Assessing the influence of environment and socio-economics on spatial and temporal patterns of COPD hospitalisation in Christchurch: a GIS approach.

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# Table of Contents

Abstract 7  
Abbreviations 7  

## Introduction 8  

### Chapter One – Chronic Obstructive Pulmonary Disease 12  
1. Introduction to COPD 12  
   1.1 Prevalence 12  
   1.2 Morbidity and Mortality 13  
   1.3 Economic Burden 13  
2. Physical Burden 14  
   2.1 Dyspnea and Chronic Cough 14  
   2.2 Airflow Limitation and air trapping 14  
   2.3 Gas exchange abnormalities 15  
   2.4 Mucus Hypersecretion 15  
3. Exacerbations 16  
   3.1 Introduction to Exacerbations 16  
   3.2 Identifying Exacerbations 17  
   3.3 Seasonality of Exacerbations 17  
4. Factors contributing to COPD 19  
   4.1 Smoking 19  
   4.2 Exposure to Particles 20  
   4.3 Genes 21  
   4.4 Age and Gender 21  
   4.5 Socio-Economic status 22  
5. Diagnosis of COPD 23  
6. Treatment of COPD 23  
7. Summary 24  

### Chapter Two – Air Pollution 25  
1. Background to Air Pollution 25  
   1.1 Particulate Matter 25  
   1.2 Sulphur Dioxide 26  
   1.3 Nitrogen Oxide 27  
   1.4 Carbon Dioxide 27  
2. Meteorological Effects 27  
3. Health Impacts of Air Pollution 28  
   3.1 Introduction to Air Pollution 28  
   3.2 Indoor Air Pollution and Health 28  
   3.3 Air Pollution and COPD 29  
   3.4 Air Pollution and COPD Exacerbations 29
4. The Geographies of Socio-Economics and Pollution
   4.1 Introduction to Socio-Economics and Pollution 31
   4.2 Environmental Justice 32
5. Christchurch’s Air Pollution Problem 33
6. Summary 34

Chapter Three – GIS and Health 35
1. Spatial Patterns 35
2. Mapping Spatial Variation of Socio-Economic Variables 36
3. Estimating Air Pollution with LUR 37
4. Correlation with Environmental Variables 39
5. GIS for COPD Hospitalisations 41
6. Summary 41

Chapter Four – Methods 42
1. Descriptive Statistics 42
2. Spatial Analysis 42
   2.1 Spatial Autocorrelation 42
   2.2 Land Use Regression 43
   2.3 Mapping Deprivation 44
   2.4 Mapping other Socio-Economic variables 44
   2.5 Ordinary Least Squares Regression 45
   2.6 Hospitalisation Predictions with Ordinary Least Squares 46
   2.7 Geographic Weighted Regression 46
   2.8 Geographic Weighted Regression Output Descriptions 47
3. Temporal Analysis 48
   3.1 Air Pollution Data 48
   3.2 Hospitalisation Events 48
4. Summary 49

Chapter Five – Results 50
1. Introduction 50
   1.1 Introduction 50
   1.2 Descriptive Statistics 51
   1.3 Spatial Autocorrelation 53
   1.4 Land Use Regression models 54
   1.5 Hospitalisation and Predicted Risk of Pollution Exposure 55
   1.6 Deprivation and Hospitalisation 55
   1.7 Maori/Pacific Islanders and Hospitalisation 56
   1.8 Regular Smokers and Hospitalisation 58
   1.9 Elderly and Hospitalisation 59
Chapter Six – Discussion

1. Spatial Analysis Questions

1.1 What is the spatial distribution of COPD patients, and explanatory variables, in Christchurch?

1.2 What are the socio-economic and demographic determinants of air pollution exposure? How are predicted air pollution exposures spatially correlated with hospitalisation, deprivation, Maori/Pacific Islanders, smoking rates or old age?

1.3 To what degree are the LUR pollution models a reliable means of predicting rates of hospitalisation?

2. Temporal Analysis Questions

1.1 How do meteorological conditions (such as wind speed and temperature) affect air pollution levels at different temporal scales?

1.2 To what degree are air pollution levels correlated to rates of hospitalisation? Are these relationships stronger at hourly, daily, weekly or monthly scales?

1.3 Can we predict rates of hospitalisation from air pollution levels?

1. Future Research
List of Figures and Tables.

Figure 1. Air trapping .............................................................. 14
Figure 2. Mucus Hypersecretion .................................................... 15
Figure 3. Particulate Matter sizes .................................................. 25
Figure 4. Spatial Autocorrelation ................................................... 35
Figure 5. Geographic Weighted Regression ..................................... 39
Figure 6. Meshblocks and Area Units ............................................ 42
Figure 7. Christchurch Suburb Maps ............................................. 51

Table 1. Ethnicity of patients ....................................................... 52
Table 2. Smoking prevalence among hospitalised patients .................. 52
Table 3. Averages ages among hospitalised patients .......................... 52
Table 4. Ethnicity and Hospitalisations .......................................... 53
Table 5. Moran’s I results ........................................................... 54

Figure 8. LUR maps ................................................................. 54
Figure 9. Pollution and hospitalisation maps .................................... 55
Figure 10. Deprivation and hospitalisation map .................................. 56
Figure 11. Maori/Pacific Islander and hospitalisation map .................... 57
Figure 12. Regular smokers and hospitalisation map ........................... 58
Figure 13. Elderly percentages and hospitalisation map ........................ 59

Table 6. OLS Model with Socio-Economic Variables .......................... 60
Table 7. OLS Model with Socio-Economic Variables Coefficients ............ 60
Table 8. OLS Model with Socio-Economic Variables and Summer LUR model .................. 61
Table 9. OLS Model with Socio-Economic Variables and Summer LUR model Coefficients ...... 61
Table 10. OLS Model with Socio-Economic Variables and Winter LUR model ............. 62
Table 11. OLS Model with Socio-Economic Variables and Winter LUR model Coefficients ...... 62
Table 12. OLS Predictions using Socio-Economic Variables .................... 62
Table 13. OLS Predictions using Socio-Economic Variables and Summer LUR model ............ 63
Table 14. OLS Predictions using Socio-Economic Variables and Winter LUR model ............. 63
Table 15. OLS Testing the Predictive Strength of Individual Variables ............ 64
Table 16. GWR Dependent Variable: Winter LUR pollution map at meshblock level ............... 65
Table 17. GWR Dependent Variable: Summer LUR pollution map at meshblock level ............. 65
Table 18. GWR Dependent Variable: Deprivation Index at meshblock level ..................... 66
Table 19. GWR Dependent Variable: Hospitalisation at meshblock level ....................... 66
Table 20. Dependent Variable: Hospitalisation at area unit level .................... 67
Table 21. Multivariate GWR for Hospitalisation 250m bandwidth .................. 67
Table 22. Multivariate GWR for Hospitalisation 500m bandwidth ........................................ 67
Table 23. Multivariate GWR for Hospitalisation 1000m bandwidth ........................................ 67
Table 24. Temporal Bivariate correlations .................................................................................. 68

Figure 14. Weekly Hospitalisations and PM10 ........................................................................... 69
Figure 15. Weekly Hospitalisations and PM2.5 ....................................................................... 70
Figure 16. Weekly Hospitalisations and wind speed averages .................................................. 70
Figure 17. Weekly Hospitalisations and temperature averages ................................................ 70
Figure 18. Weekly temperature and wind speed averages ......................................................... 70
Figure 19. Annual Pollution and Hospitalisation Patterns ....................................................... 71
Abstract

This thesis examines links between environmental conditions and the socio-economic and demographic determinants of Chronic Obstructive Pulmonary Disease (COPD) in Christchurch, New Zealand. COPD is a progressive condition characterised by reduction in lung function and limited mobility. Tobacco smoking and exposure to air pollution are considered the main causal factors. COPD is the fifth leading cause of mortality globally, and is projected to become the third by 2030. Geographic Information Systems serves a vital role in furthering understanding of the spatial patterns of the condition, and the prevalence of associated variables across space. The aim of this thesis is to engage GIS tools to assess the spatial and temporal patterns of COPD hospitalisation events in relation to air pollution distribution, based on land-use regression modelling, and associated socio-economic variables. To test for the effects of these socio-economic variables, census data was included relating to deprivation levels, and percentages of Maori/Pacific Islanders, smokers and elderly of the population at meshblock and area unit levels. Temporal analysis sought to find correlation between monitored particulate matter, hospitalisation rates and the temperature and wind speed patterns that may affect this. Geographic Weighted Regression (GWR) models found that higher hospitalisation rates were spatially correlated with meshblocks containing high predicted air pollution and deprivation. Maori/Pacific Islander and smoking percentages were moderately associated with hospitalisation event distribution. Temporally, it was found that hospitalisation events occur most predominately in periods of high pollution, lower temperatures and still wind conditions. This thesis is the first research to include spatial and temporal techniques to explore COPD hospitalisation patterns in Australasia.

Abbreviations

COPD - Chronic Obstructive Pulmonary Disease
FEV - Forced Expiratory Volume
FVC - Forced vital capacity
GIS – Geographic Information Systems
GWR – Geographically Weighted Regression
LUR – Land-use Regression
OLS – Ordinary Least Squares
Introduction

This thesis examines links between environmental conditions and the socio-economic and demographic determinants of chronic obstructive pulmonary disease (COPD) in Christchurch, New Zealand. COPD is a progressive condition characterised by reduced air circulation and lung capacity. COPD is now the world’s fifth largest cause of death (Schirnhofer et al, 2007) and is projected to grow substantially over the coming decades (Mannino & Buist, 2007). Much of the research into COPD has focused on the characteristics of sufferers of COPD (Fukuchi et al, 2004), the suspected causes (Eisner, 2010; Dominici et al, 2006 & Zanobetti et al, 2014), exacerbations (Gudmundsson et al, 2012) and various treatments (Bonini and Usmani, 2015). Broad conclusions drawn from research indicate that COPD is most likely caused by smoking and exposure to air pollutants (WHO, 2000) and tends to be more common among the elderly (Halbert et al, 2006). Ethnicity and deprivation are also significant when considering smoking rates and exposure to air pollution (Been, 1993, 1995, 1997; Hnizdo et al, 2004 and Glover et al, 2013), which will be explored extensively in subsequent chapters. Scientists have postulated that air pollution may increase irritation of the lung, thus triggering an exacerbation, episodes of heightened symptom severity (Dominici et al, 2006; Zanobetti et al, 2014). Therefore, the influence of environmental factors beyond smoking is important. Spatial and temporal variance in these environmental factors can be used to predict COPD prevalence (Muralidharan et al, 2014). Of these two dimensions, temporal variation appears regularly in COPD research (Baillargeon et al, 2013; Rabe et al, 2013 and Hurst et al, 2009). Studies have found that COPD exacerbations occur more frequently in winter, in accordance with many other respiratory and cardiovascular conditions (Bhowmik et al, 2000; Garcia-Gutierrez et al, 2013; and Public Health England, 2013). Exploration into spatial variation of COPD in relation to air pollution exposure is relatively recent and limited (Schikowski et al, 2005), and this research represents the first in Australasia that focuses on the spatial correlation of COPD prevalence and air pollution, with a previous project only mentioning COPD briefly in the context of wider health issues (Ahmad et al, 2010). Studies which simultaneously explore both spatial and temporal trends in COPD research, in relation to air pollution, as appeared in Chan et al (2015) are rarer still. Chan et al (2015) used mortality as a measure of prevalence, and not hospitalisation. This thesis extends the growing global body of emerging research on spatial and temporal trends. This will be discussed in more depth in the literature review (Chapters 1, 2 & 3).
Statement of Research problem

This thesis focusses on the COPD hospitalisations of Christchurch, a city of 341,469 (Statistics New Zealand, 2013) on New Zealand’s South Island. Christchurch experiences high air pollution levels relative to its size and low population density (Kingham et al, 2011), exceeding clean air targets for particulate matter levels on 19 days of 2014 (Let’s Clear the Air, 2015). This project evaluates the evidence for whether spatial and temporal variations of air pollution appear to influence COPD hospitalisation. This research is the first to engage spatial and temporal analysis to understand patterns of COPD hospitalization in New Zealand, and has extended a limited global body of research in this area.

The overall aim of this thesis can be best summarised as: determining whether environmental and socio-economic factors influence spatial and temporal variation in COPD hospitalisation in Christchurch.

Research Questions

The following questions have been formulated to allow for in-depth analysis of spatial and temporal patterns of COPD hospitalisation in reference to the environmental and demographic variables. The format of spatial analysis being followed by temporal analysis, first shown with these questions will appear in Methods, Results and Discussion. The order of these questions will be adhered to throughout the thesis, by first focussing on spatial relationships before shifting to a temporal focus.

Spatial

1. What is the spatial distribution of COPD patients, and explanatory variables, in Christchurch?

2. What are the socio-economic and demographic determinants of air pollution exposure? How are predicted air pollution exposures spatially correlated with hospitalization, deprivation, Maori/Pacific Islanders, smoking rates or old age?

3. To what degree are the LUR models a reliable means of predicting rates of hospitalisation?

Temporal

1. How do meteorological conditions (such as wind speed and temperature) affect air pollution levels at different temporal scales?

2. To what degree are air pollution levels correlated to rates of hospitalisation? Are these relationships stronger at hourly, daily, weekly or monthly scales?

3. Can we predict rates of hospitalisation from fluctuations in air pollution levels?
Thesis Structure

The initial chapters of this thesis are dedicated to establishing a background on which this study will be founded. Chapter One focuses on Chronic Obstructive Pulmonary Disease, and provides background details on the physical burdens experienced by sufferers, and the subsequent economic burdens on wider communities. The characteristics of patients are also explored, with particular reference to the factors that cause COPD development, namely old age, tobacco smoking and those exposed to air pollution whether that be in residential or occupational environments. These demographic vulnerabilities aid the direction of this thesis, by identifying what may be the most significant socio-economic influence on spatial and temporal patterns of COPD hospitalisation.

Chapter Two focuses on air pollution, how it affects health broadly and specifically COPD, and the demographics often more likely exposed to higher rates of air pollution. It introduces the concept of environmental justice, a theory that proposing that more deprived communities tend to contain higher levels of pollution, a factor that will be tested for its potential influence on the spatial distribution of hospitalised COPD patients in Christchurch.

Chapter Three is an overview of the application of Geographic Information Systems (GIS) in researching wider health and COPD-related topics, justifying why these tools are essential for COPD research. The tools, based on previous works, are applied to find spatial correlation between environmental and socio-economic variables, revealing how predominately hospitalisation events occur in neighbourhoods that have high air pollution, deprivation or significant populations of Maori/Pacific Islanders, regularly smoking, and elderly residents. The previous research applications of GIS, mentioned in Chapter Three, have aided this thesis in decided what techniques were most suitable. GIS presents the most applicable method of exploring if environmental variables are associated with COPD hospitalisations.

Contained within Chapter Four, the methods section, are the geospatial and statistical techniques this thesis will be utilising. In determining the spatial distribution of pollution exposure risk, a Land-use Regression (LUR) model is built to compensate for a limited number of air quality monitoring sites. Each meshblock will be assigned values for predicted pollution, hospitalisation rates, deprivation, and the percentages of residents who are Maori/Pacific Islanders, elderly or regular smokers. Ordinary Least Squares and Geographic Weighted Regression with ArcMap 10.2 was used to determine strengths of correlations between COPD hospitalisation and the pollution and socio-economic variables. Temporal data holds hourly measurements in particulate matter, provided by two ECAn monitoring sites. Pollution counts will be compared to the wind speed and temperature data to study how weather
conditions affect the levels of pollution. These variables will be compared to COPD hospitalisation rates from September 2014 to August 2015 to determine if correlation exists.

Chapter Five, the results, will present the maps, tables and graphs, generated through ArcMap 10.2 and Excel. Following the structure of earlier chapters, spatial results will be presented first, ordered in accordance with the relevant questions. Temporal analysis is then explored through correlation statistics, presented in table and graph format to reveal relationships with hospitalisation rates, fluctuating pollution levels and temperature and wind speed.

Chapter Six offers in-depth discussion to answer the questions, providing assessment on how these variables are aligned with COPD hospitalisation. The results will be assessed in light on environmental justice, the theory that more polluted areas tend to contain more deprived residents. If this is observed in Christchurch, it will confirm socio-economic vulnerabilities identified in earlier research. The reliability of land-use regression is assessed as means of predicting seasonal pollution estimations across the city. The discussion of temporal aspects focuses on how air pollution fluctuations may affect COPD hospitalisation, and how the meteorological effects may influence the exposure experienced by residents. The Future Research section reflects on the implications and limitations of this thesis to aid the design of new research. Chapter Seven concludes the thesis by summarising the findings and implications. Firstly, let’s now move on to Chapter One.
Chapter One – Chronic Obstructive Pulmonary Disease

1. Introduction to COPD

Chronic Obstructive Pulmonary Disease (COPD) is a lung condition characterised by airflow obstruction and breathing limitation (GOLD, 2014). The airflow obstruction typically worsens with time and is not fully reversible, although deterioration occurs at varying rates. Although the disease is incurable, regular visits to health services and appropriate treatment can slow degeneration in lung function considerably. Early detection will therefore extend the time in which a patient can experience a mobile lifestyle. COPD is a widespread disease and is the fifth leading cause of death worldwide, projected to become the third by 2030 (Schirhofer et al, 2007). COPD is a considerable and climbing economic and social liability.

COPD prevalence, morbidity, and mortality differ worldwide, and effects different societal groups (Logez et al, 2006; Mathers et al, 2006). The disease’s prevalence is blamed predominately on tobacco smoking, although in many urban environments, exposure to outdoor pollution (traffic and industry) and indoor pollution (biomass burning) are also considered main COPD risk factors (Salvi and Barnes, 2009). Air pollution as a factor for COPD will be the focus of this thesis, and more detail will be provided in Chapter Two (page 25) in regards to the specific effects of air pollution on health. The prevalence of COPD is expected to continue escalating in future decades as higher populations are subjected to these risks, intensified by longer life expectancies resulting in longer exposure times (Salvi and Barnes, 2009).

1.1 Prevalence

Current COPD prevalence data shows considerable difference due to variation in survey approaches and diagnostic criteria. The most conservative estimations of prevalence are those based on medical diagnosis. Additionally, most national data shows that of the adult population in the United States, only 6% has been officially diagnosed with COPD, when true prevalence is expected to be much higher (Halbert et al, 2006). This indicates the extent of the disease’s widespread under-recognition and under-treatment (van den Boom et al, 1998). Despite these difficulties, data regarding COPD prevalence is becoming more obtainable. A meta-analytical review of studies undertaken in 28 nations between 1990 and 2004 (Halbert et al, 2006) offers evidence that COPD occurrence is demonstrably higher in smokers and former smokers than non-smokers, in mature demographics, and higher in males than females.
1.2 Morbidity and Mortality

One method of comprehending prevalence is monitoring morbidity, which is a measure of reliance on physicians, emergency departments and hospitalisations. Studies have found that morbidity augments with age (Fukuchi et al, 2004; Schirnhofer et al, 2007) but this may be affected by the existence of comorbid diseases, such as cardiovascular disease, musculoskeletal impairment, and diabetes, which are associated with COPD and may impact the health of patients, as well as inhibit COPD management (GOLD, 2014). Morbidity is dependent on the severity of symptoms present, and is therefore most intense during episodes referred to as acute exacerbations (page 16).

Mortality is the occurrence of death caused by COPD (Pena et al, 2000; Talamo et al, 2007). Like morbidity, mortality is difficult to define due to the existence of comorbid diseases. Mortality offers inadequate perspectives on the daily burden of the condition, as it ignores how the disease affects patients’ lives. It is also affected by under-recognising the extent of the disease, as not all COPD patients die because of COPD. However, even from using mortality as a measure of COPD suffering, it remains one of the leading causes of deaths, and is expected to be the third leading cause of death globally by 2030 (Mathers et al, 2006).

1.3 Economic burden

The economic burden of COPD is considerable. The direct cost of COPD in the European Union is approximately 6% of the overall health expenditure, and it accounts for 56% (38.6 billion Euros) of the health budget’s allocation to respiratory conditions (Mannino et al, 2003). Across the Atlantic Ocean, the estimated direct cost of COPD in the United States is $29.5 billion, with an additional $20.4 billion attributed to related indirect costs (National Heart, Lung and Blood Institute, 2009). COPD acute exacerbations, when symptoms peak to levels often requiring hospitalisation, account for the largest amount of the total COPD burden on the health care system (GOLD, 2014).

There exists a clear relationship between economic burden and the severity of COPD. As COPD is a progressive disease, costs typically increase with time. The distribution of costs changes as the disease progresses. Costly reliance on hospitalisation and emergency oxygen supply tends to occur at later stages of COPD development (GOLD, 2014). In developing countries, the impact of COPD on workplace and home productivity may be more important than medical costs. As the health sector may not offer continuing support care services for chronically affected individuals in developing countries, COPD may lead to heightened rates of unemployment for both the individual with COPD and a relative now house-ridden to provide support. Human capital is often touted as the most important national asset for developing nations and the wider economic burden of COPD may gravely threaten their economies (GOLD, 2014.)
2. Physical burdens

COPD is characterised by reduction in airflow and lung capacity, affecting mobility. This is often the result of a complex and multi-faceted set of symptoms affecting the lungs and air passages, including dyspnea, chronic cough, air flow limitation and air trapping, gas exchange abnormalities, mucus hypersecretion and pulmonary hypertension. The following sections are dedicating to describing the physiology of these symptoms.

