0.012	-0.001	0.465	0.045	-0.133	-0.049	-0.057
O_3 concentration	0.007	0.009	0.000	-0.430	-0.040	0.129	0.040	0.047

Table C.1 Correlation matrix for variables for England (2 of 2)

	Lives in urban area	Lives below poverty line	IMD score	NO$_2$ conc.	PM$_{10}$ conc.	PM$_{2.5}$ conc.	NO conc.	O$_3$ conc.
Lives in urban area	1							
Lives below poverty line	0.158	1						
IMD Score	0.317	0.458	1					
NO$_2$ concentration	0.421	0.198	0.376	1				
PM$_{10}$ concentration	0.302	0.080	0.158	0.714	1			
PM$_{2.5}$ concentration	0.282	0.069	0.124	0.717	0.959	1		
NO concentration	0.317	0.164	0.308	0.958	0.718	0.700	1	
O$_3$ concentration	-0.344	-0.172	-0.314	-0.954	-0.619	-0.677	-0.907	1

Table C.2 Correlation matrix for variables for England excluding London (1 of 2)

	Asthma	Wheezing	Child is female	Ethnicity	Child is obese	Mother is employed	Mother has asthma	Mother smokes
Asthma	1							
Wheezing	0.453	1						
Child is female	-0.056	-0.050	1					
Ethnicity	0.002	-0.004	0.017	1				
Child is obese	0.034	0.030	0.013	0.045	1			
Maternal employment	-0.027	-0.025	0.000	-0.262	-0.020	1		
Maternal asthma	0.129	0.101	0.015	-0.092	0.011	-0.017	1	
Maternal smoking	0.051	0.038	-0.025	-0.147	0.031	-0.118	0.070	1
Lives in urban area	0.037	0.033	0.001	0.178	0.029	-0.094	0.011	0.089
Lives below poverty line	0.051	0.030	0.006	0.342	0.038	-0.484	0.016	0.194
IMD Score	0.052	0.036	-0.004	0.415	0.057	-0.305	0.005	0.151
NO_2 concentration	0.017	0.016	0.009	0.398	0.037	-0.154	-0.023	0.008
PM_{10} concentration	-0.004	0.019	0.001	0.159	0.012	-0.083	0.005	0.007
$PM_{2.5}$ concentration	-0.011	0.015	-0.004	0.166	0.010	-0.078	-0.005	-0.007
NO concentration	0.015	0.015	0.012	0.378	0.031	-0.139	-0.023	-0.002
O_3 concentration	-0.012	-0.008	-0.009	-0.338	-0.027	0.116	0.018	0.007

Table C.2 Correlation matrix for variables for England excluding London (2 of 2)

	Lives in urban area	Lives below poverty line	IMD score	NO_2 conc.	PM_{10} conc.	$PM_{2.5}$ conc.	NO conc.	O_3 conc.
Lives in urban area	1							
Lives below poverty line	0.171	1						
IMD Score	0.328	0.470	1					
NO_2 concentration	0.431	0.226	0.418	1				
PM_{10} concentration	0.253	0.058	0.121	0.513	1			
$PM_{2.5}$ concentration	0.225	0.049	0.084	0.552	0.935	1		
NO concentration	0.338	0.200	0.371	0.965	0.485	0.508	1	
O_3 concentration	-0.323	-0.172	-0.305	-0.935	-0.423	-0.544	-0.908	1

Table C.3 Correlation matrix for variables for London only (1 of 2)

	Asthma	Wheezing	Child is female	Ethnicity	Child is obese	Mother is employed	Mother has asthma	Mother smokes
Asthma	1							
Wheezing	0.469	1						
Child is female	-0.049	-0.050	1					
Ethnicity	-0.009	-0.006	-0.060	1				
Child is obese	0.021	0.027	0.033	0.056	1			
Maternal employment	-0.005	-0.011	-0.008	-0.125	0.001	1		
Maternal asthma	0.176	0.105	0.020	-0.063	-0.010	0.019	1	
Maternal smoking	0.036	0.059	-0.018	-0.148	0.056	-0.075	0.055	1
Lives in urban area	-0.013	0.008	-0.001	0.050	0.015	0.001	-0.048	-0.016
Lives below poverty line	0.032	0.004	-0.010	0.274	0.043	-0.464	0.023	0.135
IMD Score	-0.018	-0.031	-0.006	0.319	0.071	-0.207	0.019	0.094
NO_2 concentration	-0.020	0.000	-0.034	0.226	0.002	-0.138	-0.013	-0.011
PM_{10} concentration	-0.027	-0.006	-0.012	0.141	0.017	-0.134	-0.009	0.020
$PM_{2.5}$ concentration	-0.028	-0.006	-0.012	0.106	0.014	-0.122	-0.004	0.020
NO concentration	-0.017	0.001	-0.031	0.203	0.002	-0.137	-0.012	-0.013
O_3 concentration	0.021	0.007	0.037	-0.226	-0.001	0.133	0.011	0.017

Table C.3 Correlation matrix for variables for London only (2 of 2)

	Lives in urban area	Lives below poverty line	IMD score	NO_2 conc.	PM_{10} conc.	$PM_{2.5}$ conc.	NO conc.	O_3 conc.
Lives in urban area	1							
Lives below poverty line	0.021	1						
IMD Score	0.059	0.391	1					
NO_2 concentration	0.091	0.240	0.543	1				
PM_{10} concentration	0.132	0.229	0.498	0.752	1			
$PM_{2.5}$ concentration	0.129	0.198	0.441	0.683	0.984	1		
NO concentration	0.085	0.228	0.513	0.987	0.782	0.726	1	
O_3 concentration	-0.054	-0.233	-0.526	-0.983	-0.652	-0.587	-0.966	1

Appendix D: Further multilevel models

Table D.1 Asthma area level multilevel models – PM_{10} only

ASTHMA	All England OR		95% CI		Excluding London OR		95% CI		London only OR		95% CI	
Child is female	0.45	***	0.36	0.55	0.45	***	0.34	0.55	0.43	**	0.23	0.76
Ethnicity												
White	REF				REF				REF			
Mixed	1.64	*	0.94	2.64	2.09	*	1.07	3.68	1.69		0.42	4.55
Indian	0.92		0.48	1.57	1.01		0.43	2.01	1.04		0.24	2.68
Pakistani & Bangladeshi	0.67	*	0.41	1.02	0.78		0.49	1.25	0.70		0.19	2.01
Black	0.68	*	0.38	1.05	0.66		0.24	1.49	1.24		0.45	2.54
Other	0.77		0.31	1.56	1.59		0.36	4.40	0.74		0.15	2.33
Child is obese	1.68	***	1.25	2.22	1.84	***	1.29	2.55	1.25		0.54	2.46
Mother is employed	1.23	**	1.05	1.43	1.20	*	0.98	1.43	1.49		0.91	2.33
Mother has asthma	9.48	***	7.29	12.41	8.20	***	5.97	11.10	36.23	***	13.87	78.17
Mother smokes	1.27	*	1.01	1.57	1.27	*	1.02	1.56	1.63		0.81	3.11
Lives in urban area	1.26		0.89	1.78	1.34		0.87	2.01	4.96		0.11	27.75
Lives below the poverty line	1.28	**	1.05	1.52	1.24	*	1.01	1.51	1.36		0.80	2.27
IMD (level of deprivation)												
1	REF				REF				REF			
2	1.47		0.88	2.18	1.84	*	1.02	3.05	0.90		0.17	2.83
3	1.47		0.92	2.30	1.48		0.79	2.39	0.49		0.08	1.62
4	1.83	**	1.16	2.77	1.96	*	1.10	3.30	2.45		0.37	8.30
5	2.85	***	1.80	4.27	3.29	***	1.75	5.28	0.77		0.08	2.48
6	3.25	***	1.98	4.79	3.28	***	1.68	5.36	0.44		0.05	1.51
7	2.92	***	1.86	4.26	3.40	***	1.91	5.53	0.84		0.10	3.26
8	3.54	***	2.30	5.17	4.52	***	2.58	7.19	0.97		0.10	3.41

	OR		95% CI		OR		95% CI		OR		95% CI	
9	3.68	***	2.39	5.54	5.01	***	2.54	8.00	0.28		0.04	0.82
10	4.10	***	2.41	6.33	4.26	***	2.04	7.08	0.55		0.07	1.79
PM$_{10}$ (level of pollution)												
low	REF				REF				REF			
mid-low	1.04		0.78	1.33	0.98		0.72	1.26	1.44		0.74	2.92
mid-high	0.96		0.71	1.26	1.23		0.90	1.63	2.93	**	1.17	6.27
high	0.90		0.63	1.18	1.25		0.87	1.79	2.01		0.73	4.44

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.001	0.002	0.04	0.002	0.0004	0.003	0.05	0.002	0.001	0.01	0.06
Level 2: Individual	19.12	17.47	20.77	85.32	19.05	17.31	21.29	85.27	26.40	19.45	35.15	88.92
Level 1: Wave				14.64				14.68				11.02

Table D.2 Wheeze area level multilevel models – PM$_{10}$ only

	All England				Excluding London				London only			
WHEEZE	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.64	***	0.57	0.72	0.66	***	0.57	0.74	0.60	***	0.44	0.79
Ethnicity												
White	REF				REF				REF			
Mixed	1.14		0.83	1.50	1.14		0.78	1.59	1.44		0.84	2.38
Indian	1.10		0.80	1.47	1.00		0.66	1.47	1.52		0.80	2.54
Pakistani & Bangladeshi	0.90		0.71	1.13	0.96		0.73	1.22	0.76		0.41	1.35
Black	0.88		0.66	1.17	0.67	*	0.39	1.06	1.40		0.90	2.18
Other	0.90		0.55	1.40	0.99		0.46	1.77	1.14		0.55	2.23
Child is obese	1.46	***	1.20	1.75	1.43	***	1.14	1.73	1.67	*	1.03	2.59
Mother is employed	0.86	**	0.76	0.97	0.88	*	0.79	0.99	0.76	*	0.56	1.00
Mother has asthma	2.70	***	2.34	3.12	2.61	***	2.25	2.99	3.46	***	2.18	5.26
Mother smokes	1.26	**	1.12	1.44	1.19	**	1.05	1.36	1.91	***	1.29	2.72

	OR	sig	95% CI low	95% CI high	OR	sig	95% CI low	95% CI high	OR	sig	95% CI low	95% CI high
Lives in urban area	1.33	*	1.07	1.62	1.34	***	1.11	1.58	1.01		0.37	2.77
Lives below the poverty line	1.03		0.90	1.17	1.05		0.92	1.19	0.94		0.66	1.32
IMD (level of deprivation)												
1	REF				REF				REF			
2	1.02		0.79	1.30	1.26		0.97	1.61	0.49	*	0.21	0.94
3	1.08		0.83	1.38	1.17		0.89	1.53	0.84		0.40	1.52
4	1.20		0.92	1.57	1.21		0.93	1.55	0.87		0.40	1.65
5	1.32	*	1.03	1.68	1.41	**	1.10	1.80	0.58	*	0.26	1.09
6	1.31	*	1.01	1.70	1.29	*	1.00	1.70	0.46	**	0.19	0.85
7	1.20		0.93	1.54	1.40	**	1.10	1.81	0.51	*	0.22	0.99
8	1.02		0.78	1.32	1.21		0.92	1.59	0.54	*	0.24	1.04
9	1.06		0.81	1.37	1.25	*	0.97	1.66	0.62		0.26	1.21
10	1.27	*	0.97	1.67	1.40	**	1.05	1.87	0.32	***	0.12	0.67
PM_{10} (level of pollution)												
low	REF				REF				REF			
mid-low	1.03		0.89	1.19	1.05		0.90	1.24	1.09		0.72	1.57
mid-high	0.90		0.77	1.04	0.99		0.83	1.18	1.40		0.90	2.09
high	0.79	***	0.67	0.91	1.05		0.86	1.27	0.99		0.61	1.49

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.002	0.001	0.003	0.05	0.002	0.001	0.01	0.07	0.04	0.01	0.11	1.14
Level 2: Individual	4.13	3.77	4.55	55.67	4.11	3.70	4.53	55.51	4.63	3.30	5.92	58.48
Level 1: Wave				44.28				44.42				40.38

Table D.3 Asthma area level multilevel models – PM$_{2.5}$ only

ASTHMA	All England				Excluding London				London only			
	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.45	***	0.36	0.56	0.46	***	0.36	0.57	0.45	**	0.20	0.82
Ethnicity												
White	REF				REF				REF			
Mixed	1.60		0.86	2.68	1.99	*	1.04	3.49	1.56		0.40	3.91
Indian	0.84		0.42	1.44	1.03		0.45	1.99	0.91		0.21	2.71
Pakistani & Bangladeshi	0.69		0.40	1.08	0.76		0.48	1.15	0.65		0.15	1.60
Black	0.62	*	0.34	1.05	0.79		0.27	1.94	1.31		0.47	2.84
Other	0.71		0.31	1.48	1.49		0.33	4.18	0.67		0.12	2.07
Child is obese	1.65	**	1.19	2.21	1.86	***	1.33	2.59	1.19		0.54	2.31
Mother is employed	1.24	**	1.04	1.47	1.21	*	1.00	1.42	1.51		0.92	2.33
Mother has asthma	9.41	***	7.02	12.37	8.17	***	6.01	11.16	31.03	***	12.34	67.50
Mother smokes	1.29	*	1.03	1.62	1.29	*	1.03	1.57	1.52		0.74	2.64
Lives in urban area	1.26		0.91	1.94	1.27		0.84	1.80	0.25	**	0.06	0.58
Lives below the poverty line	1.28	**	1.05	1.52	1.24	*	1.00	1.51	1.42		0.84	2.27
IMD (level of deprivation)												
1	REF				REF				REF			
2	1.37		0.84	2.14	1.79	**	1.11	2.94	0.75		0.19	2.04
3	1.35		0.85	2.09	1.44	*	0.95	2.44	0.49		0.10	1.37
4	1.67	*	1.03	2.66	1.92	***	1.24	3.16	2.21		0.62	5.81
5	2.59	***	1.59	4.23	3.15	***	1.91	5.05	0.82		0.20	2.28
6	3.00	***	1.83	4.69	3.18	***	1.98	5.44	0.40	*	0.09	1.21
7	2.63	***	1.53	4.13	3.36	***	2.04	5.48	0.78		0.18	2.31
8	3.09	***	1.90	4.83	4.40	***	2.71	7.28	0.99		0.25	2.76
9	3.31	***	1.97	5.30	4.83	***	2.91	8.30	0.32	*	0.06	1.10
10	3.56	***	2.06	5.99	4.18	***	2.34	7.31	0.50		0.09	1.64

