Daily air pollution levels and asthma; exploring the influence of time-activity patterns on personal exposure in Al Jubail Industrial City, Saudi Arabia

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Abstract

Title: Daily air pollution levels and asthma; exploring the influence of time-activity patterns on personal exposure in Al Jubail industrial city, Saudi Arabia

Introduction: Air pollution is a known risk factor for adverse cardio-respiratory health effects. In the last few years, epidemiological studies have provided evidence that exposure to air pollution can aggravate symptoms in asthmatic patients. Some epidemiological studies have used ambient air pollution levels based on fixed-site monitoring (FSM) data to evaluate the short-term effects of ambient air pollution levels on asthma-related emergency department visits (AEDv) using time-series analysis. In the recent past, technology has greatly improved, making it possible to carry out personal monitoring of indoor and outdoor microenvironments (ME). While the existing literature on time-activity patterns (TAP) and ME exposures for populations in the USA and Europe keeps on growing, little research on this topic has been carried out in the Middle East. This study was designed to (i) investigate the statistical association between exposure to air pollution and AEDv, and (ii) identify factors that influence personal exposure in different ME in Jubail Industrial City, Saudi Arabia.

Methods: Daily number of AEDv, air pollution levels (particulate matter ($PM_{2.5}$ and PM_{10}), sulphur dioxide (SO_2), carbon monoxide (CO) nitrogen oxides (NO_x)) and weather variables (temperature and relative humidity) were obtained from the Royal Commission of Al Jubail Industrial City for the period between 2007 and 2011. Data were analysed using a time series approach, which involved application of a generalised linear model (GLM). Relative risks (RRs) were estimated using Poisson regression, while controlling weather variables, day of the week and holiday indicator for lag times of 0 - 7 days. RRs and 95% confidence intervals (CIs) in AEDv were calculated with each increment of inter-quartile range (IQR) change of each pollutant.

Furthermore, to explore the influence of different ME on personal exposure levels, 27 students aged between 16-18 years were recruited and asked to record their detailed movements using a time-activity diary at 15-minute intervals over a period of 24 hours. The students were asked to carry a small backpack containing

a personal air monitor to measure their personal exposure to $PM_{2.5}$, and a GPS device to help identify ME including travelling, outdoors, at school and at home.

Results: The association between AEDv and change in the quantity of SO_2, NO_2, $PM_{2.5}$ and PM_{10} remained positive and statistically significantly after adjustment in the multi-pollutants model. The RR (in percent %) of AEDv increased by 5.4% (95% CI: 2.4, 8.5) at lag 2 for SO_2 , 3.4% (95% CI: 0.8, 6.1) at lag 3 for NO_2, 4.4% (95% CI: 2.4, 6.6) for $PM_{2.5}$ and 2.2% (95% CI: 1.3, 3.2) for PM_{10} at lag 0 per IQR change in pollutants, 2.0ppb, 7.6ppb, 36μg/m^3 and 140μg/m^3 respectively. No significant associations between AEDv and CO were found.

The time activity diary revealed that most of the students' time was spent indoors (88.6%). The FSM median (IQR) $PM_{2.5}$ level, 51.0μg/m^3 (34.0-74.2), was significantly higher than personal median $PM_{2.5}$ level, which was 30.0μg/m^3 (20.9-42.4) (Wilcoxon p-value<0.001). Total personal outdoor median concentration of $PM_{2.5,}$ 44.4μg/m^3 (31.1-59.5), was significantly higher than total personal indoor concentration, which was 28.3μg/m^3 (19.2-40.2) (p-value<0.001). There was a significant but weak correlation between FSM and personal monitor $PM_{2.5}$ levels when indoors (Spearman's rank correlation=0.228, p-value < 0.001 n=544), but not at outdoors microenvironment.

Conclusion: Current levels of ambient air pollution were associated with AEDv in Al Jubail. While there appear to be similarities between TAP in this small population sample from the Middle East and Europe/USA, the exposure levels in this industrial city appear to be very high, compared to the WHO air quality guidelines. The validity of FSM data as a proxy for personal exposure to PM needs to be characterised so that the exposure error associated with this proxy measure is better understood.

Dedication

This piece of work is dedicated to:

My parents

My wife and my son

My sisters & brothers

With my sincere thanks for your love, patience and support.

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It has always been my dream to pursue a PhD degree. My dream came true the day I became a PhD student at Newcastle University. It is difficult to express my appreciation for the people who have supported me throughout my time at the university. This work could not have been completed without the help and support of several people.

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Statement of contribution

This is to declare that the work contained in this thesis comprises original work conducted by the student under the supervision of Dr Richard McNally, Dr Anil Namdeo and Dr Susan Hodgson.

This thesis has not been submitted for the award of any other degree at any other institution.

Conference Awards and Publications

Conference Awards:

1. **Best Poster Award from the Applied Epidemiology Research Day**, for the work title “Association between daily air pollution levels and asthma emergency department visits in Al Jubail Industrial City, Saudi Arabia” October 2014, Newcastle – UK

2. **Best Poster Award from the 7th UK & Ireland Occupational & Environmental Epidemiology Meeting**, for the work titled “Exploring the Influence of Time-Activity Patterns on Personal Exposure to $PM^{2.5}$ in different microenvironments” March 2013, Edinburgh – UK

3. **Best Poster Award from the 6th Saudi Scientific International Conference**, for the work titled “Exploring the Influence of Time-Activity Patterns on Personal Exposure to PM2.5 in different microenvironments” Oct 2012 at the Brunel University, London – UK

Conference Publications:

1. Salem AlBalawi, Dr. Susan Hodgson, Dr. Anil Namdeo & Dr. Richard McNally. **Association between daily air pollution levels and asthma emergency department visits in Al Jubail Industrial City, Saudi Arabia.** The 26th Annual International Society for Environmental Epidemiology, August 2014, Seattle, USA

2. Salem AlBalawi, Dr. Susan Hodgson, Dr. Anil Namdeo & Prof. Tanja Pless-Mulloli. **Personal versus fixed-site monitoring for assessing $PM^{2.5}$ exposure in an industrial city, Saudi Arabia.** Environment and Health – Bridging South, North, East and West, N: 5212, P-3-04-17, August 2013, Basel, Switzerland.

3. Salem AlBalawi, Dr. Susan Hodgson, Dr. Anil Namdeo & Prof. Tanja Pless-Mulloli. **Exploring the Influence of Time-Activity Patterns on Personal Exposure to $PM^{2.5}$ in different microenvironments.** The 7th UK & Ireland Occupational & Environmental Epidemiology, March 2013, Edinburgh, UK.

4. Salem AlBalawi, Prof. Tanja Pless-Mulloli, Dr. Susan Hodgson & Dr. Anil Namdeo. **Air Pollution and Health; Exploring the Influence of Time-Activity Patterns on Exposure in Al Jubail Industrial City, Saudi Arabia.** (SIC05) Abs.no. 169, Page:57 ISBN 978-0-9569045-0-8. June 2011 Coventry, UK.

My poster presentations are shown in Appendix A**,** Conferences Publications.

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List of Abbreviations

Term	Definition
AEDv	Asthma-related Emergency Department Visits
AIC	Akaike Information Criterion
APHEA	Air pollution and Health: a European Approach
AQGs	Air Quality Guidelines
AQGs	Air Quality Guidelines
BC	Black Carbon
CAA	Clean Air Act
CI	Confidence Interval
CO	Carbon Monoxide
Df	Degrees of Freedom
Dos	Day of Study
dow	Day of The Week
EA	Emergency Admission
ED	Emergency Department
EDVs	Emergency Department Visits
EHA	Emergency Hospital Admission
EU	European Union
EXPOLIS	Air Pollution Exposure Distributions within Adult Urban Populations in six European cities
GAM	Generalised Additive Model
GAM	Generalized Additive Models
GLM	Generalised Linear Model
GLM	Generalized Linear Model

GPS	Global Positioning System
H	Holiday Indicator
HA	Hospital Admission
ICD	International Classification for Diseases
IQR	Inter-Quartile Range
ISAAC	International Study of Asthma and Allergies in Childhood
NA	Non-Available
NAAQS	National Ambient Air Quality Standards
NMMAPS	National Mortality and Morbidity Air Pollution Studies
NO_2	Nitrogen Dioxide
Ns	Natural Cubic Spline
O_3	Ozone
OR	Odds Ratio
PACF	Partial Autocorrelation Functions
PAH	Polycyclic Aromatic Hydrocarbons
Pb	Lead
PM	Particulate Matter
$PM_{0.1}$	Particulate Matter with diameter of 0.1 micrometres or less
PM_{10}	Particulate Matter with diameter of 10 micrometres or less
$PM_{2.5}$	Particulate Matter with diameter of 2.5 micrometres or less
PR	Poisson Regression
R^2	Pseudo-Squared
RCEP	United Kingdom Royal Commission on Environmental Pollution
RCER	Royal Commission Environmental Regulations in Al Jubail

RH	Relative Humidity
RR	Relative Risk
SO_2	Sulphur Dioxide
T	Temperature
TSA	Time-Series Analysis
TSP	Total Suspended Particles
USEPA	United States Environmental Protection Agency
VOCs	Volatile Organic Compounds
WD	Wind Direction
WHO	Word Health Organization
WS	Wind Speed
X^2	Chi-Squared
$\mu g/m^3$	Microgram per cubic meter
µm	Micrometres

CHAPTER ONE

Introduction

Chapter:1 Introduction

1.1 Rational for the study

The association between air pollution exposure and the risks to human health has been a public health concern for over the past decades. This has mainly been due to the three severe air pollution episodes: in the Meuse Valley in 1930 (Firket, 1936), in Donora, Pennsylvania in 1948 (Ciocco and Thompson, 1961) and during the London smog of December 1952 (Ministry of Public Health (1954)). These episodes showed associations between the levels of air pollutants and mortality caused by a combination of industrial pollution sources and adverse weather conditions (Voelkel and MacNee, 2008). Despite this early recognition of the dangers of air pollution and continuous improvements in air quality in large parts of the world over the past decades, poor air quality remains a challenge in many urban areas, particularly in emerging and developing countries (Michelle *et al.*, 2004).

According to current research of the World Health Organization (WHO), air pollution exposure contributes to one in eight (12.5%) of total global deaths (WHO, 2015). The WHO reported that about 7 million people died as a result of air pollution exposure in 2012 (WHO, 2015). WHO estimated that there were about 4.3 million deaths caused by indoor air pollution and about 3.7 million deaths from outdoor air pollution in 2012, of which nearly 90% were in developing countries (WHO, 2015). This finding more than doubles previous estimates and confirms that air pollution, both indoors and outdoors, is the largest single environmental risk to health affecting everyone in various parts of the world (WHO, 2013; WHO, 2015).

Asthma is becoming the most common long-term respiratory disease among children in the UK (Asthma-UK, 2015). The International Study of Asthma and Allergies in Childhood (ISAAC) undertook its latest survey between 1999 and 2004. ISAAC found that about 14% of the world's children were likely to have had asthmatic symptoms in the last year and, crucially, the prevalence of childhood asthma varies widely between countries, and between centres within countries studied (Network, 2014). In addition, studies by ISAAC found that between 1999 and 2004, the prevalence of asthma in children aged 6–7 years and 13–14 years increased from less than 5% to over 20% in European samples (WHO, 2007).

Despite its high prevalence, the cause of childhood asthma remains unclear. Epidemiological studies have suggested that there are multiple genetic and environmental risk factors for asthma and interactions between genes and the environment are likely to be important (see Chapter 2). The environmental factor on which this study will focus is exposure to air pollution.

The new research by WHO (2015) reveals a stronger link between both indoor and outdoor air pollution exposure and respiratory and cardiovascular diseases (WHO, 2015), including evidence that exposure to air pollution can aggravate symptoms in asthmatic patients. Some of these studies have evaluated the short-term effects of particulate matter on asthma attacks and emergency department visits (WHO, 2014a). The asthma exacerbation is most likely due to airway inflammation and hyper-responsiveness (WHO, 2014a). Recently, emphasis has been placed on the need for research regarding disease exacerbation associated with acute exposure to air pollutants (HEI, 2010).

Epidemiological studies of air pollution fall into four types: time series; case crossover; panel and cohort studies. The time series, case-crossover and panel studies are more appropriate for acute effects estimation while the cohort studies are used for acute and chronic effects combined (Dominici *et al.*, 2003a; Peng and Dominici, 2008; Tadano *et al.*, 2012). Based on early epidemiologic evidence, the impact of air pollution on public health was observed mainly from studies of extreme episodes, when high air pollution levels over several days were accompanied by noticeably large increases in mortality and morbidity (Michelle *et al.*, 2004). As such high levels became less frequent, more formal time-series analysis was recognized as one of the most important tools for studying the health effects of air pollution (Michelle *et al.*, 2004; Chen and Kan, 2008).

The time-series approach assesses the effects of short-term changes in air pollution on acute health events by estimating associations between day-to-day variations in both air pollution levels and in mortality and morbidity counts (Michelle *et al.*, 2004; Chen and Kan, 2008; Peng and Dominici, 2008). Thus, the data for daily time-series analysis include daily measures of the number of health events (e.g., daily asthma emergency department visits), concentrations of air pollutants (e.g., carbon monoxide (CO); nitrogen dioxide (NO_2); particulate matter

(PM_{10} and $PM_{2.5}$), and; sulphur dioxide (SO_2)), and weather variables (e.g., daily temperature and relative humidity) within the same population in a geographically defined area. This approach is a type of ecologic study because of the focus on population-averaged health outcomes and exposure levels. Since observations are made within the same population, the influence of many confounding factors can thus be avoided (Chen and Kan, 2008). For example, the age distribution and smoking history are not likely confounders for time-series studies on the expected number of deaths or morbidity on any given day, since they do not vary on short-term timescales (e.g., day to day) and are not associated with air pollution levels (Schwartz *et al.*, 1993; Michelle *et al.*, 2004; Tadano *et al.*, 2012).

Regression models are generally the method of choice in time-series studies to estimate the change in risk for a health outcome, such as mortality or morbidity counts, associated with a unit change in ambient air pollution levels on a short-term basis (Dominici *et al.*, 2003a; Michelle *et al.*, 2004; Tadano *et al.*, 2012). The concentration of air pollutants is included in the model lagged for zero days (i.e. current day) to a few days (multiple lag), which describes the change in the relative risk in a multi-day period after a given day's increase in air pollution (Michelle *et al.*, 2004; Peng and Dominici, 2008). In particular, it might be reasonable to assume that at the population level, an increase in air pollutants (e.g., particulate matter) on a given day leads to an increase in morbidity (e.g., daily asthma emergency department visits) which is distributed smoothly over multiple days into the future (Michelle *et al.*, 2004; Peng and Dominici, 2008).

Most epidemiological studies of the health effects of air pollution have relied on concentrations measured at fixed-site monitors as an estimate population exposure to ambient air pollution (Michelle *et al.*, 2004; Oezkaynak *et al.*, 2013). This is because it is difficult to accurately estimate exposures for individual study participants, particularly within the limits set by feasibility, participant burden, and cost (Zeger *et al.*, 2000). Accordingly, misclassification of exposure is a well-recognized inherent limitation of these studies (Blair *et al.*, 2007; White *et al.*, 2008). The consequences of exposure misclassification varies with the study design and is dependent on the spatial and temporal aspects of the design, as well as the aims of each study (Rom and Markowitz, 2007; Oezkaynak *et al.*, 2013). In addition, the degree to which refined exposure estimates (e.g. including

location of individuals) influence predictions of health outcomes (e.g., long-term vs short-term or acute vs chronic exposure effects), depends on study-specific characteristics including epidemiological study design (e.g., time-series vs cohort). In time-series studies, only population-level exposure estimates are needed, as the focus is entirely on quantifying a temporal effect. Whereas in individual-level studies, such as cohort studies, exposure estimates for individual are preferable (Oezkaynak *et al.*, 2013).

Exposure misclassification from using fixed site monitor data as a proxy for personal exposure is due, in part, to the fact that people spend a large proportion of their time indoors, where air quality may be very different from that measured outdoors, by the fixed site monitors. The WHO (2006a) report concluded that the association between personal and outdoor concentrations could actually be stronger in relatively polluted locations, since high outdoor levels may obscure the influence of indoor sources and increase the relative association of outdoor particles to personal exposure. In addition, time-activity patterns can also significantly influence exposure, regardless of outdoor concentrations (WHO, 2006a). In the recent past, technology has greatly improved, making it possible to conduct detailed personal monitoring in both indoor and outdoor environments. This has allowed more precise estimation of personal exposure, which is important since people spend a large part of their time in indoor environments. While there is a growing literature on time-activity patterns and microenvironments exposures for populations in North America and Western Europe, little research on this topic has been carried out in the Middle East. There remains a need for studies in cities of developing countries, where levels of air pollution and meteorological conditions are different from North America and Western Europe (Gouveia and Fletcher, 2000).

This study will investigate the association between exposure to air pollution and asthma-related hospital visits, and identify factors that influence personal exposure in Al Jubail Industrial City. To the best of my knowledge, no study of this kind has been conducted in an industrial city in Saudi Arabia. Such a study will fill important gaps in our understanding of the influence of time-activity patterns and microenvironments on personal exposure in this setting.

1.2 Overall aim

The aim of this study is to: investigate the association between air pollution and asthma-related hospital visits in Al Jubail Industrial City, identify factors that influence personal exposure, and assess how the relationship between air pollution and asthma-related hospital visits varies when using different types of exposure estimates.

1.3 Hypotheses

This study was designed to investigate two main hypotheses:

1. Short-term exposure to temporally variable ambient air pollution is associated with asthma-related emergency department visits (AEDv) in Al Jubail industrial City.
2. Individual time-activity patterns and exposure to specific microenvironments will better explain personal air pollution exposure than data from local fixed-site ambient air pollution monitors.

1.4 Objectives of the Study

A: Literature review

I. Daily air pollution and asthma-related emergency department visits

II. Time-activity patterns/microenvironments and drivers of personal exposure

B: Research phases

Phase one: Investigate the statistical association between daily air pollution levels and AEDv by using time-series analysis.

Phase two: Collection and analysis of microenvironment and time-activity pattern data. This phase has two sub-objectives;

I. To identify factors that influence personal exposure to air pollution.

II. To estimate exposure error that is introduced by using fixed-site monitoring stations as a proxy for personal exposure.

Phase three: Investigate whether the associations observed from time-series analysis of AEDv differ when correcting ambient $PM_{2.5}$ levels using data from the personal monitoring campaign.

CHAPTER TWO

Scientific Background

Chapter:2 Scientific Background

2.1 Introduction

In this chapter, two main topics are discussed. The first section describes the relationship between air pollution and health effects, and provides definitions of the relevant terms. It also includes a literature review of asthma prevalence in the Middle East and in European countries. In this section, existing and on-going studies of time-series analysis that were used to study the relationship between air pollution levels and asthma-related emergency department visits are discussed. The second section is a general discussion of human exposure to air pollution. Then there is a focus on microenvironments and time-activity patterns, and their influence on personal exposure. In addition, I discussed the relationship between personal exposure and indoor as well as outdoor concentrations.

2.2 Literature search methods

The first objective of this study was to carry out a literature review of existing research in the following relevant fields. The first is the field of air pollution exposure and asthma-related emergency department visits, focusing on literature published since the WHO review, which included studies pre 2003. The second is the field of time-activity patterns/microenvironments and drivers of personal exposure. To achieve this, relevant literature was identified via a search of Ovid, Scopus, Medline, Pub med, Web of knowledge and Google Scholar database. The literature was searched using various search strategies, as shown in Table 2.1. The following main terms were used: air pollution, asthma, hospital admission, emergency department visits, time-activity pattern and microenvironment as shown in Table 2.2. The search was limited to publications in English for the years 1977 to 2015. The research evidence is summarised and critiqued in the following sections.

Table 2.1: Sources of information for the literature review

Search Strategy	Source
Database Search	Ovid, Scopus, Medline, Pub med, Web of knowledge and Google Scholar
Cross-Referencing	Checking other reviews, reference lists and hand searching
Journal Hand Search	Exposure Science and Environmental Epidemiology, Environmental Health Perspectives, Epidemiology, Environmental Health, The New England Journal of Medicine.
Search within Publishers	Elsevier (Science Direct),Wiley Inter Science, BMJ, Blackwell
Search within Organizations	WHO, DOH, EPA, ISAAC, EMRO, COMEAP and NMMAPS
Conference Proceedings	The International Society of Exposure Science (ISES) and The International Society for Environmental Epidemiology (ISEE)
SDI/Auto Alert service	Elsevier, Ovid, Scopus, Web of knowledge and Google Scholar

Table 2.2: Key words used for the selection of studies

Key words	Number of hits
(air pollutants + asthma)	3,301
(indoor air pollution + asthma)	1,866
(ambient air pollution + asthma)	855
(outdoor air pollution + asthma)	495
(indoor air pollution + asthma exacerbation)	148
(ambient air pollution + asthma exacerbation)	124
(ambient air pollution + asthma + hospital admission)	97
(air pollution + asthma + emergency department visits)	75
(ambient air pollution + asthma + emergency department visits)	22
(outdoor air pollution + asthma +emergency department visits)	14
(air pollution + asthma + hospital admission)	305
(outdoor air pollution + asthma exacerbation)	59
(outdoor air pollution + asthma + hospital admission)	41
(air pollution + schoolchildren)	348
(air pollution + schoolchildren + asthma)	146
(time-activity patterns + air pollution exposure)	100
(time activity + schoolchildren + asthma)	8
(time-activity + microenvironment + air pollution exposure)	43
(time-activity + microenvironment + air pollution)	44
(time-activity + microenvironment)	53
(time-activity + microenvironment)	52
(time-activity patterns + microenvironment)	31
(microenvironment + schoolchildren + asthma)	1
(air pollution + GPS)	110
(time-activity + GPS)	9

2.3 Air Pollution and Health Definitions

The term *ambient air* is generally understood to mean “the air to which general public has access...” (The United States Environmental Protection Agency (USEPA, 2010b). The word *pollution* is defined by the UK Royal Commission on Environmental Pollution (RCEP) as “the introduction by man into the environment of substances or energy liable to cause hazard to human health, harm to living resources and ecological systems, damage to structure or amenity or interference with legitimate use of the environment”(RCEP, 1984). This is a general definition which covers numerous types of environmental pollution including water, air and soil pollution.

The USEPA has defined the term *air pollution* as “the presence of contaminants or pollutant substances in the air that interfere with human health or welfare, or produce other harmful environmental effects” (Vallero, 2008). The World Health Organization (WHO) has defined *air pollution* as “contamination of the indoor or outdoor environment by any chemical, physical or biological agent that modifies the natural characteristics of the atmosphere” (WHO, 2015). These definitions encompass the possible effects of air pollution on both human health and/or the environment. According to WHO, the term *Health* is defined as “a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity” (WHO, 1948). This definition has not been changed since 1948 (WHO, 1948).

2.4 Classifications and Sources of Air Pollution

Air pollutants can be classified as either primary or secondary; or as gaseous or particulate (WHO, 2006a). Pollutants may be released into the atmosphere (primary air pollutants) or formed within the atmosphere itself (secondary air pollutants) (WHO, 2006a; USEPA, 2010a). Primary air pollutants are those that are released into the atmosphere directly from the source of the pollutant and retain the same chemical form. The ash produced by the burning of solid waste is an example of a primary air pollutants (WHO, 2006a; USEPA, 2010a). Secondary air pollutants are those that are formed within the atmosphere itself by chemical reactions of precursor or primary emissions, such as ozone (O_3), which is created from organic vapours released at a petrol station, for example (WHO, 2006a; USEPA, 2010a).

Gaseous and particulate classifications relate to the physical forms of pollutant (USEPA, 2010a). Gaseous air pollutants are those present as gases or vapours which can be readily taken into human lung (USEPA, 2010a). These gaseous pollutants include substances such as carbon monoxide (CO), nitrogen dioxide (NO_2), sulphur dioxide (SO_2) and volatile organic compounds (VOCs) (USEPA, 2010a).

Particulate air pollutants contain material matter in liquid or solid particles, such as pollen, dust and smoke, and cover a range of different sizes (WHO, 2006a). Overall, the smaller the size of the particle, the stronger its likely effects on public health. This is because small particles can be more easily inhaled. *Particulate matter* (PM) can be categorized in sizes depending on their predicted penetration into the lung. *Coarse particles* (PM_{10}) are inhalable particles with diameter of 10 micrometres (μm) or less. *Fine particles* ($PM_{2.5}$) are inhalable particles with diameter of 2.5μm. *Ultrafine particles* ($PM_{0.1}$) are a subset of inhalable $PM_{2.5}$ and particles less than 0.1μm in diameter (USEPA, 2010a).

Any activity that causes pollutants to be emitted into the atmosphere can be referred to as a source of air pollution. These activities can result from natural sources (biogenic sources) such as volcanoes, which can emit gases and particulate matter into air (USEPA, 2010a), or human-generated sources (anthropogenic sources), which are further categorized as mobile or stationary sources (USEPA, 2010a). Mobile sources comprise a wide range of

transportation modes such as road vehicles, trains, ships etc. (USEPA, 2010a). On the other hand, stationary sources, also known as fixed-site sources, include industrial and household emissions. (WHO, 2006a).

2.5 Air Pollution Standards and Guidelines

The quality of air is considered to be an essential ingredient for all living organisms and is important for public health and well-being (WHO, 2006c). Improving air quality will enhance the quality and longevity of life (Walters, 2010) and will have important impacts on economic development (Autrup, 2010). Air quality standards dictate the acceptable levels of air pollutants that are not be exceeded in a given time and area (WHO, 2006a; USEPA, 2010a). The term standard means “a set of laws or regulations that limit allowable emissions [...] of air quality beyond a certain limit” while the term guidelines means “a set of recommended levels against which to compare air quality from one region to another over time” (Yassi et al., 2001).

In the last few decades, many organizations have developed different standards and guidelines for the control of air quality. In the United States of America (USA), after the Clean Air Act (CAA) milestone 1970, the USEPA established National Ambient Air Quality Standards (NAAQS) that set standards for six principal air pollutants: NO_2, SO_2, O_3, CO, PM and Lead (Condliffe and Morgan, 2009). In Europe, the European Union (EU) developed a series of framework directives for controlling ambient air pollution, and identified twelve air pollutants for regulation (NO_2, SO_2, CO, $PM_{2.5}$, PM_{10}, O_3, Lead, Benzene, Arsenic, Cadmium, Nickel, and Polycyclic Aromatic Hydrocarbons (PAH)) (Abhishek Tiwary, 2010). The WHO has designed Air Quality Guidelines (AQGs), based on results from expert evaluation of recent scientific literature, to protect the large majority of individuals from the health impact of air pollution (WHO, 2006c).

The current study was conducted in Al Jubail Industrial City in Saudi Arabia, for which the Royal Commission Environmental Regulations (RCER) is the sole governmental and independent body having a wide array of responsibilities within Al Jubail Industrial City, including regulating environment, community, industrial and other related services. The RCER is also responsible for controlling pollution associated with development and operation of the industrial city. The Royal Commission has, thus, developed and adopted regulations, standards and guidelines to control all types of substances emitted, discharged, or deposited, and noise generated within Al Jubail Industrial City (RCER, 2010).

Many organizations have independently developed standards and guidelines based on different pollutants and concentrations as shown in Table 2.3. In Al Jubail Industrial City, the RCER-recommended ambient air quality standards are applicable to protect the environment of the industrial city from adverse impacts (RCER, 2010). The WHO, EU and USEPA are considered to be the most influential groups. EU standards have to be met by all the Member States, while WHO guidelines have no statutory basis, but may be used by authorities that do not have national standards (Abhishek Tiwary, 2010). Furthermore, the standard-setting procedure can be determined by the feasibility and costs of enforcing and applying the standards. This may lead to a standard above or below the respective recommended guideline value, as can be observed from the air quality standards of countries around the world included in Table 2.3 (WHO, 2006a).

Table 2.3: Selected national and regional air quality standards and guidelines

Pollutants	Averaging Period	RCER Jubail[1]	WHO[2]	USEPA[3]	EU[4]	China[5]	India[5]	Mexico[5]
PM_{10} $\mu g/m^3$	24 hours	150	50	150	50	150	100	120
	1 year	50	20	50	40	100	60	50
$PM_{2.5}$ $\mu g/m^3$	24 hours	35	25	35	-	-	-	65
	1 year	15	10	15	25	-	-	15
SO_2 $\mu g/m^3$	1 hour	730	-	-	350	-	-	-
	24 hours	365	20	366	125	150	80	341
	1 year	80	-	78		60	60	78
NO_2 $\mu g/m^3$	1 hour	660	200		200	120		395
	24 hours	-	-	-	-	80	80	-
	1 year	100	40	100	40	40	60	-
CO mg/m^3	1 hour	40	-	40		-	-	-
	8 hours	10	-	10	10	-	-	-

[1] Royal Commission Environmental Regulations in Al Jubail, Saudi Arabia (RCER, 2010)
[2] World Health Organization (WHO, 2006a)
[3] National Ambient Air Quality Standards (NAAQS) (USEPA, 2014b)
[4] European Commission, 2015 (EC, 2015)
[5] (Abhishek Tiwary, 2010)

2.6 Health Effects of Ambient Air Pollution

There is robust scientific evidence showing that exposure to air pollutants is associated with both acute and chronic health effects (WHO, 1999). These health effects can result from a variety of air pollutants depending on the duration and frequency of exposure; and, with respect to the particulate pollutants, the size of pollutants (WHO, 1999). Whereas some of these effects can be related to long-term exposure, others are related to short-term exposure (WHO, 1999). The WHO has summarised the health effects attributed to short-term and long-term exposure to air pollution as shown in Table 2.4 (WHO, 2006a). The effects attributed to short-term and acute exposure to air pollution are more feasibly seen by using data on such occurrences as daily mortality and hospitalisation, while studies on long-term exposure usually investigate the chronic effects of air pollution e.g. Disability Adjusted Life Years (DALY) and Chronic Obstructive Pulmonary Disease (COPD) (COMEAP, 2000; WHO, 2006a).

Table 2.4: Health effects of air pollution attributed to short and long-term exposure

Effects attributed to short-term exposure
Daily mortality
Respiratory and cardiovascular hospital admissions
Respiratory and cardiovascular emergency visits
Respiratory and cardiovascular primary care visits
Use of respiratory and cardiovascular medications
Days of restricted activity
School/Work absenteeism
Acute symptoms (wheezing, coughing, phlegm production, respiratory infections)
Physiological changes (e.g. lung function)
Effects attributed to long-term exposure
Disability Adjusted Life Years (DALY) lost
Reductions in life expectancy as healthy life is lost
Mortality due to cardiovascular and respiratory diseases
Chronic respiratory disease incidence and prevalence (asthma, COPD, chronic pathological changes)
Chronic changes in physiological functions
Lung cancer
Chronic cardiovascular disease
Intrauterine growth restriction (low birth weight, intrauterine growth retardation, small for gestational age)

Source: Adapted from (WHO, 2006a)

Health effects associated with air pollution can be demonstrated using a pyramid as shown in Figure 2.1. At the top of the pyramid are the most severe but less common effects, and at the bottom are the mildest but more common effects. When exposed to air pollution, the severity of health effects increases as the proportion of population affected decreases (WHO, 2006a). This pyramid shows how the effects that are perhaps easiest to measure (premature mortality and hospital admissions) represent only a small proportion of the total health burden following exposure.

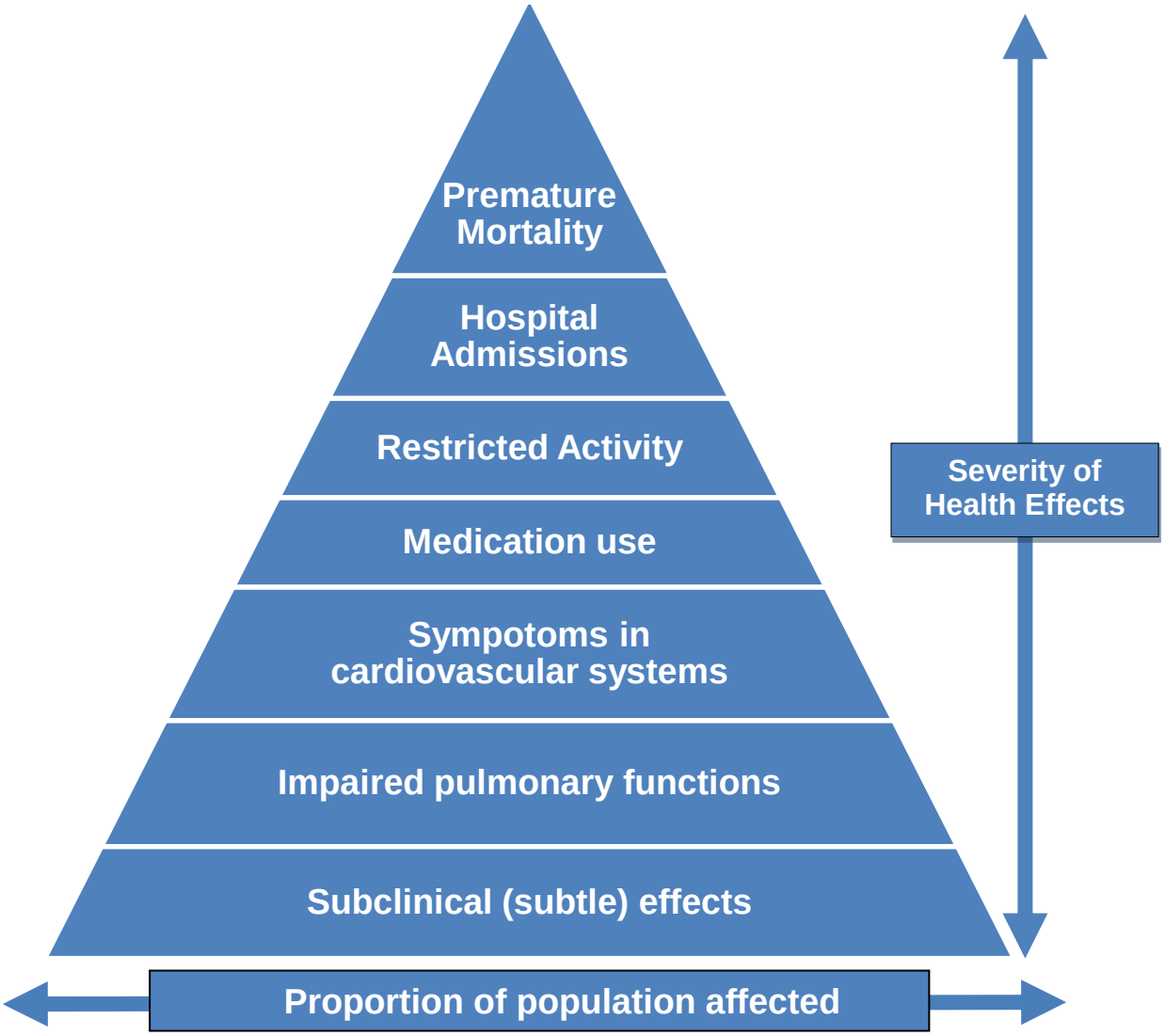


Figure 2.1: Pyramid of health effects associated with air pollution

Adapted from (WHO, 2006a)

2.7 Air Pollution and Mortality

Several studies on air pollution and health have reported an association between air pollution exposure and mortality (Anderson, 2009; COMEAP, 2009; Autrup, 2010; Chen *et al.*, 2010; Cao *et al.*, 2011), but the majority of these studies have been conducted in developed countries (Autrup, 2010; Cao *et al.*, 2011). The results from these studies indicate that an increase in mortality was found in more polluted areas (Autrup, 2010). The earliest evidence relates to the famous London smog, in December 1952, which showed an association between daily changes in particles and sulphate and daily changes in mortality (Fenger, 2009).

