Health Effects of Noise and Air Pollution
Empirical Investigations

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Abstract

The assessment of the relationship between pollution emissions and health has direct economic implications. Health status is an important factor influencing worker productivity, and hence economic growth, as well as impacting on individual well-being. We implement various strategies to disentangle the relationship between short-term noise and air pollution exposure and health. In two studies we look at airports, which are sources of both environmental stressors. In the first study we use an administrative dataset on all hospitalisations in England, the Hospital Episode Statistics (HES). We compare hospital visits between people living within certain noise levels near airports to people living further away. In the second study we focus on prescription drugs in regions around London Heathrow airport. This study exploits a trial performed over five months at Heathrow airport that redirected approaching aircraft to reduce early morning noise in designated areas. A third study implements an instrumental variable approach, where the endogenous variable of daily levels of air pollution is instrumented with daily indicators of wind direction. In this case, the health outcomes investigated are again HES visits.

Informed by the medical literature, this thesis focuses on three different health categories: nervous, circulatory and respiratory. The results of the first study show statically significant increases in visits for nervous and respiratory outcomes for people living near airports. Furthermore, we observe a substitution of admissions from elective to emergency hospitalisations. The study that exploits the Heathrow airport trial shows that prescribed medication usage is significantly correlated with air traffic around that airport. Compared to the control regions, we observe a significant decrease in prescribed drugs for respiratory and nervous system conditions in the areas affected by a reduction in air traffic. The third study on daily variation of air pollution, finds a statistically significant increase in nervous emergency hospital visits. Across the three different approaches, nervous conditions are the mostly affected. These conditions include sleep disturbance, attention deficits and other stress-related diseases.
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Abbreviations

2SLS Two-Stage Least Squares

ACR Average Causal Response

A&E Accident and Emergency or emergency room in the UK

CIPS Continuous Inpatient Spells

DD Difference-in-Differences

GP General Practitioner doctor or physician in the UK

HES Hospital Episode Statistics

IMD Index of Multiple Deprivation

IV Instrumental Variable

LAD Local Authority District - a census unit in England

LATE Local Average Treatment Effect

LIML Limited Information Maximum Likelihood

LSOA Lower-layer Super Output Area - a census unit in England

MSOA Middle-layer Super Output Area - a census unit in England

OLS Ordinary Least Squares

WHO World Health Organization
Nomenclature

CO  Carbon monoxide

Lday Day equivalent noise level measured over the 12-hour period 0700-1900 hours

Lden Day-evening-night equivalent noise level

Leq Equivalent continuous noise level meter - sound pressure level in dB

NO₂ Nitrogen dioxide

NOₓ Oxides of nitrogen

O₃ Ozone

PM Particulate matter

PM_{0.1} Particulate matter with a maximum diameter of 0.1 µm

PM_{10} Particulate matter with a maximum diameter of 10 µm

PM_{2.5} Particulate matter with a maximum diameter of 2.5 µm

SO₂ Sulphur dioxide
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Chapter 1

Introduction

1.1 Motivation and Research Aims

The assessment of the relationship between pollution emissions and health has direct economic implications. Health status is an important factor influencing worker productivity, and hence economic growth, as well as impacting on individual well-being. The study of the relationship between environmental exposure and its adverse health effects is a well-documented field in environmental, epidemiological and medical research. The aim of this thesis is to provide a quantitative contribution to the existing findings on the assessment of the relationship between human health and environmental exposure. This dissertation adds to the existing economics literature by investigating the causality of the path that leads from short-term air and noise pollution exposure to adverse health effects for an entire population. This relationship is investigated in the three empirical chapters of the thesis.

The analysis in the three chapters have common features in that they investigate the impact of some source of pollution on a measure of health outcomes. They focus on three conditions that are known in the medical literature to be affected by pollution, nervous, circulatory and respiratory conditions; the reasons for choosing these are outlined in Chapter 2. The three chapters are all based on variations across
geographies to environmental exposure, although the level of geographical detail varies. The chapters vary in terms of the health measures used, hospital visits or medications, and the source of pollution, proximity to air traffic and particulate emissions more generally. The empirical analysis is based on regressions but the identification strategy differs between the three studies. Taken together they enhance the understanding of the impact of environmental exposure on health.

The first empirical chapter (Chapter 3) builds on the observation that airports are sources of both, noise and air pollution. The chapter investigates whether people living close to airports in England increase attendance to hospital for nervous, circulatory or respiratory conditions compared to residents further away from these sources of pollution. Specifically, the study examines visit rates using hospitalisation data from the 2008 to 2014 Hospital Episode Statistic data (HES). Since the levels of noise and air pollution are higher near airports, there might be a difference in hospital visits between people living close and those living further away from airports. This chapter will therefore use OLS techniques to assess this difference. Furthermore, this study examines the pattern of substitutions between emergency and planned visits for the same two subgroups. Differences in the type of admission can have a substantial economic impact on the National Health Service (NHS), where emergency visits are more expensive than elective visits.

The second piece of empirical analysis (Chapter 4) provides quasi-experimental evidence on the health effects of air traffic. In this case, health effects are measured as general practitioner (GP) prescribed medicines for nervous, circulatory or respiratory conditions. Air traffic is geographically modeled as the density of aircraft landing at London Heathrow airport. The quasi-experimental setting is provided by a trial undertaken during early morning hours between November 2012 and March 2013, which affected all residents underneath Heathrow approach paths. The empirical analysis for this chapter distinguishes between a treatment group, which is affected by the changes introduced by the trial, and a control group, which is not. The estimations are carried out adopting a difference-in-differences (DD) technique that
Chapter 1. Introduction

captures the causal effect of the air traffic variation.

The final empirical chapter (Chapter 5) considers the short-term impact of air pollution. In this chapter, while environmental noise exposure is not examined, there is a local focus on daily exposure to particulate matter (PM), which is one of the most commonly studied pollutants. The causal investigation here is based on the identification of an instrumental variable (IV) to overcome endogeneity issues related to local measures of pollution levels. We instrument daily local PM levels with daily local wind direction and we estimate the effects of these variations on hospital visits. Once more, the relevant conditions are those related to the nervous, the circulatory or the respiratory systems. As in Chapter 3, hospital visits data are taken from the 2008 to 2014 HES datasets, which allow to distinguish between types of admissions. Similarly to Chapter 3, we investigate the interplay between planned and unplanned visits under the new setting of daily exposure to PM.

1.2 Structure of the Thesis

The thesis is structured as follows. Prior to undertaking the empirical investigations, it is necessary to introduce the context and theoretical frameworks of background research; this is the subject of Chapter 2. This begins with the economic modeling of exposure and health, followed by reviews of the relevant environmental and medical research. As we discussed above, the subsequent three empirical chapters investigate the relationship between air and noise pollution and health. Chapter 3 examines the health effects of living in the proximity of airports, Chapter 4 exploits an exogenous variation in air traffic to assess the relationship between environmental exposure and medication spending and Chapter 5 adopts an instrumental variable approach to estimate the change in hospitalisation rates induced by local variations in daily levels of air pollution. Finally, Chapter 6 discusses the findings and their policy relevance and directions for future research.
Chapter 2

Environmental Exposure and Health

This chapter discusses the theoretical framework of the health effects of environmental exposure and the causality of their relationship. These are the concepts underpinning the analyses exposed in the other chapters of this dissertation. This chapter is divided in six sections. While Section 2.1 introduces the theoretical framework of this thesis, Section 2.2 reviews the quasi-experimental evidence. Subsequently Section 2.3 introduces airports as sources of both air and noise pollution. Furthermore, Section 2.4, Section 2.5 and Section 2.6 survey the current medical literature on the health effects of environmental hazards. Section 2.7 concludes summarising the major points that are crucial to the understanding of the rest of the dissertation.

2.1 Theoretical Framework

Understanding the health effects of environmental exposure represents a crucial task for economic research. Providing citizens with a good quality of life is one of the main challenges of modern society. Health is a major factor that influences life quality. Classic economic theory models each individual’s health stock with
the health production function. In 1972, Michael Grossman, the pioneer of the conceptualisation of health capital, described the investment in health capital as one form of investment in human capital (Grossman, 1972), which is the worker’s stock of innate or acquired knowledge and skills that have economic value (Schultz, 1961; Becker, 1962). More recently, Grossman expanded the human capital model of the demand of health to include health in the individual utility function (Grossman, 2000). Thus, health does not only affect future productivity, but it also contributes directly to personal satisfaction.

With environmental exposure having an effect on health, it ought to take part to both the health production function and utility function. Among the environmental externalities, air and noise pollution have been at the centre of the academic and political debate for a long time. There are still substantial concerns on how to assign noise and air pollution levels to individuals and how to account for the duration of exposure and for individual mobility. Air and noise pollution measurements can be biased by residential sorting, avoidance behaviour and environmental confounding factors. In the spirit of Graff Zivin and Neidell (2013), we present extended versions of the health production function and the utility function to take into account individual environmental exposure, health capital and all those factors influencing the endogenous nature of air and noise pollution.

The new environmental health production function takes the following form:

$$ H = H(M(\phi), \phi(P, A)) $$  \hspace{1cm} (2.1)

where $P$ is ambient pollution levels; $M$ is medical care (post-exposure); $\phi$ represents illness episode and $A$ indicates avoidance behaviour (pre-exposure).

Given that individual health directly affects individual preferences, the new individual utility function takes the following form:

$$ U = U(C, L, H) $$  \hspace{1cm} (2.2)
which represents personal preferences over consumption \((C)\), leisure \((L)\) and health \((H)\).

The marginal effect of pollution on population health can be calculated as the derivative of the health production function of equation (2.1) with respect to pollution \((P)\) as follows:

\[
\frac{dH}{dP} = \left( \frac{\partial H}{\partial M} \frac{\partial M}{\partial \phi} + \frac{\partial H}{\partial \phi} \right) \left( \frac{\partial \phi}{\partial P} + \frac{\partial \phi}{\partial A} \frac{\partial A}{\partial P} \right)
\]  

(2.3)

The right-hand side of this equation comprises two factors: while \((I')\) represents the degree to which individual illness is translated into population’s health status, \((II')\) corresponds to the net impact of pollution on illness episodes depending on individual exposure (pure biological effect of pollution plus avoidance behaviour).

Although equation (2.3) cannot be empirically calculated due to data limitations, it offers a clear conceptual benchmark to guide our empirical investigations. Figure 2.1 shows a simplification of the basic underlying theoretical model of this thesis.

This research embodies all the variables described above with the aim to assess the interaction between environmental exposure, health and socioeconomic status (SES). We have identified five main relationships of interest: (i) from environmental hazard to illness and health capital; (ii) from environmental hazard to SES and human capital; (iii) SES and human capital as a confounder in the relationship between environmental hazard and health; (iv) from illness and health capital to SES and human capital; (v) avoidance behaviour and residential sorting. These relationships will be discussed in more detail in the subsequent subsections.
Subsection 2.1.1 describes (1), Subsection 2.1.2 describes (2), Subsection 2.1.3 describes (3), Subsection 2.1.4 describes (4) and Subsection 2.1.5 describes (5).

2.1.1 From environmental hazard to health capital and illness

Evaluating the effects of environmental hazards on health is the primary interest of this research. In order to estimate the environmental exposure effects on health capital, several types of health outcomes are investigated in the empirical literature: (i) mortality; (ii) morbidity and (iii) fetal health. Due to data availability many studies predominantly look at mortality. The essential concern in this case is called ‘harvesting’ (Graff Zivin and Neidell, 2013). This is the displacement of short-term mortality. It refers to situations where deaths from pollution include both, healthy individuals and already ill people. Whereas it is often impracticable to make this distinction with the data at hand, it makes a big difference when considering the impact on society. The loss borne by society is substantially smaller if pollution only affects people with prior health conditions. In this case, pollution anticipates death by days or weeks. For an individual already in poor health, pollution only increases their likelihood of dying (even though their poor health would have led to an earlier death anyway, compared to a healthy person).
Ultimately, changes in population morbidity might provide a more appropriate read-out of both chronic and acute conditions induced by adverse environmental exposure. Hospitalisations, medication usage, doctor visits, blood pressure level, emergency room visits and cognitive performance metrics are examples of measured morbidity (Brunekreef and Holgate, 2002; Basner et al., 2014). In addition, there is some evidence that fetal health is affected by pollution (Currie, 2009). Janet Currie (2009) shows the presence of a causal correlation between environmental exposure and the reduction of birth weight and gestational age.

Economics can contribute extensively to overcome limitations of the epidemiological studies on pollution and health. In fact, econometrics provides empirical tools that can capture how people respond to ambient pollutants, moving house or changing their behaviour, for instance, otherwise difficult to estimate in the setting of medical research. Moreover, it gives the power to test the causality and validity of both the pure biological effect of pollution - $\frac{\partial \phi}{\partial P}$ of equation (2.3) - and the consequences of environmental hazard exposure on national human capital (Graff Zivin and Neidell, 2013).

### 2.1.2 From environmental hazard to SES and human capital

There is a rich economics literature that draws on Grossman and Krueger’s seminal paper *Economic Growth and the Environment* (Grossman and Krueger, 1995). It develops the idea that low income leads to higher pollution rates in an initial phase, followed by a phase of improved pollution when income is higher. This relationship is represented by the inverted-U shaped environmental Kuznet curve and its one-way causality has been the subject of much criticism (Dasgupta et al., 2002) since environmental hazards can actually affect wages and utility. Hence, this relationship identifies a likely poverty trap (Graff Zivin and Neidell, 2013). This assumption complements the theory that sees human capital as the engine of economic growth (Schultz, 1961; Nelson and Phelps, 1966; Romer, 1986). Thus,
environmental safeguard is no longer viewed as a lagging factor of national economic
development - such as a tax imposed on the market players - but as an essential
investment aimed to protect human capital.

Government policies are in place to regulate environmental hazards. Graff Zivin and
Neidell (2013) propose a mathematical model to account for government policies
in the context of environmental quality and improved welfare. Within this context,
optimal regulation ($R$) is intuitively related to individuals’ willingness-to-pay to
improve their welfare. Building on the health production function (2.1) and the
utility function (2.2), they describe such a relationship with the following equation:

$$
\frac{\partial P}{\partial R} = \left( \frac{\partial w}{\partial H} + \frac{\partial U}{\partial H} \lambda \right) \frac{dH}{dP} + \frac{\partial A}{\partial P} + \frac{\partial M}{\partial P} \lambda
$$

(2.4)

where $c_j$ is the price of good $j$ (with $j$ taking one of the following three values: $R$
for regulation; $A$ for avoidance behaviour; $M$ for medical care); $w$ is wage and $\lambda$
is the Lagrangian multiplier. The left-hand side of the equation represents the social
marginal cost of regulation ($R$) and the right-hand side represents the willingness-to-
pay to reduce pollution. The first component ($I$) measures the impact of pollution-
driven morbidity on earnings and utility. The second component ($II$) constitutes
avoidance costs and the third ($III$) identifies medical expenditure due to pollution.
To conclude, this equation crucially decomposes the main elements of environmentally
effective public policies.

2.1.3 SES and human capital as confounders in the relationship between environmental hazard and health

The synergy of two major mechanisms allegedly explains how environmental exposure
conduces to social health inequalities (O’Neill et al., 2003; Forastiere et al., 2007;
Bolte et al., 2009; Deguen and Zmirou-Navier, 2010). These two mechanisms are
called *differential exposure* and *differential vulnerability*. The former depends on three continuous variables: exposures at work (where low-wage jobs correlate to poor air and noise quality); outdoor pollutants and noise; indoor pollutants, noise and allergens. The latter is composed of several social and health determinants: existing medical conditions; genetic susceptibility; access to health care; access to fresh food; violence and stress. The contemporaneous presence of exposure and vulnerability explains the cause of the confounding action of socioeconomic variables on the health effects of pollution (O’Neill et al., 2003; Forastiere et al., 2007).

### 2.1.4 From health capital and illness to SES and human capital

The relationship between health and income is one of the most famous positive correlations (see Figure 2.2) (Bloom and Canning, 2000). It is well established that health conditions affect economic growth. At the national level, expenditures on public health are higher for richer countries. Whereas at the individual level, the quality of food, housing and medical care depends on personal wealth.

On the one hand, the health status of each person has direct effects on country productivity, such as earlier retirement, “absenteeism” and “presenteeism”, where the last two terms are borrowed from the occupational health literature (Burton et al., 1999). The former implies a short-term situation of missing days at work due to illness. The latter, instead, refers to a reduction of the on-the-job productivity and work quality.

On the other hand, there are several indirect mechanisms that describe the effects of health on human capital. Firstly, the level of cognitive skills and school attendance conditions the quality of education received. By definition, human capital is improved through schooling and school absenteeism, due to illness, hinders human capital accumulation (Grossman and Kaestner, 1997). Secondly, healthier people are more
likely to invest in education and attain higher levels since their return can be amortized over a longer working life. Thirdly, once in the labour market, improvement in life expectancy may lead people to save for their allegedly longer retirement period, therefore raising the levels of investment (Weil, 2007). In contrast, however, a longer life is associated with increased morbidity and multiple-morbidity among the elderly, which implies more out-of-pocket health expenditure (Schoenberg et al., 2007). Moreover, if an individual experiences an adverse health shock while still working, there will be a change of future time horizon. This, in turn, will impact their preferences over work and leisure. In order to maximise their utility under the new health conditions, individual’s labour supply will be affected subject to their lifetime budget constraints and their expectations regarding future health (Chatterji and Tilley, 2002).

In summary, improvements in population health lead to economic growth and there exists a mutual reinforcement between productivity and health (Bloom and Canning, 2000). In fact, income boosts investments in population health thanks to advanced knowledge of health improving behaviours. A recent empirical research confirmed
this two-way causation through a panel data analysis of life expectancy and GDP data in OECD countries (French, 2012).

### 2.1.5 Avoidance behaviour and residential sorting

Avoidance behaviour and residential sorting are mechanisms that explain why pollution is an endogenous variable. Namely, pollution exposure can be modified in a variety of ways according to the scale and nature of the environmental hazards (e.g., moving home or changing way of transport). This confounding effect is described in the \((II^\prime)\) component of equation (2.3). The term \(\partial \phi / \partial A \times \partial A / \partial P\) explicitly shows to what extent health conditions can be obviated by actively altering pollution exposure.

Non-random assignment is another source of endogeneity and has strong correlation with the individual socioeconomic status. It is one of the attributes of the environmental quality assessment that people consider when choosing a place to live. Using the words of Charles Tiebout sixty years ago, “people vote with their feet” also due to environmental quality concerns (Tiebout, 1956). This phenomenon has been more recently re-tested by Banzhaf and Walsh (2008)’s empirical work. Employing a difference-in-differences strategy looking at changes in pollution emissions and changes in neighborhood demographics, they tested for environmentally motivated migration patterns. They find evidence of an increase in the relative population associated with improvements in air quality. Moreover, they find evidence of a relationship between local pollution and emigration of richer households and/or immigration of poorer households over the decade 1990 to 2000.
2.2 Quasi-experimental evidence

Estimating the causal impact of pollution on health meets several limitations. Equation (2.3) synthesises all the issues related to empirical research. To begin with, in order to infer on pollution (P, which can equally refer to either noise or air pollution), we need to take into account its endogenous nature. As we mentioned above, there are substantial concerns on how to assign pollution levels to individuals, how to account for the duration of exposure and for individual mobility. Moreover, residential sorting, avoidance behaviour \((\partial \phi / \partial A \times \partial A / \partial P)\) and environmental confounding factors need to be considered since they bias the estimation of the effects of pollution. Not to mention controversies on health outcome, e.g., identification of relevant illness \((\phi)\), its measurement and the final valuation of population health \((\partial H / \partial \phi)\), including the medical expenditure dimension \((\partial H / \partial M \times \partial M / \partial \phi)\). Within the published research there is evidence of attempts to tackle these issues.

The literature on changes in pollution, resulting from either unexpected events or inter-temporal variation of pollution rates, has a long tradition, starting with the analysis of the health effects of the London killer smog days in December 1952 (Logan, 1953). Subsequently, changes in pollution levels from the closure of a US steel mill in 1986 inspired several similar experimental strategies (Pope III, 1989; Ransom and Pope III, 1995). However, this stream of literature entirely concentrates on air pollution disregarding the health effects of environmental noise.

Alternatively a few studies exploit the introduction of policies, regulations or specific events, which in turn affect emissions of air pollutants and noise. In this case, the focus is on the source of pollution, being either car traffic (Friedman et al., 2001; Currie and Walker, 2011; Knittel et al., 2016), diesel school buses (Beatty and Shimshack, 2011), airport activities (Schlenker and Walker, 2016; Boes et al., 2013), electricity-generating units (Deschenes et al., 2012), boat traffic (Moretti and Neidell, 2011) or oil refineries (Lavaine and Neidell, 2017).
There are many publications that deal with avoidance behaviour suggesting techniques and strategies to account for it. For instance, Matthew Neidell (2009) finds a decrease in attendance to outdoor leisure facilities (a zoo and an observatory in Los Angeles) subsequent to the breach of an $O_3$ threshold, which is communicated through smog alerts. He regresses attendance rates on days, either just above or just below, the $O_3$ threshold, to estimate the causal relationship between air quality alerts and avoidance behaviour.

There is also evidence on the impact of pollution exposure on labour market endpoints. A very innovative paper is one published by Graff Zivin and Neidell (2012), which looks at the productivity (measured as corp pieces collected per hour) of seasonal workers. Exploiting seasonal variations of $O_3$ and the peculiarity of this workforce, they account for avoidance behaviour. They find that a relatively small decrease in $O_3$ concentrations corresponds to a significant increase in worker productivity.

### 2.3 Airports

Generally a single source of pollution is responsible for two or more types of emissions: for instance, cars and aircraft are sources of both, noise and air pollution. In the UK, aviation emissions accounted for 6% of total greenhouse gas emissions in 2014\(^1\). Specifically, airport operations are responsible for significant emissions of several pollutants, such as particulate matter (PM) of various sizes, carbon monoxide and dioxide (CO and $CO_2$), nitrogen oxides ($NO_2$ and $NO_x$) and sulphur dioxide ($SO_2$) (Masiol and Harrison, 2014). In addition, aircraft are at their noisiest when taking off or landing, making noise a major concern for the communities living near airports. Moreover, often airports lie close to large urban areas and provide an attractive setting to explore the relationship between their emissions and population health.

There are several environmental studies that specifically focus on airport-related emissions.

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\(^1\)The Committee on Climate Change - https://www.theccc.org.uk.
pollution. Researchers from MIT’s Laboratory for Aviation and the Environment implemented a concentration-response function using emissions estimates for UK airport activities (Yim et al., 2013). To calculate mortality risk they employ a pollution-health ratio previously found in a series of quasi-experimental studies involving the closure of a steel mill in the Utah valley in the US (Pope III, 1989; Pope III et al., 1995).

Remaining in the UK, a five-day shutdown, due to the Icelandic volcano eruptions in April 2010, allowed researchers at the Environmental Research Group, King’s College London, to isolate nitrogen oxides (NO₂ and NOₓ) concentrations generated by London Heathrow airport (Carslaw et al., 2012). They estimated the impact of airport operations on the annual mean to be about 16 µgm⁻³ for NOₓ and 8.5 µgm⁻³ for NO₂. Under EU directives, based on the WHO guidelines (WHO, 2006), the annual mean values should not exceed 30 and 40 µgm⁻³, respectively. Remarkably, emissions from Heathrow airport are a sizeable proportion of overall pollution in its vicinity. The relative positions of monitoring sites along Heathrow airport boundaries and the known meteorologic conditions created the perfect setting for Carslaw and colleagues to identify the pollution share injected by this specific source and the related road traffic. Comparing pollution business-as-usual patterns to measured ones, they find a decrease in pollution levels associated with the inactivity of the airport. Although they technically use a difference-in-differences approach, they do not formally employ one; thus confounding a causal interpretation of their findings.

2.4 Air Pollution and Health

One of the most documented fields pertains to air pollution and its adverse health effects. The profusion of publications is justified by the complexity of the topic, which still has many unanswered questions.

The pathways that link pollution as a mixture of several agents to health endpoints are
not clearly understood. What we know from the medical and experimental literature is the interaction mechanisms between each single air pollutant and the human body (see Appendix 2.A for a thorough description of pollutants). Some of air pollution components, such as $O_3$, $NO_x$ and $PM_{10}$, have a common property that characterises the overall health impact. In fact, they all activate oxidative stress that causes cell damage and death (Brunekreef and Holgate, 2002). Pope III and Dockery (2006) focus specifically on the consequences of fine particles exposure. The authors summarise how PM interacts with all kind of human tissues, and thus significantly affecting human morbidity. They also highlight the role that a number of socioeconomic (e.g., education, housing, healthcare) and personal (e.g., pre-existing respiratory and cardiovascular diseases, diabetes, medication use) characteristics play in affecting the scale of susceptibility. Genetic specificity triggers environmental exposure differently as well (Elliott, 2014). In addition, $NO_2$ triggers lung infection (Brunekreef and Holgate, 2002). Exposure to ozone is also known to induce inflammatory responses in the lungs. This generates a complex chain of events involving the nervous system and leading to an increased responsiveness to asthma for patients with hypersensitive airways (Schierhorn et al., 2002).

Epidemiological research unravels the relationship between short-term variations in pollutant concentrations and the occurrence of respiratory morbidity. There is strong evidence of symptom exacerbations of asthma and other respiratory diseases, transient worsening in lung function and increased respiratory infections, all resulting in more visits to general practitioners and hospitals (Brunekreef and Holgate, 2002; Li et al., 2012).

In their meta-analysis on the ozone-mortality relationship, Levy et al. (2005) report a differential effect during summer and winter. Due to the seasonal nature of ozone (i.e. higher levels with warmer weather), an increase of 10 $\mu g/m^3$ of its concentration is linked to an increase of 0.43% in mortality during summer and a decrease of 0.02% in mortality during winter. They also see that same-day ozone effects are stronger than lagged effects. This could be explained by the high variation in its concentrations
due to its facility to react with other pollutants and to transform into different ones.