PM2.5 (level of pollution)	OR		95% CI		OR		95% CI		OR		95% CI	
low	REF				REF				REF			
mid-low	1.15		0.87	1.48	1.20		0.90	1.55	1.21		0.62	2.18
mid-high	1.04		0.76	1.37	1.32	*	0.98	1.66	1.65		0.68	3.27
high	1.15		0.83	1.66	1.30	*	0.97	1.70	2.01		0.80	4.22

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0003	0.002	0.03	0.001	0.0003	0.002	0.03	0.06	0.005	0.16	1.76
Level 2: Individual	19.23	17.18	22.15	85.39	18.63	17.01	20.62	84.99	24.60	18.58	31.90	88.20
Level 1: Wave				14.58				14.98				10.04

Table D.4 Wheeze area level multilevel models – PM2.5 only

	All England				Excluding London				London only			
WHEEZE	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.64	***	0.58	0.72	0.65	***	0.57	0.73	0.62	***	0.45	0.83
Ethnicity												
White	REF				REF				REF			
Mixed	1.13		0.84	1.47	1.14		0.80	1.58	1.43		0.75	2.44
Indian	1.11		0.79	1.50	0.99		0.64	1.50	1.46		0.79	2.41
Pakistani & Bangladeshi	0.90		0.72	1.13	0.95		0.73	1.23	0.76		0.40	1.30
Black	0.84		0.63	1.11	0.64	*	0.38	1.09	1.44		0.92	2.18
Other	0.88		0.53	1.33	1.03		0.47	1.90	1.09		0.58	1.93
Child is obese	1.46	***	1.20	1.74	1.42	**	1.14	1.74	1.65	*	1.03	2.50
Mother is employed	0.88	*	0.79	0.98	0.89	*	0.80	1.00	0.77	*	0.57	1.01
Mother has asthma	2.67	***	2.31	3.06	2.62	***	2.24	3.07	3.47	***	2.28	5.16
Mother smokes	1.26	***	1.11	1.42	1.19	**	1.04	1.36	1.88	**	1.33	2.61
Lives in urban area	1.36	***	1.14	1.67	1.31	***	1.09	1.62	1.40		0.21	6.66
Lives below the poverty line	1.05		0.93	1.17	1.04		0.92	1.17	0.95		0.70	1.31

IMD (level of deprivation)

IMD	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high
1	REF				REF				REF			
2	1.02		0.79	1.28	1.34	*	1.00	1.77	0.47	**	0.23	0.85
3	1.09		0.88	1.37	1.24		0.91	1.61	0.88		0.47	1.62
4	1.21		0.94	1.51	1.29	*	0.98	1.69	0.88		0.44	1.61
5	1.31	**	1.05	1.65	1.52	**	1.16	1.99	0.60	*	0.31	1.07
6	1.30	*	1.02	1.62	1.39	**	1.06	1.84	0.44	**	0.22	0.79
7	1.18		0.92	1.47	1.51	**	1.14	2.04	0.53	*	0.25	1.00
8	1.01		0.79	1.26	1.30		0.97	1.71	0.58	*	0.28	1.07
9	1.03		0.77	1.29	1.37	*	1.00	1.82	0.61		0.29	1.22
10	1.24	*	0.97	1.55	1.55	**	1.11	2.15	0.33	***	0.14	0.66

$PM_{2.5}$ (level of pollution)

	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high
low	REF				REF				REF			
mid-low	1.03		0.87	1.19	1.07		0.90	1.27	0.99		0.68	1.40
mid-high	0.94		0.79	1.09	1.02		0.84	1.19	1.04		0.65	1.56
high	0.82	**	0.67	0.97	1.06		0.89	1.28	0.97		0.58	1.47

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.004	0.001	0.01	0.11	0.001	0.0005	0.003	0.04	0.08	0.02	0.20	2.51
Level 2: Individual	4.16	3.74	4.56	55.82	4.21	3.80	4.70	56.15	4.47	3.42	5.60	57.60
Level 1: Wave				44.07				43.81				39.89

Table D.5 Asthma area level multilevel models – NO only

ASTHMA	All England				Excluding London				London only			
	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.46	***	0.37	0.56	0.45	***	0.36	0.58	0.47	**	0.24	0.85
Ethnicity												
White	REF				REF				REF			
Mixed	1.64	*	0.94	2.72	2.17	*	0.99	3.92	1.63		0.41	4.85
Indian	0.95		0.46	1.71	1.15		0.51	2.26	1.00		0.28	2.59
Pakistani & Bangladeshi	0.70	*	0.48	1.00	0.82		0.51	1.31	0.63		0.17	1.84
Black	0.64		0.36	1.14	0.75		0.25	1.64	1.26		0.52	2.71
Other	0.78		0.32	1.62	1.73		0.35	5.44	0.71		0.16	2.01
Child is obese	1.67	**	1.17	2.25	1.86	***	1.32	2.58	1.20		0.51	2.38
Mother is employed	1.22	**	1.04	1.44	1.18	*	1.01	1.39	1.50		0.88	2.43
Mother has asthma	9.15	***	6.91	12.61	8.05	***	5.71	10.93	33.36	***	11.77	79.43
Mother smokes	1.27	*	1.03	1.53	1.25	*	1.03	1.54	1.53		0.75	2.71
Lives in urban area	1.28		0.82	1.84	1.49	**	1.12	1.97	0.02	***	0.00	0.06
Lives below the poverty line	1.28	**	1.06	1.52	1.24	*	1.01	1.49	1.42		0.81	2.42
IMD (level of deprivation)												
1	REF				REF				REF			
2	1.40		0.92	2.09	1.74	*	1.00	2.78	0.78		0.20	1.89
3	1.36		0.88	2.03	1.44		0.75	2.38	0.56		0.11	1.49
4	1.69	**	1.09	2.50	1.97	*	1.02	3.27	2.16		0.59	5.59
5	2.59	***	1.69	3.81	3.21	***	1.77	5.42	0.85		0.20	2.15
6	3.04	***	2.03	4.37	3.29	***	1.79	5.18	0.43	*	0.11	1.11
7	2.72	***	1.86	4.04	3.40	***	1.81	5.47	0.87		0.24	2.26
8	3.23	***	2.10	4.78	4.46	***	2.29	7.13	1.32		0.32	3.63
9	3.34	***	2.15	5.12	4.72	***	2.66	7.19	0.35	*	0.07	1.01
10	3.72	***	2.46	5.79	4.13	***	2.16	6.58	0.54		0.11	1.50

NO (level of pollution)									
low	REF			REF			REF		
mid-low	1.09	0.86	1.40	0.96	0.70	1.37	1.14	0.53	2.14
mid-high	1.07	0.81	1.40	0.75	0.50	1.10	1.13	0.47	2.49
high	0.96	0.70	1.28	1.00	0.66	1.40	1.84	0.64	4.30
	Mean	95% CI	VPC	Mean	95% CI		VPC	Mean	95% CI	VPC
Level 3: MSOA	0.001	0.0003	0.001	0.02	0.002	0.0003	0.01	0.07	0.002	0.001	0.005	0.06
Level 2: Individual	18.80	16.57	21.05	85.10	18.58	16.75	20.68	84.96	24.72	18.88	32.43	88.26
Level 1: Wave				14.88				14.97				11.68

Table D.6 Wheeze area level multilevel models – NO only

	All England				Excluding London				London only			
WHEEZE	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.64	***	0.57	0.71	0.65	***	0.57	0.72	0.61	**	0.44	0.82
Ethnicity												
White	REF				REF				REF			
Mixed	1.21		0.91	1.58	1.14		0.80	1.57	1.37		0.75	2.22
Indian	1.18		0.85	1.61	1.03		0.70	1.49	1.38		0.79	2.17
Pakistani & Bangladeshi	0.97		0.76	1.20	0.99		0.76	1.26	0.73		0.36	1.26
Black	0.93		0.68	1.24	0.65	*	0.39	1.06	1.32		0.86	2.00
Other	0.95		0.60	1.48	0.98		0.44	1.81	1.06		0.53	1.93
Child is obese	1.44	**	1.17	1.74	1.44	**	1.15	1.78	1.60	*	0.97	2.45
Mother is employed	0.87	**	0.78	0.96	0.89	*	0.78	1.01	0.77	*	0.58	1.01
Mother has asthma	2.70	***	2.36	3.13	2.65	***	2.30	3.05	3.31	***	2.15	4.89
Mother smokes	1.25	***	1.10	1.42	1.20	**	1.04	1.37	1.82	***	1.30	2.53
Lives in urban area	1.44	***	1.18	1.74	1.54	***	1.25	1.95	0.83		0.05	2.06
Lives below the poverty line	1.03		0.91	1.16	1.04		0.90	1.20	0.98		0.70	1.33

IMD (level of deprivation)

	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high
1	REF				REF				REF			
2	1.03		0.80	1.32	1.25		0.92	1.64	0.51	*	0.23	0.99
3	1.09		0.85	1.39	1.20		0.91	1.58	0.91		0.42	1.77
4	1.22		0.94	1.56	1.22		0.93	1.62	1.03		0.46	2.03
5	1.34	*	1.04	1.67	1.42	**	1.08	1.85	0.67		0.30	1.30
6	1.31	*	1.01	1.63	1.30	*	1.00	1.66	0.50	*	0.23	1.03
7	1.17		0.88	1.49	1.42	**	1.06	1.84	0.62		0.25	1.30
8	1.05		0.81	1.37	1.23		0.92	1.60	0.64		0.26	1.29
9	1.08		0.83	1.38	1.27		0.95	1.68	0.68		0.29	1.46
10	1.36	**	1.05	1.76	1.44	**	1.06	1.88	0.38	*	0.12	0.81

NO (level of pollution)

	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high	Est	Sig	95% CI low	95% CI high
low	REF				REF				REF			
mid-low	0.89		0.76	1.04	0.85	*	0.71	1.00	0.77		0.51	1.12
mid-high	0.81	*	0.68	0.97	0.78	**	0.64	0.94	0.93		0.58	1.45
high	0.68	***	0.56	0.83	0.85		0.69	1.04	0.83		0.51	1.33

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.002	0.0005	0.003	0.05	0.001	0.0005	0.001	0.02	0.20	0.02	0.59	5.86
Level 2: Individual	4.13	3.75	4.61	55.69	4.16	3.78	4.54	55.86	4.33	2.96	6.23	56.83
Level 1: Wave				44.26				44.12				37.31

Table D.7 Asthma area level multilevel models – O_3 only

ASTHMA	All England OR		95% CI		Excluding London OR		95% CI		London only OR		95% CI	
Child is female	0.46	***	0.36	0.56	0.46	***	0.36	0.56	0.44	**	0.22	0.77
Ethnicity												
White	REF				REF				REF			
Mixed	1.61		0.90	2.73	2.11	*	0.98	3.82	1.66		0.46	5.28
Indian	0.86		0.44	1.47	1.06		0.44	2.32	0.95		0.22	2.55
Pakistani & Bangladeshi	0.67	*	0.42	0.98	0.81		0.49	1.22	0.64		0.17	1.72
Black	0.60	*	0.32	0.98	0.73		0.25	1.69	1.31		0.47	2.88
Other	0.75		0.24	1.62	1.69		0.36	4.73	0.75		0.16	2.44
Child is obese	1.68	***	1.23	2.26	1.86	***	1.32	2.52	1.17		0.50	2.29
Mother is employed	1.23	*	1.02	1.47	1.18	*	0.99	1.40	1.51		0.90	2.31
Mother has asthma	9.27	***	6.97	12.57	8.36	***	6.23	11.53	32.29	***	12.61	66.33
Mother smokes	1.29	**	1.05	1.57	1.27	*	1.00	1.57	1.48		0.75	2.57
Lives in urban area	1.19		0.83	1.60	1.36	*	1.00	1.92	0.03	***	0.00	0.16
Lives below the poverty line	1.27	**	1.04	1.51	1.22	*	0.99	1.49	1.46		0.81	2.35
IMD (level of deprivation)												
1	REF				REF				REF			
2	1.41		0.90	2.23	1.75	*	0.95	2.87	0.84		0.22	2.12
3	1.35		0.84	2.04	1.47		0.77	2.71	0.53		0.11	1.42
4	1.68	*	1.05	2.57	1.95	**	1.11	3.64	2.89	*	0.87	7.41
5	2.63	***	1.69	3.86	3.24	***	1.84	5.61	0.98		0.23	2.53
6	3.06	***	1.97	4.67	3.34	***	2.00	5.98	0.57		0.11	1.60
7	2.74	***	1.72	4.31	3.44	***	2.01	6.20	1.16		0.34	3.02
8	3.32	***	2.16	5.04	4.57	***	2.58	8.22	1.56		0.35	4.57
9	3.53	***	2.13	5.50	4.94	***	2.72	9.21	0.42	*	0.09	1.15
10	3.79	***	2.18	6.31	4.50	***	2.40	10.03	0.76		0.13	2.34

O₃ (level of pollution)

	OR	95% CI		OR	95% CI		OR	95% CI	
low	REF			REF			REF		
mid-low	0.93	0.68	1.17	0.96	0.69	1.30	0.95	0.42	1.78
mid-high	1.09	0.80	1.44	0.98	0.68	1.32	0.84	0.31	1.70
high	0.92	0.66	1.24	1.11	0.76	1.56	1.41	0.48	3.23

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.01	0.001	0.01	0.16	0.002	0.001	0.005	0.08	0.01	0.001	0.03	0.19
Level 2: Individual	19.12	17.12	21.83	85.32	18.94	17.00	20.95	85.20	24.05	18.95	29.29	87.97
Level 1: Wave				14.52				14.72				11.84