2.1 Dyspnea and Chronic Cough

Dyspnea, perhaps the most common symptom of COPD, is also a major cause of disability and anxiety accompanying the disease. Typically, patients describe dyspnea as an increased difficulty in breathing, and the sensation of not receiving sufficient air supply, leading to gasping and ‘air hunger.’ Chronic cough, often the first symptom of COPD to develop (Georgopoulas et al, 1991), is frequently discounted by the patient as being an expected consequence of smoking and/or environmental exposures. Initially, the cough may be sporadic, but later is present every day, often throughout the day (Burrows et al, 1965). In some cases, significant airflow limitation may develop without the presence of a cough. COPD sufferers commonly produce small quantities of tenacious sputum after coughing bouts. Minor symptoms may also be experienced, not necessarily specific to COPD, and usually more distinctive in asthma, including wheezing and chest tightness. Such symptoms can vary within a single day or over numerous days (Simon et al, 1990). Audible wheeze may occur at throat-level, and although it may not negatively influence lung function, it may have psychological effects. Conversely, inspiratory and expiratory wheezing can exist at lower levels of the respiratory system. Exertion is regularly followed by chest tightness, caused by muscular contraction (Elliott et al, 1991). An absence of wheezing or chest tightness does not necessarily exclude the existence of COPD, while their existence may be misdiagnosed as asthma in many COPD cases.

2.2 Airflow Trapping and Hypertension.

![Figure 1. A diagram demonstrating how inflammation reduces air flow the lungs and lung capacity (Thomas et al, 2013).](image)

Airway obstruction caused by inflammation and sputum production is associated with reduced lung capacities (Figure 1). This obstruction progressively traps air in peripheral alveoli during exhalation. Alveolar attachments in the lungs are damaged when
COPD intensifies. Hyperinflation limits breathing capacity, particularly during exercise (dynamic hyperinflation), increasing dyspnea and affecting mobility. Further, the muscle recoil characteristics of respiratory muscles are lost, leading to increased inflammation. Research suggests that hyperinflation arrives early in COPD development, and is the leading catalyst for dyspnea. (O’Donnell et al, 2007; 2009) Pulmonary hypertension, a common symptom of COPD, refers to the narrowing, blocking or destroying of small lung arteries. Subsequently, the flow of blood through the lungs is made more difficult, raising blood pressure in those arteries. With this building pressure, the heart’s right lower chamber (right ventricle) is required to work harder, leading to its weakening and eventual failure. Pulmonary hypertension tends to develop at later stages of COPD development. There is an inflammatory response in vessels similar to that seen in the airways and evidence of endothelial cell dysfunction. The reduction of the pulmonary capillary bed, essentially the surface area of the inner lung, during emphysema is also thought to contribute to heightened pressure in lung’s blood circulation (Peinado et al, 2008).

2.3 Gas exchange abnormalities

Generally, gas transfer for oxygen and carbon dioxide worsens in COPD over time. Abnormalities in gas exchange result in respiratory failure hypoxemia and hypercapnia, the first being when an insufficient supply of oxygen is entering the blood, but carbon dioxide levels remain normal. Hypoxemia is most frequent type of failure in respiratory illness, associated with almost all serious pulmonary diseases involving fluid filling or alveoli collapse. Secondly, hypercapnia occurs when there is too much carbon dioxide lingering in the bloodstream, accompanied by near normal or insufficient amounts of oxygen. Reductions in alveolar ventilation and a reduced pulmonary surface area further deteriorate breathing (Rodriguez-Roisin et al, 2009).

2.4 Mucus hypersecretion

![Normal bronchial tube](image)

![Inflamed bronchial tube](image)

*Figure 2. Mucus hypersecretion blocks airways, reducing air flow, as shown when comparing a COPD sufferer’s airways (right) to that of a healthy airway (left) (Pacific Heart and Lung Institute, 2015).*

Mucus hypersecretion, the production of sputum in the lungs and bronchial airways (Figure 2), leads to the blocking of airways and chronic productive cough. Mucus hypersecretion often occurs due to a growing number of goblet cells and inflamed submucosal glands after the airways have been irritated by viruses, bacteria and other particulates, such as tobacco smoke (Burgel
and Nadel, 2004). Because of these conditional triggers, not all COPD patients experience mucus hypersecretion in chronic capacities.

Sputum production is often difficult to evaluate because patients may swallow sputum rather than expelling it. It has been found that these habits are controlled considerably by variations in culture and gender. Bronchiectasis, the abnormal widening of the bronchi or their branches, triggering risk of infection, may be an underlying condition of sufferers who produce large amounts of sputum. The presence of purulent sputum (containing pus, composed of white blood cells, cellular debris, dead tissue, serous fluid, and viscous liquid) indicates an increase in inflammation (Hill et al, 1999). The development of this may indicate the triggering of an exacerbation (Stockley et al, 2000).

3. COPD Exacerbations

3.1 Introduction to COPD Exacerbations

An exacerbation (also referred to as an acute exacerbation) is a sudden worsening of COPD symptoms that typically lasts for several days. Prompted by bacterial or viral infection, or ambient pollution, sufferers may experience intensified respiratory symptoms resulting in increased inflammation (Rodriguez-Roisin, et al 2000). Patients with moderate-to-severe cases of COPD suffer two to three exacerbations per year (Donaldson et al, 2006). During exacerbations there are increased cases of hyperinflation, gas trapping and dyspnea. Comorbidities such as pneumonia, thromboembolism (blood clotting) and acute heart failure may be induced by COPD exacerbations (Parker et al, 2005).

An exact definition for exacerbations has not yet been settled in medical literature. A commonly cited definition has been provided by Anthonisen et al (2002), whereby an exacerbation is considered to exist when 3 major clinical symptoms exist: the worsening of dyspnea, mounting sputum production and the existence of sputum purulence, containing white blood cells, cellular debris, dead tissue, serous fluid, and viscous liquid (mucus). According to this definition if all these symptoms of COPD are present, the exacerbation is considered severe; two symptoms indicate moderate exacerbation, while one symptom represents mild exacerbation. There are limits to this definition however, as no following research studies have determined the association between number of symptoms and severity (Parker et al, 2005; Kanner et al, 2001).

Exacerbations often require hospitalisation, affecting symptoms and the functioning of respiratory symptoms that often requires weeks of recovery time. It was concluded from a study of 101 patients with moderate to severe COPD that full recovery following exacerbation was not attained by 25% of patients (Seemungal et al, 2000). Exacerbations are known to increase the speed of decline in lung function and drastically heighten the risk of mortality in more serious cases. Exacerbations of COPD can be
triggered by numerous factors. The most regular causes are respiratory infections due to bacteria or virus, or particulate matter. Bronchoscopy studies have shown that over 50% of sufferers have increased bacteria in their lower airways during exacerbations (Monso et al, 1999; Pela et al, 1998).

3.2 Identifying Exacerbations

Exacerbation diagnoses rely solely on the patient’s self-identification of symptom deterioration and their subsequent decision to seek medical support. Patients must recognise when dyspnea, chronic cough, and/or sputum production reach intensities above regular severity. The immediate role of health services is to reduce the impact of the exacerbation, and then seek ways to reduce risk of future exacerbation (Decramer et al, 2009; Celli et al, 2008).

The rate of mortality is higher in COPD patients who suffer exacerbations. Research involving of 256 patients in Scandinavian nations discovered that 79% died in the 9 years following their exacerbations (Gudmundsson et al, 2012). Increased risk of mortality was connected with older age, poor lung function, low BMI and diabetes. While COPD is non-curable, risk of exacerbations is reducible through the cessation of smoking, and the implementation of influenza and pneumococcal vaccines. The education of patients with regards to current therapy techniques and treatment with long-acting inhaled bronchodilators also has a significant impact on reducing the likelihood of exacerbation (Wyka et al, 2012). More details on mitigation treatment techniques for COPD will be provided later in this chapter (Part 6, page 23).

3.3 Seasonality of Exacerbations

Winter months have long been established as placing extra pressures on health services (Public Health England, 2013). In temperate countries in the mid-latitudes, such as New Zealand, winter months also bring higher counts of respiratory viruses (Bhowmik et al, 2000; Garcia-Gutierrez et al, 2013). Hospital admissions, mortality and visits to doctors among COPD patients climb in winter (Calderón-Larrañaga et al, 2011; Organisation for Economic Co-operation and Development, 2011), accompanied by worsening health-related quality of life, anxiety and depression. Increases in admissions for COPD have been observed across Europe: Spain (Ballester et al, 1999), Finland (Vilkman et al, 1996), Portugal (Monteiro et al, 2013), and the UK (Brims et al, 2011). The phenomenon has also been observed in the US (Mapel et al, 2005), South Korea (Lim et al, 2012) and Hong Kong (Ko et al, 2007). This increase in mortality and morbidity during winter places a heavy burden on health and care services (Rabe et al, 2007).

Exacerbations are more frequent in the winter, although it is unknown whether severity increases. A comprehensive study (Donaldson et al, 2006) attempting to explore seasonal variation in exacerbations involved 307 COPD patients in London. The patients contributed data over the course of at least one year, from November 1, 1995 to November 1, 2009. Sufferers were required to keep records of any changes in symptom intensity each morning, using diary cards. Dyspnea and sputum production
were considered major symptoms, while minor symptoms were wheeze, sore throat, cough nasal discharge, sore throat, and cough. Time outside of their houses was also recorded in the diaries. The prediction of an approaching exacerbation was factored by following patterns of symptom intensity. If more than two consecutive days featured increases in two major symptoms or one major with one minor symptom, it was considered an exacerbation (Donaldson et al, 2006; Johnstone et al, 2010). Exacerbations typically required hospitalisation and were more frequent in winter months. During winter months when the average temperature was 6.6°C, the patients suffered 55.6% more exacerbations compared with summer months, when the average temperature was 16.7°C. Recovery time for symptoms was also influenced by seasonality, but the relationship was less significant. During warmer months, median recovery was 9 days compared with 10 days in colder seasons. A similar pattern emerged when considering the recovery from only major symptoms: 7 days in summer months compared with 9 days in winter months. The number of exacerbation days was considerably higher in colder seasons at 26 days compared with 11 days in the warmer seasons. In the cold seasons, there was a significant climb in the percentage of exacerbations requiring hospitalisation; 88 of 1,052 exacerbations (8.4%) required hospitalisation in winter while 31 of 676 exacerbations (4.6%) exacerbations required hospitalisation in the summer (Donaldson et al, 2006).

An even larger study covered all hospitalisations in Scotland for COPD from 2001–2010, (McAllister et al, 2013) found a synergistic relationship between winter and deprivation. Hospitalisation increased in winter compared to summer, and deprived areas experienced heightened seasonal differences. Seasonal variation also was found to control the length of hospital stays. In studying 153,401 hospitalisation between 1987 and 1998 in Finland (Kinnunen et al, 2002), the average length of stay was 12.3 days in colder months and 11.0 days in warmer ones. It was speculated that in colder climates during winter, accompanied by fewer sunlight hours, regular visits to doctors or rehabilitation centres declined due to inaccessibility.

A fall in the significance of seasonality on COPD exacerbations has not been expressed in recently published papers, although the overall influence of winter on respiratory diseases is falling. From 1977-1994, study data revealed decline in increase in deaths per winter (Donaldson and Keatinge, 1997). The reason for this is considered to be population adaption rather than climatic change bringing higher temperatures (Public Health England, 2014). Additionally, decrease in all facets of mortality during winter could be due to improving fuel efficiency and housing insulation, societal dietary improvement, affordable home heating, and government schemes, although the exact reasons remain undisclosed (Public Health England, 2014).
4. Factors for COPD

4.1 Smoking

A study (Eisner, 2010) revealed that the role of smoking in causing COPD ranged from 9% to 97.9%. Studies in Sweden and Denmark attributed 76.2% and 74.6% cases of COPD to smoking, respectively (Liu et al, 2015). WHO (2000) estimated that in the high-income countries, 73.0% of COPD mortality was related to smoking, whereas only 40.0% related to smoking in the low and middle income countries. Due to its dominant role in the development of the respiratory disease, an understanding of smoking distribution and prevalence is vital in COPD research. Many studies have discovered higher prevalence of smoking associated with lower socioeconomic groups and lower educational achievement (Crone et al, 2003; Haustein, 2006). The higher number of smokers in deprived neighbourhoods may be due to social disadvantages or the effect of the area’s characteristics (Shohaimi et al, 2003).

In New Zealand, as of 2013, over 700,000 New Zealanders smoke on a regular basis, constituting approximately 21.1% of men and 18.9% of women (Action on Smoking and Health, 2013). This has fallen from 907,500 in 1990, which constituted 28% and 27% for men and women respectively. In 1990, half of the adult Maori community regularly smoked, although that had been reduced to 40.4 percent by 2006/2007 (Action on Smoking and Health, 2013). New Zealand Europeans have experienced the largest decrease in smoking prevalence, with rates in Maori and Pacific Island communities undergoing less substantial decrease. Among males, smoking prevalence is approximately 22% for Europeans, 40.4% for Maori, 34.7% for Pacific Islanders and 20.1% for Asians. Among women, in the same order of ethnicities, prevalence is approximately 20.2%, 49.7%, 28.5% and 5.2% (Action on Smoking and Health, 2013).

As has been observed globally (Crone et al, 2003; Haustein, 2006), tobacco smoking is more frequent amid low socioeconomic communities, and furthers socioeconomic and ethnic inequalities in New Zealand (Glover et al, 2010). This pattern was first forged in the 1980s by tobacco control policies, which successfully began reversing the high prevalence of smoking in high socioeconomic communities, but had little effect on others. Non-smokers who inhabit deprived areas are also more likely to be exposed to higher levels of second hand smoke, and are four times more likely to die from smoking than non-smokers in less deprived areas (Glover et al, 2013). Lower socioeconomic groups were also less likely to quit smoking, and failure was more probable if they attempted to do so. Harmful health outcomes related to tobacco-related disease is high among New Zealanders of lower socioeconomic status. According to the Ministry of Health, if social inequalities are not addressed, these negative health indicators will continue to create socio-economic disparities (Glover et al, 2013).

According to Blakely et al (2006) these disparities are expected to grow, especially in regards to ethnicity, with particular effect on mortality. They advance the notion that addressing socio-economic gaps and enforcing further tobacco control is the most
credible means of reducing ethnic inequalities in health. In addition to having the highest smoking prevalence, Maori and Pacific communities in New Zealand are also overrepresented among lower socio-economic groups (Glover et al, 2010).

While tobacco smoking is the most recognised risk for the development of COPD, non-smokers may also suffer from the disease, with chronic airflow limitation affecting their lifestyles (Behrendt, 2005; Celli et al, 2005; Eisner et al, 2010; Lamprecht et al, 2011). Beyond smoking, cross-sectional epidemiological studies identify associational rather than causal relationships between the disease and potential risks. Several longitudinal studies of COPD have monitored affected populations for up to 20 years (Anthonisen et al, 2002), however none has followed the lifecycle of COPD from pre-diagnosis to death, or has included the pre-and perinatal periods which may be important in shaping a patient’s future COPD risk. Ergo, current understanding of risk factors for COPD remains speculative, but research is improving understanding.

4.2 Exposure to particles

Occupational exposures, including organic and inorganic dusts, chemicals and fumes are also influential factors in developing COPD. A study analysing 10,000 working adults (aged 30-75) in the US projected that 19.2% of all COPD diagnoses were directly attributable to occupational conditions, and 31.1% among COPD diagnoses of non-smokers (Hnizdo et al, 2004). Risks from workplace exposures in less regulated regions of the globe, such as Africa or South East Asia, are expected to be significantly higher than reported in more regulated environments, such as Europe and North America, where studies are numerous. Evidence continues to swell that indoor pollution attributable to wood/coal burning for cooking or heating in inadequately ventilated houses is a critical risk factor for the disease. One study (Halbert et al, 2006) estimated prevalence of COPD to be 8.4% in rural regions and 10.2% in urban areas, suggesting that ambient air pollution may not be as influential to the disease’s development as smoking and internal home heating.

However, the higher concentrations of air pollution found in urban environments are detrimental to all humans, particularly those with existing heart or lung disease. The role of indoor air pollution in causing COPD specifically is unclear, and again rests on associational studies as opposed to directly causational relationships (Boman et al, 2003). Ambient pollution derived from the combustion of fossil fuels, principally traffic emissions in urban areas, is associated with declining lung health generally (Ezzati, 2006; Sezer et al, 2006). Whether the respiratory challenges linked to air pollution are more influenced by short time-frame/high-peak exposures or long time-frame/low-level exposure is yet to be fully understood (Abbey et al, 1998). The role of air pollution on health, and Christchurch’s specific air pollution characteristics, will be explored in more detail in Chapter Two.
4.3 Genes

COPD results from an interaction between environmental conditions and genetic vulnerabilities (Stoller and Aboussouan, 2005). Although tobacco smoking is the established main risk for the development of COPD, there is significant evidence that genetic factors impact the development of COPD in reaction to smoking (McCloskey et al, 2001). Among unrelated people with similar smoking histories, not all will suffer from COPD, suggesting variation in genetic susceptibility to the disease. The role of genetic factors in non-smokers with COPD has been less widely studied, but several assessments of evidence suggest that genetics is of equal significance to smokers and non-smokers alike. The diagnosis of COPD is determined by decreased expiratory airflow; this is possibly caused by different pathophysiological processes controlled by genes and causing variation in patients’ phenotypes (Castaldi et al, 2010; Cho et al, 2010; Pillai et al, 2009). Bronchial smooth muscle cell growth, inflammatory narrowing of peripheral airways and loss of elastic recoil may affect individuals in varying extents, but more genetic research is required (Repapi et al, 2010; Wilk et al, 2009).

4.4 Age and gender

Age is regularly highlighted as a risk factor for COPD, but scientists remain uncertain if aging alone leads to COPD or if age brings cumulative exposure to risk throughout life. One study used a global average of 7.6% to represent those affected by COPD. The paper claimed that 3.1% of those under 40 were affected by COPD, rising to 10% for those over 40, and 14.2% were affected over 65 (Halbert et al, 2006). Old age and comorbidity are partly responsible for misdiagnosis and under-treatment of COPD. Older COPD patients generally have poor health status due to comorbidities and reduced mobility (Tayde and Kumar, 2013). In New Zealand, the mean age of COPD patients who were hospitalised between July 2008 and June 2013 was 63.4 for Maori/Pacific Islanders and 72.2 for Europeans (Milne and Beasley, 2015). In the United Kingdom, the average age of COPD patients had plateaued at 70 over the previous decade (James, 2011), although the average age of diagnosis has decreased to the mid-fifties, and the average age of mortality has increased to the mid-seventies.

A majority of research from developing countries indicates that the prevalence and mortality of COPD is higher among males than females. This affects global averages, where 9.8% of males are said to suffer from COPD, while 5.6% of females are affected. Conversely, data from developed countries (National Heart, Lung and Blood Institute, 2009; Mannino et al, 2002) reveal near-equality between genders in regards to the prevalence and mortality of COPD, likely due to shifting distributions of cigarette smoking habits. Cigarette smoking was rare among women in the early 20th Century, but started increasing due to targeted marketing in the 1920s. Several research studies have found that females are more physically susceptible to the damaging pulmonary effects of tobacco smoke than men (Foreman et al, 2011; Lopez et al, 2010; Sorheim et al, 2010). This is due to females generally having less respiratory muscle, and as a result pollutants and irritants affect a larger proportion of pulmonary
surface. Furthermore, evidence suggests that women would benefit more from the cessation of smoking, but often have more difficulty in quitting and staying smoke-free, particularly those who are older (Beckett, 2004). However, women with COPD are 1.5 times more likely to be non-smokers, but are susceptible to second-hand smoke and indoor and outdoor pollution (Centers for Disease Control and Prevention, 2011).

Differences in vulnerability to COPD between genders may also be influenced by occupational exposure to particulate matter. Traditionally, males hold jobs in industry or mining that feature major exposure to dusts and fumes that potentially cause respiratory disease (Mehta et al, 2012). Gender disparities in regards to occupation may be the factor behind variation in disease susceptibility in both developed, but especially, developing countries. The history of COPD affecting more men has led to women being more likely to be misdiagnosed as having asthma, remain undiagnosed, or feel they are receiving insufficient time and attention from medical staff (Ancochea et al, 2013). The shifting trend away from being a male-dominated disease is especially evident in the United States, where more women now die from COPD than men (Price et al, 2010).

**4.5 Socio-economic Status**

Lower socio-economic status has been consistently and significantly linked with poorer COPD outcomes (Prescott and Vestbo, 1999). The conditions of homes associated with lower socio-economics, include dampness, a high population of dust mites and the use of gas or fire for cooking. These are typically related to respiratory symptoms and reduced lung function (Prescott and Vestbo, 1999). Exposure to outdoor air pollution is a supplementary risk factor to lung function more likely to be suffered by subjects of lower socioeconomic areas (Been 1993, 1995 and 1997), due to geographies of property values, a concept referred to as environmental justice, which will be explored in Chapter Two (page 32).

Further, due to the close association between low socio-economics and lower education, exposure to occupational pollution also significantly influences respiratory disease among the poor (Kanervisto et al, 2011). This influence appears to exist at all ages, as they are present before occupational exposure has had accumulative negative effect on lung function. The strength of socio-economic status’ influence on lung function, and other characteristics of COPD, is dominated by that of smoking. The relationship is important as it signifies controlling risks in the development of the disease. It has become essential for health sector research to consider socio-economics as an independent risk factor whose constituents need to be identified. With many Western nations showing increasing disparities between wealthier and poorer citizens (Cingano, 2014), the influence of socio-economics on COPD development will only intensify.
5. Diagnosis of COPD

Unlike the identifying of exacerbation, which depends on heightened rates of symptoms, the diagnosis of general COPD by medical professional rests on identifying mild symptoms. This is considerably challenging, and is a reason for the believed under-diagnosis of COPD. If patients do seek medical support, spirometry is a common technique to assess lung capability (Zwar et al, 2011). Patients exhale into a spirometer machine, for the recording of two measurements. Firstly, the volume of breath exhaled in one second (called the Forced Expiratory Volume, FEV1) provides some indication of air flow obstruction when considered against predicted normal values based on age, BMI, gender and ethnic origins (Hole et al, 1996; Young et al, 2009). The second measurement, FVC (Forced vital capacity) is the volume of air that can be exhaled after full inspiration measured in litres. Often FEV and FVC are combined to form the FEV1/FVC ratio (Hardie et al, 2002). Lower values of both measurements indicate likelihood of COPD. Spirometry can also be utilised to expose COPD before symptoms have come into full effect, or observe how effective treatment is post-diagnosis (Pellegrino et al, 2005). Further diagnosing measures include chest X-rays, able to reveal the presence of emphysema. An X-ray can also identify the existence of other lung problems or heart failure. CT scans of lungs can also discover if emphysema is present, and determine if lung surgery for COPD is viable (Sverzellati et al, 2007).