Among the most common air pollutants, particulate matter is responsible for the greatest risk for human health (WHO, 2013). PM$_{2.5}$ is associated with cardiovascular risk. Brook et al. (2010) review studies on the matter and highlight a 0.4 to 1% increase in relative risk for daily cardiovascular mortality per 10 µg/m$^3$ 24-hour PM$_{2.5}$ concentration elevation. Cohort studies show that long-term exposure is characterised by a higher risk between 1.06 to 1.76% per 10 µg/m$^3$ increase in PM$_{2.5}$ levels. Brook et al. (2010) add that the risk is not equally distributed within a population. There are sensitive groups that have a stronger negative response to pollution - the elderly and those with existing conditions, specifically with coronary artery or structural heart disease.

Hoek et al. (2013) update Brook et al. (2010)’s review adding evidence on specific causes of death and on respiratory mortality. Although they confirm the significant effect on circulatory mortality, they do not find any significant increase in respiratory mortality associated with PM$_{2.5}$ levels, which may be due to mortality displacement. Hoek’s review also reports a 5.5% increase in overall mortality due to a 10 µg/m$^3$ increase in NO$_2$. The same review also stresses the role of ischemic heart disease and myocardial infarction as the specific causes of death that drive the effect on mortality. Overall, they call for a more uniform definition of exposure and confounding factors selection that would help to compare health estimates across studies. For instance, there is evidence of heterogeneity between geographic regions for the relationship between particulates and health. This variability highlights the importance of controlling for confounding factors that could systematically weaken the estimated effects. Ultimately, some caution is necessary when extrapolating risk estimates to regions with different pollutant levels (Shah et al., 2013).

There are a few recent papers that adopt a multi-pollutant approach. They all take different strategies on how to include a pollution mixture in the analysis. It is worth mentioning a study in France, which adopts a regression of an artificially created
pollution index on hospital admissions for respiratory diseases (Chardon et al., 2007). Chardon and colleagues implemented a weighted sum of $O_3$ and $NO_2$ with the aim to obtain a more representative indicator of the kind of pollution that reacts with light. Even though their results are stronger for ozone-only levels, they suggest the adoption of artificial indicators as a way to explore health effects.

WHO calls for a strong epidemiological assessment of the impact of the mixture of air pollutants on human health (WHO, 2013). This strengthens the belief built on the results of a London study on particle metrics monitored between 2000 and 2005 (Atkinson et al., 2010) where particle number concentrations, instead of the type of pollutant, appeared to play a large part in the adverse interaction with human health\(^2\).

Air pollution is also particularly harmful to children’s health (Currie, 2013). Compared to adults, their respiratory and immune systems are in the delicate phase of development, they spend more time outdoor and they have higher ventilation rates (WHO, 2013; Beatty and Shimshack, 2014).

### 2.5 Ambient Noise and Health

Traffic noise exposure has been found to have a non-auditory impact on human health. It increases mortality and the prevalence of strokes, myocardial infarction (heart attack) and cardiovascular morbidity as well as affects the endocrine system, perceived annoyance, cognitive impairment and sleep disturbance (Huss et al., 2010; Correia et al., 2013a; Hansell et al., 2013; Basner et al., 2014).

The human body can respond through direct and indirect pathways to acute exposure to noise. The latter refers to the path from perceived nuisance to emotional stress

\(^2\)Due to the 1952 London killing fog episode (Logan, 1953), London has historically hosted world-leading research groups specialised in monitoring air pollution levels. One of these is the *Environmental Research Group*, King’s College London, that manages the London Air Quality Network - http://www.londonair.org.uk - and co-authored the aforementioned paper by Atkinson et al. (2010).
reactions. The direct pathway consists of the autonomic physiological stress triggered by the interaction between the central auditory system and the central nervous system. Even at low noise levels, this is considered to be the prevalent mechanism in sleeping individuals (Basner et al., 2014). Observations on chronically exposed populations showed an effect on the metabolism and the deterioration of the cardiovascular system (Basner et al., 2014). Sleep disturbance is regarded as the most harmful effect of environmental noise exposure. Occasional incidents as low as 33 dB $L_{A\text{max}}$ at night can induce various physiological reactions during sleep, such as tachycardia, body movements and awakenings (Basner et al., 2014). There is conflicting evidence on the size of these effects, which differ depending on whether elderly, children or people with existing conditions are examined (van Kamp and Davies, 2013).

Noise source is a fundamental contributor in reaction to noise. Different sources hold different acoustic characteristics: frequency, sound level, duration, intensity and psychoacoustic measures. For instance, at the same average night noise level, aircraft noise is found to trigger a higher level of annoyance than other transportation noise (Working Group on Health and Socio-Economic Aspects, 2004). In this relationship, gender and age play the role of effect modifiers.

Studies on noise effects date back to the 1970s (Ando et al., 1975). Initially laboratory settings were promoted, followed by field experiments with a focus on airports (Cohen et al., 1981; Chen and Chen, 1993; Evans et al., 1995). The findings on the harmful effects of noise on blood pressure were comparable to findings on cognitive impairment. However, it should be noted that levels of noise were much higher and less regulated back then. Over the years, qualitative research played an increasingly important role in documenting individuals’ reaction to noise.

The effects of aircraft noise on cognitive impairment, annoyance and hypertension have been explored using a combination of research methods in three significant European projects: 5A - Attitudes to Aircraft Annoyance Around Airports (Heaver, 2002); HYENA - Hypertension and Exposure to Noise near Airports (Floud et al.,
2011) and RANCH - Road Traffic and Aircraft Noise Exposure and Children’s Cognition (Clark et al., 2013). Through the use of surveys, the first project finds some degree of correspondence but no exact coincidence between standard noise metrics and aircraft annoyance (Heaver, 2002). The HYENA cross-sectional study detects a positive correlation between aircraft noise and the consumption of anxiolytic and antihypertensive medications in European countries. The third longitudinal study focuses specifically on children and observes significant effects of environmental noise on reading comprehension and annoyance. Specifically, children tend to express higher degrees of annoyance than adults given the same exposure level (Clark et al., 2013).

2.6 Noise and Air Pollution and Health

The interaction between various indicators of pollution and their effects on human health is an understudied field (van Kamp and Davies, 2013). Publications on the contemporaneous effects of noise and air pollution are rare. Usually these studies independently estimate the exposure effects on health controlling for either stressor. As such, there is a gap in the understanding of the synergistic impact of both environment factors. An exception is an analysis by van Kempen et al. (2012) of the well exploited RANCH study on cognitive outcomes for school children aged 9-11 years. The researchers conclude that effects of noise and pollution are mainly independent. Nevertheless, they do detect some interaction. They find that high noise levels confound the effect of NO$_2$ on cognitive outcomes, although the physiological mechanisms are yet to be explored.

A few recent population studies designed for Greater London in the UK, Vancouver in Canada and Switzerland agree on the independent effects of air and noise exposure. However, they suggest to take into account either factor when estimating health effects (Tonne et al., 2016; Gan et al., 2012; Huss et al., 2010; Vienneau et al., 2015; Eze et al.,
Moreover, there is also a wide range of health endpoints analysed, making a comparison between studies a difficult task. These go from self-reported health and annoyance (Klæboe et al., 2000; Cohen et al., 2008) to myocardial infarction (Huss et al., 2010) and cognitive performance (van Kempen et al., 2012). In their review on the cognitive and psychological effects of environmental exposure, Tzivian et al. (2015) define the two different roles of long-term air and noise exposure in modifying adult neurocognitive functions and mood disorders, respectively. Stansfeld (2015) also finds an association between noise and depression and anxiety disorders when controlling for air pollution exposure as well. Both reviews stress the necessity to understand the underlying biological mechanisms.

In recent years, there has been increasing attention towards the relationship between transportation noise and air pollution with incidence of diabetes. Air pollution, through its inflammatory effect on the respiratory system, could mediate a chronic insulin resistance and subsequent onset of diabetes (Eze et al., 2015). Eze et al. (2017) find a strong effect of long-term exposure of road and aircraft noise on diabetes risk BUT no effect of NO₂ and railway noise exposure. Noise may affect diabetes through stress and sleep quality and quantity disturbance, which in turn affect behaviour and metabolism (glucose regulation and increased adiposity) (Cappuccio et al., 2010).

### 2.7 Conclusion

The evidence on the relationship between environmental pollutants and health indicates the presence of negative effects. Both short- and long-term exposure impact on human health through different channels. However, due to data limitations and methodological constraints, environmental and medical research have left many unanswered questions and the causal nature of this relationship is still under investigation. Firstly, personal exposure to ambient pollution is difficult to estimate given that people in Western societies spend 90% of their time indoors. Moreover, with
long-term exposure the events, which are known as chronic health effects, include only partially the acute effects due to short-term exposure.

Although mortality is often used as a measure of health, estimating its day-to-day variation related to pollution levels could overcount deaths. In fact, affected people may have died anyway from different causes and the pollution impact may only anticipate rather than cause fatalities (Brunekreef and Holgate, 2002). As we have seen in the previous sections, in the last two decades scholars have tried to tackle these issues, harnessing different research designs and exploiting various kinds of data on both environmental exposure and health endpoints.

The next chapters use different strategies and add to the existing economics literature by investigating the causality of the path that leads short-term air and noise pollution exposure to adverse health effects for an entire population.
2.A Appendix

Air Pollutants

Air pollution can be defined as a composition of different gaseous elements and airborne particles. From a health perspective, its principal and commonly monitored constituents are: carbon monoxide (CO), ozone (O₃), sulphur dioxide (SO₂), nitrogen dioxide (NO₂), oxides of nitrogen (NOₓ) and particulate matters (PM₁₀, PM₂.₅ and PM₀.₁³). These can be divided between primary pollutants, which are directly released into the air by combustion of fossil fuels (such as ozone and oxides of nitrogen) and secondary pollutants, which are formed by the interaction of different components in the atmosphere, such as ozone. Particulate matter is a mixture of constituents that differ in size and source (Newby et al., 2015). Many Western countries have regulations in place to keep these pollutants monitored. Table 2.A1 reports the European standards in place. Both, in the US and in Europe there are extensive networks of monitoring stations that continuously record ground levels of pollutants.

Table 2.A1: EU standards. Maximum concentrations allowed when averaged over time (WHO, 2000).

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>1h</th>
<th>8h</th>
<th>24h</th>
<th>Annual</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM₁₀ (µg/m³)</td>
<td>-</td>
<td>-</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>PM₂.₅ (µg/m³)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>25</td>
</tr>
<tr>
<td>PM₁₀–₂.₅ (µg/m³)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>PM₀.₁ (µg/m³)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>NO₂ (µg/m³)</td>
<td>200</td>
<td>-</td>
<td>-</td>
<td>40</td>
</tr>
<tr>
<td>NOₓ (µg/m³)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>30</td>
</tr>
<tr>
<td>CO (mg/m³)</td>
<td>-</td>
<td>10</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>O₃ (µg/m³)</td>
<td>-</td>
<td>120</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>SO₂ (µg/m³)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>20</td>
</tr>
</tbody>
</table>

³PM₁₀, PM₂.₅ and PM₀.₁ are particles with a maximum diameter of 10, 2.5 and 0.1 µm (micron), respectively. By comparison, a strand of hair has a width between 10 to 200 µm.
CO is mainly emitted into the atmosphere as a product of incomplete combustion. In terms of absolute concentrations CO is the most prevalent of the toxic air pollutants. Its concentrations are expressed in mg/m$^3$ (milligram - i.e. one thousandth of a gram - per cubic meter). This is a larger unit compared to all other pollutants, which are measured in µg/m$^3$ (microgram - i.e. one millionth of a gram - per cubic meter). The highest share of CO comes from slow moving vehicles. A smaller share results from processes involving combustion of organic matter, e.g., power stations and waste incinerators (European Parliament and Council of the European Union, 2000).

Ozone is a bluish gas with a detectable smell. It is formed principally by a complicated series of chemical reactions, started by sunlight, through a reaction of nitrogen oxides (NO$_x$) and volatile organic compounds (VOCs). These chemical responses are not instant, but can take up to a few hours or even days. Once ozone is generated it may endure for several days and it can travel long distances. Therefore, O$_3$ measured at a specific location may have arisen from VOC and NO$_x$ emissions hundreds of kilometres away. Thus, maximum levels are measured downwind of the emission source. In urban areas, where concentrations of traffic emissions may be high, nitric oxide (NO) from exhaust emissions may react with ozone to form nitrogen dioxide (NO$_2$), thereby reducing ozone concentrations (European Parliament and Council of the European Union, 2002).

Sulphur dioxide is a colourless gas, which results from the combustion of sulphur-containing fossil fuels (principally coal and heavy oils) and the smelting of sulphur containing ores (a mineral). Volcanoes and oceans are the major natural sources of SO$_2$ and it can be transported over large distances. After being released in the atmosphere, SO$_2$ is further oxidized and is associated with other pollutants in droplets or solid particles of various sizes. Sulphur dioxide and its oxidation products are cleared from the atmosphere through rain and dry deposition (European Parliament and Council of the European Union, 2008).

Oxides of nitrogen indicates the sum of nitrogen monoxide (NO) and nitrogen dioxide
(NO₂) expressed in µg/m³. NO₂ is a brown reactive gas with a perceptible smell, which occurs as a primary as well as a secondary pollutant. As a primary pollutant, NO₂ is mainly emitted from the tailpipe of diesel vehicles, especially when they move slowly. As a secondary pollutant, NO₂ is mainly formed by oxidation of nitric oxide, which is produced by burning fuel at high temperatures (European Parliament and Council of the European Union, 2008).

PM₁₀ are classified as inhalable or thoracic particles given their capability to penetrate the lower respiratory system. PM₂.₅ are known as fine or respirable particles and can penetrate into the gas-exchange region of the lung (Brunekreef and Holgate, 2002). Ultrafine particles (PM₀.₁) are particles with a diameter smaller than 0.1 µm and have a very short life (minutes to hours). They rapidly coagulate or condensate to form larger aggregates usually not bigger than PM₂.₅. Ultrafine particles are not regulated by official standards. However, there is growing interest in understanding the role they play for human health. In fact, due to their small size, they are more likely to pass from the lung into the blood (Pope III and Dockery, 2006).
Chapter 3

Living in the Proximity of Airports and Health

SUMMARY. Airports are sources of both noise and air pollution, which are known to have a negative effect on health. Looking at the population living close to the busiest airports in England, we investigate the relationship between distance to airports and hospital admissions. In this study, we look at residents within an average of 5 km from the first seven busiest airports in England and we compare their hospitalisation rates for nervous, circulatory and respiratory conditions to those of the population living further away from airports. The results show statistically significant increases in both elective and emergency visits for nervous and respiratory outcomes. Furthermore, this study contributes to the existing health economics literature estimating the substitution of admissions from elective to emergency hospitalisations. Under the short-term conditions of this research, we find statistically significant results of a shift of admissions from elective to emergency near airports for circulatory and respiratory conditions.
Chapter 3. Living in the Proximity of Airports and Health

3.1 Introduction

The aim of this chapter is to explore the relationship between health and exposure to air and noise pollution between 2008 and 2014. Airports are sources of both environmental factors and we look specifically at population living in the vicinity of major airports.

Noise has a strong subjective component: how noise is perceived differs from one person to another. Perception varies with noise intensity, frequency and duration and with individual sensitivity, which may confound the causal relationship running from noise to health. These factors complicate the challenging process involved in evaluating its impact on people’s well-being (Lawton and Fujiwara, 2016). There are acknowledged effects triggered by noise; some are auditory (usually related to occupational and recreational noise and not involved with ambient noise) while others are non-auditory. The latter span from perceived disturbance and annoyance, cognitive impairment in children, sleep disturbance and cardiovascular health. The medical literature explains the biological mechanisms underpinning these effects. For instance, the direct pathway that links noise exposure, even at low levels, to arousals of the autonomic nervous system is the predominant mechanism in sleeping individuals (Basner et al., 2014).

Airports emit air pollutants (such as nitrogen dioxide, carbon dioxide and particulate matter) through a variety of activities: from aircraft taxiing in their premises to landing and taking off. People that live in the proximity of airports are the most affected by their negative externalities. As reviewed in Chapter 2, the increasing numbers of epidemiological studies of the last two decades have identified cardiovascular and respiratory system diseases as the main health endpoints related to environmental exposure (Hoek et al., 2013).

This chapter seeks to provide estimates of the health effects of living in proximity to airports. It explores the relationship between exposure to airport noise as well as
air pollution and hospital admissions. We look at three broad categories of health conditions, i.e. nervous, circulatory and respiratory, which are proposed by the medical literature on pollution exposure and health.

We compare health outcomes of those living near airports to those living further away. Specifically, we compare patients within a certain noise threshold (with an average distance of 5 km from the airport) to patients who live between 10 to 30 km from the airport.

England and its seven busiest airports provide the right setting for our research question. First, England includes Heathrow airport, one of the biggest airports in the world for passenger traffic that lies within a highly populated area. Second, we have access to an administrative dataset that holds visit-level information on all patients in the country from 2008 to 2014. This data enables us to assign to the outcomes of interest the exact local area of residence, age group and gender.

We find significant effects on several health outcomes. Our econometric model, which consists of a log-linear regression of health outcomes on proximity to airports, suggests that hospital admissions for nervous disorders increased for residents near airports. They also show a pattern of shifts from elective to emergency admissions for the circulatory and respiratory classes.

The rest of the chapter is laid out as follows. Section 3.2 outlines the empirical specifications while Section 3.3 describes the data. This is followed by Section 3.4, which presents and discusses our results. The chapter concludes with final remarks in Section 3.5 and detailed tables of results are reported in Appendix 3.A.

### 3.2 Method

We use regression analysis to investigate the relationship between the main health outcomes and their environmental determinants. We assume that the process linking
the determinants and our main outcome is best described by the following log-linear regression equation.

\[
\ln \text{Visits}_{ih} = \alpha_{lh} + \delta_{lh} \text{close}_i + X_i' \beta_{i lh} + \text{demographic}_i^{lh} + \text{quartile}_i^{lh} + \epsilon_{ih} \tag{3.1}
\]

where \(\ln \text{Visits}_{ih}\) is the natural logarithm of the rate of visits by unit of observation \(i\) (which has regional and demographic components and is composed of LSOA\(^1\), age group and gender) and quarterly time \(t\) per method of admission \(l\) and for one of the health endpoints \(h\). More precisely, \(l\) can be either elective or emergency admissions. As suggested by the medical literature reviewed in Chapter 2, \(h\) refers to visits for one of the following three primary diagnoses: nervous, cardiovascular or respiratory system. Patients’ ages are grouped into standard five broad categories: 0 to 4, 5 to 19, 20 to 49, 50 to 64, 65 and over years old\(^2\).

The binary variable of main interest, \(\text{close}_i\), is equal to 1 when the LSOA is within 55 Lden or 57 Lday airport noise contours. \(X_i\) represents a vector of controls at the LSOA level: a set of indicators for the level of rural or urban land of local area (i.e. urban, town, village or hamlet sorted from the most to least populated area), indicators for the broader regions (i.e. North West, East Midlands, West Midlands, East of England, London, South East and South West) and indicators for the deciles of twelve different local indices of deprivation. The terms \(\text{demographic}_i\) and \(\text{quartile}_i\) represent demographic characteristics (i.e. age group, gender and all their possible interactions) and quarterly time effects, respectively. Finally, \(\epsilon_{ih}\) is the error term for the method of admission \(l\) and the health outcome \(h\).

Equation (3.1) represents a grouped data regression where the groups are given by the units of observation (i.e. by LSOA, gender and age group). In order to maximise the efficiency of the estimator and to produce coefficients identical to those potentially

\(^1\)A Lower-layer Super Output Area, or LSOA, is a census unit. There are more than 32,000 LSOAs in England, which on average include 650 households and a population of 1,500.

\(^2\)The first and the last age groups are those where the most treatments occur. We have added three categories in between to allow for varying susceptibility of diseases.
obtained using the underlying microdata sample, equation (3.1) is weighted by the population size of the unit of observation. In addition, the standard errors are robust to heteroskedasticity as well as to within unit of observation correlation. In fact, the null of constant variance of the Breusch-Pagan test for heteroskedasticity was rejected for all subsamples. To test for within cluster correlation, we estimate the intraclass correlation coefficients (of both, outcome and errors). We find evidence of within unit of observation correlation and, as a result, account for these sources of bias by adopting heteroskedasticity- as well as cluster-robust standard errors that allow for non-constant variance and within unit of observation correlation.

To detect effects specific to certain sub-populations, equation (3.1) is also estimated for a series of subsamples: (i) regions of the four airports near London (Heathrow, Gatwick, Stansted and Luton - see Figure 3.2); (ii) regions of the three major airports away from London (Manchester, Birmingham and Bristol - see Figure 3.2); (iii) males and females; (iv) for each age group separately; (v) and for two broader categories of urban or rural land levels, where the classes town, village and hamlet are pooled together to indicate a broader rural category.

The intrinsic differences between emergency and elective visits suggest that these two types of hospital admissions carry different information on the kind of health effects of environmental exposure. By the nature of elective admissions, they need to be booked in advance and planned. They are therefore more likely to capture long-term and chronic effects. Emergency admissions are more likely to be associated with acute conditions generated by short-term spikes of pollution.

Although equation (3.1) is implemented differentially for the two types of admissions, we want to explore how they interact. We propose to investigate the interaction between elective and emergency visits within the same diagnosis group introducing a
new model that takes the following form:

\[
\ln(EMvisits_{it}^h) = \alpha^h + \delta_h(close_i \times ELvisits_{it}^h) + X_i'\beta_h + \text{demographic}_i^h + \text{quartile}_t^h + \epsilon_{it}^h
\]  

(3.2)

where \(\ln(EMvisits_{it}^h)\) is the natural logarithm of the rate of emergency visits by unit of observation \(i\) (which is again composed of LSOA, age group and gender) and \(t\) and \(h\) still represent quarterly time and one of the health endpoints (nervous, circulatory, respiratory), respectively. The term \(ELvisits_{it}^h\) corresponds to the percentage of elective visits by unit of observation \(i\). When \(close_i\) is equal to 1 the interaction term \(close_i \times ELvisits_{it}^h\) represents the rate of elective visits for the population living near an airport. When \(close_i\) is 0, it represents the population living further away from an airport. The rest of the equation is exactly as in equation (3.1), with the same weights and cluster-robust standard errors.

### 3.3 Data

#### 3.3.1 Health Outcomes

The main data used is an administrative dataset on individual hospital admissions in England: the Hospital Episode Statistics (HES). HES data is released by NHS England, formerly Health and Social Care Information Centre (HSCIC). Within HES data, this study specifically employs admitted patient care (APC) visits, also known as inpatient visits, from April 2008 to April 2014. Inpatients refer to those patients that are assigned a hospital bed either for a day case surgery or for a minimum of an overnight stay. Precisely, inpatients incidents included in our dataset can be divided into two broad categories by method of admission: elective (booked or planned) or emergency (via A&E services, via physician or consultant outpatient clinic). Within emergency and elective admissions, we select three health sections of interest: nervous, cardiovascular and respiratory. As reviewed in Chapter 2 of
this thesis, these have been suggested by the epidemiological literature on the health
effects of exposure to air and noise pollution.

Our dataset builds on hospital visits, which, in the HES language, are indicated as
“continuous inpatient spells” (CIPS). HES data is stored as one record per episode,
where an episode is a period of care under one consultant. The CIPS are comprised
of sequences of spells from the patient’s first admission to hospital to the same
patient’s final discharge at home, taking into account transfers to other hospitals as
part of the patient’s care. Although a CIPS corresponds usually to one episode, it
can as well include more than one and our measure of visits takes into account this
possibility.

The same HES dataset also carries information on individual age, gender, census unit
of residence and day of hospitalisation. The smallest unit of residence provided is
the Lower-layer Super Output Area (LSOA) for the 2001 Census in England. LSOA
boundaries are designed in a way to give some social homogeneity. As a result, the
extension of a LSOA is inversely proportional to the population density: LSOA
territories are smaller in highly populated areas and are larger in rural areas where
dwellings are sparser. Each LSOA in England is associated with a land use indicator
(i.e. urban, town, village, hamlet) and an index of multiple deprivation (IMD) that
returns the level of deprivation of the local area.

This composite dataset was then aggregated to obtain a quarterly visit rate by the
unit of observation given by age group, gender and LSOA. The four pairs of plots
of Figure 3.1 give a visual summary of the main variables. The graphs delve into
the patterns underlying the method of admission within the visits data series from
the second quarter of 2008 until the first quarter of 2014. They all show the average
quarterly visit rate per LSOA population (counted by age groups and gender) by
diagnosis category and method of admission. This rate is the health measure that
we use as the outcome variable in the current study.

---

To assemble the CIPS (i.e. the visits for this study), we use the algorithm designed by the
Centre for Health Economics at the University of York.
Figure 3.1: Average quarterly visit rates per LSOA population by method of admission and diagnosis category, with their 95% confidence intervals.
Summary Statistics of Health Data

The graphs in the first panel of Figure 3.1 plot the averages by quarter of the year. There is some fluctuations for circulatory elective admissions with a reasonably stable trend from second quarter of 2012. Although it is difficult to explain the cause of this variability, it shows time-related noise that needs to be controlled for.

Panel (b) of Figure 3.1 shows U-shaped curves that represent the relationship between age group and average admission rate by unit of observation. Unsurprisingly, admission rates are higher at the extremes, for the 0 to 4 years old and over 65 years old groups.

The third panel of Figure 3.1 looks at admissions by urban to rural indicators, with urban (to the left) denoting the most populated LSOA and hamlet (to the right) the least populated LSOA. Interestingly, there is a downward trend for both circulatory and respiratory admissions going from urban to rural areas. This is an indicator of a lower number of circulatory and respiratory visits for those people living in more rural areas. This trend does not appear to be true for nervous admissions, which have a stationary behaviour, especially for the elective class. As a result, land use does not seem to influence the admission rates for the nervous class of ailments.