Table D.8 Wheeze area level multilevel models – O₃ only

	All England				Excluding London				London only			
WHEEZE	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.64	***	0.57	0.72	0.65	***	0.58	0.73	0.60	***	0.43	0.79
Ethnicity												
White	REF				REF				REF			
Mixed	1.18		0.86	1.58	1.15		0.80	1.60	1.36		0.74	2.26
Indian	1.17		0.86	1.56	1.02		0.66	1.47	1.46		0.81	2.32
Pakistani & Bangladeshi	0.95		0.74	1.19	0.98		0.75	1.24	0.75		0.39	1.30
Black	0.89		0.66	1.20	0.67		0.37	1.10	1.38		0.86	2.06
Other	0.92		0.58	1.37	1.01		0.48	1.84	1.06		0.54	1.82
Child is obese	1.46	***	1.20	1.75	1.44	**	1.14	1.77	1.67	*	0.98	2.57
Mother is employed	0.87	**	0.78	0.97	0.89	*	0.79	1.01	0.76	*	0.57	1.02
Mother has asthma	2.71	***	2.34	3.10	2.61	***	2.25	3.04	3.40	***	2.20	5.24
Mother smokes	1.26	***	1.12	1.42	1.20	**	1.06	1.37	1.89	***	1.31	2.68
Lives in urban area	1.41	**	1.10	1.70	1.39	**	1.09	1.71	14.51	**	1.13	67.91
Lives below the poverty line	1.04		0.92	1.16	1.05		0.92	1.20	0.95		0.67	1.30

	Model 1 Mean		CI low	CI high	Model 2 Mean		CI low	CI high	Model 3 Mean		CI low	CI high
IMD (level of deprivation)												
1	REF				REF				REF			
2	1.06		0.83	1.33	1.21		0.90	1.57	0.47	**	0.22	0.86
3	1.13		0.89	1.43	1.13		0.84	1.55	0.83		0.42	1.43
4	1.25	*	0.97	1.56	1.19		0.90	1.54	0.88		0.43	1.50
5	1.37	**	1.07	1.71	1.38	*	1.04	1.81	0.60		0.29	1.11
6	1.37	**	1.07	1.69	1.26		0.95	1.68	0.45	**	0.22	0.84
7	1.23		0.95	1.55	1.37	*	1.03	1.82	0.52	*	0.26	0.93
8	1.09		0.83	1.38	1.19		0.91	1.55	0.56	*	0.26	1.06
9	1.12		0.86	1.44	1.21		0.90	1.61	0.59		0.28	1.15
10	1.41	**	1.08	1.81	1.42	*	1.03	1.90	0.31	***	0.13	0.58
O$_3$ (level of pollution)												
low	REF				REF				REF			
mid-low	1.15		0.97	1.36	1.01		0.84	1.21	0.91		0.61	1.35
mid-high	1.39	***	1.18	1.65	1.06		0.87	1.27	0.98		0.60	1.54
high	1.33	**	1.09	1.60	1.12		0.91	1.35	1.02		0.61	1.58

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0002	0.002	0.02	0.002	0.001	0.003	0.05	0.07	0.004	0.20	2.08
Level 2: Individual	4.16	3.82	4.56	55.84	4.07	3.69	4.48	55.29	4.30	3.31	5.81	56.68
Level 1: Wave				44.14				44.66				41.24

Table D.9 Asthma interaction multilevel models – PM_{10} only

ASTHMA	All England				Excluding London				London only			
	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.45	***	0.37	0.55	0.46	***	0.35	0.57	0.44	***	0.21	0.76
Ethnicity												
White	REF				REF				REF			
Mixed	1.95	**	1.15	3.20	2.15	*	0.98	3.98	1.76		0.47	4.75
Indian	1.04		0.56	1.74	1.28		0.54	2.77	1.04		0.29	2.53
Pakistani & Bangladeshi	0.77		0.51	1.10	0.86		0.53	1.31	0.78		0.21	2.13
Black	0.96		0.57	1.63	0.81		0.27	1.78	1.32		0.54	2.70
Other	1.02		0.43	2.18	1.50		0.34	4.32	0.96		0.21	2.70
Child is obese	1.71	***	1.27	2.23	1.81	***	1.29	2.43	1.20		0.51	2.30
Mother is employed	1.22	**	1.03	1.44	1.20	*	0.99	1.42	1.42		0.90	2.11
Mother has asthma	9.34	***	6.92	12.69	8.52	***	5.85	12.21	29.10	***	12.13	55.56
Mother smokes	1.29	*	1.05	1.57	1.31	**	1.06	1.58	1.46		0.76	2.58
Lives in urban area	1.72	***	1.23	2.63	1.72	***	1.28	2.27	1.13		0.28	3.15
Lives below the poverty line	1.29	**	1.07	1.55	1.27	*	0.99	1.57	1.80	*	0.96	2.97
IMD (level of deprivation)	1.02	***	1.01	1.02	1.02	***	1.01	1.03	0.99		0.96	1.02
PM_{10} (level of pollution)	0.90	***	0.87	0.92	0.94	***	0.89	0.98	0.88	***	0.80	0.94
Poverty*IMD	1.00		0.99	1.01	1.00		0.99	1.01	1.00		0.96	1.04
Poverty*PM_{10}	0.99		0.91	1.07	0.94		0.85	1.05	1.14		0.80	1.65
IMD*PM_{10}	1.00		1.00	1.00	1.00		1.00	1.01	1.01		0.99	1.03
Poverty*IMD*PM_{10}	1.00		0.99	1.00	1.00		0.99	1.01	0.96	**	0.93	0.99
	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0003	0.004	0.04	0.001	0.0005	0.003	0.04	0.06	0.003	0.23	1.71
Level 2: Individual	18.80	17.20	20.39	85.11	18.75	16.80	21.27	85.07	21.90	15.91	26.78	86.94
Level 1: Wave				14.85				14.89				11.35

Table D.10 Wheeze interaction multilevel models – PM_{10} only

WHEEZE	All England OR		95% CI		Excluding London OR		95% CI		London only OR		95% CI	
Child is female	0.64	***	0.57	0.72	0.66	***	0.58	0.75	0.61	***	0.46	0.82
Ethnicity												
White	REF				REF				REF			
Mixed	1.10		0.82	1.46	1.12		0.78	1.57	1.29		0.72	2.20
Indian	1.02		0.74	1.37	0.94		0.63	1.35	1.51		0.88	2.37
Pakistani & Bangladeshi	0.88		0.70	1.09	0.90		0.70	1.13	0.81		0.40	1.48
Black	0.80		0.57	1.06	0.60	*	0.34	0.95	1.26		0.81	1.85
Other	0.82		0.51	1.26	0.94		0.43	1.75	1.06		0.52	1.84
Child is obese	1.44	***	1.19	1.74	1.43	**	1.14	1.77	1.62	*	1.00	2.49
Mother is employed	0.87	*	0.78	0.98	0.89	*	0.79	1.01	0.77	*	0.58	1.03
Mother has asthma	2.71	***	2.34	3.15	2.61	***	2.21	3.05	3.22	***	2.04	4.67
Mother smokes	1.25	***	1.11	1.40	1.17	*	1.02	1.35	1.80	**	1.26	2.51
Lives in urban area	1.25	*	1.02	1.52	1.25	*	1.02	1.48	2.25		0.31	13.01
Lives below the poverty line	1.04		0.91	1.17	1.05		0.91	1.21	1.08		0.75	1.49
IMD (level of deprivation)	1.01	**	1.00	1.01	1.01	***	1.00	1.02	0.99		0.98	1.01
PM_{10} (level of pollution)	0.99		0.96	1.02	1.02		0.97	1.06	1.01		0.97	1.05
Poverty*IMD	0.99	*	0.99	1.00	0.99	*	0.99	1.00	0.98		0.95	1.00
Poverty*PM_{10}	0.99		0.93	1.04	0.97		0.90	1.05	1.25	*	0.99	1.58
IMD*PM_{10}	1.00	*	1.00	1.00	1.00		1.00	1.00	1.00		0.99	1.01
Poverty*IMD*PM_{10}	1.00		1.00	1.00	1.00		1.00	1.01	0.97	***	0.95	0.99
	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.002	0.001	0.005	0.06	0.001	0.0003	0.001	0.02	0.01	0.001	0.07	0.38
Level 2: Individual	4.14	3.79	4.57	55.70	4.12	3.67	4.59	55.61	4.24	3.33	5.36	56.32
Level 1: Wave				44.24				44.37				43.30

Table D.11 Asthma interaction multilevel models – PM$_{2.5}$ only

ASTHMA	All England				Excluding London				London only			
	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.46	***	0.36	0.58	0.46	***	0.36	0.59	0.43	**	0.22	0.75
Ethnicity												
White	REF				REF				REF			
Mixed	1.83	*	1.07	2.91	2.17	*	1.08	3.89	1.79		0.46	4.65
Indian	1.08		0.54	1.98	1.30		0.50	2.64	1.00		0.23	2.58
Pakistani & Bangladeshi	0.79		0.53	1.12	0.82		0.46	1.33	0.71		0.17	2.01
Black	0.87		0.49	1.40	0.81		0.29	1.82	1.39		0.55	3.01
Other	0.94		0.38	2.03	1.53		0.31	4.31	0.76		0.18	2.28
Child is obese	1.71	***	1.22	2.30	1.88	***	1.36	2.57	1.15		0.52	2.18
Mother is employed	1.23	**	1.03	1.44	1.21	*	1.00	1.44	1.55		0.91	2.40
Mother has asthma	9.38	***	6.94	12.31	8.49	***	6.34	11.23	34.77	***	11.60	96.10
Mother smokes	1.32	**	1.08	1.59	1.33	*	1.04	1.67	1.49		0.77	2.51
Lives in urban area	1.71	***	1.20	2.33	1.82	*	1.12	2.60	0.10	***	0.01	0.33
Lives below the poverty line	1.29	**	1.05	1.58	1.27	*	1.03	1.58	1.76	*	0.90	3.07
IMD (level of deprivation)	1.01	***	1.01	1.02	1.02	***	1.01	1.03	1.00		0.96	1.03
PM$_{2.5}$ (level of pollution)	0.92	***	0.89	0.95	0.93	*	0.83	1.00	0.73	***	0.67	0.81
Poverty*IMD	1.00		0.99	1.01	1.00		0.99	1.01	1.00		0.95	1.05
Poverty*PM$_{2.5}$	0.96		0.88	1.06	0.96		0.84	1.10	1.30		0.78	2.11
IMD*PM$_{2.5}$	1.00		1.00	1.00	1.004		1.00	1.01	1.01		0.99	1.04
Poverty*IMD*PM$_{2.5}$	1.00		0.99	1.00	1.00		0.99	1.00	0.95		0.91	1.00
	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0001	0.003	0.04	0.002	0.001	0.01	0.07	0.01	0.001	0.02	0.19
Level 2: Individual	19.12	17.11	21.35	85.32	19.11	16.99	22.12	85.32	24.21	16.85	33.89	88.04
Level 1: Wave				14.64				14.61				11.77

Table D.12 Wheeze interaction multilevel models – PM$_{2.5}$ only

WHEEZE	All England OR		All England 95% CI		Excluding London OR		Excluding London 95% CI		London only OR		London only 95% CI	
Child is female	0.64	***	0.57	0.71	0.65	***	0.57	0.73	0.61	***	0.44	0.82
Ethnicity												
White	REF				REF				REF			
Mixed	1.11		0.83	1.46	1.11		0.77	1.55	1.35		0.76	2.23
Indian	1.04		0.76	1.43	0.95		0.63	1.40	1.48		0.86	2.35
Pakistani & Bangladeshi	0.88		0.71	1.10	0.89		0.70	1.10	0.85		0.47	1.44
Black	0.81		0.59	1.08	0.61	*	0.35	0.97	1.32		0.84	1.92
Other	0.83		0.53	1.21	0.96		0.47	1.74	1.08		0.53	1.88
Child is obese	1.45	***	1.20	1.74	1.43	***	1.15	1.77	1.63	*	1.01	2.52
Mother is employed	0.87	**	0.78	0.96	0.88	*	0.78	0.98	0.76	*	0.56	1.01
Mother has asthma	2.70	***	2.30	3.09	2.57	***	2.17	3.01	3.31	***	2.19	4.84
Mother smokes	1.24	**	1.09	1.40	1.16	**	1.03	1.32	1.80	***	1.24	2.53
Lives in urban area	1.27	*	1.02	1.50	1.30	**	1.07	1.55	1.92		0.51	5.52
Lives below the poverty line	1.03		0.89	1.16	1.06		0.92	1.21	1.05		0.77	1.40
IMD (level of deprivation)	1.01	*	1.00	1.01	1.01	***	1.00	1.01	0.99		0.97	1.01
PM$_{2.5}$ (level of pollution)	0.97	***	0.94	0.99	1.00		0.94	1.05	0.99		0.90	1.13
Poverty*IMD	0.99	*	0.99	1.00	0.99	*	0.98	1.00	0.98		0.95	1.01
Poverty*PM$_{2.5}$	1.00		0.94	1.06	0.99		0.91	1.08	1.27		0.91	1.69
IMD*PM$_{2.5}$	1.00	*	0.99	1.00	1.00		0.996	1.00	1.00		0.98	1.01
Poverty*IMD*PM$_{2.5}$	1.00		1.00	1.01	1.00		1.00	1.01	0.97	**	0.94	0.99
	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0002	0.001	0.02	0.001	0.0003	0.002	0.02	0.10	0.03	0.32	2.99
Level 2: Individual	4.05	3.65	4.49	55.19	4.07	3.63	4.65	55.32	4.17	3.35	5.14	55.88
Level 1: Wave				44.79				44.66				41.13