6. Treatment of COPD

Identification, reducing and control of risk factors are the best methods of preventing COPD development. The cessation of smoking, although often a complicated and psychologically challenging task, is the single most effective way of reducing risk of COPD progression (Pelkonen et al, 2001). Secondly, exposure to occupational and environmental air pollutants should be reduced as much as possible (Becklace et al, 1989). No medication is effective in completely reversing the progressive nature of COPD. However, symptoms can be mitigated with the correct application of medication to limit risk of exacerbation. Bronchodilator drugs commonly prescribed in the management of COPD, administered with inhalers. Bronchodilators expand and smoothen air passages in the lung to ease respiration. There exist both long and short-acting relief medications that can be used together and independently (Wyka et al, 2012). Alternative medical practices include vaccinations against influenza, oxygen therapy, pulmonary rehabilitation and surgery. Vaccinations against influenza reduce serious illness and mortality in COPD patients by approximately 50% and should be administered before and during winter months when comorbidity vulnerabilities are high. Oxygen therapy is the use of breathing apparatus that, if administered for over 15-18 hours a day, can aid the mobility of severely affected COPD patients. The primary goal of oxygen therapy is to increase baseline arterial partial pressure or achieve oxygen saturation (McDonald, 2014). The components of pulmonary rehabilitation differ widely but generally involve exercise training, nutritional advice and education (Nici et al, 2006; Schols et al, 1998). As the conditions and characteristics of each patient vary significantly, it is recommended that patients follow the specific regimen designed by their doctors. Lung volume reduction is a palliative procedure aimed to improve respiratory function by expanding lung cavities that have been compressed by dyspnea, and has been shown to improve quality of life for several years in patients that were deemed suitable to receive it (Young et al, 1999).

In appropriate cases, unilateral or bilateral lung transplants can help patients with severe COPD.
Home management of COPD exacerbations involves increasing the frequency or dosage of self-applied medication in accordance with symptoms. One of the most significant challenges facing diagnosed COPD patients is correct adherence to medication. A Lung Health Study of 5, 887 COPD patients (Anthonisen et al, 1994) was undertaken determining adherence by weighing inhaler cannisters and allowing patients to self-report, an often inaccurate method. Inhaler adherence declined over time from over 60% to below 50% five years later (Anthonisen et al, 1994). Another study of 11, 376 COPD patients in the last year of life found only 52% used any medication (Jung et al, 2009). Non-adherence or inadequate adherence results in exacerbations, and expensive hospitalisation, that may have been preventable. This demonstrates that diagnosis is not the turning point for COPD sufferers, and that challenges continue. Various psychological barriers exist as to why many COPD patients do not follow prescribed medication regimen (Restrepo et al, 2008). The first of which is the perception that no obvious link exists between medication and immediate symptom reduction. Some patients do not believe their case of COPD is serious enough to grant such treatment, or worry that reliance on medication will weaken its effectiveness at times of acute exacerbation. Complexity in applying medication, especially when comorbidities need to be addressed, may confuse patients, particularly those more senior. Patient depression, the relationship with their health professional, and access to medication, support and follow-up appointments, may also influence adherence (Restrepo, 2008).

Recent improvements in technology are allowing for the monitoring of adherence to move beyond canister weighing and patient reports, both of which are limited by inaccuracies (Bonini and Usmani, 2015). Inhalers that record use and communicate this data to a database, helps to inform the health sector of how patients are adhering to medication. This information has been used to discredit earlier adherence tests, by discovering that patients often emptied their inhalers before an appointment to appear as if correct adherence had been achieved, without knowing the actualisations had been temporally-stamped. Bonini and Usmani (2015) call on this data to be used to further the understanding of the relationship between regular and irregular, or frequent and infrequent inhaler use with exacerbation rates. Use of this technology will form one of the most significant steps forward in COPD research in decades.

7. Summary

COPD is a lung condition characterised by airflow obstruction and breathing limitation. The condition typical worsens with time and is not fully reversible (GOLD, 2014). COPD is the leading cause of death worldwide, and is projected to become the third by 2030 (Schirnhofer et al, 2007). The disease is blamed predominately on tobacco smoking, and indoor and outdoor air pollution. Air pollution is also a leading trigger of exacerbation, when symptoms worsen for several days. Air pollution, and its observed correlation with COPD (Hnizdo et al, 2004), forms the main focus of this thesis and will be explored further in Chapter Two (page 25).
Chapter Two – Air Pollution

1. Background to Air Pollution

The main environmental factor by which this thesis will compare COPD prevalence is that of air pollution. This chapter explores the various types of air pollution found in urban areas, and the negative impacts it has on health, specifically COPD. To aid in our understanding of temporal statistics, the effects of pollution levels on COPD exacerbation are included, as are the meteorological effects of wind speed and temperature. Also contained within this chapter are the concepts of environmental justice, essentially how poor socio-economic demographics may be more likely, through cultural and economic pressures, to inhabit areas more prone to high pollution levels (Been, 1993, 1995, 1997). The chapter concludes with particular reference to the air pollution and socio-economics of Christchurch city.

Air pollution is a broad term that encapsulates the presence of any substance in the atmosphere that causes harm to human health including fumes, dust, mist and smoke. If there is adequate concentration for a sufficient time, the particles threaten to be adverse to humans and property, flora and fauna, or whichever interferes with satisfactory life. Air pollutants can arise from both manmade and natural means (Wang et al, 2004). Pollutants are generally classified into two groups. Primary pollutants are the consequence of fuel combustion by home heating, industry and traffic. Secondary pollutants are those produced through chemical and photochemical reactions of primary pollutants, especially when mixed with water (Daly & Zannetti, 2007).

1.1 Particulate Matter

Officially, Particulate Matter (PM) is considered to be any dispersed material, whether in solid or liquid form, under 500 um (Figure 3). The term ‘dust’ generally represents particles that range from 1 to 200 um in size. Fume is a secondary pollutant, characterised by very fine solid or liquid particles that arise from reactions of chemicals and gaseous condensation. Smoke denotes fine particles which result from incomplete combustion of material such as coal, wood or petroleum (EPA, 2003).

![Figure 3. A diagram to demonstrate the sizes of particulate matter compared to human hair (EPA, 2016).](Image)
Particles between 0.5 and 10 um, referred to as thoracic particles, penetrate the lungs. These particles cause the greatest harm when deposited inside the lungs, whether as primary or secondary pollution. If concentrations of particulates in the atmosphere are above 400 ug/m3, especially when conjoined with sulphuric oxides, risk of respiratory infections, bronchitis, asthma and pneumonia is heightened substantially. Many particles that have been produced through carbon combustion, specifically those which contain Polycyclic Aromatic Hydrocarbons (PAH) are suspended carcinogens (Brown et al, 2013).

Air pollution in urban areas that are especially small (PM2.5), below 2.5um in diameter, differ in chemical composition to larger particles. PM 2.5 contain proportionately higher levels of water and acid-forming chemicals such as sulphate and nitrate, and trace metals too, such as chromium, cobalt, copper, iron, magnesium, selenium and zinc (Qui et al., 2013). Smaller particles infiltrate buildings easily and are reasonably consistent in their dispersal across the urban zones that produce them. Scientists now acknowledge that fine particles are additionally injurious for respiratory health due to their capacity to reach deeper into the lungs and affect greater surface area (Henschel et al, 2012). Therefore, toxic pollutants often accumulate to higher levels in the lungs (Henschel et al, 2012). In response to these discoveries, the World Health Organisation (WHO) now recommends the use of PM2.5, as opposed to coarse particulate matter, when monitoring concentration of urban pollution (WHO, 2013).

The major health effects from PM are increased mortality, the aggravation of pre-existing lung and cardiovascular disease, hospitalisation and restricted activity, resulting in lost work and school days. The most affected demographics, when exposed to particulate matter are the elderly and infants, those who suffer asthma, COPD, bronchitis, cardiovascular diseases and pneumonia. Research suggests that any contact is harmful based on the level of concentration, and cause drowsiness (Tanvir and Begum, 2010).

1.2 Sulphur Dioxide

Sulphur Dioxide is a toxic irritant when inhaled, particularly in the case of asthma sufferers. SO2 directly affects the upper airways within minutes of inhalation. Symptoms include wheezing, chest tightness, coughing and shortness of breath, related to the decreasing in bronchial capacity and airway restrictions. Inhalation during exercise may lead to severer symptoms. Epidemiological research has revealed considerable associations between daily average SO2 levels and mortality from respiratory and cardiovascular disease. Increases in hospitalisations are also evident for asthma, COPD and other respiratory conditions. These diseases, and lung cancer, are associated with long-term exposure to SO2 (Viegi, 2006).
1.3 Nitrogen Oxide

The relationship between Nitrogen Oxide inhalation and reduced airway function is well established, although it is considered reversible. Sensitivity to natural allergens may also be influenced by exposure to NO2. Recent epidemiological studies have demonstrated that NO2 exposure is also linked to climbing daily mortality and respiratory-related hospitalisations (Vagaggini et al, 1996). Research suggests that NO2 intensifies susceptibility to other irritants, such as ozone and particulates. The health implications of NO2 at low concentrations are not fully established and conflict exists in results regarding a threshold of adversity (Vagaggini et al, 1996).

1.4 Carbon Dioxide

The health implications of exposure to carbon dioxide are now well defined in research literature (Tran, 2009; Rice, 2003). Low concentrations of carbon dioxide are associated with heavy breathing, sweating and increased heart rates. Higher concentrations of carbon dioxide, cause acute poisoning, leading to eventual nausea, loss of consciousness and even death at CO levels of over 40% (Tran, 2009; Rice, 2003).

2. Meteorological Effects

Levels of air pollution depend not only upon shifting variation in emissions but also meteorological conditions. Weather can alter the concentrations of air pollution at hourly scales (Seinfeld and Pandis, 2006). Generally, windy conditions aid in the dispersal of pollution, whilst calm conditions allows for the build-up of pollution. Coastal and open areas tend to experience higher exposure to winds, therefore reducing lingering levels of pollution (Melas et al, 1995). Wind direction is also an important factor on a wider scale, if residential zones are situated downwind of an industrial area they will be more exposed to pollutants (Seinfeld and Pandis, 2006).

Atmospheric inversion controls the dispersion of pollutants by limiting vertical mixing (Melas et al, 1995). Inversion layers can be formed in a number of ways (Bourcier et al, 2003). Firstly, elevated subsidence is a common form of inversion, generally related to subtropical anti-cyclones where compressions warms the air as it falls in a high pressure system and reaches a temperature higher than that of the air below. An inversion will occur if the temperature increase is at a sufficient level (Bourcier et al, 2003). The subsidence is instigated by air flowing down to replace the air underneath, which has the high-pressure area. Secondly, another common type of inversion is radiation. The earth’s surface cools at night when losing energy through radiation. This radiation is obstructed on cloudy nights due to absorption by water vapour, where much of the energy is returned (Marthews et al, 2011). In contrast, on clear nights, ground cooling occurs quickly due to less obstruction to radiating energy. With a cooling ground, bordering air temperature also decreases. As is common on cold clear nights, the air temperature directly above ground becomes colder than the air above, generating an inversion, which typically forms at dusk. As the night continues, the inversion
expands to continually higher elevations; sometimes stretching a few hundred metres before morning sun heats the earth again, breaking up the inversion. Finally, advective inversion is generated when warm air drifts over cold surfaces (ground-based) or air (elevated). For example, elevated advective inversion occurs when a warm land breeze is forced over a hill range whilst a cool sea breeze flows at low level in the opposite direction (Admassu and Wubeshet, 2005). Air pollution is often associated with lower temperatures, when use of home heating increases, and commuters are less compelled to walk or cycle, instead opting for their vehicles (Tin Tin et al, 2012).

3. Health Impacts of Air Pollution

3.1 Introduction to Health and Air Pollution

The established link between air pollution and health effects forms the central premise of this thesis. The evidence that ambient air pollution has adverse health effects, and leads to increased mortality, is supported by a large number of epidemiological studies across the globe, although precise biological mechanisms continue to be unknown (Pope and Dockery, 2006). Inflammation of airways, in a similar means to cigarette smoking, is the most agreed upon effect of inhaling air pollution.

Besides concentration of air pollution, the temporality of exposure is also an important factor in determining risk of poor health outcomes. Different groups within a population are more susceptible to the effects of air pollution, such as elderly people with existing respiratory and cardiovascular diseases (Le Tertre et al, 2002; Staffogia, 2010), asthmatics (Strickland et al, 2010), and infants (Gent, 2009). It remains unresolved whether the source of air pollution has a significant impact on the levels of adversity caused.

Particulate pollutants damage airways in variety of ways, including the narrowing of bronchial airways, pulmonary and systemic inflammation, amplification of viral infections, and reduction in airway ciliary activity. Evidence suggests that the relationship between air pollutants and lung damage is causal, although a causal association between air pollution and COPD remains more difficult to verify, as a long-term study involving spirometry tests has not yet been undertaken (Staffogia, 2010).

3.2 Indoor Air Pollution and Health

The most common indoor air pollutants include tobacco smoke, particulate matter, nitrogen oxide, carbon monoxide, volatile organic compounds and biological allergens. As mentioned, smoking often considered the most significant factor in the development of many lung diseases, including COPD (Eisner, 2010). Indoor environments also contain exhaled tobacco smoke, referred to as second-hand smoke. A cross-sectional Chinese report (Ko, 2011) involving 15379 people over 50, who had never smoked, discovered a relationship between COPD risk and self-reported exposure to second-hand smoking at home and work.
(Ko, 2011). Similarly, a study in the USA involving 2113 adults aged from 55 to 75, revealed a link between second-hand smoke exposure and physician’s diagnoses of chronic bronchitis, emphysema and COPD (Eisner, 2005).

Approximately half of the world’s population uses biomass (wood, dung and coal) as the primary source of home heating, cooking and lighting. The inhalation of second-hand smoke and biomass smoke are averse to health, with the first containing at least 250 harmful chemicals (Kurmi et al, 2012). Exposure to biomass smoke has been associated with local scarring and pigment deposition in the lung parenchyma. Liu et al (2001) revealed that exposure to biomass smoke in rural China, among non-smokers, was considerably related to prevalence of COPD. A number of papers have drawn associations between indoor air pollution and increasing symptoms (Regalado et al, 2006), acute exacerbations, reliance on hospital services (McGowan et al, 2002) and mortality (Fairley, 1990).

3.3 Air Pollution and COPD

Not many research studies have investigated the causal nature between outdoor air pollutants and diagnosed COPD patients. A study involving 4,775 women, inhabiting the Rhine-Ruhr valley of central western Germany, discovered the prevalence of COPD was 4.5%, mostly influenced by PM10 and exposure to traffic (Schikowski et al, 2005). Proximity to roads strengthened likelihood of COPD development; for women living within 100m of a main arterial route, COPD was 1.79 times more likely than for those living away. These findings were further vindicated by a follow-up study in the same region, which discovered that among a subgroup of 402 women in 2008-2009, improving air quality accompanied decreasing rates of COPD (Schikowski et al, 2010). Contrarily, some papers have found very little evidence of correlation between air pollution and COPD. A paper involving 2644 adults in Nottingham, UK found no significant association between proximity to nitrogen oxide and traffic pollution and reduced lung function, or COPD (Pujades-Rodriguez et al, 2009). A wide-reaching Danish study (Andersen, 2011), involving 57053 patients, from 1993-2006, discovered associations between first admissions to hospital for COPD exacerbation and exposure to traffic pollution. COPD prevalence was related to the 35-year average nitrogen oxide level. While evidence of lung damage associated with air pollution is well supported, the direct causal relationship between pollution and COPD remains elusive. It is recommended that studies follow subjects from birth to gain complete understanding of COPD development (Andersen, 2011).

3.4 Air Pollution and COPD exacerbations

Some previous studies have revealed associations between outdoor air pollution and more severe symptoms, increasing exacerbations and subsequent hospitalisations (Arbex et al, 2009). Studies on a wide-scale in North America and Europe revealed considerable relationships between air pollution concentrations and hospital admissions. Research into hospitalisations for
cardiovascular and lung diseases in population over 65 in US cities, found a 2.5% increase in COPD admissions for a 10 um/m3 climb in PM10 (Zanobetti et al, 2014). Similarly, more research from the USA discovered that the same concentration of PM2.5 was associated with a 0.9% increase for COPD hospitalisations. (Dominici et al, 2006).

The relationship between air pollution and hospital admissions for COPD has been well established in literature (Baillargeon. 2013). Overall, international studies have found that increases in fine particular matter were correlated with increases in COPD hospitalisations (Zhu et al, 2013; Tsai, 2013; Ghozikali et al, 2016), as were levels of carbon monoxide (Tian, 2014). A similar, but reduced, trend was found in Sydney (Morgan et al, 1998). Sydney’s annual average for PM10 is 20ug, notably lower than what is found in Northern hemisphere studies, where averages are in the range of 35-48ug. This suggests that particulate matter at any level may increase COPD hospitalisation.

A study conducted by Anderson et al (1997), in association with the ‘Air Pollution on Health: a European Approach’ project studied the cities of Amsterdam, Barcelona, London, Milan, Paris and Rotterdam. The association between a 50 um/m3 increase in daily pollution averages and COPD admissions was explored. Results for SO2, black smoke, particulate matter, nitrogen oxide and ozone each were associated with higher COPD admissions, after lagging 1-3 days. A study in Rome (Fusco et al, 2001) found that CO and nitrogen oxide were linked with acute symptoms for respiratory disease. It was further noted that for all ages, a rise in carbon dioxide of 1.5 mg/m3 was associated with a 4.3% increase in COPD admissions. A study from Norfolk, a rural county of England, which features lower pollutant concentrations than urban areas, discovered that frequency of hospital admissions for COPD exacerbation was directly tied to fluctuating concentrations of CO & NO, as has been had been observed in urban areas (Sauerzapf et al, 2009). A study in Hong Kong used 119,225 COPD admissions from 2000-2004, observing the risk of hospital admissions for increases in SO, NO, PM10 and PM2.5. All were associated with increases in hospitalisation, particulate matter most predominately, although often increased hospitalisations occurred 0-5 days after peaks in pollution concentration (Ko et al, 2007). Sunyer et al (1991) assessed the rate of COPD hospitalisation in Barcelona Spain during 1985-1986, and found increases in hospitalisations rose by 0.02 and 0.01 for each milligram of SO and black smoke per cubic metre, respectively, and by 0.11 for each milligram of CO per cubic metre, following meteorological and temporal adjustment. Similarly, a time-series from Sao Paulo, Brazil involved 1769 sufferers of COPD, discovered that PM10 and SO concentrations were linked to hospitalisations (Arbex et al, 2009). Increases of 28.3 mg/m3 in PM10 and 7.8mg/m3 in SO, resulted in a 6-day cumulative climb in admissions of 19% and 16%, respectively. One notable exception was a study of COPD admission to 14 Canadian hospitals from the 1990s to the early to the early 2000s. Of the 400,000 hospital visits studied, no overall positive correlation could be found between air pollution and hospitalisation (Stieb et al, 2009).

The understanding of the true effects of pollution on COPD is limited by under-diagnosis, mostly attributable to patients who do not seek emergency treatment. The effect of air pollution on COPD was investigated by collecting diary symptoms records from
95 COPD patients in London, UK. Significant relationships were discovered between symptoms and increases in PM10, NO and black smoke (Peacock et al 2011).

A number of studies have found that air pollution increases risk of COPD mortality (Gan et al, 2013; Meng et al, 2013). A study of COPD patients over the age of 65 in 29 European cities discovered that a 10 ug/m³ increase in PM10 and black smoke was related with daily rises in mortality of 0.8% and 0.6% respectively (Aga et al, 2003). Particulates were found to have more significant associations with mortality than gases such as NO and ozone.

The overall conclusion indicates that the causes of COPD from air pollution remain unconfirmed, but there is more significance in relation to exacerbation. Fewer studies have examined the effects of indoor air pollution and the subsequent aggravation of symptoms among patients with pre-existing COPD. In a recent research study of 809 COPD patients in the USA, exposure to second-hand smoke was linked with poorer COPD outcomes. Exposure to second-hand smoke was associated with increased rates of exacerbations (Eisner et al, 2010). Exposure to indoor pollutants was linked with increases in respiratory symptoms and heightened the risk of exacerbation (Hansel et al, 2013).

4. The Geographies of Socio-Economics and Pollution

4.1 Introduction to Socio-Economics and Pollution

As established in Chapter One, the relationship between lower socioeconomic status and COPD is well established. Additionally, as covered in this chapter, air pollution has adverse effects on lung functionality and negatively impacts COPD. Completing this three-way correlation is socio-economic status, a major factor in the level of exposure, and subsequent health problems (Adler and Stewart, 2010). It has been found that air pollution concentration can be distinguished by residential segregation based on class, and that pollution production is generally clustered in less affluent areas. Marshall (2008) studied environmental inequality in California’s South Coast air basin, using linear regression based on a mobility-based exposure model for 25,064 locals. For all pollution types besides ozone, average exposures are more substantial for ethnic minorities, those on lower incomes and those living in denser urban areas. In many cases, these three variables are mutually inclusive. There was a 50% increase in exposure for deprived minorities over affluent ethnic majorities. Other studies also suggest that areas containing higher rates of deprivation and minority populations share a larger burden of air pollution exposure and risk (Bell et al, 2005; O’Neill 2003).