The last pair of plots of Figure 3.1 separate the admission rates by gender. Female admissions are lower for all categories of admissions. For both, males and females, respiratory visits suffer an increase for emergency admissions. Consequently, gender induces some differential effects on admissions and, as common in the economics literature, it is included in the model as a control variable.

Overall these graphs constitute supporting evidence for a distinction between elective and emergency admissions, a point that is assessed using equation (3.2).
3.3.2 Proximity to Airport

To define vicinity to airports, we adopt a measure of population exposure to noise pollution. Defining exposure to noise is technically easier than assigning air pollution levels to local areas. For this reason, geographically, the areas affected by airport activities’ by-products are defined using a noise metric. However, we assume that the population within these areas is also exposed to pollutants emitted by the same source.

The indicator of exposure to noise is the noise contour, which is the boundary line that defines regions exposed to a certain level of noise. Specifically, the population living within a given $x$ noise contour is exposed to a level of noise of $x$ or higher. The metrics often used to describe these contours are $Leq$ (equivalent continuous noise level meter) and $Lden$ (level of noise averaged across the day, evening and night). The former follows all noise fluctuations for a given period of time, then it is calculated an average energy\footnote{It is not a simple arithmetic average because it measures decibels which are in a logarithmic scale.} or $Leq$ value in decibels, dB. $Lden$ is an average measured over the 24 hours period that adds noise penalties to the most sensitive hours: 5 dB are added to the levels between 1900 and 2300 hours and 10 dB are added to the levels between 2300 and 0700 hours. Given that there is a noise contour for different noise levels, the next step is to pick the right curve: how close to an airport is close enough for people to be affected by noise?

When scheduling their operations, airports in the UK need to abide by government regulations to limit the population’s exposure to noise. Traditionally, the “57 dB summer $Leq$ contour” is set as the threshold above which ‘significant community annoyance’ begins (Brooker et al., 1985). Another threshold often adopted is the “55 dB $Lden$ contours”. In Europe this latter level is used to describe highly annoyed people, which represents 10-28% of the population (European Commission, 2002; European Environment Agency, 2010). Statistically, this means that there are people...
Figure 3.2: Location of the seven busiest airports in England.

On the left, region boundaries and location of all the seven busiest airports in England. On the right, a magnified map of the region around London, with its airports and their related LSOAs. From NE going anti-clockwise: Stansted, Luton, Heathrow and Gatwick airports. Blue-shaded LSOAs touch the noise contours (solid red lines around an airport). Pink-shaded LSOAs lie within 10-30 km from the airports and are used as control areas.

Table 3.1: First seven busiest airports in England with IATA (International Air Transport Association) code and 2015 total movements, sorted by average 2015 annual passenger traffic.

<table>
<thead>
<tr>
<th>Airport name</th>
<th>IATA code</th>
<th>Flights (thousand)</th>
<th>Passengers (million)</th>
</tr>
</thead>
<tbody>
<tr>
<td>London Heathrow</td>
<td>LHR</td>
<td>469.7</td>
<td>75</td>
</tr>
<tr>
<td>London Gatwick</td>
<td>LGW</td>
<td>262.5</td>
<td>40.3</td>
</tr>
<tr>
<td>Manchester</td>
<td>MAN</td>
<td>163.6</td>
<td>23.1</td>
</tr>
<tr>
<td>London Stansted</td>
<td>STN</td>
<td>144.5</td>
<td>22.5</td>
</tr>
<tr>
<td>London Luton</td>
<td>LTN</td>
<td>85.7</td>
<td>12.3</td>
</tr>
<tr>
<td>Birmingham</td>
<td>BHX</td>
<td>89.8</td>
<td>10.2</td>
</tr>
<tr>
<td>Bristol</td>
<td>BRS</td>
<td>54.7</td>
<td>6.8</td>
</tr>
</tbody>
</table>

exposed to a lower degree of noise that feel highly annoyed and there will be people within the contour that experience less annoyance than others.

This study focuses on the noise contours around the busiest airports in England to investigate the relationship between vicinity to airport and hospital admissions. We selected the first seven busiest airports due to data availability in the regions covered by the HES data series. These are: London Heathrow, London Gatwick, Manchester, London Stansted, London Luton, Birmingham and Bristol. Figure 3.2 shows their location and Table 3.1 sorts the airports by 2015 annual passenger traffic. For these airports, we looked at noise contours around the aforementioned 55 dB
Lden or 57 dB Lday thresholds\(^5\). Unfortunately, it was not possible to find maps for the same noise levels for all airports. Therefore, we use 55 dB Lden contour maps for Manchester, Luton, Birmingham and Bristol airports. The 57 dB Lday contour maps are used for Heathrow, Gatwick and Stansted airports. We spatially interpolated noise contours to match LSOA boundaries. The LSOAs have been divided into three groups (see also Figure 3.2): (i) LSOAs within the noise threshold, which represent the ‘treated’ group (in blue); (ii) LSOAs outside the threshold noise contours, which are used as control (in pink); (iii) LSOAs in a buffer zone between ‘treated’ and controls, which we excluded from the analysis.

Summary Statistics of Geographical Data

Table 3.1 shows that there are inherent differences among airports. Between the last and the first airport in that list there is a 17 fold difference in passenger traffic. Besides this, airports hold different shares of intercontinental flights, known to be the largest and noisiest aircraft. Figure 3.3 shows that Heathrow (LHR), for instance, has the highest percentage of intercontinental flights compared to the other airports.

\[\text{Figure 3.3: Trends in intercontinental, within Europe and domestic flight shares by airport.}\]

\(^5\)It is mandatory for airports to provide noise contour maps and noise action plans every five years (UK Government, 2006).
Figure 3.4: Average quarterly visit rates per LSOA population by method of admission, diagnosis category and airport group, with their 95% confidence intervals.

This is accounted for by the noise contours, which by definition cover the area exposed to a certain level of noise. For instance, Figure 3.2 clearly shows that the noise contour (i.e. the region delimited by the red solid line) around Heathrow covers a broader area compared to the contours around the other airports.

Figure 3.4 distinguishes between admission rates by proximity to airports. Airports are divided into two groups: those around London (labeled as “London area” and including Heathrow, Gatwick, Stansted and Luton) and those far away from London (labeled as “Outside London” and comprising of Manchester, Birmingham and Bristol). This distinction is both geographical (see the map of Figure 3.2) and operational. Precisely, the London area accounts for the highest levels of air traffic in Europe and is seen as an interconnected compact network (Hess and Polak, 2006). This figure shows that the most substantial differences between the two groups are for elective circulatory admissions and for emergency respiratory admissions. While the former are higher for patients living in the area of London, the latter are higher for residents away from the region around London. This might indicate a larger incidence of chronic circulatory conditions near the capital as well as a larger incidence of acute respiratory conditions outside London.
Table 3.2: Summary statistics.

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>[SD]</th>
<th>Away</th>
<th>[SD]</th>
<th>Close</th>
<th>[SD]</th>
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<tr>
<td>LSOAs</td>
<td>10,053</td>
<td></td>
<td>9,901</td>
<td></td>
<td>152</td>
<td></td>
</tr>
<tr>
<td>Visits</td>
<td>3,437,192</td>
<td></td>
<td>3,381,897</td>
<td></td>
<td>55,295</td>
<td></td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nerv - elective</td>
<td>0.009</td>
<td>[0.012]</td>
<td>0.009</td>
<td>[0.012]</td>
<td>0.009</td>
<td>[0.008]</td>
</tr>
<tr>
<td>Nerv - emergency</td>
<td>0.008</td>
<td>[0.010]</td>
<td>0.008</td>
<td>[0.010]</td>
<td>0.008</td>
<td>[0.006]</td>
</tr>
<tr>
<td>Circ - elective</td>
<td>0.015</td>
<td>[0.138]</td>
<td>0.015</td>
<td>[0.140]</td>
<td>0.010</td>
<td>[0.008]</td>
</tr>
<tr>
<td>Circ - emergency</td>
<td>0.014</td>
<td>[0.020]</td>
<td>0.014</td>
<td>[0.020]</td>
<td>0.013</td>
<td>[0.011]</td>
</tr>
<tr>
<td>Resp - elective</td>
<td>0.008</td>
<td>[0.010]</td>
<td>0.008</td>
<td>[0.010]</td>
<td>0.008</td>
<td>[0.007]</td>
</tr>
<tr>
<td>Resp - emergency</td>
<td>0.016</td>
<td>[0.022]</td>
<td>0.016</td>
<td>[0.022]</td>
<td>0.016</td>
<td>[0.015]</td>
</tr>
<tr>
<td><strong>Covariates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 to 4 yrs</td>
<td>6.54%</td>
<td>[18.81]</td>
<td>6.53%</td>
<td>[18.81]</td>
<td>6.76%</td>
<td>[18.55]</td>
</tr>
<tr>
<td>65+ yrs</td>
<td>41.97%</td>
<td>[38.64]</td>
<td>41.97%</td>
<td>[38.65]</td>
<td>41.88%</td>
<td>[37.89]</td>
</tr>
<tr>
<td>females</td>
<td>47.38%</td>
<td>[37.45]</td>
<td>47.38%</td>
<td>[37.46]</td>
<td>47.28%</td>
<td>[36.88]</td>
</tr>
</tbody>
</table>

Table 3.3: Correlation coefficients of all indices of deprivation.

<table>
<thead>
<tr>
<th>IMD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.95</td>
<td>1</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>3</td>
<td>0.34</td>
<td>0.95</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>4</td>
<td>0.78</td>
<td>0.78</td>
<td>0.80</td>
<td>1</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>5</td>
<td>0.58</td>
<td>0.88</td>
<td>0.85</td>
<td>0.89</td>
<td>0.72</td>
<td>1</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>6</td>
<td>0.50</td>
<td>0.72</td>
<td>0.64</td>
<td>0.59</td>
<td>0.42</td>
<td>0.56</td>
<td>1</td>
<td></td>
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<tr>
<td>7</td>
<td>0.47</td>
<td>0.39</td>
<td>0.30</td>
<td>0.20</td>
<td>0.21</td>
<td>0.33</td>
<td>1</td>
<td></td>
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</tr>
<tr>
<td>8</td>
<td>0.91</td>
<td>0.95</td>
<td>0.87</td>
<td>0.75</td>
<td>0.78</td>
<td>0.64</td>
<td>0.41</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>0.90</td>
<td>0.91</td>
<td>0.83</td>
<td>0.64</td>
<td>0.81</td>
<td>0.66</td>
<td>0.41</td>
<td>0.85</td>
<td>1</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>10</td>
<td>0.73</td>
<td>0.73</td>
<td>0.72</td>
<td>0.91</td>
<td>0.66</td>
<td>0.41</td>
<td>0.19</td>
<td>0.73</td>
<td>0.62</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>0.72</td>
<td>0.73</td>
<td>0.78</td>
<td>0.93</td>
<td>0.69</td>
<td>0.35</td>
<td>0.17</td>
<td>0.67</td>
<td>0.58</td>
<td>0.73</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>-0.45</td>
<td>-0.48</td>
<td>-0.41</td>
<td>-0.21</td>
<td>-0.42</td>
<td>-0.50</td>
<td>0.11</td>
<td>-0.47</td>
<td>-0.57</td>
<td>-0.21</td>
<td>-0.17</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>0.72</td>
<td>0.70</td>
<td>0.58</td>
<td>0.40</td>
<td>0.40</td>
<td>0.51</td>
<td>0.66</td>
<td>0.60</td>
<td>0.71</td>
<td>0.76</td>
<td>0.40</td>
<td>0.34</td>
<td>-0.57</td>
</tr>
</tbody>
</table>

1 Overall Index of Multiple Deprivation
2 Income
3 Employment
4 Education, Skills and Training
5 Health Deprivation and Disability
6 Crime
7 Barriers to Housing and Services
8 Income Deprivation Affecting Children Index
9 Income Deprivation Affecting Older People
10 Children and Young People Sub-domain
11 Adult Skills Sub-domain
12 Geographical Barriers Sub-domain
13 Wider Barriers Sub-domain

Table 3.2 presents summary statistics of the main variables used in this study for all regions as well as for the two sub-regions identified: away from airports (i.e. controls) and close to airports (i.e. ‘treated’). Sample consists of approximately 3.5 million visits, of which only around 55,300 are of people living closer to airports. This is due to the smaller subsamples where approximately 9,900 LSOAs are away from airports while only 152 LSOAs are close to airports. On average, there are no sensible differences between rates of visits for the different macro-regions. A noticeable exception is given by circulatory elective visits, which shows a high degree of variability. While the visits for this class are about 30% lower in the regions close
to airports, the standard deviations of the same visit category away from airports are more than nine fold bigger than their point estimates.

The first covariate summarised in Table 3.2 is the index of multiple deprivation (IMD) decile. Although the overall IMD does not appear in equation (3.1), it helps to give an idea of the average level of deprivation of the local areas included in this study. The lower the IMD decile, the higher the level of deprivation and, hence, on average the LSOAs closer to airports have higher level of deprivation: 5.349 versus 5.506 for the LSOAs less close to airports. We observe that areas near airports have more young children (6.76% of population), less elderly people (41.88%) and fewer females (47.28%) compared to areas further away from airports (6.53%, 41.97% and 47.38% for the respective categories).

All the indices of deprivation included in equations (3.1) and (3.2) and the overall IMD are listed in Table 3.3. This table also reports their correlation coefficients. Each index captures different types of deprivation with certain indices more correlated between each other than others. For instance, the overall index of deprivation highly correlates especially with the income and employment indices (2 and 3 in Table 3.3, respectively) and the two sub-income indices (8 and 9 in Table 3.3, respectively). On the opposite, the barriers to housing and services index\(^6\) (7 in Table 3.3) has low correlation with all other indices. We excluded three of all the indices available, which describe the quality of the local environmental, both indoor and outdoor. The model of equation (3.1) was implemented including several combinations of sub-groups of the indices. All models yielded similar results.

\(^6\)The barriers to housing and services is composed of several indicators: household overcrowding, district level rate of acceptances under the homelessness provisions of the 1996 Housing Act, difficulty of access to owner-occupation, road distance to a GP surgery, road distance to a general store or supermarket, road distance to a primary school and road distance to a Post Office (see https://data.gov.uk/dataset/imd-barriers).
3.4 Results and Discussion

For the pooled samples, the results of the log-linear regression equation (3.1) are presented in Table 3.4. This table reports the estimates of the coefficient of interest $\delta_{lh}$ in the same equation (3.1)$^7$. We control for time effects and demographic effects which represent indicators for year quartile and demographic characteristics (i.e. age group and gender), i.e. $demographic_i$ and $quartile_i$ in equation (3.1), respectively. Socio-geographic effects summarise the contribution of the vector of controls $X_i$ at the LSOA level. As specified in the previous section, these controls are the indicators for the level of rural or urban land of local area, the indicators for the broader government regions and the indicators for the deciles of the local IMD indices.

Table 3.4: Relationship between living near airports (i.e. within 55 Lden or 57 Lday noise contours) and hospital admission rate by admission type.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Elective</th>
<th></th>
<th>Emergency</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nervous</td>
<td>Circulatory</td>
<td>Respiratory</td>
<td>Nervous</td>
</tr>
<tr>
<td>All airports</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>0.055**</td>
<td>-0.038***</td>
<td>-0.023</td>
<td>0.017</td>
</tr>
<tr>
<td></td>
<td>(0.027)</td>
<td>(0.013)</td>
<td>(0.017)</td>
<td>(0.017)</td>
</tr>
<tr>
<td>Time effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Demographic effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Socio-geog. effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Observations</td>
<td>246,305</td>
<td>469,485</td>
<td>244,231</td>
<td>188,262</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.410</td>
<td>0.552</td>
<td>0.629</td>
<td>0.634</td>
</tr>
</tbody>
</table>

| London area       |          |          |           |          |          |           |
| close to airport  | 0.019    | -0.059***| -0.082*** | -0.032   | -0.023   | -0.031    |
|                   | (0.034)  | (0.018)  | (0.026)   | (0.026)  | (0.018)  | (0.021)   |
| Time effects      | ✓        | ✓        | ✓         | ✓        | ✓        | ✓         |
| Demographic effects | ✓        | ✓        | ✓         | ✓        | ✓        | ✓         |
| Socio-geog. effects | ✓        | ✓        | ✓         | ✓        | ✓        | ✓         |
| Observations      | 153,517  | 270,049  | 145,578   | 100,790  | 298,440   | 382,670    |
| Adjusted $R^2$    | 0.443    | 0.608    | 0.650     | 0.678    | 0.702     | 0.703      |

| Outside London    |          |          |           |          |          |           |
| close to airport  | 0.132*** | -0.015   | 0.062***  | 0.073*** | 0.064*** | 0.079***   |
|                   | (0.042)  | (0.016)  | (0.014)   | (0.019)  | (0.014)  | (0.016)    |
| Time effects      | ✓        | ✓        | ✓         | ✓        | ✓        | ✓         |
| Demographic effects | ✓        | ✓        | ✓         | ✓        | ✓        | ✓         |
| Socio-geog. effects | ✓        | ✓        | ✓         | ✓        | ✓        | ✓         |
| Observations      | 92,788   | 199,436  | 98,653    | 87,472   | 236,956   | 329,152    |
| Adjusted $R^2$    | 0.358    | 0.476    | 0.605     | 0.583    | 0.622     | 0.634      |

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.

$^7$The equation being log-linear, its estimated coefficient $\delta$ can be interpreted as the $\exp(\delta) - 1 \times 100$ percentage change in the outcome variable given a 1-unit change in the covariate of interest, which in this case corresponds to the effects on the population living near airports. Table 3.A4, Table 3.A5 and Table 3.A6 in the Appendix provide the full set of estimates for the main specification, by region.
Given the well known negative correlation between environmental exposure and health, we would expect the rates of admission for relevant categories to increase for those people living close to airports. For the nervous class, there is indeed a statistically significant increase by around 5.5% for elective visits (column 1). Also for the respiratory class (column 6) there is a significant increase of 2.3 percentage points in emergency visits. However, living close to an airport appears to have a negative effect on circulatory elective admissions (column 2), with a 3.8% decrease in visits. This negative result for planned visits raises concerns over its interpretation. It is not clear how vicinity to airport could actually decrease elective hospitalisations. The model explained by equation (3.2) provides some plausible explanation that will be discussed later in this section.

Another feature detected in Table 3.4 is that while all statistically significant results are positive for outside London areas, they are negative for the estimates of London airports. The next Subsection 3.4.1 provides more detail on the interpretation of these contradicting results.

In addition, all statistically significant negative results of Table 3.4 concentrate in the elective type of admissions, i.e. in the first three columns. The pattern seems to suggest a migration of visits from elective to emergency admissions. We test for this hypothesis estimating equation (3.2), for which we presents the results in Table 3.5.

Table 3.5 shows the parameter estimates of the coefficient $\delta$ of the interaction term $close_i \times ELvisits_{it}$ of equation (3.2). This term can represent elective visits for patients that live either close or further away from an airport, depending on whether the unit of observation $i$ pertains to the area close to an airport (when $close_i$ is equal to 1) or to the area further away (when $close_i$ is equal to 0), respectively.

Column 1 of Table 3.5 reports that a 1% change in nervous elective visits corresponds to a 12.2 percentage change in the same emergency visits for patients living close to airports and to a 13.1 percent change for those living further away from airports. For circulatory conditions the difference in changes across airport proximity groups
Table 3.5: Substitution effect between elective and emergency admissions.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Emergency visits rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nervous (1)</td>
</tr>
<tr>
<td>Elective vis (%) close to airport</td>
<td>0.115***</td>
</tr>
<tr>
<td></td>
<td>(0.028)</td>
</tr>
<tr>
<td>Elective vis (%) away from airport</td>
<td>0.123***</td>
</tr>
<tr>
<td></td>
<td>(0.025)</td>
</tr>
<tr>
<td>Time effects</td>
<td>✓</td>
</tr>
<tr>
<td>Demographic effects</td>
<td>✓</td>
</tr>
<tr>
<td>Socio-geog. effects</td>
<td>✓</td>
</tr>
<tr>
<td>Observations</td>
<td>31,881</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.581</td>
</tr>
</tbody>
</table>

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.

is larger, with around 6.2% and 1.0% increases in emergency visits for patients living close or away from airports, respectively, correlating with a 1 percentage point increase for elective admissions. The most marked results belong to the respiratory category, where a 1% change in elective visits is associated with about 35.7% increase in emergency admissions close to airports against a 25.2% increase away from airports.

To summarise, our results appear to suggest that patients in our sample have a preference for emergency admissions over elective. In particular, near airports this preference is stronger for circulatory and respiratory conditions. Given the decreases in elective hospital admissions for patients close to airports, this behaviour might be explained by a differential effect of environmental exposure on access to health with some migration of admissions from planned to emergency for circulatory and respiratory conditions. We will come back to this issue in Chapter 5.

3.4.1 Results by Subsample

Due to the large number of subsamples (by diagnosis category as well as by airport group, age group, gender and land use category), there is a series of estimates of the coefficient of interest, $\delta_{th}$, in equation (3.1). To give a visual summary of the results of all these regression equations, the two plots in Figure 3.5 (one for elective and one for emergency visits) show all statistically significant coefficients (below the 10%, 5%
Chapter 3. Living in the Proximity of Airports and Health

(a) Elective admissions.

(b) Emergency admissions.

Figure 3.5: Statistically significant estimates of $\delta_{lh}$ in equation (3.1) by subsample.
and 1% significance levels), where the missing information indicates non-significant results. Three tables in Appendix 3.A report all estimates for all subsamples: Table 3.A1 reports estimates for the gender subsamples, Table 3.A2 illustrates the results for the rural and urban sub-groups, and Table 3.A3 shows the results by different age groups.

Plot (a) of Figure 3.5 for elective visits shows that the points are scattered above and below the zero line, suggesting a mixed effect of environmental exposure. Going from elective to emergency admissions (i.e. from plot (a) to plot (b)), the estimates show several patterns.

Looking at all regions, for elective admissions there is a distinction in configuration between nervous and the other two diagnosis groups. While nervous conditions are predominantly characterised by positive estimates, the estimates of the other groups are exclusively negative. This situation changes when moving to graph (b), where only emergency estimates are represented. In this case, excluding the subsample of young children, the effect of adverse environmental exposure on nervous visits disappears. Due to vicinity to airports, elective circulatory visits suffer a decrease in admissions. The same happens to the young children subsample for emergency visits. However, males and patients from urban areas increase their emergency circulatory visits if living close to airports compared to those from the same categories but living further away. Respiratory estimates have a mixed allocation in the emergency graph. Although they remain negative for the subsample of young children, they are in the positive quadrant for the overall sample and the urban subsample.

The patterns change if we select areas close to London (the middle graphs of both plots, in panel (a) and in panel (b) of Figure 3.5). For this subsample, overall, vicinity to airports and rates of visits for elective circulatory and respiratory admissions move in opposite direction. However, one notable exception is observed in the elderly subsample. Around London airports, the population aged over 65 show positive estimates for elective nervous visits as well as emergency circulatory visits.
Those areas close to airports outside the London area (i.e. Manchester, Birmingham and Bristol) are characterised by a completely different scenario. This is illustrated by the third graph in panels (a) and (b) of Figure 3.5. For both elective and emergency admissions, the estimates concentrate in the positive half of the plots with a couple of notable departures from this pattern. The two age group subsamples for the over 50 year-olds exhibit negative estimates for the elective circulatory class. The 50 to 64 subsample equally experiences a decrease in elective admissions for respiratory conditions, whereas the over 65 subsample is characterised by an increase in elective admissions for nervous reasons.

Vicinity to airports appears to affect the two broad regions differentially. The areas around London are generally negatively affected, while there is a consistent positive increase in visits for the three conditions of interest across the majority of subsamples in the areas outside London. These apparently contradicting results raise questions on the validity of the model represented by equation (3.1). The adjusted $R^2$ for all fitted models are generally high, which suggest that the covariates explain reasonably well the variation detected in the outcome. However, there are potential endogeneity problems in identifying the effect of proximity to airports and hospital admissions.

There might be unobserved unit of analysis characteristics that influence both airport proximity and hospital admission rates. For instance, regarding distance to the closest hospital, there might be a conflicted scenario for the areas around London compared to those in the rest of England. A possible situation is that around London there are more hospitals in the “control” group (i.e. further away from airports) rather than in the “treatment” group and, on the contrary, in the other areas hospitals could have a higher density around airports. This would motivate patients outside London and closer to hospital to exploit hospital facilities more often than they analogous patients near airports in the London area. In this case, the patient’s distance from the hospital could act as a confounder in the relationship between proximity to airport and hospitalisations. We address this issue looking at hospital positions and

\footnote{Propper et al. (2007) show that distance to hospital matters differently for elective and
we see that they are predominantly located in the “control” areas across England. We conclude that this factor does not affect the estimates differently.

The interpretation of these results remains difficult. Our model seems to have some omitted variables that confound the health effects of by-products of airports. In the next chapters, we take different approaches to explore this issue and try to measure the causal health effects of environmental exposure.

### 3.5 Conclusion

We provide the first estimates of the effects of proximity to airports on several types of hospital admissions. For the circulatory and respiratory classes, we show that exposure to airport emissions augments the rates of emergency admissions compared to rates of elective hospitalisations. This suggests a migration from elective to emergency admissions for patients living near airports. Surprisingly, the results show a different response for the nervous class, which is characterised by an increase of admissions for elective visits versus a decrease for emergency admissions.

Overall, in the areas around non-London airports, proximity to airport increases all visits rates. However, around London airports our results show statistically significant negative estimates for the three classes of interest: nervous, circulatory and respiratory.