Recent large cohort studies, using a common analysis protocol, have been conducted in developed countries, for example the National Mortality and Morbidity Air Pollution Studies (NMMAPS) in the United States based upon 90 cites, and the Air pollution and Health: a European Approach (APHEA), based upon 29 European cities. The studies show that daily variations in PM pollution (measured as PM_{10} and $PM_{2.5}$), even at low levels of exposure, are associated with cardiovascular and respiratory mortality (Brunekreef, 2010).

The estimated percentage change in health outcome per 10µg/m^3 increase in pollutant levels showed slight variation in all-cause mortality, ranging from 0.41% in Asia and US to 0.6% in Europe for PM_{10}, whereas it was 0.35% for Asia and 0.40% in Europe for SO_2 (Autrup, 2010). The stronger association with mortality was for $PM_{2.5}$ rather than for PM_{10} or TSP (Autrup, 2010). Other studies of long-term exposure using a prospective cohort design show similar results, and allow prediction of average life expectancy in years associated with given levels of air pollution (Martuzzi *et al.*, 2002).

These studies also show that children and infants are at increased risk of contracting respiratory disease following exposure to air pollution (Autrup, 2010). These results may be attributed to the fact that the body burden is higher for children and infants than for adults due to a relatively high ventilation rate, as well as increased susceptibility due to the developing respiratory system (Ashmore and Dimitroulopoulou, 2009).

2.8 Asthma Prevalence in Europe

Asthma is defined as a chronic (long-lasting) condition of the respiratory system characterized by inflammation of the air passages (WHO, 2005a). Symptoms of Asthma include difficulty in breathing, wheezing, coughing and tightness in the chest (WHO, 2005a). The underlying process includes chronic inflammation of the airways, reversible obstruction of the flow of air in and out of the airways, and the tendency of the airways to over-react to stimuli (Network, 2014). Asthma most commonly develops in early childhood, and more than three-quarters of children who develop asthma symptoms before age 7 no longer have symptoms by age 16. However, asthma can develop at any stage in life, including in adulthood (Network, 2014).

According to the Global Asthma Report 2014, an estimated 334 million are living with asthma. It is becoming the most common long-term respiratory disease among children (Asthma-UK, 2015). A survey done by the International Study of Asthma and Allergies in Childhood (ISAAC) found that about 14% of the world's children were likely to have had asthmatic symptoms (Network, 2014). In 1999–2004, the prevalence of asthma in children across the European study centres varied from less than 5% to over 20% (WHO, 2007). Figure 2.2 shows asthma prevalence in children across European countries, as reported in the ISAAC study (Phase Three, 1999–2004) (WHO, 2007). However, the data presented in Figure 2.2 are not indicative of prevalence in all European countries as only selected centres (represented by cities/regions) participated in the study. Thus, the intra-country comparison shows the differences between the centres. The highest prevalence of asthma symptoms in children aged 6–7 years (>20%) and 13–14 years (>25%) were found in Ireland and the United Kingdom. The lowest asthma rates for both age groups were found in Albania (<5%) (Network, 2014).

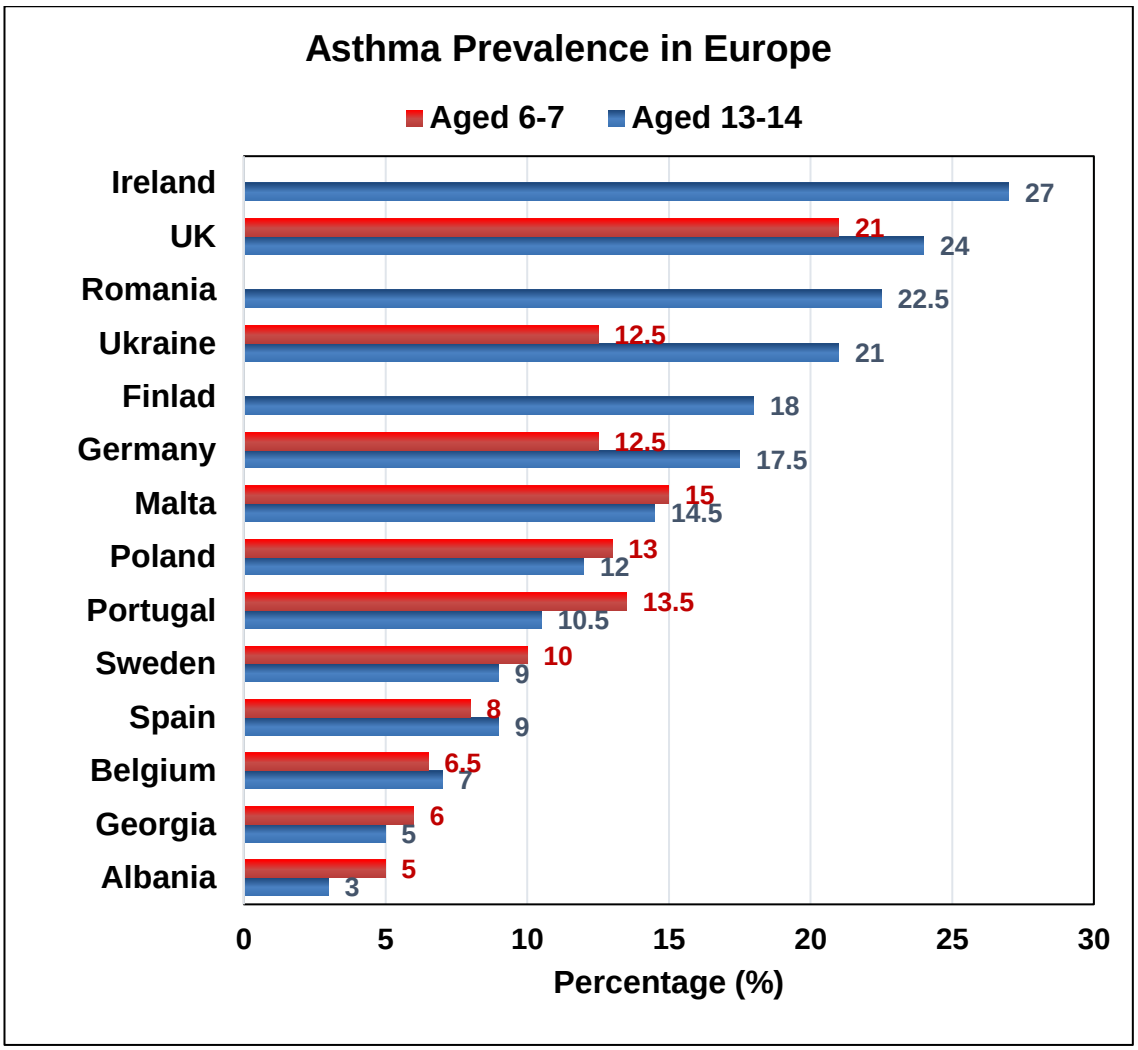


Figure 2.2: Prevalence of asthma symptoms in children aged 6–7 years and 13–14 years, ISAAC Phase Three, 1999–2004

Source: (WHO, 2007)

Note. As the data were collected from specific centres only, prevalence figures are not country-representative.

2.9 Factors Affecting Asthma

A wide variety of factors are known to affect asthma, but not one specific cause, either biological or environmental, has been identified (Network, 2014). Studies indicate there is a complex gene-by-environment interaction. (WHO, 2007). A possible explanation is the “hygiene hypothesis”. This suggests that increased hygiene and the resulting lack of exposure to various microorganisms in early life affect the immune system so that individuals’ ability to fight off certain diseases is weakened and they are more susceptible to autoimmune diseases, such as asthma (WHO, 2007).

Asthma used to be thought of as an allergic disease, where allergen exposure causes sensitisation, and continued exposure leads to the processes in the airway which lead to asthma symptoms (Network, 2014). An increasing trend in the prevalence of asthma and allergies is particularly apparent in urban areas, where children have been found to have more allergic reactions to outdoor and indoor allergens (WHO, 2007). However, some occupational causes of asthma do not appear to involve allergy. These non-allergic mechanisms are currently not well-understood (Network, 2014).

Environmental factors that may provoke asthma attacks include inhaled allergens (commonly dust mites and animal fur; less commonly pollens, moulds, and allergens encountered in the workplace); and inhaled irritants (cigarette smoke, fumes from cooking, heating or vehicle exhausts, cosmetics, and aerosol sprays), and medicines (including aspirin) (Network, 2014).

The use of fossil fuels, as well as higher volumes of road traffic in cities, is thought to contribute to asthma. Recent evidence supports a causal relationship between exposure to air pollution and exacerbation of asthma (WHO, 2007). There is little evidence, however, to support a causal association between the prevalence or incidence of asthma and air pollution in general (WHO, 2007).

Additionally, there seems to be a parallel development between climate change and the increasing prevalence of asthma and allergies in children (WHO, 2007). As warmer temperatures and early spring are related to increased airborne pollen, sensitization to pollen allergens is likely to have doubled during the last three decades, particularly in young people in many areas in Europe (WHO,

2007). Environmental factors are much more likely than genetic factors to have caused the large increase in the numbers of people in the world with asthma (Network, 2014). This is because the rapid increase in incidence of a disease across many different countries and populations shows that it is not likely to be due to genetic factors.

There is increasing evidence and awareness about the relation between asthma development or exacerbation and indoor and outdoor environmental exposure (WHO, 2006b). Indoor exposure to dampness, dust mites and fungal allergens may account for 20% of asthma prevalence (WHO, 2006b). Indoor smoke from solid fuels and environmental tobacco smoke are also significant triggers of asthma symptoms and attacks (WHO, 2006b). Outdoor environmental exposures, such as exposure to poor air quality are also known to exacerbate asthma (WHO, 2006b). In 1995 the Committee on the Medical Effects of Air Pollution (COMEAP) concluded that 'exposure to ambient concentrations of air pollutants is associated with an increase in exacerbations of asthma in those who already have the condition', and more recent evidence has only served to confirm this (COMEAP, 2010).

The WHO (2006) estimated that 44% of the total global disease burden from asthma is related to the environment (WHO, 2006b) while in the USA, 30% of asthma exacerbations among children were due to environmental pollution (EHIB, 2010). The estimate for environmental exposures do not include outdoor exposure to pollen, as this is not realistically modifiable (WHO, 2006b).

2.10 Asthma Prevalence in Saudi Arabia and Gulf Countries

A number of studies have been conducted to investigate the prevalence of asthma among schoolchildren in Saudi Arabia. A study by Al Frayh *et al.* (2001) investigated the changing prevalence of asthma in schoolchildren between 1986 and 1995. The prevalence of asthma in comparable populations using ISAAC questionnaire increased from 8% in 1986 to 23% in 1995, a period of 9 years (Al Frayh et al., 2001). In a follow-up study, Al Frayh (2005) also compared the prevalence of asthma in schoolchildren during the year 2002 in three regions in Saudi Arabia: central, western and eastern regions. The prevalence of asthma in schoolchildren was reported to be 33.7%, 17.7 and 14.1 in eastern, central and western regions, respectively. A comparison of the cumulative prevalence rates for childhood asthma in this 2002 study (21.7%) with those of the previous study in 1995 (23%) showed a slight downward trend. The authors suggested that there is only a small chance that genetic factors could contribute to the increasing prevalence of asthma over a relatively short period. They proposed that the change in the environment directly or indirectly could be responsible for the observed increase in the prevalence of asthma. Variations in asthma prevalence between rural and urban areas as well as within urban areas have been noted in the epidemiological literature (Amoah *et al.*, 2012; Jie *et al.*, 2013). A study by Hijazi *et al.* (1998) compared the prevalence of asthma in schoolchildren living in urban and rural areas of Saudi Arabia. A significantly greater prevalence of asthma was found in urban areas (17.7%) than in rural areas (4.9%) (Hijazi et al., 1998). The reason for the differences in asthma prevalence in rural and urban areas may be due to the fact that populations have different lifestyles and cultures, as well as different environmental exposures and different genetic backgrounds (Subbarao *et al.*, 2009; Jie *et al.*, 2013). Only one study has compared the prevalence of asthma in industrialised urban areas with non-industrialised rural areas in Saudi Arabia (Al-Shairi and Al-Dawood, 1999). The prevalence of asthma was significantly higher among schoolchildren living in an industrialised urban environment (13.9%) than those living in non-industrialised rural areas (8%). The authors hypothesised that the urban-rural differences in prevalence of asthma among schoolchildren in Saudi Arabia relate to environmental and lifestyle factors. These observations suggested that

environmental factors play an important role in the variations of the prevalence of asthma among schoolchildren.

Table 2.5 shows prevalence of asthma in the Gulf Region, which was found in the extant literature from 1993 to 2015. The highest prevalence of asthma within Saudi Arabia was found in the Eastern region (33.7%). This prevalence was higher than that of the surrounding Gulf Countries. The prevalence of asthma in other countries was as follows: Kuwait (22.4%), Iraq (22.3%) and (19.8%) for both Oman and Qatar, while lower prevalence was found in Emirates (13%) and Iran (9.8%). Figure 2.3 shows the prevalence of asthma in Gulf Countries sorted from the highest to the lowest. In addition, Figure 2.4 shows the locations of each country in the Gulf region on a map. The difference in prevalence of asthma among these Gulf countries might be due to differences in socioeconomic status, climate, air pollution, exposure to respiratory infections, life style, allergen concentration, social habits, diet and nutrition, and diverse awareness of physicians and about the diagnosis of asthma (Subbarao *et al.*, 2009; Amoah *et al.*, 2012; Jie *et al.*, 2013; Network, 2014).

Table 2.5: Prevalence of asthma in Saudi Arabia and Gulf countries

Reference	Location (Country/City)	Prevalence (%)	Age (years)	Year
Saudi Arabia				
Al Frayh (2005)	Eastern region	33.7	6-14	2002
	Central region (Riyadh)	17.7		
	Western region	14.1		
Hijazi *et al.* (1998)	Jeddah-Urban area	17.7	12	1998
	Rural area	4.9		
Al-Dawood (2002)	Al-Khubar	9.5	6-15	1995
Al Frayh *et al.* (2001)	Hail & Gizan	23.0	8-16	1995
	Riyadh & Jeddah	8.0		1986
Al-Shairi and Al-Dawood (1999)	Industrialised Urban Area	13.9	6-15	1993
	Non-Industrialised Rural Area	8.0		
Bener *et al.* (1993)	National	6.8	7-12	1990
Kuwait				
Abal *et al.* (2010)	National	22.4	5-7	2009
Owayed *et al.* (2008)	National	15.6	13-14	2002
Awadh Behbehani *et al.* (2000)	National	16.8	13-14	1996
Asher (1998)	National	17.5	13-14	1996
Iraq				
Al-Thamiri *et al.* (2005)	Baghdad	22.3	6-12	2002
Qatar				
Janahi *et al.* (2006)	National	19.8	6-14	2004
Oman				
Al-Rawas *et al.* (2008)	National	19.8	13-14	2001
		10.6	6-7	
Al-Riyami *et al.* (2003)	National	20.7	13–14	1995
		10.5	6-7	
United Arab Emirates				
Alsowaidi *et al.* (2010)	Al-Ain	13.0	13–19	2008
Al-Maskari *et al.* (2000)	National	13.0	6-13	2000
Bener *et al.* (1994)	Al-Ain	13.6	6-14	1993
Iran				
Shakurnia *et al.* (2010)	Ahvaz	9.8	13-14	2007
		6.8	6-7	

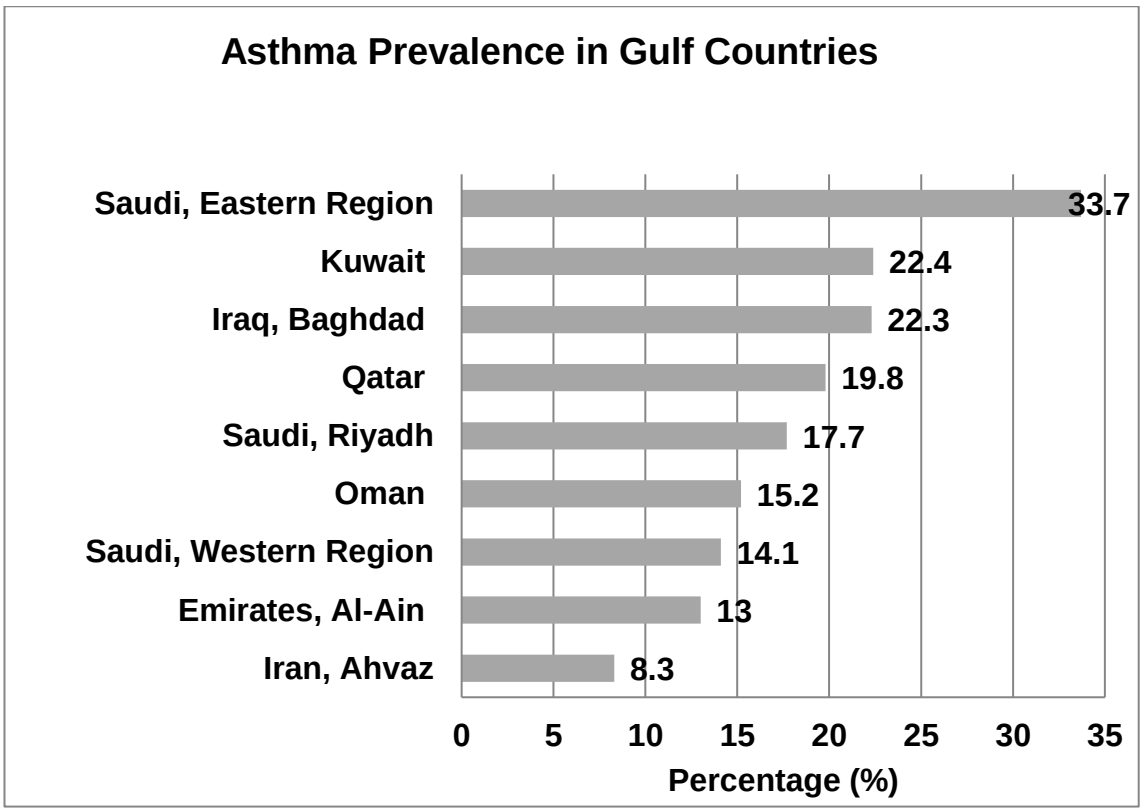


Figure 2.3: Prevalence of asthma symptoms in children aged 6–14 years in Gulf countries in the Middle East, 1993–2010

Figure 2.4: Gulf Countries (Saudi Arabia, Kuwait, Iraq, Iran, Qatar, Emirates and Oman)
Source: (Google Maps, 2015)

2.11 Asthma-related Emergency Department Visits and Air Pollution Levels

In the last few decades, epidemiological studies have shown that exposure to air pollution can aggravate symptoms in asthmatic patients (WHO, 2005a; COMEAP, 2010). Some of these studies have evaluated the short-term effects of air pollution on asthma attacks and visits to emergency department (WHO, 2005a). For such studies, time-series analyses have been most appropriate to use to estimate the influence of daily variations in air pollutant levels on daily counts of asthma-related visits to emergency department within a geographically defined population (Tobías *et al.*, 1999; WHO, 2005a; Wilkinson, 2006).

2.11.1 Early studies (1990 -2003)

A summary of early time-series analyses of air pollution and hospital admissions or visits to emergency department is given in Table 2.6. The WHO report (2005) summarized the epidemiological studies published in the period between1990 and 2003 that used time-series analysis (short-term effects) to study the association between particulate matter (PM_{10}) and hospital admissions or visits to emergency department for asthmatic children (WHO, 2005a). PM_{10} was found to be associated with an increase of 1.5% (95% Confidence Interval (CI) 0.1-2.8) in asthma admissions per 10µg/m^3 increase as shown in Figure 2.5 (WHO, 2005a). Similarly, Wong *et al.* (1999) analysed asthma-related hospital admissions in 1994 and 1995 in Hong Kong, considering PM_{10} together with other pollutants. The analysis revealed a strong relationship between PM_{10} levels and asthma-related visits (Wong *et al.*, 1999). In Seattle, USA, asthma related visits to emergency department for children during 15 months in 1995 and 1996 were evaluated in relation to PM_{10}, where asthma admissions were found to increase by 14% (95%CI: 5.0-23.0) per inter-quartile range (IQR) (11.6µg/m^3) increase in PM_{10} (Norris *et al.*, 1999). Previous studies concerning fine particles ($PM_{2.5}$), most of which were conducted outside Europe, are very limited and more controversial. Understanding of the specific influence of particles of different sizes on health can thus be said to be limited (WHO, 2005a).

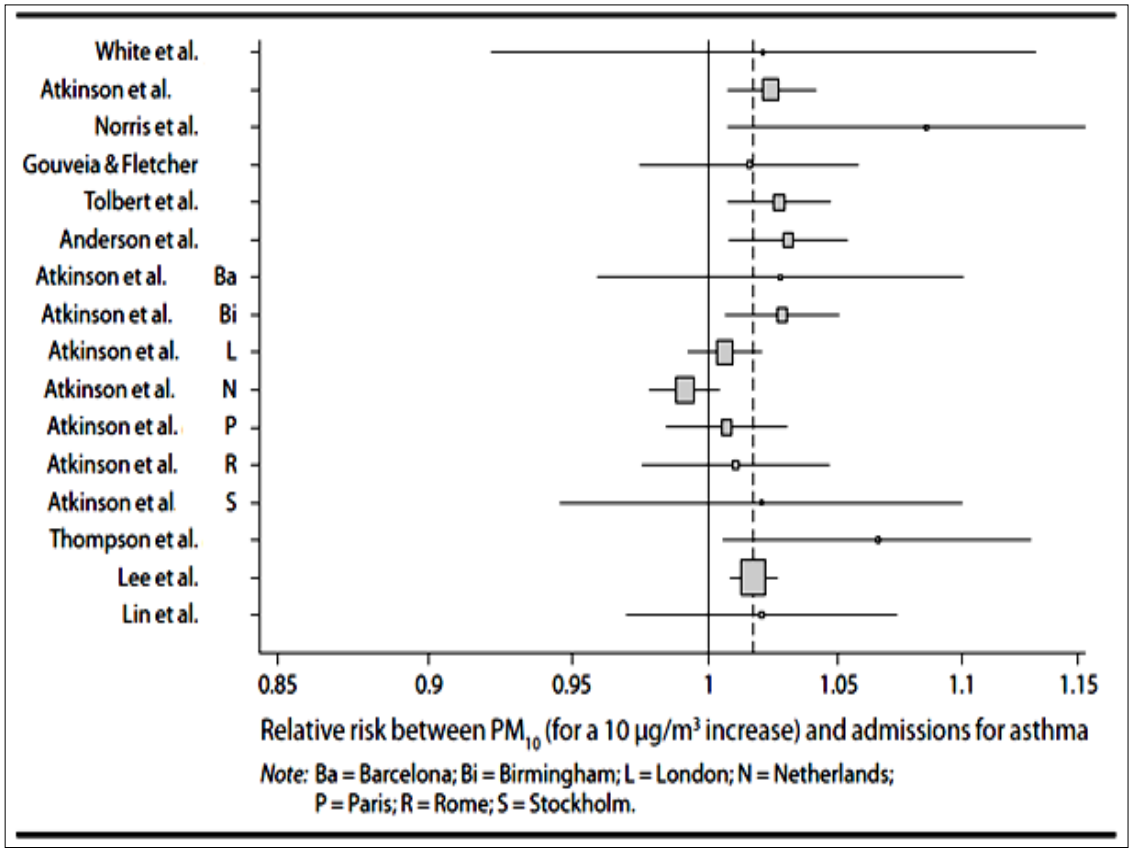


Figure 2.5 Time-series studies on PM_{10} and hospital admissions or visits to emergency department
Source: (WHO, 2005a)

Few studies have reported the relationship between NO_2 and asthma-related hospital admissions (Castellsague *et al.*, 1995; Sunyer *et al.*, 1997; Norris *et al.*, 1999; Wong *et al.*, 1999). Sunyer *et al.* (1997) conducted a study on four cities (Barcelona, Helsinki, London & Paris), in which they considered adults aged between 15-64 years, and children aged between 0-15 years for the period between 1986 and 1992. This study showed a statistically significant effect of nitrogen dioxide (NO_2), with an estimated 2.9% increase (range 0.3-5.5%) per 50µg/m^3 at a cumulative lag of three days (Sunyer *et al.*, 1997). Only one report was available from Asia. Wong *et al.* (1999) analysed asthma-related emergency hospital admissions in Hong Kong for the period between 1994 and 1995. This report showed a similar effect, with an estimated 2.6% increase (range 1.1-4.2%) per 10µg/m^3 (Wong *et al.*, 1999). Castellsague *et al.* (1995) examined the effects of NO_2 on asthma-related emergency room visits during summers and winters of 1985 to 1989 in Barcelona, Spain. NO_2 was associated with asthma-related visits, with an estimated 4.5% increase in summer (range 0.9-8.1%) and 5.6% (range 1.1-10.4) per 25µg/m^3 (Castellsague *et al.*, 1995). Conversely, Norris *et al.* (1999) reported no significant relationship between asthma-related visits to emergency department and NO_2.

For sulphur dioxide (SO_2), Sunyer *et al.* (2003), in the APHEA-2 dataset of European cities (Birmingham, London, Milan, Paris, Rome, Stockholm), and The Netherlands for the period between1988 and 1997, found that daily exposure to SO_2 was associated with an increase in the number of daily asthma-related emergency admissions in children but not for adults. In contrast to earlier findings, however, no significant effect of SO_2 exposure on asthma-related visits was found in previous studies for all ages (Castellsague *et al.*, 1995; Norris *et al.*, 1999; Wong *et al.*, 1999). Given the high correlation of SO_2 with other pollutants, such as PM and CO, it is difficult to determine whether these associations were due to SO_2 itself or to other pollutants emitted from fuel combustion processes (Sunyer *et al.*, 2003). However, the results of most controlled-chamber experiments have consistently shown that asthmatics are more sensitive to SO_2 than non-asthmatics (Castellsague *et al.*, 1995; WHO, 2006a).

Black smoke (BS) was found to have no statistically significant association with asthma admissions (4.6% increase in admissions per 50µg/m^3) (Sunyer *et al.*, 1997). One study carried out in Seattle, USA, reported an association

between CO and asthma-related visits, with an estimated 10.0% increase in admissions (range 10.2-11.9%) per IQR (0.6 ppm) (Norris *et al.*, 1999).

Studies conducted outside Europe on Ozone (O_3) tend to show an increase in asthma-related hospital admissions with increase in O_3, while some studies in Europe have found an apparent protective effect of this pollutant. The results for studies on effects of Ozone are more controversial, owing to issues concerning the design of time-series studies of this pollutant; final conclusions cannot therefore be drawn, although there is some evidence supporting an effect (WHO, 2005a).

Finally, many of the studies reviewed by the WHO report showed a stronger effect in asthmatic children than adults, indicating greater susceptibility in children to outdoor pollutant components (WHO, 2005a).

Table 2.6: Previous studies that used time-series analysis to show association between air pollution and asthma-related admissions

Authors	Location	Period	Age years	Study Design	Pollutant	Conc. ($\mu g/m^3$)	Lag day	Asthma-related admissions 95% CI	RR (%)
WHO (2005a)	EU	1990-2003	All ages	Meta-analysis	PM_{10}	↑10	-	1.015 (CI:1.010, 1.020)	1.5
Sunyer *et al.* (2003)	APHEA-2 Project Birmingham London, Milan, Paris, Rome, Stockholm, and The Netherlands	1988-1997	0-14	TSA*	SO_2	↑10	-	1.013 (CI: 1.004, 1.022)	1.3
			15-64				-	NO Effects	-
Wong *et al.* (1999)	Hong Kong	1994-1995	All ages	TSA*	NO_2	↑10	0-3	1.026 (CI: 1.010, 1.042)	2.6
					SO_2	-	-	Not sig.	-
					PM_{10}	↑10	0-3	1.015 (CI: 1.002, 1.028)	1.5
					O_3	↑10	0-2	1.031 (CI: 1.017,1.046)	3.1
Norris *et al.* (1999)	Seattle, USA	1995-1996	0-18	TSA*	PM_{10}	↑IQR 11.6	-	1.14 (CI: 1.05, 1.23)	14.0
					CO	↑IQR 0.6 ppm	-	1.10 (CI: 1.02, 1.19)	10.0
					NO_2	-	-	Not sig.	-
					SO_2	-	-	Not sig.	-
Sunyer et al. (1997)	APHEA-1 Project Barcelona, Helsinki, London and Paris	1986-1992	0-64	TSA*	NO_2	↑50	3	1.029 (CI: 1.003, 1.055)	2.9
					BS	-	-	Not sig.	-
					O_3	-	-	Not sig.	-
Castellsague et al. (1995)	Barcelona, Spain	1985 - 1989	All ages	TSA*	NO_2	↑25	-	Summer: 1.045 (CI: 1.009, 1.081)	4.5
							-	Winter: 1.056 (CI: 1.011, 1.104)	5.6
					SO_2	-	-	Not sig.	-
					O_3	-	-	Not sig.	-
Schwartz et al. (1993)	Seattle, USA	NA	All ages	TSA*	PM_{10}	↑30	0-4	1.12 (CI: 1.04, 1.20)	11.2

*TSA = Time-Series Analysis, ↑ = increase in pollutant concentrations

2.11.2 Recent studies

There exist limited papers reporting on time-series analysis for air pollution and asthma-related visits to emergency department for the period between 2003 to date. The majority of the studies published in this period confirm the WHO report and previous findings (WHO, 2005a). A summary of recent time-series analyses of air pollution and hospital admissions or visits to emergency department is given in Table 2.7.

Several sources have identified that an increase in asthma-related admissions is associated with a 10μg/m^3 increase in PM_{10} (Galan *et al.*, 2003; Ko *et al.*, 2007; Giovannini *et al.*, 2010; Nastos *et al.*, 2010; Samoli *et al.*, 2011), with an increase in IQR of daily mean levels (Lee *et al.*, 2006b) and with other value increases (Bell *et al.*, 2008; Tadano *et al.*, 2012) in PM_{10} levels. In Sao Paulo, Brazil, Tadano *et al.* (2012) reported a statistically significant effect for PM_{10}, with an estimated 5.0% increase in asthma-related hospital admissions per 90μg/m^3 at a cumulative lag of three days. Similarly, Bell *et al.* (2008) found a significant positive connection between cumulative lag of three days of PM_{10} with asthma-related hospital admissions. Overall, the recent studies estimated the effects to range from 0.1% to 7.4% increase of asthma-related hospital admissions per a 10μg/m^3 increase in PM_{10} level, (Galan *et al.*, 2003; Ko *et al.*, 2007; Giovannini *et al.*, 2010; Nastos *et al.*, 2010; Samoli *et al.*, 2011). Samoli *et al.* (2011) indicated that the increasing risk in Athens, Greece, was higher in winter and desert dust days.

Some studies reported on the relationship between exposure to fine PM fraction ($PM_{2.5}$) and hospital visits for asthma patients (Lee *et al.*, 2006b; Ko *et al.*, 2007; Bell *et al.*, 2008; Mar *et al.*, 2010; Silverman and Ito, 2010; Li *et al.*, 2011). These studies indicate that an increase in $PM_{2.5}$ level by 7-20μg/m^3 increased cases of asthma-related admissions by 3%-9%.

Other studies have considered the relationship between NO_2 levels and asthma-related admissions (Galan *et al.*, 2003; Lee *et al.*, 2006b; Ko *et al.*, 2007; Giovannini *et al.*, 2010; Li *et al.*, 2011; Cirera *et al.*, 2012). Most of these studies have reported a significant positive association between NO_2 levels and asthma-related hospital visits, with an increase of 9-27μg/m^3 of NO_2 causing an estimated increase of 0.2%-9.0% of asthma-related admissions. However, two studies

reported by Bell *et al.* (2008) and Samoli *et al.* (2011) found limited evidence for an association between the level of NO_2 and hospital admissions.

Recent studies have reported a positive association between an increase in the level of SO_2 and asthma-related hospital admissions (Galan *et al.*, 2003; Lee *et al.*, 2006b; Li *et al.*, 2011; Samoli *et al.*, 2011; Cirera *et al.*, 2012).

A positive relationship was also reported in several studies between an increase in the concentration of O_3 and emergency department visits among asthma patients (Galan *et al.*, 2003; Lee *et al.*, 2006b; Ko *et al.*, 2007; Bell *et al.*, 2008).

Others factors have also been found to be related with increased asthma-related admissions, such as diurnal changes in humidity and temperature (Kim *et al.*, 2014) (Mireku *et al.*, 2009). A study conducted by Kim *et al*. (2014) reported that a 1-unit increase in temperature led to a 3.5% (95% CI 0.7, 6.4%) increase in asthma-related visits to the emergency department. According to Mireku *et al.* (2009), fluctuations in humidity, but not barometric pressure, appear to influence asthma-related emergency department visits. Other studies have also reported associations between ambient pollen levels and various measures of asthma morbidity (D'Amato *et al.*, 2010; Darrow *et al.*, 2012).