Several studies have reported associations between air pollution and lower socio-economics. Jerrett et al (2001) interpolated estimates of air pollution exposure in Hamilton, Canada and compared it with socioeconomic and demographic census data. This revealed that significant associations existed between low socioeconomic status and exposure to air pollution. Similar patterns
have been documented in Europe. On a continental scale, PM10 was recorded as being higher in less developed European countries, reflecting the historic West/East divide in Europe. These trends are highlighted best by Bulgaria, one of the most deprived nations in Europe, which contains 4 of the continent’s five most polluted urban environments, the exception being another eastern European city, Krakow in Poland (Richardson et al, 2013). The association between air pollution and lower socio-economics has been observed globally, such as in Norway (Naess et al, 2007), Brazil (Filluel and Harrabi, 2004), Canada (Perry et al, 2011), England (McLoed et al, 2000) and India (Holian, 2014).

4.2 Environment Justice

The reasons for the associations between deprivation, exposure to pollution and health are complex and cyclical. A commonly used term to summarise this relationship is ‘environmental justice,’ which offers five interpretations as to why such patterns exist. The first, and simplest, is the contention that corporate industries choose to build factories in neighbourhoods of weaker political sway in order to avoid organised opposition to pollution. Hamilton (1993; 1995) discovered industry planned to expand their processing of hazardous wastes in areas with lower voter turnout. Evidence suggests that immigrants and ethnic minorities are generally less politically engaged (Adamson, 2006) and tend to live in clustered communities. This aids the explanation as to why pollution, and subsequent health effects, may affect ethnic minorities to a greater extent.

Linked with this, the second interpretation suggests that firms are attracted to these areas due to generally low land prices, access to main arterial routes and proximity to suppliers due to the benefits found in clustering business activities (Banzhaf, 2008). The third interpretation blames government for not enforcing environmental standards and regulations equally. Government decisions for enforcing standards more strongly in certain areas could be based on support attainable from more politically engaged populations (Banzhaf, 2008). Government enforcement agencies might lack incentives to enforce standards unless to do so by stakeholders. Agencies are more likely to respond to pressure from better organised, connected and politically powerful citizens.

The fourth interpretation speculates that market responses to property prices control who inhabits the area. If pollution or other environmental problems deriving from industry are evident, wealthier residents will be financially able to leave driving down property values and allowing for the immigration of the relatively poor. This process was first defined by law professor Vicki Been in a series of studies (1993; 1995 & 1997). It was later validated by the modelling work of Banzhaf and Walsh (2008). The commonly cited association between immigrants or ethnic minorities and deprivation is another potential catalyst for these groups inhabiting areas of poorer health outcomes. This interpretation is also most applicable to major road networks, themselves large contributors of toxic pollutants. Proximity to this pollution and road noise will act as an incentive for wealthier residents to leave. This will also result in falls in property values, and the immigration of poorer populations.
The final interpretation is that the geographic distribution of local pollution emission is a result of negotiations between industry and local residents, where companies compensate neighbourhoods for hosting protested factories (Hamilton, 1993; 1995). Ronald Coase (1960) claimed that firms would choose to locate themselves in neighbourhoods that would be willing to accept lower payments as compensation, typically the poor.

5. Christchurch’s Air Pollution Problem

Particulate matter from domestic solid fuel heating contributes 80% of Christchurch’s air pollution (Canterbury District Health Board, 2012). Pearce et al (2006) discovered that PM10 levels were not uniform across the urban area, but were clustered in central areas compared to surrounding suburbs. Of these suburbs, higher exposures were measured in the north and east of the city centre. This result is based upon spatial variation in pollution production due to denser housing and higher traffic counts in central areas, at least in pre-earthquake times. Variations in localised meteorological conditions because of wind and the effects of foothills also influenced pollution distribution. The total air pollution levels in Christchurch range from 0.06 ug m\(^{-3}\) to 31.02ug m\(^{-3}\) annually, with a mean of 14.26 ug m\(^{-3}\). On average, thresholds for pollution levels were surpassed on 3.30% of days (12 days per year) in the years before their publication (Pearce et al, 2006). In 2010, Christchurch experienced 15 high pollution days, while this increased to 32 in 2011, 19 in 2012, and 23 in 2013 (ECan, 2014).

In Pearce et al’s study (2006) two variables were used to investigate socioeconomic differences in exposure; household income and deprivation. Low-household income and deprivation was correlated with higher pollution exposure, and lower pollution was associated with richer households (Pearce et al, 2006). Patterns for domestic and vehicle pollution also followed these socioeconomic patterns (Pearce et al, 2006). Finally, the average number of days when pollution in each area unit exceeds the recommended 24 hour means pollution threshold (50 ug m\(^{-3}\)) was compared to the Deprivation Index. The average number of exceedance grew from the least deprivation areas (1.81% or 6.60 days per year) to the most deprived (5.56% or 20.31 days per year) (Pearce et al, 2006).

Wind field analysis in Christchurch discovered that the Western and South-western regions of the city receive fresh cold air draining from the Southern Alps and the Port Hills (Kossmann and Sturman, 2004). Pollution monitoring found the Port Hills sites to have lower rates of particulate matter due to this elevation-induced draining (Wilson et al, 2006).

To reduce Christchurch’s air pollution problem, restrictions have been introduced to limit the use of wood and coal fires (Young et al, 2013). Current legislation rules that wood fireplaces cannot be installed in new or rebuilt homes, or homes that residents move to. Wood burners over the age of 15 years cannot be used between April and September each year. Instead, alternative heating technology must be used such as pellet fires, heat pumps, and fluid gas fire or low-emission burners. Proposed changes allow for low emission burners that produce 0.5g of particulate matter per kilogram of wood burnt, as opposed to those which produce between 1-5g. Bowing to pressure, ECan air commissioner David Bedford relaxed rules for the vulnerable on
16/06/2012, allowing quake-affected residents to use an open fire or older wood burner. Bedford said that the earthquake had affected air quality, but that Christchurch was not on track to meet targets anyway. The effect of the earthquakes can be demonstrated by high pollution days, when Christchurch exceeds guidelines for pollution levels. In 2010, Christchurch experienced 15 high pollution days, while this increased to 32 in 2011, 19 in 2012, and 23 in 2013 (ECan, 2014). Although timeframes are contested due to technological and judicial complications, it is expected that pollution caused by fireplaces will eventually decrease (Young et al, 2013). If the findings of this thesis indicate a relationship between air pollution and COPD, this will reduce a significant factor in the diseases development.

6. Summary

Air pollution is a broad term that encapsulates the presence of any substance in the atmosphere that causes harm to human health including fumes, dust, mist and smoke. Air pollution has a particularly strong effect on elderly people with existing respiratory and cardiovascular diseases (Staffo gia, 2010), asthmatics (Strickland et al, 2010), and infants (Gent, 2009). Air pollution has also been found to be correlated with COPD, and the severity and frequency of acute exacerbation (Eisner et al, 2010; Stieb et al, 2009). Theories have been presented that claim lower-socio economic areas are more likely, through economic pressure, to contain higher rates of air pollution emission (Banzhaf, 2008). By their nature, environmental variables related to COPD call on spatial and temporal concepts to aid in their description and explanation. GIS is the most suitable means of exploring the relationship between air pollution exposure and COPD hospitalisation patterns. Chapter Three will demonstrate how Geographic Information Systems have been applied to health and COPD research.
Chapter Three – GIS and Health Science

Geographic Information Systems (GIS) are becoming an increasingly important tool in the advancement of health science. GIS has aided research into the prevalence and spatial patterns of various health conditions, and the associated socio-economic and environmental factors. A growing body of research is using GIS to find how disease and influential factors interact spatially (Andersen et al, 2011; Peacock et al, 2011; Ghozikali et al, 2016). GIS is multi-faceted, and contains many tools that can be applied to answer important geographic questions. This chapter explores international and local papers that have utilised spatial autocorrelation, land use regression, Ordinary Least Squares regression and Geographically Weighted Regression to improve understanding of health conditions, including COPD, and associated factors, such as deprivation, ethnicity and smoking.

1. Spatial Patterns

Often research involving spatial analysis begins with an assessment of the spatial autocorrelation of features (Figure 4). This refers to the degree to which spatial variables and their associated data values tend to be clustered together (positive spatial autocorrelation) or dispersed (negative spatial autocorrelation). Moran’s I is one index used to measure spatial autocorrelation, and ranges from -1 (dispersed) to +1 (clustered). Zero represents patterns demonstrating random distribution. Identification of clustering is important in spatial analysis as it reveals that influences exist beyond the single variable, paving the way for more complex spatial analysis. This technique, which will be detailed in the Methods chapter (page 42), has been used extensively to describe patterns relating to variables featured in this thesis, including air pollution and COPD distribution. For example, Moran’s I has been used to assess the spatial patterns of cardio-respiration diseases in Brazil (Requia and Roig, 2015). It was found that clustering existed across all ages, albeit weakly, with a Moran’s I value of 0.1, and the under 5 years old demographic exhibited the most statistically significant clustering with a Moran’s I value of 0.25. My own thesis will also apply this technique to assess clustering of COPD patients.

There exist two categories of spatial autocorrelation testing; global and local. Global assumes homogeneity across the region, essentially that points which are close together have similar values on average. Global tests do not provide indication of where

![Figure 4. Diagram demonstrating the three broad categories of spatial autocorrelation (O’Sullivan and Unwin, 2003).](image-url)
clustering is occurring, a significant aspect of spatial analysis. Local spatial autocorrelation tests (Anselin, 1995) seek to avoid these limitations, and test for the extent to which points that are close to a specific point share similar values. Both local and global spatial autocorrelation are limited in only evaluating single variables. If a variable is clustered, it suggests that other variables may be influencing it. Although it is not possible to have definitive evidence of these causal relationships, as exploratory spatial analysis is dependent on the identification of potentially associated variables.

2. Mapping Spatial Variation of Socio-Economic Variables

This thesis focuses on variables relating to the rates of deprivation, smoking, old age and Maori/Pacific Islander population in Christchurch. This particular combination, when combined with COPD hospitalisation and air pollution data is unique. Deprivation mapping, the spatial distribution and analysis of indicators of human wellbeing and poverty within a region, is useful in widening understanding factors behind disease (Williams et al, 2004). Deprivation maps provide an example of how socio-economic indicators can reveal spatial patterns at a variety of scales. Choropleth maps allow visual comparison, allowing for the identification of spatial trends, clusters, or other patterns. The spatial representation of these socio-economic variables can therefore compliment regression analysis to further the understanding of background influences.

Deprivation is a multidimensional phenomenon, including economic, social and other aspects of human wellbeing. The selected variables may be monetary or non-monetary indicators such as the percentage of households within a specified income bracket (Pearce et al, 2007) or the proportion of households without access to sanitation. Data used to construct a deprivation map are typically drawn from population censuses, household surveys, or spatial databases in which values are fixed to specific locations (Salmond and Crampton, 2002). Increasingly, poverty mapping relies on data from many sources. Data may differ in its coverage, collection method and level of resolution all of which may have methodological implications (Tobias et al, 2001). The level of resolution of data used in deprivation mapping may be fine (household level) or relatively coarse (averages for Census or administrative units). In New Zealand, the Deprivation Index includes specific questions pertaining to economic security; including the reliance on welfare, charities and food banks, as well as lifestyle sacrifices such as not heating homes or replacing leaking shoes, at meshblock level (Atkinson et al, 2013).

Research highlights the ability of GIS to aid public service in understanding the characteristics of the communities they serve. A number of studies have incorporated deprivation mapping into disease distribution research (Finkelstein et al, 2005; Williams et al, 2004), allowing for the better understanding of where diseases occur more predominately and potentially how these challenges could be addressed. Besides deprivation scores, other variables can also be held within spatial features and used for geographic correlation. A recent study (Fu, 2014) sought to explore the geographic patterns of tobacco smoking and high alcohol consumption in India. Data was obtained through census surveys at district and postal code levels. Cluster maps were also
produced both of univariate patterns and bivariate correlation, finding significance in the first but not the latter. A Quebec study (Guend and Rondeau, 2009) identified the density of elderly people across the region with census data, and compared it to the density of doctors. From census and survey data, the characteristics of the elderly residents could also be incorporated into the study, such as obesity rates and deprivation level. It found these were correlated, and associated with the likelihood of having fewer family doctors available.

Ethnicity can also be included in census-based studies, which can aid public health services by widening understanding of ethnic inequalities. A New Zealand study (Kruger et al, 2012) sought to find spatial relationships between geocoded dentistry practises and area units ranked on the percentage of Maori and Pacific Islanders in the adult population. Results found dentists were likely to be located in communities with lower Maori and Pacific Islander populations. These areas were also associated with higher socio-economics and better oral health. This aligns with inverse care law, a concept first proposed by Tudor Hart (1971) which recognises that often the availability of good medical or social care tends to be located in areas with lower levels of need. Lower-socio economic areas are therefore neglected. These papers represent a carefully chosen subset of relevant examples on how GIS is used in public, and the range of variables that could be used in this thesis. However, this thesis will also use environmental data, namely air pollution monitoring, and will utilise Geographic Information Systems to build prediction maps.

3. Estimating Air Pollution with LUR

Due to limitations in monitoring air pollution across urban areas at a fine scale, land-use regression (LUR) is used as means of estimating the air quality based on the prevalence of likely emitters. The LUR model calculates a number of regressions in order to determine the correlation of recorded air pollution concentrations and potential predictor variables held as spatial data. Potential predictors for high pollution used in previous studies include the proximity to industrial or commercial land and being relatively distance from green space and water areas (Chen, 2012).

LUR modeling has become, over the last decade, the preferred method of assessing spatial variation in exposure to pollutants. In relying on spatial data, LUR models are subjected to the same effects associated with these methods, namely the scale and aggregation errors. Confidence in the predicting values depends on how well they align with the measured sites (Chen, 2012). If confidence in LUR models is assumed, they can be applied to other urban areas with similar geography and prediction data (such as info on home-heating/traffic etc.), but where a limited number of pollution measurements have been taken. Most of the earlier studies recommend using models built specifically for each area calculated, despite spatial distributions being generally well described by transferred models. When a LUR model is applied to a city with limited air pollution monitoring sites, an understanding of localised potential pollutant sources in the form of spatial data is essential (Chen, L. 2012).
A LUR study from Hamilton, Canada (Jerrett, 2009) found association in relation to NO2 air pollution and a variety of cardiovascular and respiratory illnesses. Land-use categories included commercial, industrial, green space, residential, industrial and water space. Population and housing densities were calculated using census variables, and road network data pertaining to traffic densities. Further variables included a digital elevation model where low-lying areas were considered more likely to hold pollution in meteorological models. Wind direction measurements from local windsock recordings were incorporated, as sheltered areas were more likely to contain higher pollution concentrations. Industrial NO2 producers were added as points. Predictor variables were generated by circular buffers ranging from 100m to 600m. Separate summer and winter regression models were produced to provide more temporally accurate descriptions of air pollution distribution.

A land-use regression model was built to estimate long-term residential exposure to air pollution in Germany (Vossoughi et al, 2014). This predicted exposure was based on traffic indicators and individual monitoring sites which were interpolated to cover the Ruhr and Borken area (Germany) in which 402 elderly women lived. They were monitored for lung inflammation, and their individual conditions were compared to the pollution they were exposed to. Results revealed that there was a high variability in the severity of lung inflammation experienced. High lung inflammation was associated with increased pollution exposure. The authors (Vossoughi et al, 2014) expected the same pattern to exist in the wider elderly population.

LUR modelling has not been applied a significant amount in New Zealand research. It first appeared in a study (Kingham et al, 2008), where LUR models were used to predict PM10 exposure on a national level, using household fire densities, industrial emission estimations, traffic densities and meteorological measurements. Predictions were compared to monitoring sites and correlation was found, validating the model. LUR modelling was undertaken in Auckland (Kingham et al, 2013) to test the ability of the model to predict air pollution at fine spatial scale. Two sites were used, Otahuhu at 10km² and Mangere at 70km², which were divided into gridded area units containing their own variable values. The geographic variables used to build prediction models were proximity to roads and highways, and the sums of traffic density and road lengths within each cell, deemed to have a positive impact on air pollution. Proximity to coast and elevation, deemed to be negatively associated with pollution, were also included. As with the earlier model (Kingham et al, 2008), monitored results from 26 sites in Mangere and 32 in Otahuhu found that pollution was higher nearer to roads. The modelling for Mangere was relatively well aligned with monitored sites, but the modelling for the smaller Otahuhu deviated from it. The findings indicated that challenges arise in building LUR at finer scales. The authors hypothesized that smaller areas contain lower variability, resulting in lower probability of strong contrasts than when measuring larger areas (Kingham et al, 2013). They suggested that LUR may not be as effective in the range of 4-30km² study areas. Christchurch’s urban extent covers approximately 1,400km² so the issue of scale is not expected here. Additionally, LUR models have been most widely applied to larger urban environments rather than single suburbs (Hoek et al, 2008).
4. Correlation with Environmental Variables

Regression is used to find spatial correlation with multiple variables. A common statistical modelling technique used in spatial sciences is that of Ordinary Least Squares (OLS). Generally, a dependent variable is compared against a variety of independent variables outputting correlation coefficients, usually engaging a global model. The term ‘global’ implies that the spatial data used to calculate single statistics does not consider the surrounding area. In other words, the prime assumption in a global model is that the relationship between the dependent and independent variables are spatially consistent (stationary). More specifically, the global model assumes that the same stimulus provokes the same response in all parts of the study region. Although, in reality, the relationships between dependent and independent variables is often non-stationary and varies geographically (Cressie, 1993). There is an expectation that different processes are at work if non-stationarity exists within the study region. Standard global modelling techniques, such as ordinary least squares (OLS) linear regression, which is still commonly used in spatial research, cannot recognise non-stationarity. OLS conceals regional variation in the potential correlation between dependent and independent variables. A major problem with this technique when applied to spatial data is that the processes being examined are assumed to be constant over space - that is, one model fits all. Research and subsequent public policy based on global results, with concealed non-stationarity, will be unreliable (Ali et al, 2007). To combat these spatial problems, Geographic Weighted Regression is a statistical technique that allows for the modelling of variables over space. GWR extends beyond OLS linear regression models by challenging the assumption of non-stationarity, building separate models and local parameter estimates for each geographic feature using a differential weighting scheme (Figure 5). Fotheringham et al (2002) used the analogy of ‘spatial microscope’ when referring to GWR’s ability to acknowledge non-stationarity. This approach emphasises the differences across a map, and identifies localised ‘hotspots.’

Geographic Weighted Regression

![Regression Point Diagram](image)

*Figure 5. Values closer to the kernel centre are more influential than distant points (Harris, 2009).*

GWR has been applied to a number of health-related studies, including COPD. Comparisons between both ordinary least squares and geographically weighted regression were explored in a recent Taiwanese study (Chan et al, 2014), which used ecological risk
factors thought to influence COPD. Smoking rates, area deprivation index, tuberculosis exposure, ethnic aborigines, density of healthcare facilities, air pollution and altitude were considered on both models to evaluate their effects on mortality. Global and local Moran’s I were used for examining spatial autocorrelation and identifying clusters. Overall, males’ COPD mortality rate was approximately three times higher than females. The GWR results revealed median coefficients of smoking rates, percentage of ethnic minorities; PM10 and altitude were positively associated with COPD mortality in both males and females. Potentially linked with Tudor Hart’s theory of inverse care law (1971), the density of healthcare facilities was found to be negatively associated with COPD mortality. The overall adjusted R-squares were found to be approximately 20% higher in the geographic weighted regression than the ordinary least squares (Chan et al, 2014).

A study in England (Nacul et al, 2011) aimed to identify areas where undiagnosed sufferers of COPD are statistically more likely to live. The diagnosed number is only 765,000. The research again drew light on ‘global’ regression holding the belief in consistently across the study area. The authors performed GWR modelling to investigate the spatial variation between the density of diagnosed COPD patients and other predictor variables deemed related to COPD prevalence, including age group, gender, ethnicity, smoking prevalence, including age group, gender, ethnicity, smoking prevalence, area of residence (rural, suburban and urban) and deprivation. The authors tested for spatial non-stationarity by running classical regression and GWR using a Monte-Carlo simulation. The GWR results were visualised in mapping format to show the geographic variation in the diagnosed prevalence: expected prevalence relationship. Two GWR models were run, the first simply being the relationship of diagnosed to expected, and the second a bivariate regression involving diagnosed to expected and primary care density. Overall estimates of the number of COPD sufferers over 15 years old in England was 3.5% m, or 1.4 million. Geographically, a north-south gradient was found, reinforcing established findings that COPD is associated with deprivation; the urban north of England experiencing more relative economic hardship than the south. Comparison of correlation coefficients between classical regression results, and those of GWR, confirmed that GWR modelled the data more accurately than OLS regression.

Similar to the focus of this thesis, GWR has studied relationships between respiratory-related hospitalisations among 0-15 year olds, deprivalion, and PM10 exposure. Based in Leicester, the findings indicated that significant global relationships exist between children’s hospitalisation and deprivalion, ethnic minorities, and PM10 road-transport emissions. Local Indicators of Spatial Association (LISA) and (GWR) discovered significant variations across the dataset. These were related to the combination of residential traffic pollution and deprivalion. The author’s make the claim that wealthier urban communities tended to produce higher levels of pollution from private vehicles, despite residing in relatively low pollution areas, echoing the previously mentioned inequalities of environmental justice. (Jephcote and Chen, 2012).
5. GIS for COPD Hospitalisations

There is limited research that focuses specifically on the spatial association between air pollution and COPD hospitalisations. The most relevant to this thesis comes from a recent study based in Jinan, China, using 414 hospitalisation events (Wang et al, 2015). The home and working addresses were studied in the analysis, plotted as points across the Jinan city extent. In this study, LUR was not used, rather kriging method in ArcMap 10.1 that interpolated the monthly recordings of air pollution from fifteen monitoring stations. Spatial correlation calculations were undertaken to assess whether higher pollution was clustered with hospital events. Results revealed that points of residence were significantly spatially correlated to concentrations of SO2, PM10, NO2, CO and O3. A dataset of non-smokers was run as a means of testing the results, and also arrived at similar results, as did those for workplace location. After adjusting for potential confounders in the model, only PM10 remained the most significant type of air pollution to influence hospitalisation. In Los Angeles, between 1993 and 1999, a generalised least squares model found that COPD hospitalisation was more prevalent in areas associated with immigrant populations and deprivation, and higher numbers of tobacco outlets (Lipton and Banerjee, 2007).