These results raise questions over either the validity of the model or its interpretation. A number of data issues need to be considered. First, we cannot measure the length of personal exposure. Although HES data provides a patient identifier which allows to track the same patient over time, we decide to aggregate the quarterly number of visits by unit of observation. This feature of the hospital data gives the residence of emergency admissions. In fact, patients are more likely to travel longer distances for planned visits compared to emergency (and maternity). The same study demonstrates that the level of deprivation affects the distance travelled, where patients coming from most deprived areas tend to travel shorter distances for their elective care (Propper et al., 2007). To account for this confounding factor, both our models control for the local area deprivation level.
the patients at the time of the visit, but we do not know when the patient moved there and for how long he has lived at the same address, therefore we are not able to exploit this information. Second, we use simple OLS, which does not rule out the presence of unobserved unit of aggregation characteristics that could influence both airport proximity and hospital admission rates. We briefly take into consideration that distance to hospital might be one these factors. However, the homogeneous distribution of (NHS) healthcare facilities does not seem to influence the heterogeneous results across regions and type of admissions. Third, proximity to airport can be considered an endogenous variable. Therefore, patients’ residence might well affect their health outcomes through other channels that are not taken into account by our model.

In order to disentangle these issues, the next Chapter 4 and Chapter 5 adopt strategies that investigate the causality of the relationship between environment exposure and health.
### 3.A Appendix

#### Table 3.A1: Relationship between living near airports and hospital admission rates by gender and method of admission.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Elective</th>
<th></th>
<th></th>
<th>Emergency</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nervous (1)</td>
<td>Circulatory (2)</td>
<td>Respiratory (3)</td>
<td>Nervous (4)</td>
<td>Circulatory (5)</td>
<td>Respiratory (6)</td>
</tr>
<tr>
<td>1. Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All airports</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>0.042</td>
<td>-0.032**</td>
<td>-0.045*</td>
<td>0.006</td>
<td>0.002</td>
<td>0.024</td>
</tr>
<tr>
<td>Observations</td>
<td>113857</td>
<td>268972</td>
<td>130160</td>
<td>89913</td>
<td>295715</td>
<td>364080</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.470</td>
<td>0.581</td>
<td>0.647</td>
<td>0.674</td>
<td>0.690</td>
<td>0.699</td>
</tr>
<tr>
<td>London area</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>-0.029</td>
<td>-0.058***</td>
<td>-0.112***</td>
<td>-0.049</td>
<td>-0.044*</td>
<td>-0.039</td>
</tr>
<tr>
<td>Observations</td>
<td>72772</td>
<td>156083</td>
<td>77439</td>
<td>48102</td>
<td>165762</td>
<td>196507</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.505</td>
<td>0.633</td>
<td>0.671</td>
<td>0.715</td>
<td>0.721</td>
<td>0.726</td>
</tr>
<tr>
<td>Outside London</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>0.177***</td>
<td>-0.001</td>
<td>0.057***</td>
<td>0.076**</td>
<td>0.061***</td>
<td>0.087***</td>
</tr>
<tr>
<td>Observations</td>
<td>41085</td>
<td>112889</td>
<td>52721</td>
<td>41811</td>
<td>129953</td>
<td>167573</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.413</td>
<td>0.510</td>
<td>0.623</td>
<td>0.631</td>
<td>0.648</td>
<td>0.666</td>
</tr>
<tr>
<td>2. Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All airports</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>0.060*</td>
<td>-0.047***</td>
<td>0.002</td>
<td>0.025</td>
<td>0.029*</td>
<td>0.022</td>
</tr>
<tr>
<td>Observations</td>
<td>132448</td>
<td>112889</td>
<td>52721</td>
<td>41811</td>
<td>129953</td>
<td>167573</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.359</td>
<td>0.500</td>
<td>0.608</td>
<td>0.594</td>
<td>0.637</td>
<td>0.645</td>
</tr>
<tr>
<td>London area</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>0.051</td>
<td>-0.059***</td>
<td>-0.044</td>
<td>-0.013</td>
<td>0.003</td>
<td>-0.023</td>
</tr>
<tr>
<td>Observations</td>
<td>80745</td>
<td>113966</td>
<td>68139</td>
<td>52688</td>
<td>132678</td>
<td>186163</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.390</td>
<td>0.561</td>
<td>0.629</td>
<td>0.644</td>
<td>0.675</td>
<td>0.679</td>
</tr>
<tr>
<td>Outside London</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>close to airport</td>
<td>0.102*</td>
<td>-0.032</td>
<td>0.070***</td>
<td>0.070***</td>
<td>0.065***</td>
<td>0.071***</td>
</tr>
<tr>
<td>Observations</td>
<td>51703</td>
<td>86547</td>
<td>45932</td>
<td>45661</td>
<td>107003</td>
<td>161579</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.321</td>
<td>0.416</td>
<td>0.586</td>
<td>0.534</td>
<td>0.589</td>
<td>0.600</td>
</tr>
</tbody>
</table>

For all models:
- Time effects: ✓ ✓ ✓ ✓ ✓ ✓
- Demographic effects: ✓ ✓ ✓ ✓ ✓ ✓
- Socio-geog. effects: ✓ ✓ ✓ ✓ ✓ ✓

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
Table 3.A2: Relationship between living near airports and hospital admission rates, by rural/urban geography and method of admission.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Elective</th>
<th>Emergency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nervous (1)</td>
<td>Circulatory (2)</td>
</tr>
<tr>
<td>1. Rural</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All airports close to airport</td>
<td>0.002</td>
<td>-0.071**</td>
</tr>
<tr>
<td></td>
<td>(0.047)</td>
<td>(0.028)</td>
</tr>
<tr>
<td>Observations</td>
<td>87908</td>
<td>161692</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.325</td>
<td>0.481</td>
</tr>
<tr>
<td>London area close to airport</td>
<td>-0.030</td>
<td>-0.083***</td>
</tr>
<tr>
<td></td>
<td>(0.057)</td>
<td>(0.029)</td>
</tr>
<tr>
<td>Observations</td>
<td>10545</td>
<td>87929</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.345</td>
<td>0.545</td>
</tr>
<tr>
<td>Outside London close to airport</td>
<td>0.082</td>
<td>-0.058</td>
</tr>
<tr>
<td></td>
<td>(0.092)</td>
<td>(0.046)</td>
</tr>
<tr>
<td>Observations</td>
<td>38363</td>
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<td>Adjusted $R^2$</td>
<td>0.327</td>
<td>0.428</td>
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<td>2. Urban</td>
<td></td>
<td></td>
</tr>
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<td>All airports close to airport</td>
<td>0.077**</td>
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<td>(0.033)</td>
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<tr>
<td>Observations</td>
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<td>Adjusted $R^2$</td>
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<tr>
<td>London area close to airport</td>
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<td></td>
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<td>(0.017)</td>
</tr>
<tr>
<td>Observations</td>
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</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.488</td>
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</tr>
<tr>
<td>Outside London close to airport</td>
<td>0.163***</td>
<td>-0.007</td>
</tr>
<tr>
<td></td>
<td>(0.048)</td>
<td>(0.018)</td>
</tr>
<tr>
<td>Observations</td>
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<td>125673</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.404</td>
<td>0.509</td>
</tr>
</tbody>
</table>

For all models

<table>
<thead>
<tr>
<th></th>
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<th>✓</th>
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<tr>
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Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.

Table 3.A3: Health impacts of living near airports, by age group.

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<tr>
<th>Variable</th>
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<td></td>
<td>Nervous (1)</td>
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</tr>
<tr>
<td>1. Age group: 0 to 4 years old</td>
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<tr>
<td>All airports close to airport</td>
<td>-0.118**</td>
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<tr>
<td>Observations</td>
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<tr>
<td>Adjusted $R^2$</td>
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<tr>
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<td>(0.138)</td>
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<td>Adjusted $R^2$</td>
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Continued on next page
## Chapter 3. Living in the Proximity of Airports and Health

### 2. Age group: 5 to 19 years old

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**All airports**

- close to airport: -0.063, -0.137, -0.036, 0.023, -0.129, 0.004
- Observations: 20428, 6139, 53141, 15962, 7386, 66098
- Adjusted R²: 0.187, 0.215, 0.189, 0.228, 0.340, 0.185

**London area**

- close to airport: -0.178, -0.395*, -0.150*, -0.089**, -0.319***, -0.211**
- Observations: 11581, 3473, 30368, 8260, 4168, 34360
- Adjusted R²: 0.180, 0.206, 0.152, 0.184, 0.275, 0.164

**Outside London**

- close to airport: 0.096*, 0.054, 0.086***, 0.065*, 0.071, 0.137***
- Observations: 8847, 2666, 22773, 7702, 3218, 31738
- Adjusted R²: 0.248, 0.313, 0.285, 0.321, 0.463, 0.259

### 3. Age group: 20 to 49 years old

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<td>0.248</td>
<td>0.269</td>
<td>0.255</td>
<td>0.217</td>
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**All airports**

- close to airport: 0.070*, -0.030*, -0.030, 0.009, -0.007, 0.010
- Observations: 87537, 118280, 83825, 58180, 84869, 119443
- Adjusted R²: 0.135, 0.168, 0.248, 0.269, 0.255, 0.217

**London area**

- close to airport: 0.032, -0.111***, -0.096***, -0.071**, -0.097***, -0.049**
- Observations: 55777, 66940, 50616, 31532, 46722, 67379
- Adjusted R²: 0.134, 0.142, 0.238, 0.256, 0.251, 0.200

**Outside London**

- close to airport: 0.142**, 0.046*, 0.075***, 0.087***, 0.090***, 0.076***
- Observations: 31760, 51340, 33209, 26648, 38147, 52064
- Adjusted R²: 0.136, 0.163, 0.263, 0.250, 0.232, 0.194

### 4. Age group: 50 to 64 years old

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<td>0.209</td>
<td>0.285</td>
<td>0.206</td>
<td>0.218</td>
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**All airports**

- close to airport: -0.030, -0.059***, -0.065***, -0.028, -0.003, -0.019
- Observations: 68436, 146411, 40774, 34307, 132921, 103291
- Adjusted R²: 0.119, 0.143, 0.209, 0.285, 0.206, 0.218

**London area**

- close to airport: -0.049, -0.047***, -0.097***, -0.036, -0.013, -0.019
- Observations: 43156, 82817, 24124, 18056, 72379, 54561
- Adjusted R²: 0.127, 0.165, 0.201, 0.304, 0.223, 0.234

**Outside London**

- close to airport: 0.012, -0.069***, -0.040**, -0.017, 0.009, -0.019
- Observations: 25280, 63594, 16650, 16251, 60542, 48730
- Adjusted R²: 0.122, 0.127, 0.219, 0.278, 0.198, 0.214

Continued on next page
## Chapter 3. Living in the Proximity of Airports and Health

### 5. Age group: over 65 years old

#### All airports

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<th>Nervous</th>
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<th>Respiratory</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
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<td>0.084*</td>
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#### London area

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<td>Observations</td>
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#### Outside London

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**Notes:** *p < .1, **p < .05, ***p < .01. Cluster-robust standard errors in parentheses.

### Time effects
- ✓ = included
- ✗ = excluded

### Demographic effects
- ✓ = included
- ✗ = excluded

### Socio-geog. effects
- ✓ = included
- ✗ = excluded

### Table 3.A4: Health impacts of living near airports, all regions.

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<tr>
<td>2011 Q2</td>
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<tr>
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Chapter 3. Living in the Proximity of Airports and Health

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## Chapter 3. Living in the Proximity of Airports and Health

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Notes: Sub-d: Sub-domain. * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
Table 3.A5: Health impacts of living near airports, around London.

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Chapter 3. Living in the Proximity of Airports and Health

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### Chapter 3. Living in the Proximity of Airports and Health

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### Chapter 3. Living in the Proximity of Airports and Health

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Table 3.A6: Health impacts of living near airports, outside London.

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### Chapter 3. Living in the Proximity of Airports and Health

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### Chapter 3. Living in the Proximity of Airports and Health

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</table>

Notes: Sub-d: Sub-domain. * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
Chapter 4

The Medical Cost of Air Traffic Pollution: Evidence from Changes in Flight Patterns

SUMMARY. This chapter investigates health externalities generated by air transportation. As a source of exogenous variation, we use an unannounced five-month trial that changed early morning patterns of aircraft landings at London Heathrow airport. We observe that prescribed medication usage significantly correlates with air traffic in four local regions around the airport. Compared to the control regions, we observe a significant and substantial decrease in prescribed drugs for respiratory and central nervous system conditions in the two areas subject to a reduction in air traffic. Our findings suggest a causal adverse impact of air traffic on health conditions.

4.1 Introduction

The study of the relationship between environmental exposure and its adverse health effects is a well-documented field in environmental, epidemiological and medical
research. This chapter contributes to the still limited, but growing, field of studies using exogenous variation to investigate the causal effect of pollution on health (see Cesur et al., 2017; Deryugina et al., 2016 for recent examples, and Graff Zivin and Neidell, 2013 for a review).

We present new evidence on the health impact of airports as major sources of air and noise pollution (Wolfe et al., 2017; Schlenker and Walker, 2016). Specifically, we focus on prescription drugs in regions exposed to a change in patterns of plane landings around a global aviation hub, located within a large metropolitan area, London Heathrow airport. We make use of a trial implemented over five months (between November 2012 and March 2013) that redirected approaching aircraft to reduce early morning noise in designated areas. This trial had the critical and unique feature of occurring at daybreak, between 4:30 am and 6:00 am, when targeted residents are most likely to be at home and therefore exposed to the full impact of the changed flight paths.

We select local areas differentially affected by changes in air traffic during the trial. By exploiting the systematic changes induced by the trial, we implement a difference-in-differences approach using communities not affected as control group. Our main measure of a health effect is the use of prescribed drugs for the five months of the trial, compared to the same outcome in earlier periods. We focus on three broad types of diseases that, as suggested by the medical literature, are aggravated by environmental pollution: central nervous, circulatory and respiratory conditions.

Our main contribution to the literature is establishing new and concrete results linking medical conditions to air and noise pollution exposure in a causal framework. We do so by exploiting unique context and data.

First, the nature of the trial allows to overcome avoidance behaviours - people may rationally avoid places exposed to increased pollution - that plague earlier literature. The trial has the unique feature of only applying to people at home before sunrise. Second, by using data on medicines prescribed by doctors to their patients, we infer
direct evidence on health conditions rather than relying on self-reported health.

Finally, by quantifying the impact of airports on medicines usage, the chapter adds to the burgeoning literature trying to credibly estimate the impacts of transport congestion locations on air pollution and health outcomes using natural experiments, such as for airports (Schlenker and Walker, 2016), ports (Moretti and Neidell, 2011), tollbooths and traffic congestion (Currie and Walker, 2011).

Overall, during the trial there was a decrease in monthly prescription expenditure on central nervous system and respiratory medication by 5.8% and 3.3% respectively\(^1\). These results are more pronounced for areas most affected by the changing flight patterns. We find only weak effects for cardiovascular diseases.

We test the main results by checking whether similar prescription changes happened for other diseases known to be unrelated to air and noise pollution (infections and musculoskeletal conditions). We cannot detect any significant changes over the same period. The results are also robust to changes in the time periods chosen for both the trial and the control group, especially for central nervous conditions.

We further submit our initial results to a series of additional robustness tests, which confirm our findings. Our results, by suggesting a causal link between air traffic and health, also have financial implications for health spending. Back-of-the-envelope computations suggest a potential annual saving of 5 millions GBP (6.3 millions USD) for prescribed medicines in respiratory and nervous conditions, in the areas most affected by changes in air traffic.

This chapter is structured as follows. The next Section 4.2 gives some background information on airports, noise, air quality and health. Section 4.3 describes the institutional setting and the empirical strategy of our research and Section 4.4 describes the data. Section 4.5 presents and discusses the results. Section 4.6

\(^1\)In Britain medical prescriptions are subsidised by the National Health Service (NHS) and arise from visits to physicians, known as general practitioner (GP) doctors. This is in contrast to the reimbursement systems that occur in countries with medical insurance schemes.
summarises the findings and discusses implications for policy.

4.2 Noise, Air Quality, Health and Airports

4.2.1 Air and Health

The pathways that link pollution to health outcomes, which may comprise a mixture of several agents and processes, are not clearly understood. But we do understand, from the clinical and medical literature, some of the mechanisms of interaction between each single air pollutant and the human body. Generally, air pollutants have the common property of activating oxidative stress that causes cell damage and death. More specifically, there is strong evidence of symptom exacerbations of cardiovascular diseases such as arrhythmia and myocardial infarction, as well as asthma and other respiratory diseases, transient worsening in lung function, and increased respiratory infections, which all result in more visits to general medical practitioners (GPs, i.e. physicians in the US) and hospitals (Brunekreef and Holgate, 2002; Li et al., 2012). Furthermore, the combination of air pollutants, rather than their separate action, seems to play a large part in the adverse interaction with human health (WHO, 2013).

Health effects of pollution can be categorised as resulting from short or long-term exposure. In the former case, the events are known as acute health effects or effects of daily variations in pollution. These consist of the impact of air pollution levels on a given day on the health of people on that same day or on the following days - usually no more than seven. In the latter case, the events are known as chronic health effects and partially include the acute effects, measured as changes in mortality rates.

A recent WHO review of health aspects of air pollution (WHO, 2013) provides evidence that short and long-term adverse effects of particulate matters, ozone and NO$_2$ can occur at concentrations lower than those set by official guidelines. In
fact, even in the UK, a country where the current levels of pollutants are generally low, it has been shown that mortality rates respond to pollution variation at any concentration level (Janke et al., 2009).

As we discussed in Chapter 2, individual behaviour can confound the causal link between exposure and health, for instance by moving home, deciding to stay indoor or changing mode of transport. The lack of random assignment to different environmental exposure weakens the results reported in the epidemiology research. Economics literature suggests empirical approaches to overcome this issue. For instance, Currie et al. (2009) look at how exposure to air pollution explain school absenteeism variation adopting an econometric strategy which allows to address potentially confounding factors. Although employing different variables, this study uses a similar research design.

4.2.2 Noise and Health

There is strong evidence that noise, defined as undesirable sound, impinges on human health. Among the adverse effects of this stressor, we focus on those non-auditory effects - i.e. those health effects other than tinnitus and hearing loss, triggered by environmental noise. In their recent review, Basner et al. (2014) identified several outcomes: annoyance, cardiovascular disease, cognitive impairment and sleep disturbance. People react to various levels of noise when it interferes with daily activities or sleep. They experience a range of effects of varying severity, from anger, displeasure and exhaustion to stress-related symptoms.

Both, laboratory and field studies, suggest two biological pathways for the relationship between noise and circulatory system diseases: a direct and an indirect pathway. Both pathways relate to stress hormone reactions. The former predominantly occurs during sleep and results in a non-conscious physiological stress from interactions between the central auditory system and areas of the central nervous system. The indirect pathway refers to the combined effect of the noise level and its provenance
on the degree of annoyance. The relative perceived discomfort then triggers stress hormone response.

There are many epidemiological studies drawing on large administrative sources of health outcomes to investigate the effects of noise on health. For example, two different studies similarly looked at the effects of noise around airports on cardiovascular hospital admissions rate (Correia et al., 2013b; Hansell et al., 2013).

Correia et al. (2013b) adopted a multi-airport approach, linking noise contours of 89 US airports with hospital admission rates. Looking at a specific sensitive group, the elderly, they found an increase of 3.5% in circulatory admission rates associated with an increase of 10 dB noise exposure. Hansell et al. (2013) focused on the Heathrow airport region specifically. They found that exposure to higher noise levels increased mortality and the prevalence of strokes, coronary heart disease and cardiovascular disease for both hospital admissions and mortality.

In a recent review paper the focus was on nervous conditions (Tzivian et al., 2015). Tzivian and colleagues selected studies on mental health effects of exposure to air and noise pollution and reported a positive association with anxiety, depression and impaired activities of daily living, among other outcomes.

Although these cross-sectional studies control for some of the confounding factors that could be associated with the relevant outcomes, such as socio-economic status and individual overall health conditions, they do not unequivocally prove causation between environmental factors and health. For example, they assume that exposure to noise happens mainly at the individual’s home address. However, a large proportion of the population spend most of their day outside their home, thus raising problems of exposure bias. Economists have adopted quasi-experimental techniques to tackle some of these issues (Graff Zivin and Neidell, 2013).

Many economic studies exploit exogenous shocks to emissions to estimate the related

\[\text{Noise exposure is measured in decibels (dB), a logarithm scale that ranks noise pressure levels and its metric is the Equivalent Continuous Sound Level, or Leq.}\]
health effects. However, these typically focus on air pollution levels (Currie and Walker, 2011; Beatty and Shimshack, 2011; Schlenker and Walker, 2016 among others). A notable exception is a study on exposure to noise around Zurich airport (Boes et al., 2013). Using an individual fixed-effects strategy and change in flights regime, they found that daytime exposure to an increase in aircraft noise (measured yearly) significantly affects self-reported sleeping problems and headaches episodes.

The use of quasi-experimental evidence published thus far have focused on the causal relationship between air quality and a series of health outcomes, from low birth weight and asthma episodes to mortality. Our project adds to this literature by including the impact of the combined action of air and noise pollution.

This chapter specifically focuses on London Heathrow airport activities. In addition to being exposed to increased levels of air pollution, communities located near major airports such as Heathrow also suffer increased exposure to noise pollution, which can have similarly negative impacts on health.

In the UK the Civil Aviation Authority (CAA) on behalf of the Department for Transport produces noise contours maps to estimate the size of the areas subject to different noise levels (Lee et al., 2014). For Heathrow airport, Lee et al. (2014) calculated that in 2013 about 266,000 and 421,000 people were exposed to significant community annoyance. The large number of residents affected is due to the proximity of Heathrow to a highly urbanised area. In fact, Heathrow lies within the boundaries of Greater London (an unusual location for a major international hub)\(^3\).

\(^3\)The initial location was chosen for military purpose during WWII, without foreseeing its expansion into the one of the world’s top four busiest civilian airports (from "The History of Heathrow", The Independent, 1 March 2011.)
4.3 Method

4.3.1 Institutional Setting

In order to address its noise externalities, Heathrow airport explores ways to reduce them through a number of adjustments and measures. For instance, it encourages the use of quieter planes especially during sensitive hours, promotes quieter operating procedures and working with local communities it provides individual home insulation (Heathrow Airport Limited, 2013). The Early Morning Arrivals Trial (EMAT) in 2012 and 2013 was introduced to provide noise respite to specific communities living near Heathrow airport.

Our analysis focuses on this intervention. During five months, from 5th November 2012 to 31st March 2013, Heathrow airport ran the trial in collaboration with the noise pressure group HACAN (Heathrow Association for the Control of Aircraft Noise), British Airways and NATS (formerly National Air Traffic Services). The main feature of the trial was the identification of four pairs of exclusion zones (two to the east and two to the west of Heathrow), which were designed to be free of aircraft movements during the night and early morning in alternate weeks for the duration of the trial, redirecting the night flights to other areas (Tucker et al., 2013).

Night quota restrictions reduce landings at Heathrow between 11:30 pm and 6:00 am. However airlines, responding to travellers preferences for early morning landings, allocate nearly all those landing slots between 4:30 am to 6:00 am. This pattern translates into one aircraft landing every four to ten minutes during those crucial 90 minutes when sleep is likely to be disrupted. In addition these early morning landings are typically transcontinental large bodied jets which are noisier than the average landing at other times of the day.

Figure 4.A1 and Figure 4.A2 in the Appendix allow for a visual inspection of the flight tracks, which compares the baseline time span (November 2011 to March 2012)
to the trial period (November 2012 to March 2013). It also identifies five geographical zones in the Greater London (GL) area experiencing varied exposure as a result of the trial. We label them as follows: GLW1 and GLW2 to the west of Heathrow and GLE1, GLE2 and GLE3 to the east of the airport. The average height of the areas is 10 miles and the average width is 5 miles.

There is considerable variation in the exposure to early morning aircraft noise for affected sub-populations. For instance, the region called GLE2 was free of flights during its exclusion weeks; it was overflown more on the other weeks but overall experienced a reduction.

Our data on prescriptions being available on a monthly basis only, we need to rely on the combined total monthly early morning flights. One climate related characteristic helps us, however, to build prior expectations of the magnitude and direction of exposure. Aircraft have to land into the wind when their speed exceeds 5 knots. In South East England 70% of the year the wind direction is west to east. This little known pattern implies that, as opposed to the intended regular planned alternation between landing from the west one week and the east the following, more than 70% of planes typically land flying over central London (from the east). The first four months of our trial did follow this pattern: between 4.30 am and 6.00 am, 1,300 planes landed flying over central London and 559 landed from the west. The effect of the tracks modifications during the trial is therefore much more likely to be noticeable on overflown residents living to the east of the airport.

We chose two areas grouped together as control group. These correspond to regions to the north and south of Heathrow, outside of the approach path corridor, bounded to the west and to the east by the regions involved in the trial (drawn as trapeziums on Figure 4.1). Residents in the control areas have remained unaffected by changes in air traffic throughout the trial period.

Residential sorting does not seem to be an issue within this setting thanks to two inherent attributes of the trial. The first is suggested by the name of the trial: the
Early Morning Arrivals Trial. We assume that most people are at home between 4.30 am and 6.00 am and are in light sleep hours where deep sleep is infrequent\(^4\). Secondly, no advance notification about the start of the trial was given to residents (Tucker et al., 2013). The organisations involved decided to communicate the implementation of the change only after the first week of the on-going trial, and then to collect feedback from residents through media and meetings. Therefore, it is unlikely that people relocated due to this unexpected temporary change.