Table D.13 Asthma interaction multilevel models – NO only

ASTHMA	All England OR		95% CI		Excluding London OR		95% CI		London only OR		95% CI	
Child is female	0.45	***	0.35	0.55	0.46	***	0.36	0.57	0.43	*	0.23	0.84
Ethnicity												
White	REF				REF				REF			
Mixed	1.91	**	1.13	3.04	2.10	*	0.98	3.67	1.99		0.58	5.10
Indian	1.17		0.60	2.02	1.24		0.53	2.52	1.20		0.35	3.07
Pakistani & Bangladeshi	0.85		0.53	1.27	0.83		0.50	1.30	0.97		0.22	2.92
Black	0.99		0.56	1.67	0.83		0.24	2.00	1.46		0.62	2.98
Other	1.07		0.45	2.16	1.67		0.33	4.73	1.02		0.24	2.61
Child is obese	1.70	**	1.25	2.25	1.87	***	1.32	2.56	1.14		0.48	2.31
Mother is employed	1.23	**	1.04	1.46	1.20	*	0.99	1.45	1.48	*	0.99	2.21
Mother has asthma	9.15	***	7.00	12.05	8.29	***	6.07	11.11	29.06	***	11.55	60.60
Mother smokes	1.31	**	1.06	1.59	1.30	**	1.06	1.58	1.48		0.72	2.53
Lives in urban area	1.66	***	1.22	2.24	1.63	***	1.25	2.22	0.12	***	0.01	0.41
Lives below the poverty line	1.34	**	1.07	1.60	1.30	*	1.05	1.58	1.99	*	1.09	3.46
IMD (level of deprivation)	1.02	***	1.01	1.02	1.02	***	1.01	1.03	0.98		0.94	1.01
NO (level of pollution)	0.97	**	0.95	0.99	0.99		0.94	1.02	1.01		0.95	1.07
Poverty*IMD	1.00		0.99	1.02	1.00		0.99	1.01	1.02		0.97	1.07
Poverty*NO	1.01		0.98	1.04	1.01		0.95	1.06	0.99		0.91	1.08
IMD*NO	1.00		1.00	1.00	1.00		1.00	1.00	1.00		1.00	1.01
Poverty*IMD*NO	1.00		1.00	1.00	1.00		1.00	1.00	0.99	***	0.98	0.99

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.002	0.001	0.005	0.05	0.003	0.001	0.005	0.09	0.002	0.0004	0.01	0.06
Level 2: Individual	18.62	16.84	20.92	84.99	18.75	16.48	21.41	85.07	23.47	17.42	30.36	87.71
Level 1: Wave				14.96				14.84				12.23

Table D.14 Wheeze interaction multilevel models – NO only

WHEEZE	All England				Excluding London				London only			
	OR		95% CI		OR		95% CI		OR		95% CI	
Child is female	0.64	***	0.58	0.71	0.65	***	0.57	0.74	0.59	***	0.42	0.78
Ethnicity												
White	REF				REF				REF			
Mixed	1.14		0.85	1.48	1.11		0.77	1.52	1.35		0.77	2.23
Indian	1.07		0.75	1.42	0.95		0.62	1.37	1.42		0.79	2.37
Pakistani & Bangladeshi	0.88		0.71	1.12	0.91		0.70	1.14	0.82		0.43	1.38
Black	0.84		0.61	1.12	0.60	*	0.34	0.96	1.33		0.83	2.04
Other	0.86		0.55	1.31	0.93		0.43	1.78	1.16		0.58	2.11
Child is obese	1.44	***	1.18	1.75	1.41	***	1.13	1.73	1.62	*	1.02	2.50
Mother is employed	0.89	*	0.80	0.99	0.89	*	0.79	1.02	0.75	**	0.57	0.96
Mother has asthma	2.71	***	2.33	3.14	2.61	***	2.22	3.05	3.41	***	2.10	5.20
Mother smokes	1.24	***	1.09	1.38	1.17	*	1.03	1.32	1.85	***	1.26	2.57
Lives in urban area	1.29	**	1.06	1.53	1.34	***	1.13	1.60	3.19		0.19	11.16
Lives below the poverty line	1.06		0.92	1.21	1.06		0.91	1.21	1.08		0.78	1.48
IMD (level of deprivation)	1.01	**	1.00	1.01	1.01	***	1.00	1.01	0.98	*	0.97	1.00
NO (level of pollution)	0.99		0.98	1.01	1.00		0.98	1.02	1.02		0.99	1.05
Poverty*IMD	0.99		0.99	1.00	0.99	*	0.99	1.00	0.99		0.96	1.02
Poverty*NO	1.00		0.98	1.02	0.99		0.96	1.03	1.01		0.96	1.06
IMD*NO	1.00		1.00	1.00	1.00		1.00	1.00	1.00		1.00	1.00
Poverty*IMD*NO	1.00		1.00	1.00	1.00		1.00	1.00	1.00	**	0.99	1.00
	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.001	0.0004	0.001	0.02	0.001	0.0003	0.002	0.02	0.13	0.02	0.33	3.84
Level 2: Individual	4.13	3.77	4.52	55.67	4.14	3.69	4.65	55.70	4.35	3.31	6.03	56.95
Level 1: Wave				44.31				44.28				39.21

Table D.15 Asthma interaction multilevel models – O_3 only

ASTHMA	All England OR		95% CI		Excluding London OR		95% CI		London only OR		95% CI	
Child is female	0.45	***	0.37	0.56	0.46	***	0.35	0.59	0.42	**	0.21	0.76
Ethnicity												
White	REF				REF				REF			
Mixed	1.73	*	1.04	2.64	2.18	*	1.12	4.03	1.95		0.58	5.18
Indian	0.94		0.48	1.61	1.18		0.54	2.34	1.06		0.32	2.64
Pakistani & Bangladeshi	0.74		0.46	1.10	0.82		0.51	1.24	1.01		0.29	2.55
Black	0.75		0.42	1.19	0.78		0.30	1.73	1.54		0.66	3.05
Other	0.86		0.37	1.67	1.38		0.32	3.86	0.91		0.23	2.49
Child is obese	1.68	**	1.21	2.22	1.85	***	1.35	2.55	1.08		0.45	2.09
Mother is employed	1.21	*	1.00	1.44	1.18	*	0.98	1.40	1.44		0.92	2.14
Mother has asthma	8.96	***	6.82	11.78	7.91	***	5.58	10.70	29.61	***	11.61	61.00
Mother smokes	1.30	**	1.04	1.61	1.31	**	1.07	1.61	1.42		0.71	2.55
Lives in urban area	1.45	**	1.07	1.98	1.65	***	1.23	2.29	1.06		0.14	3.07
Lives below the poverty line	1.32	**	1.09	1.59	1.29	**	1.05	1.56	1.96	*	1.08	3.22
IMD (level of deprivation)	1.01	***	1.00	1.02	1.02	***	1.01	1.03	0.97	*	0.94	1.00
O_3 (level of pollution)	1.01		0.99	1.02	1.02	***	1.01	1.05	0.94		0.82	1.09
Poverty*IMD	1.01		1.00	1.02	1.00		0.99	1.01	1.03		0.98	1.08
Poverty*O_3	1.00		0.95	1.05	0.97		0.91	1.03	1.12		0.92	1.36
IMD*O_3	1.00		1.00	1.00	1.00		1.00	1.00	1.00		0.99	1.01
Poverty*IMD*O_3	1.00		1.00	1.00	1.00		1.00	1.01	1.03	***	1.01	1.04

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.002	0.001	0.005	0.49	0.001	0.0003	0.002	0.03	0.02	0.003	0.07	0.71
Level 2: Individual	18.19	16.49	20.03	83.79	18.24	16.38	20.11	84.72	23.15	17.30	30.02	87.56
Level 1: Wave				15.72				15.25				11.73

Table D.16 Wheeze interaction multilevel models – O_3 only

WHEEZE	All England OR		All England 95% CI		Excluding London OR		Excluding London 95% CI		London only OR		London only 95% CI	
Child is female	0.65	***	0.58	0.72	0.65	***	0.57	0.74	0.61	**	0.45	0.80
Ethnicity												
White	REF				REF				REF			
Mixed	1.10		0.79	1.43	1.11		0.76	1.58	1.38		0.76	2.28
Indian	1.06		0.77	1.44	0.96		0.60	1.46	1.45		0.84	2.33
Pakistani & Bangladeshi	0.88		0.69	1.11	0.91		0.71	1.13	0.79		0.42	1.39
Black	0.81		0.59	1.08	0.62	*	0.35	1.00	1.30		0.83	1.92
Other	0.87		0.53	1.31	0.96		0.43	1.85	1.07		0.53	1.85
Child is obese	1.43	***	1.17	1.72	1.43	***	1.14	1.75	1.58	*	0.98	2.43
Mother is employed	0.88	*	0.79	0.99	0.89	*	0.79	1.00	0.76	*	0.57	1.04
Mother has asthma	2.68	***	2.27	3.09	2.60	***	2.21	3.09	3.25	***	2.15	4.67
Mother smokes	1.24	***	1.11	1.40	1.19	**	1.03	1.35	1.76	***	1.21	2.37
Lives in urban area	1.28	***	1.08	1.51	1.30	**	1.06	1.58	4.02	*	0.72	16.04
Lives below the poverty line	1.05		0.93	1.20	1.05		0.90	1.20	1.14		0.80	1.57
IMD (level of deprivation)	1.01	**	1.00	1.01	1.01	*	1.00	1.01	0.99		0.97	1.01
O_3 (level of pollution)	1.01		1.00	1.02	1.00		0.98	1.02	0.97		0.91	1.02
Poverty*IMD	1.00		0.99	1.00	0.99	*	0.99	1.00	0.99		0.96	1.02
Poverty*O_3	1.01		0.98	1.05	1.01		0.97	1.06	1.00		0.89	1.11
IMD*O_3	1.00		1.00	1.00	1.00		1.00	1.00	1.00		0.99	1.00
Poverty*IMD*O_3	1.00		1.00	1.00	1.00		1.00	1.00	1.01	**	1.00	1.02

	Mean	95% CI		VPC	Mean	95% CI		VPC	Mean	95% CI		VPC
Level 3: MSOA	0.01	0.001	0.01	0.17	0.001	0.0002	0.001	0.02	0.004	0.001	0.01	0.12
Level 2: Individual	4.07	3.74	4.48	55.32	4.11	3.68	4.67	55.56	4.24	3.32	5.26	56.30
Level 1: Wave				44.51				44.42				43.58

Appendix E: **Differences in IMD score and air pollution concentrations for England, England excluding London, and London only**

Table E.1 Differences in area level variables for the different geographies

	All England			Excluding London			London only		
	min.	mean	max.	min.	mean	max.	min.	mean	max.
IMD score	0.8	25.2	81.6	0.8	24.8	81.6	2.2	27.3	62.3
NO_2 conc.	1.9	21.9	60.7	1.9	19.2	60.7	12.1	35.7	56.2
PM_{10} conc.	12.0	19.9	26.6	12.0	19.3	26.0	17.9	22.8	26.6
$PM_{2.5}$ conc.	5.8	12.5	19.2	5.8	12.1	19.2	11.2	14.9	18.3
NO conc.	0.1	6.9	40.7	0.1	5.0	37.9	1.6	16.9	40.7
O_3 conc.	11.8	26.8	37.5	11.8	27.6	37.5	14.5	22.7	30.5

Bibliography

ACHAKULWISUT, P., BRAUER, M., HYSTAD, P. & ANENBERG, S. C. 2019. Global, national, and urban burdens of paediatric asthma incidence attributable to ambient NO2 pollution: estimates from global datasets. *The Lancet Planetary Health,* 3**,** e166-e178.

AL GHOBAIN, M. O., AL-HAJJAJ, M. S. & AL MOAMARY, M. S. 2012. Asthma prevalence among 16-to 18-year-old adolescents in Saudi Arabia using the ISAAC questionnaire. *BMC Public Health,* 12**,** 1-5.

ANDERSEN, Z. J., LOFT, S., KETZEL, M., STAGE, M., SCHEIKE, T., HERMANSEN, M. N. & BISGAARD, H. 2008. Ambient air pollution triggers wheezing symptoms in infants. *Thorax,* 63**,** 710-716.

ARATHIMOS, R., GRANELL, R., HENDERSON, J., RELTON, C. L. & TILLING, K. 2017. Sex discordance in asthma and wheeze prevalence in two longitudinal cohorts. *PloS one,* 12**,** e0176293.

ASHER, M. & WEILAND, S. 1998. The International Study of Asthma and Allergies in Childhood (ISAAC). ISAAC Steering Committee. *Clinical and experimental allergy: Journal of the British Society for Allergy and Clinical Immunology,* 28**,** 52-66; discussion 90.

ASSARI, S. & MOGHANI LANKARANI, M. 2018. Poverty Status and Childhood Asthma in White and Black Families: National Survey of Children's Health. *Healthcare,* 6**,** 62.

BACON, S. L., BOUCHARD, A., LOUCKS, E. B. & LAVOIE, K. L. 2009. Individual-level socioeconomic status is associated with worse asthma morbidity in patients with asthma. *Respiratory research,* 10**,** 1-8.

BASAGAÑA, X., SUNYER, J., KOGEVINAS, M., ZOCK, J. P., DURAN-TAULERIA, E., JARVIS, D., BURNEY, P. & ANTO, J. M. 2004. Socioeconomic status and asthma prevalence in young adults: The European community respiratory health survey. *American Journal of Epidemiology,* 160**,** 178-188.

BECK, A. F., SANDEL, M. T., RYAN, P. H. & KAHN, R. S. 2017. Mapping neighborhood health geomarkers to clinical care decisions to promote equity in child health. *Health Affairs,* 36**,** 999-1005.

BELL, M. L., EBISU, K. & BELANGER, K. 2007. Ambient air pollution and low birth weight in Connecticut and Massachusetts. *Environmental health perspectives,* 115**,** 1118.

BELL, M. L., ZANOBETTI, A. & DOMINICI, F. 2013. Evidence on vulnerability and susceptibility to health risks associated with short-term exposure to particulate matter: a systematic review and meta-analysis. *American journal of epidemiology,* 178**,** 865-876.

BEUTHER, D. A., WEISS, S. T. & SUTHERLAND, E. R. 2006. Obesity and asthma. *American journal of respiratory and critical care medicine,* 174**,** 112-119.