An Indian study of 3,592 individual patients required the collection of questions of respiratory health, home heating techniques used and performed pre and post bronchodilator spirometry (Muralidharan et al, 2014). Using GIS tools, two spatial groups were defined: those living within 500m of major highways and those living beyond that threshold. Spatial statistics and logistic regression analysis was used to determine the spatial distribution of COPD and other geographic risk factors. Results found that COPD subjects residing within 500m of the highway are randomly distributed 72% of individuals residing within 500m used liquefied petroleum gas, which is considered relatively less harmful than biomass home heating, and have strong prevalence of COPD and proximity to highways. Individuals residing over 500m from highways show significantly clustered patterned, and have a higher prevalence of COPD which is strongly associated with the use of biomass fuel. Therefore, the prevalence of COPD among LPG users is significantly associated with the proximity to highways while those residing away from highways show three-fold prevalence due to use of biofuels. By this result, it appears as though home heating within homes has a stronger influence than traffic proximity, though this largely depends on the distant threshold used and the arrangement of the fireplace and chimney.

6. Summary

This chapter demonstrates the wide and multi-faceted applicability of GIS in health research. Health problems, and the variables that may cause or exacerbate them, are best understood and treated when their geographic characteristics are understood. The various geographic techniques that have been applied in GIS and health research, including spatial autocorrelation, deprivation and variable mapping, LUR, OLS and GWR will become the central techniques for this thesis on the basis of the literature discussed in the previous two chapters. Chapter Four will explain more specifically how the techniques are applied and how data collection was undertaken.
CHAPTER FOUR - Methods

In order to answer the aims and objectives, the thesis methodology will be based on the techniques used by previous researchers which were described in the previous chapter. The thesis will build on such research, by using spatial analysis to determine if a relationship exists between COPD hospitalisation rates and the distribution of air population and demographic variables. This will be combined with temporal statistical analysis to determine if air pollution and climate influence hospitalisation rate over a 12 month period. This thesis is the first piece of research that combines both spatial and temporal analysis to study COPD patterns in New Zealand.

1. Descriptive Statistics

The research process begins with an exploration of the data to find whether or not there exists any discernible trends in the selected population in regards to average age, gender, smoking status and number of hospitalisations suffered in the 12 month period from September 2014 to August 2015. Calculations will be performed by Excel and presented in table format. The calculations will determine the number of hospitalisations that each ethnic groups suffered, and how average the average ages of both non-smokers and smokers, and males and females, relates to the hospitalisation rates. This will indicate if any trends exist in who is experiencing hospitalisations, before the study focuses on where they occur most predominately.

2. Spatial Analysis

![Figure 6. Meshblock (left) and area unit (right) features with light green representing the Christchurch area that was studied.](image)

2.1 Spatial Autocorrelation

The next stage of the data exploration is determining whether patterns exist in the spatial data. Using Moran’s I calculations in ArcMap 10.2 results will reveal if patient points are spatially autocorrelated in particular areas, which will suggest geographic
principles are influencing distribution. Also explored will be the meshblocks containing the variables deemed to be associated with COPD, namely smoking rates, deprivation, Maori/Pacific population and elderly population. The meshblocks making up the Christchurch area were downloaded from coordinates.com. Each meshblock is assigned a numeric code, which will be matched to the Census data for that meshblock. Area Unit codes also accompany both meshblock and Census data, representing aggregated zones each comprised of a number of meshblocks. Regression is applied to both meshblock and area unit features (Figure 6).

Before regression analysis occurs, all meshblocks will be tested for spatial autocorrelation using the Moran’s I tool in ArcMap, and then the meshblocks containing the highest rates of each variable will be selected for testing to determine if extremes are clustered. For deprivation, smoking rates, Maori/Pacific Islanders and elderly populations, the top 25% of meshblocks will be tested for each variable.

Moran’s I and Z score values will be produced to demonstrate strength of clustering (ArcGIS Pro, 2015). Moran’s I values range from -1 to +1. -1 represents negative spatial autocorrelation, where similar values are not clustered. This is also referred to as a checkerboard pattern (as was demonstrated in Figure 1, Chapter Three). Zero typically indicates no autocorrelation. Values closer to +1 indicate positive autocorrelation, and that similar values are clustered. Z score values indicate whether or not the null hypothesis (the assumption that no clustering exists) can be rejected. If the Z score falls within -1.96 and +1.96, the null hypothesis cannot be rejected. Very high or low z scores indicate very unlikely that the observed pattern is aligned with the null hypothesis, and is likely clustered. P values help to determine the significance of the results. All hypothesis tests ultimately use a p-value to weigh the strength of the evidence. The p-value is a number between 0 and 1 and interpreted in the following way: a small p-value (usually below 0.05) indicates strong evidence against the null hypothesis so it can be rejected. A large p-value (above 0.05) indicates weak evidence against the null hypothesis should be accepted. P-values close to 0.05 are considered marginal (ArcGIS Pro, 2015).

### 2.2 Land Use Regression

Due to the limitations of having only two sites supplying air pollution data, land use regression will be used to provide a distribution of expected air pollution exposures in Christchurch. The urban map will use 3,473 meshblock polygons based on the 2013 Census boundaries (Figure 1) of the Christchurch area. These meshblocks will be generalized to 123 area unit sizes (Figure 6) with the dissolve tool in ArcMap 10.2, which will ascribe them with the averaged or sum values of the meshblocks. Both meshblock and area units were used throughout the spatial calculations that will be described in this Methods Chapter. The Statistics New Zealand Census 2013 provides information on the number of households heated predominantly by wood or coal fires. To avoid population bias, counts will be converted to percentages, and then a decile index, where 1 represents the meshblocks containing fewest coal and wood burners, and 10 represents those with the most. The second other major contributor to air pollution, road traffic, will be provided using traffic counts provided by the Christchurch City Council, featuring daily averages for every public road and street. Using spatial joins in ArcMap 10.2, meshblocks will be assigned the sums of all traffic counts within their boundaries. Again, values will be converted to a decile index, with 10 representing highest rates of traffic.
Two maps will be built from this data to represent summer (November, December, January, February, March) and winter (May, June, July, August, September) seasonality, weighted in accordance with the perceived likelihood that the influence of wood and coal heating is has significantly higher influence in winter than in summer. For winter, home heating and traffic will be multiplied by 0.7 and 0.3 respectively, and added. For summer, these numbers were reversed. The pollution estimations of April and October are expected to be transitional between summer and winter models. Although traffic counts are assumed to be lower in summer due to warmer temperatures encouraging pedestrian and cycle transport, the influence is still considered stronger due to a more drastic reduction in coal and wood combustion.

With only the two monitored sites in Christchurch for particulate matter, it is impossible to validate the variation in air pollution to a level of certainty attained in research that features over 40 sites. Both sites will be used to validate the predicted exposures in the LUR models; however confidence in the produced maps is based primarily on the dominance of home heating and traffic pollution in other land-use regression models. Christchurch does not feature any significant industrial areas, so this particular land-use category was excluded from analysis. Proximity to road networks, a common feature in previous research studies, is deemed to be equally represented by traffic densities at meshblock level.

2.3 Mapping Deprivation

Also using these meshblocks, the Deprivation Index 2013 (Atkinson et al, 2013) will be spatially joined, assigning each polygon with a value. Again, 10 represent the most deprived areas of the city. This has been included as a means of testing if patterns in COPD patient distribution and air pollution are associated with that of deprivation, and whether the theory of environmental justice is deemed an important consideration. The aim of the Deprivation Index is “to identify a small set of indicators of an individual’s deprivation that is appropriate for all ethnic groups and can be combined into a single and simple index of individual socioeconomic deprivation” (Atkinson et al, 2013). The index uses eight YES/NO questions pertaining to employment, clothing, access to food, treating costs and reliance on community organisations.

2.4 Mapping other Socio-Economic Variables

The New Zealand Census (Statistics New Zealand, 2013) also provides the other demographic variables deemed to be associated with COPD. Percentage population above 60 will be used as the means of identifying meshblocks with elderly populations. The percentage of residents identifying as Maori or Pacific Islander at meshblock level will be used, as will the percentages of residents who regularly smoke.

Maps will be produced using meshblocks centroid points of varying sizes to represent the number of hospitalisations experienced within that respective meshblock. Therefore, these points will not represent individual patients. Points will be chosen to differentiate from the choropleth maps, which will be shaded in regards to the percentage of residents within the individual
meshblocks who are over 60, Maori/Pacific Islanders and regular smokers. The Results section (Chapter Five, page 50) will present these maps for visualisation purposes, and then proceed to regression calculations.

2.5 Ordinary Least Squares

The first calculations will use OLS techniques in Excel, with hospitalisation as the dependent variable, and deprivation, smoking, Maori/Pacific Islander and elderly rates as the independent variables. To test the models, winter and summer pollution predictions will be included individually. This method only considers correlation within meshblocks and area units, and does not consider surrounding areas as geographic weighted regression does. The results will produce a number of values which help in the understanding of the model’s strength, namely the R squared, Adjusted R squared, coefficients, Standard Errors and p-values.

The R-squared value is a measure of goodness of fit (Miles, 2014). Its value varies from 0.0 to 1.0, with the higher values demonstrating strength of correlation. It may be interpreted as the proportion of dependent variable variance accounted for by the regression models. The denominator for the R2 computation is the sum of squared dependent variable values. Adding an extra explanatory variable for the model does not alter the dominator, but does alter the numerator; this gives the impression of improvement in model fit that may not be real (ESRI, 2009a).

The adjusted R-squared is a modified version of R-squared that has been adjusted for the number of variable in the model (Miles, 2014). The adjusted R-squared only increases if the new variable improves the model more than would be expected by random. It decreases when a variable strengthens the model, whereas the adjusted R-square will decrease if a variable weakens the model. The adjusted R-square can be negative, but it’s usually not. It is always lower than the regular R-square (ESRI, 2009a).

Regression coefficients represent the mean change in the response variable for one unit of change in the predictor variable while holding other predictors in the model constant. Standard errors represent the average distance that the observed values fall from the regression line. Smaller values are better because it indicates that the observations are closer to the fitted line. The p-value for each variable tests the null hypothesis that the coefficient is equal to zero (no effect). A low p-value (<0.05) indicates that you can reject the null hypothesis. In other words, a predictor that has a low p value is likely to be meaningful addition to the model because changes in the predictor’s value are related to changes in the response variable. Conversely, a larger p-value suggests that changes in the predictor are not associated with changes in the response (Miles, 2014; ESRI, 2009a).
2.6 Hospitalisation Predictions with Ordinary Least Squares

The equation, \( y = a + bx \), was used to test for the predictive power of the variables based on the deviation from the regression line, where \( y \) = predicted or criterion variable, \( x \) = predictor variable, \( a \) = y-intercept — regression constant and \( b \) = slope — regression coefficient. The OLS models will be tested for their predictive powers in two suburbs that demonstrate disparate values. In the equation each variable value in a model output is multiplied by the coefficient for that variable. The resulting numbers and intercept coefficient are added producing a hospitalisation prediction value. If the model’s variables are influential, the resulting hospitalisation prediction values are expected to be higher where variable values are higher, and lower where variable values are lower. The first test will include only socio-economic variables, while the second and third will determine the influence of summer and winter pollution values when those variables are included. Finally, to test individual variable strength artificial scenarios, Suburb 1 and Suburb 2, will be created where all but one variable, the variable being tested, will be assigned different values. This will allow the strength of each variable to be ascertained.

2.7 Geographic Weighted Regression

Geographic Weighted Regression (GWR) extends beyond OLS linear regression models by considering spatial non-stationarity, estimating separate models and local parameter estimates for each geographic feature in the data based on a subset of the data using a differential weighting scheme. GWR functions by shifting a kernel from one feature in a data set to the next, whether that is a point or the centroid of a polygon, until all features have been assigned calculations. As the kernel rests on a sample feature, all surrounding features within the specified distance threshold are included in calculations. A regression model is then fitted to that subset of the data, giving most weight to the values closest features to the centre. Overall coefficients are gained through the averaging of localised individual calculations. Specified user input controls the bandwidth distance or number of neighbours in each local equation. In the GWR model localised calculations can be gained for any feature, which creates a map revealing a continuous and varying surface of values (ESRI, 2009b).

In ArcMap 10.2, Geographic Weighted Regression will be calculated for a range of variables to find potential bivariate and multivariate correlations. Initially the winter and summer ratios of predicted exposure at meshblock level, based on an index built from traffic and home heating densities, will be used as dependent variables. Independent variables will include annual hospitalisation rates, the season rate pertaining to the dependent variable, deprivation indices, and the percentages of the population (from the 2013 Census) who identified as Maori/Pacific, sixty years old or above and those who smoked. The Geographic Weighted Regression will calculate strength of correlation from the surrounding values, using meshblock centroids. All independent variables will be entered and weighted separately. Threshold distances will be set to 250m, 500m and 1000m
bandwidths in order to explore which distance best reflects the environmental risk factors for each location. All these threshold distances have been applied in previous research (Boehmer et al, 2013; Patel et al, 2011 & Olaide et al, 2014).

In addition, deprivation will also be entered as a dependent variable, and compared with smoking rates, Maori/Pacific Islanders, and hospitalisations. Again, separate regressions will be run at run at 250m, 500m and 1000m to find which distance had the most significant influence.

2.8 Geographic Weighted Regression Output Descriptions

Geographic Weighted Regression produces message window diagnostics, which are published in a supplementary table along with summarised information regarding the model variables and parameters. Briefly, for future reference in the Results section of this thesis (Chapter Five, page 50), descriptions of both tabular outputs will be described here.

Residual Squares are the sum of the model’s residual squares. A residual is the difference between observed values and those estimated by the GWR model. Smaller residuals indicate the model is closer to the fitted line (ESRI, 2009b).

The Effective Number value reflects a trade-off between the variance of the fitted values and the bias in the coefficient estimates, and is dependent on the size of the bandwidth. As the bandwidth approaches infinity, the geographical weights for every observation near 1, and coefficient estimates will be very similar to those of a global OLS model. For very large bandwidth sizes, the effective number of coefficients is closer to the actual numbers; local coefficient estimations will have reduced variance but will be relatively biased. Alternatively, if the bandwidth distance is near zero, the geographical weights for all observations also near zero, except for the regression point itself. For extremely small bandwidths, the effective number of coefficient estimates will have a large variance but low bias. The effective number is applied to a variety of diagnostic measures (ESRI, 2009b).

The sigma value is the standard deviation for the residuals and is an estimate of the accuracy of the dependent variable being measured. Smaller sigma values are preferred in statistical analysis (ESRI, 2009b).

The AICc, Akaike information criterion, is a measure of model performance, useful to comparing different regression models. The model receiving the lowest AICc value is considered to provide a better fit to the observed data. AICc is not an absolute measure of goodness of fit, however, but is considered a useful method of comparing models containing different independent variables, as long as the dependent variable remains the same. Comparing the AICc of a GWR model to that of an OLS model is a way of demonstrating the potential benefits of using a weighted technique, and can be used to justify such a decision (ESRI,
Several discharge Accompanying Although Times 3.2 Celsius) COPD PM10 Hourly be entrenched Standards The from air, small ECan’s sizes collected Christchurch’s Environment 3.1 3.

2009b). These measurements will be included in the Chapter Five, the Results, as a means of demonstrating variable influence and model strength.

3. Temporal Analysis

3.1 Air Pollution Data

Environment Canterbury has ten permanent air pollution monitoring sites across the region, three of which are within Christchurch’s urban extent. Only Woolston and St Albans supplied the suitable range of data for this research. A year’s data was collected from 23.08.2014 to 23.08.2015, as means of revealing complete variation in seasonality. All three particulate matter sizes (PM coarse, PM10 and PM2.5) were collected.

ECan’s sites use a TEOM (Tapered Element Oscillating Microbalance) with a FDMS (Filter Dynamic Measurement System). A small pump sucks air through a filter at a constant rate. The weight of the filter is altered by the level of particulate matter in the air, in turning changing the frequency of a small vibrating element in the machine. The amount of particulate matter is calculated from the changed vibration, and the data is sent to ECan via the phone lines.

The data from ECan is measured in micrograms (millionth of a gram) per cubic metre µg/m3. The National Environmental Standards set in 2004 permit one day a year for levels of PM10 to breach 50 micrograms per cubic metre. There are currently no entrenched targets for PM2.5 in New Zealand, but a monitoring guideline was set in 2002 as being 25 µg/m3. No guidelines could be found regarding PM coarse, however the same units of measurements were used by ECan.

Hourly data was downloaded from ECAN’s website in Excel CSV format (ECan Data Catalogue, 2015). Averages of PM coarse, PM10 and PM2.5 were taken at St Albans and Woolston to provide an as accurate as possible indication of air pollution affecting COPD patients across the wider Christchurch area. Accompanying the hourly pollution data are temperature averages (degrees Celsius) and wind speed averages. These are included to test potential meteorology effects that were explored earlier.

3.2 Hospitalisation events

Times of hospitalisation were received from the Canterbury District Health Board from 1 September 2014 to 31 August 2015. Although the times are recorded to minute precision they were assigned to the matching hour in the air pollution spreadsheet. Accompanying data for each event includes a numerical patient ID, ethnicity, gender, age, domicile, admission time and date, discharge time and date, General Practitioner and associated clinic, current smoking status and the transport method to hospital. Several of these details were used to gain generalised descriptions of the study group.
Excel spreadsheets contained the hourly averages of PM Coarse, PM10 and PM2.5, accompanied by hourly wind speed and temperature averages. A further column, mostly empty at an hourly scale, contained the occasional value pertaining to the number of hospitalisations that occurred within the hour. The correlation tool in Excel performed bivariate statistical analysis between two columns of variables. This same analysis was carried out again with daily, weekly and monthly averages for all variables. Graphs were constructed of air pollution and hospitalisations at weekly and monthly scales, presented in the results section. Hourly and daily patterns were deemed too sparse, taking up too much space for a thesis. A summary table of correlation for all temporal scales and variable combinations is included in the results section.

4. Summary

The methods included in this chapter were specifically chosen to answer the aims and objections of this thesis. Pollution estimations will be built from summer and winter LUR models based on fireplace and traffic density. A variety of spatial tools will be engaged to find correlation between hospitalisation and socio-economic variables. OLS will be used initially, before GWR is engaged to test if patterns are significant at localized levels, using bandwidth thresholds of 250m, 500m and 1000m. Finally, regression calculations will be applied to temporal data to seek for correlation of particulate matter levels with hospitalisation rates, temperature and wind speed.
CHAPTER FIVE – Results

1. Introduction

The results chapter aims to answer the questions laid out in the thesis introduction (page 9) using the techniques first described in the Chapter Three and more closely detailed in the Chapter Four. The Results chapter begins with descriptive statistics, providing information pertaining to the 1,420 patients, their gender, range of ages, smoking rates and the number of hospitalisations experienced. In accordance with the Methods section, spatial results will follow, and then temporal results.

To answer the first question, the spatial results first explore spatial autocorrelation of individual variables, revealing that patient points and meshblocks are not clustered as features. However, patient points assigned to high rates of hospitalisation are clustered. More deprived meshblocks are also clustered, as are the meshblocks which contain high rates of smoking and Maori/Pacific communities. The meshblocks with high percentages of elderly people were found to be randomly distributed.

The second question refers to the strength of the correlation between the variables. Choropleth maps were used, containing hospitalisation rates and values relating to the prevalence of deprivation, Maori/Pacific Islanders, regular smoking, and elderly, as well as the summer and winter LUR pollution estimations. Deprivation and air pollution distribution appear the most spatially correlated with hospitalisation when inspecting the produced maps. Moderate visual alignment also occurred with the Maori/Pacific Islander and smoking maps that follow. Meshblocks containing higher percentages of elderly did not appear to be correlated with the hospitalisation. OLS and GWR tests were run on bivariate and multivariate relationships between the variables, confirming deprivation and air pollution distribution as being the most significantly associated with hospitalisation, and the best variables to use for predicting hospitalisation. Hospitalisation was correlated with rates of deprivation, smoking, and Maori/Pacific. GWR results were stronger than those OLS had produced, particularly for area unit bivariate correlation.

The final spatial question refers to the strength of the land use regression model in predicting rates of hospitalisation, assessed qualitatively. The land-use regression results are shown, revealing the summer and winter land use regression maps, and how weighting traffic and home fires in accordance to their seasonal influence affects which areas of the city are deemed to experience higher pollution exposure. Patient points are overlaid, demonstrating that hospitalisation appear to be spatially aligned with predicted high exposure.