### 4.3.2 Empirical Strategy

The goal of this chapter is to assess the impact of changes in aircraft emissions on health outcomes for those people living underneath flight paths. To simultaneously isolate causal effects of the flight changes and control for confounding factors, we explore GP prescribing differences between communities that experienced the flight change and communities that did not, outside and during the trial. The empirical

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\(^4\)Night sleep is divided in a series of cycles made of Rapid Eyes Movements (REM) and non REM episodes. During last cycles before daybreak, REM periods significantly increase which implies shallow sleep (Klemm, 2011).
design adopted here is a standard difference-in-differences (DD) approach. The strong assumption that needs to hold for this model to be valid is the so-called parallel paths assumption: non-affected regions provide information on the expected health outcome trends for affected regions, had changes not occurred. This is discussed further in Section 4.5.

The epidemiological literature on the detrimental impact of noise and air pollution on health suggests to focus on medical conditions related to central nervous, cardiovascular and respiratory systems. The health outcome of interest is monthly spending on each of those three therapeutic classes. This comprises medications to aid circulation and breathing, and for the central nervous system includes anti-depressants and drugs to treat insomnia. These conditions characterise the health outcomes of the multiple time period DD regression model, which takes the following form:

\[
\ln SPENDING^j_{it} = \delta_j (TRIAL_t \times TREAT_i) + \gamma^j_k + \lambda_t + \sum_s X_{sit} \beta^j_s + \varepsilon^j_{it}, \quad (4.1)
\]

where \(\ln SPENDING^j_{it}\) is the natural logarithm of the total spending on prescription medicines for one of the three classes of conditions of interest \((j)\) per thousand patients in each practice \((i)\), and month \((t)\). The causal effect of the trial on medication spending is captured by the coefficient \(\delta\) of the interaction term, with \(TRIAL_t\) taking value 1 for the trial months (November 2012 to March 2013) and 0 for the baseline months (November 2011 to March 2012) and \(TREAT_i\) taking value 1 for treated practices and 0 for control practices.

The model includes monthly time effects \((\lambda_t)\) and region effects \((\gamma_k)\), where the region \(k\), which contains practice \(i\), refers to more narrowly defined geographical areas as explained below. \(X_{it}\) represents a series of \(s\) controls including index of multiple deprivation (IMD) scores to account for socio-economic levels; practice proportions of patients by gender and age; the practice proportion of GPs by age and GPs who qualified in countries other than the UK and finally the number of GPs per thousand.
Table 4.1: List of variables.

<table>
<thead>
<tr>
<th>Category</th>
<th>Variable</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Outcome</strong></td>
<td></td>
</tr>
<tr>
<td>GP spending</td>
<td>Central nervous, circulatory and respiratory systems related monthly medication spending</td>
</tr>
<tr>
<td><strong>Covariates</strong></td>
<td></td>
</tr>
<tr>
<td>Socio-economic</td>
<td>Index of Multiple Deprivation at LSOA† level</td>
</tr>
<tr>
<td>GP density</td>
<td>GPs per thousand patients</td>
</tr>
<tr>
<td>GP characteristics</td>
<td>Non-UK qualified; females; &lt;30 yrs; 30-49 yrs; 50-64 yrs</td>
</tr>
<tr>
<td>Patient characteristics</td>
<td>Females; 5-14 yrs; 45-64 yrs; 65-74 yrs; 75-84 yrs; &gt;85 yrs</td>
</tr>
</tbody>
</table>

†LSOA: Lower Layer Super Output Area, socio-geographical area with an average of 1,500 residents.

patients. Table 4.1 summarises the list of variables. The last term, \( \varepsilon_{jt} \) represents an idiosyncratic disturbance term.

We estimate the model in equation (4.1) for different macro-regions: first all areas grouped together, then regions GLE1, GLE2, GLE3 and GLW1 individually\(^5\). In the first case, we estimated the overall effect of the trial. The remaining estimates show the effect by smaller geographical areas that from a visual inspection seemed to experience consistently distinct air traffic changes. The analysis of these variations is discussed in Section 4.5.

### 4.4 Data

Monthly general practice prescriptions are drawn from the Health and Social Care Information Centre (HSCIC) for the period from November 2011 to October 2013\(^6\).

The aim is to capture conditions induced and exacerbated by environmental exposure that are treated by medications rather than in emergency rooms or hospital visits. The key variables for our analysis are the practice code (unit of observation) and its postcode, the medication identifier, the month of prescription and the Net Ingredient

\(^5\)GL stands for Greater London, then E is east, W is west. GLW2 is not estimated separately due to the low number of practices in this region.

\(^6\)The datasets are released under the terms of the Open Government Licence and can be downloaded freely online at: GP practice prescribing data - Presentation level, https://data.gov.uk/dataset/prescribing-by-gp-practice-presentation-level.
Cost (NIC - the basic cost of a drug that adjusts for the size/quantity of the medication). We matched the practice postcodes data with the trial regions.

The locations of all practices within the Heathrow airport trial areas are shown in Figure 4.1. Each medication lies within a specific therapeutic class, called BNF (British National Formulary) chapter. The three categories selected for our analysis are nervous, circulatory and respiratory systems. In addition we extracted data for infections and musculoskeletal and joint diseases to use as placebo conditions in order to test the robustness of our results.

The logarithm transformation of the practice spending per thousand patients is the main outcome used in our analysis. It summarises information on monthly expenditure by practice aggregated at medication category level. In the publicly funded British health system (NHS), this adjusted measure of practice spending corresponds to prescribed medications consumed in countries where health systems relies on private medical insurances.

The practice postcode is used to match the practice with the six trial regions (five treatment trapeziums to the east and west and one control - the aggregated areas to the north and south of Heathrow, see Figure 4.1). We assume that people tend to register with one of the practices closer to their home\(^7\). Therefore, we expect GP prescribing to be a good measure of medication spending for patients living within the same trial region of the practice. Using GIS (Geographic Information System) tools (QGIS software, Google Earth and Maps Engine) we geocode the practices' location in order to assign them to the trial areas (see Figure 4.1).

We include the Index of Multiple Deprivation (IMD) data\(^8\) to control for local socio-economic levels - this is a multidimensional composite index including dimensions related to income, employment, health, education and crime.\(^9\) We match all practice’s

\(^7\)This idea is confirmed by a recent study on the trade-off between practice quality and patient distance in England (Santos et al., 2017).

\(^8\)IMD data at LSOA level for 2011 are freely accessible and are provided under the Open Government licence.

\(^9\)The four constituent nations of the UK have each developed their own index of multiple
Figure 4.2: Average monthly number of days and flights per landing direction. Vertical lines delimit the trial period.

postcode with the respective Lower layer Super Output Areas (LSOAs), which are socio-geographical areas with an average of 1,500 residents. Our dataset reports a minimum IMD score of 0.99 for the least deprived areas and a maximum IMD score of 66.21 for the most deprived areas.

We gather yearly information on practice characteristics by using General Practice Workforce data. It contains patients headcount and its breakdown by age and gender as well as the number of GPs, their age, gender and country of qualification.10

As discussed in the previous section, in South East England wind is predominantly westerly. This is especially important when looking at landing planes at Heathrow since above 5 knots they need to land into the wind regardless of the scheduled landing direction. Introducing a monthly wind switch variable that returned the deprivation (IMD). These have been built to identify small area concentrations of deprivation, and are based on methodology developed at the University of Oxford Social Disadvantage Research Centre (Noble et al., 2006)

10See https://www.england.nhs.uk/gp/gpfv/workforce/
monthly proportion of nights when wind speed exceeded this threshold does not change the results because of collinearity with the month dummies. Besides the general wind prevalence, the upper panel of Figure 4.2 shows that March 2013 (a month that falls in the trial period) dramatically deviated from the usual pattern. The number of nights when planes came from the west of Heathrow (i.e. wind blew from east) outweighed the number of nights with planes landing from the east. This contradicted the westerly wind direction prevalence. We address this issue in Subsection 4.5.3 dropping March 2013 and exploring the effects on GP spending of a reduced four-month trial period (November 2012 to February 2013 only).

Table 4.2 reports the summary statistics for the data we used, broken down by control and treatment groups. Overall we are able to use 802 practices for which we can link the prescribing data to the variables listed in Table 4.1. The practices excluded are specialist clinics, hospitals and out-of-hours services that do not have a patient list. Overall we dropped around 24% of providers, which is similar to other studies using the same data (Rowlingson et al., 2013).

4.5 Results and Discussion

4.5.1 Landing Patterns

As discussed in Section 4.3, the trial implemented a weekly switch between two sets of air traffic exclusion zones, which we term 'odd' and 'even' weeks below. The aim was to provide early morning noise respite to the population living in the vicinity of Heathrow airport. A very detailed report on the flight patterns during the trial is available (Tucker et al., 2013); here we visually summarise its main findings.

In Figure 4.A1 and Figure 4.A2, the top panel of both figures represents the map of all landing tracks during the five-month period on the year before the trial. The second and the third panels show the aircraft tracks of planes landing at Heathrow on
Table 4.2: Sample descriptive statistics, monthly averages, Nov 2012 - Mar 2013 (during the trial) and Nov 2011 - Mar 2012 (before the trial).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Control</th>
<th>GLE1</th>
<th>GLE2</th>
<th>GLE3</th>
<th>GLW1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of practices</td>
<td>802</td>
<td>393</td>
<td>197</td>
<td>154</td>
<td>21</td>
<td>31</td>
</tr>
<tr>
<td>Number of MSOAs</td>
<td>444</td>
<td>213</td>
<td>120</td>
<td>83</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>IMD scores</td>
<td>27.63</td>
<td>23.40</td>
<td>32.88</td>
<td>36.77</td>
<td>15.65</td>
<td>16.87</td>
</tr>
<tr>
<td></td>
<td>(13.77)</td>
<td>(13.27)</td>
<td>(10.46)</td>
<td>(10.08)</td>
<td>(7.79)</td>
<td></td>
</tr>
<tr>
<td>GPs per 1,000 patients</td>
<td>0.70</td>
<td>0.68</td>
<td>0.80</td>
<td>0.65</td>
<td>0.60</td>
<td>0.69</td>
</tr>
<tr>
<td></td>
<td>(0.34)</td>
<td>(0.31)</td>
<td>(0.43)</td>
<td>(0.27)</td>
<td>(0.21)</td>
<td>(0.26)</td>
</tr>
<tr>
<td>Patients per practice</td>
<td>6,550</td>
<td>6,233</td>
<td>7,074</td>
<td>6,112</td>
<td>7,771</td>
<td>7,717</td>
</tr>
<tr>
<td></td>
<td>(3,912)</td>
<td>(3,820)</td>
<td>(3,897)</td>
<td>(3,648)</td>
<td>(4,105)</td>
<td>(4,370)</td>
</tr>
<tr>
<td>% patients:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- females</td>
<td>49.14</td>
<td>49.46</td>
<td>49.05</td>
<td>48.24</td>
<td>51.24</td>
<td>48.34</td>
</tr>
<tr>
<td></td>
<td>(3.98)</td>
<td>(3.18)</td>
<td>(5.40)</td>
<td>(4.06)</td>
<td>(1.43)</td>
<td>(2.37)</td>
</tr>
<tr>
<td>- children (4 to 14 yrs)</td>
<td>10.88</td>
<td>11.14</td>
<td>9.32</td>
<td>11.95</td>
<td>12.56</td>
<td>10.80</td>
</tr>
<tr>
<td></td>
<td>(2.96)</td>
<td>(2.68)</td>
<td>(3.22)</td>
<td>(2.60)</td>
<td>(2.24)</td>
<td>(2.67)</td>
</tr>
<tr>
<td>- elderly (over 85)</td>
<td>1.26</td>
<td>1.42</td>
<td>0.92</td>
<td>1.09</td>
<td>2.02</td>
<td>1.49</td>
</tr>
<tr>
<td></td>
<td>(0.80)</td>
<td>(0.81)</td>
<td>(0.52)</td>
<td>(0.84)</td>
<td>(0.82)</td>
<td>(0.80)</td>
</tr>
<tr>
<td>Prescribed medicines, spending per 1,000 patients:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Nervous system</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>before the trial</td>
<td>1,768</td>
<td>1,747</td>
<td>1,813</td>
<td>1,715</td>
<td>2,099</td>
<td>1,762</td>
</tr>
<tr>
<td></td>
<td>(882)</td>
<td>(632)</td>
<td>(1,324)</td>
<td>(786)</td>
<td>(389)</td>
<td>(734)</td>
</tr>
<tr>
<td>during the trial</td>
<td>1,592</td>
<td>1,575</td>
<td>1,618</td>
<td>1,496</td>
<td>2,069</td>
<td>1,734</td>
</tr>
<tr>
<td></td>
<td>(715)</td>
<td>(579)</td>
<td>(987)</td>
<td>(627)</td>
<td>(336)</td>
<td>(717)</td>
</tr>
<tr>
<td>- Circulatory system</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>before the trial</td>
<td>1,458</td>
<td>1,538</td>
<td>1,222</td>
<td>1,466</td>
<td>2,003</td>
<td>1,503</td>
</tr>
<tr>
<td></td>
<td>(558)</td>
<td>(547)</td>
<td>(479)</td>
<td>(584)</td>
<td>(429)</td>
<td>(519)</td>
</tr>
<tr>
<td>during the trial</td>
<td>957</td>
<td>985</td>
<td>835</td>
<td>953</td>
<td>1,433</td>
<td>1,041</td>
</tr>
<tr>
<td></td>
<td>(350)</td>
<td>(327)</td>
<td>(327)</td>
<td>(362)</td>
<td>(342)</td>
<td>(351)</td>
</tr>
<tr>
<td>- Respiratory system</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>before the trial</td>
<td>1,063</td>
<td>1,075</td>
<td>967</td>
<td>1,068</td>
<td>1,381</td>
<td>1,232</td>
</tr>
<tr>
<td></td>
<td>(398)</td>
<td>(381)</td>
<td>(369)</td>
<td>(440)</td>
<td>(244)</td>
<td>(431)</td>
</tr>
<tr>
<td>during the trial</td>
<td>1,068</td>
<td>1,068</td>
<td>985</td>
<td>1,065</td>
<td>1,432</td>
<td>1,289</td>
</tr>
<tr>
<td></td>
<td>(401)</td>
<td>(383)</td>
<td>(380)</td>
<td>(427)</td>
<td>(258)</td>
<td>(477)</td>
</tr>
</tbody>
</table>

Notes: Standard deviations in parenthesis. IMD refers to the Index of Multiple Deprivation. MSOA: Middle Layer Super Output Areas, which are geographies with a mean population of around 7,700.

Odd and even weeks during the trial. Since data on medication spending is available in the form of monthly datasets, we aggregated the second and third panels and interpreted the trial as a monthly event composed by a combination of alternated weekly changes. Below we describe how these monthly events are different for each region of interest.

The control regions (outlined above and below the airport on the maps in Figure
4.1 and Figure 4.A2) included those regions that were not affected by changes implemented during the trial. The GLE1 area (see Figure 4.A1) experienced an overall notable reduction in air traffic on odd weeks and a slight increase on even weeks of the trial, with a reduction overall in each month of the trial. Similarly GLE2 (see Figure 4.A1), an area generally subject to heavy early morning air traffic, saw some increase in traffic on the odd weeks and an important drop on the even weeks. These are the two regions most affected by the trial. The last region to the east of Heathrow is GLE3 (see Figure 4.A1); if we distinguished the northern from the southern region, the latter experienced an overall increase in air traffic and specifically a sharp increase in traffic on even weeks.

From the second and third panels of Figure 4.A2 we can see that the GLW1 area was characterised by a serious increase in air traffic on the odd weeks and a decrease on the other weeks, implying an overall increase in early morning air traffic. The GLW2 area (see Figure 4.A2) saw a drastic reduction of air traffic on odd weeks and almost no change on even weeks. However interesting this area might be, it contains only six GP practices in a mainly rural region.

These are the broad regions identified by the trial final report. However, we assume that the level of variation occurred at a lower regional dimension. Our observations are at the practice level but the environmental quality may be common to groups of practices. This is supported by the fact that noise and air pollution levels vary at a refined level. Maps of noise contours provided by the Civil Aviation Authority draw a picture of how much variation there is from one street to a few streets apart. This suggests using a geographical unit smaller than the broad regions but larger than practice level. We use the Middle Layer Super Output Areas, MSOAs, in which environmental quality is likely to be more homogeneous\footnote{MSOAs enclose between 5,000 to 15,000 residents, with an average of 7,700 population as of Census 2011. Each MSOA includes a minimum of one and a maximum of seven practices.} (Lee et al., 2014). Our unit of observation (practices) is smaller than the MSOAs which could bias our standard-errors, as documented by Moulton (1986). Failure to take account of this
clustering dimension could lead to a downward bias of the standard errors. The main specification of equation 4.1 controls for these potential common group variations by adopting cluster-robust standard errors, where the number of clusters (MSOAs) is large (between 227 to 444, see Table 4.2).

We check for possible standard error bias and calculate the intraclass correlation coefficients (ICC) of errors and covariate (i.e. TRIAL_t × TREAT_i, the main regressor of interest)\(^1\). In fact, the correct standard error can be biased by a quantity which depends on the magnitude of those coefficients, on the number of clusters and on the size of the clusters\(^2\). We obtain very small ICC of covariate (0.073) and zero ICC of errors. This suggests standard error bias may not be a major concern. However, we decide to maintain the more conservative cluster adjusted standard errors, rather than those obtained with the commonly used robust adjustment. These are the main results reported in this chapter but later in Subsection 4.5.3 we discuss in detail a series of alternative specifications and corrections to standard errors.

Besides the regional variations due to the trial, we need to keep in mind that wind speed and wind direction affect the landing provenance regardless of the planned schedule. In other words, ideally during the trial there should have been a regular weekly switch between planes landing from the east (i.e. over London) and planes landing from the west (i.e. over Reading). The reality however departs from the forecast due to changing atmospheric conditions. When wind speed is above 5 knots, planes always land into the wind. As we have already mentioned, in South East England on average wind is westerly 70% of the year. We therefore expect more robust results for the three areas to the east of Heathrow - GLE1, GLE2 and GLE3 - as for these regions there was a significant reduction during weeks when they experienced respite (see Figure 4.2). This westerly preference of planes landing over

\(^{12}\)This can be done using the \texttt{loneway} command in \textsc{Stata} (StataCorp 2014).

\(^{13}\)The so-called Moulton factor, which tells how much larger the corrected standard error would be compared to an unadjusted standard error. With unbalanced group sizes, this is given by:

\[
\frac{SE(\hat{\beta}_1)}{SE(\hat{\beta}_1)} = \left(1 + \left[\frac{V(n_g)}{\bar{n}} + \bar{n} - 1\right] \rho_x \rho_\varepsilon \right)^{\frac{1}{2}},
\]

where \(n_g\) is the size of group \(g\); \(V(n_g)\) is the variance of group sizes, \(\bar{n}\) is the average group size and \(\rho_\varepsilon\) and \(\rho_x\) are the ICC of errors \(\varepsilon\) and covariate \(x\), respectively.
London was observed during the first four months of the trial.

To summarise, the trial included four broad areas where we can investigate the impacts on medical prescriptions of changes in air traffic during early mornings for five months. What can we expect to be the relationship between the variations in population exposure to noise and air pollution and monthly medication spending? The impact will depend crucially on the population density of the affected areas. Those areas where there appeared to be a significant reduction in air traffic during the trial, GLE1 and GLE2, were in fact the most densely populated, as illustrated in Figure 1 by their high GP practice density. Therefore we might expect an overall reduction in medical prescriptions. Our GP practice data are at a much more refined geographical level and so the regressions will ascertain if significant reductions can be detected.

4.5.2 Effect of the Trial by Health Condition and Region

Our analyses focus on the effects of the trial on central nervous, circulatory and respiratory system ailments. The previous literature showed that these conditions are associated with air and noise pollution exposure.

An investigation of the parallel paths assumption is given by Figure 4.4.3, Figure 4.4.4, Figure 4.4.5 and Figure 4.4.6 where trends of monthly spending by thousand patients are adjusted by percent of female patients, percent of old patients (85+ years old) and IMD scores of the small socio-geographical areas. They show the patterns of medication spending in control and several treatment groups and generally suggest no differences in trends. Therefore, we take this as supporting evidence that the parallel paths assumption holds.

Table 4.3 summarises regression estimates using equation (4.1) by health condition for the whole sample for the main variable of interest, $TRIAL_t \times TREAT_i$, which is a trial indicator equals to 1 for all practices within treated areas and during the five
Table 4.3: Trial effect on medication spending per 1,000 patients by therapeutic class for all regions.

<table>
<thead>
<tr>
<th></th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRIAL × TREAT</td>
<td>-0.058***</td>
<td>0.020</td>
<td>-0.033*</td>
</tr>
<tr>
<td>(Adjusted)</td>
<td>(0.020)</td>
<td>(0.017)</td>
<td>(0.018)</td>
</tr>
</tbody>
</table>

Adjusted $R^2$ 0.655 0.829 0.687
Observations 7832 7834 7834
Clusters 444 444 444
Months 10 10 10

Notes: Cluster-robust standard errors in parentheses. The clustering dimension is MSOA, where each cluster has a minimum of 1 and a maximum of 7 practices. * $p < .1$, ** $p < .05$, *** $p < .01$

months of the trial and to 0 for the same five months one year earlier.\(^{14}\)

The first column of Table 4.3 shows the results for the central nervous system, a therapeutic class related to the treatment of sleep loss, concentration deficits and other stress-related diseases. The estimate is significantly negative overall for the regions involved in the trial. This showed the greatest reduction in spending of 5.8% during the trial.

The second column of Table 4.3 shows the estimates for cardiovascular system medication spending. For this class of conditions, this indicates that the trial had no overall significant effect on all regions involved in the flight-path variations. As we show in Table 4.4, the coefficient estimates are significantly positive around 7.2% for GLE3 and only slightly significant for GLE1 and GLE2. The weak results here probably reflect the more long term nature of these conditions that make it difficult to identify impacts from short term changes as in our trial.

Column 3 of Table 4.3 reports the results for respiratory system conditions. The five-month trial reduced the spending on respiratory medication by 3.3%.

On average a negative effect on central nervous and respiratory system conditions seem to dominate. The explicit purpose of the systematic flight paths variations set up by Heathrow airport was to reduce the population exposed to high noise and air

\(^{14}\)Full regression results are available in the Appendix Table 4.A1, Table 4.A2 and Table 4.A3. The regression analysis was repeated for each broad treatment region and included all the atmospheric, socio-economic, GP and patient controls listed in Table 4.1.
pollution levels during sensitive hours. The results from Table 4.3 for all regions seem to confirm an overall decrease in medication spending caused by the trial.

The trial final report documented the comments received by local communities after the trial was conducted (Tucker et al., 2013). The response was mixed, residents outside the areas of predictable respite expressed vocal complaints of increased air traffic and annoyance. However, other communities perceived a decrease in early morning noise and positively assessed the trial. Therefore it is worthwhile focusing on the regional results in more detail, which are given by Table 4.4.

The GLE1 area reports significant effects mainly for the nervous system class. In fact, there are negative changes in GP spending of 7.7% for nervous system conditions. Although that area also shows an increase in circulatory spending by 4%, this estimate is statistically significant at the 90% confidence level. The GLE2 region is characterised by a marked decrease in air traffic during its respite weeks and it produced the clearest picture. The almost complete reduction in landing aircraft prevailed over the increase in flights in alternate weeks. In fact during the trial, monthly GP spending decreased by around 11%, 3.6% and 6.8% for nervous, circulatory and respiratory conditions, respectively. Evidently, the results for the GLE2 area indicate that residents benefited from the weekly respite during early morning hours. It appears that two weeks per month of air traffic suspension were enough to reduce monthly prescription spending on all conditions.

For GLE3 as a whole we find a 4.7% significant increase for those medicines related to the central nervous conditions. From the maps in Figure 4.A1 and Figure 4.A2 we can see that the change differently affected the northern and the southern part of GLE3. To investigate the effect of the trial on the two regions of GLE3 we separately estimate the model for the two areas. The results - not reported here - show that prescribing practices in the northern part drive the change, in contrast to our expectations that the southern part experienced the most increase in medication spending. The two main concerns are the reduction in the number of observations
and in the areas extension. Having smaller regions opens the issue of patient sorting. In fact residents of one side of the region could easily be registered with a GP on the other side, with a maximum distance from the southern to the northern part of 10 miles. This division also results in small numbers of practices, sixteen for GLE3 north and just five practices for GLE3 south. Therefore, we keep GLE3 as a whole and we apply the estimates to the entire region. We find a 4.7% and a 7.2% significant increase for those medicines related to the central nervous and circulatory conditions, respectively.

Table 4.4: Trial effect on medication spending per 1,000 patients by therapeutic class and treatment regions.