BIBI, H., SHOSEYOV, D., FEIGENBAUM, D., GENIS, M., FRIGER, M., PELED, R. & SHARFF, S. 2004. The relationship between asthma and obesity in children: is it real or a case of over diagnosis? *Journal of Asthma,* 41**,** 403-410.

BLANC, P. D., YEN, I. H., CHEN, H., KATZ, P. P., EARNEST, G., BALMES, J. R., TRUPIN, L., FRIEDLING, N., YELIN, E. H. & EISNER, M. D. 2006. Area-level socio-economic status and health status among adults with asthma and rhinitis. *European Respiratory Journal,* 27**,** 85-94.

BLOOM, C. I., SAGLANI, S., FEARY, J., JARVIS, D. & QUINT, J. K. 2019. Changing prevalence of current asthma and inhaled corticosteroid treatment in the UK: population-based cohort 2006–2016. *European Respiratory Journal,* 53.

BONINGARI, T. & SMIRNIOTIS, P. G. 2016. Impact of nitrogen oxides on the environment and human health: Mn-based materials for the NO_x abatement. *Current Opinion in Chemical Engineering,* 13**,** 133-141.

BOOGAARD, H., WALKER, K. & COHEN, A. J. 2019. Air pollution: the emergence of a major global health risk factor. *International health,* 11**,** 417-421.

BOWATTE, G., LODGE, C., LOWE, A. J., ERBAS, B., PERRET, J., ABRAMSON, M. J., MATHESON, M. & DHARMAGE, S. C. 2015. The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies. *Allergy,* 70**,** 245-256.

BRADSHAW, J. & HOLMES, J. 2010. Child poverty in the first five years of life. *Children of the 21st century: the first five years. The UK Millennium Cohort Series,* 2**,** 13-31.

BRAMAN, S. S. 2006. The global burden of asthma. *Chest,* 130**,** 4S-12S.

BRAUBACH, M., SAVELSBERG, J. & ORGANIZATION, W. H. 2009. Social inequalities and their influence on housing risk factors and health: a data report based on the WHO LARES database.

BRIGGS, D., ABELLAN, J. J. & FECHT, D. 2008. Environmental inequity in England: Small area associations between socio-economic status and environmental pollution. *Social Science and Medicine,* 67**,** 1612-1629.

BROOK, R. D., FRANKLIN, B., CASCIO, W., HONG, Y., HOWARD, G., LIPSETT, M., LUEPKER, R., MITTLEMAN, M., SAMET, J., SMITH JR, S. C. & TAGER, I. 2004. Air pollution and cardiovascular disease: A statement for healthcare professionals from the expert panel on population and prevention science of the American Heart Association. *Circulation,* 109**,** 2655-2671.

BROPHY, S., COOKSEY, R., GRAVENOR, M. B., MISTRY, R., THOMAS, N., LYONS, R. A. & WILLIAMS, R. 2009. Risk factors for childhood obesity at age 5: analysis of the millennium cohort study. *BMC public health,* 9**,** 1-7.

BROWNE, W. J. 2015. MCMC estimation in MLwiN. *Centre for Multilevel Modelling, University of Bristol*.

BRUNEKREEF, B. & HOLGATE, S. T. 2002. Air pollution and health. *Lancet,* 360**,** 1233-1242.

BUIS, M. L. 2010. Stata tip 87: Interpretation of interactions in nonlinear models. *The stata journal,* 10**,** 305-308.

BURKE, W., FESINMEYER, M., REED, K., HAMPSON, L. & CARLSTEN, C. 2003. Family history as a predictor of asthma risk. *American journal of preventive medicine,* 24**,** 160-169.

BURNETT, R. T., DEWANJI, A., DOMINICI, F., GOLDBERG, M. S., COHEN, A. & KREWSKI, D. 2003. On the relationship between time-series studies, dynamic population studies, and estimating loss of life due to short-term exposure to environmental risks. *Environmental health perspectives,* 111**,** 1170-1174.

BUSH, A. 2007. Diagnosis of asthma in children under five. *Primary Care Respiratory Journal,* 16**,** 7-15.

CAI, Y., ZIJLEMA, W. L., DOIRON, D., BLANGIARDO, M., BURTON, P. R., FORTIER, I., GAYE, A., GULLIVER, J., DE HOOGH, K. & HVEEM, K. 2016. Ambient air pollution, traffic noise and adult asthma prevalence: a BioSHaRE approach. *European Respiratory Journal***,** ERJ-02127-2015.

CAILLAUD, D., LEYNAERT, B., KEIRSBULCK, M. & NADIF, R. 2018. Indoor mould exposure, asthma and rhinitis: findings from systematic reviews and recent longitudinal studies. *European Respiratory Review,* 27.

CAKMAK, S., HEBBERN, C., CAKMAK, J. D. & VANOS, J. 2016. The modifying effect of socioeconomic status on the relationship between traffic, air pollution and respiratory health in elementary schoolchildren. *Journal of Environmental Management,* 177**,** 1-8.

CARSON, C., SACKER, A., KELLY, Y., REDSHAW, M., KURINCZUK, J. & QUIGLEY, M. 2013. Asthma in children born after infertility treatment: findings from the UK Millennium Cohort Study. *Human Reproduction,* 28**,** 471-479.

CAUDRI, D., WIJGA, A., SCHIPPER, C. M. A., HOEKSTRA, M., POSTMA, D. S., KOPPELMAN, G. H., BRUNEKREEF, B., SMIT, H. A. & DE JONGSTE, J. C. 2009. Predicting the long-term prognosis of children with symptoms suggestive of asthma at preschool age. *Journal of Allergy and Clinical Immunology,* 124**,** 903-910. e7.

CESARONI, G., FARCHI, S., DAVOLI, M., FORASTIERE, F. & PERUCCI, C. A. 2003. Individual and area-based indicators of socioeconomic status and childhood asthma. *European Respiratory Journal,* 22**,** 619-624.

CHANG, J., DELFINO, R., GILLEN, D., TJOA, T., NICKERSON, B. & COOPER, D. 2009. Repeated respiratory hospital encounters among children with asthma and residential proximity to traffic. *Occupational and environmental medicine,* 66**,** 90-98.

CHEN, B. & KAN, H. 2008. Air pollution and population health: a global challenge. *Environmental health and preventive medicine,* 13**,** 94-101.

CHEN, K., GLONEK, G., HANSEN, A., WILLIAMS, S., TUKE, J., SALTER, A. & BI, P. 2016. The effects of air pollution on asthma hospital admissions in Adelaide, South Australia, 2003–2013: time-series and case–crossover analyses. *Clinical & Experimental Allergy,* 46**,** 1416-1430.

CHEN, L., VILLENEUVE, P. J., ROWE, B. H., LIU, L. & STIEB, D. M. 2014. The Air Quality Health Index as a predictor of emergency department visits for ischemic stroke in Edmonton, Canada. *Journal of Exposure Science and Environmental Epidemiology,* 24**,** 358-364.

CHI, G. C., HAJAT, A., BIRD, C. E., CULLEN, M. R., GRIFFIN, B. A., MILLER, K. A., SHIH, R. A., STEFANICK, M. L., VEDAL, S., WHITSEL, E. A. & KAUFMAN, J. D. 2016. Individual and neighborhood socioeconomic status and the association between air pollution and cardiovascular disease. *Environmental Health Perspectives,* 124**,** 1840-1847.

CHRISTEN, P. & CHURCHES, T. Secure health data linkage and geocoding: Current approaches and research directions. National e-Health Privacy and Security Symposium, 2006. Brisbane.

CLOUGHERTY, J. E. 2010. A growing role for gender analysis in air pollution epidemiology. *Environmental health perspectives,* 118**,** 167-176.

CLOUGHERTY, J. E., LEVY, J. I., KUBZANSKY, L. D., RYAN, P. B., SUGLIA, S. F., CANNER, M. J. & WRIGHT, R. J. 2007. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. *Environmental Health Perspectives,* 115**,** 1140-1146.

CONNELLY, R. & PLATT, L. 2014. Cohort Profile: UK Millennium Cohort Study (MCS). *International Journal of Epidemiology,* 43**,** 1719-1725.

CROMAR, K. R., GHAZIPURA, M., GLADSON, L. A. & PERLMUTT, L. 2020. Evaluating the US Air Quality Index as a risk communication tool: Comparing associations of index values with respiratory morbidity among adults in California. *PloS one,* 15**,** e0242031.

CROUSE, D. L., ROSS, N. A. & GOLDBERG, M. S. 2009. Double burden of deprivation and high concentrations of ambient air pollution at the neighbourhood scale in Montreal, Canada. *Social Science and Medicine,* 69**,** 971-981.

CURTIS, L., REA, W., SMITH-WILLIS, P., FENYVES, E. & PAN, Y. 2006. Adverse health effects of outdoor air pollutants. *Environment international,* 32**,** 815-830.

DAVIDSON, R., ROBERTS, S. E., WOTTON, C. J. & GOLDACRE, M. J. 2010. Influence of maternal and perinatal factors on subsequent hospitalisation for asthma in children: evidence from the Oxford record linkage study. *BMC pulmonary medicine,* 10**,** 1-8.

DENTON, M., PRUS, S. & WALTERS, V. 2004. Gender differences in health: a Canadian study of the psychosocial, structural and behavioural determinants of health. *Social science & medicine,* 58**,** 2585-2600.

DHARMAGE, S. C., PERRET, J. L. & CUSTOVIC, A. 2019. Epidemiology of asthma in children and adults. *Frontiers in pediatrics,* 7**,** 246.

DI GENOVA, L., PENTA, L., BISCARINI, A., DI CARA, G. & ESPOSITO, S. 2018. Children with obesity and asthma: which are the best options for their management? *Nutrients,* 10**,** 1634.

DICK, S., FRIEND, A., DYNES, K., ALKANDARI, F., DOUST, E., COWIE, H., AYRES, J. G. & TURNER, S. W. 2014. A systematic review of associations between environmental exposures and development of asthma in children aged up to 9 years. *BMJ Open,* 4**,** e006554.

DOCKERY, D. W. & POPE III, C. A. 1994. Acute respiratory effects of particulate air pollution. *Annual Review of Public Health.*

DOHERTY, R. M., HEAL, M. R., WILKINSON, P., PATTENDEN, S., VIENO, M., ARMSTRONG, B., ATKINSON, R., CHALABI, Z., KOVATS, S. & MILOJEVIC, A. 2009. Current and future climate-and air pollution-mediated impacts on human health. *Environmental health,* 8**,** 1-8.

DRISCOLL, K. A., VOLKENING, L. K., HARO, H., OCEAN, G., WANG, Y., JACKSON, C. C., CLOUGHERTY, M., HALE, D. E., KLINGENSMITH, G. J. & LAFFEL, L. 2015. Are children with type 1 diabetes safe at school? Examining parent perceptions. *Pediatric Diabetes,* 16**,** 613-620.

ELLEN, I. G., MIJANOVICH, T. & DILLMAN, K.-N. 2001. Neighborhood effects on health: exploring the links and assessing the evidence. *Journal of urban affairs,* 23**,** 391-408.

ELO, I. T. 2009. Social class differentials in health and mortality: Patterns and explanations in comparative perspective. *Annual Review of Sociology.*

ERNST, P., DEMISSIE, K., JOSEPH, L., LOCHER, U. & BECKLAKE, M. R. 1995. Socioeconomic status and indicators of asthma in children. *American Journal of Respiratory and Critical Care Medicine,* 152**,** 570-575.

ESPOSITO, S., TENCONI, R., LELII, M., PRETI, V., NAZZARI, E., CONSOLO, S. & PATRIA, M. F. 2014. Possible molecular mechanisms linking air pollution and asthma in children. *BMC pulmonary medicine,* 14**,** 1-8.

ESTRADA, R. D. & OWNBY, D. R. 2017. Rural asthma: current understanding of prevalence, patterns, and interventions for children and adolescents. *Current allergy and asthma reports,* 17**,** 37.

FAIRBURN, J., SCHÜLE, S. A., DREGER, S., KARLA HILZ, L. & BOLTE, G. 2019. Social inequalities in exposure to ambient air pollution: a systematic review in the WHO European Region. *International journal of environmental research and public health,* 16**,** 3127.

FECHT, D., FISCHER, P., FORTUNATO, L., HOEK, G., DE HOOGH, K., MARRA, M., KRUIZE, H., VIENNEAU, D., BEELEN, R. & HANSELL, A. 2015. Associations between air pollution and socioeconomic characteristics, ethnicity and age profile of neighbourhoods in England and the Netherlands. *Environmental pollution,* 198**,** 201-210.

FINKELSTEIN, M. M., JERRETT, M., DELUCA, P., FINKELSTEIN, N., VERMA, D. K., CHAPMAN, K. & SEARS, M. R. 2003. Relation between income, air pollution and mortality: A cohort study. *CMAJ,* 169**,** 397-402.

FITZSIMONS, E. & PONGIGLIONE, B. 2017. Prevalence and Trends in Overweight and Obesity in Childhood and Adolescence Findings from the Millennium Cohort Study, with a Focus on Age 14. *Centre for Longitudinal Studies Working paper,* 16.

FLANIGAN, C., SHEIKH, A., DUNNGALVIN, A., BREW, B. K., ALMQVIST, C. & NWARU, B. I. 2018. Prenatal maternal psychosocial stress and offspring's asthma and allergic disease: a systematic review and meta-analysis. *Clinical & Experimental Allergy,* 48**,** 403-414.

FONT, A., GUISEPPIN, L., BLANGIARDO, M., GHERSI, V. & FULLER, G. W. 2019. A tale of two cities: is air pollution improving in Paris and London? *Environmental Pollution,* 249**,** 1-12.

FORASTIERE, F., STAFOGGIA, M., TASCO, C., PICCIOTTO, S., AGABITI, N., CESARONI, G. & PERUCCI, C. A. 2007. Socioeconomic status, particulate air pollution, and daily mortality: Differential exposure or differential susceptibility. *American Journal of Industrial Medicine,* 50**,** 208-216.