The first temporal question sought to determine the influence of meteorology on air pollution at different temporal scales, and was found through correlation calculations in Excel. The results found that air pollution is highest in colder periods, and episodes of lower wind speed. The second question sought to determine strengths of correlation with particulate matter (PM Coarse, PM10 and PM2.5 and hospitalisation rates. The results revealed, that air pollution levels were significantly and strongly correlated with rates of hospitalisation, and were presented in a bivariate table. The second aspect of this question, pertaining to which temporal scale gave the strongest results, found that weekly and monthly averages were the most significant. The final temporal question, relies on qualitative assessment on the reliability of air pollution fluctuations in predicting hospitalisation rates for the health sector and will be answered more thoroughly in the Chapter Six (page 72).
1.2 Descriptive Statistics

Firstly, report some basic descriptive statistics to better illuminate the study population before progressing to the spatial and temporal hospitalisation results as it is important to understand the demographic characteristics of those in this spatial epidemiological study. Notably, due to the under-diagnosis of COPD, and that only hospitalised patients were included, there is likely to be a considerably larger number of COPD sufferers in Christchurch than the 1,420 people in this study. The table below (Table 1) reveals the ethnic demographics of the 1,420 sufferers, of which 1,113 are New Zealand Europeans, 127 are Maori, 27 are Pacific Islanders of various origins, 11 are East Asian and 4 are Middle Eastern. Overall, 777 are female, with an average age of 72, and 642 are male with an average age of 67.
### Ethnicity of Hospitalised Patients

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Count of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand European</td>
<td>1,113</td>
</tr>
<tr>
<td>Maori</td>
<td>127</td>
</tr>
<tr>
<td>Pacific Islanders</td>
<td>27</td>
</tr>
<tr>
<td>East Asian</td>
<td>11</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>1,420</strong></td>
</tr>
</tbody>
</table>

*Table 1: Patient Ethnicity*

### Smoking Prevalence among Hospitalised Patients

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Zealand European</td>
<td>18%</td>
<td>18%</td>
</tr>
<tr>
<td>Maori/Pacific Islanders</td>
<td>29%</td>
<td>49%</td>
</tr>
</tbody>
</table>

*Table 2: Percentage of Europeans and Maori/Pacific Islanders who smoke.*

As seen in Table 2, 18% of both NZ European males and females are currently smokers. 29% and 49% of Maori males and females respectively, are smokers. When exploring the average ages of patients who have suffered a varying number of hospitalisations, some patterns emerge. Those who smoke are generally hospitalised at younger ages. This is especially evident in females (Table 3).

### Average ages and Hospitalisations

<table>
<thead>
<tr>
<th>Number of Hospitalisations</th>
<th>Male average ages</th>
<th>Female average ages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-smoking</td>
<td>Smoker</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>1</td>
<td>67</td>
<td>63</td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>67</td>
</tr>
<tr>
<td>3</td>
<td>71</td>
<td>68</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>68</td>
</tr>
<tr>
<td>5+</td>
<td>62</td>
<td>68</td>
</tr>
</tbody>
</table>

*Table 3: Average ages for male and female patients, according to their smoking status and number of hospitalisations suffered.*
The descriptive statistics in relation to ethnicity reveal that the majority within all ethnic groups have only suffered one hospitalisation over the twelve month period (Table 4). It seems that, relative to the total patient population of each ethnic group, the relationship between ethnicity and number of hospitalisations is uniform. Age and ethnicity are not out rightly associated with increased hospitalisation within the COPD group (Table 4) and therefore the results will move to focus on geographic patterns to ascertain if this has influence over the distribution of hospitalisations.

**Ethnicity and Hospitalisations**

<table>
<thead>
<tr>
<th>Number of Hospitalisations</th>
<th>East Asian</th>
<th>European</th>
<th>Maori</th>
<th>Middle Eastern</th>
<th>Pacific Islanders</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4</td>
<td>588</td>
<td>43</td>
<td>2</td>
<td>20</td>
<td>669</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>130</td>
<td>14</td>
<td>1</td>
<td>5</td>
<td>153</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>44</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>52</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>17</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>12</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>7 - 14</td>
<td>1</td>
<td>10</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>15</td>
</tr>
</tbody>
</table>

*Table 4. The total in each ethnic group who experienced hospitalisations during the twelve month period.*

**1.3 Spatial Autocorrelation – Moran’s I results**

Meshblocks containing COPD patients appear to be randomly distributed across the urban area (Table 5), only clustered in suburbs that are themselves confined, such as Heathcote, Redcliffs and Sumner on Banks Peninsula (Figure 7). Moran’s I results confirm that there does not appear to be spatially autocorrelated features. However, when applying Moran’s I for rate of hospitalisations clustering becomes more evident receiving a Moran’s I value of 0.89.

Moran’s I results also indicate that the meshblocks with the highest rates of deprivation, smoking and Maori/Pacific populations are clustered. All Z-scores indicate it is very unlikely that the observed patterns are random, and the p values for hospitalisation, deprivation, smoking and Maori/Pacific Islander reveal marginal significance.
Moran’s I Results

<table>
<thead>
<tr>
<th>Christchurch Patient Points</th>
<th>Moran’s I</th>
<th>Z score</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Christchurch patient points</td>
<td>0.43</td>
<td>-0.78</td>
<td>0.067</td>
</tr>
<tr>
<td>Points with highest hospitalisation</td>
<td><strong>0.89</strong></td>
<td><strong>4.93</strong></td>
<td><strong>0.045</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Christchurch Meshblock</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Christchurch meshblocks</td>
</tr>
<tr>
<td>Most deprived meshblocks</td>
</tr>
<tr>
<td>Highest smoking rates</td>
</tr>
<tr>
<td>Highest Maori/Pacific population</td>
</tr>
<tr>
<td>Highest elderly population</td>
</tr>
</tbody>
</table>

*Table 5. Moran’s I results. Most significant values are in bold.*

1.4 Land Use Regression Pollution Maps

![Predicted risk of pollution exposure over summer](image1)

![Predicted risk of pollution exposure over winter](image2)

*Figure 8. LUR models reveal predicted seasonal distributions of particulate matter in Christchurch.*

Firstly, the land-use regression (LUR) models reveal the predicted distribution of pollution exposure based on a carefully selected set of variables, including traffic density and home heating, the largest producers of particulate...
matter in Christchurch (Figure 8). Due to the weighting ratios of traffic and home heating (wood and coal fires are deemed rare in summer) the distribution is seasonally variable. Validation with the air pollution sites found that Woolston received higher recorded levels of pollution than St Albans and was assigned higher grades of predicted exposure across all maps.

The summer pollution model (Figure 8) is more influenced by traffic volumes, higher in Linwood and stretching out into the Eastern suburbs of Woolston, Bromley and Aranui. The northern suburb of Belfast, with its proximity to northern arterial routes also has high pollution predictions. Pollution affects Ilam, Fendalton and Merivale, but not to the severity as in the east. Areas with lower road and property densities, rural zones have lower risk of pollution, as do suburban fringes such as Sumner (south east), Hoon Hay (south west) and Redwood (north). The winter pollution (Figure 8), more influenced by wood fires, is predicted to be higher in the southern and eastern suburbs of Woolston, Bromley and Aranui, and again in the northern suburb of Belfast. Rural surroundings received low pollution exposure rates. Most notably of the urban areas, the Western central suburbs of Fendalton and Merivale are predicted to be less exposed to air pollution, particularly in the winter, relative to those surrounding them.

1.5 Hospitalisation and Predicted risk of pollution exposure.

![Summer predicted pollution and hospitalisations](image1)
![Winter predicted pollution and hospitalisations](image2)

*Figure 9. Points representing summer and winter hospitalisations overlaying the LUR maps.*

No COPD patients in this study live in the Central Business District, and of those that inhabit central Western suburbs such as Ilam and Fendalton, they experience fewer hospitalisations. Before regression is undertaken visual descriptions indicate that hospitalisation patterns are similar to those of predicted pollution exposure, particularly for
the winter LUR model. Hospitalisation events are most predominant to the east of the city, especially in the suburbs of Linwood and Aranui. Some hospitalisation points exist in North New Brighton to the far-east and to the southern suburb of Hornby. The summer hospitalisation maps seem to be more distributed across the Christchurch area, without the obvious clustering of winter hospitalisation.

1.6 Deprivation and Hospitalisation

![Figure 10. Hospitalisation points overlaying the Deprivation Index map. 1 represents the least deprived meshblocks, and 10 the most deprived.](image)

The maps of deprivation (Figure 10) in Christchurch show perhaps the most spatially significant patterns of all maps produced. Quantile maps featuring colour ramps representing deprivation reveal that it is highest in the eastern (Bromley, Aranui and Linwood) and south-eastern suburbs (Woolston). There are smaller clusters of deprivation in the northern suburb of Belfast and the western fringes of Hornby. Hill suburbs of Cashmere, Redcliffs and Sumner demonstrate low levels of deprivation, as do Ilam, Fendalton and Merivale to the northwest of the CBD. Overlaid hospitalisation points appear to follow these patterns. Hospitalisation points are predominately clustered around the eastern suburbs, although parts of the north and south-west also contain clustering. Ilam, Fendalton, and Merivale, assigned low deprivation values, contain very few hospitalisations.
1.7 Maori/Pacific Islanders and Hospitalisation

The distribution of Maori and Pacific people (Figures 11) is similar to the deprivation pattern. Higher percentages of Maori and Pacific Islanders are found predominately in eastern (Bromley, Aranui and Linwood) and south-eastern suburbs (Woolston) where population percentages are 30 - 60%. There are high percentages found in the furthest north suburbs too (Figures 13 & 14), to the north of Belfast, as well as to the west, in Hornby. The rate of hospitalisation in Hornby is not as high, however, as the hospitalisation in other areas containing high Maori/Pacific populations.

The central Western suburbs of Ilam, Fendalton and Merivale do not contain considerable percentages of Maori/Pacific peoples (generally 0 - 9%). The hill suburbs also contain lower percentages of Maori and Pacific peoples, low rates of hospitalisation. However, there exist several rural outlying meshblocks that do contain higher percentages of Maori/Pacific Islanders residents, yet no hospitalisations.
1.8 Regular Smokers and Hospitalisations

The percentage of regular smokers (Figure 12) is again similar to the patterns of previous maps, particularly for deprivation, observed most predominately in the east (Bromley, Aranui and Linwood) and south (Woolston) suburbs where more than 30% are classified as regular smokers. The south-western suburb of Hornby, as well as Belfast to the north, also feature high rates of regular smoking. Lyttelton receives a higher percentage of regular smokers, above 20%, deviating from the Port Hills trends that it is more aligned with in other variables.

The residents of the wealthier suburbs, Ilam, Fendalton and Merivale, and Port Hills suburbs of Cashmere and Sumner, generally are far less likely to smoke (0-4%), and hospitalisation rates also follow this pattern. Again the distribution of hospitalisation rates is similar to the deprivation and Maori/Pacific patterns in the east. There are outlying meshblocks in rural areas which contain the highest bracket of regular smoking percentages (30%), yet experienced no hospitalisation.
1.9 Elderly percentages and Hospitalisations

Conversely, the distribution of elderly people (those over 60) does not follow any of the patterns of earlier variables (Figure 13). There are no discernible suburbs where elderly people are the majority, and even those containing relatively high numbers do not appear clustered. This is in alignment with the Moran’s I results (Table 5) which also discovered the layer was randomly distributed. The hospitalisation rates also show no discernible relationship with patterns of elderly percentages.

Unlike other demographic variables, the percentages of elderly people on the Port Hills do not differ substantially from those in the lower city suburbs. Rural boundaries to the city, generally containing fewer counts of smoking, deprivation and Maori/Pacific Islander population, received similar percentages for elderly as did most of the suburbs.
2. Ordinary Least Squares

Area unit models that sought to explain hospitalisation, the dependent variable, with deprivation, smoking, Maori/Pacific Islander and old age, as the independent variables (Table 6). This returned a weak Adjusted R squared value of 0.346.

2.1 OLS Model with Socio-Economic Variables

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple R</td>
<td>0.606926299</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R Square</td>
<td>0.368359533</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R Square</td>
<td>0.346947991</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard Error</td>
<td>6.529520929</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>123.00</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Table 6. Ordinary Least Square results at area unit returned a positive but weak Adjusted R-squared.*

<table>
<thead>
<tr>
<th></th>
<th>Coefficients</th>
<th>Standard Errors</th>
<th>t Stat</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-10.59258067</td>
<td>3.313545021</td>
<td>-3.196751697</td>
<td>0.001783931</td>
</tr>
<tr>
<td>Deprivation</td>
<td>2.138436133</td>
<td>0.506083596</td>
<td>4.225460276</td>
<td>4.72164E-05</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.165925916</td>
<td>0.244513221</td>
<td>-0.67859691</td>
<td>0.498722008</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>0.385614348</td>
<td>0.233943858</td>
<td>1.648320031</td>
<td>0.101947132</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.461737028</td>
<td>0.133461853</td>
<td>3.459692928</td>
<td>0.000753362</td>
</tr>
</tbody>
</table>

*Table 7. Accompanying predictive statistics.*

Coefficient scores (Table 7) reveal that the likelihood of an individual hospitalisation will increase with a climb of 2.1 deciles in the deprivation index, 0.16% in smoking, 0.38% in Maori/Pacific Islander population and 0.46% in old age. Standard errors reveal that the explanatory variable of old age falls closest to the fitted line, and deprivation furthest, and this is repeated for p-values. P-values being above 0.05 suggest that changes in the predictor are not associated with changes in the response. The same model was run again (Table 8, overpage), this time adding summer predicted pollution exposure as an independent variable.
2.2 OLS Model with Socio-Economic Variables and Summer LUR model

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple R</td>
<td>0.617230089</td>
</tr>
<tr>
<td>R Square</td>
<td>0.380972983</td>
</tr>
<tr>
<td>Adjusted R Square</td>
<td>0.348954344</td>
</tr>
<tr>
<td>Standard Error</td>
<td>6.519482985</td>
</tr>
<tr>
<td>Observations</td>
<td>123.00</td>
</tr>
</tbody>
</table>

Table 8. Adding the summer LUR map as an independent variable to the original OLS model.

When including the summer predicted pollution exposure variable (Table 8), the explanatory variables returned an adjusted R2 of 0.348, similar to the original (Table 6). Only the coefficient of deprivation rises significantly (2.6 on the index) with each climb (+1) in pollution exposure, and smoking rises 0.67% (Table 9). Again, standard errors indicate that old age falls closest to the fitted line, and only the p-values of deprivation, smoking and old age represent indicate significance.

<table>
<thead>
<tr>
<th></th>
<th>Coefficients</th>
<th>Standard Errors</th>
<th>t Stat</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-15.06389</td>
<td>4.416079515</td>
<td>-3.411145552</td>
<td>0.000891037</td>
</tr>
<tr>
<td>Deprivation</td>
<td>2.635454891</td>
<td>0.750231434</td>
<td>3.512855864</td>
<td>0.000632811</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.678380738</td>
<td>0.429231913</td>
<td>3.512855864</td>
<td>0.000632811</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>0.366082108</td>
<td>0.237987106</td>
<td>1.538243454</td>
<td>0.126712456</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.392616309</td>
<td>0.141039141</td>
<td>2.783740069</td>
<td>0.006276129</td>
</tr>
<tr>
<td>Summer Pollution</td>
<td>0.64497382</td>
<td>0.671330877</td>
<td>0.215949877</td>
<td>0.829406093</td>
</tr>
</tbody>
</table>

Table 9. Accompanying predictive statistics.

When using the winter predicted pollution exposure variable (Table 10, overpage), the explanatory variables returned an adjusted R² of 0.35, again marginally stronger than the origin. Once again (Table 11), the coefficient of deprivation rises significantly (2.55 on the index) with each climb in pollution exposure rank (+1), smoking rises 0.59% and winter pollution climbs +0.44 on the predicted pollution rank. The standard error scores reveal that the elderly variable is closest to the fitted line, followed by Maori/Pacific Islanders. Only deprivation and elderly variables scored significant p values, while smoking, Maori/Pacific Islanders and winter pollution received p values that suggest that changes in the predictor are not associated with changes in the response.
2.3 OLS Model with Socio-Economic Variables and Winter LUR model

| Multiple R | 0.62091596 |
| R Square | 0.3853663 |
| Adjusted R Square | 0.353754041 |
| Standard Error | 6.495406769 |
| Observations | 123.00 |

Table 10. Adding the winter LUR map as an independent variable to the original OLS model.

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>Standard Errors</th>
<th>t Stat</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-14.18876066</td>
<td>4.406527799</td>
<td>-3.219941257</td>
</tr>
<tr>
<td>Deprivation</td>
<td>2.557497766</td>
<td>0.655068647</td>
<td>3.904167565</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.595146872</td>
<td>0.434526651</td>
<td>-1.369644119</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>0.316227546</td>
<td>0.237375616</td>
<td>1.332182099</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.360139202</td>
<td>0.14456325</td>
<td>2.491186613</td>
</tr>
<tr>
<td>Winter Pollution</td>
<td>0.740259082</td>
<td>0.461893225</td>
<td>0.953162025</td>
</tr>
</tbody>
</table>

Table 11. Accompanying predictive statistics.

2.4 OLS Predicted Hospitalisation Rates

The equation \( y = a + bx \) (described in Chapter Three, page 45) allows for the models’ predictive power to be tested.

**OLS Predictions using Socio-Economic Variables**

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>Variable values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Linwood</td>
</tr>
<tr>
<td>Intercept</td>
<td>-10.59258067</td>
</tr>
<tr>
<td>Deprivation</td>
<td>2.138436133</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.165925916</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>0.385614348</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.461737028</td>
</tr>
</tbody>
</table>

**Predicted Hospitalisations:** 25.66974088 4.895198316

Table 12. Predicted hospitalisations higher in Linwood based on socio-economic variables

The first OLS model (Table 12) contained the four socio-economic variables for Linwood and Fendalton, chosen as they demonstrated disparity. The values for these suburbs were derived by averaging three area units each. Each variable value is multiplied by the coefficient for that variable. The resulting numbers and the intercept coefficient are added producing a prediction of the number of hospitalisations predicted for both suburbs. The first model predicts 25.6 annual hospitalisations in Linwood and 4.89 hospitalisations in Fendalton, indicating these variables are significant in identifying geographic disparity between two locations with different socio-economic variables. According to real data however, sufferers in Linwood had 85 hospitalisations over the 12 month study period,
drastically more than this model predicts. Fendalton patients had 5 hospitalisation events over this time. This suggests that other variables influence high hospitalisation rates, so LUR models were included.

**OLS Predictions using Socio-Economic Variables and Summer LUR model**

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>Variable values</th>
<th>Linwood</th>
<th>Fendalton</th>
<th>Linwood</th>
<th>Fendalton</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deprivation</td>
<td>2.635454891</td>
<td>8.37</td>
<td>1.565</td>
<td>22.05875744</td>
<td>4.124486904</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.678380738</td>
<td>22.8</td>
<td>8.277</td>
<td>15.46708083</td>
<td>5.614957368</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>0.366082108</td>
<td>19.85</td>
<td>4.215</td>
<td>7.266729844</td>
<td>1.543036085</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.392616309</td>
<td>15</td>
<td>19.8</td>
<td>5.889244635</td>
<td>7.773802918</td>
</tr>
<tr>
<td>Summer Pollution</td>
<td>0.54497382</td>
<td>6.9</td>
<td>4.2</td>
<td>3.760319358</td>
<td>2.288890044</td>
</tr>
</tbody>
</table>

**Predicted Hospitalisations**

<table>
<thead>
<tr>
<th>Linwood</th>
<th>Fendalton</th>
</tr>
</thead>
<tbody>
<tr>
<td>39.3782421</td>
<td>6.28128332</td>
</tr>
</tbody>
</table>

*Table 13. Predicted hospitalisations higher in Linwood based on socio-economic variables and Summer LUR model*

When summer pollution values are included in the equation, as they were in the next model (Table 13), the difference between Fendalton and Linwood hospitalisation predictions is even more significant, at 39.3 and 6.2 respectively. According to the real data, Linwood patients suffered 31 hospitalisations over the warmer months, while only one hospitalisation event is recorded in Fendalton. The inclusion of the Summer LUR allows for more accuracy in the prediction of hospitalisation events. This indicates that the summer LUR is a relatively strong factor in determining geographic patterns of hospitalisation.

**OLS Predictions using Socio-Economic Variables and Winter LUR model**

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>Variable values</th>
<th>Linwood</th>
<th>Fendalton</th>
<th>Linwood</th>
<th>Fendalton</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interception</td>
<td>-14.188760</td>
<td>-14.18876066</td>
<td>-14.18876066</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deprivation</td>
<td>2.55749777</td>
<td>8.37</td>
<td>1.565</td>
<td>21.4062563</td>
<td>4.002484004</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.5951468</td>
<td>22.8</td>
<td>8.277</td>
<td>13.56934868</td>
<td>4.92603066</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>0.3162275</td>
<td>19.85</td>
<td>4.215</td>
<td>6.277116788</td>
<td>1.332899106</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.3601392</td>
<td>15</td>
<td>19.8</td>
<td>5.40208803</td>
<td>7.1307562</td>
</tr>
<tr>
<td>Winter Pollution</td>
<td>0.7402590</td>
<td>5.084</td>
<td>3.19</td>
<td>3.763477173</td>
<td>2.361426472</td>
</tr>
</tbody>
</table>

**Predicted Hospitalisations**

<table>
<thead>
<tr>
<th>Linwood</th>
<th>Fendalton</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.22952631</td>
<td>5.564835781</td>
</tr>
</tbody>
</table>

*Table 14. Predicted hospitalisations higher in Linwood based on socio-economic variables and Winter LUR model*

When the winter LUR model is included in the equation (Table 14), the difference is again well pronounced, predicting 35.3 hospitalisations for Linwood and 4.68 hospitalisations for Fendalton. The real data reveals that 43 hospitalisation events occurred in Linwood over the colder months, and 4 occurred in Fendalton. Winter pollution estimates were slightly more accurate in their prediction of hospitalisation events.
2.5 Testing Variables’ Predictive Power

To test which variables are more influential, two fictitious suburbs were created for a series of tests, featuring the same coefficients as the previous models. Socio-economic variables used the intercept and coefficients of Table 12. For summer pollution, Table 13’s intercept coefficient was added, and for winter pollution, Table 14’s intercept coefficient was added. These coefficients were multiplied as they were in previous tests with variable values, only this time these variables were invented. In each test, the suburbs shared identical values for all but one variable. When a variable was being tested, the two suburbs were assigned different values for that variable. The consistent values for each socio-economic variable were as follows: deprivation 5/5, Maori/Pacific 20/20, smoking 15/15, and elderly 15/15. When an individual variable was tested, the disparate values were as follows: deprivation 1/10, Maori/Pacific 15/20, smoking 15/20, elderly 15/20, and winter and summer pollution values were set to 5/10 to test for variable strength. The test results are revealed below (Table 15).