<table>
<thead>
<tr>
<th>Region</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
</tr>
<tr>
<td>GLE1</td>
<td>TRIAL × TREAT</td>
<td>-0.077*** (0.027)</td>
<td>0.040* (0.022)</td>
</tr>
<tr>
<td></td>
<td>Adjusted R²</td>
<td>0.634</td>
<td>5845</td>
</tr>
<tr>
<td></td>
<td>Observations</td>
<td>5843</td>
<td>0.824</td>
</tr>
<tr>
<td></td>
<td>Clusters</td>
<td>333</td>
<td>333</td>
</tr>
<tr>
<td></td>
<td>Months</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>GLE2</td>
<td>TRIAL × TREAT</td>
<td>-0.105*** (0.023)</td>
<td>-0.036* (0.020)</td>
</tr>
<tr>
<td></td>
<td>Adjusted R²</td>
<td>0.691</td>
<td>5374</td>
</tr>
<tr>
<td></td>
<td>Observations</td>
<td>5374</td>
<td>0.809</td>
</tr>
<tr>
<td></td>
<td>Clusters</td>
<td>296</td>
<td>296</td>
</tr>
<tr>
<td></td>
<td>Months</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>GLE3</td>
<td>TRIAL × TREAT</td>
<td>0.047** (0.022)</td>
<td>0.072*** (0.019)</td>
</tr>
<tr>
<td></td>
<td>Adjusted R²</td>
<td>0.675</td>
<td>4110</td>
</tr>
<tr>
<td></td>
<td>Observations</td>
<td>4110</td>
<td>0.801</td>
</tr>
<tr>
<td></td>
<td>Clusters</td>
<td>229</td>
<td>229</td>
</tr>
<tr>
<td></td>
<td>Months</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>GLW1</td>
<td>TRIAL × TREAT</td>
<td>0.046 (0.036)</td>
<td>0.048 (0.031)</td>
</tr>
<tr>
<td></td>
<td>Adjusted R²</td>
<td>0.665</td>
<td>4205</td>
</tr>
<tr>
<td></td>
<td>Observations</td>
<td>4205</td>
<td>0.793</td>
</tr>
<tr>
<td></td>
<td>Clusters</td>
<td>227</td>
<td>227</td>
</tr>
<tr>
<td></td>
<td>Months</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

Notes: Cluster-robust standard errors in parentheses. The clustering dimension is MSOA, where each cluster has a minimum of 1 and a maximum of 7 practices. 10 months correspond to 5 in the baseline period (Nov 2011- Mar 2012) plus 5 in trial period (Nov 2012- Mar 2013). * p < .1, ** p < .05, *** p < .01
For the GLW1 region, the coefficient estimates are positive as expected due to an overall increase in air traffic. However, they are not statistically significant. As previously discussed and as shown in the Figure 4.2, we know that wind is predominantly westerly which implies that the majority of the flights landed over the three other areas. This, combined with the sparse population density and low number of practices in this region, could explain the lack of significant results.

To conclude, our estimates suggest that decreases in air traffic were responsible for the health effects. The identification of these effects is aided by the fact that the groups with the higher number of practices, hence more densely populated, and the higher percentage of landing aircraft happened to be the two regions that experienced an important reduction in air traffic during the trial.

### 4.5.3 Robustness Tests

We introduce a number of robustness tests to further investigate our main results. These are summarised in Table 4.5, where the top panel reports the coefficient estimates of the main specification from Table 4.3.

Panel 1 reports the estimates of the coefficient $\delta$ of equation (4.1) with heteroskedasticity-robust standard errors. As expected the standard errors are lower, raising the statistical significance relative to the variant with MSOA clusters. In panel 2 of Table 4.5 we change the cluster dimension to a more aggregated level, the four trial zones: GLE1, GLE2, GLE3 and GLW1. The significance levels are comparable to the previous panel with larger standard errors. Therefore the results are robust to alternative error term variance corrections.

For each outcome group we repeat the analysis for all the 24 months of available data and find smaller coefficients with similar levels of significance (see panel 3 of Table 4.5). The second panel of Figure 4.2 (see page 85) shows the well known seasonal pattern of flights with the majority of landings in the summer months. As
the trial was during the off season it seems preferable to compare landings with the same period one year earlier.

The structure of DD panel data raises concerns over serial correlation. The literature does not give unequivocal guidance over the resolution of this potential problem. One reference paper by Bertrand et al. (2004) highlights that, within the DD setting, the combined presence of long time series and the use of the period of treatment indicator imposes very little variation over time causing serious potential issues of serial correlation. A common solution is to aggregate the observations across time periods. Therefore we average across all five months for the year before the trial and all five months during the trial period, equivalent to using two cross-sections. We estimate equation (4.1) with this new two-period set up and we obtain the coefficient estimates for the regressor of interest $TRIAL_t \times TREAT_i$ reported in panel 4 of Table 4.5. We can see that the size and the direction of the effects does not change, however the statistical significance is affected. With such a large reduction in observations it is difficult to obtain very precise estimates. The less restrictive alternative of adding a time trend to equation (4.1) does not substantially affect the nervous coefficient, although it does impact on the significance of the respiratory coefficient (see panel 5 of Table 4.5). An intermediate approach is to include area-by-time trends as these allow for region specific shocks (see panel 6 of Table 4.5). In this case the nervous coefficient is larger and highly significant but the value of the circulatory coefficient drops.

As we mentioned earlier, March 2013 showed an unusual wind direction pattern, see Figure 4.2 on page 85. To overcome possible issues caused by the easterly wind prevalence in that specific month, we decide to exclude observations for March 2013 and consequently for March 2012. The results in panel 7 of Table 4.5 suggest that this deviation from the usual wind direction pattern does not significantly affect our original estimates.

We also experiment with alternative regional groupings, given that they are differ-
entially affected by the landing patterns. The results are shown in panels 8 to 11 of Table 4.5. In panel 8 we include only observations for GLE1 and GLE2, which as previously discussed and clearly shown in Table 4.4, report the most significant results. We estimate the trial coefficients with these two regions grouped together, keeping the same control region and omitting the GLE3 and GLW1 areas. We, therefore, assess the impact of the trial on regions that experienced a visible decrease in air traffic. As expected, the estimates increase in absolute value. GP spending decrease most for central nervous system medication, from 5.8% in the original pooled estimate to 7.6%. For respiratory medication, the overall decrease in GP spending goes from 3.3% in the original estimation to 3.9%.

Table 4.5: Robustness tests for all regions involved in the trial.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nervous (1)</th>
<th>Circulatory (2)</th>
<th>Respiratory (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main - MSOAs clusters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( TRIAL \times TREAT )</td>
<td>-0.058*** (0.020)</td>
<td>0.020 (0.017)</td>
<td>-0.033* (0.018)</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.655</td>
<td>0.829</td>
<td>0.687</td>
</tr>
<tr>
<td>Observations</td>
<td>7832</td>
<td>7834</td>
<td>7834</td>
</tr>
<tr>
<td><strong>1. No clusters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( TRIAL \times TREAT )</td>
<td>-0.058*** (0.013)†</td>
<td>0.020** (0.010)†</td>
<td>-0.033*** (0.011)†</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.655</td>
<td>0.829</td>
<td>0.687</td>
</tr>
<tr>
<td>Observations</td>
<td>7832</td>
<td>7834</td>
<td>7834</td>
</tr>
<tr>
<td><strong>2. Trial zones as clusters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( TRIAL \times TREAT )</td>
<td>-0.058** (0.018)</td>
<td>0.020 (0.026)</td>
<td>-0.033** (0.014)</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.655</td>
<td>0.829</td>
<td>0.687</td>
</tr>
<tr>
<td>Observations</td>
<td>7832</td>
<td>7834</td>
<td>7834</td>
</tr>
<tr>
<td><strong>3. Full period of 24 months</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( TRIAL \times TREAT )</td>
<td>-0.035*** (0.011)</td>
<td>0.005 (0.009)</td>
<td>-0.020** (0.009)</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.655</td>
<td>0.812</td>
<td>0.665</td>
</tr>
<tr>
<td>Observations</td>
<td>18801</td>
<td>18802</td>
<td>18804</td>
</tr>
<tr>
<td><strong>4. Averaging across time</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( TRIAL \times TREAT )</td>
<td>-0.055** (0.022)</td>
<td>0.023 (0.019)</td>
<td>-0.028 (0.018)</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.611</td>
<td>0.83</td>
<td>0.694</td>
</tr>
<tr>
<td>Observations</td>
<td>1569</td>
<td>1569</td>
<td>1569</td>
</tr>
<tr>
<td><strong>5. With time trend</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( TRIAL \times TREAT )</td>
<td>-0.059*** (0.019)</td>
<td>-0.023 (0.016)</td>
<td>-0.024 (0.017)</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.642</td>
<td>0.816</td>
<td>0.681</td>
</tr>
</tbody>
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Continued on next page
Continued from previous page

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<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
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<tbody>
<tr>
<td></td>
<td>Observations</td>
<td>7832</td>
<td>7834</td>
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</table>

### 6. With area-specific time trends

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<tbody>
<tr>
<td></td>
<td></td>
<td>-0.081*** (0.017)</td>
<td>-0.164*** (0.017)</td>
<td>-0.014 (0.014)</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.641</td>
<td>0.784</td>
<td>0.68</td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>7832</td>
<td>7834</td>
<td>7834</td>
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### 7. Dropping obs for March 2012 and 2013

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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>-0.058*** (0.021)</td>
<td>0.019 (0.017)</td>
<td>-0.033* (0.019)</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.646</td>
<td>0.827</td>
<td>0.679</td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>6266</td>
<td>6267</td>
<td>6267</td>
<td></td>
</tr>
</tbody>
</table>

### 8. GLE1 & GLE2

<table>
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<th>$TTrail \times TREAT$</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>-0.076*** (0.022)</td>
<td>0.013 (0.018)</td>
<td>-0.039** (0.019)</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.654</td>
<td>0.827</td>
<td>0.678</td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>7317</td>
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### 9. GLE1, GLE2 & GLE3

<table>
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<tr>
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<th>$TTrail \times TREAT$</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>-0.067*** (0.021)</td>
<td>0.017 (0.018)</td>
<td>-0.036** (0.018)</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.656</td>
<td>0.83</td>
<td>0.683</td>
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### 10. GLE3 & GLW1

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<td></td>
<td></td>
<td>0.047* (0.025)</td>
<td>0.059*** (0.021)</td>
<td>0.006 (0.019)</td>
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<tr>
<td>Adjusted $R^2$</td>
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### 11. GLW1 & GLW2

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<tbody>
<tr>
<td></td>
<td></td>
<td>0.042 (0.033)</td>
<td>0.038 (0.028)</td>
<td>0.005 (0.021)</td>
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<tr>
<td>Adjusted $R^2$</td>
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</table>

### 12. No atorvastatin

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</thead>
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</table>

### 13. Placebos

<table>
<thead>
<tr>
<th></th>
<th>Musculoskeletal</th>
<th>Infections</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$TTrail \times TREAT$</td>
<td>-0.023 (0.019)</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
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<td>0.399</td>
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<tr>
<td>Observations</td>
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<td>7833</td>
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</table>

Notes: Cluster-robust standard errors in parentheses if not otherwise specified.

† Heteroskedasticity-robust standard errors. * $p < .1$, ** $p < .05$, *** $p < .01$

We next report results for all regions to the east of Heathrow, adding GLE3 to the previous specification (see panel 9 of Table 4.5). This confirms the same estimates reported in row 7. The magnitude reduces as we would anticipate considering that
in the GLE3 area we see some increase in air traffic during the trial. Keeping observations for GLE3 and GLW1, groups all areas that had an overall increase in air traffic during the trial (panel 10). For these regions, we see a significant increase in circulatory medication spending by around 6%, as well as a positive change of 4.7% for nervous spending.

This important result shows that the gains in some areas were, to some extent, counterbalanced by increased spending in regions overflown more heavily during the trial. Moreover, this lends additional support to our identification strategy that relies on early morning changes in landing patterns. Additionally, we report the results pooling together all regions to the west of Heathrow (see panel 11 of Table 4.5). For this specification we retrieve data for the GLW2 area that was excluded for the analysis due to too few practices. The signs remain positive for the three therapeutic classes, but the coefficients are not statistically significant.

We detect a substantial decrease in spending from June 2012 onwards for circulatory system diseases. We discover that in May 2012 the patent of a medicine widely used to control cholesterol levels (atorvastatin) expired inducing a 93% reduction in its price. Consequently the NHS advised GPs to switch to atorvastatin\textsuperscript{15}. This change is likely to have been driven by the drop in the medicine price rather than in a decrease in the quantity prescribed. To account for the possibility that the switch to the generic medicine has been differentially adopted in the treated and control groups, we add a further set of outcomes: cardiovascular diseases spending excluding atorvastatin medicines. Panel 12 of Table 4.5 shows results for all circulatory medicines other than atorvastatin to rule out a possible confounding effect caused by this drug. The coefficient estimate changes in size but remains statistically insignificant, as for the coefficients of the main specification.

Finally, we run a series of regressions as placebos using health outcomes that are deemed unlikely to be affected by air quality or noise changes. We identify infections

\textsuperscript{15}See http://www.pulsetoday.co.uk/price-of-atorvastatin-plummets-93-as-patent-end.
and musculoskeletal and joint diseases as such ‘placebos’, considered unaffected by either noise or air pollution exposure. Panel 13 of Table 4.5 shows the results of this analysis. The estimates for both therapeutic classes are found to be statistically insignificant, hence providing further support for our identification strategy.

4.5.4 Impacts on Health Spending

We next investigate the economic significance of our results. Table 4.6 shows back-of-the-envelope calculations of changes in monthly prescribing costs due to the implementation of the trial by region, which generated an overall decrease in spending by GP practices.

For instance, for the GLE1 and GLE2 regions we find a 7.6% reduction in monthly spending on nervous system conditions per thousand patients (see Table 4.5). On average a practice based in GLE1 or GLE2 has 6,600 patients and recorded about 1,760 GBP (2,200 USD) monthly spending per thousand patients (derived from Table 4.2). From these figures, we calculate the monthly change in spending per practice, and we multiply it by 351 - the total number of the practices within the GLE1 and GLE2 regions (see Table 4.2). The result of this calculation is shown in Table 4.6 and adds up to about 310,000 GBP (390,000 USD) saved in monthly spending for the whole GLE1 and GLE2 regions only for the nervous system therapeutic class.

To put this number in context, we calculate the monthly saving in these regions arising from the substitution to atorvastatin following the expiration of the patent in May 2012, as described above. This suggests about 110,000 GBP savings per month from this one drug alone. Therefore our estimate of the savings from the trial for the entire nervous system class of drugs, 310,000 GBP, seems realistic.

We similarly calculate the cost savings for respiratory conditions, which was generally significant but less robust, and we add these to the nervous system savings. Looking at all the regions involved in the trial, we calculate an overall net monthly saving
of about 420,000 GBP (525,000 USD). Had the flights reduction been adopted permanently, the NHS would have saved around 5 millions GBP (6.3 USD millions) per year in respiratory and nervous system prescribing costs. To put this figure into perspective, we can calculate the total annual prescribing spend in the trial area. In 2013 in England the prescribing spend was at 142 GBP (178 USD) per person\(^{16}\). Multiplying this by the 403 practices times the average number of patients per practice, we obtain about 410 millions GBP (513 millions USD), which consists of an estimate of the annual total prescribing spend in the trial regions. Therefore, the estimated savings account for 1.23\% of the total prescribing spending. We should also note that these are likely to be conservative figures since in our practice sample we rule out all those practices that do not have a patient list (e.g., specialist clinics, out-of-hours services and hospitals - which account for about 24\% of all practices).

To complete the figure of the induced monetary saving, we should add the reduced costs of GP time due to the likely lower number of visits by patients to request prescriptions. However we do not have sufficient data to estimate this. In addition, there are likely to be indirect benefits, such as reduced absenteeism and related gains in productivity. Combining these with the direct reduction in medical spending is likely to lead to much greater savings.

**Table 4.6:** Monthly change in prescribing costs (GBP) induced by the five-month systematic flight paths variation.

<table>
<thead>
<tr>
<th></th>
<th>Overall(^{†})</th>
<th>GLE1 &amp; GLE2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Practices</td>
<td>403</td>
<td>351</td>
</tr>
<tr>
<td>Thousand patients per practice</td>
<td>7.17</td>
<td>6.59</td>
</tr>
<tr>
<td>Nervous</td>
<td>-309,481</td>
<td>-310,152</td>
</tr>
<tr>
<td>Respiratory</td>
<td>-110,745</td>
<td>-91,817</td>
</tr>
<tr>
<td><strong>TOT</strong></td>
<td><strong>-420,226</strong></td>
<td><strong>-401,969</strong></td>
</tr>
</tbody>
</table>

\(^{†}\)Results for the MSOAs within the areas of GLE1, GLE2, GLE3 and GLW1. GP spending for the cardiovascular therapeutic class is omitted since no significant results were detected.

4.6 Conclusion

The aim of this chapter is to estimate the health externalities generated by air and noise pollutants from aircraft. We exploit a five-month trial that took place around London Heathrow airport from November 2012 to March 2013. The trial involved changes in patterns of aircraft landings during early morning hours (4.30 am to 6.00 am). Health effects are measured through changes in medication prescribing by GP practice. We find a statistically significant response of monthly medication spending on central nervous and respiratory system conditions to these changes, and weak effects for circulatory conditions. Crucially, significant reductions in prescription spending on nervous and respiratory conditions are detected for the regions that experienced a drop in air traffic during the trial.

This quasi-experimental approach suggests a causal impact of aircraft air and noise pollution on human health. By relying on a quasi-experimental research design, we complement previous epidemiology-based studies that find negative associations between aircraft noise and health around major airports (Clark et al., 2012).

This study also illustrates the benefits of using publicly available data to estimate some of the direct costs from adverse environmental exposure imposed on society, whose costs are often borne by the public health system. Our calculations suggest a sizeable direct impact on GP spending in the areas affected. These estimates do not include the reduced costs of avoided GP visits, the gain in patients well-being, and impacts on individual worker productivity through absenteeism or less effective effort in the workplace. Our findings suggest that small variations to air traffic exposure during critical hours affect health and this could inform environmental policy.
4.A Appendix

The first two figures of this Appendix, Figure 4.A1 and Figure 4.A2, illustrate aircraft tracks to the east and to the west of Heathrow airport (labelled as LHR), respectively. For both figures, the first panel refers to the baseline period 2011/2012 (~45 nights) and second (~44 nights) and third (~41 nights) panels for the trial period 2012/2013. The tracks show aircraft landing at Heathrow with inner and outer exclusion zones operative, which are the shaded areas in the second and third panels for both figures, respectively. The maps of Figure 4.A1 show five macro-regions involved in the study: the control zones are to the north and south of LHR; and to the east of LHR there are GLE1, GLE2 and GLE3. The maps of Figure 4.A2, besides the same control regions, show two more macro-regions involved in the study: to the west of LHR there are GLW1 and GLW2. Flight tracks analysis from the trial final report (Tucker et al., 2013). All areas on these maps are approximative.
Figure 4.A1: Aircraft tracks to the east of Heathrow airport.
Figure 4.A2: Aircraft tracks to the west of Heathrow airport.
Figure 4.A3: GLE1 area. Average monthly practice medication spending related to different categories of medication adjusted by IMD score, percent of female patients and percent of the elderly (85 and above years old). The dashed vertical lines indicate the trial period from November 2012 to March 2013.
Figure 4.A4: GLE2 area. Average monthly practice medication spending related to different categories of medication adjusted by IMD score, percent of female patients and percent of the elderly (85 and above years old). The dashed vertical lines indicate the trial period from November 2012 to March 2013.
Figure 4.A5: GLE3 area. Average monthly practice medication spending related to different categories of medication adjusted by IMD score, percent of female patients and percent of the elderly (85 and above years old). The dashed vertical lines indicate the trial period from November 2012 to March 2013.
Chapter 4. The Medical Cost of Air Traffic Pollution

(a) **Nervous system.**

(b) **Circulatory system.**

(c) **Respiratory system.**

**Figure 4.A6:** GLW1 area. Average monthly practice medication spending related to different categories of medication adjusted by IMD score, percent of female patients and percent of the elderly (85 and above years old). The dashed vertical lines indicate the trial period from November 2012 to March 2013.
Table 4.A1: Trial effect on nervous system medication spending per 1,000 patients by treatment regions.

<table>
<thead>
<tr>
<th>Treatment Region</th>
<th>All</th>
<th>GLE1</th>
<th>GLE2</th>
<th>GLE3</th>
<th>GLW1</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRIAL x TREAT</td>
<td>-0.058***</td>
<td>-0.077***</td>
<td>-0.105***</td>
<td>0.047**</td>
<td>0.046</td>
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<td></td>
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<td>(0.027)</td>
<td>(0.023)</td>
<td>(0.022)</td>
<td>(0.036)</td>
</tr>
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<td></td>
<td>(0.090)</td>
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<tr>
<td>Dec 2011</td>
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<td>0.034***</td>
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<td>(0.008)</td>
<td>(0.008)</td>
<td>(0.009)</td>
<td>(0.009)</td>
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<tr>
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<td>(0.008)</td>
<td>(0.009)</td>
<td>(0.010)</td>
<td>(0.010)</td>
</tr>
<tr>
<td>Feb 2012</td>
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<td>(0.008)</td>
<td>(0.009)</td>
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<td>(0.010)</td>
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<td>(0.015)</td>
<td>(0.015)</td>
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<td>(0.015)</td>
<td>(0.015)</td>
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<td>(0.003)</td>
<td>(0.003)</td>
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<td>4.523***</td>
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<tr>
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<td>(1.114)</td>
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<td>(0.910)</td>
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<td>(1.770)</td>
<td>(0.927)</td>
<td>(0.994)</td>
<td>(0.999)</td>
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<td>Patients 45-64 years old</td>
<td>5.144***</td>
<td>5.522***</td>
<td>1.113</td>
<td>1.455*</td>
<td>2.094**</td>
</tr>
<tr>
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<td>(2.035)</td>
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<td>(2.464)</td>
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<td>Patients 75-84 years old</td>
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<td>0.705</td>
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</tr>
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<td>(3.569)</td>
<td>(3.039)</td>
<td>(3.286)</td>
<td>(3.611)</td>
</tr>
<tr>
<td>Patients over 85 years old</td>
<td>16.049***</td>
<td>18.110***</td>
<td>6.680</td>
<td>10.632**</td>
<td>16.046***</td>
</tr>
<tr>
<td></td>
<td>(5.331)</td>
<td>(5.413)</td>
<td>(5.153)</td>
<td>(4.884)</td>
<td>(5.814)</td>
</tr>
<tr>
<td>GP females</td>
<td>0.021</td>
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<td>-0.081</td>
<td>-0.078</td>
</tr>
<tr>
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<td>(0.060)</td>
<td>(0.049)</td>
<td>(0.056)</td>
<td>(0.054)</td>
</tr>
<tr>
<td>GP up to 30 years old</td>
<td>0.377***</td>
<td>0.386**</td>
<td>0.331**</td>
<td>0.297*</td>
<td>0.365*</td>
</tr>
<tr>
<td></td>
<td>(0.171)</td>
<td>(0.189)</td>
<td>(0.145)</td>
<td>(0.151)</td>
<td>(0.186)</td>
</tr>
<tr>
<td>GP qualified outside UK</td>
<td>0.178**</td>
<td>0.180**</td>
<td>0.150**</td>
<td>0.156**</td>
<td>0.142**</td>
</tr>
<tr>
<td></td>
<td>(0.072)</td>
<td>(0.083)</td>
<td>(0.069)</td>
<td>(0.071)</td>
<td>(0.068)</td>
</tr>
<tr>
<td>constant</td>
<td>4.714***</td>
<td>4.903***</td>
<td>4.133***</td>
<td>4.417***</td>
<td>4.214***</td>
</tr>
<tr>
<td></td>
<td>(0.434)</td>
<td>(0.518)</td>
<td>(0.394)</td>
<td>(0.493)</td>
<td>(0.511)</td>
</tr>
</tbody>
</table>

Notes: Cluster-robust standard errors in parentheses. * p < .1, ** p < .05, *** p < .01.

Observations: 7832, 5843, 5374, 4110, 4205

Adjusted $R^2$: 0.655, 0.634, 0.691, 0.675, 0.665
Table 4.A2: Trial effect on circulatory medication spending per 1,000 patients by treatment regions.

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>GLE1</th>
<th>GLE2</th>
<th>GLE3</th>
<th>GLW1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial x Treat</td>
<td>0.020</td>
<td>0.040*</td>
<td>-0.036*</td>
<td>0.072***</td>
<td>0.048</td>
</tr>
<tr>
<td>(0.017)</td>
<td>(0.022)</td>
<td>(0.020)</td>
<td>(0.019)</td>
<td>(0.031)</td>
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Notes: Cluster-robust standard errors in parentheses. * $p < .1$, ** $p < .05$, *** $p < .01$
Table 4.A3: Trial effect on respiratory medication spending per 1,000 patients by treatment regions.

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<td>GP 50-64 years old</td>
<td>-0.011</td>
<td>0.012</td>
<td>-0.032</td>
<td>-0.010</td>
<td>-0.005</td>
</tr>
<tr>
<td></td>
<td>(0.050)</td>
<td>(0.059)</td>
<td>(0.051)</td>
<td>(0.055)</td>
<td>(0.055)</td>
</tr>
<tr>
<td>GP qualified outside UK</td>
<td>-0.023</td>
<td>0.025</td>
<td>-0.039</td>
<td>0.015</td>
<td>0.038</td>
</tr>
<tr>
<td></td>
<td>(0.052)</td>
<td>(0.052)</td>
<td>(0.056)</td>
<td>(0.062)</td>
<td>(0.061)</td>
</tr>
<tr>
<td>constant</td>
<td>4.404***</td>
<td>4.593***</td>
<td>3.720***</td>
<td>4.228***</td>
<td>4.171***</td>
</tr>
<tr>
<td></td>
<td>(0.361)</td>
<td>(0.403)</td>
<td>(0.386)</td>
<td>(0.533)</td>
<td>(0.519)</td>
</tr>
</tbody>
</table>

Notes: Cluster-robust standard errors in parentheses. * p < .1, ** p < .05, *** p < .01.

Observations: 7834 5845 5374 4110 4205

Adjusted $R^2$: ✓ ✓ ✓ ✓ ✓
Chapter 5

Air Pollution and Hospital Admissions: an Instrumental Variable Approach

SUMMARY. This chapter investigates the effects of daily variations in wind direction on air pollution levels and hospitalisation rates for nervous, circulatory and respiratory conditions using administrative data from England spanning from 2008 to 2014. Adopting an instrumental variable approach, we show that short-term exposure to particulate matter increases emergency admission rates for nervous ailments. We also find some substitution effects of hospital admissions from planned to unplanned visits, especially for circulatory and respiratory conditions.

5.1 Introduction

Estimating the health effects of daily variations of air pollution is a major issue of all countries. While there is much interest in meeting international standards of pollution levels, quantifying the health burden borne by citizens remains a concern of epidemiologists as well as health and environmental economists.
Although England generally meets the pollution thresholds imposed by the EU, there is evidence that people still die from exposure to air pollution (Janke et al., 2009). In this chapter, we estimate the health impact of local and short-term exposure to air pollution exploiting daily variations in wind direction from 2008 to 2014. We employ an instrumental variable approach with the aim to contribute to the existing economics literature on the causal effects of environmental exposure on health outcomes.