Table 2.7: Recent studies that used time-series analysis to show association between air pollution and asthma-related admissions

Authors	Location	Period	Age years	Study Design	Pollutant	Conc. ($\mu g/m^3$)	Lag day	Asthma-related admissions 95% CI	RR (%)
Samoli et al. (2011)	Athens, Greece	2001 - 2004	0-14	TSA*	PM_{10}	↑10.0	0	1.025 (CI:1.006, 1.508)	2.5
					SO_2	↑10.0	0	1.059 (CI:1.008, 1.113)	5.9
					O_3	-	-	Not sig.	-
					NO_2	-	-	Not sig.	-
Silverman and Ito (2010)	New York, USA	1999 - 2006	All ages	TSA*	$PM_{2.5}$	↑IQR 12.0	0-1	1.090 (CI:1.060, 1.120)	9.0
					O_3	↑IQR 22.0 ppb	0-1	1.090 (1.060, 1.120)	9.0
Nastos et al. (2010)	Athens, Greece	2001 - 2004	5-14	TSA*	PM_{10}	↑10.0	1	1.034 (CI:1.018, 1.050)	3.4
Giovannini et al. (2010)	Milan, Italy	2007 - 2008	0-14	TSA*	CO	↑1mg/m^3	1	1.105 (CI:1.005, 1.149)	10.5
					NO_2	↑10.0	1	1.002 (CI:1.000, 1.004)	0.2
					PM_{10}	↑10.0	0	1.001 (CI:1.000, 1.003)	0.1
					O_3	-	-	Not sig.	-
Ko et al. (2007)	Hong Kong	2000 - 2005	All ages	TSA*	NO_2	↑10.0	0-4	1.028 (CI:1.021, 1.034)	2.8
					O_3	↑10.0	0-5	1.034 (CI:1.029, 1.039)	3.4
					PM_{10}	↑10.0	0-5	1.019 (CI:1.015, 1.024)	1.9
					$PM_{2.5}$	↑10.0	0-5	1.021 (CI:1.015, 1.028)	2.1
					SO_2	-	-	Not sig.	-
Cirera et al. (2012)	Cartagena, Spain	1995 - 1998	All ages	TSA*	SO_2	↑10.0	4	1.061 (CI:1.014, 1.110)	6.1
					NO_2	↑10.0	4	1.026 (CI:1.004, 1.049)	2.6
					TSP	-	-	Not sig.	-
					O_3	-	-	Not sig.	-
Li et al. (2011)	Detroit, USA	2004 - 2006	2-18	TSA*	NO_2	↑IQR 9.6 ppb	5	1.038 (CI:1.005, 1.072)	3.8
					SO_2	↑IQR 4.0 ppb	5	1.040 (CI:1.017, 1.064)	4.0
					CO	↑IQR 0.3 ppm	5	1.023 (CI:1.004, 1.043)	2.3
					$PM_{2.5}$	↑IQR 9.2	5	1.036 (CI:1.014, 1.059)	3.6
Mar et al. (2010)	Tacoma, USA	1999 - 2002	All ages	TSA*	$PM_{2.5}$	↑IQR 7.0	2	1.04 (CI:1.01, 1.07)	4.0
					CO	↑IQR 0.7 ppm	2	1.03 (CI:1.00, 1.06)	3.0
Lee et al. (2006b)	Hong Kong	1997 - 2002	0-18	TSA*	PM_{10}	↑IQR 33.4	4	1.074 (CI:1.056, 1.093)	7.4
					$PM_{2.5}$	↑IQR 20.6	4	1.066 (CI:1.045, 1.087)	6.6
					SO_2	↑IQR 11.1	5	1.015 (CI:1.002, 1.027)	1.5
					NO_2	↑IQR 27.1	3	1.090 (CI:1.072, 1.109)	9.0
					O_3	↑IQR 23.0	3	1.056 (CI:1.041, 1.079)	5.6
Galan et al. (2003)	Madrid, Spain	1995 - 1998	All ages	TSA*	PM_{10}	↑10.0	3	1.039 (CI:1.010, 1.068)	3.9
					SO_2	↑10.0	3	1.029 (CI:0.997, 1.062)	2.9
					NO_2	↑10.0	3	1.033 (CI:1.013, 1.054)	3.3
					O_3	↑10.0	1	1.045 (CI:1.018, 1.073)	4.5
Bell et al. (2008)	Taipei, Taiwan	1995 - 2002	All ages	TSA*	O_3	↑10.0ppb	0-3	1.076 (CI:1.029, 1.125)	7.6
					CO	-	-	Not sig.	-
					PM_{10}	↑28.0	0-3	1.045 (CI:1.007, 1.084)	4.5
					$PM_{2.5}$	-	-	Not sig.	-
					NO_2	-	-	Not sig.	-
					SO_2	-	-	Not sig.	-
Tadano et al. (2012)	Sao Paulo, Brazil	2007 - 2008	All ages	TSA*	PM_{10}	↑90.0	3	1.05	5.0

*TSA = Time-Series Analysis, ↑ = increase in pollutant concentrations

2.12 Human Exposure to Air Pollution

The term *human exposure* was defined by Ott (1982) as "the event when a person comes into contact with a pollutant of a certain concentration during a period of time". This means that exposure requires both the pollutant and the person to be present (Ashmore and Dimitroulopoulou, 2009) and is represented by the following equation:

$$E_i = \sum_{j}^{J} C_j \times t_{ij}$$

Equation 1

where *"E_i"* is the time-weighted integrated exposure for person '$_i$' over the specified time period, *"C_j"* is the pollutant concentration in microenvironment '$_j$', and "J" the total number of microenvironments that person '$_i$' visited (Watson AY *et al.*, 1988).

The term *dose* refers to the amount of pollution that enters a contact host in a specified time duration, while *concentration* is defined as the amount of pollutant per unit volume inside a given environmental medium (WHO, 2006a). Therefore, exposure needs both concentration and dose to be present and this can occur along the source-receptor pathway as shown in Figure 2.6 (WHO, 2006a; Ashmore and Dimitroulopoulou, 2009). As shown in Figure 2.6, the source may be industry refineries and automobile exhaust, which can emit a variety of contaminants into the air, water and soil, leading to environmental concentrations. Dispersion takes place into so-called "microenvironments" such as indoors or outdoors at home, school, work, during transit and in other locations. Where people come into contact with the environmental contaminants, exposure is considered to have occurred. There are two main routes of exposure which are known as intake and uptake routes by which pollutants cross the boundary from outside to inside the body (USEPA, 1992). Intake is via inhalation through the respiratory system and via ingestion through the gastrointestinal system (USEPA, 1992). The other main route of uptake includes dermal absorption of the chemical through the skin or eye (USEPA, 1992; WHO, 2006a). (WHO, 2006a; Nieuwenhuijsen, 2015). The factors determining exposure include, for example, duration of time spent in different indoor and outdoor

locations, ventilation rate and weather conditions. The exposure route(s) of a contaminant and the amount of uptake (dose) depends on, for example, the biological, chemical, and physical characteristics of the contaminants, location and activity of the person, and the person themselves. The reaction with the body boundary through exposure routes (inhalation, ingestion, dermal) may have an impact on health (Nieuwenhuijsen, 2015). Most epidemiological and toxicological studies in the field of air pollution have focused on respiratory and cardiovascular effects which are likely to occur via inhalation exposure (WHO, 2006a).

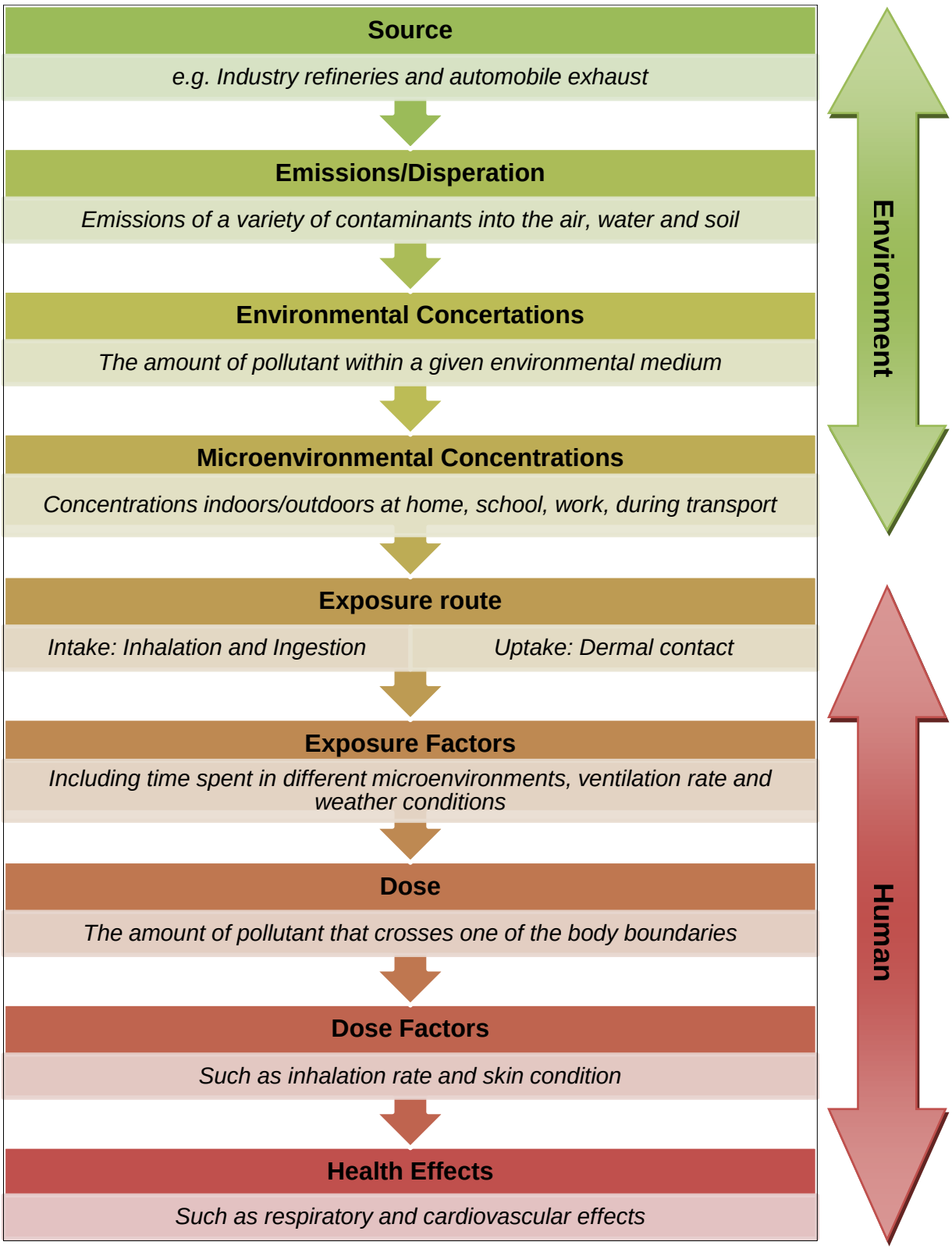


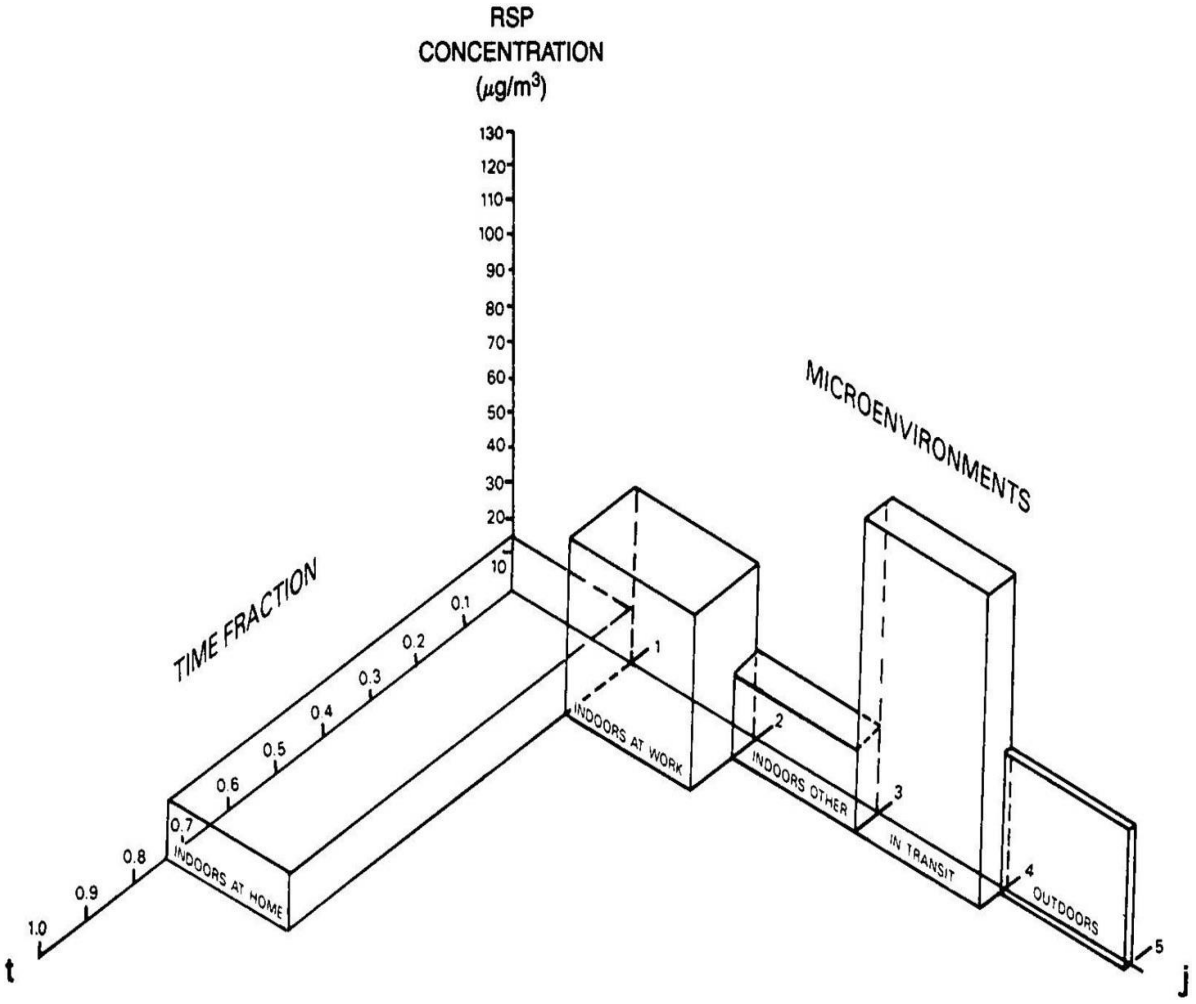
Figure 2.6: source-receptor pathway

Adapted from: (WHO, 2006a; Nieuwenhuijsen, 2015)

2.13 Microenvironments and Time-Activity Patterns

A *microenvironment* (ME) is defined as "a small space in which human contact with a pollutant takes place, and which can be treated as a well-characterized, relatively homogenous location with respect to pollutant concentrations for a specified time period" (USEPA, 2014a). Microenvironments include indoors/outdoors at home, school, work, during transport and in other locations. Most air pollution measurements have been made in the outdoor community air microenvironment. Nevertheless, because most people are outside for a relatively small fraction of the time, and because the amount of air pollution that penetrates indoors is modified by building characteristics, these outdoor measurements are of marginal value in estimating the actual exposures of humans to many air pollutants (Watson AY *et al.*, 1988).

The concept of a time-weighted integrated exposure is illustrated in Figure 2.7. A unit width is indicated on the *j* axis for each of five microenvironments: indoors at home, indoors at work, indoors in other locations, in transit, and outdoors. The concentration of respirable particles (RSP) is displayed on the Y-axis, and the fraction of time that person *i* spends in each microenvironment over the 24-hr period is plotted on the *t* axis. The volumes of the boxes shown in Figure 2.7 represent contributions from each of the five microenvironments to the time-weighted integrated exposure. The contribution of each microenvironment is represented mathematically in the table at the bottom of Figure 2.7 Even though respirable particle concentration was low inside the home, it contributed significantly to the time-weighted exposure because this person spent 18 out of 24 hr in this microenvironment. Conversely, the relatively higher respirable particle concentration outdoors made only a minor contribution to the time-weighted exposure because this person was outdoors for less than half an hour during the 24-hr period. This illustrates the general problems associated with attempts to define the limits of microenvironments that are sufficiently homogeneous, to identify which among them are the significant contributors to integrated exposure, and to measure or estimate both the pollutant concentration C_j and the average time, t_{ij}, the subject spends in the microenvironment (Watson AY *et al.*, 1988).



Microenvironment Type	RSP Concentration (C_j, μg/m^3)	Time Fraction[a] (t_{Ij})	$C_j \times t_{Ij}$ (μg/m^3)	Microenvironment Contribution[b] to E_i (%)
Indoors at Home	15	0.75	11.25	47
Indoors at Work	50	0.15	7.5	31
Indoors, Other	25	0.04	1	4
In Transit	90	0.04	3.6	15
Outdoors	40	0.02	0.8	3
			$E_i = \sum C_j \times t_{ij} = 24.15$ μg/m^3	

a Fraction of 24 hr spent in each microenvironment.
b Percentage that each microenvironment contributes to the 24-hr, time-weighted, integrated exposure (E_i).

Figure 2.7**:** Example of the relative contributions from specific microenvironments to an individual's time-weighted, integrated exposure to respirable particles (RSP).

Source adapted from (Watson AY *et al.*, 1988)

In some regions, seasons affect microenvironment-associated exposures. For example, a study by Kornartit et al. (2010) conducted in north London in 2009 illustrated the contribution to exposure of different microenvironments between summer and winter seasons as shown in Figure 2.8. This Figure 2.8 shows a summary of personal exposure in microenvironments, and illustrates the significance of indoor sources (Kornartit et al., 2010).

Time-activity patterns can be used in conjunction with air pollution levels in different microenvironments to generate a measure of total human exposure (WHO, 2006a). The true level of exposure depends on the individual activities of the person (WHO, 1999). Figure 2.9 shows different profiles of time-activity patterns for typical days of a full-time worker, homemaker with young children, retired person and schoolchild (WHO, 1999).

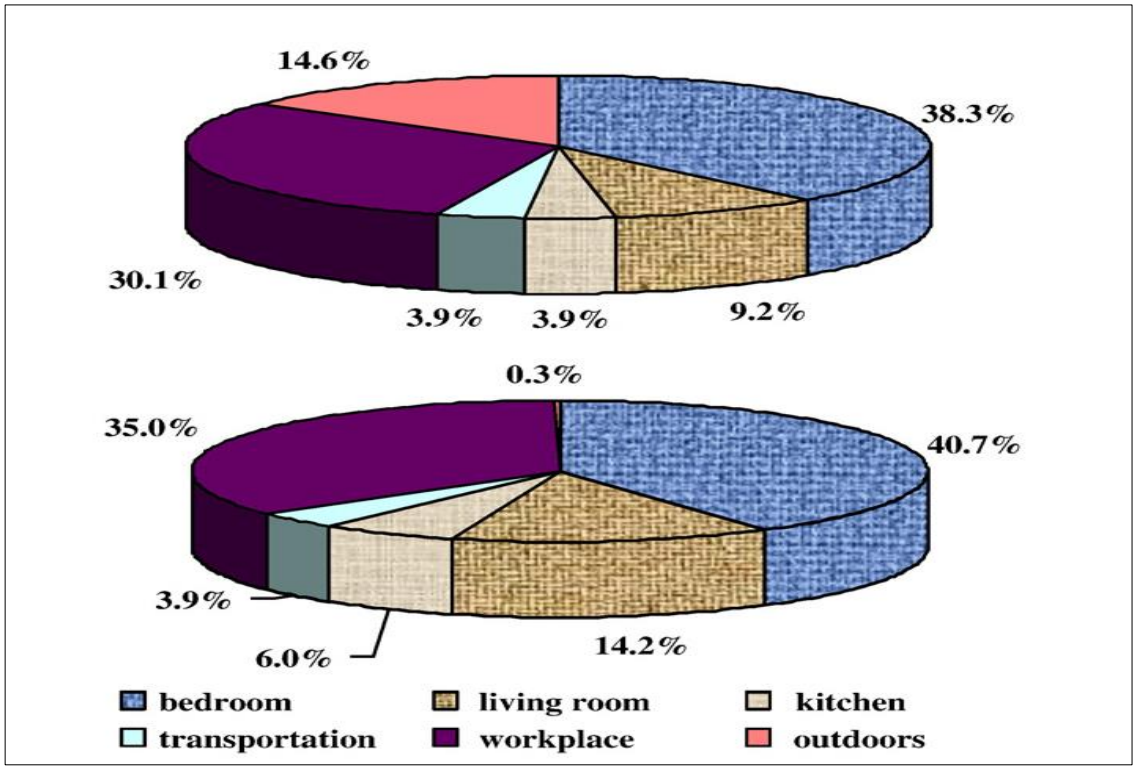


Figure 2.8: Distribution of personal exposure in different microenvironments during summer (above) and winter (below) Source: (Kornartit *et al.*, 2010)

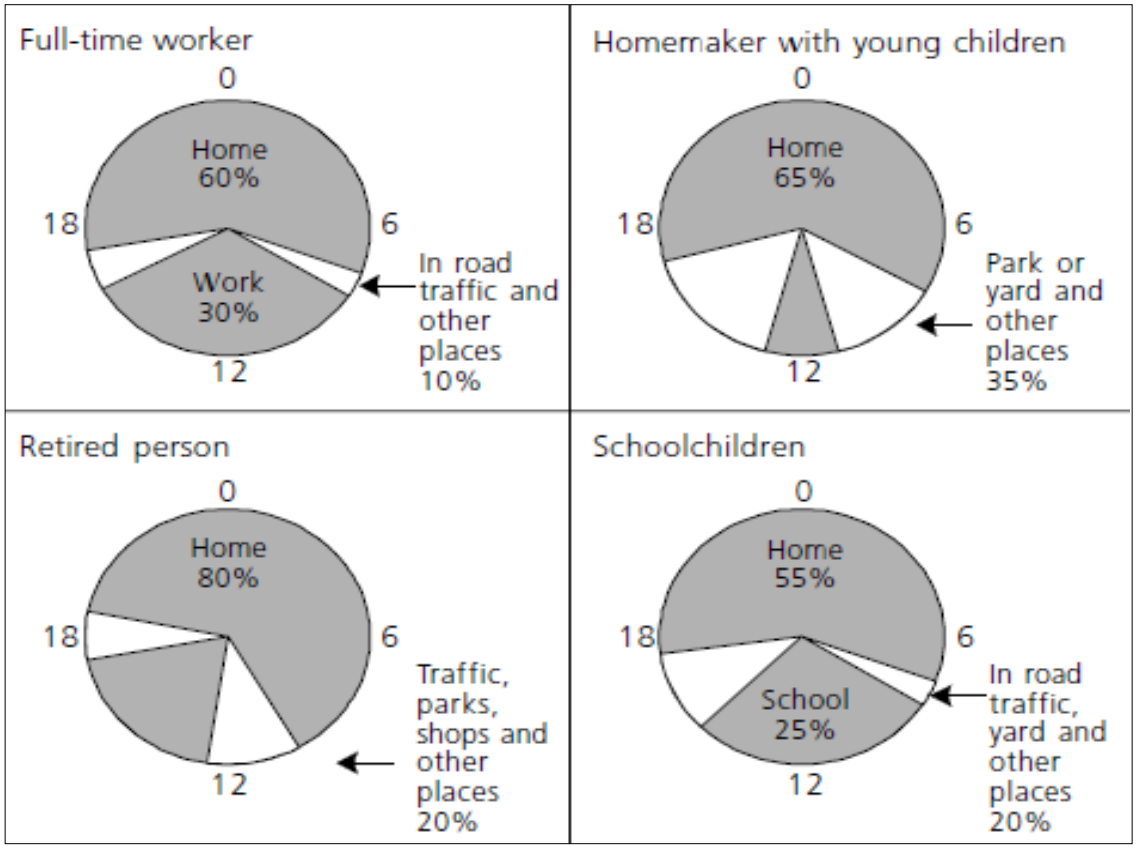


Figure 2.9: Examples of Time-activity profiles for typical 24-hour days Source: (WHO, 1999)

Many epidemiological studies have used outdoor concentrations of air pollutants as a surrogate for human exposure (Briggs, 2005; Ashmore and Dimitroulopoulou, 2009; Autrup, 2010; Avery *et al.*, 2010). A common feature of such studies is their reliance on ambient fixed-site measurement stations as proxies for personal exposure (Briggs, 2005; Violante *et al.*, 2006; Tsai *et al.*, 2008; Gerharz *et al.*, 2009; Avery *et al.*, 2010). In reality, these fixed-site monitoring stations alone may not provide good estimates of personal exposure. This is because individuals spend most of their time indoors (Ashmore and Dimitroulopoulou, 2009; Gerharz *et al.*, 2009; Avery *et al.*, 2010; Kornartit *et al.*, 2010), where they are exposed to various sources of air pollutants that are different from those outdoors (Gerharz *et al.*, 2009; Avery *et al.*, 2010). Several studies, particularly those conducted in Europe and North America, indicate that on average, people spend 87.0% (range 81.5%-94.5%) of their time indoors, 7.0% (range 3.4%-14.1%) outdoors and 5.3% (ranged 2.5%-8.0%) in transport as shown in Table 2.8 (Jantunen *et al.*, 1998; Burke *et al.*, 2001; Lai *et al.*, 2004; Wu *et al.*, 2005; Kim *et al.*, 2006; Johannesson *et al.*, 2007; Braniš and Kolomazníková, 2010; Mohammadyan, 2011; Michikawa *et al.*, 2014; Wang *et al.*, 2014). Therefore, total personal exposure results from a combination of personal experiences in different microenvironments (WHO, 2006a).

Table 2.8: Selected studies presenting total time spent indoors, outdoors and in transport

Author	Location (City/Country)	Total Time spent (%)		
		Indoor	Outdoor	Transport
Michikawa *et al.* (2014)	6 Japanese cities	84.0	-	-
Wang *et al.* (2014)	Guangzhou, China	81.5	-	-
Mohammadyan (2011)	Bradford, UK	90.8	3.4	4.7
Braniš and Kolomazníková (2010)	Prague, Czech	84.3	10.6	5.1
Johannesson *et al.* (2007)	Gothenburg, Sweden	94.5	4.0	2.5
Kim *et al.* (2006)	Toronto, Canada	88.6	5.3	6.1
Wu *et al.* (2005)	Alpine-CA, USA	82.6	14.1	3.3
Lai *et al.* (2004)	Oxford, UK	89.5	3.8	6.7
Jantunen *et al.* (1998)	6 European cites	88.0	4.0	8.0
Burke *et al.* (2001)	Philadelphia-PA, USA	83.0-91.0	10.8	6.2
Overall mean (Range)		**87.0** (81.5-94.5)	**7.0** (3.4-14.1)	**5.3** (2.5-8.0)

2.14 Relationship between Personal Exposure, Indoor, Outdoor and Transport Concentrations

The WHO (2006a) summarised the key factors that determine the association between outdoor and indoor air pollution concentrations. The first is the spatial variation in ambient air concentration, which is related to the geographical distribution and type of emission source (point or line). For some pollutants, such as carbon monoxide, the concentration is simply a gradient, decreasing with increasing distance from the source. In contrast, the formation of secondary air pollutants shows little variation on the local scale. Weather conditions, such as wind speed, wind direction and solar radiation, can determine the dispersion and transfer of pollutants. Secondly, the amount of an outdoor generated pollutant penetrating into the indoor environment has a significant effect on the association between outdoor and indoor concentrations. This depends on the penetration coefficient, the ventilation rate, and the rate of decay. The penetration factor from outdoor to indoor air has been shown to vary for the different particle size fractions, with the smallest particles penetrating almost completely from outside to inside. For a typical home with an air exchange rate of 0.75 air changes per hour and no indoor sources, the estimated average concentration of fine PM indoors is about 65% of the outdoor concentration. For NO_2 the figure is approximately 40–50%. For O_3 and SO_2, this ratio is generally much lower. Finally, indoor sources such as cooking, heating sources and smoking can significantly contribute to total human exposure (WHO, 2006a).

2.14.1 Personal exposure to $PM_{2.5}$ in different microenvironments

Fine particulate matter ($PM_{2.5}$) has been the focus of much attention of recent studies (Kaur *et al.*, 2007) due to the National Ambient Air Quality Standards (NAAQS) being revised by the US Environment Protection Agency (EPA), leading to the introduction of $PM_{2.5}$ regulations (Kaur *et al.*, 2007; USEPA, 2014b). Another reason is because of the increasing number of studies that suggest that $PM_{2.5}$ has greater toxicity (Donaldson *et al.*, 2001; Donaldson *et al.*, 2002; Kaur *et al.*, 2007). In comparison to studies investigating exposure to gaseous pollutants, far fewer exposure studies examining fine particulate matter have been carried out across different microenvironments. This has been mainly due to logistical and practical issues of sampling various microenvironments, the availability of appropriate portable personal monitoring equipment with suitable detection limits and the associated financial constraints (Kaur *et al.*, 2007). However, the number of studies examining personal exposure concentrations in different microenvironments have increased over the last decade for particulate mass, and more recently, ultrafine particle counts (Kaur *et al.*, 2007).

Personal exposure studies covering continuous exposures over one or more days often use time-activity diaries to record the time spent across different microenvironments (e.g. indoor, outdoor and transport). Many of these studies focus on ETS (Environmental Tobacco Smoke), but only the non-ETS influenced samples are reviewed here, as they are more relevant to the aims and objectives in this study. Table 2.9 shows a a typical example of those relevant studies that used personal monitoring device to measure personal $PM_{2.5}$ concentration across different microenvironments (e.g. indoor, outdoor and transport). Many of these studies have been conducted in developed countries (Burke *et al.*, 2001; Kousa *et al.*, 2002; Lai *et al.*, 2004; Brunekreef *et al.*, 2005; Kaur *et al.*, 2006; Tsai *et al.*, 2008; Kornartit *et al.*, 2010; Wichmann *et al.*, 2010; Zuurbier *et al.*, 2010). A study in Philadelphia, USA, indicated that indoor-residential $PM_{2.5}$ exposures had the most influence on total $PM_{2.5}$ exposures compared to other $PM_{2.5}$ microenvironments (Burke *et al.*, 2001). Figure 2.10 illustrates a summary of expected exposure to $PM_{2.5}$ for a simulated population of Philadelphia based on available measurements of outdoor, indoor and personal measurements, accompanied with details of exposure due to time spent in various microenvironments and activities (Burke *et al.*, 2001). For the majority of the

population in Philadelphia, outdoor and in-vehicle exposure contributes little to total exposure, while indoor residential exposure is the most significant contributor of total exposure to $PM_{2.5}$ (Burke *et al.*, 2001). Results from the EXPOLIS study (2002) suggested that ambient fixed-site monitoring provides better estimates of $PM_{2.5}$ exposure for relatively inactive individuals who stay at home, than for active, working adults (Kousa et al., 2002).

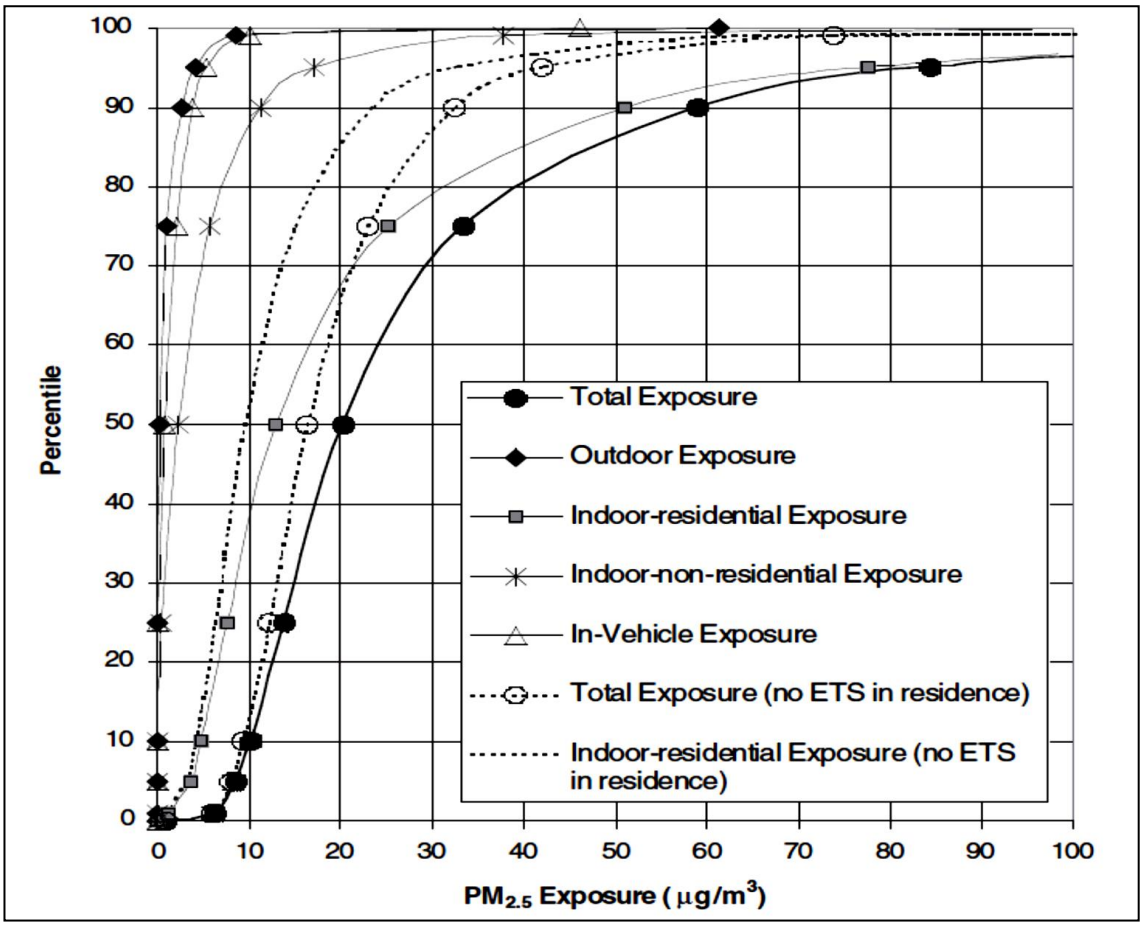


Figure 2.10: Cumulative frequency distributions of daily total and microenvironmental $PM_{2.5}$ exposures for the simulated population of Philadelphia, USA
Source: (Burke *et al.*, 2001)

Table 2.9: Selected studies which presented personal exposure to $PM_{2.5}$ in different microenvironments

Author	Location	Method (Measuring time)	Period	Age (n)	Mean (Median) $PM_{2.5}$ Concentrations ($\mu g/m^3$)						
					School	Work	Home	Total Indoor	Total Outdoor	Transport	Personal
Wichmann et al. (2010)	Stockholm, Sweden	-	2003-2004	6-11 (18)	8.1 (8.3)	-	10.1 (10.0)	8.4 (7.9)	9.4 (8.1)	-	-
Wang et al. (2014)	Guangzhou-China	(pDR-1500 personal dust monitor)	2010	6-13 (216)	AM[1] 63.2 (57.5)	-	AM[1] 118.8 (117.4)	-	-	-	-
Mohammadyan, (2005) and (2011)	Bradford, UK	(48h)	2002-2003	40	-	GM[2] 27.3	GM[2] 19.0	-	-	-	30.3
Jantunen et al. (1998) and Marino M (2002)	6 European Cites	EXPOLIS Study	-	25-55	-	GM[2] 40.4	GM[2] 26.4	-	-	-	GM[2] 24.0
Vallejo et al. (2004)	Mexico City	(13h)	2002	21-40 (40)	(93.3)	-	(54.5)	(70.8)	(89.1)	-	-
Johannesson et al. (2007)	Gothenburg, Sweden	(24h)	2000	30 (23-51)	-	-	(8.6)	-	-	-	(8.4)
Rojas-Bracho et al. (2002)	Santiago, Chile	(24h)	1998-1999	(20 children)	-	-	-	68.5 (61.0)	68.1 (60.9)	-	59.5 (57.5)
Kim et al. (2006)	Toronto, Canada	(24h)	1999-2001	49-80 (28)	-	-	-	-	-	-	22.0 (14.0)
Lim et al. (2012)	Seoul, Korea	(SidePak)3days	-	(2 adults)	-	-	13.4 (11.6)	19.5	21.0 (18.6)	18.6 (16.8)	19.8 (15.4)
Borgini et al. (2011)	Milan, Italy	(SidePak) (21h)	2006	High school pupils (90)	-	-	-	79.3	77.9	-	75.9 (58.1)
Yassin et al. (2012)	Kuwait	(Dust Track)11 houses	2012	-	-	-	-	46.0	-	-	-
Brains and Kolomaznikova (2010)	Prague, Czech	(DustTrak) One Healthy Person	-	-	-	-	-	AM[1] 15.1 (7.7)	AM[1] 13.5 (8.3)	14.2 (11.7)	AM[1] 14.9 (8.0)
Pekey et al. (2010)	Kocaeli City, Turkey	15 homes	2006	-	-	-	-	S[3] 29.8 W[4] 24.4	S[3] 23.5 W[4] 21.8	-	-
Lai et al. (2004)	Oxford, UK	(48h)	1998-2000	25-55 (50)	-	-	-	-	-	-	GM[2] 13.2

AM[1]=Arithmetic Mean, GM[2]=Geometric Mean, S[3]=Summer, W[4]=Winter

Few studies have been conducted to explore the relationship between indoor, outdoor and personal exposure within developing countries. A study conducted in Bangkok, Thailand, suggested that ambient fixed-site monitors were able to capture the daily variation of indoor PM levels and even personal exposure (Tsai et al., 2000). Another study that was conducted in Santiago, Chile, found that personal exposure of children was strongly associated with indoor and outdoor $PM_{2.5}$ levels. However, correlations for NO_2 were weaker, probably because of the presence of gas cooking stoves in all homes (Rojas-Bracho et al., 2002). A study in four Mexican cities, however, did show that the best predictors of personal nitrogen dioxide exposure were outdoor levels and time spent outdoors, due to the specific characteristics and personal behaviour of the people in these Mexican cities (Ramirez-Aguilar et al., 2002).