### Testing the Predictive Strength of Individual Variables

<table>
<thead>
<tr>
<th>When all values are consistent</th>
<th>Suburb 1</th>
<th>Suburb 2</th>
<th>Suburb1</th>
<th>Suburb2</th>
</tr>
</thead>
<tbody>
<tr>
<td>except:</td>
<td>Values</td>
<td>Values</td>
<td>Values</td>
<td>Values</td>
</tr>
<tr>
<td>Deprivation</td>
<td>1</td>
<td>10</td>
<td>17.22683</td>
<td>27.91</td>
</tr>
<tr>
<td>Smoking</td>
<td>15%</td>
<td>20%</td>
<td>17.22683</td>
<td>18.05</td>
</tr>
<tr>
<td>Maori/Pacific</td>
<td>15%</td>
<td>20%</td>
<td>15.29</td>
<td>17.22</td>
</tr>
<tr>
<td>Over 60</td>
<td>15%</td>
<td>20%</td>
<td>17.22</td>
<td>19.53</td>
</tr>
<tr>
<td>Summer Pollution</td>
<td>5</td>
<td>10</td>
<td>22.04496</td>
<td>26.9472</td>
</tr>
<tr>
<td>Winter Pollution</td>
<td>5</td>
<td>10</td>
<td>19.99283</td>
<td>26.6516</td>
</tr>
</tbody>
</table>

Table 15. Predicted hospitalisations when all but one variable are consistent.

If all variables are equal except deprivation, a more deprived suburb is predicted to have 27 hospitalisations, considerably higher than the 17 predicted in more affluent areas. Other variables were weaker than deprivation but remained influential on predicted hospitalisation rates. If all variables are the same except percentage who are elderly, the suburb containing more elderly residents will have slightly higher hospitalisation rates. Percentages of regular smoking and Maori/Pacific Islanders also had a slight positive increase on the number of predicted hospitalisations.

Tests were then run to test summer and winter pollution exposure for two fictitious suburbs where all other variables were equal. The suburb with the highest pollution count was predicted to have more hospitalisations. The gap widens slightly with the winter pollution exposure, demonstrating that is more influential than the summer model. Individual variables are not as capable of predicting hospitalisation rates as our previous models.
3. Geographic Weighted Regression

3.1 Bivariate Correlations

The first bivariate correlations test the LUR model’s predicted pollution exposure with socio-economic variables. For winter predicted values (Table 16), weighted regression performed better at lower distance thresholds of 250m and 500m, rather than 1000m, which includes a wider array of variability in its scope. The adjusted R squared correlation at 500m thresholds between winter predicted exposure and hospitalisations, winter hospitalisations, deprivation, Maori/Pacific, age and smoking rates are 0.73, 0.74, 0.33, 0.35, 0.36 and 0.3 respectively. The rates of hospitalisation are more closely tied to the winter’s pollution predictions, with other variables providing positive albeit weak explanatory power.

### Dependent Variable: Winter LUR pollution map at meshblock level.

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>250m</th>
<th>500m</th>
<th>1000m</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deprivation</td>
<td>Adjusted R2</td>
<td>0.39</td>
<td>0.33</td>
</tr>
<tr>
<td>Hospitalisation</td>
<td>Adjusted R2</td>
<td><strong>0.91</strong></td>
<td><strong>0.73</strong></td>
</tr>
<tr>
<td>Maori/Pacific Islander</td>
<td>Adjusted R2</td>
<td><strong>0.51</strong></td>
<td>0.35</td>
</tr>
<tr>
<td>Over 60</td>
<td>Adjusted R2</td>
<td><strong>0.51</strong></td>
<td>0.36</td>
</tr>
<tr>
<td>Smoking</td>
<td>Adjusted R2</td>
<td>0.42</td>
<td>0.33</td>
</tr>
</tbody>
</table>

*Table 16. Bivariate regression results produced some strong Adjusted R2 values, significant highlighted in bold.*

The summer model (Table 17) also produced positive spatial correlation, although the p values are slightly weaker. The same order of independent variables when compared with summer predicted values (replacing the second with summer hospitalisation) returned the following R square results at 500m thresholds: 0.68, 0.30, 0.22, 0.28, 0.32 and 0.27 respectively.

### Dependent Variable: Summer LUR pollution map at meshblock level.

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>250m</th>
<th>500m</th>
<th>1000m</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deprivation</td>
<td>Adjusted R2</td>
<td>0.25</td>
<td>0.22</td>
</tr>
<tr>
<td>Hospitalisation</td>
<td>Adjusted R2</td>
<td><strong>0.90</strong></td>
<td><strong>0.68</strong></td>
</tr>
<tr>
<td>Maori/Pacific Islander</td>
<td>Adjusted R2</td>
<td>0.43</td>
<td>0.28</td>
</tr>
<tr>
<td>Over 60</td>
<td>Adjusted R2</td>
<td>0.49</td>
<td>0.32</td>
</tr>
<tr>
<td>Smoking</td>
<td>Adjusted R2</td>
<td>0.35</td>
<td>0.27</td>
</tr>
</tbody>
</table>

*Table 17. Bivariate regression results produced some strong Adjusted R2 values, significant highlighted in bold.*
Dependent Variable: Deprivation Index at meshblock level.

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>250m</th>
<th>500m</th>
<th>1000m</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjusted R2</td>
<td>0.95</td>
<td>0.86</td>
<td>0.69</td>
</tr>
<tr>
<td>Maori/Pacific Islander</td>
<td>0.83</td>
<td>0.73</td>
<td>0.63</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.77</td>
<td>0.69</td>
<td>0.60</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.21</td>
<td>0.15</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*Table 18. Bivariate regression results produced some strong Adjusted R2 values, highlighted in bold.*

When using deprivation as an independent variable (Table 18), positive correlation was found when compared to the dependent variables of hospitalisation, Maori/Pacific Islanders and smoking as follows 0.86, 0.87, 0.73 and 0.69. Deprivation is a stronger predictor of these variables than pollution models. Elderly percentages are not strongly associated with deprivation. Meshblocks with higher concentrations of hospitalisations (Table 19) are moderately associated with deprivation, Maori/Pacific Islanders, smoking, elderly and predicted pollution exposures.

Dependent Variable: Hospitalisation at meshblock level.

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>250m</th>
<th>500m</th>
<th>1000m</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deprivation</td>
<td>0.53</td>
<td>0.21</td>
<td>0.09</td>
</tr>
<tr>
<td>Maori/Pacific Islander</td>
<td>0.51</td>
<td>0.19</td>
<td>0.09</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.55</td>
<td>0.23</td>
<td>0.08</td>
</tr>
<tr>
<td>Over 60</td>
<td>0.23</td>
<td>0.18</td>
<td>0.07</td>
</tr>
<tr>
<td>Winter Pollution*</td>
<td>0.51</td>
<td>0.18</td>
<td>0.06</td>
</tr>
<tr>
<td>Summer Pollution *</td>
<td>0.67</td>
<td>0.22</td>
<td>0.06</td>
</tr>
</tbody>
</table>

*Table 19. Bivariate regression results produced mildly strong Adjusted R2 values, highlighted in bold.*

*Summer hospitalisations were compared to summer LUR maps, winter hospitalisations were compared to winter LUR maps.*
Bivariate correlations at Area Unit Level

The results of bivariate correlations at area unit level are presented here. Broadly the patterns are similar, with higher deprivation in the east. Only the 1000m bandwidth was suitable, as smaller bandwidths often did not reach beyond the polygon borders of the polygon under calculation. Area units with higher hospitalisation were strongly associated with all independent variables, except elderly (Figure 27).

Dependent Variable: Hospitalisation

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>1000m Bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deprivation</td>
<td>Adjusted R²</td>
</tr>
<tr>
<td>Maori/Pacific Islander</td>
<td>Adjusted R²</td>
</tr>
<tr>
<td>Smoking</td>
<td>Adjusted R²</td>
</tr>
<tr>
<td>Over 60</td>
<td>Adjusted R²</td>
</tr>
<tr>
<td>Winter Pollution</td>
<td>Adjusted R²</td>
</tr>
<tr>
<td>Summer Pollution</td>
<td>Adjusted R²</td>
</tr>
</tbody>
</table>

Table 20. Bivariate regression results for hospitalisation produced strong Adjusted R² values, highlighted in bold.

3.2 Multivariate Geographically Weighted Regression models

While bivariate calculations help to provide descriptive indicators of spatial correlation, the combined strength of the independent variables is best test with multivariate regression. The results of GWR found the model to be stronger when geographic weighting takes place. With hospitalisations acting as dependent variable, and deprivation, smoking and Maori/Pacific Islanders as the independent variables, a 1000m bandwidth returned an Adjusted R² score of 0.64 (Table 23). Following the trend set by bivariate correlation, 500m and 250m bandwidths were stronger again, receiving 0.73 (Table 22) and 0.70 (Table 21), respectively. Lower Sigma and AICc values represent statistically stronger models and re-establish that 500m and 250m bandwidths are the most accurate. When old age was included in these models, the Adjusted R was 0.01, indicating this variable is not geographically significant, as Table 20 suggested.

Dependent Variable: Hospitalisation

<table>
<thead>
<tr>
<th>Bandwidth</th>
<th>250m</th>
<th>Bandwidth</th>
<th>500m</th>
<th>Bandwidth</th>
<th>1000m</th>
</tr>
</thead>
<tbody>
<tr>
<td>Residual Squares</td>
<td>164.645035</td>
<td>Residual Squares</td>
<td>774.59263</td>
<td>Residual Squares</td>
<td>1347.0844</td>
</tr>
<tr>
<td>Sigma</td>
<td>0.460398</td>
<td>Sigma</td>
<td>0.59665</td>
<td>Sigma</td>
<td>0.677197</td>
</tr>
<tr>
<td>AICc</td>
<td>7756.75666</td>
<td>AICc</td>
<td>6869.6565</td>
<td>AICc</td>
<td>7270.369</td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>0.709822</td>
<td>Adjusted R²</td>
<td>0.734135</td>
<td>Adjusted R²</td>
<td>0.648333</td>
</tr>
</tbody>
</table>

Table 21. 250m bandwidth  Table 22. 500m bandwidth  Table 23. 1000m bandwidth
4. Temporal Statistics

There exist a number of significant temporal correlations in the air pollution and hospitalisation data, to answer the first temporal questions. The second is Temporal correlation was most prominent at monthly and weekly averages (Figure 31).

4.1 Bivariate correlations

<table>
<thead>
<tr>
<th></th>
<th>PM10</th>
<th>PM2.5</th>
<th>Windspeed</th>
<th>Temperature</th>
<th>Hospitalisations</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM Coarse</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months</td>
<td>0.204</td>
<td>-0.244</td>
<td>0.315</td>
<td>0.309</td>
<td>-0.051</td>
</tr>
<tr>
<td>Weeks</td>
<td>0.312</td>
<td>-0.138</td>
<td>0.066</td>
<td>0.14</td>
<td>-0.069</td>
</tr>
<tr>
<td>Days</td>
<td><strong>0.666</strong></td>
<td>0.172</td>
<td>0.017</td>
<td>0.038</td>
<td>0.316</td>
</tr>
<tr>
<td>Hours</td>
<td><strong>0.732</strong></td>
<td>0.231</td>
<td>-0.005</td>
<td>0.014</td>
<td>-0.005</td>
</tr>
<tr>
<td>PM10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months</td>
<td>0.961</td>
<td>-0.853</td>
<td>-0.807</td>
<td><strong>0.554</strong></td>
<td></td>
</tr>
<tr>
<td>Weeks</td>
<td>0.89</td>
<td>-0.588</td>
<td>-0.553</td>
<td>0.366</td>
<td></td>
</tr>
<tr>
<td>Days</td>
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<td>-0.154</td>
<td>-0.295</td>
<td>0.223</td>
<td></td>
</tr>
<tr>
<td>Hours</td>
<td><strong>0.819</strong></td>
<td>-0.074</td>
<td>-0.147</td>
<td>0.015</td>
<td></td>
</tr>
<tr>
<td>PM2.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months</td>
<td>-0.905</td>
<td>-0.881</td>
<td>0.554</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weeks</td>
<td>-0.645</td>
<td>-0.662</td>
<td>0.425</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days</td>
<td>-0.216</td>
<td>-0.438</td>
<td>0.157</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours</td>
<td>-0.102</td>
<td>-0.227</td>
<td>0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Windspeed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months</td>
<td><strong>0.901</strong></td>
<td></td>
<td><strong>0.535</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weeks</td>
<td><strong>0.682</strong></td>
<td></td>
<td>-0.391</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days</td>
<td>0.316</td>
<td></td>
<td>-0.135</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours</td>
<td>0.123</td>
<td></td>
<td>-0.017</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months</td>
<td>-0.765</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weeks</td>
<td>-0.623</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days</td>
<td>-0.259</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours</td>
<td>-0.022</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 24. Bivariate correlations in temporal data. Significant correlation values are in bold.
Daily and hourly correlations were weaker, but followed similar trends (Table 24). Particulate matter sizes 10 and 2.5 were significantly correlated, with p values of 0.96, 0.89, 0.83 and 0.81 at monthly, weekly, daily and hourly scales, respectively. PM was significantly negatively correlated with temperature. Following the same order of temporal scale outlined above, PM10 was correlated with temperature at p values of -0.80, -0.55, -0.29 and -0.147, respectively. Similarly, PM2.5 received the following regression results: -0.81, -0.662, -0.438 and -0.22, when correlated with temperature. Of all the hours recorded, the target guidelines were exceeded in 5.61% of them for PM2.5, and in 3.98% for PM10.

Windspeed also proved significantly negatively correlated with PM10 (-0.85, -0.588, -0.154, -0.074) and PM2.5 (-0.95, -0.64, -0.21, -0.102). Therefore, the higher the temperature and wind speed, the lower the level of air pollution. Interestingly, PM coarse values adhered to the same order of significance in regards to temporal scale, but were positively correlated, albeit much more weakly, with temperature and wind speed.

Levels of air pollution at month and week averages (Table 24) were positively and significantly correlated with hospitalisations. PM10 (0.55, 0.36) and PM2.5 (0.55, 0.42). Daily and hourly averages being more sparsely distributed in time were not significant at the p (0.05) level. Temperature was significantly negatively correlated with hospitalisation rates. Adhering to the same temporal scale, the correlation was as follows -0.76, 0.623, -0.25 and -0.022. From this, colder months and week contained heightened rates of hospitalisation. The overall impact of air quality and meteorology on hospitalisation suggests that sustained periods of similar conditions have a larger influence than rapid fluctuations.

4.2 Weekly Bivariate Graphs

Scatterplots are a visual demonstration of the linear correlations that may exist between a series of variables (Figures 14-18). An equation is included which describes how well the data fits with the regression line, expressed as a correlation coefficient, referred to as Adjusted R2. The points represent individual weeks from September 2014 to August 2015.
Figure 15. Adjusted $R^2$ between hospitalisation and PM2.5 was 0.178, indicating relatively weak linear correlation.

Figure 16. Adjusted $R^2$ between hospitalisation and wind speed was 0.10873, indicating relatively weak linear correlation.

Figure 17. Adjusted $R^2$ between hospitalisation and temperature was 0.42288, indicating moderate linear correlation.

Figure 18. Adjusted $R^2$ between hospitalisation and temperature was 0.5162, indicating moderate linear correlation.
Annual Pollution and Hospitalisation Patterns

This graph (Figure 19) reveals the annual fluctuations in PM10, PM25 and hospitalisations. It can be seen from this graph, that the fine particulate matter (PM2.5 and PM10) is understandably well-correlated. Micrograms per cubic metre are revealed on the right axis, and hospitalisations are on the left. It is important to note that the graph begins in September. The initial peak is spring, followed by a reduction in pollution and a steadening in hospitalisation over summer. Approaching autumn the plot lines begin to climb again, peaking in winter for all three variables. The figure contains labels indicating what times of year were contained within LUR summer and winter models, and the two transitionary months of April and October.

5. Conclusion

The results revealed that features in themselves are not spatially autocorrelated, but higher rates of hospitalisation, deprivation, smoking and Maori/Pacific Islanders are clustered. Maps demonstrate that these values appear aligned, vindicated weakly by ordinary least squares and more significantly by geographic weighted regression, particularly at closer bandwidth distances. Areas of the city in contrast to other variables was not correlated with air pollution in both seasonal models. The eastern suburbs have higher rates of deprivation, smoking, Maori/Pacific Islanders and hospitalisation rates. Temporally, air pollution is negatively correlated with fluctuations in temperature and wind speed, and is positively correlated to hospitalisation. All questions can be answered with the produced results.
CHAPTER SIX – Discussion

This research is the first to combine spatial and temporal techniques to explore the relationship between air pollution of COPD hospitalisation. It therefore represents a valuable extension to recent research into the geographic patterns of COPD, particularly in New Zealand where research remains relatively scarce compared to that occurring in the Northern Hemisphere, which constituted most of this thesis’ literature review in earlier chapters. The results indicate that there are significant relationships between air pollution, hospitalisation and the demographic variables of deprivation, Maori/Pacific Islanders and smoking rates. Elderly population distribution was not significantly correlated with hospitalisation and air pollution. The purpose of this chapter is to evaluate the questions relating to the aims and objections of this research. The discussion will ponder the strength of the research in answering these questions, attempting to explain why the results eventuated as they did and what can be improved to ensure future studies continue to shed more light on this growing global disease.

1. Spatial Analysis Questions

1.1 What is the spatial distribution of COPD patients, and explanatory variables, in Christchurch?

While the centroid points representing COPD patients did not receive Moran’s I values that signified spatial autocorrelation, those assigned with the highest hospitalisation were clustered. The maps (Figure 8, page 54) provided visual representation of where this clustering was, predominantly in the Eastern suburbs of Aranui, Bromley and Linwood, and to the South in Woolston. The Moran’s I results for meshblock data also found that deprivation, smoking and Maori/Pacific Islander communities were all clustered. They were predominately in eastern suburbs, although outliers existed for smoking and Maori/Pacific variables in outlying rural suburbs. Meshblocks containing high percentages of elderly residents were not clustered, and were scattered all over Christchurch’s urban area. This reveals that old age is not a significant factor in determining the distribution of COPD patients in Christchurch, and that the elderly are as socio-economically varied as the wider population. Although COPD is a disease that disproportionately affects older demographics, the distribution of elderly residents is likely too random for this pattern to emerge. It must be noted that the patients involved in this study are only the ones who were admitted to hospital within twelve months, and not the total number of COPD sufferers in Christchurch. The literature that claimed deprivation, smoking and Maori/Pacific Islanders (described in Chapter Two) are linked has been vindicated, as their clustered patterns occur in the same eastern and southern suburbs, as well as in Belfast to the south and Hornby in the
far-west. With the observational aspect of analysis indicating association, the following question will discuss whether there was statistical correlation between the variables and the potential reasons why this is so.

1.2 What are the socio-economic and demographic determinants of air pollution exposure? How are predicted air pollution exposures spatially correlated with hospitalisation, deprivation, Maori/Pacific Islanders, smoking rates or old age?

The winter and summer patterns in predicted pollution exposures are strikingly different, but both were correlated, in reference to the first question, to most independent variables. Suburbs with higher populations of Maori/Pacific Islanders, such as Aranui, Bromley and Linwood (refer to Suburb Map in Chapter Five) tend to experience higher rates of smoking and deprivation, a phenomenon well aligned with the findings of Glover et al (2010). The 2013 Census data revealed that the CBD had a lower residential population than surrounding suburbs, and also a lower density of fireplaces. Therefore, the LUR winter model did not assign the CBD high values of predicted pollution. The CBD received higher pollution prediction values relative to surrounding suburbs in summer, when traffic is believed to be the major cause of air pollution. More affluent suburbs, such as Fendalton, Merivale and Ilam, were less polluted than other suburbs in winter. It is postulated that this is the result of fewer fireplaces and the economic security to engage other forms of residential heating such as heat pumps and underfloor heating, as well as heat saving techniques of insulation and double-glazing windows (Canterbury District Health Board, 2012). Poorer areas of Christchurch perhaps rely more on wood fires as an affordable means of heating. Rural areas surrounding Christchurch simply do not have the density of houses, and therefore population, to produce significant amounts of particulate matter or high traffic counts. The geographic correlation between lower deprivation and air pollution in Christchurch further validates the findings of Pearce et al (2006).

Strong correlation between deprivation, Maori/Pacific Islander communities and smoking existed in the census data, affecting the south and east most acutely. It is expected that these patterns will become increasingly polarized as the socio-economic gaps widen and cessation among Maori/Pacific Islanders slows compared to European ethnicities (Blakely, 2006). Rural areas containing high rates of Maori/Pacific Islanders and smoking, but no hospitalisation suggest that the patterns are more geographically dependent on air pollution and deprivation.

Smoking rates among the patients in this study are not significantly different from wider national averages (Action on Smoking and Health, 2013). For New Zealand European males and females the smoking rates were both 18% of the study group, and for Maori/Pacific Islanders the male and female smoking rates were 29% and 49% respectively. National smoking rates for New Zealand Europeans adults are 18%, while it’s 40.4% for Maori adults and 34.7% for
Pacific Islander adults. This indicates that not all patients are not experiencing hospitalisation because they smoke, in fact most do not smoke. Further, the rates of hospitalisation suffered for each ethnicity were not disproportionately biased towards Maori/Pacific Islanders. Instead the proportions were relatively equal for all ethnic groups. Maps revealed that there are communities with significant Maori/Pacific Islander populations where no hospitalisation events occurred. This suggests that there is not necessarily a causal link between smoking and Maori/Pacific Islanders and increased risk of hospitalisation. Instead the most significant factor is a geographic one. Deprived and polluted suburbs are better aligned with hospitalisation events, and also happen to contain higher rates of smoking and Maori/Pacific Islanders.