Instrumental variables provide a good solution to the issue of endogeneity that characterises pollution exposure. Other studies have adopted the same identification strategy to investigate the relationship between pollution exposure and health. In particular, among these, two studies look at the short-time effect on hospitalisations. Moretti and Neidell (2011) instrument exposure to ozone with daily boat traffic at the ports of Los Angeles. They find that increases in ozone levels lead to increases in hospitalisations for respiratory conditions. Schlenker and Walker (2016) exploit daily variations in taxi time at airports in California to model exposure to carbon monoxide and estimate its effects on emergency admissions. They find that daily pollution increments raise hospitalisation rates for respiratory and heart-related conditions. To our knowledge, the current study is the first to adopt an instrumental variable approach using UK data.

Among the most common air pollutants, particulate matter (PM) is responsible for the greatest risk for human health (WHO, 2013). Its adverse impact is due to its various toxic components and depends on its size. As discussed in Chapter 2, the smaller the size of the diameter of the particles, the deeper they can travel into the lungs. The most dangerous particles are $\text{PM}_{2.5}$ and $\text{PM}_{0.1}$\footnote{$\text{PM}_{2.5}$ and $\text{PM}_{0.1}$ are particles with a maximum diameter of 2.5 and 0.1 $\mu$m (micron), respectively.} that can transfer into the alveolus, thus reaching the circulatory system (Brunekreef and Holgate, 2002).

The previous chapters have looked at either the combined adverse effects of exposure to air and noise pollution (Chapter 3) or at the health effects of noise (Chapter
This chapter primarily focuses on short-term exposure to air pollution in the form of fine particulate matter (PM$_{2.5}$) and its effects on hospitalisations. Although this study does not assess noise effects, the health categories involved are the same investigated in the previous chapters: nervous, circulatory and respiratory. While the last two are the ones traditionally associated with air pollution exposure, the inclusion of nervous conditions needs some further justification.

There is growing evidence that exposure to PM triggers effects on the central nervous system. For example, Wang et al. (2017) review the neurodevelopmental and neurodegenerative diseases induced by inhalation of PM. Through eyes, nose and mouth PM can reach the central nervous system and activate different responses. The main reactions studied are systemic inflammations (which lead to stroke, neurodegenerative disease and sickness behaviour) and toxicity mechanisms due to the physical and chemical composition of the particles that directly reach the brain (Block and Calderón-Garcidueñas, 2009). The experimental results are mainly obtained under chronic exposure to fine particulates (Heusinkveld et al., 2016). This study focuses on short-term exposure to PM$_{2.5}$ (which readings are routinely recorded compared to PM$_{0.1}$) and tries to shed some light on its acute health effects.

The rest of the chapter is structured as follows: Section 5.2 explains the identification strategy, Section 5.3 provides a detailed description of the data used in this study and Section 5.4 presents and discusses the results - Appendix 5.A provides some additional information. Section 5.5 summarises and concludes the study.

### 5.2 Method

To estimate the causal relationship between short-run daily variations in fine particulate matter and hospitalisations, we start by introducing the following OLS regression
Chapter 5. Air Pollution and Hospital Admissions: an IV Approach

\[ Y_{kt} = \alpha + \sum_{\tau=t-1}^{t+1} \beta_{\tau} PM_{2.5k\tau} + \sum_{\tau=t-1}^{t+1} WEATHER_{k\tau} + X'_{kt}\eta + \lambda_t + \gamma_k + \epsilon_{kt} \] (5.1)

where the dependent variable \( Y_{kt} \) is the logarithm of the number of visits per geographical unit \( k \) at time \( t \). Specifically, the rate of hospitalisations is calculated over a 3-day period, which consists of day \( t \) and the following two days. This correction reduces the plausible effect of morbidity misplacement due to the short-run effect of pollution fluctuation. We split the sample by elective and emergency hospitalisations, as well as by different classes of diagnosis: nervous, circulatory and respiratory. The main coefficient of interest is \( \beta_t \), the coefficient on \( PM_{2.5} \) levels at time \( t \). By including the lead and lag on \( PM_{2.5} \), we control for the influence of the following and the previous day pollution variation. We include weather fixed effects with \( WEATHER_{kt} \) which consists of indicators for daily wind speed deciles, daily maximum and minimum temperature (divided in 3 degree Celsius-wide bins) as well as total daily rain deciles. Weather data is lagged and leaded by one period as it is done for pollution data. \( X_{kt} \) represents a vector of covariates that vary by Local Authority Districts (LADs) and time (in particular by year), which consist of indicators for the age group (0 to 4, 5 to 19, 20 to 49, 50 to 64 and over 64), gender and interaction dummies between age and gender. The regression also includes time \( (\lambda_t) \) and region \( (\gamma_k) \) fixed effects, which consist of the commonly used day of the week, month and year dummies as well as LAD indicators.

Estimates of all equations are weighted by the number of population by LAD, age group and gender and have LAD clusters-robust standard errors. The weights help to maximise the efficiency of the estimator and to produce the same coefficients to those potentially obtained using the underlying microdata sample. The standard errors

\[ \text{In this and the following equations, } PM_{2.5} \text{ levels are scaled such as } 10 \, \mu g/m^3 \text{ corresponds to the unit of } PM_{2.5}. \]

\[ \text{A Local Authority District (LAD) is a broader region than the LSOA or MSOA already mentioned in this thesis. In England there are 326 districts, which account for populations between 25,000 to 1,1 million.} \]
are robust to heteroskedasticity as well as to within unit of observation (i.e. LAD, age group and gender) correlation since we found evidence of both sources of bias. In fact, the Breusch-Pagan test for heteroskedasticity rejected the null hypothesis of constant variance for all types of outcome. In addition, an analysis of the intraclass correlation coefficients (of both, covariate of interest and errors) highlighted the presence of within unit of observation standard error correlation.

As we discussed in Chapter 2, pollution is endogenous and introduces error bias. Another related issue is the bias due to omitted variables, which are crucial to capture a causal effect. In this work, these refer to preferences and behaviours of the population that modify their exposure to adverse environmental factors. A common solution to these concerns is the adoption of an instrumental variable (IV) approach.

We adopt an instrument suggested by Deryugina et al. (2016), daily variations in local wind direction, which changes exposure to particulate matter. Although Figure 5.4 will be discussed later in the chapter (see page 125), it is useful to briefly mention it here to explain this choice of the instrument. Panel (a) of Figure 5.4 shows that local wind generally increases the local levels of PM$_{2.5}$. This figure represents an econometric depiction of the fact that PM$_{2.5}$ is transported by wind, which we treat as an exogenous variable. Deryugina et al. (2016) employ this instrument for US data on hospitalisations for the elderly. Our study looks at the entire population within the local regions that include PM$_{2.5}$ monitoring stations and we split the health data series by condition category.

In practice, this instrument translates into four instruments, one wind direction indicator for each quadrant of wind direction: (1) 0 to 90 degrees or North/North-East/East, (2) 90 to 180 degrees or East/South-East/South, (3) 180 to 270 degrees or South/South-West/West and (4) 270 to 360 degrees or West/North-West/North. The instruments are calculated as an average across all monitoring stations within LADs. Having constant instruments reduces the measurement error of the estimates of the effect of local wind direction on local pollution levels. More specifically, local
sources of pollution have less influence on pollution levels if wind direction and pollution readings are averaged within LAD. Without imposing a local constant effect, only a portion of the population might live downwind from a local source of pollution, hence adding bias to the estimation of the health effects of pollution exposure.

The IV estimator that identifies the Local Average Treatment Effect (LATE) is the ratio of the instrument coefficient of a reduced form equation over the instrument coefficient of a first stage equation. The former is the regression of the outcome on the instrument while the latter is the regression of the endogenous variable on the instrument. The estimates of the IV are carried out using the two-stage least squares (2SLS) procedure. In practice, this procedure first calculates the first stage regression. Subsequently, the 2SLS calculates a different regression of the outcome on the newly produced coefficient, which represents the instrumented endogenous variable. This procedure allows to obtain the correct standard errors, to add control variables and to include multiple instruments, as in our case.

In our context of heterogeneous treatment effects, the estimation of LATE with IV is subject to four conditions: (i) **first stage**, the instrument has a causal effect on the endogenous variable (in this case, PM$_{2.5}$); (ii) **exclusion restriction**, the instrument affects the outcome only through its effect on the endogenous variable; (iii) **independence assumption**, the instrument is uncorrelated with the omitted variables, which means that it is as good as randomly assigned; (iv) **monotonicity**, within this context of a multivalued treatment variable (i.e. PM$_{2.5}$), this assumption implies that a change in the instruments value causes all individuals to either increase treatment intensity, or to be unaffected.

To check that the selected instruments respect the first IV assumption on the relationship between the instrument and the endogenous variable, we calculate the first stage equation. This allows to see if there is a strong relationship between wind

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4Here the so-called “treatment variable” is the PM$_{2.5}$ endogenous variable.
direction and pollution levels. This regression represents the effect of the instruments on pollution levels and is estimated with the following equation.

\[ PM_{2.5kt} = \alpha + \sum_{\tau=t-1}^{t+1} \sum_{b=1}^{3} \phi_{b\tau} WINDDIR_{kt}^{90b} + \sum_{\tau=t-1}^{t+1} WEATHER_{k\tau} + X'_{kt} \eta + \lambda_t + \gamma_k + \epsilon_{kt} \]  

(5.2)

where \( WINDDIR_{kt}^{90b} \) are the indicators of wind direction quadrant and \( b \) can be any integer between 1 to 4. The \( b^{th} \) indicator is equal to 1 if the daily average wind direction in LAD \( k \) falls within the 90-degree interval \([90b - 90, 90b)\) and 0 otherwise. These indicators at time \( t \) are the excluded instruments. The omitted category is the last quarter of wind direction (i.e. \([270, 360)\), with \( b \) equal to 4) when wind blows from West/North-West/North. The rest of the equation, the weights and the standard errors are the same as in equation (5.1).

Employing four instruments to model wind direction restricts the effect of wind direction on pollution levels to be constant within each wind direction quadrant. We decide to follow the path suggested by Deryugina et al. (2016) and use this number of instruments in order to reduce the computational burden imposed by a higher number of instruments, which would also imply a proportional increase in the number of leaded and lagged \( WINDDIR \) variables. We investigate the adoption of different combinations of instruments, for instance Table 5.A2 in the Appendix shows the estimates of equation (5.2) for the main model with wind direction quadrants as well as for a modified model with 10-degree wind direction bins.

To disentangle the exclusion restriction assumption, we can compare the first stage with the reduced form. For this study, the latter consists of the OLS regression of the health outcomes on the instrument and the covariates included in equation (5.1). The following equation represents the reduced form.

\[ Y_{kt} = \alpha + \sum_{\tau=t-1}^{t+1} \sum_{b=1}^{3} \phi_{b\tau} WINDDIR_{kt}^{90b} + \sum_{\tau=t-1}^{t+1} WEATHER_{k\tau} + X'_{kt} \eta + \lambda_t + \gamma_k + \epsilon_{kt} \]  

(5.3)
where the dependent variable is again the logarithm of the number of visits per geographical unit $k$ at time $t$. The right-hand side of the equation is identical to equation (5.2). The aim of the reduced form is to estimate the effect of the instruments on the outcome. The results of equations (5.2) and (5.3) will be discussed in Section 5.4.

To test for the independence assumption, we check covariate balance with the instruments switched off or on, i.e. either equal to 0 or to 1. Table 5.1 reports the covariates related to individuals’ characteristics. No change is above 0.6 percentage points of the average, therefore we can conclude that overall there is an equal share of LAD characteristics between inactive and active indicators of wind direction. The third assumption on the instruments being randomly assigned seems to be satisfied.

The final assumption on monotonicity needs to be applied to each instrument of this IV model. When the treatment is multi-valued, as in our case of pollution levels, instead of the classical LATE, the IV estimator measures an “average causal response” (ACR) (Angrist and Imbens, 1995). However, if monotonicity does not hold, the estimates are uninterpretable. This study uses local LAD averages of pollution levels as well as local LAD changes in wind direction. Consequently, a change in wind direction for a given LAD will have a monotonic impact on PM$_{2.5}$ intensity for all age groups and gender units within the same LAD.

Using the notations introduced by Angrist and Pischke (2008), let $D_{1i}$ be $i$’s treatment

### Table 5.1: Covariate balance for different wind direction instruments.

<table>
<thead>
<tr>
<th>Category</th>
<th>Wind blowing from:</th>
<th>$N/NE/E$</th>
<th>$E/SE/S$</th>
<th>$S/SW/W$</th>
<th>$W/NW/N$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0 0</td>
<td>1 1</td>
<td>0 0</td>
<td>1 1</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td>0 to 4</td>
<td>5 to 19</td>
<td>20 to 49</td>
<td>50 to 64</td>
</tr>
<tr>
<td>0 to 4</td>
<td></td>
<td>7,647.6</td>
<td>19,827.9</td>
<td>28,147.8</td>
<td>23,746.5</td>
</tr>
<tr>
<td>5 to 19</td>
<td></td>
<td>7,634.8</td>
<td>19,761.8</td>
<td>28,090.6</td>
<td>23,776.4</td>
</tr>
<tr>
<td>20 to 49</td>
<td></td>
<td>7,635.3</td>
<td>19,770.7</td>
<td>28,042.3</td>
<td>23,727.0</td>
</tr>
<tr>
<td>50 to 64</td>
<td></td>
<td>7,659.0</td>
<td>19,879.6</td>
<td>28,174.3</td>
<td>23,770.3</td>
</tr>
<tr>
<td>over 65</td>
<td></td>
<td>7,621.7</td>
<td>19,755.2</td>
<td>28,152.3</td>
<td>23,705.7</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td>0 to 4</td>
<td>5 to 19</td>
<td>20 to 49</td>
<td>50 to 64</td>
</tr>
<tr>
<td>0 to 4</td>
<td></td>
<td>7,343.1</td>
<td>19,025.4</td>
<td>27,637.6</td>
<td>23,501.2</td>
</tr>
<tr>
<td>5 to 19</td>
<td></td>
<td>7,336.4</td>
<td>19,014.2</td>
<td>27,712.8</td>
<td>23,496.6</td>
</tr>
<tr>
<td>20 to 49</td>
<td></td>
<td>7,389.2</td>
<td>19,018.5</td>
<td>27,671.8</td>
<td>23,456.6</td>
</tr>
<tr>
<td>50 to 64</td>
<td></td>
<td>7,337.7</td>
<td>19,031.9</td>
<td>27,642.6</td>
<td>23,526.5</td>
</tr>
<tr>
<td>over 65</td>
<td></td>
<td>7,337.2</td>
<td>19,054.2</td>
<td>27,654.6</td>
<td>23,504.2</td>
</tr>
</tbody>
</table>
status when the wind direction instrument is switched on (i.e. when $Z_i = 1$) and $D_{0i}$ be $i$’s treatment status when the instrument is turned off (i.e. when $Z_i = 0$). In our case, we assume monotonicity holds for $D_{1i} \geq D_{0i}$. In practice, this would mean that short-run daily wind variation affects residents’ health negatively or not at all, and it does not improve their health.

5.3 Data

5.3.1 Air Pollution and Atmospheric Conditions

Environmental data was obtained through the London Air Quality Network managed by the Environmental Research Group at King’s College London, which provides hourly data on several pollutants as well as atmospheric conditions like temperature, rain, wind speed and wind direction among others. We took into consideration one pollutant: particulate matter with a diameter below 2.5 $\mu g/m^3$ ($PM_{2.5}$). As we mentioned in Section 5.1, $PM_{2.5}$ represents a major risk for human health compared to other measures of pollution (WHO, 2013).

Data is gathered by air pollution monitors in England. We selected 105 monitors that have enough information on the environmental variables of interest. These monitoring sites lie within three different government regions and twenty-two Local Authority Districts (LADs): 8 sites sit within the boundaries of East of England (3 LADs), 24 within South East (8 LADs) and 73 within Greater London (11 LADs). Figure 5.1 displays the position of monitoring stations within their correspondent LADs in England. On average there are four monitoring stations per LAD.

Wind direction is provided in the form of the degree from which the wind blows. For instance, this means that a value of 90 degree corresponds to wind blowing from East towards West. We create four indicators, one for each quadrant of wind direction.

Environmental data covers the period from 2008 to 2014 to match the period of the
data series on hospital admissions. LAD-level pollution and weather measures were created averaging across daily means of the hourly monitor readings recorded within the LAD.

The annual average limit set by WHO for PM$_{2.5}$ is 25 $\mu$g/m$^3$ (WHO, 2000). Figure 5.2 shows the monthly and yearly aggregated trends in PM$_{2.5}$. For the monitored areas included in this study, there is no breach of this threshold and annual averages are relatively low around 15 $\mu$g/m$^3$. One unit of PM$_{2.5}$ corresponds to around 15 percent of the average concentration during the period 2008-2014. Monthly averages express more variations with four peaks above WHO’s ceiling. These peaks are visible also in the graphs by LAD of Figure 5.3. We produced the same graphs for the annual averages in PM$_{2.5}$, which are not shown here, and none of the LAD exceeds the internationally set limit$^5$. This shows a common trend of a few high

Figure 5.1: Twenty-two selected LADs (shaded) with monitoring stations (dots) in England.

$^5$The only LAD that gets close to the annual limit is Westminster with a PM$_{2.5}$ annual average of 24.56 $\mu$g/m$^3$ in 2011. Figure 5.3 shows that this LAD is characterised by relatively high readings of PM$_{2.5}$ monthly averages.
Figure 5.2: Monthly and annual trends in PM$_{2.5}$ air pollution, 2008-2014.

The dashed line represents the annual WHO’s limit of 25 µg/m$^3$.

Figure 5.3: Monthly trends in PM$_{2.5}$ air pollution by LAD, 2008-2014.

The dashed line represents the annual WHO’s limit of 25 µg/m$^3$. 
### Table 5.2: Summary statistics, 2008-2014.

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Std Dev</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM 2.5 (µg/m³)</td>
<td>15.03</td>
<td>10</td>
<td>-1.13</td>
<td>86.42</td>
</tr>
<tr>
<td>LAD population, tot</td>
<td>179,286.1</td>
<td>50,506.97</td>
<td>76,192</td>
<td>266,924</td>
</tr>
<tr>
<td>LAD population, 0-4</td>
<td>13,980.83</td>
<td>4,627.95</td>
<td>5,131</td>
<td>22,966</td>
</tr>
<tr>
<td>LAD population, 5-19</td>
<td>35,791.78</td>
<td>10,780.86</td>
<td>16,280</td>
<td>54,258</td>
</tr>
<tr>
<td>LAD population, 20-49</td>
<td>52,174.19</td>
<td>21,165.72</td>
<td>17,413</td>
<td>11,0836</td>
</tr>
<tr>
<td>LAD population, 50-64</td>
<td>45,385.16</td>
<td>12,947.05</td>
<td>18,734</td>
<td>71,086</td>
</tr>
<tr>
<td>LAD population, over 65</td>
<td>31,954.16</td>
<td>8,594.96</td>
<td>17,740</td>
<td>52,603</td>
</tr>
<tr>
<td>LAD population, female</td>
<td>88,910.36</td>
<td>24,868.06</td>
<td>38,517</td>
<td>133,005</td>
</tr>
<tr>
<td>LAD population, male</td>
<td>90,375.77</td>
<td>25,721.47</td>
<td>37,597</td>
<td>139,080</td>
</tr>
<tr>
<td>Nervous 3-day admission rate, elective</td>
<td>0.89</td>
<td>0.65</td>
<td>0.18</td>
<td>12.05</td>
</tr>
<tr>
<td>Nervous 3-day admission rate, emergency</td>
<td>0.72</td>
<td>0.47</td>
<td>0.18</td>
<td>8.65</td>
</tr>
<tr>
<td>Circulatory 3-day admission rate, elective</td>
<td>1.9</td>
<td>11.24</td>
<td>0.18</td>
<td>1,423.62</td>
</tr>
<tr>
<td>Circulatory 3-day admission rate, emergency</td>
<td>1.7</td>
<td>1.48</td>
<td>0.18</td>
<td>14.86</td>
</tr>
<tr>
<td>Respiratory 3-day admission rate, elective</td>
<td>0.88</td>
<td>0.68</td>
<td>0.18</td>
<td>12.32</td>
</tr>
<tr>
<td>Respiratory 3-day admission rate, emergency</td>
<td>1.86</td>
<td>1.89</td>
<td>0.18</td>
<td>33.22</td>
</tr>
</tbody>
</table>

Unit of observation is LAD-age group-gender-day. All rates are per 10,000 population in the relevant subsample.

Values over the year but an overall abiding by the WHO’s annual limit.

#### 5.3.2 Hospital Admissions

The measure of health adopted in this study is the same as used in Chapter 3, hospital admission rates by a unit of aggregation, which exploits the features of the Hospital Episode Statistics (HES) administrative data. The unit of aggregation used in our analysis is formed by the LAD population stratified by age group and gender. The health measure is formed by daily hospital admission rates by unit of aggregation. For each quarter of the year, we calculate the number of visits for a specific diagnosis group and each unit of aggregation. The types of diagnosis selected are those suggested by the epidemiological and environmental literature and are the same used throughout this thesis: nervous, circulatory and respiratory conditions. Date of visit, type of admission, age, gender, primary diagnosis and LAD of residence are obtained for all patients from 2008 to 2014.

Our main estimation sample consists of 1,210,320 observations at the LAD-age
group-gender-day level. This figure reduces to 928,578 when merging the health
dataset with the environmental data series. Table 5.2 presents descriptive statistics
for the population sample. There are on average 180,000 residents in each LAD.

Table 5.2 also reports the daily mean concentration of PM$_{2.5}$, which is 15.03 mi-
icrograms per cubic meter with a standard deviation of 10. The minimum PM$_{2.5}$
value is negative but small. This is due to technical characteristics of the monitoring
stations$^6$ and means that the pollution level is 0 or very close to 0. Negative values in
the sample represent 0.02% of the total readings and we decided to keep the original
values in order not to influence the measurement error of the monitoring devices
only for a portion of the observations.

The average 3-day admission rate for the nervous class is relatively low with 0.89
and 0.72 visits per 10,000 residents within the unit of aggregation for elective and
emergency visits, respectively. The rates are higher especially for the circulatory
class, that registers some peaks (6 in total) above 1,000 visits across three days for a
sample of 10,000 people. All these extreme events concentrate in September 2009
and we could not identify the reason for this. However, the rate of elective circulatory
visits grows almost continuously from the minimum of 0.18 to the maximum of 1,423.
It is therefore difficult to pick a cut-off above which we can consider the number of
events to be out of the ordinary and we decided to keep all observations.

5.4 Results and Discussion

We begin by graphically analysing the first stage equation (5.2) and reduced form
equation (5.3). Figure 5.4 is based on the use of more instruments than in the
reported estimates below, in order to give a more detailed graphical picture. We use
10-degree bins wind direction instead of the 90-degree we use when implementing

$^6$“Sometimes a fine particulate PM$_{2.5}$ monitor will report negative values for pollution. A PM$_{2.5}$
monitor may have recorded a loss of material from its filter to the air, for example, a very minute
amount of moisture may have evaporated during the sample period resulting in a small negative
value” (http://www.mass.gov/eea/docs/dep/air/aqi/aqi.htm).
Chapter 5. Air Pollution and Hospital Admissions: an IV Approach

the 2SLS\(^7\). Panel (a) of Figure 5.4 displays the pollution wind direction pattern from 2008 to 2014 for residents of the aforementioned 22 LADs. The figure clearly shows that when wind blows from East/South-East (i.e. from the sea and the European continent) and North-West/North (i.e. from inside England) the levels of PM\(_{2.5}\) are at their highest.

Panel (b) of Figure 5.4 displays average 3-day hospitalisation rates by wind direction for the same sample used to construct panel (a). This panel is a graphical depiction of the reduced form, which is the regression of the dependent variable on the instruments and any covariates in the model. Panel (b) shows that the largest effects on visits are for emergency nervous conditions when wind blows from North-West/North. This figure also shows that the peaks around East/South-East shown in the first panel disappear. There is an environmental explanation for this lack of parallelism between the first stage and reduced form trends.

Wind coming from East/South-East comes from the sea and carries minerals and sea salt from that area. These are considered innocuous for human health and the European Commission suggests to deduct the contribution of this natural particulate

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\(^7\)The estimates of the 2SLS using the different sets of instruments yield similar results. Table 5.A2 in the Appendix shows the first stage estimates with both options: 90-degree and 10-degree indicators.
matters from the total levels of PM$_{2.5}$ (Commission Staff Working Paper, 2011). In the area around London, Walton et al. (2015) attributed to sea salt a share of 0.55 µg m$^{-3}$ for the year 2010. Interestingly, this is the level of the peak of PM$_{2.5}$ in panel (a) of Figure 5.4. Indeed, wind carries particles from the sea, which are not harmful to health, as it is shown in panel (b) of Figure 5.4 where the peaks disappear.

With the PM$_{2.5}$ natural share reduction, the reduced form of the nervous emergency visits parallels the wind direction pattern in PM$_{2.5}$, suggesting the two patterns are closely related. Because individuals’ exposure to daily variations in wind direction is unlikely to be related to their latent preferences, we can conclude that the only reason for the hospital visits variations are the PM$_{2.5}$ variations in the wind direction pattern. Remarkably, this can be seen as the proof of the exclusion restriction assumption, which states that for wind direction to be a good instrument, the only way wind direction can affect hospitalisations is through exposure to pollution.

Figure 5.A1 in the Appendix, similarly to Figure 5.4, shows first stage and reduced form representations but splits the sample by LAD. From these more refined plots it is harder to see as clear patterns as in Figure 5.4 and our preferred model remains the one that keeps the LADs pooled together.