2.14.2 Personal exposure and modes of transport

Personal exposures to air pollution in the transport microenvironment are considered as one of the high-exposure periods among various daily activities (Kaur *et al.*, 2007; Karanasiou *et al.*, 2014). Several studies pointed out that people spend an average of 5.2% of their time commuting as shown in Table 2.8 (Jantunen *et al.*, 1998; Burke *et al.*, 2001; Marino M, 2002; Lai *et al.*, 2004; Wu *et al.*, 2005; Kim *et al.*, 2006; Johannesson *et al.*, 2007; Braniš and Kolomazníková, 2010; Mohammadyan, 2011). This is also confirmed by the WHO report on the health effects of traffic-related air pollution, which showed that individuals spend approximately 1-1.5 hours per day commuting during their regular journeys in many countries (WHO, 2005b). Therefore, individuals may gain a significant contribution to their daily total personal exposure during these regular journeys (Kaur *et al.*, 2007; Karanasiou *et al.*, 2014).

Very few studies related to personal exposure have included comparisons of $PM_{2.5}$ concentrations between different transport modes. The search for my thesis was limited to studies that measured personal exposure to $PM_{2.5}$ when commuting by three different modes: walking, by car and by bus, as they are more relevant to the aims and objectives of this study. Table 2.10 shows a typical example of these studies reporting on $PM_{2.5}$ exposures when commuting by these transit modes. These studies vary in terms of combination of modes of transport studied, types of route and geographical setting (e.g. roadside, background). Some other major factors that potentially influence results, such as weather conditions and different levels of 'background' pollution (i.e. regional and far-travelled air pollution) are not shown in Table 2.10. Most of the identified studies compare only two travel modes. It is important to note that the studies by Kaur and Nieuwenhuijsen (2009), Vallejo et al. (2004), Tsai et al. (2008) and Zuurbier et al. (2010) did not undertake simultaneous monitoring between modes, nor did they cover the same route. Results are therefore specific to each setting and do not represent the ideal basis for comparison. Only one study by Gulliver and Briggs (2007) simultaneously monitored walking and in-car personal exposure to $PM_{2.5}$ on the same route perhaps providing a better basis for drawing conclusions on concentrations of pollutants between the modes of transport studied. Since commuting by walking is a common form of transport globally, it is surprising that limited studies on personal exposure to $PM_{2.5}$ have studied this mode.

Table 2.10: Selected studies that presented personal exposure to $PM_{2.5}$ for different modes of commuting

Author	Location	Period	N. (Age)	Equipment	Modes of Commuting Mean (Median) $PM_{2.5}$ Conc. ($\mu g/m^3$)			Total Transport
					Walk	Car	Bus	
Gulliver and Briggs (2007)	Leicester, UK	Jan-Mar 2005	1-2	Light scattering (OSIRIS & DUSTMATE)	10.9 (11.1)	8.3 (7.2)	-	-
Kaur and Nieuwenhuijsen (2009)	London, UK	Apr-May 2003	4	Gravimetric (HFPS)	(25.3)	(32.4)	(34.1)	(31.4)
Vallejo et al. (2004)	Mexico City	Apr-Aug 2002	40 (21-40)	Light scattering (pDR)	-	(64.2)	(101.7)	-
Tsai et al. (2008)	Taipei, Taiwan	-	2 adults	Gravimetric (DUST-check)	-	22.1	38.5	-
Zuurbier et al. (2010)	Arnhem, Netherlands	2007-2008	(34)	Light scattering (pDR)	-	114.8 (73.6)	68.7 (39.1)	-

2.15 Other Methods of Exposure Models

Recent literature has reported on the use of several innovative techniques designed to increase the accuracy in describing spatial variations in air pollution in relation to population location, in order to better estimate levels of exposure, or reduce estimation uncertainty (Zhang and Lioy, 2002). Jerrett et al (2005) reviewed and assessed existing exposure models for air pollution used in exposure studies (Jerrett *et al.*, 2005). They categorised the exposure methods into six classes depending on their level of complexity: 1) proximity models, 2) dispersion models, 3) land use regression models, 4) interpolation models, 5) integrated meteorological-emission models, and 6) hybrid models (Jerrett *et al.*, 2005). The authors noted that proximity models are relatively crude estimators, because none of the parameters influencing the dispersion process of pollutants are generally considered. Dispersion models require considerable time, resources and expertise that may not be readily available, whereas land use regression models provide comparatively poor temporal resolution and do not reflect seasonality variables well (Briggs *et al.*, 2002; Jerrett *et al.*, 2005). Hybrid models, where two or more of exposure methods are joined together, were recommended as they can provide measurement validation, whilst the weaknesses depend on the combination of models used (Zou *et al.*, 2009). Overall, all these methods are usually combined with some sort of “overlay analysis” in GIS with a dataset containing population data to obtain levels of exposure for individuals or subgroups (Jerrett *et al.*, 2005).

Space-time modelling differs from the previously mentioned methods by additionally incorporating the location of the individuals. The methods described above either apply the location of the population in question as a statistical measure (such as the population density for an area), or the individuals are simply assigned an exposure based on the levels at their place of residence. Neither of these approaches reflects the true exposure well, since most individuals spend most of their waking time elsewhere or, when they are at home, tend to spend most of their time indoors where pollutant levels do not correspond to the outdoor levels most often applied in exposure studies (Briggs, 2005). Numerous exposure studies have attempted to explain the complexity of this problem, and have thus begun to apply time-space models in which the exposure at different

microenvironments, and the time and duration spent in these environments, are taken into account (Briggs, 2005).

An example of this space-time modelling approach is the study carried out by Gulliver and Briggs (2005) which used STEMS model (Space-Time Exposure Modelling System) to simulate the exposures of 50 schoolchildren as they travelled between home and school in Northampton, UK. Each subject provided a time-activity diary with home and school locations; GIS was used to identify the shortest walking route and extract pollution concentrations developed by combining dispersion and traffic models. This model was designed to simulate the exposure of people as they move through a changing air pollution field. The model integrates data on source activity, pollutant dispersion and individual travel behaviour to derive individual or group-level exposures to air pollution during journeys (Gulliver and Briggs, 2005).

In recent years, Bayesian approaches for spatial prediction of air pollution have been developed (Wikle *et al.*, 1998; Kibria *et al.*, 2002; Sahu *et al.*, 2006; Cocchi *et al.*, 2007). These Bayesian models have been further developed by Zidek et al (2007) and Shaddick et al (2008) who presented a two-step Bayesian model that (i) estimates the individual exposure by combining diaries of activities and time spent in each microenvironment and (ii) links the probability distribution of the individual exposure to the values of the health outcome (Zidek *et al.*, 2007) (Shaddick *et al.*, 2008; Blangiardo *et al.*, 2011). More recently, univariate spatio-temporal hierarchical models were proposed to combine monitoring data and the output from a local-scale air pollution model for health risk assessment (Pirani *et al.*, 2014). The main reason for using Bayesian methods is that it enables the combination of different data sources, e.g. monitoring data, modelled outputs and covariates derived from spatial analysis tools such as GIS, to be modelled in a flexible framework which accounts for estimates of the uncertainties associated with the aggregated pollution levels (Sahu, 2012).

Indoor Models of Exposure

There are three major groups of exposure methods that may be used to model exposure to indoor air pollutants: statistical regression models, microenvironmental (ME) models (indoor air quality models based on mass-

balance equations) and computational fluid dynamics (CFD) methods (Chaloulakou *et al.*, 2003; Milner *et al.*, 2011; Shilpa and Lokesh, 2013). Indoor CFD models have been used to model the spatial and temporal variations in indoor pollutant concentrations at an extremely fine scale (typically 0.01 m to 1 m diameter grid cells) (Milner *et al.*, 2011; Shilpa and Lokesh, 2013). However, data input requirements and user expertise for CFD tools may be extensive (Milner *et al.*, 2011; Shilpa and Lokesh, 2013). Therefore, while CFD may be useful as a means for looking at air distribution within the indoor environment at extremely fine spatial and temporal scales, is not considered appropriate for generic population exposure modelling (Milner *et al.*, 2011; Branco *et al.*, 2013; Shilpa and Lokesh, 2013).

A statistical regression model is a technique that seeks to explain air pollutant levels at a certain location using one or more explanatory variables. In this method, linear and nonlinear regression techniques are used to relate indoor exposure to its determinants (Chaloulakou *et al.*, 2003; Shilpa and Lokesh, 2013). The development of a regression model is usually based on monitoring campaigns measuring indoor and outdoor concentrations of air pollutants (Chaloulakou *et al.*, 2003). In addition, regression models are generally applied to model long-term exposures (Milner *et al.*, 2011). For example, Wu *et al.* (2005) used statistical regression to model exposure to $PM_{2.5}$ of asthmatic children in California and found that a multiple linear regression model with fixed-site concentrations as the main predictor had better predictive power (R^2=0.41) than a three microenvironment model (R^2=0.11). Another study by Gauvin *et al.* (2002) also used multiple linear regression methods to model exposure to $PM_{2.5}$ in children in France and observed that 36% of the variance of children $PM_{2.5}$ personal exposure is explained.

Alternatives to the regression approach are Mass-balance models specifically designed to simulate average indoor air pollutant concentration as a function of outdoor concentration, building characteristics and indoor sources (Milner *et al.*, 2011; Shilpa and Lokesh, 2013; Desauziers *et al.*, 2015). The underlying principle of all air pollution mass balance models is that indoor concentrations are determined as a function of key building characteristics, like the infiltration of outdoor pollution into buildings, indoor source strengths and the physical properties of the air pollutants (Milner *et al.*, 2011; Shilpa and Lokesh, 2013). The

advantage of mass balance models is that they are based on physical and chemical principles, thus they represent to some extent the underlying atmospheric processes (Mumovic and Santamouris, 2013). A disadvantage is the large amount of information required for the input parameters. This level of detail on various building characteristics and internal sources is usually not available for epidemiological studies (Milner *et al.*, 2011). A further disadvantage is that mass balance models need to be parameterised and run individually for each building or room, which is time consuming and will limit the size of a study (Mumovic and Santamouris, 2013).

2.16 Exposure Misclassification

Exposure misclassification is a well-recognized inherent limitation of many epidemiologic studies on environment and health (Blair *et al.*, 2007; White *et al.*, 2008). Epidemiological studies of the health effects of air pollution have estimated exposure using a variety of exposure assessment approaches. These approaches range from basic descriptions of geographic backgrounds to detailed assessments of personal exposures based on measurements made with small monitoring devices (Rom and Markowitz, 2007). Estimates of population exposure to ambient air pollution have traditionally relied on concentrations measured at fixed-site monitors as a surrogate for personal exposure (Michelle *et al.*, 2004; Oezkaynak *et al.*, 2013). This method assumes that all individuals within the community will experience the same exposure and ignores differences in time-activity patterns, indoor and outdoor concentrations, and sub spatial variability (Michelle *et al.*, 2004; Rom and Markowitz, 2007). As such, this approach is likely to introduce exposure misclassification, especially for pollutants that are spatially heterogeneous, such as those associated with traffic emissions (e.g., carbon monoxide and nitrogen oxides) (Oezkaynak *et al.*, 2013).

Fixed-site monitors do not account for a multitude of factors that influence personal exposure to air pollutants, such as exposures near emissions sources, variations in infiltration of outdoor/indoor environments, indoor sources of air pollution, and the time individuals spend away from their home or in other near-source microenvironments (e.g., in vehicles). (Rom and Markowitz, 2007; Oezkaynak *et al.*, 2013). Moreover, the dose of an inhaled pollutant depends on the rate and pattern of breathing and for inhaled particles (Rom and Markowitz, 2007).

The extent of exposure misclassification further depends on the spatial and temporal aspects of the design, as well as the aim of each study (Rom and Markowitz, 2007). For example, the degree to which refined exposure estimates (e.g. including location of individuals in the exposure assessment) influence predictions of health outcomes (e.g., long-term vs short-term or acute vs chronic exposure effects), depends on study-specific characteristics including epidemiological study design (e.g., time-series vs cohort). In time-series studies for example only population-level exposure estimates are needed, as the focus

is entirely on quantifying a temporal effect. Whereas in individual-level studies, such as cohort studies, exposure estimates for individuals are required (Oezkaynak *et al.*, 2013).

Other approaches have been developed to consider and potentially correct for the impact of exposure misclassification, including the use of personal monitors to estimate individual exposure and to account for daily time-activity patterns in different microenvironments (Rom and Markowitz, 2007). However, personal monitors have their own set of limitations, such as weather condition is altered by wearing a monitor, the feasibility of obtaining long-term exposures, and burden on study subjects (Rom and Markowitz, 2007). A critical assessment of exposure misclassification and modelling issues for different sampling designs, including recommended data collection and novel statistical methods, is still lacking in the literature (Dominici *et al.*, 2003b; Oezkaynak *et al.*, 2013).

2.17 Chapter Summary

In this chapter, the relationships between air pollution and health, and definitions of relevant terms were described. An overview of air pollution standards was provided, and despite continuous improvements in air quality in large parts of the world over the past decades, poor air quality remains a challenge in many urban areas, particularly in emerging and developing countries (Michelle *et al.*, 2004).

The health effects of air pollution can result from a variety of air pollutants depending on the duration and frequency of exposure; and, with respect to the particulate pollutants, the size of pollutants (WHO, 1999). Some health effects are related to long-term exposure, others to short-term exposure (WHO, 1999), but together, indoor and outdoor air pollution is now recognised as the largest single global environmental risk to health (WHO, 2013; WHO, 2015).

The asthma prevalence in Europe, Saudi Arabia and Gulf Countries were reviewed in the current study. There is increasing evidence and awareness about the relation between asthma development or exacerbation and indoor and outdoor environmental exposure (WHO, 2006b).

Epidemiological studies of air pollution fall into four types: time series; case crossover; panel and cohort studies. The time series, case-crossover and panel studies are more appropriate for acute effects estimation while the cohort studies are used for acute and chronic effects combined (Dominici *et al.*, 2003a; Peng and Dominici, 2008; Tadano *et al.*, 2012). The time-series approach assesses the effects of short-term changes in air pollution on acute health outcomes by estimating associations between day-to-day variations in both air pollution levels and in mortality and morbidity counts (Michelle *et al.*, 2004; Chen and Kan, 2008; Peng and Dominici, 2008). To date, there has been limited research in the Middle East on the relationship between daily air pollution levels and health, and no studies on air pollution and asthma-related emergency department visits using time-series analysis. There remains a need for studies in cities of developing countries, where levels of air pollution and meteorological conditions are different from North America and Western Europe (Gouveia and Fletcher, 2000).

A general discussion of human exposure to air pollution was described. Time-activity patterns can significantly influence air pollution exposure (WHO, 2006a). In the recent past, technology has greatly improved, making it possible to conduct detailed personal monitoring in both indoor and outdoor environments. This has allowed more precise estimation of personal exposure, which is important since people spend a large part of their time in indoor environments. While the existing literature on time-activity patterns and microenvironment exposures for populations in North America and Western Europe keeps on growing, little research on this topic has been carried out in the Middle East. To the best of my knowledge, no study on microenvironments concentrations and time-activity patterns has been conducted in an industrial city in Saudi Arabia. Such a study is necessary to fill important gaps in our understanding of the influence of time-activity patterns and microenvironments on personal exposure in such a setting.

2.18 Thesis Overview

The thesis is organized into eight main chapters:

Chapter One, “Introduction”, provides the background to my thesis, and outlines the aim and objectives.

Chapter Two, “Scientific Background” comprises two main topics. The first topic describes the relationship between air pollution and health, and then focuses on asthma prevalence in the Middle East and in European countries. In addition, it provides a review of the previous and current time-series analyses that were used to study the relationship between air pollution levels and asthma-related emergency department visits. The second topic is a general discussion of approaches used to assess human exposure to air pollution. Here the focus is on microenvironments and time-activity patterns, and their influence on personal exposure to air pollution. Finally, the relationship between personal exposure to air pollution and indoor as well as outdoor concentrations is presented.

Chapter Three, “Materials and Methods”, presents the research protocol, methods and tools of the study. The first section gives information about the general study design and location. The second section provides details of the first phase of the study, a time-series analysis to investigate the statistical association between air pollution and asthma-related emergency department visits, followed by the second phase, collection and analysis of data on microenvironment and time-activity pattern. The methods of subject recruitment, research tools, and data analysis are described for each phase.

Chapter Four, “Results of Time-Series Analysis”, includes the descriptive analysis of air pollutants, meteorological variables and asthma-related emergency department visits. This is followed by detailed results of the single-pollutant model and the multi-pollutant model, as well as relative risk analysis.

Chapter Five, “Results of Time-Activity Patterns and Microenvironments”, presents the descriptive results of the questionnaire and personal exposure analysis. This is followed by detailed results of students’ activities and time spent in each microenvironment. In addition, it presents personal exposures in different microenvironments. This is followed by an analysis of the variability of $PM_{2.5,}$

using data from a fixed-site monitor and other variables to show the prediction power of these proxy variables. The last section presents the relationship of AEDv and ambient PM2.5 levels converted from personal monitoring campaign

Chapter Six “Discussion of Time-Series Analysis” discusses the findings under three main headings, the first being air quality exceedance in Al Jubail industrial City. The second is the relative risk of time-series analysis. This is followed by a discussion of the limitations and strengths of the time-series analysis.

Chapter Seven, “Discussion of Time-Activity Patterns and Microenvironments”, discusses the findings under four main headings: total time-spent at indoors, outdoors and commuting; personal exposure in different microenvironments, and; comparison between personal and fixed-site monitoring of $PM_{2.5}$ exposure levels. Finally, a discussion of the limitations and strengths of the study is presented.

Chapter Eight, “Conclusion” and Recommendations”, provides an overall conclusion of the thesis and recommendations for future studies and policy makers.

Finally, the references and appendices are provided at the end of the thesis.

CHAPTER THREE

Materials and Methods

Chapter:3 Materials and Methods

3.1 Introduction

This chapter gives information about the general study design and location. The next section provides details of the first phase of the study, a time-series analysis to investigate the statistical association between air pollution and asthma-related visits to emergency department, followed by the second phase, the collection and analysis of microenvironment and time-activity pattern data. The methods of subject recruitment, research tools and data analysis are described for each phase.

3.2 General Study Design

In this study, a time-series analysis was undertaken using existing routinely collected health data from medical record for asthmatic patients who visited the Emergency Department in Al Jubail for the period between 2007 and 2011, and was linked to ambient air pollution data from fixed-site monitoring stations. The aim was to investigate the relationship between exposure to air pollution and Emergency Department visits. Fieldwork was then undertaken in Al Jubail to explore the relationship between time-activity patterns, microenvironments and drivers of personal exposure in students aged between 16 and 18 years.

3.3 Study Location

This study was set in Al Jubail Industrial City, which is located in the Eastern Province. The Eastern province is the largest province in Saudi Arabia. It is located to the east of the country on the Arabian Gulf coast, and has land borders with Kuwait, Bahrain, Qatar, the United Arab Emirates, Oman, and Yemen. The Kingdom's main oil and gas fields, onshore and offshore, are mostly located in the Eastern Province. Figure 3.1 shows Saudi Arabia and the location of the Eastern province (RCJY, 2009).

In 1975, Al Jubail Industrial City was designated as a site for a new industrial city by the Saudi government, and has seen a rapid expansion and industrialization since. The Seventh Census Report on Al Jubail Industrial City, prepared in 2009, gives a resident population of 105,367 (RCJY, 2009). Figure 3.2 illustrates the population by sex and age group, and indicates that there is male bias in the working-age population since industrial workers are mainly males.

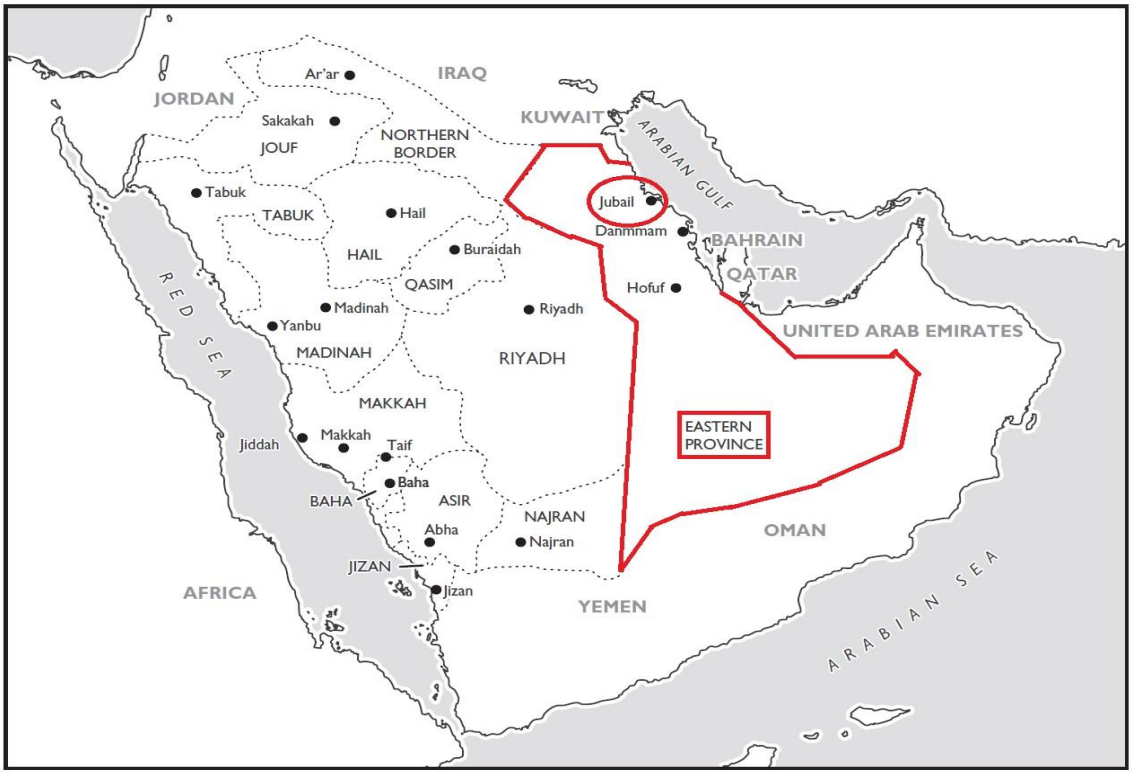


Figure 3.1: Al Jubail Industrial City, Eastern Province, Saudi Arabia
Source: Adapted from (Vincent, 2008)

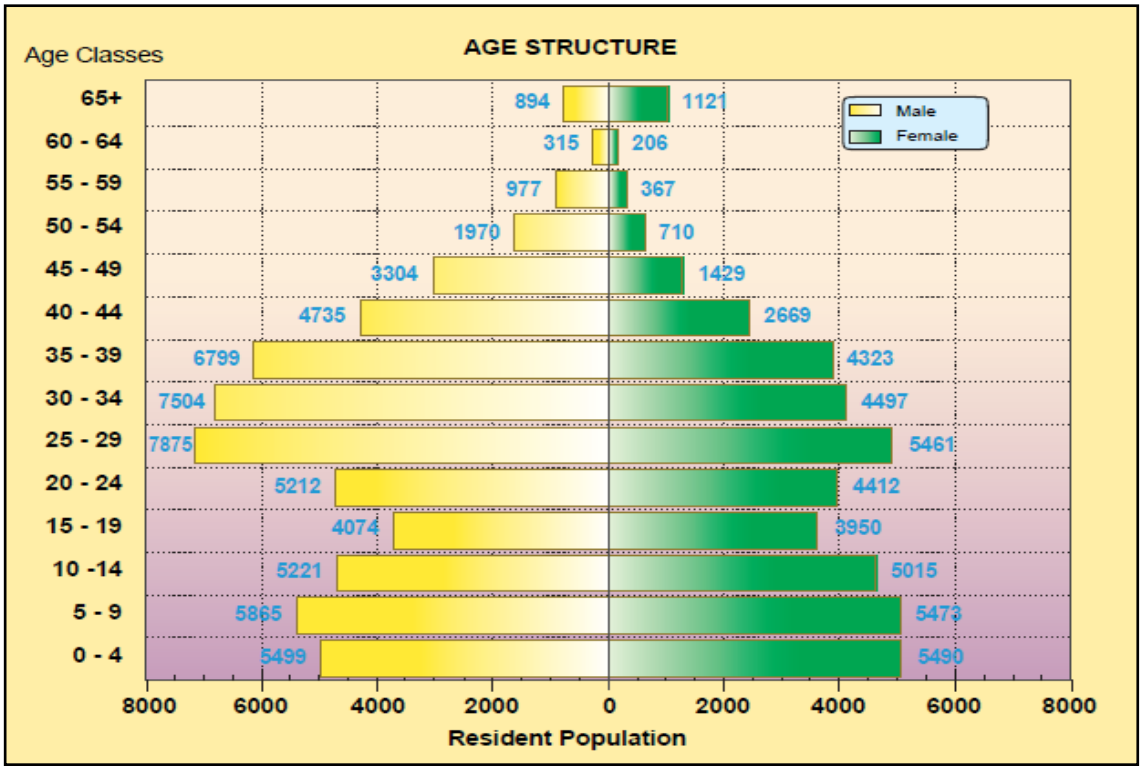


Figure 3.2: Residential population by sex and age group, in Al Jubail Industrial City

Source: (RCJY, 2009)

3.4 Study Phases and Methods

3.4.1 Phase One: Time-series analysis

The main objective of this phase is to investigate the statistical association between air pollution and asthma-related hospital admissions in Al Jubail Industrial City in Saudi Arabia. Time-series analysis was used to evaluate the health effects of air pollution by assessing associations between daily variations (short-term) of air pollution levels and health events (such asthma emergency department visits) (Tobías *et al.*, 1999; WHO, 2005a; Wilkinson, 2006; Tadano *et al.*, 2012). This involved collecting data on asthma emergency department visits, air pollution data and meteorological data from Al Jubail Industrial City.

3.4.1.1 *Data on asthma-related visits to emergency department*

The health data for the time-series analysis were collected from the Royal Commission Health Service Program in Al Jubail Industrial City, Saudi Arabia, which is responsible for the Royal Commission Hospital. The health data are stored in a central-computerised database in the Medical Records Department. Relevant records were identified based on a discharge diagnosis of asthma using the International Classification for Diseases, 9th revision (ICD-9 code 493). The health data included the date and time of admission and discharge, identification number for each patient, sex, age and nationality. The data obtained were on asthma-related visits to emergency department for the period between 1st January 2007 and 31st December 2011, for all ages. A total number of 8434 daily asthma-related emergency department visits (AEDv) occurred during the study period with no missing or duplicate values. Since the present study is interested in overall AEDv, all visits were counted as independent, so the dataset included repeat visits made by the same patient to the same emergency department.

3.4.1.2 *Air pollution and meteorological data*

Hourly monitor-based data for the period of study were obtained from the Royal Commission Environmental Control Department in Al Jubail, Saudi Arabia. The air pollution and meteorological data were collected from seven fixed-site monitoring stations as shown in Figure 3.3. The monitored pollutants include: PM_{10}, $PM_{2.5}$, SO_2, NO_2 and CO. These same stations also measured meteorological conditions including temperature (T), relative humidity (RH), wind speed (WS) and wind direction (WD). Data from these stations are transmitted wirelessly after every 5 minutes, 24 hours a day to a central computer and are then stored in a central-computerized database in the Royal Commission Environmental Control Department in Al Jubail. The data obtained were for the period between 1st January 2007 and 31st December 2011.

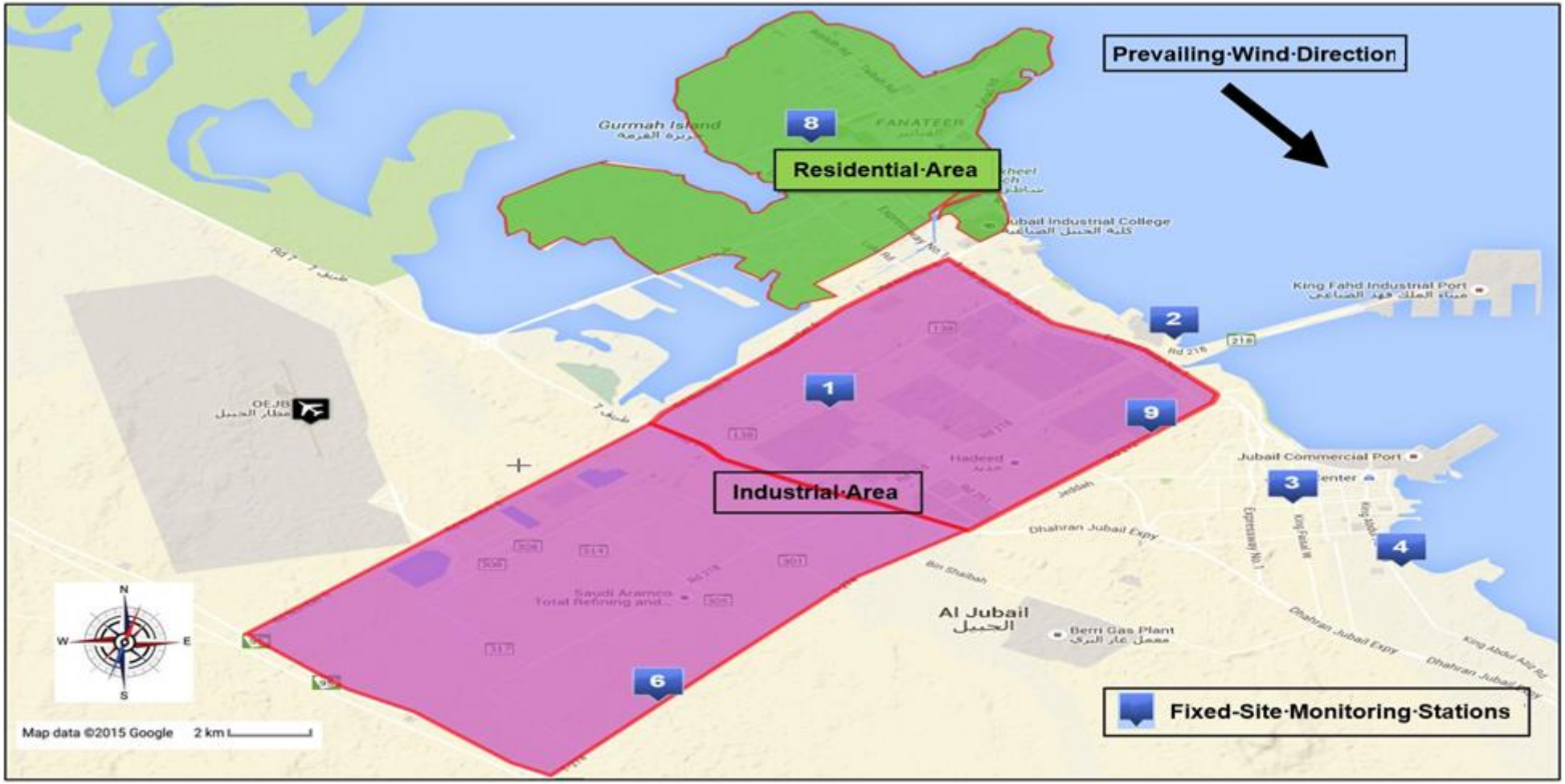


Figure 3.3: Al Jubail industrial area, community area and fixed-site monitoring stations
Source: Map data Google, 2015

3.4.1.3 *Selection of fixed-site monitoring station*

The residential community is located in the north of the industrial zone, and the prevailing wind blows from the north-west as shown earlier in Figure 3-3, the locations of the fixed-site monitoring stations, as well as the close proximity of Jubail industrial area to the residential community area (Map data Google, 2015). There is only one fixed-site monitoring station located within the community area (site 8), which called the residential fixed-site, while the other fixed-site monitors are located in the industrial zone (sites 1, 2, 6 and 9) and two sites (3 and 4) to the south far from both the residential and industrial areas.