FORNO, E. & CELEDÓN, J. C. 2009. Asthma and ethnic minorities: socioeconomic status and beyond. *Current opinion in allergy and clinical immunology,* 9**,** 154.

FORNO, E. & CELEDÓN, J. C. 2017. The effect of obesity, weight gain, and weight loss on asthma inception and control. *Current opinion in allergy and clinical immunology,* 17**,** 123.

FORNO, E., LESCHER, R., STRUNK, R., WEISS, S., FUHLBRIGGE, A., CELEDÓN, J. C. & GROUP, C. A. M. P. R. 2011. Decreased response to inhaled steroids in overweight and obese asthmatic children. *Journal of Allergy and Clinical Immunology,* 127**,** 741-749.

FOVERSKOV, E. & HOLM, A. 2016. Socioeconomic inequality in health in the British household panel: Tests of the social causation, health selection and the indirect selection hypothesis using dynamic fixed effects panel models. *Social Science and Medicine,* 150**,** 172-183.

GAUDERMAN, W. J., VORA, H., MCCONNELL, R., BERHANE, K., GILLILAND, F., THOMAS, D., LURMANN, F., AVOL, E., KUNZLI, N. & JERRETT, M. 2007. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet,* 369**,** 571-577.

GIBSON, M., PETTICREW, M., BAMBRA, C., SOWDEN, A. J., WRIGHT, K. E. & WHITEHEAD, M. 2011. Housing and health inequalities: a synthesis of systematic reviews of interventions aimed at different pathways linking housing and health. *Health & place,* 17**,** 175-184.

GONZALEZ-BARCALA, F.-J., PERTEGA, S., SAMPEDRO, M., LASTRES, J. S., GONZALEZ, M. A. S. J., BAMONDE, L., GARNELO, L., CASTRO, T. P., VALDÉS-CUADRADO, L. & CARREIRA, J.-M.

2013. Impact of parental smoking on childhood asthma. *Jornal de pediatria,* 89**,** 294-299.

GOODMAN, A., WILKINSON, P., STAFFORD, M. & TONNE, C. 2011. Characterising socio-economic inequalities in exposure to air pollution: A comparison of socio-economic markers and scales of measurement. *Health and Place,* 17**,** 767-774.

GRAHAM, A. M., PRINGLE, K. J., ARNOLD, S. R., POPE, R. J., VIENO, M., BUTT, E. W., CONIBEAR, L., STIRLING, E. L. & MCQUAID, J. B. 2020. Impact of weather types on UK ambient particulate matter concentrations. *Atmospheric Environment: X,* 5**,** 100061.

GRAY, A. M. 1982. Inequalities in health. The Black Report: a summary and comment. *International Journal of Health Services,* 12**,** 349-380.

GRIFFITHS, L. J., LYONS, R. A., BANDYOPADHYAY, A., TINGAY, K. S., WALTON, S., CORTINA-BORJA, M., AKBARI, A., BEDFORD, H. & DEZATEUX, C. 2018. Childhood asthma prevalence: cross-sectional record linkage study comparing parent-reported wheeze with general practitioner-recorded asthma diagnoses from primary care electronic health records in Wales. *BMJ open respiratory research,* 5**,** e000260.

GRINESKI, S. E., STANISWALIS, J. G., PENG, Y. & ATKINSON-PALOMBO, C. 2010. Children's asthma hospitalizations and relative risk due to nitrogen dioxide (NO 2): effect modification by race, ethnicity, and insurance status. *Environmental research,* 110**,** 178-188.

GUNIER, R. B., HERTZ, A., VON BEHREN, J. & REYNOLDS, P. 2003. Traffic density in California: socioeconomic and ethnic differences among potentially exposed children. *Journal of Exposure Science & Environmental Epidemiology,* 13**,** 240-246.

GUO, Y., LI, S., TIAN, Z., PAN, X., ZHANG, J. & WILLIAMS, G. 2013. The burden of air pollution on years of life lost in Beijing, China, 2004-08: retrospective regression analysis of daily deaths. *Bmj,* 347.

GUPTA, R. P., MUKHERJEE, M., SHEIKH, A. & STRACHAN, D. P. 2018. Persistent variations in national asthma mortality, hospital admissions and prevalence by socioeconomic status and region in England. *Thorax,* 73**,** 706-712.

HAJAT, A., HSIA, C. & O'NEILL, M. S. 2015. Socioeconomic Disparities and Air Pollution Exposure: a Global Review. *Current environmental health reports,* 2**,** 440-450.

HANSELL, A., GHOSH, R. E., BLANGIARDO, M., PERKINS, C., VIENNEAU, D., GOFFE, K., BRIGGS, D. & GULLIVER, J. 2016. Historic air pollution exposure and long-term mortality risks in England and Wales: prospective longitudinal cohort study. *Thorax,* 71**,** 330-338.

HAWKINS, S. S., COLE, T. J. & LAW, C. 2008. Maternal employment and early childhood overweight: findings from the UK Millennium Cohort Study. *International journal of obesity,* 32**,** 30-38.

HAWLEY, C., WILSON, J., HICKSON, C., MILLS, S., EKEOCHA, S. & SAKR, M. 2013. Epidemiology of paediatric minor head injury: comparison of injury characteristics with Indices of Multiple Deprivation. *Injury,* 44**,** 1855-1861.

HEIKKILÄ, K., SACKER, A., KELLY, Y., RENFREW, M. J. & QUIGLEY, M. A. 2011. Breast feeding and child behaviour in the Millennium Cohort Study. *Archives of disease in childhood,* 96**,** 635-642.

HEINRICH, J. & WICHMANN, H.-E. 2004. Traffic related pollutants in Europe and their effect on allergic disease. *Current opinion in allergy and clinical immunology,* 4**,** 341-348.

HENDERSON, I. & QUENBY, S. 2021. Gestational hypertension and childhood atopy: a Millennium Cohort Study analysis. *European Journal of Pediatrics***,** 1-9.

HILL, T. D., JORGENSON, A. K., ORE, P., BALISTRERI, K. S. & CLARK, B. 2019. Air quality and life expectancy in the United States: An analysis of the moderating effect of income inequality. *SSM-population health,* 7**,** 100346.

HILLS, J. 2007. Ends and means: the future roles of social housing in England.

HINDMARSH, G., LLEWELLYN, G. & EMERSON, E. 2017. The social-emotional well-being of children of mothers with intellectual impairment: a population-based analysis. *Journal of Applied Research in Intellectual Disabilities,* 30**,** 469-481.

HISCOCK, R., BAULD, L., AMOS, A., FIDLER, J. A. & MUNAFÒ, M. 2012. Socioeconomic status and smoking: a review. *Annals of the New York Academy of Sciences,* 1248**,** 107-123.

HOOD, E. 2005. Dwelling disparities: how poor housing leads to poor health. National Institue of Environmental Health Sciences.

HUBBARD, A. E., AHERN, J., FLEISCHER, N. L., VAN DER LAAN, M., SATARIANO, S. A., JEWELL, N., BRUCKNER, T. & SATARIANO, W. A. 2010. To GEE or not to GEE: comparing population average and mixed models for estimating the associations between neighborhood risk factors and health. *Epidemiology***,** 467-474.

HUGHES, R. A. 2012. *'Governing in hard times': the Heath government and civil emergencies– the 1972 and the 1974 miners' strikes.*

HULIN, M., CAILLAUD, D. & ANNESI-MAESANO, I. 2010. Indoor air pollution and childhood asthma: variations between urban and rural areas. *Indoor air,* 20**,** 502-514.

ISLAM, M. S., HUQ, S., AHMED, S., ROY, S., SCHWARZE, J., SHEIKH, A., SAHA, S. K., CUNNINGHAM, S., NAIR, H. & COLLABORATION, R. 2021. Operational definitions of paediatric asthma used in epidemiological studies: A systematic review. *Journal of Global Health,* 11.

JACKSON, S. E., SMITH, C., CHEESEMAN, H., WEST, R. & BROWN, J. 2019. Finding smoking hot-spots: a cross-sectional survey of smoking patterns by housing tenure in England. *Addiction,* 114**,** 889-895.

JAYAWEERA, H. & QUIGLEY, M. A. 2010. Health status, health behaviour and healthcare use among migrants in the UK: evidence from mothers in the Millennium Cohort Study. *Social science & medicine,* 71**,** 1002-1010.

JEPHCOTE, C. & CHEN, H. 2012. Environmental injustices of children's exposure to air pollution from road-transport within the model British multicultural city of Leicester: 2000-09. *Science of the Total Environment,* 414**,** 140-151.

JEPHCOTE, C., ROPKINS, K. & CHEN, H. 2014. The effect of socio-environmental mechanisms on deteriorating respiratory health across urban communities during childhood. *Applied Geography,* 51**,** 35-47.

JERRETT, M., BURNETT, R. T., BROOK, J., KANAROGLOU, P., GIOVIS, C., FINKELSTEIN, N. & HUTCHISON, B. 2004. Do socioeconomic characteristics modify the short term association between air pollution and mortality? Evidence from a zonal time series in Hamilton, Canada. *Journal of Epidemiology and Community Health,* 58**,** 31-40.

JERRETT, M., BURNETT, R. T., KANAROGLOU, P., EYLES, J., FINKELSTEIN, N., GIOVIS, C. & BROOK, J. R. 2001. A GIS - Environmental justice analysis of particulate air pollution in Hamilton, Canada. *Environment and Planning A,* 33**,** 955-973.

JERRETT, M., BURNETT, R. T., POPE III, C. A., ITO, K., THURSTON, G., KREWSKI, D., SHI, Y., CALLE, E. & THUN, M. 2009. Long-term ozone exposure and mortality. *New England Journal of Medicine,* 360**,** 1085-1095.

JERRETT, M., EYLES, J., COLE, D. & READER, S. 1997. Environmental equity in Canada: An empirical investigation into the income distribution of pollution in Ontario. *Environment and Planning A,* 29**,** 1777-1800.

JERRETT, M., MCCONNELL, R., WOLCH, J., CHANG, R., LAM, C., DUNTON, G., GILLILAND, F., LURMANN, F., ISLAM, T. & BERHANE, K. 2014. Traffic-related air pollution and obesity formation in children: a longitudinal, multilevel analysis. *Environmental Health,* 13**,** 1-9.

JIAO, K., XU, M. & LIU, M. 2018. Health status and air pollution related socioeconomic concerns in urban China. *International journal for equity in health,* 17**,** 18.

JORDAN, H., RODERICK, P. & MARTIN, D. 2004. The Index of Multiple Deprivation 2000 and accessibility effects on health. *Journal of Epidemiology & Community Health,* 58**,** 250-257.

JORGENSON, A. K., HILL, T. D., CLARK, B., THOMBS, R. P., ORE, P., BALISTRERI, K. S. & GIVENS, J. E. 2020. Power, proximity, and physiology: does income inequality and racial composition amplify the impacts of air pollution on life expectancy in the United States? *Environmental Research Letters,* 15**,** 024013.

JORGENSON, A. K., THOMBS, R. P., CLARK, B., GIVENS, J. E., HILL, T. D., HUANG, X., KELLY, O. M. & FITZGERALD, J. B. 2021. Inequality amplifies the negative association between life expectancy and air pollution: A cross-national longitudinal study. *Science of The Total Environment,* 758**,** 143705.

KAMPA, M. & CASTANAS, E. 2008. Human health effects of air pollution. *Environmental Pollution,* 151**,** 362-367.

KELLY, F. J. & FUSSELL, J. C. 2012. Size, source and chemical composition as determinants of toxicity attributable to ambient particulate matter. *Atmospheric environment,* 60**,** 504-526.

KELLY, L., BARRETT, P., MCCARTHY, F. & KHASHAN, A. 2019. OP99 Hypertensive disorders in pregnancy and childhood diagnosis of asthma. BMJ Publishing Group Ltd.

KOENIG, J. Q. 1999. Air pollution and asthma. *Journal of allergy and clinical immunology,* 104**,** 717-722.

KONTOPANTELIS, E., MAMAS, M. A., VAN MARWIJK, H., RYAN, A. M., BUCHAN, I. E., ASHCROFT, D. M. & DORAN, T. 2018. Geographical epidemiology of health and overall deprivation in England, its changes and persistence from 2004 to 2015: a longitudinal spatial population study. *J Epidemiol Community Health,* 72**,** 140-147.

KRAVITZ-WIRTZ, N., TEIXEIRA, S., HAJAT, A., WOO, B., CROWDER, K. & TAKEUCHI, D. 2018. Early-life air pollution exposure, neighborhood poverty, and childhood asthma in the United States, 1990–2014. *International journal of environmental research and public health,* 15**,** 1114.

KRÖGER, H., PAKPAHAN, E. & HOFFMANN, R. 2015. What causes health inequality? A systematic review on the relative importance of social causation and health selection. *The European Journal of Public Health,* 25**,** 951-960.

LAD, M. 2011. The English Indices of Deprivation 2010. *Neighbourhoods Statistical Release***,** 21.

LANDRIGAN, P. J. 2017. Air pollution and health. *The Lancet Public Health,* 2**,** e4-e5.

LANG, D. M., SHERMAN, M. S. & POLANSKY, M. 1997. Guidelines and realities of asthma management: The Philadelphia story. *Archives of Internal Medicine,* 157**,** 1193-1200.

LAURENT, O., PEDRONO, G., SEGALA, C., FILLEUL, L., HAVARD, S., DEGUEN, S., SCHILLINGER, C., RIVIÈRE, E. & BARD, D. 2008. Air pollution, asthma attacks, and socioeconomic deprivation: a small-area case-crossover study. *American journal of epidemiology,* 168**,** 58-65.

LAVIGNE, E., VILLENEUVE, P. J. & CAKMAK, S. 2012. Air pollution and emergency department visits for asthma in Windsor, Canada. *Canadian Journal of Public Health,* 103**,** 4-8.

LEBOLD, K. M., JACOBY, D. B. & DRAKE, M. G. 2020. Inflammatory mechanisms linking maternal and childhood asthma. *Journal of Leukocyte Biology*.

LECKIE, G. & CHARLTON, C. 2013. Runmlwin-a program to Run the MLwiN multilevel modelling software from within stata. *Journal of Statistical Software,* 52**,** 1-40.