As we mentioned in the previous section, the IV estimator can be seen as the ratio of the instrument coefficient of a reduced form equation over the instrument coefficient

### Table 5.3: OLS and IV estimates of the effect of a 10-µg/m$^3$ increase of PM$_{2.5}$ on (logarithm of) 3-day hospital admissions per diagnosis class.

<table>
<thead>
<tr>
<th></th>
<th>Nervous</th>
<th>Elective</th>
<th>Respiratory</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
<th>Emergency</th>
<th>Circulatory</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>OLS estimates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.006</td>
<td>0.004</td>
<td>0.001</td>
<td>-0.002</td>
<td>-0.002</td>
<td>-0.004*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.004)</td>
<td>(0.004)</td>
<td>(0.004)</td>
<td>(0.004)</td>
<td>(0.003)</td>
<td>(0.002)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>72751</td>
<td>113010</td>
<td>68018</td>
<td>49816</td>
<td>133350</td>
<td>191777</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.240</td>
<td>0.402</td>
<td>0.323</td>
<td>0.409</td>
<td>0.665</td>
<td>0.618</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>2SLS IV estimates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.003</td>
<td>-0.033*</td>
<td>-0.002</td>
<td>0.039**</td>
<td>-0.007</td>
<td>-0.007</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.017)</td>
<td>(0.019)</td>
<td>(0.018)</td>
<td>(0.020)</td>
<td>(0.014)</td>
<td>(0.014)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>72751</td>
<td>113010</td>
<td>68018</td>
<td>49816</td>
<td>133350</td>
<td>191777</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>208.304</td>
<td>170.506</td>
<td>191.250</td>
<td>171.582</td>
<td>337.322</td>
<td>257.255</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
of a first stage equation. Consequently, the IV estimator will be proportional to
the coefficient of the instrument in the reduced form. From panel (b) of Figure 5.4,
we can expect to see some effects for emergency nervous hospital admissions. This
would reflect the assumption that, due to the short-term exposure measured in this
study, the statistically significant results will be for emergency inpatients.

Table 5.3 reports the estimates of the OLS equation (5.1) as well as of the 2SLS
estimator\(^8\). The Wu-Hausman test for endogeneity was performed and the null of
exogenous variables was rejected at the 5% significance level. We also perform the
IV analysis using the limited information maximum likelihood (LIML) estimator,
which is an alternative to 2SLS. It is widely used when the IV is over-identified (as
in our case, with four instruments) and has a smaller bias than the 2SLS estimator
(Cameron and Trivedi, 2010). We find that LIML IV estimates, which are not
reported here, do not differ to those of the 2SLS model. This provides some evidence
of the absence of bias in the main model used in this study.

The first panel of Table 5.3 for OLS estimates shows statistically significant results
only for emergency respiratory visits. Although the sign is still negative, the effect
disappears when estimating 2SLS. Also the second panel of Table 5.3 for 2SLS
IV estimates reports a negative coefficient. In this case, this corresponds to a
3.3% decrease for elective circulatory visits over the three days after a 10-µg/m\(^3\)
increase in PM\(_{2.5}\), which is statistically significant at the 90% confidence level. More
interpretable and with a higher confidence level is the coefficient on emergency
nervous visits. The 3-day visit rate of this class raises by 3.9% when day-one registers
a 10-µg/m\(^3\) increase in PM\(_{2.5}\). F-statistics are generally very high, which suggests
that there are no weak instrument problems (Stock et al., 2002).

While the positive result on the emergency nervous class corresponds to our expec-
tations, the statistically significant negative estimate of elective circulatory visits
is puzzling. In fact, in the epidemiological tradition, the cardiovascular class is the

\(^8\)Table 5.A1 in the Appendix reports the full set of estimated coefficients for the 2SLS IV model.
main class associated with air pollution effects. In Chapter 3, we faced the same issue and we addressed it by observing the interaction between elective and emergency admissions within each diagnosis class.

We recall that the study developed in Chapter 3 exploits the same HES data series but uses a different approach to the one we use in this chapter. Chapter 3 looks at the variation in hospital admissions for people living close to airports and adopts a log-linear OLS model. Exploring the interplay between proximity to airport and type of admission, Chapter 3 finds an overall increase of admissions for the nervous conditions with some preference towards elective admissions. On the contrary, there is statistically significant migration from elective to emergency admissions for circulatory and respiratory conditions. Although we cannot apply the same identification strategy to the current study, we can find another way to explore this matter consistently to the IV model.

In order to explore the interaction between types of hospital admission, we introduce a new 2SLS model, which has exactly the same first stage equation of the main model (see equation (5.2)) but which has a different reduced form compared to equation (5.3). The only alteration of the new reduced form is a different outcome variable. Rather than the 3-day admission rates of the main model (and equation (5.3)), we employ the ratio of elective over emergency 3-day hospital admissions.

Table 5.4 reports the results of the new estimates. The coefficients of interest have

<table>
<thead>
<tr>
<th>Variable</th>
<th>Elective/emergency visits rate (%)</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM 2.5</td>
<td>0.008</td>
<td>-0.037</td>
<td>-0.024</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.031)</td>
<td>(0.024)</td>
<td>(0.024)</td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>39,742</td>
<td>115,156</td>
<td>84,133</td>
<td></td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.126</td>
<td>0.233</td>
<td>0.301</td>
<td></td>
</tr>
</tbody>
</table>

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
all non statistically significant estimates but we can still infer something from their magnitude. For the nervous, the positive coefficient indicates an increase, although small, of elective visits over emergency. The pattern changes for the other two classes of conditions. For both, circulatory and respiratory conditions, the negative coefficients point towards a decrease of elective admissions in favour of emergency visits. This is the same scenario we find in Chapter 3 and confirms the idea that short-term exposure to environmental factors substitutes planned with unplanned hospital visits for circulatory and respiratory conditions. Although we do not find evidence of this migration pattern for nervous conditions, this class is characterised by increases in both types of admissions.

5.4.1 Results by Gender and Age Groups

To explore the effect of PM\textsubscript{2.5} exposure by sub-samples of the LAD population, we modify the 2SLS model to apply it to the different age groups and gender categories. Table 5.5 and Table 5.6 report the IV estimates for these categories.

From Table 5.5, it is clear that the positive and significant results of the main model in Table 5.3 are driven by the female subsample. For this group, a 10-\(\mu\)g/m\textsuperscript{3} increase in PM\textsubscript{2.5} induces an increase in nervous visits by 2.9 and 4.5 percentage points for

<table>
<thead>
<tr>
<th></th>
<th>Elective</th>
<th>Emergency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nervous (1)</td>
<td>Circulatory (2)</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.029*</td>
<td>-0.033</td>
</tr>
<tr>
<td>(0.016)</td>
<td>(0.023)</td>
<td>(0.018)</td>
</tr>
<tr>
<td>Obs</td>
<td>37632</td>
<td>48740</td>
</tr>
<tr>
<td>F-statistic</td>
<td>119.272</td>
<td>112.672</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>-0.022</td>
<td>-0.035</td>
</tr>
<tr>
<td>(0.026)</td>
<td>(0.023)</td>
<td>(0.026)</td>
</tr>
<tr>
<td>Obs</td>
<td>35119</td>
<td>64270</td>
</tr>
<tr>
<td>F-statistic</td>
<td>132.293</td>
<td>106.331</td>
</tr>
</tbody>
</table>

Notes: * \(p < .1\), ** \(p < .05\), *** \(p < .01\). Cluster-robust standard errors in parentheses.
Table 5.6: 2SLS IV estimates of the effect of a 10-µg/m³ increase of PM$_{2.5}$ on (the logarithm of) 3-day hospital admissions per diagnosis class, by under or over 50 age groups.

<table>
<thead>
<tr>
<th></th>
<th>Elective</th>
<th></th>
<th></th>
<th>Emergency</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nervous</td>
<td>Circulatory</td>
<td>Respiratory</td>
<td>Nervous</td>
<td>Circulatory</td>
<td>Respiratory</td>
</tr>
<tr>
<td>Under 50 years old</td>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
<td>(5)</td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.025</td>
<td>-0.034</td>
<td>-0.028</td>
<td>-0.016</td>
<td>-0.024</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.029)</td>
<td>(0.028)</td>
<td>(0.019)</td>
<td>(0.021)</td>
<td>(0.022)</td>
<td>(0.017)</td>
</tr>
<tr>
<td>Obs</td>
<td>34870</td>
<td>30258</td>
<td>36430</td>
<td>23720</td>
<td>27040</td>
<td>61159</td>
</tr>
<tr>
<td>F-statistic</td>
<td>77.896</td>
<td>47.866</td>
<td>89.526</td>
<td>67.318</td>
<td>110.279</td>
<td>101.340</td>
</tr>
<tr>
<td></td>
<td>50 years old and above</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.001</td>
<td>-0.033*</td>
<td>-0.026</td>
<td>0.023</td>
<td>-0.000</td>
<td>0.009</td>
</tr>
<tr>
<td></td>
<td>(0.019)</td>
<td>(0.019)</td>
<td>(0.018)</td>
<td>(0.022)</td>
<td>(0.014)</td>
<td>(0.013)</td>
</tr>
<tr>
<td>Obs</td>
<td>31661</td>
<td>57597</td>
<td>23336</td>
<td>27369</td>
<td>71929</td>
<td>70376</td>
</tr>
<tr>
<td>F-statistic</td>
<td>156.909</td>
<td>147.092</td>
<td>92.261</td>
<td>106.990</td>
<td>161.414</td>
<td>146.993</td>
</tr>
</tbody>
</table>

Notes: * p < .1, ** p < .05, *** p < .01. Cluster-robust standard errors in parentheses.

the elective and the emergency method of admission, respectively (although only at the 10% significance level). Although the coefficients for the elective circulatory class for the gender subsamples have the same magnitude for the main sample as shown in Table 5.3, their standard errors are larger and the estimates are no longer statistically significant.

Table 5.6 reports the results of the 2SLS estimation by two broad age subsamples: under and equal or over 50 years old. Interestingly, it shows that the increase in hospitalisations for emergency nervous conditions is motivated by the attendance of the younger sub-group. In fact, this group could be highly sensitive to stress due to some awareness towards the external environment as well as due to the exposure to other sources of stress, such as work or personal circumstances. On the contrary, the reduction in elective circulatory visits is a characteristic of the older group. Although we do not find any statistically significant results for the correspondent emergency outcome, this last finding could be interpreted within the frame of the substitution of hospital admissions from elective to emergency visits.
Table 5.7: 2SLS IV estimates of the effect of a 10-µg/m³ increase of PM$_{2.5}$ on (the logarithm of) 3-day hospital admissions per diagnosis class, with different numbers of leads and lags of all instruments and weather variables.

<table>
<thead>
<tr>
<th></th>
<th>Nervous</th>
<th>Elective</th>
<th>Respiratory</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main: 1 lead 1 lag</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.003</td>
<td>-0.033*</td>
<td>-0.002</td>
<td>0.039**</td>
<td>-0.007</td>
<td>-0.007</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>72751</td>
<td>113010</td>
<td>68018</td>
<td>49816</td>
<td>133350</td>
<td>191777</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>208.304</td>
<td>170.506</td>
<td>191.250</td>
<td>171.582</td>
<td>337.322</td>
<td>257.255</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>No leads no lags</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.002</td>
<td>-0.029***</td>
<td>-0.020***</td>
<td>0.007</td>
<td>-0.009</td>
<td>-0.009</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>129423</td>
<td>158741</td>
<td>127381</td>
<td>96551</td>
<td>166666</td>
<td>246843</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>965.206</td>
<td>802.207</td>
<td>893.239</td>
<td>810.221</td>
<td>1059.360</td>
<td>1222.865</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>2 leads 2 lags</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>0.022</td>
<td>0.001</td>
<td>0.008</td>
<td>0.069*</td>
<td>-0.011</td>
<td>-0.002</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>31565</td>
<td>74802</td>
<td>26297</td>
<td>18522</td>
<td>107462</td>
<td>147656</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>93.557</td>
<td>138.388</td>
<td>79.037</td>
<td>97.662</td>
<td>305.339</td>
<td>298.581</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>3 leads 3 lags</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM 2.5</td>
<td>-0.098</td>
<td>-0.019</td>
<td>-0.061</td>
<td>0.002</td>
<td>-0.024</td>
<td>0.022</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>15428</td>
<td>52319</td>
<td>11992</td>
<td>8053</td>
<td>93173</td>
<td>121813</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>37.243</td>
<td>58.001</td>
<td>24.860</td>
<td>33.087</td>
<td>179.082</td>
<td>177.514</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.

### 5.4.2 Robustness Checks

Table 5.7 shows the 2SLS estimates with different numbers of leads and lags of the instruments and of the environmental variables. Removing leads and lags shows only negative statistically significant results for elective admissions. Therefore, not controlling for previous and next day weather conditions highlights the PM$_{2.5}$ short-term exposure effect on planned visits, which we interpret as a reduction of elective admissions in favour of emergency admissions.

Table 5.8: Placebo IV estimates of the effect of a 10-µg/m³ increase of PM$_{2.5}$ on (the logarithm of) 3-day hospital admissions per diagnosis class.

<table>
<thead>
<tr>
<th></th>
<th>Nervous</th>
<th>Elective</th>
<th>Respiratory</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
<th>Nervous</th>
<th>Circulatory</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM 2.5</td>
<td>-0.393</td>
<td>0.618</td>
<td>0.544</td>
<td>-0.226</td>
<td>0.505</td>
<td>0.016</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>72788</td>
<td>113065</td>
<td>68045</td>
<td>49839</td>
<td>133411</td>
<td>191844</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>2.602</td>
<td>1.121</td>
<td>1.475</td>
<td>1.688</td>
<td>0.395</td>
<td>0.937</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
Adding two leads and two lags reduces the significance level of all estimates, leaving a 6.9% increase in emergency hospital admissions due to a 10-µg/m³ rise in PM$_{2.5}$, which is significant at the 10% level. Adding one extra lead and lag, i.e. controlling for weather conditions on three days prior as well as the three days after the first day of exposure, removes all statistical significance of the estimates. Within our short-term exposure setting, this might indicate that the distance from the first day of measured environmental conditions (that affect pollution levels) to the last day of measured health outcome needs to be at most five days. This is consistent with the epidemiological definition of short-term exposure to pollution with being one to four days (see Dominici et al. (2003) among others).

We randomly generate instruments to control for some placebo effects. Table 5.8 shows the results of this test. F-statistics are all extremely low and no result is statistically significant, thus providing further evidence of the robustness of the wind direction instruments of the main model of this study.

### 5.5 Conclusion

Estimating the health effects of air pollution is crucial to understand the social burden of this environmental stressor. There are many obstacles to this process, from the endogeneity nature of pollution to the heterogeneity of its effects on population. Ignoring these issues leads to errors and biases in the assessment of the adverse effects of environmental exposure.

This chapter adopts an instrumental variable (IV) approach to estimate the causal effects of short-term exposure to air pollution and health outcomes. Specifically, we focus on hospital visits for nervous, circulatory and respiratory conditions from 2008 to 2014.

The population of interest are the residents within local regions hosting PM$_{2.5}$ monitoring stations. Pollution levels are instrumented by local daily variations of
wind direction. The underpinning idea is that daily wind direction variations are not predictable and depart from the average annual wind direction, hence crucially holding a random component.

Although this chapter builds on the work of Deryugina et al. (2016), it applies the methodology to a different context and uses a different health outcome. Their study uses US data and, due to data availability, they have access to information on all-cause hospitalisations for the elderly. On the contrary, our study looks at the entire population within the local regions that include PM$_{2.5}$ monitoring stations. Furthermore, our HES data contains information on the primary diagnosis of hospital admissions, which allows to split the health data series by condition category.

Using random alterations in daily wind direction, we find significant effects of pollution on hospitalisations. However, not all results are easily interpretable. Although we find statistically significant increases in visits for emergency nervous conditions, we also find significant reductions in elective circulatory visits. While emergency visits refer to those hospitalisations that result from visits to the emergency rooms, elective visits are planned and usually deal with chronic conditions. To explain this pattern, we detect some signs of a substitution effect of hospital admissions, from planned to unplanned visits especially for circulatory and respiratory conditions.
## 5.A Appendix

Table 5.A1: IV estimates of the effect of a 10-µg/m³ increase of PM$_{2.5}$ on (logarithm of) 3-day hospital admissions per diagnosis class, all estimated coefficients.

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### Chapter 5. Air Pollution and Hospital Admissions: an IV Approach

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Notes: * p < .1, ** p < .05, *** p < .01. Cluster-robust standard errors in parentheses.
Table 5.A2: First stage estimates of equation 5.2 for wind direction indicators either by 90-degree bins or 10-degree bins.

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<td>10deg bin=30</td>
<td>1.341***</td>
<td>(0.075)</td>
</tr>
<tr>
<td>10deg bin=31</td>
<td>-1.143***</td>
<td>(0.094)</td>
</tr>
<tr>
<td>10deg bin=32</td>
<td>0.460***</td>
<td>(0.098)</td>
</tr>
<tr>
<td>10deg bin=33</td>
<td>2.074***</td>
<td>(0.109)</td>
</tr>
<tr>
<td>10deg bin=34</td>
<td>7.920***</td>
<td>(0.220)</td>
</tr>
<tr>
<td>10deg bin=35</td>
<td>0.031</td>
<td>(0.174)</td>
</tr>
<tr>
<td>Observations</td>
<td>300,650</td>
<td></td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.601</td>
<td></td>
</tr>
</tbody>
</table>

Notes: * $p < .1$, ** $p < .05$, *** $p < .01$. Cluster-robust standard errors in parentheses.
(a) First stage. Average PM$_{2.5}$ levels by wind direction indicator.

(b) Reduced form. Average daily emergency hospitalisations by wind direction indicator.

Figure 5.A1: Graphical representation of the first stage and reduced form for IV estimates of the health effects of short-term exposure to PM$_{2.5}$ using 10-degree bins wind direction as instruments. By LAD. Wind from West as reference point.
Chapter 6

Conclusion

The three pieces of research introduced in the previous chapters take advantage of different data series and employ varied approaches to try and disentangle the same issue: estimating the causal link between environmental exposure, in terms of noise and air pollution, and health outcomes.

This chapter provides a summary of these studies. The next Section 6.1 compares and contrasts data, methods and findings of Chapter 3 (Living in the Proximity of Airports and Health), Chapter 4 (The Medical Cost of Air Traffic Pollution: Evidence from Changes in Flight Patterns) and Chapter 5 (Air Pollution and Hospital Admissions: an Instrumental Variable Approach). Section 6.2 concludes with implications of the findings and suggestions for future research.

6.1 Summary of Studies and Findings

The analyses of this dissertation point towards a causal estimation of the health effects of environmental exposure. The theoretical framework underpinning this thesis is based on the idea of health capital. The three studies help to quantitatively estimate the impact of environmental hazards on health capital accounting for sources of endogeneity bias and controlling for socioeconomic factors.
The three chapters adopt varied methodologies. The study described in Chapter 3 applies a simple log-linear OLS regression of quarterly hospitalisation rates on proximity to airports and a series of controls. The empirical design of Chapter 4 exploits a difference-in-differences approach (DD), which models the double difference between the changes in medication spending for the population exposed and for the population not exposed to air traffic variation before and after the changes occur. The focus of the last study revolves around the estimation of the change in hospitalisation rates as a consequence of daily fluctuations of the class of pollutants PM$_{2.5}$. To meet this target, we introduce daily changes of wind direction as the instrumental variable (IV). In Chapter 5, we show that this variable has the advantage of ruling out the endogeneity issues associated with pollution readings. Adding the instrument in a two-stage least squares (2SLS) regression, we are able to estimate the health effects of short-term exposure to air pollution.

The thesis uses two different kinds of health outcomes. The study on the effects of air traffic on health (Chapter 4) employs monthly data on medication spending by general practitioner (GP) practice (from 2011 to 2013). The other two studies, on the quarterly health effects of proximity to airports (Chapter 3) and health impact of daily variation in particulate matter (PM) levels (Chapter 5), work with the Hospital Episode Statistic (HES) dataset (for the 2008-2014 period). The HES dataset consists of administrative data on hospital admissions in England. Although there are differences in the data series used, the three chapters focus on the same three categories of health conditions: nervous, circulatory and respiratory.

The focus of the three studies shifts geographically from smaller to larger regions. The census units in Chapter 3 are Lower layer Super Output Areas (LSOAs), which on average include 650 households and a population of 1,500. Chapter 4 looks at broader regions, namely Middle layer Super Output Areas (MSOAs), which include LSOAs and enclose an average of 7,700 residents. The largest areas are included in Chapter 5; these are Local Authority Districts (LADs), that account for population between 25,000 to 1.1 million.
Although the samples of the three studies are different in terms of the observed outcome, the geography and the temporal aggregation, there is a common feature across all statistically significant results: conditions related to the central nervous system are the most affected by short-term exposure to both air and noise pollution. Chapter 3 shows that people living closer to airports experience an increase of 5.5% in quarterly nervous elective hospital visits compared to residents of more distant areas. Chapter 4 exploits a five-month trial at London Heathrow airport that resulted in an overall decrease in air traffic during sensitive hours. The general results of the DD analysis show a subsequent reduction in monthly GP spending for nervous conditions by 5.8%. Chapter 5 reports a 3.9% increase in 3-daily emergency hospitalisations for nervous ailments in response to a $10\mu g/m^3$ increase in PM$_{2.5}$.

The other two therapeutic classes, circulatory and respiratory, show mixed results across the three studies. In particular, for circulatory outcomes the estimates have an opposite sign to what is expected, and often are statistically significant. In Chapter 3 and Chapter 5 the signs of hospitalisation rates are negative, which is counterintuitive given that we want to estimate the adverse effect of air and noise pollution. In Chapter 4, although the setting presents a reduction in air traffic, the estimated monthly spending for circulatory conditions has a positive sign. For respiratory conditions there are fewer statistically significant results and we are not able to draw many conclusions.

All three strands of the research focus on short-term health effects of environmental exposure. The weak results for circulatory and respiratory conditions probably reflect the more long-term nature of these conditions that make it difficult to identify impacts from short-term changes as in our studies. Considering only the hospitalisation outcomes, Chapter 3 and Chapter 5 introduce a novel idea to formalise this hypothesis. The two studies model the interplay between elective and emergency admissions to assess a contemporaneous dynamic that may exist between the two types of admissions after environmental exposure. The model of Chapter 3 shows that there is a strong preference for emergency over elective admissions for circulatory
and respiratory conditions near airports. The same classes show a decrease of elective in favour of emergency visits in Chapter 5. Although the latter results are not statistically significant, the two models suggest that short-term exposure to environmental factors induces a substitution of planned with more expensive unplanned visits, specifically for circulatory and respiratory conditions.

6.2 Implications of Findings and Future Research

This work contributes to the existing literature on health and environmental economics by providing some estimates of the causal relationship between short-term air and noise pollution exposure and adverse health effects. Furthermore, this study has several policy implications. First, the results on nervous conditions raise awareness on the broad range of effects that air and noise pollution can trigger. The consistency of evidence found across the three studies represents a strong call for policy makers to safeguard citizens from adverse effects on the central nervous system within urban settings.

Second, the interplay between elective and emergency visits is a novelty of this thesis. For circulatory and respiratory conditions, we find that there is a substitution of elective with more expensive emergency visits for the population more exposed to air and noise pollution. This has an impact on the English public health service and deserves public attention and regulation.

Lastly, the dataset on monthly GP prescriptions used in Chapter 4 is a valuable yet under-utilised source of health-related information. Although this information is provided freely by the UK government, we could find only one paper that employs it (see Rowlingson et al. (2013)). Compared to hospital visits or other quantitative health measures, the data on prescriptions by condition class represent a crucial piece of information that allows us to specifically investigate on morbidity effects of air traffic exposure. Generally, medicine consumption affects people’s everyday life
and needs to be taken into account when estimating the social costs of environmental exposure.

Besides the contributions highlighted above, this work also carries weaknesses and leaves many open questions for future research. Most importantly, due to data limitations, we are not able to investigate the health effects on a series of population subsamples, such as people with existing conditions, which are considered among the most vulnerable groups (Brook et al., 2010; van Kamp and Davies, 2013).

Furthermore, although the air traffic trial exploited in Chapter 4 represents an ideal setting for the causal identification of health effects, we do not have enough information to quantify the reduction (or increment) of landing aircraft during the relevant period. This strategic limitation does not let us associate the beneficial (or adverse) impact on monthly medication spending with a specific number of flights that were removed from (or added to) the usual weekly schedule.

To conclude, the strengths and limitations of this work suggest that there is scope for future work on this field of research. An area for further exploration will be to find other strategies and settings to investigate the causal relationship between environmental hazards and nervous conditions. In fact, the consistency of evidence found across the three studies is a strong call for further specific assessments of this specific class of diseases.

Second, the interplay between planned and unplanned hospital visits represents a complex topic specific to all countries with a public health system and could be investigated in settings beyond the English National Health Service (NHS).

Third, we strongly encourage the study and adoption of the freely available data on GP prescriptions in order to produce further evidence on morbidity effects.

Lastly, albeit data limitations, it would be interesting to explore the more chronic and long-term causal effects of environmental exposure. In fact, with the data and time periods available, we were able to only look at short-term effects of environmental exposure.
exposure. There are other datasets that can help towards the investigation of long-
term health effects. For instance, combining Understanding Society data with the
British Household Panel Survey (BHPS) would provide up to 23 waves of annual data.
Crucially, health variables of these datasets include chronic conditions. However,
not all participants of the BHPS became part of Understanding Society, therefore
reducing the longitudinal sample size. This would endanger the economic significance
of a long-term study. Therefore, further work is necessary to find relevant data and
the correct strategy to estimate the causal health effects of long-term exposure to
air and noise pollution.
Bibliography


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