The residential fixed-site monitoring station is considered to represent air quality in the community area and has coordinates of 27° 7'54.03"N 49°31'57.02"E (Figure 3-3). The residential fixed-site monitoring station is influenced by any nearby direct sources, and is about 50 meters away from the nearest motorway. Accordingly, the residential fixed-site was selected as fulfilling the needs of the current study by reflecting the levels of air pollutants of the community area in Jubail Industrial City. However, a critical assessment was carried out to further justify the choice of residential fixed-site monitoring station. Descriptive analysis was used to assess the correlation between air pollution data from all fixed-site monitoring stations, and availability of air pollution data was also considered. T-tests were used to identify any significant differences between air pollutant levels obtained from the residential fixed-site and the other fixed-site monitoring stations.

3.4.1.4 *Wind direction Analysis*

Wind direction analysis was used to look at the variability in air pollution levels and AEDv over time in relation to wind direction, as well as to assess the choice of fixed-site monitoring station. Using the Openair toolset (Carslaw, 2015), the available meteorological data was interrogated using the windRose function and polarFreq function in R software (R.Core.Team, 2014). The windRose function summarises meteorological data to show how wind direction/speed conditions vary during the study period. The data were summarised by wind direction, typically by 22.5 degrees and by different wind speed categories and the percentage of time that the wind blew from a certain angle and wind speed range (Carslaw, 2015). The polarFreq function was used to better understand which wind directions contributed most to the overall mean pollution concentrations. By weighting the concentrations by the frequency of occasions, the wind blows from a certain direction, the conditions that dominate the overall mean concentrations can be assessed (Carslaw, 2015).

3.4.1.5 *Air quality assessment and quality assurance of the data*

The first step taken in the analysis was to subject all the data to a thorough quality assurance process as proposed by the protocol described by Katsouyanni *et al.* (1996) and (Schwartz *et al.*, 1996), who set out the methods as part of the APHEA project in order to ensure maximum comparability of results. The pollution and meteorological data were analysed, and those days for which data did not meet the completeness criteria were removed. The criteria were set at ≥75% of the hourly values per day to meet with Air Quality Standards (AQS) as proposed by the protocol (Katsouyanni *et al.*, 1996; WHO, 1999). There were still a few missing values in the pollution and meteorological data for some days. Since the time-series analysis required a complete dataset, the missing values from the community fixed-site station (number 8) were estimated using the available measurements in the other fixed-site monitoring stations in Al Jubail Industrial City on the same day. The method for imputing missing values followed the same protocol described by Katsouyanni *et al.* (1996) and (Schwartz *et al.*, 1996). The daily missing value was replaced with the mean level of the remaining stations multiplied by a correction factor, which was the ratio of the seasonal mean (three months) for the missing station to the corresponding seasonal mean for the remaining stations on that particular day (Katsouyanni *et al.*, 1996). The same method has been used for studies outside the remit of the APHEA project, for example,Tob *et al.* (1999), Wong *et al.* (1999), Ko *et al.* (2007) and Samoli *et al.* (2011).

The second step in the analysis was to use hourly air pollution data to calculate appropriate annual and daily mean values for comparison with Air Quality Standards (AQS). This affords the possibility of comparing the effects of those pollutants which exceed the annually and daily AQS with those that do not.

3.4.1.6 *Time-series analysis*

Regression models have commonly been used in time-series analyses to assess the association between one or more explanatory variables (independent, predictor variables or covariates) and a single response variable (dependent or predicted variable) (Dominici *et al.*, 2003a; Peng *et al.*, 2006; Tadano *et al.*, 2012).

Generalized Linear Models (GLM) with parametric splines and Generalized Additive Models (GAM) with non-parametric splines are commonly applied in time-series analyses of air pollution impacts on human health due to the non-linearity of the response variable (Tadano *et al.*, 2012). However, Peng *et al.* (2006) found that fully parametric and non-parametric methods perform well, with neither preferred over the other, so this does not affect the quality of the analysis.

In recent studies on impacts of air pollution on human health, where non-negative count data are used as the response variable, the GLM with Poisson regression is broadly applied (Dominici *et al.*, 2002; Tadano *et al.*, 2012). One feature of the GLM with Poisson regression is that even if all the explanatory variables are known and measured without error, there would still be considerable unexplained variability in the response variable. This is a result of the fact that even if the response variable is more precise, the Poisson process ensures stochastic variability around that expected count (Tadano *et al.*, 2012).

In the present study, the time-series analysis was conducted using Generalized Linear Models (GLM) with Poisson regression. The steps used to apply the GLM with Poisson regression were adopted from the method described by Tadano *et al.* (2012). There are five steps that need to be followed in order to fit GLM with Poisson regression, namely: temporal trends adjustment, number of degrees of freedom test, goodness of fit analysis, single/multiple pollutant models and relative risk (RR) analysis.

Step One: Long and short-term trend adjustment

This study considered both long-term trends (including seasonality) and short-term trends (including day of the week (dow) and holiday indicator (H)). The day of the week variable was considered as a categorical variable that varies from one to seven, starting on Sundays. The holiday indicator was adjusted for by adding a dichotomous variable (1 = holidays; 0 = workdays). To apply the natural cubic spline (ns) in a generalized linear model (GLM) with Poisson regression, an explanatory variable for the day of study (dos) was added to the model to consider seasonality, consisting of values from 1 to 1826, comprising of data from the five years from 1st January 2007 to 31st December 2011.

The GLM with Poisson regression was then applied in R software (R.Core.Team, 2014) with the following equation:

$$\boldsymbol{m.name} < -\boldsymbol{glm} \begin{pmatrix} \boldsymbol{AEDv} \sim \boldsymbol{ns}(\boldsymbol{dos.df}) \\ +\boldsymbol{as.factor}(\boldsymbol{dow}) \\ +\boldsymbol{as.factor}(\boldsymbol{H}) + \boldsymbol{T} + \boldsymbol{RH} \\ +(\boldsymbol{X}), \boldsymbol{database.name}, \boldsymbol{family} \\ = \boldsymbol{poisson}, \boldsymbol{na.action} = \boldsymbol{na.omit} \end{pmatrix}$$

Equation 2

Where m.name is the name given to the analysis; ns is natural cubic spline; *df* refers to degrees of freedom; *X* is the pollutant (one pollutant variable in the single pollutant model or two or more pollutant variable terms in the multi-pollutant model); database.name is the name given to the database file (Tadano *et al.*, 2012).

Step Two: The number of degree of freedom test

To apply a time-series analysis, one important decision is the degrees of freedom (*df*) to be considered in the natural cubic spline of days of the study (Peng *et al.*, 2006; Tadano *et al.*, 2012). The commonly used values range from four to eight degrees of freedom per year of data (Peng *et al.*, 2006; Tadano *et al.*, 2012). To decide which one to use, five analyses were made using four, five, six, seven and eight degrees of freedom in the model and results compared using the Akaike Information Criterion (AIC) (Tadano *et al.*, 2012). The Akaike Information Criterion (AIC) is a measure of the relative quality of statistical models for a given set of data and provides a means for model selection. The smaller the AIC, the better is the model. The AIC is automatically calculated in R software when applying the GLM algorithm and is calculated by the following Equation 3 :

$$\boxed{\boldsymbol{AIC = 2l(b) + 2(df)\,\hat{\phi}}}$$

Equation 3

Where $l(b)$ = maximum log-likelihood value for the complete model; *df* = degrees of freedom of the model and $\hat{\phi}$ = estimated dispersion parameter (Peng *et al.*, 2006; Tadano *et al.*, 2012).

As shown in Table 3.1, the model with seven degrees of freedom per year of data had the smallest AIC and therefore best fits the data.

Table 3.1: Comparison of models using the AIC with different numbers of degree of freedom (*df*) for seasonality adjustment

Number of *df* per year	PM_{10}	$PM_{2.5}$	SO_2	NO_2	CO
4	8844.5	8836.1	8864.1	8864.1	8863.3
5	8841.6	8833.1	8860.9	8861.0	8859.8
6	8819.9	8812.5	8840.4	8841.3	8839.2
#7	**8786.6**	**8779.6**	**8793.0**	**8805.6**	**8804.6**
8	8813.1	8806.1	8819.6	8832.1	8831.1

#=Best fits

Step Three: Partial autocorrelation functions

The short-term trends (days of the week (dow) and holiday indicator (H)) can lead to autocorrelation between data from one day and the previous days. One way to analyse this time trend is plotting the partial autocorrelation function (ACF) against lag days (Peng *et al.*, 2006; Tadano *et al.*, 2012).

In the Partial ACF plot, the residuals should be as small as possible, ranging from $-2n^{-1/2}$ and $2n^{-1/2}$ (dashed lines) as shown in Figure 3.4 and Figure 3.5. In epidemiological studies of air pollution, the important autocorrelations are those occurring in the first five days, which are usually caused by a decrease of health outcomes at weekends and holidays (Peng *et al.*, 2006; Tadano *et al.*, 2012). If the database has autocorrelations, then the model should consider them by including the residuals in the model. In R software (R.Core.Team, 2014), the residuals to be included are the working residuals. These residuals are returned when extracting the residuals component directly from the glm command in R software. In this study, the number of observations (n) is equal to 1826, so the lines in Partial ACF plot out of the range (-0.047 to 0.047), indicating a strong autocorrelation between data from one day and previous days.

Figure 3.4 shows the Partial ACF plot against lag days for the model with seven degrees of freedom with no residual inclusion, which indicates autocorrelations for the first five lag days. To adjust for this autocorrelation, it is necessary to include the residuals for these lag days in the model. The Partial ACF plot including residuals for Figure 3.5 shows no autocorrelation between data for the first 7 days, indicating that it is the best fitting model.

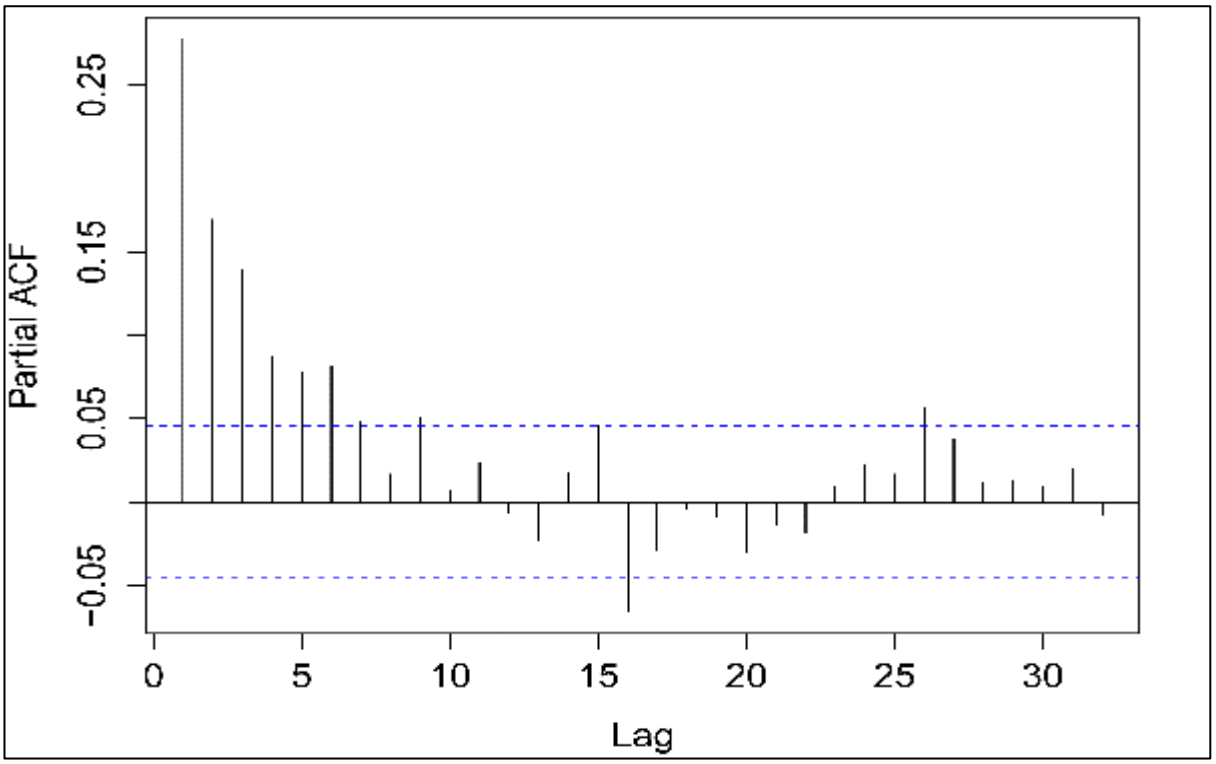


Figure 3.4: Partial ACF plot against lag days with no residuals included

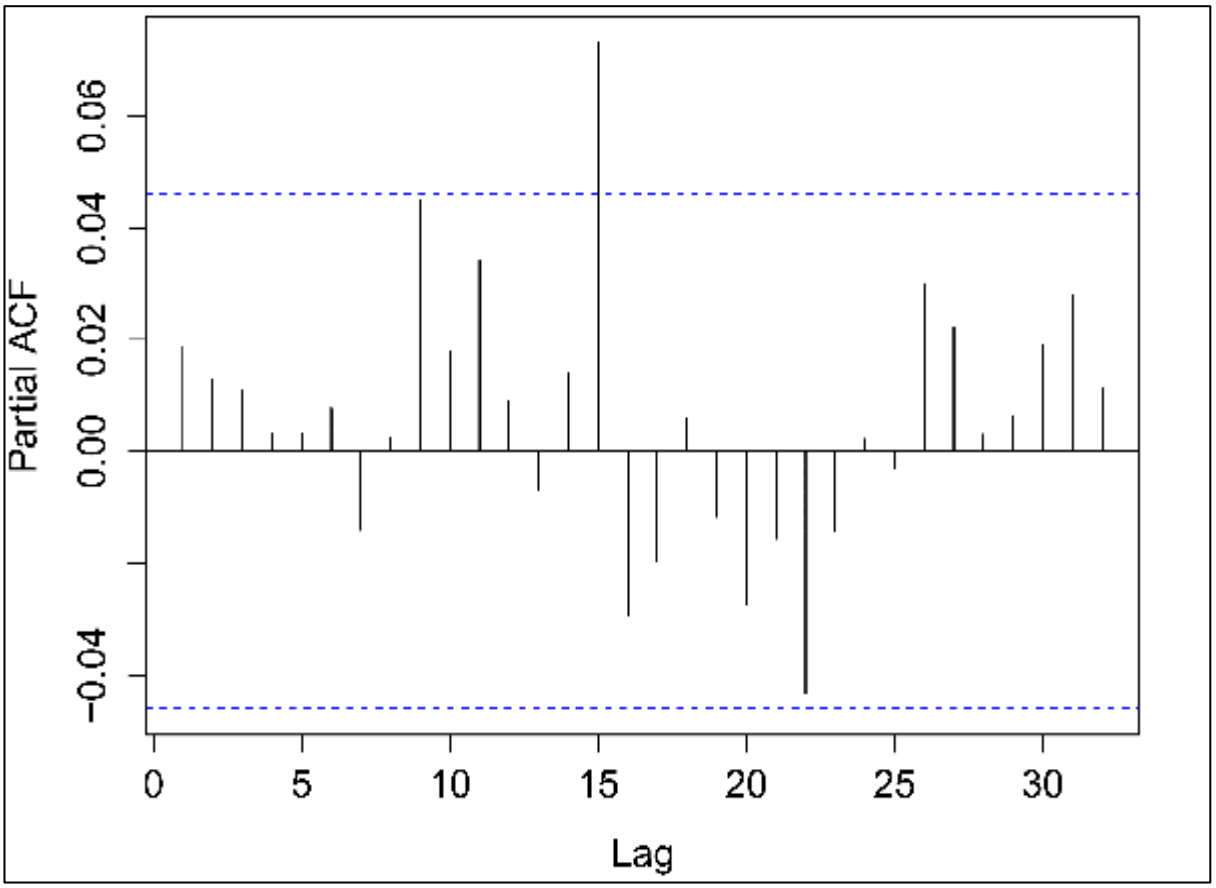


Figure 3.5: Partial ACF plot against lag days including residuals

Step Four: Single and multiple pollutant models

After adjusting the GLM with Poisson regression, including all the time trends and explanatory variables, and choosing the degrees of freedom (*df*) that best fits the data, the fitted model was tested using the pseudo (R^2) and the chi-squared (X^2) statistic to ensure that it is the best one to be applied to the single pollutant model (Peng *et al.*, 2006; Tadano *et al.*, 2012). The goodness of fit of the analyses from no lag to seven lag days is shown in Appendix G, Results of time series analysis (Tables 9-1 to 9-5 A-without residual and, B-with residuals). All of them (from no lag to seven lag days) indicated the need for residual inclusion for the first seven lag days. The models with residual inclusion did not show autocorrelations. After that, multi-pollutant models were used, where more than one pollutant showed a significant positive relationship in the single-pollutant models, and the lag that had the strongest effect was tested. (Katsouyanni *et al.*, 1996).

Multi-pollutant models used the same basic steps as the single-pollutant model (steps one, two, three and four in this chapter, section 3.3.1.6), with the inclusion of two or more pollutant variable terms (Katsouyanni *et al.*, 1996). Pollutants that were significant in the single pollutant analysis and the lag that had the strongest univariate effect were tested, using GLM with Poisson regression applied in R software with the following Equation 4:

$$m.name <- glm\left(\begin{array}{c} AEDv \sim ns(dos.df) \\ +as.factor(dow) \\ +as.factor(H) + T + RH \\ +(X1) + (X2), database.name, family \\ = poisson, na.action = na.omit \end{array}\right)$$

Equation 4

Where *m.name* is the name given to the analysis; *ns* is natural cubic spline; *df* refers to degrees of freedom; *X1 and X2* is pollutant variable terms; *database.name* is the name given to the database file (Tadano *et al.*, 2012).

Step Five: Relative Risk (RR) analysis

The relative risks of asthma-related emergency department visits for the single and multiple pollutant models considering the best fitting model were calculated. The results were expressed as per cent increases with 95% confidence intervals (95% CI) in daily asthma-related emergency department visits with each increment of inter-quartile range (IQR) change of each pollutant. The RR, its standard error and 95% confidence interval (CI) can be calculated from a 2x2 table according to Altman (1990) (See Table 3.2) . The expression that represents it is given in equation (Tadano *et al.*, 2012).

$$\boldsymbol{RR(x) = e^{0.000582x}}$$

Equation 5

Where "*X"* is the data point and "*e"* is the exp function (Tadano *et al.*, 2012).

Table 3.2: General representation of the result as a 2x2 table

	Group 1	**Group 2**	**Total**
Outcome	a	b	a+b
Present	c	d	c+d
Total	a+c	b+d	a+b+c+d

Table 3.2 shows the general layout of the 2x2 table. The RR is given by

$$\boldsymbol{RR = \frac{a/(a+b)}{c/(c+d)}}$$

With the Standard Error (SE) of the log RR being

$$\mathbf{SE\{ln(RR)\}} = \sqrt{\frac{1}{a} + \frac{1}{c} - \frac{1}{a+b} - \frac{1}{c+d}}$$

The 95% CI of the RR is computed as the antilogarithm (exp function) of the two confidence limits computed below

$$\mathbf{95\%\ CI = exp\ (ln(RR) - 1.96 \times SE\{ln(RR)\})\ to}$$

$$\mathbf{exp\ (ln(RR) + 1.96 \times SE\{ln(RR)\})}$$

3.4.2 Phase two: Microenvironment data

For this phase, the following sets of data were collected from 27 students in Al Jubail Industrial City: 24-hour data on personal $PM_{2.5}$ exposure, Global Positioning System (GPS) data and time-activity diary data. These data have been used to identify factors that influence personal exposure to $PM_{2.5}$, and to estimate exposure error introduced by using data from fixed-site monitoring stations as a proxy for personal exposure.

3.4.2.1 *Study population and design*

The specific population selected for sampling in this study survey consisted of male non-smoking students who attend public high school in the middle of Al Jubail Industrial City, Saudi Arabia. Due to political, religious and cultural reasons, female students study in separate schools from their male counterparts. Therefore, female students could not be reached by the researcher and are excluded in this study. The majority of the students are usually aged between 16 and 18 years. This age group has been selected to reflect students who are likely to have relatively well established travel patterns and able to adopt a degree of travel independence. In addition, this age range might be able to deal well with research devices, which might be cumbersome for younger age groups.

The study design was not intended to recruit a representative selection of the population, but rather to identify students' activity patterns and drivers of personal exposure in different microenvironments.

3.4.2.2 *Study procedures and recruitment*

Twenty-seven students aged between 16-18 years were recruited from Al Ahasa Secondary School, which is located in the middle of Al Jubail Industrial City. This is the final number who agreed to participate at this study. The students were selected randomly from the school. The students were divided into three groups of 10 and the researcher met with each group to give a short presentation to explain the aims and participants' role in this project. There was plenty of time for questions and answers at the end of each presentation. In addition, the students were asked to take the information sheet with them and to read it with their parents. If they then agreed to participate in the study, the students were required to sign the consent form. The parents were also required to counter-sign

on the back before any data were collected. A flow diagram explaining the recruitment process is provided in Figure 3.6. The fieldwork was planned to be undertaken over a period of 12 weeks, from February to May 2012.

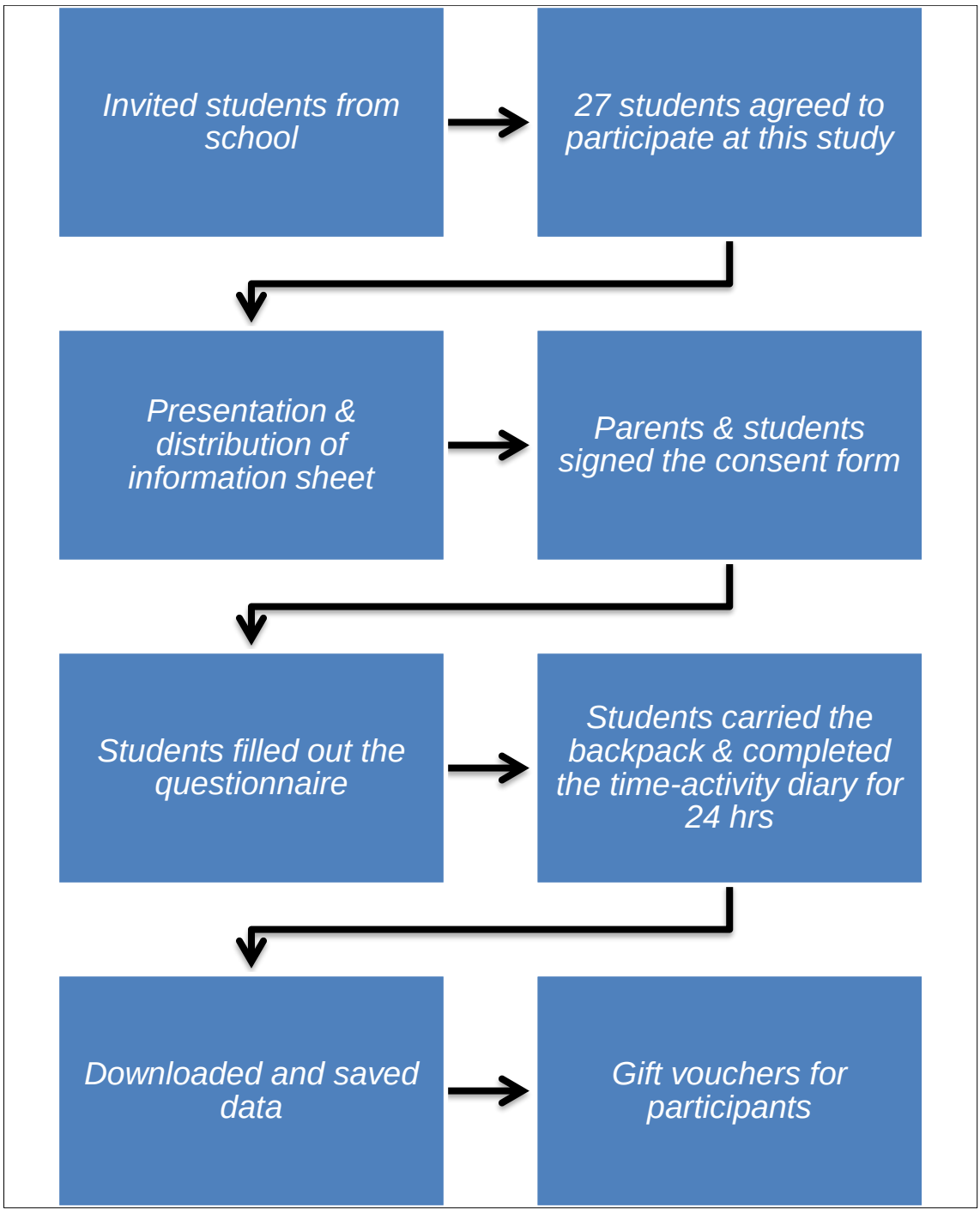


Figure 3.6: Students recruitment process

3.4.2.3 *GPS, time-activity dairy and questionnaire*

A small GPS device was used along with time-activity diary to record the detailed movements of students and the time spent in important microenvironments (including travel, outdoor, at school/work, at home, and at other locations) for over a 24-hour period. The GPS device was set to 'log the participants' location after every 5 seconds, at a geographic resolution of 10 metres, over the 24-hour study period. These data were stored on the device for subsequent download and mapping. The time activity diary required each participant to record various activities and locations that may have an effect on personal exposure, such as activity patterns (time spent indoor/outdoor, travel), modes of transport (car, bus and taxi), recreational activities (jogging, shopping) at 15 minutes intervals. Participants were also asked to note specific exposure sources, such as tobacco smoke, gas cookers and open fires. In addition, a questionnaire was used to collect details of house characteristics, lifestyle, transport and potential exposures (i.e. cooking, heating) from each participant to identify additional factors that may influence time-activity patterns, exposure to specific microenvironment and personal exposures. A personal and home characteristics questionnaire was designed to collect information on house characteristics, lifestyle, transport and potential exposures (i.e. cooking, heating) from each participant to identify factors that may influence time-activity patterns, microenvironment and personal exposures.

3.4.2.4 *Information sheet and consent form*

I developed a participant information sheet for this study to explain the aims and objectives of this study and the role of participant before and after taking part. I also developed a consent form for participants to read and sign before taking part in this study. All these documents and forms were written in English and translated to Arabic (See Appendix B, Questionnaire, Time-Activity Diary, Information Sheet & Consent Form in English and Arabic Forms**).**

3.4.2.5 *Pre-pilot study and testing the equipment*

A pre-pilot study was undertaken in Newcastle to develop the methodology and test the questionnaire and equipment to be used in the fieldwork before travelling to collect data in Saudi Arabia. This included testing three different GPS devices (Qstarz, EasilyShow and SGSII) to determine the accuracy of location and strength of signal (See Appendix C, Comparing three GPS devices (EasilyShow, Qstarz and SGSII)). In addition, I tested the SidePak AM510 Personal Aerosol Monitor, to measure $PM_{2.5}$ levels in different microenvironments. The questionnaire was pre-piloted on friends whose first language is Arabic. This allowed me to test the questionnaire language, which I translated from English to Arabic, and then translated back to English.

3.4.2.6 *Personal air monitor data*

Personal exposure to $PM_{2.5}$ was monitored in this study by using the SidePak™ (TSI Inc., Model AM510, Shoreview, MN, USA) portable (0.46 kg, 106 x 92 x70 mm) (TSI, 2012), which was chosen because of its level of precision and durability, given the budget constraints. This device has battery-operated laser photometers that express airborne particle mass concentration in mg/m^3, and it was used in other similar studies on personal exposure (Braniš and Kolomazníková, 2010; Borgini *et al.*, 2011; Lim *et al.*, 2012).

The Figure 3.7 shows a schematic process of detection of the SidePak™. A diaphragm pump provides a continuous aerosol flow stream drown though the sensing chamber. The aerosol is then flow-passed through the sensing chamber. One section of the aerosol stream is illuminated with a small beam of laser light. Particles in the aerosol stream scatter light in all directions. A lens at 90° to both the aerosol stream and laser beam collects some of the scattered light and focuses it onto a photodetector. The sensing volume of the SidePak™ AM510 is constant and is defined by the intersection of the aerosol stream and the laser beam. Mass is determined from the intensity of light scattered by the aerosol within the fixed sensing volume. Since the sensing volume is known, the information can be easily converted by the SidePak™ AM510 microprocessor to units of mass per unit volume (mg/m^3) (TSI, 2012).

The SidePak™ (AM510) is calibrated at the factory against a gravimetric reference using the respirable fraction of standard ISO 12103-1, A1 test dust (Arizona Test Dust). This test dust has a wide size distribution covering the entire size range of the SidePak™ (AM510) and is representative of a wide variety of ambient aerosols (TSI, 2012). In addition, it is recommended by TSI that the instruments be returned to the factory for cleaning and calibration on an annual basis (TSI, 2012).

At the start of each sampling day, the SidePak™ impactor was cleaned to remove particles from previous experiments, and clean grease was applied to it. The SidePak™ was zero-calibrated using the manufacturer-supplied High-Efficiency Particulate Air (HEPA) filter, its flow rate checked and synchronized prior to each experiment. The logging interval for the SidePak™ monitor was set as 1 min. The $PM_{2.5}$ concentrations are logged automatically, and can be read directly from the monitor screen. Logged data were downloaded using the TSI TrackPro software (version 4.4.0.5).

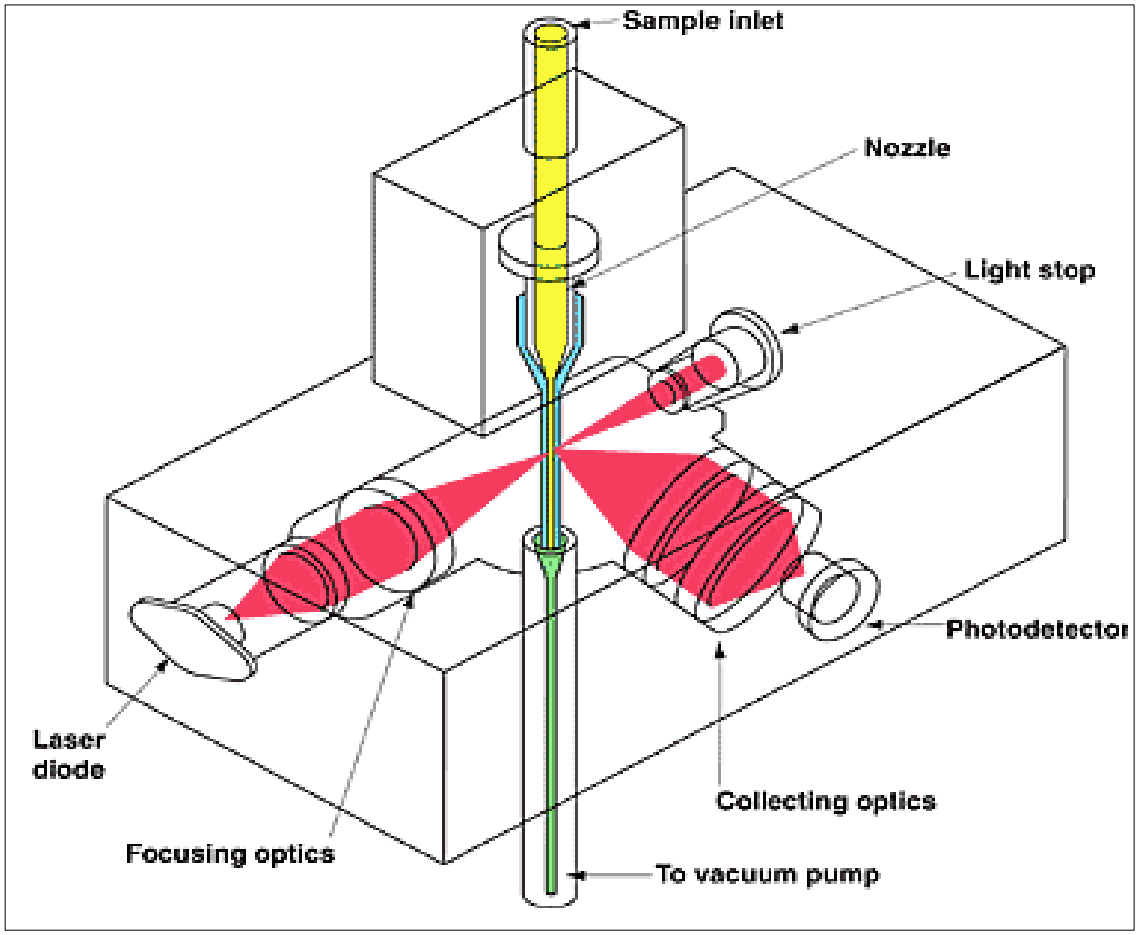


Figure 3.7: The schematic process of detection of the SidePak™ (Model AM510)
Source: (TSI (2012))

3.4.2.7 *Backpack*

Subjects participating in this study were asked to carry a small backpack containing the personal air monitor and GPS device to measure $PM_{2.5}$ levels associated with specific geographic locations, which were then categorised into meaningful microenvironments using data from the time-activity dairy. A backpack was used to house two instruments (see Figure 3.8). The personal air monitor (SidePak AM510) inlet was fitted with a tube, which was positioned near the breathing zone. The GPS device (Qstarz) tracker was placed inside the backpack. Both devices were time synchronised at the beginning of each day of the trial and were set to sample at intervals of one minute.

Participants were asked to carry this small backpack for 24 hours, including when traveling, outdoors at school/work, at home. During sports activities such as running and cycling, students were asked to continue wearing the small backpack. Where students took part in activities such as swimming, they were asked to leave the monitor at the edge of the pool, close to them. At bedtime, the monitor was to be placed next to the bed, and no time activity was required for this.

Figure 3.8: The backpack

3.4.2.8 *Analysis of phase two, microenvironment data*

The first objective of phase three was to identify factors that influence personal exposure to $PM_{2.5}$. The time activity diary provided details to categorise the participants' time over the study period into broad groupings to reflect indoor, outdoor and travel related exposures, as well as time spent at specific locations and undertaking specific activities.

Descriptive analyses have been used to describe the levels of a range of pollutant exposures and the proportion of time spent in each microenvironment.

Shapiro Wilk's test was used to assess the normality of the frequency distributions of $PM_{2.5}$ concentrations. For variables not normally distributed, non-parametric tests (Kruskal-Wallis test, to compare more than two groups and the Mann Whitney U test, to compare two groups) were used to compare personal exposure at different microenvironments. Statistically significant is defined as the 5% significance level in a two-tailed test ($p<0.05$).

As the air pollution levels had a skewed distribution, non-parametric tests were applied for the descriptive analyses. Wilcoxon-paired-sign-rank tests were used to identify any significant differences between hourly $PM_{2.5}$ levels obtained from the personal monitor and fixed-site monitoring station.