LEE, J.-T., SON, J.-Y., KIM, H. & KIM, S.-Y. 2006. Effect of air pollution on asthma-related hospital admissions for children by socioeconomic status associated with area of residence. *Archives of environmental & occupational health,* 61**,** 123-130.

LEE, S.-I. 2010. Prevalence of childhood asthma in Korea: international study of asthma and allergies in childhood. *Allergy, asthma & immunology research,* 2**,** 61-64.

LEWIS, K. M., PIKHART, H. & MORRISON, J. 2018. Does adiposity mediate the relationship between socioeconomic position and non-allergic asthma in childhood? *J Epidemiol Community Health,* 72**,** 390-396.

LIM, C. C., HAYES, R. B., AHN, J., SHAO, Y., SILVERMAN, D. T., JONES, R. R., GARCIA, C., BELL, M. L. & THURSTON, G. D. 2019. Long-term exposure to ozone and cause-specific mortality risk in the United States. *American journal of respiratory and critical care medicine,* 200**,** 1022-1031.

LIM, R. H. & KOBZIK, L. 2009. Maternal transmission of asthma risk. *American Journal of Reproductive Immunology,* 61**,** 1-10.

LIN, C., HEAL, M. R., VIENO, M., MACKENZIE, I. A., ARMSTRONG, B. G., BUTLAND, B. K., MILOJEVIC, A., CHALABI, Z., ATKINSON, R. W. & STEVENSON, D. S. 2017. Spatiotemporal evaluation of EMEP4UK-WRF v4. 3 atmospheric chemistry transport simulations of health-related metrics for NO2, O3, PM10, and PM2. 5 for 2001–2010. *Geoscientific Model Development,* 10**,** 1767-1787.

LITONJUA, A. A., CAREY, V. J., BURGE, H. A., WEISS, S. T. & GOLD, D. R. 1998. Parental history and the risk for childhood asthma: does mother confer more risk than father? *American journal of respiratory and critical care medicine,* 158**,** 176-181.

LONGFORD, N. T., PITTAU, M. G., ZELLI, R. & MASSARI, R. 2012. Poverty and inequality in European regions. *Journal of Applied Statistics,* 39**,** 1557-1576.

LUEKE, L. 2011. Devouring childhood obesity by helping children help themselves. *Journal of Legal Medicine,* 32**,** 205-220.

MACINTYRE, S., ELLAWAY, A., DER, G., FORD, G. & HUNT, K. 1998. Do housing tenure and car access predict health because they are simply markers of income or self esteem? A Scottish study. *Journal of Epidemiology & Community Health,* 52**,** 657-664.

MARMOT, M., ALLEN, J., GOLDBLATT, P., BOYCE, T., MCNEISH, D. & GRADY, M. 2010. Fair society, healthy lives. *The Marmot Review,* 14.

MARTINS, P., VALENTE, J., PAPOILA, A. L., CAIRES, I., ARAÚJO-MARTINS, J., MATA, P., LOPES, M., TORRES, S., ROSADO-PINTO, J. & BORREGO, C. 2012. Airways changes related to air pollution exposure in wheezing children. *European Respiratory Journal,* 39**,** 246-253.

MATSUI, E. C. 2014. Environmental exposures and asthma morbidity in children living in urban neighborhoods. *Allergy,* 69**,** 553-558.

MCCONNELL, R., ISLAM, T., SHANKARDASS, K., JERRETT, M., LURMANN, F., GILLILAND, F., GAUDERMAN, J., AVOL, E., KÜNZLI, N. & YAO, L. 2010. Childhood incident asthma and traffic-related air pollution at home and school. *Environmental health perspectives,* 118**,** 1021-1026.

MCDONOUGH, P. & WALTERS, V. 2001. Gender and health: reassessing patterns and explanations. *Social science & medicine,* 52**,** 547-559.

MCLAREN, D., COTTRAY, O., TAYLOR, M., PIPES, S. & BULLOCK, S. 1999. The geographic relation between household income and polluting factories. *Friends of the Earth: London*.

MCLENNAN, D., BARNES, H., NOBLE, M., DAVIES, J., GARRATT, E. & DIBBEN, C. 2011. The English Indices of Deprivation 2010. London: Department for Communities and Local Government.

MICHNA, P. & WOODS, M. 2013. RNetCDF–A package for reading and writing NetCDF datasets. *The R Journal,* 5**,** 29-36.

MILOJEVIC, A., NIEDZWIEDZ, C. L., PEARCE, J., MILNER, J., MACKENZIE, I. A., DOHERTY, R. M. & WILKINSON, P. 2017. Socioeconomic and urban-rural differentials in exposure to air pollution and mortality burden in England. *Environmental Health,* 16**,** 1-10.

MINISTRY OF HOUSING, C. & GOVERNMENT, L. 2011. The English Indices of Deprivation 2010.

MIRI, M., AVAL, H. E., EHRAMPOUSH, M. H., MOHAMMADI, A., TOOLABI, A., NIKONAHAD, A., DERAKHSHAN, Z. & ABDOLLAHNEJAD, A. 2017. Human health impact assessment of exposure to particulate matter: an AirQ software modeling. *Environmental Science and Pollution Research,* 24**,** 16513-16519.

MIRZAKHANI, H., CAREY, V. J., ZEIGER, R., BACHARIER, L. B., O'CONNOR, G. T., SCHATZ, M. X., LARANJO, N., WEISS, S. T. & LITONJUA, A. A. 2019. Impact of parental asthma, prenatal maternal asthma control, and vitamin D status on risk of asthma and recurrent wheeze in 3-year-old children. *Clinical & Experimental Allergy,* 49**,** 419-429.

MITCHELL, G. & DORLING, D. 2003. An environmental justice analysis of British air quality. *Environment and planning A,* 35**,** 909-929.

MITCHELL, R. & POPHAM, F. 2007. Greenspace, urbanity and health: relationships in England. *Journal of Epidemiology & Community Health,* 61**,** 681-683.

MÖLTER, A., AGIUS, R. M., DE VOCHT, F., LINDLEY, S., GERRARD, W., LOWE, L., BELGRAVE, D., CUSTOVIC, A. & SIMPSON, A. 2013. Long-term exposure to PM10 and NO2 in association with lung volume and airway resistance in the MAAS birth cohort. *Environmental health perspectives,* 121**,** 1232-1238.

MORAL, L., VIZMANOS, G., TORRES-BORREGO, J., PRAENA-CRESPO, M., TORTAJADA-GIRBÉS, M., PELLEGRINI, F. & ASENSIO, Ó. 2019. Asthma diagnosis in infants and preschool children: a systematic review of clinical guidelines. *Allergologia et immunopathologia,* 47**,** 107-121.

MORELLI, X., RIEUX, C., CYRYS, J., FORSBERG, B. & SLAMA, R. 2016. Air pollution, health and social deprivation: A fine-scale risk assessment. *Environmental Research,* 147**,** 59-70.

MORRILL, M. S. 2011. The effects of maternal employment on the health of school-age children. *Journal of health economics,* 30**,** 240-257.

MORRIS, S., SUTTON, M. & GRAVELLE, H. 2005. Inequity and inequality in the use of health care in England: an empirical investigation. *Social science & medicine,* 60**,** 1251-1266.

MORRISSEY, K., CHUNG, I., MORSE, A., PARTHASARATH, S., ROEBUCK, M. M., TAN, M. P., WOOD, A., WONG, P.-F. & FROSTICK, S. P. 2021a. The effects of air quality on hospital admissions for chronic respiratory diseases in Petaling Jaya, Malaysia, 2013–2015. *Atmosphere,* 12**,** 1060.

MORRISSEY, K., ESPUNY, F. & WILLIAMSON, P. 2016. A multinomial model for comorbidity in E ngland of long-standing cardiovascular disease, diabetes and obesity. *Health & social care in the community,* 24**,** 717-727.

MORRISSEY, K., SPOONER, F., SALTER, J. & SHADDICK, G. 2021b. Area level deprivation and monthly COVID-19 cases: The impact of government policy in England. *Social Science & Medicine,* 289**,** 114413.

NAEEM, A. & SILVEYRA, P. 2019. Sex differences in paediatric and adult asthma. *European Medical Journal (Chelmsford, England),* 4**,** 27.

NAEI. 2018. *National Atmospheric Emissions Inventory, UK emissions data selector* [Online]. Department for Environment, Food and Rural Affairs. Available: naei.beis.gov.uk [Accessed 15 January 2021].

NETUVELI, G., HURWITZ, B., LEVY, M., FLETCHER, M., BARNES, G., DURHAM, S. R. & SHEIKH, A. 2005. Ethnic variations in UK asthma frequency, morbidity, and health-service use: a systematic review and meta-analysis. *The Lancet,* 365**,** 312-317.

NORBÄCK, D., LU, C., ZHANG, Y., LI, B., ZHAO, Z., HUANG, C., ZHANG, X., QIAN, H., SUN, Y. & WANG, J. 2019. Sources of indoor particulate matter (PM) and outdoor air pollution in China in relation to asthma, wheeze, rhinitis and eczema among pre-school children: Synergistic effects between antibiotics use and PM10 and second hand smoke. *Environment international,* 125**,** 252-260.

NORDLING, E., BERGLIND, N., MELÉN, E., EMENIUS, G., HALLBERG, J., NYBERG, F., PERSHAGEN, G., SVARTENGREN, M., WICKMAN, M. & BELLANDER, T. 2008. Traffic-related air pollution and childhood respiratory symptoms, function and allergies. *Epidemiology,* 19**,** 401-408.

NORTHRIDGE, J., RAMIREZ, O. F., STINGONE, J. A. & CLAUDIO, L. 2010. The role of housing type and housing quality in urban children with asthma. *Journal of Urban Health,* 87**,** 211-224.

O'NEILL, M. S., JERRETT, M., KAWACHI, I., LEVY, J. I., COHEN, A. J., GOUVEIA, N., WILKINSON, P., FLETCHER, T., CIFUENTES, L., SCHWARTZ, J., BATESON, T. F., CANN, C., DOCKERY, D., GOLD, D., LADEN, F., LONDON, S., LOOMIS, D., SPEIZER, F., VAN DEN EEDEN, S. & ZANOBETTI, A. 2003. Health, wealth, and air pollution: Advancing theory and methods. *Environmental Health Perspectives,* 111**,** 1861-1870.

OCAMPO, J., GAVIRIA, R. & SÁNCHEZ, J. 2017. Prevalence of asthma in Latin America. Critical look at ISAAC and other studies. *Revista alergia Mexico,* 64**,** 188-197.

OFTEDAL, B., NYSTAD, W., BRUNEKREEF, B. & NAFSTAD, P. 2009. Long-term traffic-related exposures and asthma onset in schoolchildren in Oslo, Norway. *Environmental health perspectives,* 117**,** 839-844.

OSMAN, M. 2003. Therapeutic implications of sex differences in asthma and atopy. *Archives of disease in childhood,* 88**,** 587-590.

OSTRO, B., LIPSETT, M., MANN, J., BRAXTON-OWENS, H. & WHITE, M. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology,* 12**,** 200-208.

PALMIERI, M., LONGOBARDI, G., NAPOLITANO, G. & SIMONETTI, D. 1990. Parental smoking and asthma in childhood. *European journal of pediatrics,* 149**,** 738-740.

PANICO, L., BARTLEY, M., MARMOT, M., NAZROO, J. Y., SACKER, A. & KELLY, Y. J. 2007. Ethnic variation in childhood asthma and wheezing illnesses: findings from the Millennium Cohort Study. *International journal of epidemiology,* 36**,** 1093-1102.

PANICO, L., STUART, B., BARTLEY, M. & KELLY, Y. 2014. Asthma trajectories in early childhood: identifying modifiable factors. *PLoS One,* 9**,** e111922.

PAVORD, I. D., BEASLEY, R., AGUSTI, A., ANDERSON, G. P., BEL, E., BRUSSELLE, G., CULLINAN, P., CUSTOVIC, A., DUCHARME, F. M. & FAHY, J. V. 2018. After asthma: redefining airways diseases. *The Lancet,* 391**,** 350-400.

PEDERSEN, S. E., HURD, S. S., LEMANSKE JR, R. F., BECKER, A., ZAR, H. J., SLY, P. D., SOTO-QUIROZ, M., WONG, G. & BATEMAN, E. D. 2011. Global strategy for the diagnosis and management of asthma in children 5 years and younger. *Pediatric pulmonology,* 46**,** 1-17.

PERLIN, S. A., SEXTON, K. & WONG, D. W. 1999. An examination of race and poverty for populations living near industrial sources of air pollution. *Journal of Exposure Analysis & Environmental Epidemiology,* 9.

PEVALIN, D. J., TAYLOR, M. P. & TODD, J. 2008. The dynamics of unhealthy housing in the UK: A panel data analysis. *Housing studies,* 23**,** 679-695.

PIKE, K. C., GRIFFITHS, L. J., DEZATEUX, C. & PEARCE, A. 2019. Physical activity among children with asthma: cross-sectional analysis in the UK millennium cohort. *Pediatric pulmonology,* 54**,** 962-969.

PISANO, G. P. 1996. Learning-before-doing in the development of new process technology. *Research Policy,* 25**,** 1097-1119.

PLEWIS, I., CALDERWOOD, L., HAWKES, D., HUGHES, G. & JOSHI, H. 2007. Millennium Cohort Study: technical report on sampling. *London: Centre for Longitudinal Study, Institute of Education*.

POLIVKA, B. J. 2018. The Great London Smog of 1952. *AJN The American Journal of Nursing,* 118**,** 57-61.

POPE III, C. A. 2007. Mortality effects of longer term exposures to fine particulate air pollution: review of recent epidemiological evidence. *Inhalation toxicology,* 19**,** 33-38.

POPE III, C. A., BURNETT, R. T., THUN, M. J., CALLE, E. E., KREWSKI, D., ITO, K. & THURSTON, G. D. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Jama,* 287**,** 1132-1141.