Old age did not appear to have a large effect on the severity of COPD suffered. The literature established that old age increases the likelihood of developing COPD through accumulated exposure to particulate matter and tobacco smoke over time (Salvi and Barnes, 2009). With an average age of 70 among the patients studied, age is obviously a significant factor in increasing vulnerability to COPD generally. This project only focused on hospitalisations, however, suggesting that old age increases risk of COPD but not necessarily the intensity of exacerbation within that group, as the descriptive statistics revealed (Table 3, page 52). The distribution of elderly in Christchurch was the least clustered variable. Obviously there are elderly people of all ethnic and socio-economic backgrounds so other factors to find those most vulnerable should be considered.

Predictive values for selected suburbs of Linwood and Fendalton found that deprivation was the most significant factor in predicting hospitalisation. Maori/Pacific Islander population had a weaker influence on predicting hospitalisation. Deprivation caused more significant differential in spatial patterns than Maori/Pacific Islanders and smoking. This is considered to be a result of the outlying rural areas with higher Maori/Pacific Islanders and smoking, but very little hospitalisation. Deprivation is the strongest socio-economic predictor as it is most aligned with real hospitalisation rates.

The results indicate that the theory of environmental justice is evident in Christchurch (Jephcote C and Chen H, 2012; Been 1993, 1995, 1997). Although Christchurch is without large-scale industry, the correlation between heavy traffic and deprivation is strong. In the eastern and southern suburbs, built around the main arterial thoroughfares, pollution exposure is expected to be higher, as it is in the northern suburb of Belfast, situated at the source of the high volume northern motorway. Property values, as the literature established (Been, 1993, 1995, 1997), are affected by proximity to main roads. In contrast, less polluted and quieter streets are generally able to command higher prices. These pressures eventually lead to the relatively poor having limited housing choice, and the relatively rich having more
market choice. With socio-spatial clustering, the associated variables are established in similar patterns. Poorer areas contain higher rates of smoking and ethnic minorities more prone to various health issues, aligned with the findings of Glover et al (2010). Conversely, richer areas have the economic security to access better healthcare and education. These confounding factors are best revealed through the geographic distribution of COPD hospitalisation.

This project has focused on COPD patients who have sought medical assistance at the time of exacerbation. Underdiagnosis remains a global concern, and those with reduced access to medical services are expected to be more likely to suffer COPD undetected (Price et al, 2010). Christchurch likely has a much larger population suffering from COPD who remain undiagnosed. Without knowledge of the number, geographic location and condition of many sufferers, presents an immediate research challenge. Without education, assistance and treatment many from deprived backgrounds will suffer exacerbations and place increasing burden on already overloaded emergencies services, particularly over winter months. Deprivation typically limits the social support required in caring and providing accessibility (Brims et al, 2011). Prevention of exacerbation and control of symptoms is possible, and perhaps demonstrated by the equal number of COPD patients in wealthy areas having far fewer hospitalisations. It is assumed that non-diagnosis is more common among the poor than the rich due to international findings, which assert that socio-economics affects a person’s motivation and ability to seek medical help (MacKian, 2003). Considering the average age of COPD patients in this study is 70, it is likely their condition developed when smoking was more common and less associated with deprivation and ethnic minorities. Even if they did not smoke themselves, second-hand smoke exposure was higher historically due to smoking being permitted in public places. If the smoking trends continue in the current direction, communities containing higher percentages of Maori/Pacific Islanders, many of them also containing higher rates of deprivation, will be increasingly predominant in related health issues, although this thesis highlighted that hospitalisation rates. The Canterbury Health District Board records each hospital patient as being either a present smoker or a non-smoker, the majority of which are ex-smokers according to the data

Geographic weighted regression was found to produce more significant results than ordinary least squares by incorporating surrounding values into the calculations. The size of meshblocks may allow for variation in values close proximities. A road or boundary fence may form the border of two meshblocks, so assigning the values of an individual meshblock may not provide an accurate understanding of pollution exposure. Geographic weighting allows for more accurate depiction of potential pollution exposure. The 500m threshold is deemed the most effective for meshblock level as it balances localized accuracy in the variables being tested whilst considering neighbouring meshblocks.
1.3 *To what degree are the LUR pollution models a reliable means of predicting rates of hospitalisation?*

The summer and winter pollution models were tested for their ability to predict hospitalisation. Both models were found to increase differential patterns between Linwood and Fendalton, when included in predictions tests. The models predicted that the difference between Linwood and Fendalton would be more significant in winter. This is understandable when comparing winter and summer LUR maps. Here the difference between affluent western suburbs, such as Fendalton, and more deprived eastern suburbs, such as Linwood, is more significant in winter, when weighted more heavily for household fires. The summer LUR, weighted more heavily by traffic count density, did not differentiate eastern and western suburbs as significantly as the winter model. This is due to traffic density in Fendalton being not considerably disparate to traffic density in the east, relative to that of household fires. Both suburbs contain main arterial routes, and therefore localised pollution emissions. The theory of environmental justice proposes that properties nearer main roads tend to be less valuable (Been 1993, 1995, 1997). The suspected reason as to why traffic density does not necessarily reflect patterns of environmental justice may be due to scale. Finer detail in models may find the influence of traffic density to be stronger, rather than aggregated to meshblock and area unit scales. In these models, density of household fireplaces have influenced geographic disparity more than traffic density, but both were correlated with the socio-economic variables, predominately deprivation, and hospitalisation. Finer scale modeling in future should provide a better understanding of patient exposure to traffic.

Furthermore, while the land-use regression models allow for maps to be constructed indicating pollution exposure, these are primarily speculation and exhibit some limitations. The LUR models do not consider exposure to winds or inversion systems, which enable higher elevations to often sit above the smog (Wilson et al, 2006). Coastal areas, more exposed to wind, are expected to have air pollution dispersed more easily, and the temporal results did reveal that air pollution is lower when the wind is stronger. Therefore, it could be argued that air pollution is likely to be lower in areas where the wind is consistently strong. Christchurch, being a coastal city, likely experiences generally similar wind effects across its flat suburbs, and more complex wind systems on its hills and valley (Kossmann and Sturman, 2004). The LUR models do hold limitations, due to the few assumptions they are built on, but still provide a valid description of where pollution originates.

New fireplace legislation in Christchurch, first mentioned in the air pollution section of Chapter Two, will play an important role in future exposure risks. As the earthquake recovery continues, new and rebuilt houses will be required to seek alternative heat production techniques. This will reduce the amount of particulate matter produced and allow for less reliance on wood and coal among lower socio-economic areas. With the earthquakes most significant damage
situated in the Eastern suburbs, an opportunity for rebuilding and in some cases relocation to new subdivisions, is presented to the relatively poorer communities. This would not have occurred at such a scale without the earthquakes. Deprived households, more affected by COPD, are projected to be less exposed to particulate matter if such restrictions are monitored. The focus for clean air will then move to reducing traffic emissions, by encouraging alternative transport through the construction of walking and cycling infrastructure as well as revised bus routes. The development of new suburbs and wide scale rebuilding projects present a significant opportunity for such advances to be explored and implemented. Trends indicate that among European and Asian populations, smoking rates are decreasing. However, Maori smoking rates have plateaued and require the continued attention of the health sector.

2. Temporal Analysis Questions

2.1 How does weather (wind speed and temperature) affect air pollution levels?

High pollution is correlated with low temperatures and still wind conditions. The reason for this pattern is believed to be increased use of home fires on colder nights, as fireplaces have been found to contribute 80% of Christchurch’s air pollution (Canterbury District Health Board, 2012). Car use is more probable at these times, as colder weather has been found to be a barrier against walking and cycling commuting (Brandenburg et al, 2007), thus increasing particulate matter in the Christchurch area. Higher wind speed was associated with higher temperatures, suggesting that the air produced in warmer conditions is likely to be lower in concentration and more dispersed. Still conditions where inversion layers are more common (Marthews et al, 2011), will typically hold more pollution, triggering acute exacerbation as literature has shown (Dominici et al, 2006). Weather and air pollution data does not provide details of indoor pollution, but it is likely that indoor pollution is higher in colder conditions, when fireplaces are used. Seasonally, there remains a peak in air pollution and COPD hospitalisation over winter months. This is in accordance with a multitude of previous research that found similar patterns (Garcia-Gutierrez et al, 2013; Calderón-Larrañaga et al, 2011).
2.2 To what degree are air pollution levels correlated to rates of hospitalisation? Are these relationships stronger at hourly, daily, weekly or monthly scales?

The rate of hospitalisation appears to follow fluctuations in particulate matter in Christchurch, especially at monthly and weekly scales, indicating correlation. The vast majority of hours contained no hospitalisations, leading to weaker correlation at hourly scales. As temporal scales are larger, correlation was found to be stronger. This indicates that averages over shorter periods feature more fluctuation, where correlation between variables is weaker. Hospitalisation refers to the time when a patient is officially admitted to hospital and visited by medical professionals. The specific environmental conditions linked to the hospitalisation will be a more significant factor in the immediate hours before the triggering of the event, not necessarily when a patient is hospitalised. The time window between calling an ambulance and being treated by health professionals may vary in different scenarios, indicating that hourly scales may not be accurate, and lagging times of varying length may be a factor. More research in this area will allow for more conclusive understanding of the temporal effects on hospitalisation at finer levels of detail.

2.3 Can we predict rates of hospitalisation from fluctuations in air pollution levels?

Unlike previous spatial and temporal questions, the assessment of this research’s ability to predict hospitalisation from air pollution patterns is more qualitative. Firstly, causal relationships between air pollution as an irritant and hospitalisations cannot be concluded, as this research explores geographic correlation not physiological effects. Hospitalisations may be caused by colder temperatures and other seasonal illnesses associated with winter. One particular purpose of the temporal analysis was to assess the applicability of particulate matter levels in predicting hospitalisations. Although it cannot be used at fine temporal detail yet, due to likely variation in lag times between exacerbations and the recorded time of hospitalisation, emergency services can assume increased hospitalisation in the days or weeks when high pollution levels have been recorded. For example, Ko et al (2007) discovered lags of 0-5 days between heightened concentrations of air pollution and hospitalisation.

The results of this thesis are useful in drawing light on the broad temporal patterns of pollution, but there are some limitations to overcome, which would provide health services with more reliable information. Further details on practical implications to health professionals will be made in the Future Research section.
3. Future Research.

There are a number of limitations in this thesis that need to be explained, in order to provide recommendation to research that may follow. The first limitation is applicable to all spatial analysis involving the use of area units, and is referred to as Modifiable Areal Unit Problem (MAUP). It is a challenge occurring when spatial analysis is applied to the same data, but different aggregation schemes affect the results (Nthiwa, 2011). MAUP appears in two ways: scale and zone effects. The scale effect refers to how changing the number of areal units on a map affects the interpretation of geographic patterns. The zone effect occurs with the partitioning of an area within a map, and relates to how the size of areal units generalise spatial data by different amounts (Wong, 2004). The inclusion of area units and meshblocks in this thesis ensures that if MAUP exists in the data, the analysis of both would yield different results. This was observed, although it did not drastically influence which variables were most significant in relation to hospitalisation rates. Meshblocks, being finer in scale, are assumed to be spatially accurate in Christchurch’s urban area. Where potential errors occur with meshblocks may be on urban fringes, where lower population density allows for larger meshblocks. The pollution exposure or deprivation level experienced at an individual household within these larger meshblocks may be more characteristic of a closely neighbouring meshblock, rather than the average of their own meshblock.

![Figure 19. Diagrams to demonstrate how bandwidths of smaller distances may exclude important neighbouring features.](image)

Further, there are also issues of scale when running Geographic Weighted Regression at different bandwidth lengths. Figure 19 demonstrates how distance of bandwidths affect the number of neighbouring meshblocks involved in the calculations. It is expected that the 250m bandwidths often recorded higher regression values due to the kernel only considering individual meshblocks and not having a large enough diameter to reach the centroids of other meshblocks.
1000m bandwidths were, as demonstrated in the diagram, more generalized in the number of meshblocks included in calculations, and this increased variability produced weaker results. All area unit calculations were performed with 1000m bandwidths however, as the 500m bandwidths were suspected to not reach some neighbouring area unit centroids.

Secondly, with only two pollution monitoring sites in Christchurch, the patterns of particulate matter across the city were based on the distribution of predicted pollution emitters, namely traffic density and household fire places. The two sites provided a means of testing the model for both summer and winter, but more monitoring sites in Christchurch would allow a more accurate patterns be recorded and validated with the model. With Christchurch’s air pollution problem, the need for more extensive monitoring extends beyond my thesis to a wide range of environmental and social concerns. The pollution modelling undertaken in this project makes several assumptions that limit the accuracy. Proximity to coast, and the influence of winds, was not fully incorporated. The effect of elevation and topography was also not fully accounted for in this study but, the pollution prediction models are assumed to describe emission locatoin accurately. Hill suburbs have lower traffic and housing densities. They also have higher socio-economic status enabling cleaner heating techniques, as is observed in the LUR models for Merivale and Fendalton. It is assumed that valleys and ridge lines on the Port Hills would experience the most differentiation from the Christchurch average, whilst most of the flat land in Christchurch, where the majority live, likely does not experience drastically different conditions across its surface. Incorporating topography will allow for each household to be assigned a pollution exposure prediction at a more localised level of accuracy. Over time, it is predicted that the census will record a fall in the number of households using wood and coal for heating, as new regulations take effect (Young, 2013). With the time and resources available, this LUR model remains the most suitable means of describing predicted exposure.

Future research will enable the health sector to understand how redevelopment and migration has affected the distribution of deprivation, COPD and air pollution following the earthquakes. With the mass movement of affected people around the city, including those who left and those who have since arrived, Christchurch’s demographics are expected to demonstrate many changes over the next few years. Research must reflect these dynamic patterns, and seek new and robust methods of understanding these changes.

An innovative new direction for COPD research in Christchurch would be to supply a number of COPD patients with inhalers that record the time and location of individual inhaler actualisations using GPS equipment. This would provide an accurate description of the exact temporal and spatial conditions they experienced before each actualisation
of the inhaler, and subsequently if hospitalisations could be predicted from frequent or infrequent inhaler use. This would remove a limitation in this thesis, which currently uses home meshblocks and does not factor in movement over space. Hypothetically, someone who lives in a rural area but commutes to an industrial zone is likely to be exposed to more pollution than predicted, and vice versa. Address points are a general means of assigning a person with a predicted exposure level, and not particularly accurately for most inhabitants. For COPD patients however, using address points is still a robust approach. COPD sufferers are older, and with an average age of 70, it is fairly safe to assume that most are retired. The condition reduces movement, so patients are less active than the general population. Therefore, it is reasonable to assume that COPD patients spend more time at home and have limited mobility. Most journeys from the house would likely be for essentials, and proximity would be an important factor. There remains space for potential study that incorporates temporal and locational data in finer detail.

As mentioned, inhalers also provide temporal data that could offer insight into patient behaviour, as well as environment conditions, in the time leading to an exacerbation. This research cannot access the rate of inhaler use before an exacerbation so any association between environmental factors and hospitalisation can only be formed with the understanding that the rate of mitigation treatment is unknown. It may reflect the air pollution experienced, or have no association at all. Future research in this field will allow for such unknown to be explored. In using hospitalisations, the temporal window leading to exacerbation is slightly unpredictable. The hour of hospitalisation refers to when the patient is officially admitted to hospital and visited by medical professionals. Depending on the number of patients in emergency services at the time, the admission may be some time, potentially a few hours, after the emergency call was made, at a time when the environmental conditions perhaps had a stronger influence on the need to seek medical support. The scarcity of hospitalisations at hourly level resulted in poor correlation, so they were grouped into daily, weekly, and monthly scales, rendering the call-to-admission lag less important, but a loss of detail typical with such generalisation.

As was discovered in the literature (Rossi et al, 2002), the best means of avoiding exacerbation is adhering to prescribed long-term mitigation treatment. For doctors to ascertain the patient’s COPD severity, regular visits are also recommended, particularly over winter months. It is expected that sufferers in more deprived areas are less likely to seek regular medical attention due to having reduced accessibility (Kinnunen et al, 2002). The Christchurch earthquakes have affected the city substantially, particularly in the less affluent eastern suburbs. The exact effect on accessibility to medical centres is unknown, but suspected to be important. Those without cars, or who are unsure of their nearest medical professional, are likely to be more hesitant to make the effort of regular visits. Often mild symptoms are ignored until their search severe levels, an emergency call is then made and a subsequent hospitalisation
follows at high cost. The best method of reducing expenditure is for exacerbations to be avoided through early mitigation. Future research should focus on monitoring environmental and social patterns that determine vulnerability, including spatial patterns of air pollution, ethnicity and deprivation, as well as temporal patterns of air pollution and weather, as these have been shown to be correlated with COPD hospitalisation. Monitoring inhaler use will provide valuable insight into patient adherence, which can then determine if higher use of inhalers is associated with reduced hospitalisations. The number of COPD patients in Christchurch, and the cost of such inhalers, will mean that only a sample can be gained, but it will be a useful measure when compared with techniques in this thesis, to formulate where inhaler use and hospitalisation is highest, and at what times.

The results attained can be answered in every case. Throughout the various methods, correlation has been evident particularly for socio-economic variable of deprivation. Correlations were also found with Maori/Pacific Islander and smoking variables. Although these trends could be assumed from international studies, this thesis is the first to vindicate such patterns in Christchurch. The usefulness of these findings to the health sector is considered valuable, but not conclusive. This discussion has alluded to ways in which monitoring of air pollution and inhaler could be incorporated into future study.
CHAPTER SEVEN – CONCLUSION

COPD is a respiratory condition characterised by airflow obstruction and breathing limitation. Air flow obstruction typically worsens with time, and is not fully reversible. Regular and prompt visits to health services and appropriate treatment can slow degeneration in lung function. Early detection will extend the time in which patients can experience mobile lifestyles. COPD is widespread, and projected to become the third leading cause of death by 2030 (GOLD, 2014), although differences in survey approaches, diagnostic criteria and analytic methods can lead to considerable variation in what is considered COPD. Some estimates place official diagnosis of COPD at only 6%, indicating what is believed to be significant under-diagnosis. COPD is more prevalent among smokers and ex-smokers, the elderly and males, due to occupational exposures (Mehta et al, 2012). The economic burden of COPD is significant in developed nations (Mannino et al, 2003; National Heart, Lung and Blood Institute, 2009), and increases for each patient in relation to worsening symptoms. Reliance of emergency services and oxygen supply is costly, and could be reduced with correct treatment and regular doctors’ visits. Besides smoking, exposure to pollutants is considered a causal factor (Boman et al, 2003). Exposure to pollution is associated with socio-economic deprivation in areas, signalling the importance of geography in COPD studies (McAllister et al, 2013). This project has extended the research into the relationships between air pollution and COPD hospitalisation, and has found results that corroborate previous study findings.

Air pollution is the consequence of fuel combustion by home heating, industry and traffic. The evidence that air pollution has adverse health effects, and leads to increased mortally, is supported by a wide number of epidemiological studies across the world (Tran, 2009; Tanvir and Begum, 2010), although the biological mechanisms continue to be unknown (Pope and Dockery, 2006). Inflammation or airways, in a similar means to cigarette smoking, is the most agreed upon effect of inhaling air pollution. Not many research studies have investigated the causal nature between outdoor air pollutants and objectively defined COPD, and although correlations are regularly found, scientists suggest studies follow patients since birth and compare how exposure affects COPD development (Andersen, 2011).

Indoor pollution is also thought to affect respiration, particularly in residential houses with poor ventilation and open-hearth fires, more common in developing nations (Liu et al, 2001). One way to measure the effect of air pollution and COPD is to monitor the exposure levels and symptom intensity, namely exacerbations.

Geographic Information Systems have aided research into the spatial prevalence and patterns of COPD. A number of international examples provided have attempted to demonstrate the geographic relationship between air pollution and COPD distribution (Andersen, 2011; Schikowski et al, 2005, 2010). Hospitalisations have also been used in
international research; finding high pollution counts and proximity to roads were associated with increased risk. GIS is also important when focussing on all geographically significant data sets, such as deprivation. This information can be presented as maps, visualisation often helping to clearly demonstrate geographic concepts, although caution should be heeded at how aggregation affects spatial accuracy. The basis of these previous research papers forms the foundation of this thesis’ methods.

Land use regression was used to provide a distribution of expected air pollution exposures in Christchurch. The results indicated that meshblocks, and area units, had significant association between high predicted pollution exposure. Hospitalisation was positively associated with those poorer socio-economic variables. Old age, itself a leading risk of COPD was found not to influence the geospatial distribution of COPD severity. Geographic weighted regression performed better than ordinary least squares, acknowledging the spatial relationships are stronger at closer proximities. A more thorough distribution of air pollution monitoring sites would increase the accuracy of pollution prediction maps in Christchurch, by interpolating results or validating future LUR models. Despite these current limitations, broader patterns remain evident, particularly with reference to deprivation and pollution, informing the health sector of where more severe COPD is likely located, and where people may be living with undiagnosed COPD.

Temporally, it was found that hospitalisation events are correlated with increases in PM10 and PM2.5. Hospitalisation events occurred most predominately in days, weeks and months with lower temperatures and wind speeds. Therefore, it can be concluded that COPD hospitalisation is less likely to occur during meteorological periods when air pollution is reduced and dispersed. As air pollution is mostly caused by residential fire places in Christchurch (Canterbury District Health Board, 2012), there is higher pollution recorded over winter, and subsequently hospitalisation.

While this thesis provides evidence that environmental and demographic variables are associated with COPD hospitalisation in Christchurch, future research could take steps to ensure better understanding of patient behaviour. This project used residential property as an indicator of patient location, aggregated to meshblock level, whereas patients in reality will be more mobile. The Discussion, Chapter Six, finished with the recommendation of using monitoring inhalers to better understand temporal patterns of inhaler use before exacerbation events. Additionally, with air pollution monitoring at a finer geographic scale, exposure predictions can be built. These will enable scientists to confirm further the role of air pollution in triggering hospitalisation events. This research is the first to engage spatial and temporal analysis to understand patterns of COPD hospitalization in New Zealand, and has extended a limited global body of research in this area. As COPD becomes an increasing cause of morbidity and mortality worldwide, GIS will provide the best means of understanding the environment influencing this trend.
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