The second objective was to estimate exposure error introduced by using fixed-site monitoring stations as a proxy for personal exposure. In order to address this objective, the additional data downloaded from the community fixed-site monitoring station (monitor number eight) for the same time-period for which personal monitoring data for participants was collected. The correlations between the daily personal monitoring data and fixed-site monitoring data during each sampling period were assessed using Spearman's rank correlation coefficient. The differences between pairs of personal, indoor, outdoor and fixed-site monitoring data were calculated using the Wilcoxon signed rank test.

By incorporating the GPS data, it was possible to explore the geographic extent of the validity of the fixed-site monitor data as a proxy for personal exposure. This provided information on the characteristics of the exposure error/misclassification likely to be introduced into studies relying on local fixed-

site monitor data in this Middle East context. A multiple regression model was run to try to predict personal $PM_{2.5}$ exposure levels from the hourly fixed site monitor $PM_{2.5}$ levels and other covariates, including time slot, participant age, home characteristics (including cooking fuel, ventilation), and microenvironment (indoor versus 'other' locations). A stepwise selection method was used to exclude or include variables in a sequential process, with a statistical significance level of 95% ($p<0.05$) required for inclusion. If the fixed-site monitoring data was a good proxy for local personal exposure, it was expected that larger residuals might be seen further from the monitor location. Although the residuals have been used to give some indication of the "exposure error" from using the proxy measure, this measure is not based on just fixed-site data but also other covariates, including time, participant age, home characteristics (including cooking fuel, ventilation), and microenvironment (indoor versus 'other' locations) so 'error' is not solely due to using fixed-site data.

Hourly personal mean $PM_{2.5}$ levels were plotted in ArcGIS (ESRI, 2014) according to the mean GPS locations visited by the students across the hour on an Al Jubail base map to explore spatial patterns in exposure error. SPSS v.21 for Windows (IBM, 2012) was used to perform a stepwise multiple regression analysis for prediction of personal exposure. Microsoft Excel 2013 was used to plot the correlation between different variables.

3.4.3 Phase three: TSA of AEDv and ambient $PM_{2.5}$ levels corrected from personal monitoring campaign

The relationship between AEDv and air pollution was re-examined to investigate how the relationship would change if personal air pollution exposures (from objective two) were used instead of data from fixed-site monitor (from objective one). In order to investigate this relationship, a strategy was developed by using the $PM_{2.5}$ levels from the personal monitoring campaign to refine the ambient concentrations derived from the fixed-site monitor, which were then used in a re-run of the main time-series analysis model. The corrected ambient $PM_{2.5}$ levels ($PM_{2.5}C$) were calculated using a correction factor derived by dividing the total median $PM_{2.5}$ levels obtained from the personal exposure monitoring by the levels obtained from the fixed-site monitor. The fixed-site ambient $PM_{2.5}$ levels were then multiplied by the correction factor to create $PM_{2.5}C$. The correction factor was also checked again by dividing the lower and upper quartiles of $PM_{2.5}$ levels measured via fixed-site monitor and personal exposure to reflect the whole distribution levels in order to identify any difference possibility for the difference between fixed-site monitor and personal exposure.

The AEDv for the relevant school age group (admissions aged 6-18 years old) were extracted from the AEDv dataset. This school age group was selected to compare associations between AEDv with ambient $PM_{2.5}$ levels and between AEDv and $PM_{2.5}C$ using the main time-series analysis model. Restricting this re-analysis to school age admissions was due to the fact that the personal monitoring campaign involved only school students, and it did not seem appropriate to apply the correction factor beyond this age group in this wider analysis in Jubail Industrial City. The AEDv in this relevant school age group represents 29.2% of the total AEDv dataset. The relative risks of AEDv for this school age group were calculated using the same basic steps as the main model of time-series analysis (steps one, two, three and four in this chapter, section 3.3.1.6). The results were expressed as percent increases in daily AEDv (with 95% confidence intervals (95% CI)) per 10μg/m^3 increase of $PM_{2.5}C$.

Finally, a sensitivity analysis was carried out by using the highest and the lowest difference of $PM_{2.5}$ levels between fixed-site monitor and personal exposure in the main model of time-series analysis to see if there were any

possible differences between RRs of AEDv and to insure the reliability of this model.

3.4.4 Confidentiality

It was made clear during the recruitment process that I would not be able to 'track' the participants in the real-time when they are carrying the GPS devices, I would only know where the participants have been retrospectively. Names of individual participants will not appear on any reports, published scientific papers or presentations. This study acted in accord with Caldicott principles (Caldicott.Committee, 1999; Crook, 2003).

3.4.5 Ethical approval and risk assessment

I applied for, and secured ethical approval before undertaking the fieldwork in Saudi. The Ethics Committee of the Faculty of Medical Science at Newcastle University gave ethical approval for this project (See Appendix D, Ethical Approval Letter from FMS Ethics Committee at Newcastle University). In addition, the Royal Commission in Al Jubail Industrial City approved this work (See Appendix E, Approval Letter from Royal Commission in Al Jubail, Saudi Arabia) and I got a permission to visit a secondary school from the Education Service Program in Al Jubail to recruit students aged between 16 to 18 years. A risk assessment was undertaken at Newcastle University before fieldwork travel to Saudi. Finally, I applied for insurance indemnity to cover this project and this was also approved by Insurance Office at Newcastle University (See Appendix F, Insurance Indemnity Letter).

3.4.6 Data storage

Data collected were accessible only to the researchers, and secured in locked cabinet inside the University. Data analysis was done on a University PC, which is password-protected.

CHAPTER FOUR

Result of Time-Series Analysis

Chapter:4 Result of Time-Series Analysis

4.1 Introduction

In this chapter, the results of descriptive analysis of air pollutants, meteorological variables and asthma-related emergency department visits (AEDv) are presented. This is followed by the detailed results of the single pollutant model, the multi-pollutant model as well as relative risk analysis.

4.2 Data Availability for Fixed-Site Monitoring Stations

Table 4.1 shows the availability of daily air pollutant data for the period between 1st January 2007 and 31st December 2011. Days with >75% of the hourly values missing were counted as missing days. Data includes air pollution levels; carbon monoxide (CO), nitrogen oxides (NO_x), particulate matter ($PM_{2.5}$ and PM_{10}), sulphur dioxide (SO_2), and weather variables; temperature (T) and relative humidity (RH). The total number of days in this five-year period was 1826 days. The fixed-site monitoring station with the lowest percent missing data was the residential fixed-site monitoring station (site 8) with a total of 2.3% missing data.

For the other fixed-site monitoring stations the percent missing data ranged between 5.0% and 26.5%. The air pollutant variables with the greatest proportion of missing data were $PM_{2.5}$ at site 6 (data missing for 1539 days), SO_2 at site 2 (519 days), PM_{10} at site 1 (312 days), NO_2 at site 9 (120 days) and CO at site 2 (108 days). Whereas the variables with little missing data were NO_2 at site 2 (11 days), $PM_{2.5}$ at site 2 (17 days), CO at site 4 (17 days), SO_2 at site 4 (33 days) and PM_{10} at site 4 (78 days). These figures for missing data are as a result of removal of any days that did not meet the completeness criteria, which was set at 75% of the hourly values per day. Overall, these results showed that the residential fixed-site monitoring station (site 8) has the highest availability of data among the available fixed-site monitoring stations in Al Jubail city.

Table 4.1: Data availability of all fixed-site monitoring stations

Fixed-Site	Pollutant	N		Missing (%)
		Valid	Missing	
Site 1	PM_{10}	1514	312	20.6%
	$PM_{2.5}$	1283	543	42.3%
	NO_2	1809	17	0.9%
	SO_2	1790	36	2.0%
	CO	1793	33	1.8%
	Total	**8189**	**941**	**11.5%**
Site 2	PM_{10}	1571	255	16.2%
	$PM_{2.5}$	1809	17	0.9%
	SO_2	1307	519	39.7%
	NO_2	1815	11	0.6%
	CO	1718	108	6.3%
	Total	**8220**	**910**	**11.1%**
Site 3	PM_{10}	1700	126	7.4%
	$PM_{2.5}$	1721	105	6.1%
	SO_2	1763	63	3.6%
	NO_2	1808	18	1.0%
	CO	1796	30	1.7%
	Total	**8788**	**342**	**3.9%**
Site 4	PM_{10}	1748	78	4.5%
	$PM_{2.5}$	1535	291	19.0%
	SO_2	1793	33	1.8%
	NO_2	1809	17	0.9%
	CO	1809	17	0.9%
	Total	**8694**	**436**	**5.0%**
Site 6	PM_{10}	1632	194	11.9%
	$PM_{2.5}$	287	1539	84.3%
	SO_2	1781	45	2.5%
	NO_2	1735	91	5.2%
	CO	1783	43	2.4%
	Total	**7218**	**1912**	**26.5%**
Site 8 residential area	PM_{10}	1764	62	3.5%
	$PM_{2.5}$	1786	40	2.2%
	SO_2	1788	38	2.1%
	NO_2	1793	33	1.8%
	CO	1794	32	1.8%
	Total	**8925**	**205**	**2.3%**
Site 9	PM_{10}	1558	268	17.2%
	$PM_{2.5}$	1750	76	4.3%
	SO_2	1752	74	4.2%
	NO_2	1706	120	7.0%
	CO	1751	75	4.3%
	Total	**8517**	**613**	**7.2%**

4.3 Descriptive Statistics for Fixed-Site Monitoring Stations

Summary statistics for the daily air pollutant levels from all fixed-site monitoring stations are shown in Table 4.2. The summary statistics shown are mean, median, 25 percentile, 50 percentile and 75 percentile, because the distribution of data did not fit a normal pattern (Shapiro Wilk's test $P<0.05$). The daily median levels of CO (median range 0.41-0.55ppb) did not vary much among the monitoring sites. For NO_2, the daily median levels ranged between 13.8 and 21.21ppm, except for fixed-site number 9 (median = 8.99ppm) that has a great proportion of missing data (7.0%). For SO_2, the daily median levels by fixed-site monitoring station ranged between 1.80-3.71ppm, except fixed-site number 2 (median = 1.06ppm) that has a great proportion of missing data (39.7%). For PM_{10} and $PM_{2.5}$, the daily median levels varied greatly among the fixed-site monitoring stations, which might be due in part to there being a greater proportion of missing data for these variables, as shown in Table 4.2.

Table 4.3 shows the t-test of difference in the mean of the median air pollutant levels measured at the residential fixed-site (fixed-site 8) and the other fixed-site monitoring stations. The results indicated that they were no significant differences between air pollutant levels measured at the residential fixed-site and other fixed-site monitoring stations.

Table 4.2: Descriptive statistics for fixed-site monitoring stations

Pollutant (site)	Missing (%)	Mean	SD	Median	Percentiles		IQR	Min	Max
					25%	75%			
PM_{10}_1	20.6%	341.19	372.39	223.63	152.00	376.63	224.63	16.25	4141.33
PM_{10}_2	16.2%	37.95	106.62	10.53	7.72	19.50	11.78	2.36	1737.00
PM_{10}_3	7.4%	161.16	216.07	99.76	67.97	159.27	91.31	0.00	3049.40
PM_{10}_4	4.5%	213.12	282.53	130.85	83.01	219.17	136.16	23.26	3756.46
PM_{10}_6	11.9%	265.86	336.11	158.17	96.63	274.90	178.26	24.50	3229.95
PM_{10}_8	3.5%	220.43	311.08	128.72	81.81	221.41	139.60	0.00	3599.26
PM_{10}_9	17.2%	163.77	221.74	101.46	59.47	175.26	115.79	0.00	3441.07
PM_{10} Overall Mean	11.6%	200.50	263.79	121.87	78.37	206.59	128.22	9.48	3279.21
$PM_{2.5}$_1	42.3%	92.15	98.24	63.25	41.75	99.56	57.81	0.01	777.42
$PM_{2.5}$_2	0.9%	26.13	66.09	3.40	1.70	9.23	7.53	0.01	568.53
$PM_{2.5}$_3	6.1%	66.88	63.11	47.38	32.29	75.10	42.81	0.01	695.92
$PM_{2.5}$_4	19.0%	62.58	63.19	42.13	29.00	67.87	38.87	0.01	563.57
$PM_{2.5}$_6	84.3%	184.45	144.92	153.10	98.68	207.71	109.02	17.25	946.29
$PM_{2.5}$_8	2.2%	64.54	64.98	45.55	32.00	67.71	35.71	9.96	643.70
$PM_{2.5}$_9	4.3%	56.24	57.43	40.18	24.50	63.44	38.95	4.70	540.28
$PM_{2.5}$ Overall Mean	22.7%	79.00	79.71	56.43	37.13	84.37	47.24	4.56	676.53
SO_2_1	2.0%	4.24	3.01	3.75	2.02	5.75	3.73	0.00	27.46
SO_2_2	39.7%	1.42	1.87	1.06	0.51	1.59	1.08	0.00	31.02
SO_2_3	3.6%	3.97	3.71	2.95	1.60	5.07	3.47	0.00	42.67
SO_2_4	1.8%	3.84	2.94	3.17	2.11	4.81	2.71	0.00	38.46
SO_2_6	2.5%	2.97	2.10	2.40	1.56	3.83	2.27	0.00	15.91
SO_2_8	2.1%	3.09	1.80	2.77	1.98	3.99	2.01	0.00	14.11
SO_2_9	4.2%	10.36	15.79	4.84	2.56	10.53	7.96	0.00	177.20
SO_2 Overall Mean	8.0%	4.27	4.46	2.99	1.76	5.08	3.32	0.00	49.55

Note: To be continued on the next page

Table 4-2: Continued

Pollutant (site)	Missing (%)	Mean	SD	Median	Percentiles		IQR	Min	Max
					25%	75%			
NO_2_1	0.9%	23.61	12.31	21.21	15.30	28.61	13.31	3.21	111.67
NO_2_2	0.6%	16.84	10.13	15.15	9.36	22.21	12.85	0.05	70.44
NO_2_3	1.0%	20.19	7.72	20.27	14.52	25.38	10.86	0.03	51.70
NO_2_4	0.9%	17.39	7.28	17.68	12.60	22.20	9.60	0.00	72.99
NO_2_6	5.2%	14.78	7.79	13.80	9.37	18.72	9.35	0.00	73.62
NO_2_8	1.8%	15.52	6.40	14.45	11.04	18.75	7.71	0.07	45.95
NO_2_9	7.0%	11.11	15.34	8.99	5.41	13.21	7.80	0.00	258.23
NO_2 Overall Mean	2.5%	17.06	9.57	15.94	11.09	21.30	10.21	0.48	97.80
CO_1	1.8%	0.46	0.23	0.41	0.30	0.58	0.28	0.00	1.50
CO_2	6.3%	0.48	0.21	0.46	0.34	0.61	0.27	0.00	1.28
CO_3	1.7%	0.46	0.24	0.44	0.32	0.58	0.26	0.00	3.42
CO_4	0.9%	0.58	0.32	0.55	0.41	0.70	0.29	0.00	3.50
CO_6	2.4%	0.47	0.26	0.43	0.31	0.58	0.27	0.00	2.61
CO_8	1.8%	0.46	0.19	0.43	0.32	0.56	0.24	0.02	1.49
CO_9	4.3%	0.51	0.40	0.45	0.33	0.61	0.27	0.01	5.02
CO Overall Mean	2.7%	0.49	0.26	0.45	0.33	0.60	0.27	0.00	2.69

Table 4.3: T-test of air pollutant from all fixed-site compared to residential fixed-site monitoring stations

Pollutant	Mean	SD	t	df	Sig. (2-tailed)
PM_{10}	121.87	64.68	-0.28	6.00	0.79
$PM_{2.5}$	56.43	46.32	0.62	6.00	0.56
SO_2	2.99	1.17	0.50	6.00	0.63
NO_2	15.94	4.19	0.94	6.00	0.38
CO	0.45	0.05	1.32	6.00	0.23

4.4 Air Pollutant Correlations between the Residential Fixed-Site and All Other Fixed-Site Monitoring Stations

The correlations of air pollutants between sites were assessed using Spearman's rank correlation coefficient, as the data were not normally distributed (Shapiro Wilk's test p-value < 0.001). Table 4.4 shows Spearman's rank correlation coefficients among each air pollutant across all fixed-site monitoring stations in Jubail Industrial City for the five-year period. Daily average PM_{10} was significantly positively correlated (r = 0.714 to 0.916) between the residential fixed-site and all other fixed-site monitoring stations, except for fixed-site number 2, that has a great proportion of missing data (r = 0.03). $PM_{2.5}$ levels between the residential fixed-site and all other fixed-site monitoring stations were significantly positively correlated (r = 0.503 to 0.730), except site number 2 (r = 0.051). For daily SO_2, the correlations were significant and positively, but highly variable, ranging from weak to moderate (r = 0.064 to 0.500), again with the exception of site number 2 (r = -0.069). For daily NO_2, the correlations were significant and positive, ranging from weak to strong (r = 0.191 to 0.655). CO levels were weakly but significantly positively correlated with CO levels measured at all other fixed-site monitoring stations (r = 0.137 to 0.309).

Table 4.4: Correlations between the residential fixed-site and all other fixed-site monitoring stations

Fixed-sites		Residential Fixed-site Monitoring Station				
		PM_{10}	$PM_{2.5}$	SO_2	NO_2	CO
Site 1	Correlation	0.808**	0.725**	0.410**	0.575**	0.224**
	Sig. (2-tailed)	<0.01	<0.01	<0.01	<0.01	<0.01
	N	1722	1557	1826	1826	1826
Site 2	Correlation	0.03	0.051*	-0.069**	0.655**	0.223**
	Sig. (2-tailed)	0.16	0.03	0.01	<0.01	<0.01
	N	1640	1826	1422	1826	1822
Site 3	Correlation	0.916**	0.730**	0.500**	0.552**	0.137**
	Sig. (2-tailed)	<0.01	<0.01	<0.01	<0.01	<0.01
	N	1826	1811	1826	1826	1826
Site 4	Correlation	0.857**	0.725**	0.218**	0.535**	0.295**
	Sig. (2-tailed)	<0.01	<0.01	<0.01	<0.01	<0.01
	N	1826	1671	1826	1826	1826
Site 6	Correlation	0.839**	0.586**	0.416**	0.514**	0.309**
	Sig. (2-tailed)	<0.01	<0.01	<0.01	<0.01	<0.01
	N	1766	589	18265	1826	1826
Site 9	Correlation	0.714**	0.503**	0.064**	0.191**	0.277**
	Sig. (2-tailed)	<0.01	<0.01	0.01	<0.01	<0.01
	N	1826	1826	1826	1826	1826

**Spearman's rank correlation is significant at the 0.01 level (2-tailed)
*Spearman's rank correlation is significant at the 0.05 level (2-tailed

4.5 Wind Direction Analysis

Using the Openair toolset, the available meteorological data was interrogated, generating the wind rose plots as shown in Figure 4.1. The plot shows wind direction/speed frequencies during the study period 1st January 2007 to 31st December 2011. The data are summarised by direction, and by different wind speed categorise and the percentage of time that the wind blows from a certain angle and wind speed range. The distance from the centre of the plot, where the north-west and south-east axes cross, represents the wind speed; the further from the centre, the higher the wind speed. Wind speeds are split the intervals shown by the scale in each panel. The grey circles show the percentage frequencies. Wind speeds are most commonly between 2-6 m s^{-1} i.e. relatively low wind speeds. The direction out from the centre represents the wind direction at the time of measurement. The wind direction is dominated by north-westerly winds, with some occurrences from the south-east.

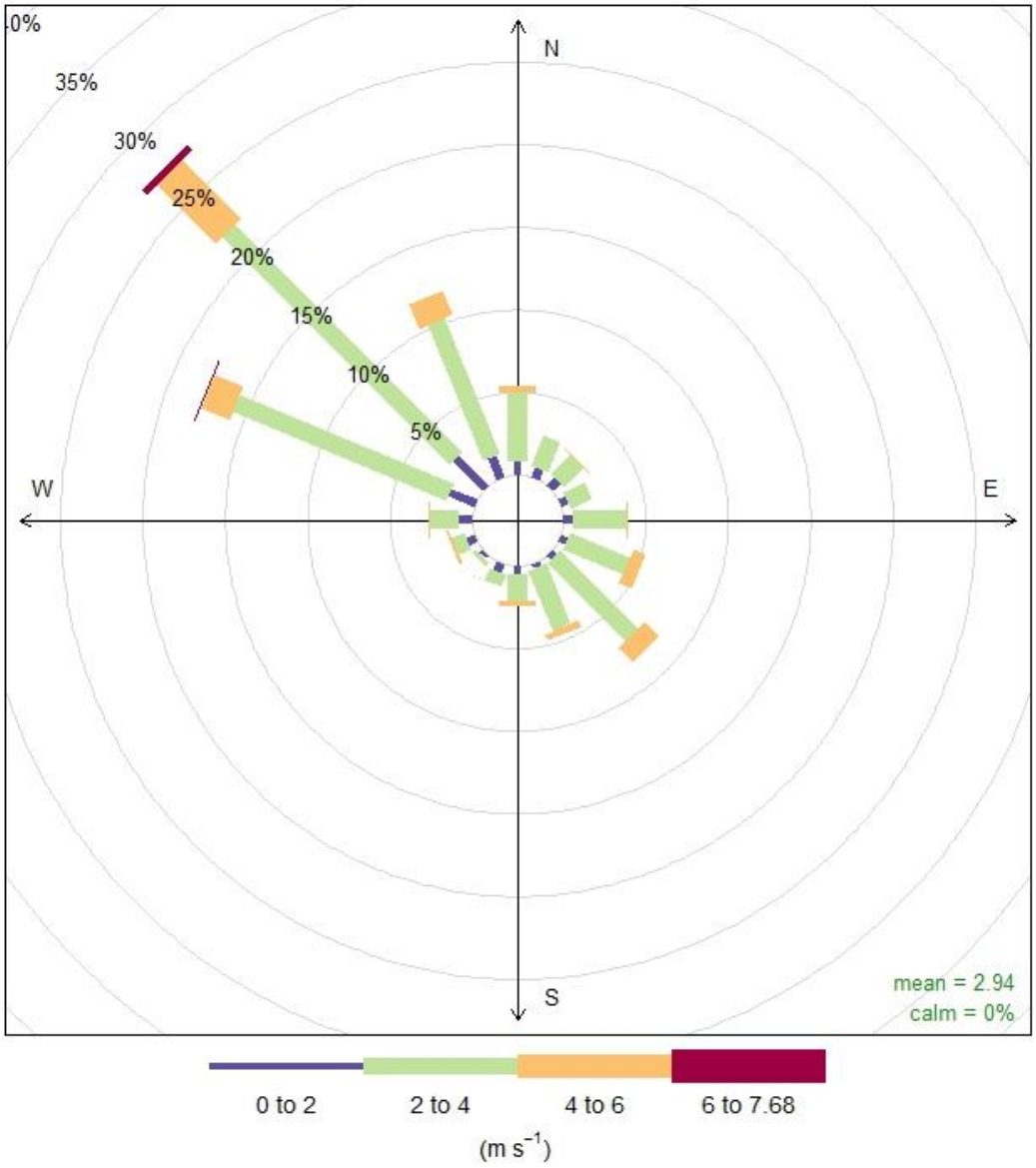


Figure 4.1: Frequency of counts (%) by wind direction

4.6 Wind Direction Contribution to AEDv and Air pollution

Using the Openair toolset, the available AEDv and air pollution data were interrogated using polar frequency plots as shown in Figure 4.2 for AEDv, and in Figures 4.3 to 4.7 for air pollutants. The plots show data over the whole study period rather than examining it on an annual, monthly, daily or diurnal basis. The polar frequency plot was used to gain an idea about the wind directions that contribute most to the overall mean of air pollution levels and AEDv. The plots show measurements weighted by their frequency of occurrence by wind direction. The colour of the chart represents the percentage contribution to overall mean of measurements, with white being lower percentage contribution and red higher percentage contribution.

As shown in Figure 4.2, the daily number of AEDv at this study setting is dominated by north-westerly winds, and the probability of their being such high daily number of AEDv from other wind directions is effectively zero. The polar frequency plots for weighted mean of PM_{10}, $PM_{2.5}$, SO_2, NO_2 and CO are shown in Figures from 4.3 to 4.7 retrospectively. These plots are very useful for understanding which wind directions control the overall mean air pollutant concentrations. The plots of weighted mean air pollutant levels highlight that annual mean concentrations are also dominated by north-westerly winds which is the prevailing wind direction (as shown previously in Figure 4.1), which explains why there is little/no contribution from south and easterly winds.

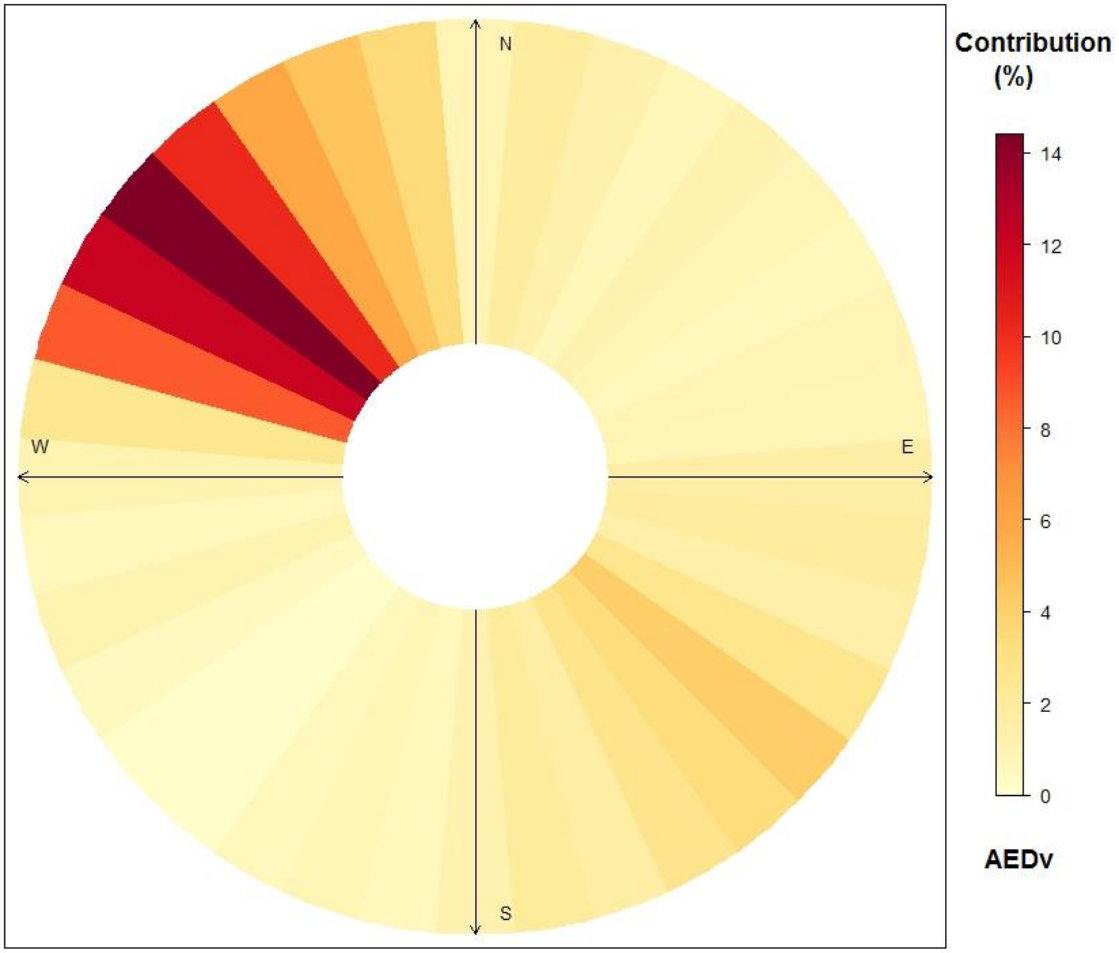


Figure 4.2: The percentage contribution of wind direction to overall mean of Asthma-related Emergency Department visits

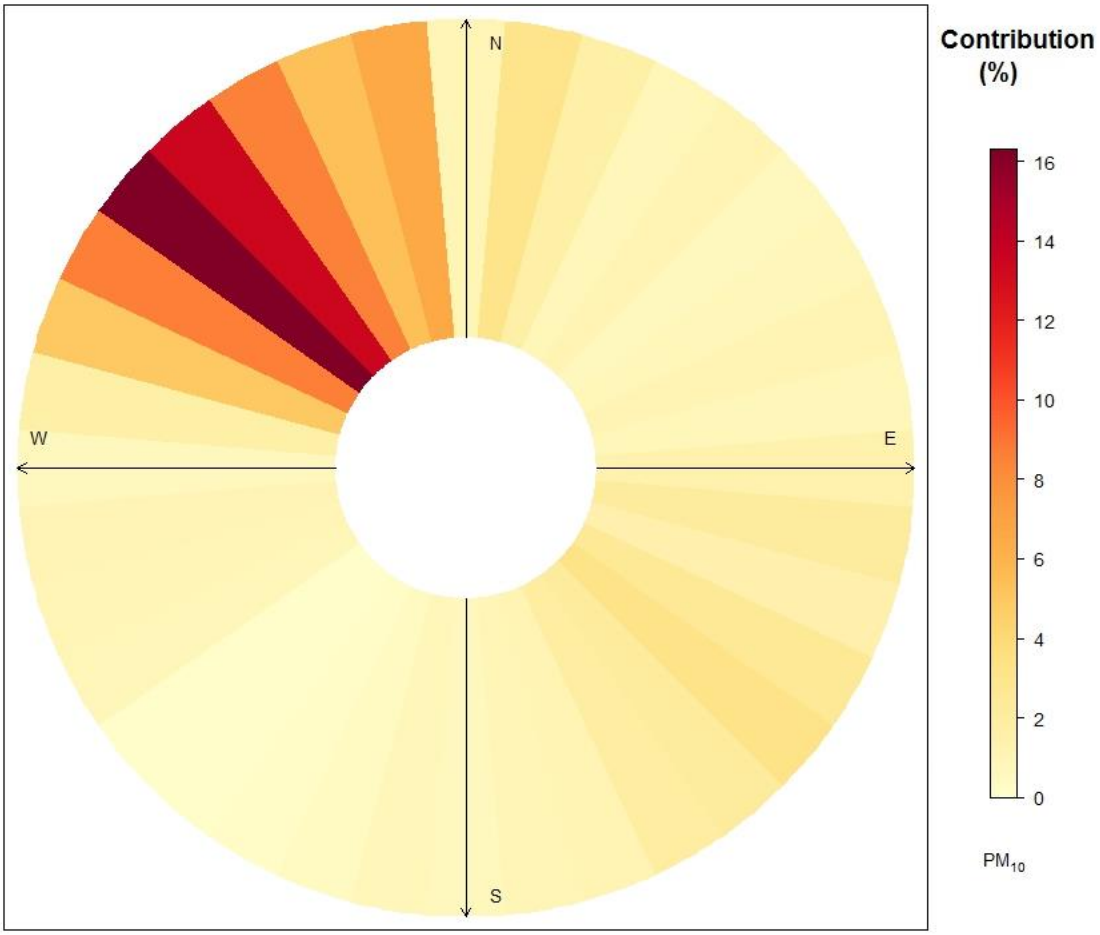


Figure 4.3: The percentage contribution of wind direction to overall mean concentrations of PM_{10}

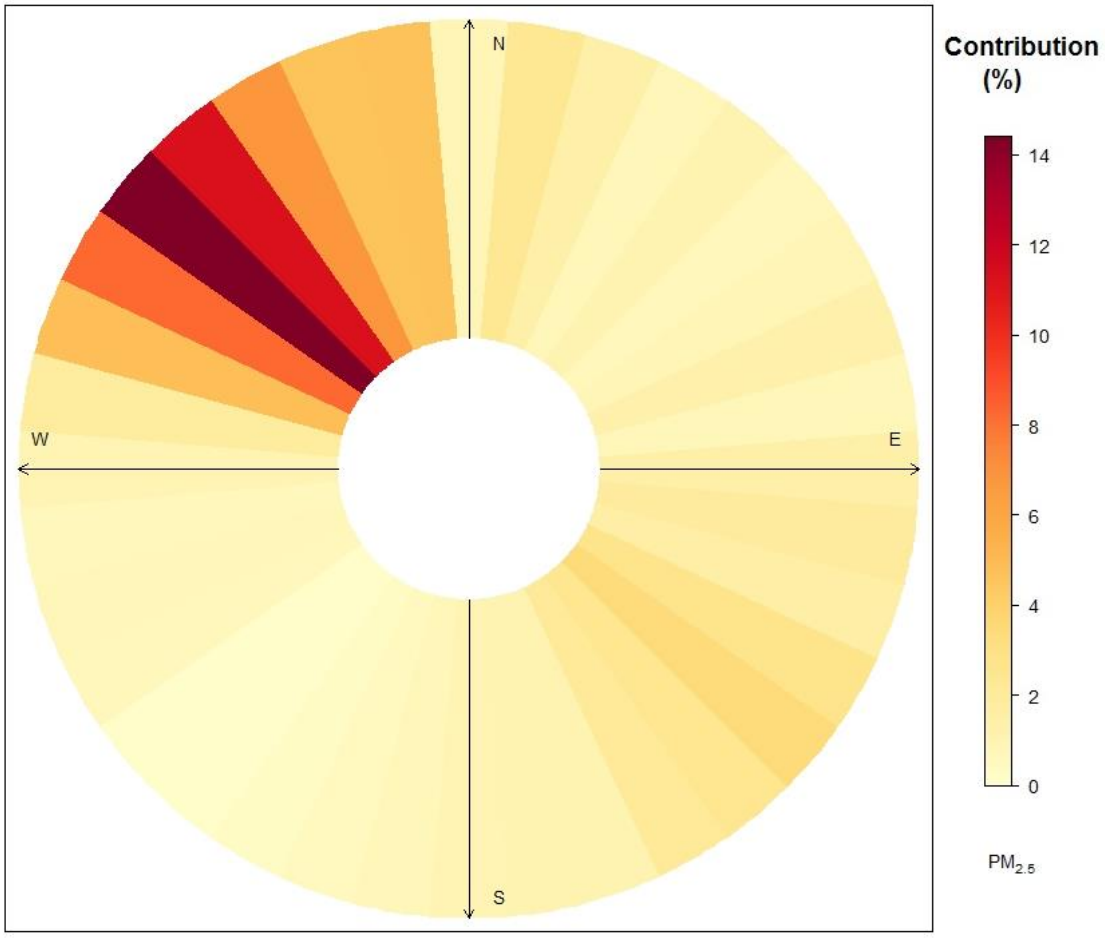


Figure 4.4: The percentage contribution of wind direction to overall mean concentrations of $PM_{2.5}$