PRATLEY, P. 2016. Associations between quantitative measures of women's empowerment and access to care and health status for mothers and their children: a systematic review of evidence from the developing world. *Social Science & Medicine,* 169**,** 119-131.

PRATT, G. C., VADALI, M. L., KVALE, D. L. & ELLICKSON, K. M. 2015. Traffic, air pollution, minority and socio-economic status: Addressing inequities in exposure and risk. *International Journal of Environmental Research and Public Health,* 12**,** 5355-5372.

PUGMIRE, J., VASQUEZ, M. M., ZHOU, M., SHERRILL, D. L., HALONEN, M., MARTINEZ, F. D. & GUERRA, S. 2014. Exposure to parental smoking in childhood is associated with persistence of respiratory symptoms into young adult life. *Journal of Allergy and Clinical Immunology,* 134**,** 962-965. e4.

QUIGLEY, M. A., CARSON, C. & KELLY, Y. 2018. Breastfeeding and childhood wheeze: Age-specific analyses and longitudinal wheezing phenotypes as complementary approaches to the analysis of cohort data. *American journal of epidemiology,* 187**,** 1651-1661.

RABINOVITCH, N., SILVEIRA, L., GELFAND, E. W. & STRAND, M. 2011. The response of children with asthma to ambient particulate is modified by tobacco smoke exposure. *American Journal of Respiratory and Critical Care Medicine,* 184**,** 1350-1357.

RASBASH, J., STEELE, F., BROWNE, W., GOLDSTEIN, H. & CHARLTON, C. 2013. A user's guide to MLwiN. *Centre for Multilevel Modelling: University of Bristol*.

REDDEL, H. K., BATEMAN, E. D., BECKER, A., BOULET, L.-P., CRUZ, A. A., DRAZEN, J. M., HAAHTELA, T., HURD, S. S., INOUE, H. & DE JONGSTE, J. C. 2015. A summary of the new GINA strategy: a roadmap to asthma control. *European Respiratory Journal,* 46**,** 622-639.

RICHARDSON, E. A., PEARCE, J. & KINGHAM, S. 2011. Is particulate air pollution associated with health and health inequalities in New Zealand? *Health and Place,* 17**,** 1137-1143.

RICHARDSON, E. A., PEARCE, J., TUNSTALL, H., MITCHELL, R. & SHORTT, N. K. 2013. Particulate air pollution and health inequalities: A Europe-wide ecological analysis. *International Journal of Health Geographics,* 12.

RODRIGUEZ-VILLAMIZAR, L. A., BERNEY, C., VILLA-ROEL, C., OSPINA, M. B., OSORNIO-VARGAS, A. & ROWE, B. H. 2016. The role of socioeconomic position as an effect-modifier of the association between outdoor air pollution and children's asthma exacerbations: An equity-focused systematic review. *Reviews on Environmental Health,* 31**,** 297-309.

RONA, R. J. 2000. Asthma and poverty. *Thorax,* 55**,** 239-244.

ROSENLUND, M., FORASTIERE, F., PORTA, D., DE SARIO, M., BADALONI, C. & PERUCCI, C. A. 2009. Traffic-related air pollution in relation to respiratory symptoms, allergic sensitisation and lung function in schoolchildren. *Thorax,* 64**,** 573-580.

ROTHMAN, K. J. 2008. BMI-related errors in the measurement of obesity. *International journal of obesity,* 32**,** S56-S59.

RUSSELL, G., RODGERS, L. R., UKOUMUNNE, O. C. & FORD, T. 2014. Prevalence of parent-reported ASD and ADHD in the UK: findings from the Millennium Cohort Study. *Journal of autism and developmental disorders,* 44**,** 31-40.

SALONEN, H., SALTHAMMER, T. & MORAWSKA, L. 2019. Human exposure to NO2 in school and office indoor environments. *Environment international,* 130**,** 104887.

SCHIKOWSKI, T., SUGIRI, D., REIMANN, V., PESCH, B., RANFT, U. & KRÄMER, U. 2008. Contribution of smoking and air pollution exposure in urban areas to social differences in respiratory health. *BMC Public Health,* 8.

SCHOLTENS, S., WIJGA, A. H., SEIDELL, J. C., BRUNEKREEF, B., DE JONGSTE, J. C., GEHRING, U., POSTMA, D. S., KERKHOF, M. & SMIT, H. A. 2009. Overweight and changes in weight status during childhood in relation to asthma symptoms at 8 years of age. *Journal of Allergy and Clinical Immunology,* 123**,** 1312-1318. e2.

SCHWARTZ, J. 2004. Air pollution and children's health. *Pediatrics,* 113**,** 1037-1043.

SCHWARTZ, J., SLATER, D., LARSON, T. V., PIERSON, W. E. & KOENIG, J. O. 1993. Particulate air pollution and hospital emergency room. *Amer. Rev. Respiratory Dis,* 147**,** 826-831.

SEATON, A. 1996. Particles in the air: the enigma of urban air pollution. *Journal of the Royal Society of Medicine,* 89**,** 604.

SETIA, M. S. 2016. Methodology series module 3: Cross-sectional studies. *Indian journal of dermatology,* 61**,** 261.

SHANKARDASS, K., MCCONNELL, R., JERRETT, M., MILAM, J., RICHARDSON, J. & BERHANE, K. 2009. Parental stress increases the effect of traffic-related air pollution on childhood asthma incidence. *Proceedings of the National Academy of Sciences,* 106**,** 12406-12411.

SHARPE, R., MACHRAY, K., FLEMING, L., TAYLOR, T., HENLEY, W., CHENORE, T., HUTCHCROFT, I., TAYLOR, J., HEAVISIDE, C. & WHEELER, B. 2019. Household energy efficiency and health: Area-level analysis of hospital admissions in England. *Environment international,* 133**,** 105164.

SHEIKH, A., STEINER, M. F., CEZARD, G., BANSAL, N., FISCHBACHER, C., SIMPSON, C. R., DOUGLAS, A. & BHOPAL, R. 2016. Ethnic variations in asthma hospital admission, readmission and death: a retrospective, national cohort study of 4.62 million people in Scotland. *BMC medicine,* 14**,** 1-9.

SHEPPARD, L., BURNETT, R. T., SZPIRO, A. A., KIM, S.-Y., JERRETT, M., POPE, C. A. & BRUNEKREEF, B. 2012. Confounding and exposure measurement error in air pollution epidemiology. *Air Quality, Atmosphere & Health,* 5**,** 203-216.

SHORE, S. A. & JOHNSTON, R. A. 2006. Obesity and asthma. *Pharmacology & therapeutics,* 110**,** 83-102.

SIMPSON, D., BENEDICTOW, A., BERGE, H., BERGSTRÖM, R., EMBERSON, L. D., FAGERLI, H., FLECHARD, C. R., HAYMAN, G. D., GAUSS, M. & JONSON, J. E. 2012. The EMEP MSC-W chemical transport model-technical description.

SKALICKÁ, V., VAN LENTHE, F., BAMBRA, C., KROKSTAD, S. & MACKENBACH, J. 2009. Material, psychosocial, behavioural and biomedical factors in the explanation of relative socio-economic inequalities in mortality: evidence from the HUNT study. *International journal of epidemiology,* 38**,** 1272-1284.

SKOBELOFF, E. M., SPIVEY, W. H., CLAIR, S. S. S. & SCHOFFSTALL, J. M. 1992. The influence of age and sex on asthma admissions. *Jama,* 268**,** 3437-3440.

SORENSEN, N. 2002. Child Benefit Quarterly Statistics – November 2001. Department for Work and Pensions.

SPENCE, J. & STEPHENSON, C. 2007. The politics of the doorstep: Female survival strategies and the legacy of the miners' strike 1984–85. *Community, Work and Family,* 10**,** 309-327.

STAMATAKIS, E., WARDLE, J. & COLE, T. J. 2010. Childhood obesity and overweight prevalence trends in England: evidence for growing socioeconomic disparities. *International journal of obesity,* 34**,** 41-47.

STATACORP 2017. Stata Statistical Software: Release 14. College Station, TX: StataCorp LP.

STOCKFELT, L., ANDERSSON, E. M., MOLNÁR, P., ROSENGREN, A., WILHELMSEN, L., SALLSTEN, G. & BARREGARD, L. 2015. Long term effects of residential NOx exposure on total and cause-specific mortality and incidence of myocardial infarction in a Swedish cohort. *Environmental research,* 142**,** 197-206.

STRONKS, K., VAN DE MHEEN, H. D. & MACKENBACH, J. P. 1998. A higher prevalence of health problems in low income groups: does it reflect relative deprivation? *Journal of Epidemiology and Community Health,* 52**,** 548-557.

SZMARAGD, C., CLARKE, P. & STEELE, F. 2013. Subject specific and population average models for binary longitudinal data: a tutorial. *Longitudinal and life course studies,* 4**,** 147-165.

TAGER, I. B., BALMES, J., LURMANN, F., NGO, L., ALCORN, S. & KÜNZLI, N. 2005. Chronic exposure to ambient ozone and lung function in young adults. *Epidemiology,* 16**,** 751-759.

TAYLOR-ROBINSON, D. C., PEARCE, A., WHITEHEAD, M., SMYTH, R. & LAW, C. 2016. Social inequalities in wheezing in children: findings from the UK Millennium Cohort Study. *European Respiratory Journal,* 47**,** 818-828.

TIWARY, A. & WILLIAMS, I. 2018. *Air pollution: measurement, modelling and mitigation*, CRC Press.

TOBLER, W. R. 1970. A computer movie simulating urban growth in the Detroit region. *Economic geography,* 46**,** 234-240.

TURNER, M. C., JERRETT, M., POPE III, C. A., KREWSKI, D., GAPSTUR, S. M., DIVER, W. R., BECKERMAN, B. S., MARSHALL, J. D., SU, J. & CROUSE, D. L. 2016. Long-term ozone exposure and mortality in a large prospective study. *American journal of respiratory and critical care medicine,* 193**,** 1134-1142.

UNGAR, W. J., COPE, S. F., KOZYRSKYJ, A. & PATERSON, J. M. 2010. Socioeconomic factors and home allergen exposure in children with asthma. *Journal of Pediatric Health Care,* 24**,** 108-115.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2020. Millennium Cohort Study: Longitudinal Family File, 2001-2018. 4th Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021a. Millennium Cohort Study: Fifth Survey, 2012. 5th Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021b. Millennium Cohort Study: Fourth Survey, 2008. 8th Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021c. Millennium Cohort Study: Geographical Identifiers, Fifth Survey, 2001 Census Boundaries: Secure Access. 2nd Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021d. Millennium Cohort Study: Geographical Identifiers, Fourth Survey: Secure Access. 2nd Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021e. Millennium Cohort Study: Geographical Identifiers, Second Survey: Secure Access. 2nd Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021f. Millennium Cohort Study: Geographical Identifiers, Third Survey: Secure Access. 2nd Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021g. Millennium Cohort Study: Second Survey, 2003-2005. 10th Edition ed.: UK Data Service.

UNIVERSITY OF LONDON, I. O. E., CENTRE FOR LONGITUDINAL STUDIES 2021h. Millennium Cohort Study: Third Survey, 2006. 8th Edition ed.: UK Data Service.

VALAVANIDIS, A., FIOTAKIS, K. & VLACHOGIANNI, T. 2008. Airborne particulate matter and human health: Toxicological assessment and importance of size and composition of particles for oxidative damage and carcinogenic mechanisms. *Journal of Environmental Science and Health - Part C Environmental Carcinogenesis and Ecotoxicology Reviews,* 26**,** 339-362.

VIENO, M., HEAL, M., HALLSWORTH, S., FAMULARI, D., DOHERTY, R., DORE, A., TANG, Y., BRABAN, C., LEAVER, D. & SUTTON, M. 2014. The role of long-range transport and domestic emissions in determining atmospheric secondary inorganic particle concentrations across the UK. *Atmospheric Chemistry and Physics,* 14**,** 8435-8447.

VIENO, M., HEAL, M., WILLIAMS, M., CARNELL, E., NEMITZ, E., STEDMAN, J. & REIS, S. 2016. The sensitivities of emissions reductions for the mitigation of UK PM2. 5. *Atmospheric Chemistry and Physics,* 16**,** 265-276.

VIOLATO, M., PETROU, S. & GRAY, R. 2009. The relationship between household income and childhood respiratory health in the United Kingdom. *Social Science and Medicine,* 69**,** 955-963.

WEINMAYR, G., ROMEO, E., DE SARIO, M., WEILAND, S. K. & FORASTIERE, F. 2010. Short-Term effects of PM10 and NO2 on respiratory health among children with asthma or asthma-like symptoms: A systematic review and Meta-Analysis. *Environmental Health Perspectives,* 118**,** 449-457.

WHEELER, B. W. & BEN-SHLOMO, Y. 2005. Environmental equity, air quality, socioeconomic status, and respiratory health: A linkage analysis of routine data from the Health Survey for England. *Journal of Epidemiology and Community Health,* 59**,** 948-954.

WHITROW, M. J. & HARDING, S. 2010. Asthma in Black African, Black Caribbean and South Asian adolescents in the MRC DASH study: a cross sectional analysis. *BMC pediatrics,* 10**,** 1-7.

WICKHAM, S., ANWAR, E., BARR, B., LAW, C. & TAYLOR-ROBINSON, D. 2016. Poverty and child health in the UK: using evidence for action. *Archives of disease in childhood,* 101**,** 759-766.

WILSON, A. M., WAKE, C. P., KELLY, T. & SALLOWAY, J. C. 2005. Air pollution, weather, and respiratory emergency room visits in two northern New England cities: an ecological time-series study. *Environmental research,* 97**,** 312-321.

WRIGHT, R. J., MITCHELL, H., VISNESS, C. M., COHEN, S., STOUT, J., EVANS, R. & GOLD, D. R. 2004. Community violence and asthma morbidity: the Inner-City Asthma Study. *American journal of public health,* 94**,** 625-632.

ZURAIMI, M., THAM, K., CHEW, F., OOI, P. & DAVID, K. 2008. Home exposures to environmental tobacco smoke and allergic symptoms among young children in Singapore. *International archives of allergy and immunology,* 146**,** 57-65.