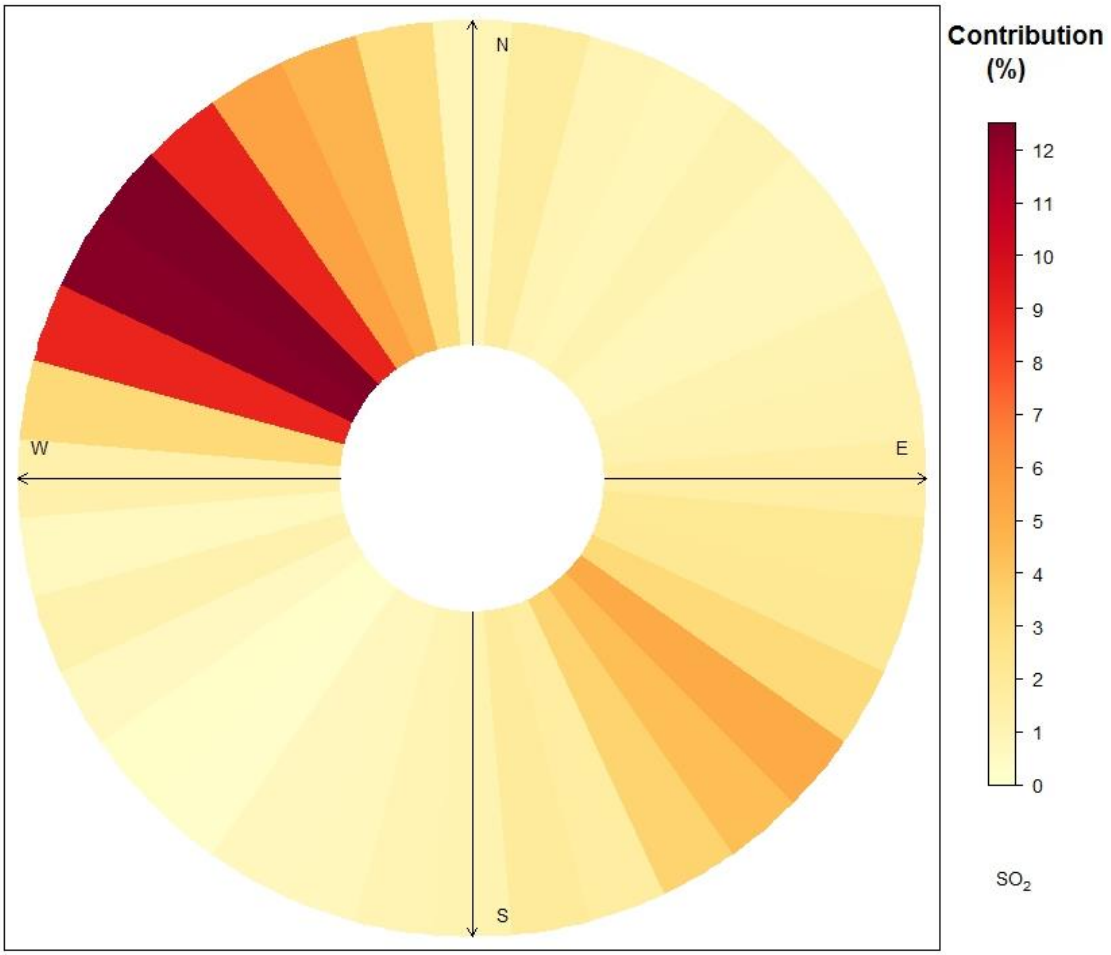


Figure 4.5: The percentage contribution of wind direction to overall mean concentrations of SO_2

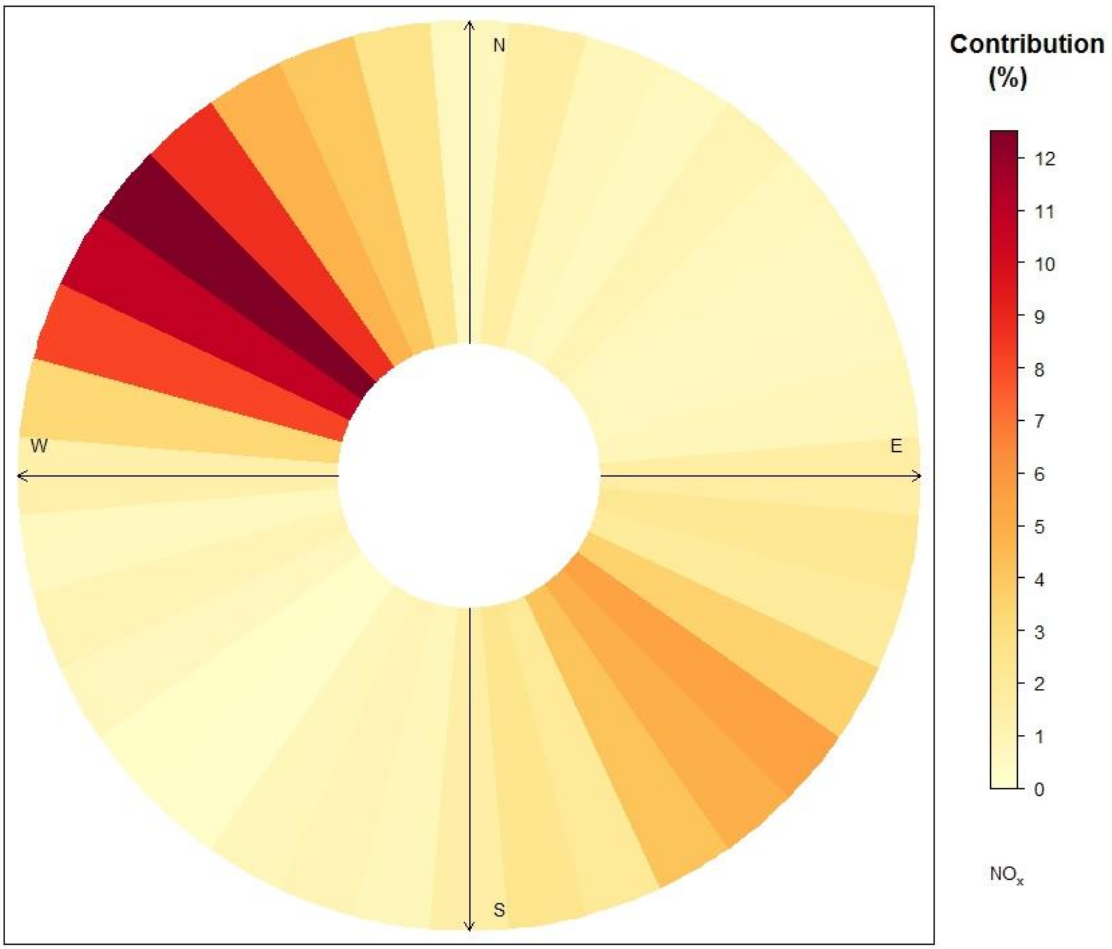


Figure 4.6: The percentage contribution of wind direction to overall mean concentrations of NO_2

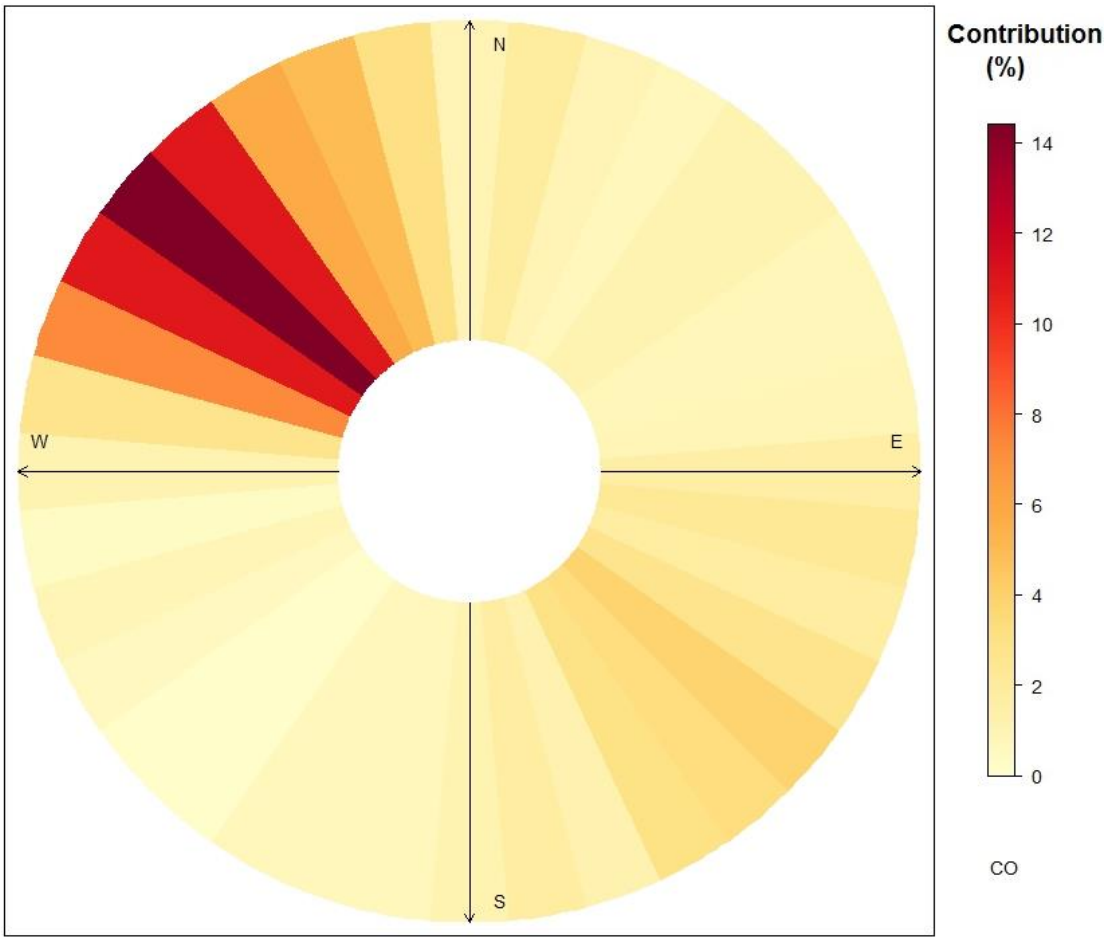


Figure 4.7: The percentage contribution of wind direction to overall mean concentrations of CO

4.7 Descriptive Analysis of Daily AEDv and Air Pollutants and Meteorological Data

Table 4.5 shows the descriptive statistics for the daily AEDv, air pollutants and meteorological data (temperature and relative humidity) for the study period,

There was no missing data for asthma-related emergency department visits (i.e AEDv data were available for all 1826 days in the study period). The variables with a great proportion of missing data were PM_{10} (62 days) and $PM_{2.5}$ (40 days), while the variables with little missing data were the weather variables: temperature and relative humidity (both 33 days). These figures for missing data are as a result of removal of any days that did not meet the completeness criteria, which was set at 75% of the hourly values per day.

Table 4.5: Descriptive statistics for daily AEDv, air pollution and weather variables

		AEDv	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	SO_2 (ppb)	NO_2 (ppb)	CO (ppm)	Temp (C^o)	RH (%)
Total number of days		1826	1764	1786	1788	1793	1794	1793	1793
Missing days*		0	62	40	38	33	32	33	33
Mean		4.60	220.16	64.61	3.06	15.48	0.46	26.39	49.69
Median		4	129.56	45.63	2.74	14.39	0.43	27.53	49.69
Std. Deviation		3.30	308.49	64.52	1.80	6.37	0.19	8.02	15.18
Minimum		0	2.00	9.96	0.01	0.07	0.02	6.33	12.94
Maximum		28	3599.26	643.70	14.11	45.95	1.49	39.13	95.30
Quartiles	**25%**	2	81.60	32.13	1.96	11.04	0.32	19.21	37.69
	50%	4	129.56	45.63	2.74	14.39	0.43	27.53	49.69
	75%	6	222.35	68.41	3.95	18.63	0.57	33.97	61.26
IQR		4	140.76	36.28	1.99	7.59	0.25	14.76	23.57

*Days excluded due to missing data (≥75% of the hourly values per day)

4.8 Time-Series Plot of Asthma-related Emergency Department visits

A total number of 8434 daily asthma-related emergency department visits (AEDv) occurred during the study period. The time-series plots of daily asthma-related emergency department visits revealed a prominent seasonal cycle as shown in Figure 4.8. The annual average of AEDv did not show any yearly trend during the study period, as shown in Figure 4.9. The distribution of AEDv by season and day of the week is presented in Figure 4.10 and Figure 4.11 respectively. The number of asthma visits was lower in the warm season (spring and summer), and uniformly higher in winter and fall. With regard to day of the week, admissions during weekdays (from Saturday to Wednesday) were relatively constant, whereas weekends (Thursday and Friday) showed the highest number of visits per day.

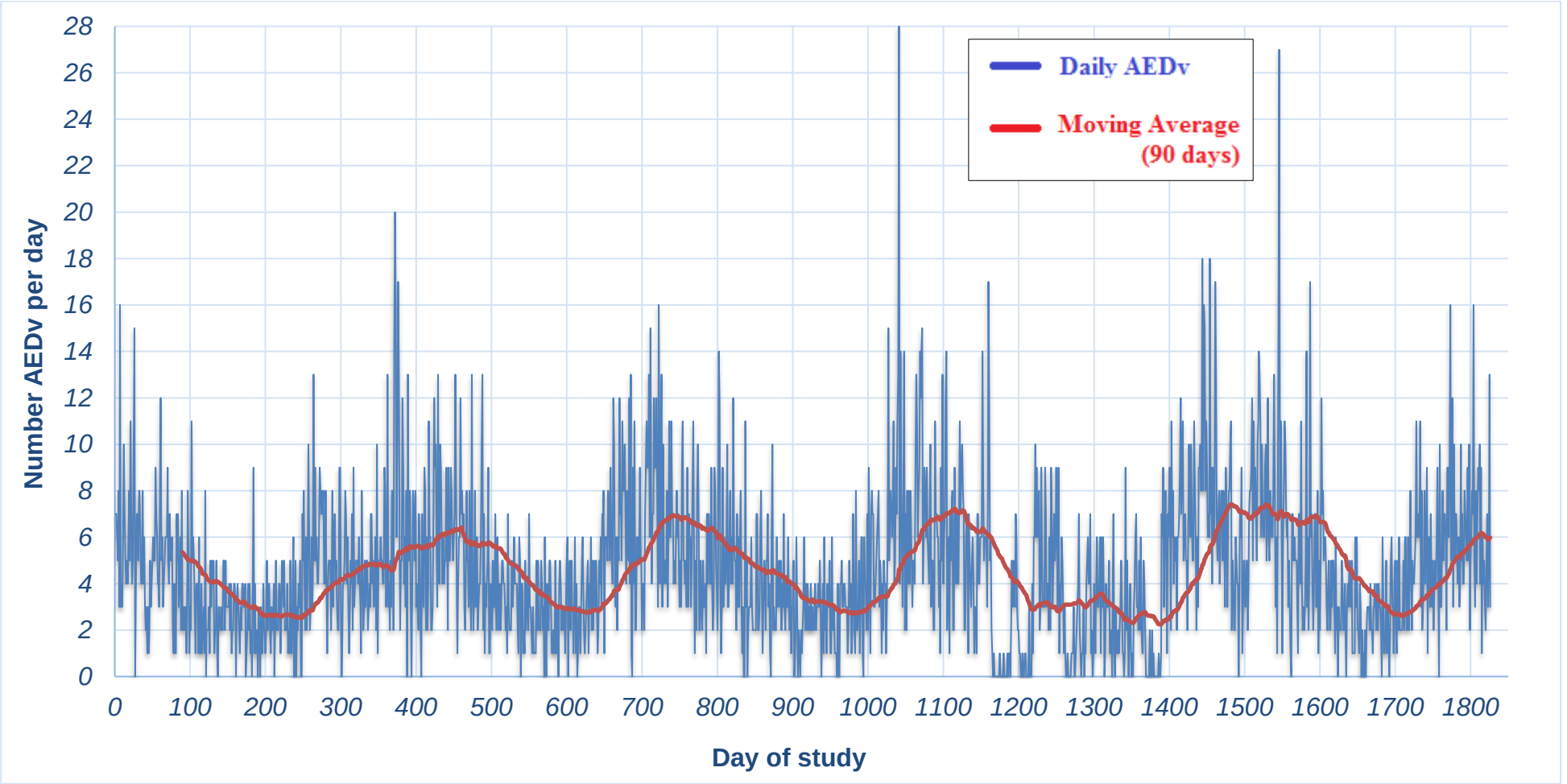


Figure 4.8: Time-series of daily asthma-related emergency department visits for the period 2007-2011

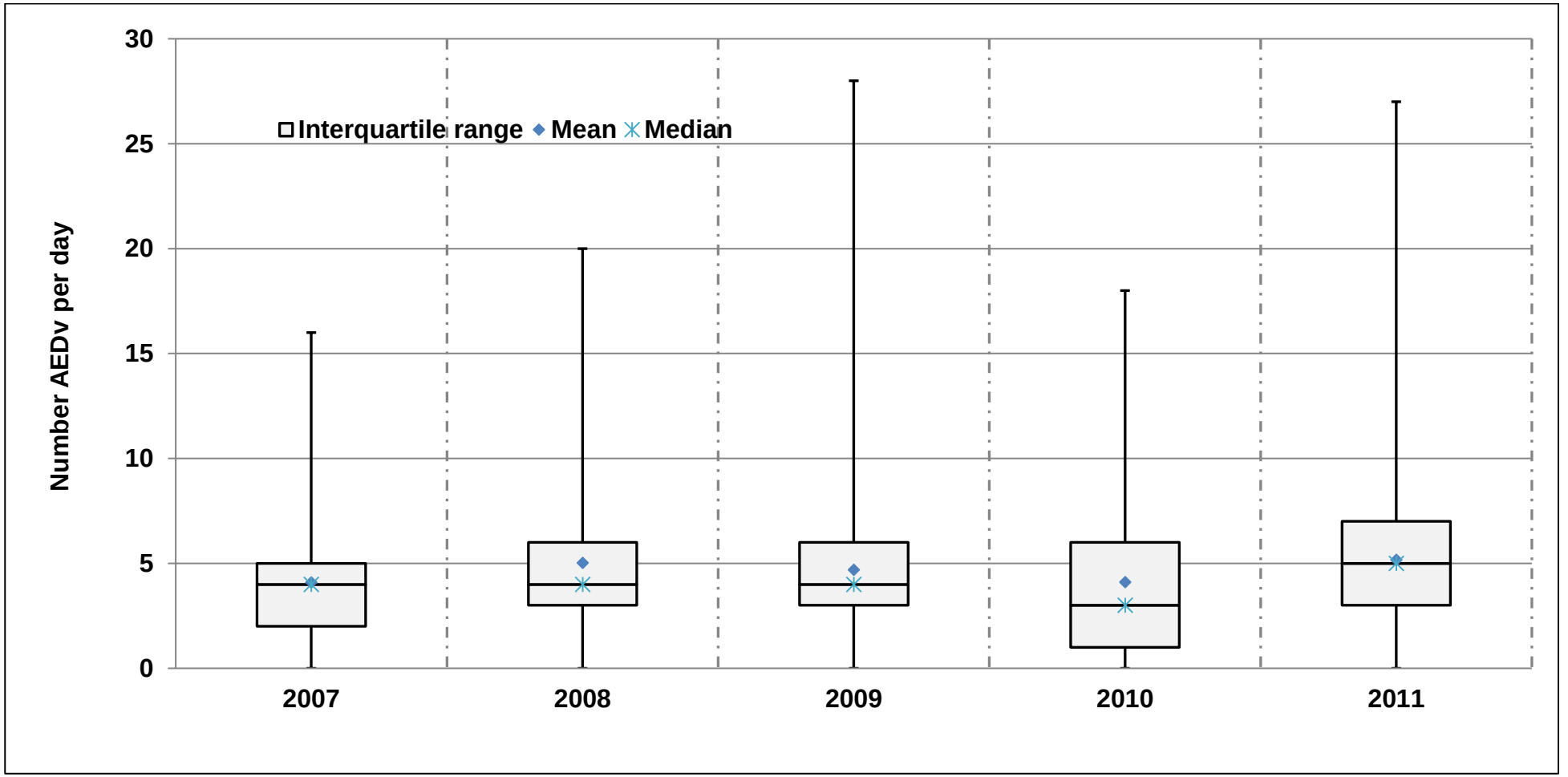


Figure 4.9: Annual average and variability of asthma-related emergency department visits for the period 2007-2011

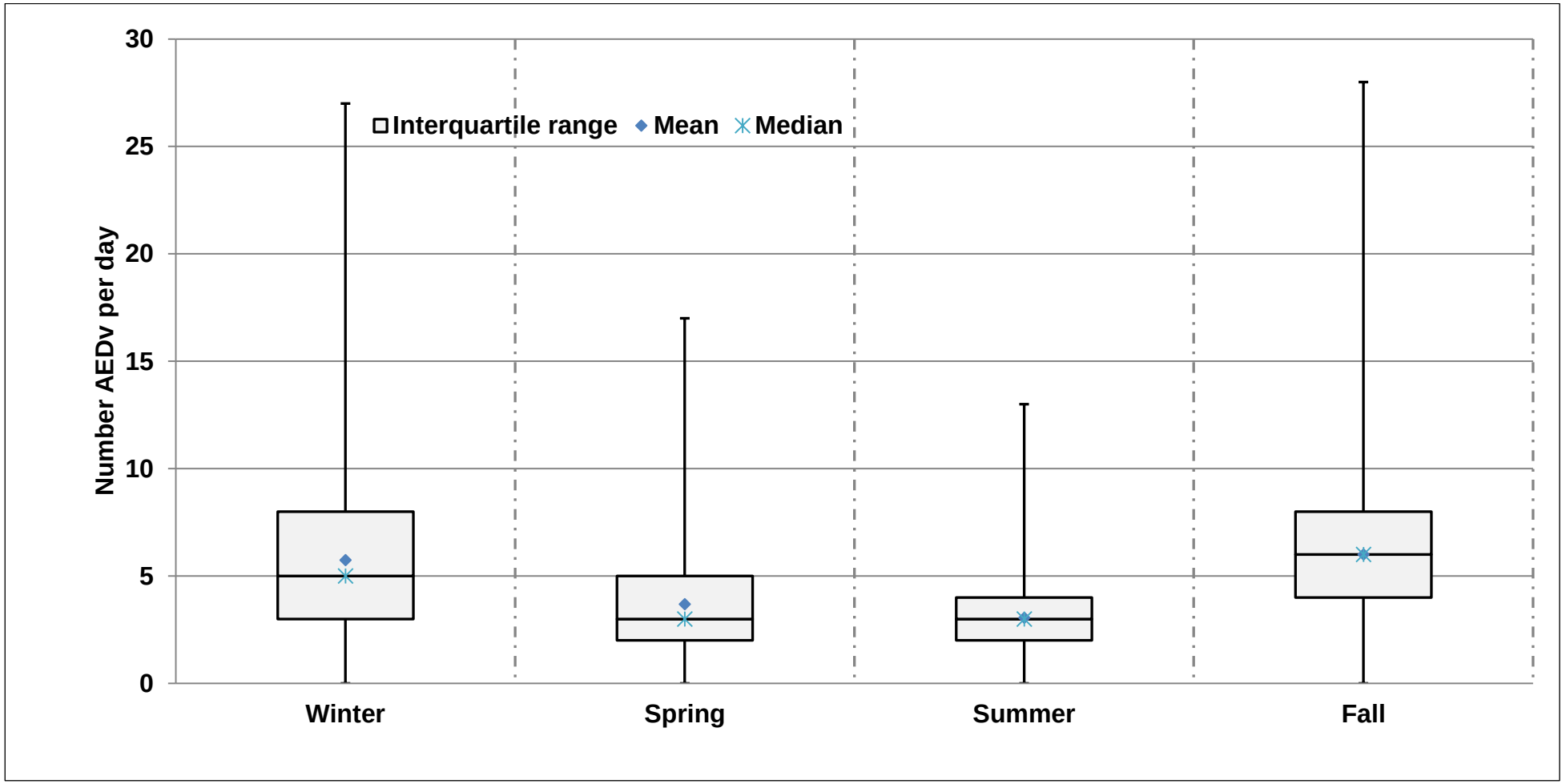


Figure 4.10: Seasonal average and variability of asthma-related emergency department visits for the period 2007-2011

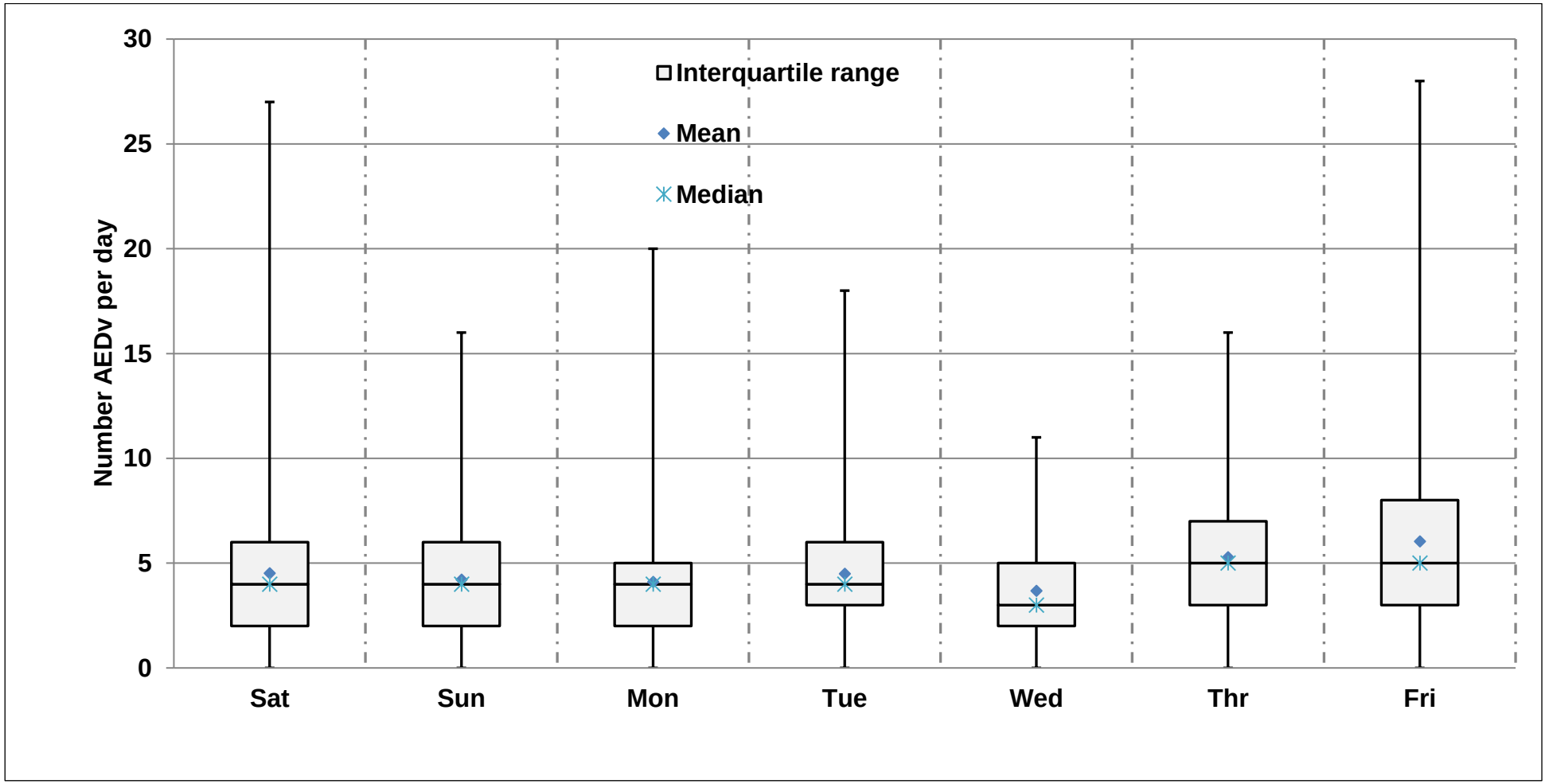


Figure 4.11: Day of week average and variability of asthma-related emergency department visits for the period 2007-2011

4.9 Time-Series Plot per Day of Study among Environmental Variables

The day-to-day variations in air pollutants levels are shown in Figure 4.12 to Figure 4.16. PM_{10}, $PM_{2.5}$ and NO_2 show clear seasonal patterns. SO_2 levels decreased over the last year of the study period. However, CO did not show any seasonally or yearly trend but variability in CO appears to decrease over the study period.

Weather variables are shown in Figure 4.17 for temperature and in Figure 4.18 for relative humidity. As shown on these figures, weather variables follow the typical seasonal patterns seen in Al Jubail Industrial City.

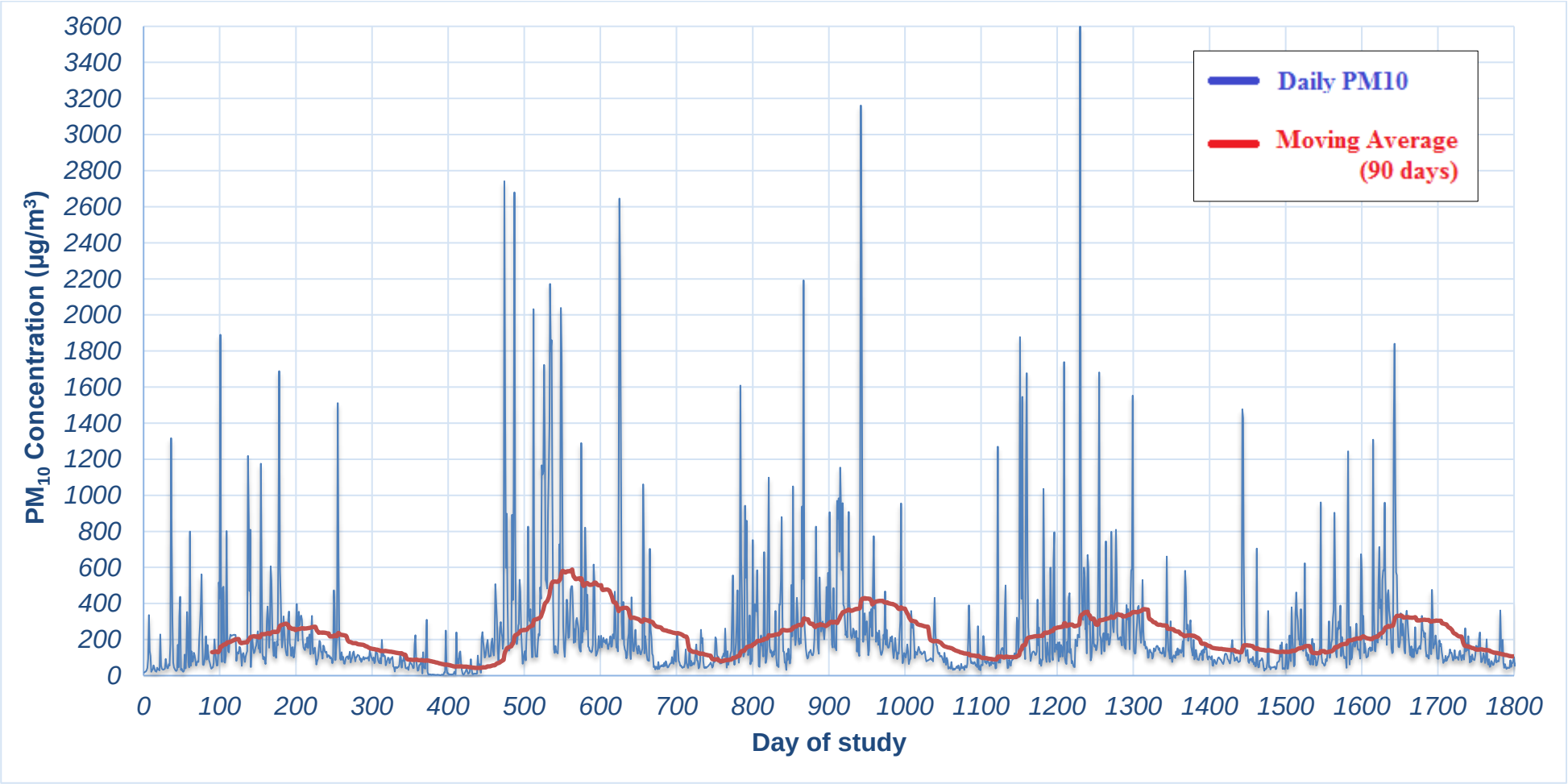


Figure 4.12: Time-series of daily average PM_{10} for the period 2007-2011

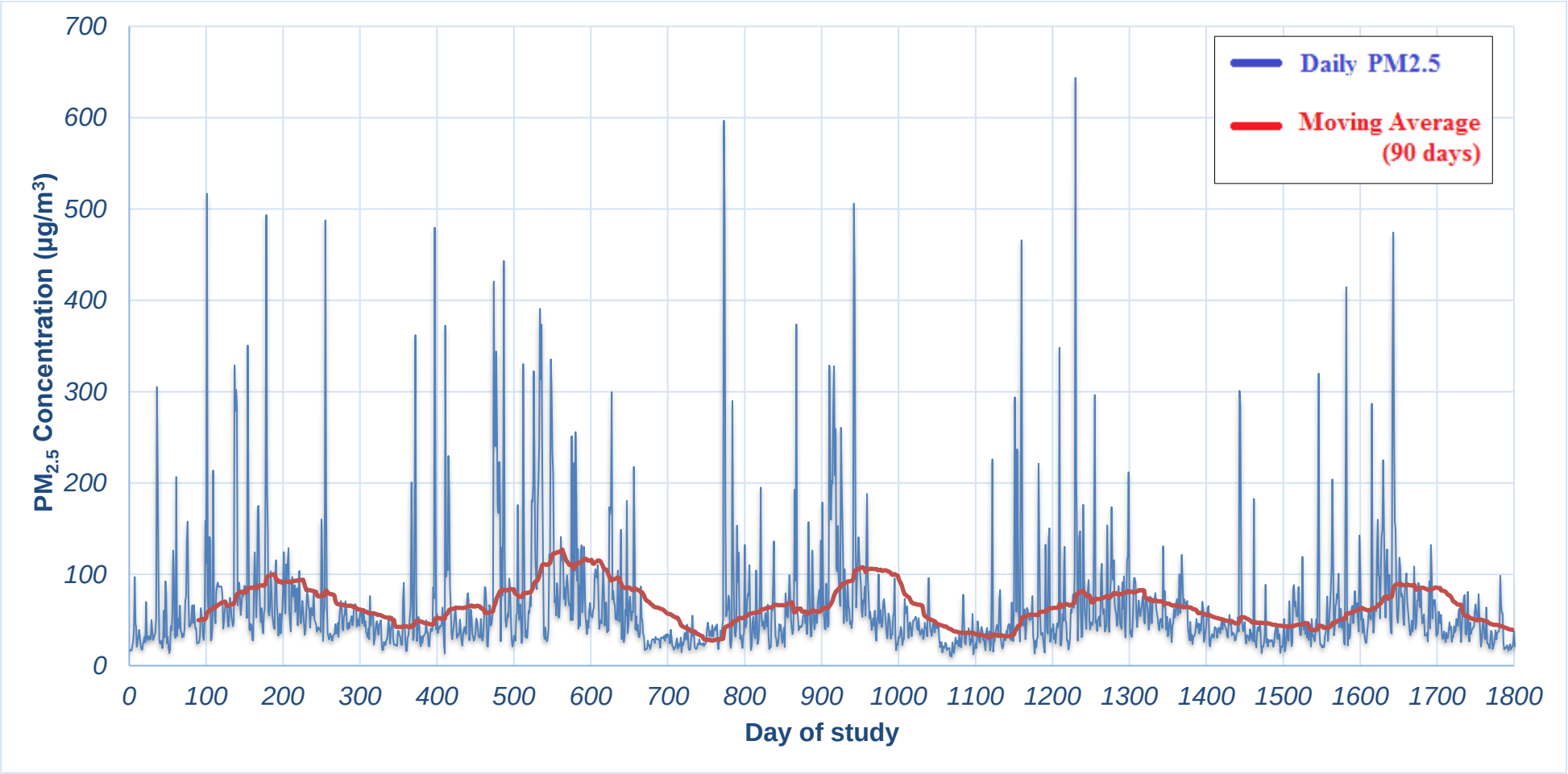


Figure 4.13: Time-series of daily average $PM_{2.5}$ for the period 2007-2011

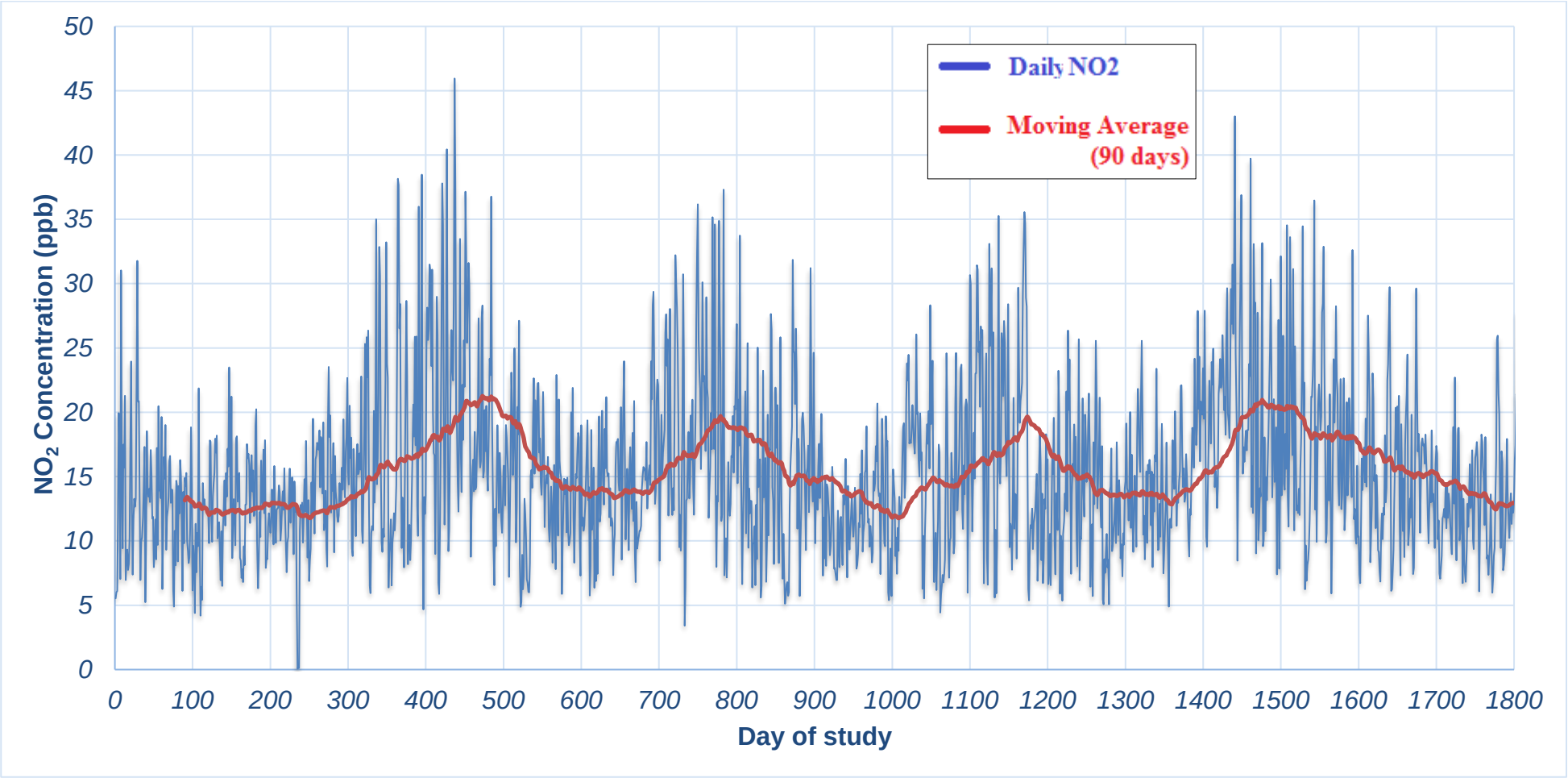


Figure 4.14: Time-series of daily average NO_2 for the period 2007-2011

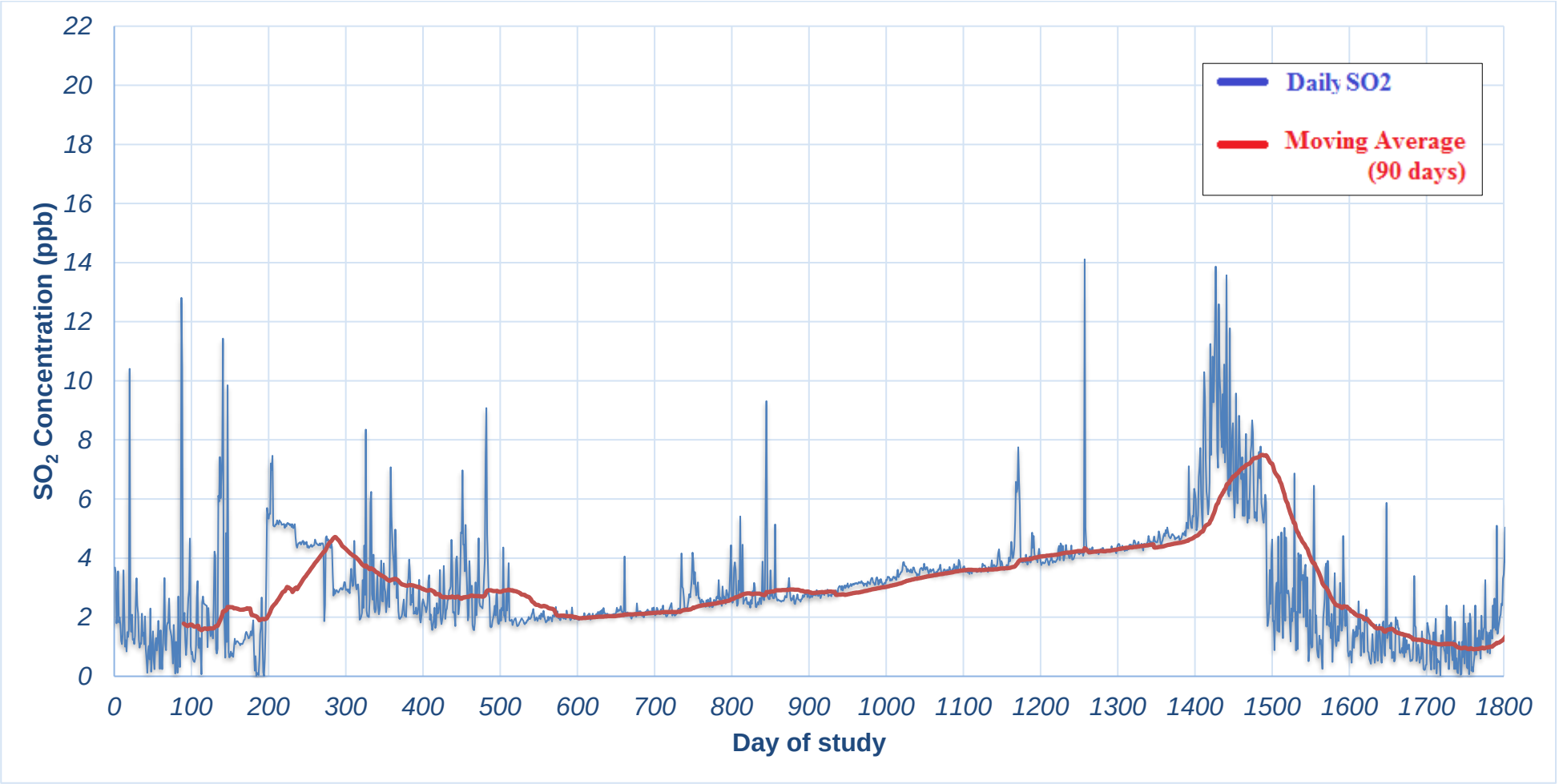


Figure 4.15: Time-series of daily average SO_2 for the period 2007-2011

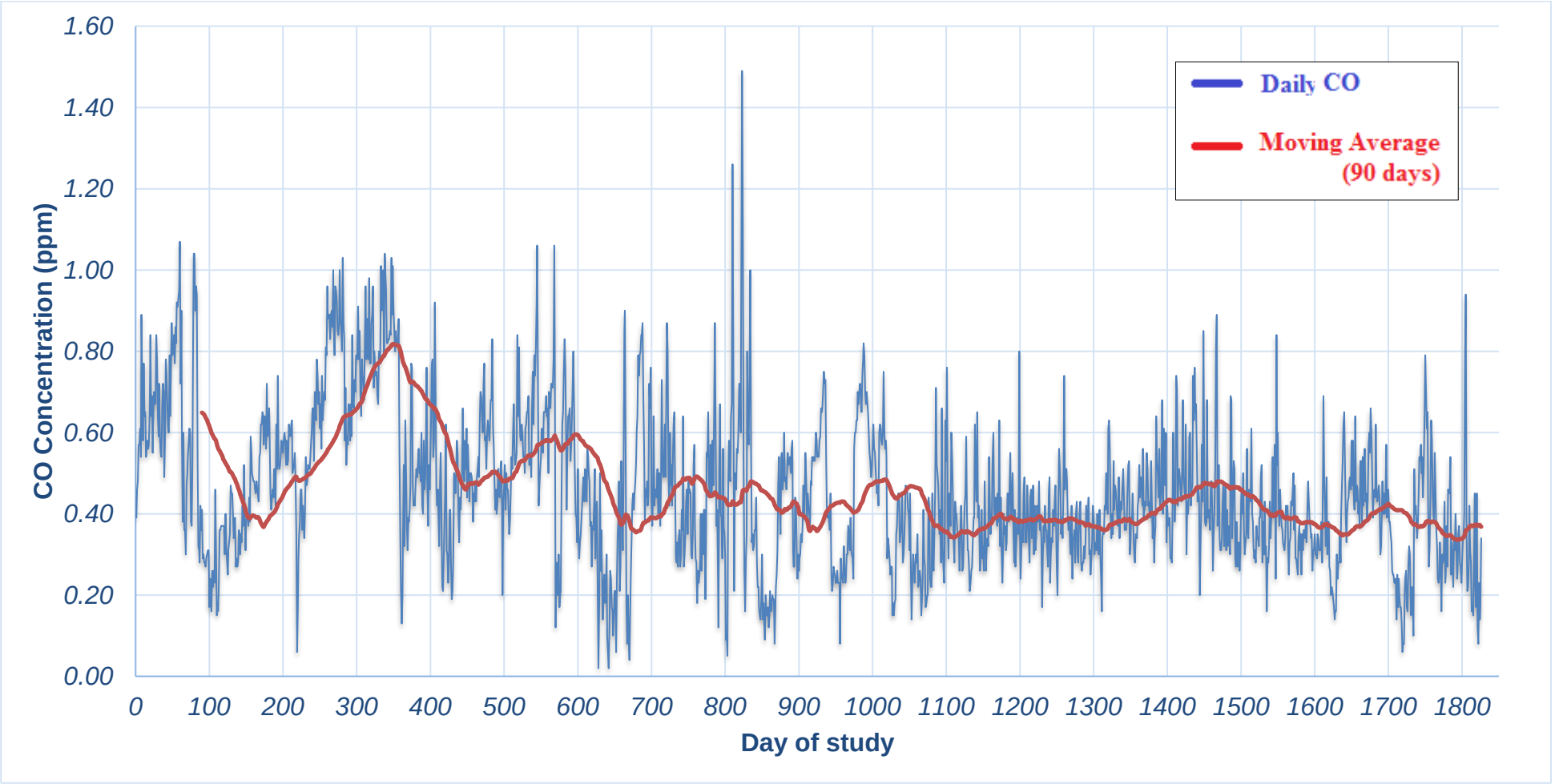


Figure 4.16: Time-series of daily average CO for the period 2007-2011

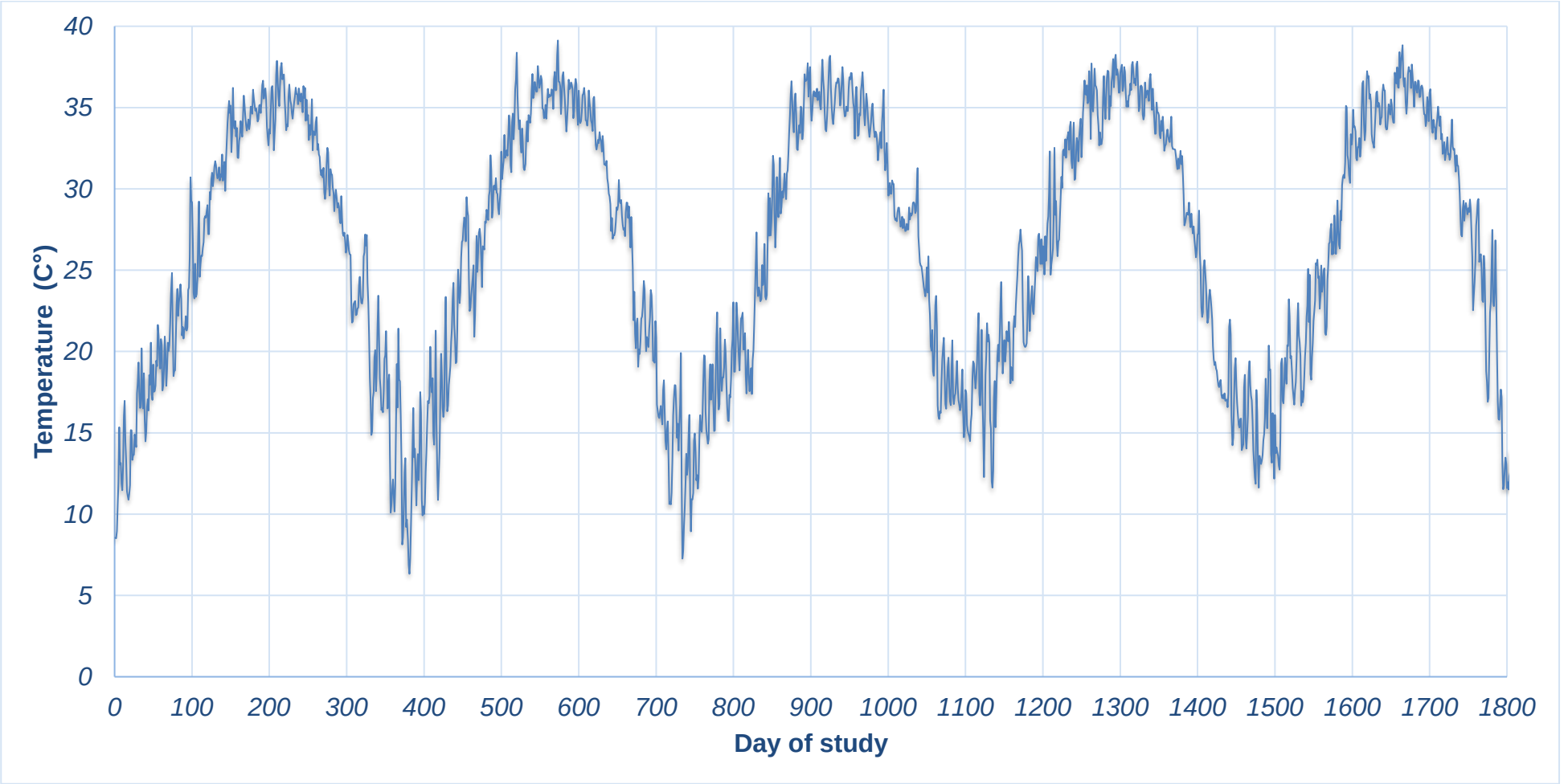


Figure 4.17: Time-series of daily average temperature for the period 2007-2011

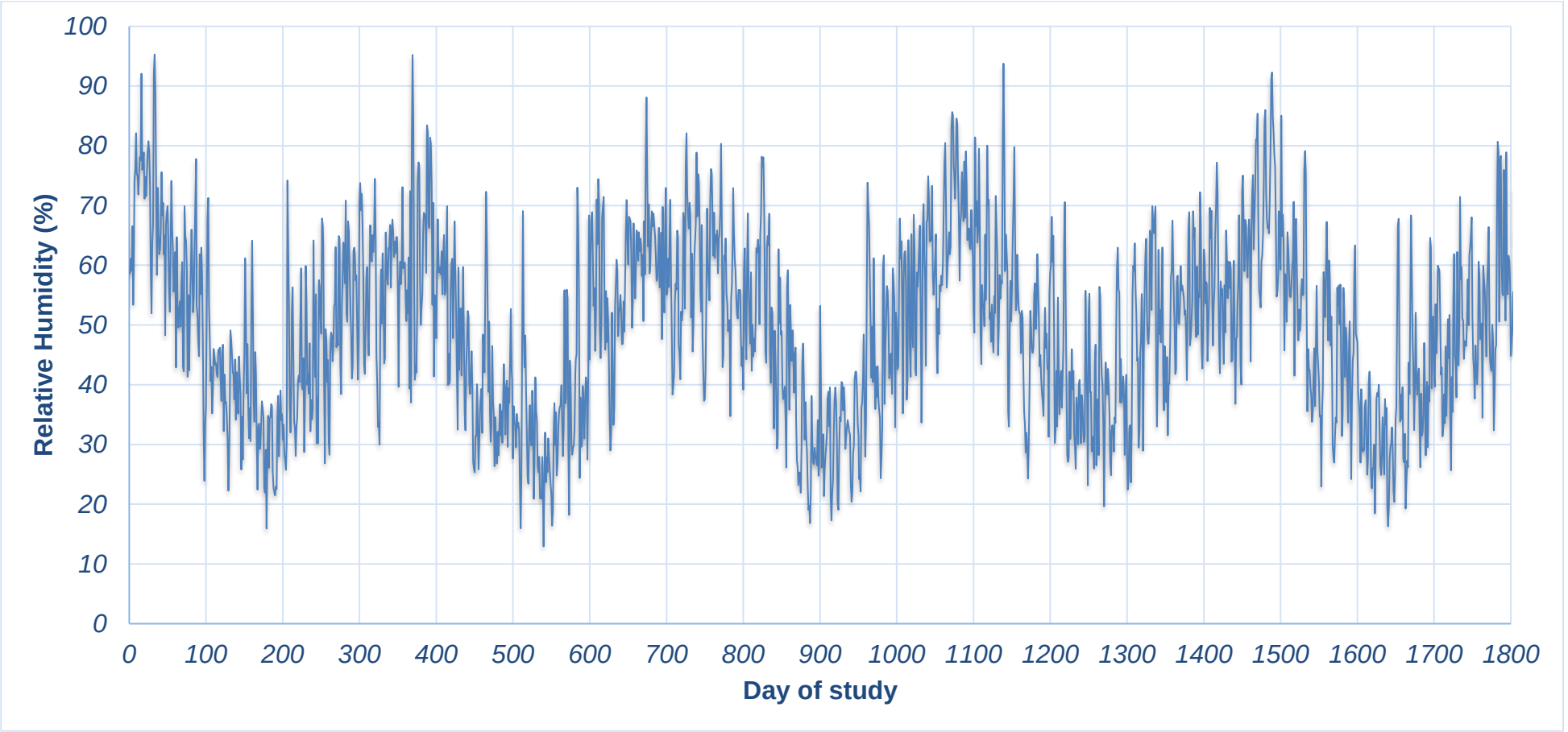


Figure 4.18: Time-series of daily average RH for the period 2007-2011

4.10 Air Quality Exceedance

Comparisons of recorded air quality with Al Jubail Air Quality Standard (AQS) and WHO Air Quality Guidelines (AQG) are shown in Table 4.6. SO_2, $PM_{2.5}$ and PM_{10} exceeded the daily Al Jubail AQS and WHO AQG limits, while $PM_{2.5}$ and PM_{10} also exceeded annual Al Jubail AQS and WHO AQG limits for each of the five years (Table 4.6).

Table 4.6: Daily and annual air quality exceedance

Variables	Averaging Period	Al Jubail AQS	WHO AQQ
PM_{10}	Daily	757 (41.5%) days exceeded limit value (15 μg/m^3)	1618 (88.6%) days exceeded limit value (50μg/m^3)
	Annual	All five years exceeded limit value (50μg/m^3)	All five years exceeded limit value (2 μg/m^3)
$PM_{2.5}$	Daily	1269 (69.5%) days exceeded limit value (35μg/m^3)	1623 (88.9%) days exceeded limit value (25μg/m^3)
	Annual	All five years exceeded limit value (15μg/m^3)	All five years exceeded limit value (10μg/m^3)
SO_2	Daily	Did not exceed limit value	43 (2.3%) days exceeded limit value (7.7ppb)
	Annual	Did not exceed limit value	Did not exceed limit value
NO_2	Daily	Did not exceed limit value	Did not exceed limit value
	Annual	Did not exceed limit value	Did not exceed limit value
CO	Daily	Did not exceed limit value	Did not exceed limit value
	Annual	Did not exceed limit value	Did not exceed limit value

4.11 Correlation among air pollution, weather variables and asthma-related emergency department visits (AEDv)

The correlations among air pollution, weather variables and asthma-related emergency department visits (AEDv) were assessed using Spearman's rank correlation coefficient as the data were not normally distributed (Shapiro Wilk's test p-value < 0.001). Table 4.7 shows Spearman's rank correlation coefficients among air pollution, weather variables and AEDv in Al Jubail Industrial City for the five-year period. AEDv were negatively correlated PM_{10}, $PM_{2.5}$, CO and temperature, and positively correlated with NO_2, SO_2 and relative humidity. A strong positive correlation was observed between PM_{10} and $PM_{2.5}$ (r=0.816).

Table 4.7: Spearman's correlation coefficients between air pollution and AEDv in Al Jubail Industrial City for the period 2007-2011

Spearman's Correlation		CO	NO_2	PM_{10}	$PM_{2.5}$	SO_2
AEDv	Correlation coefficients	-0.04	**0.077****	**-0.255****	**-0.237****	0.02
	Sig. (2-tailed)	0.05	0.00	0.00	0.00	0.43
CO	Correlation coefficients		**0.258****	**-0.086****	0.02	0.01
	Sig. (2-tailed)		0.00	0.00	0.38	0.74
NO_2	Correlation coefficients			**-0.206****	**-0.117****	**0.217****
	Sig. (2-tailed)			0.00	0.00	0.00
PM_{10}	Correlation coefficients				**0.816****	**-0.068****
	Sig. (2-tailed)				0.00	0.00
$PM_{2.5}$	Correlation coefficients					**-0.077****
	Sig. (2-tailed)					0.00

N=1826
**Spearman's rank correlation is significant at the 0.01 level (2-tailed)
*Spearman's rank correlation is significant at the 0.05 level (2-tailed)

4.12 Correlation between AEDv and Weather Variables

The correlations among asthma-related emergency department visits and weather variables were assessed using Spearman's rank correlation coefficient as the data were not normally distributed (Shapiro Wilk's test p-value < 0.001). Table 4.8 shows Spearman's rank correlation coefficients among AEDv and weather variables in Jubail Industrial City for the five-year period. AEDv were negatively correlated with temperature and wind direction, and positively correlated with relative humidity.

Table 4.8: Spearman's correlation coefficients among weather variables and AEDv in Jubail Industrial City for the period 2007-2011

Spearman's Correlation		Temp	RH	WD
AEDv	Correlation coefficients	-0.437**	0.277**	-0.014
	Sig. (2-tailed)	0.000	0.000	0.553
Temp	Correlation coefficients		-0.625**	0.000
	Sig. (2-tailed)		0.000	0.996
RH	Correlation coefficients			-0.125**
	Sig. (2-tailed)			0.000

N=1826
**Spearman's rank correlation is significant at the 0.01 level (2-tailed)

4.13 Single-pollutant models

Table 4.9 summarises the results of the single-pollutant time-series analysis for asthma-related emergency department visits with up to seven lag days. All pollutants studied, except CO, were associated with an increase in daily asthma-related emergency department visits. An increase in inter-quartiles range (IQR) of daily mean concentrations of PM_{10} and $PM_{2.5}$ were associated with an increase in daily asthma-related emergency department visits on the same day (lag 0) and the following day (lag 1). For SO_2, an increase in IQR of daily mean levels (2.0ppb) was associated with increase in daily asthma-related emergency department visits on the same day to following three days (lag 0 to lag 3). For NO_2, an increase in IQR of daily mean levels (7.6ppb) was associated with an increase in daily asthma-related emergency department visits on the previous one (lag 1) and three days before admission (lag 3). The most significant increase in asthma-related emergency department visits were; on the same day of admission (lag 0) for PM_{10} and $PM_{2.5}$, on the previous two days of admission (lag 2) for SO_2 and on the previous three days of admission (lag 3) for NO_2.

Plots of the relative risks of asthma-related emergency department visits against air pollutants concentrations in the single pollutant model are shown in Figure 4.20 to Figure 4.22.

Table 4.9: Relative risks (95% CI) for AEDv per IQR increase in pollutants concentration for 0-7 lag days in the single-pollutant model

Pollutant	Lag days	RR per Inter-Quarter Range (IQR) increase			t-value	
		RR	95% Confidence Interval			
PM_{10} IQR *(140µg/m³)*	**0#**	**1.023**	**1.014**	**1.033**	**4.71**	*****
	1	1.011	1.001	1.022	2.19	*
	2	1.003	0.992	1.014	0.51	
	3	0.996	0.985	1.007	-0.68	
	4	0.997	0.985	1.008	-0.60	
	5	1.004	0.993	1.016	0.73	
	6	1.003	0.992	1.014	0.54	
	7	0.984	0.973	0.996	-2.65	
$PM_{2.5}$ IQR *(36µg/m³)*	**0#**	**1.037**	**1.026**	**1.049**	**6.31**	*****
	1	1.019	1.007	1.031	3.09	*
	2	0.997	0.985	1.010	-0.42	
	3	0.989	0.976	1.002	-1.62	
	4	0.995	0.982	1.009	-0.70	
	5	1.006	0.993	1.020	0.92	
	6	0.995	0.982	1.009	-0.70	
	7	0.975	0.962	0.989	-3.47	
SO_2 IQR *(2.0ppb)*	0	1.040	1.010	1.071	2.61	*
	1	1.052	1.022	1.083	3.44	*
	2#	**1.058**	**1.028**	**1.089**	**3.84**	*****
	3	1.039	1.009	1.070	2.54	*
	4	1.004	0.997	1.011	2.12	
	5	1.003	0.996	1.010	2.38	
	6	1.002	0.995	1.009	3.20	
	7	1.003	0.996	1.010	2.09	
NO_2 IQR *(7.6ppb)*	0	1.001	0.975	1.029	0.10	
	1	1.031	1.005	1.058	2.38	*
	2	1.015	0.990	1.042	1.18	
	3#	**1.036**	**1.010**	**1.062**	**2.71**	*****
	4	1.002	0.998	1.005	1.07	
	5	1.012	0.986	1.038	0.87	
	6	1.008	0.982	1.034	0.62	
	7	1.010	0.984	1.037	0.78	
CO IQR *(0.25ppm)*	0	0.963	0.933	0.993	-2.38	
	1	0.962	0.932	0.993	-2.42	
	2	0.982	0.952	1.014	-1.12	
	3	1.007	0.975	1.039	0.41	
	4	0.983	0.952	1.014	-1.09	
	5	0.999	0.968	1.031	-0.05	
	6	0.990	0.960	1.022	-0.62	
	7	0.987	0.960	1.014	-0.95	

*Statistically Significant (*p*-value<0.001)
 # = The better model after control for temperature, relative humidity, and indicator variables for day of the week and holidays

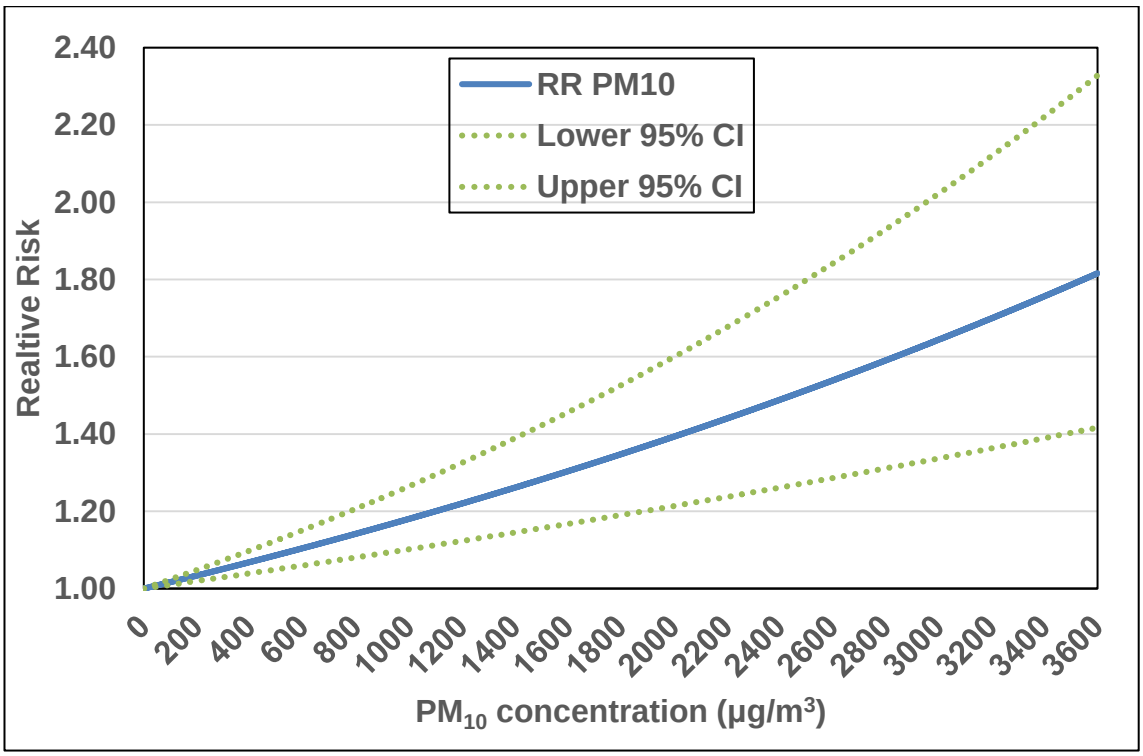


Figure 4.19: Estimates of relative risk of AEDv by PM_{10} concentration (the dashed lines are the 95% confidence interval) in the single-pollutant model

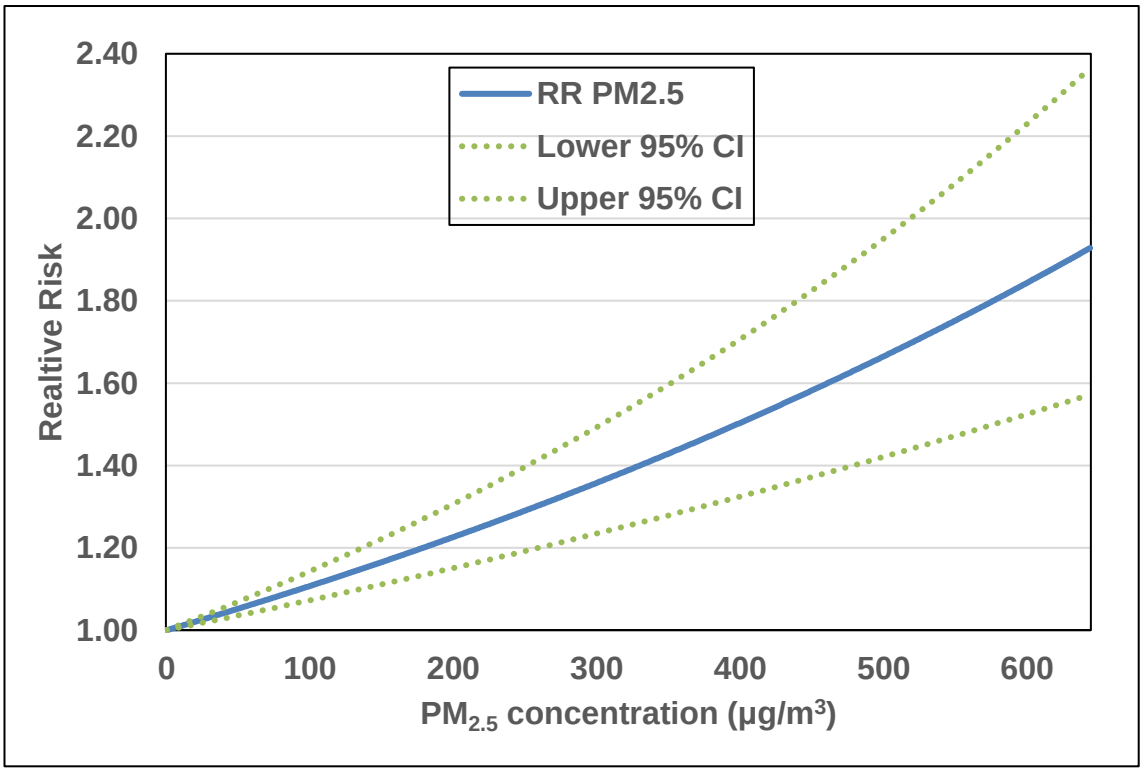


Figure 4.20: Estimates of relative risk of AEDv by $PM_{2.5}$ concentration (the dashed lines are the 95% confidence interval) in the single-pollutant model

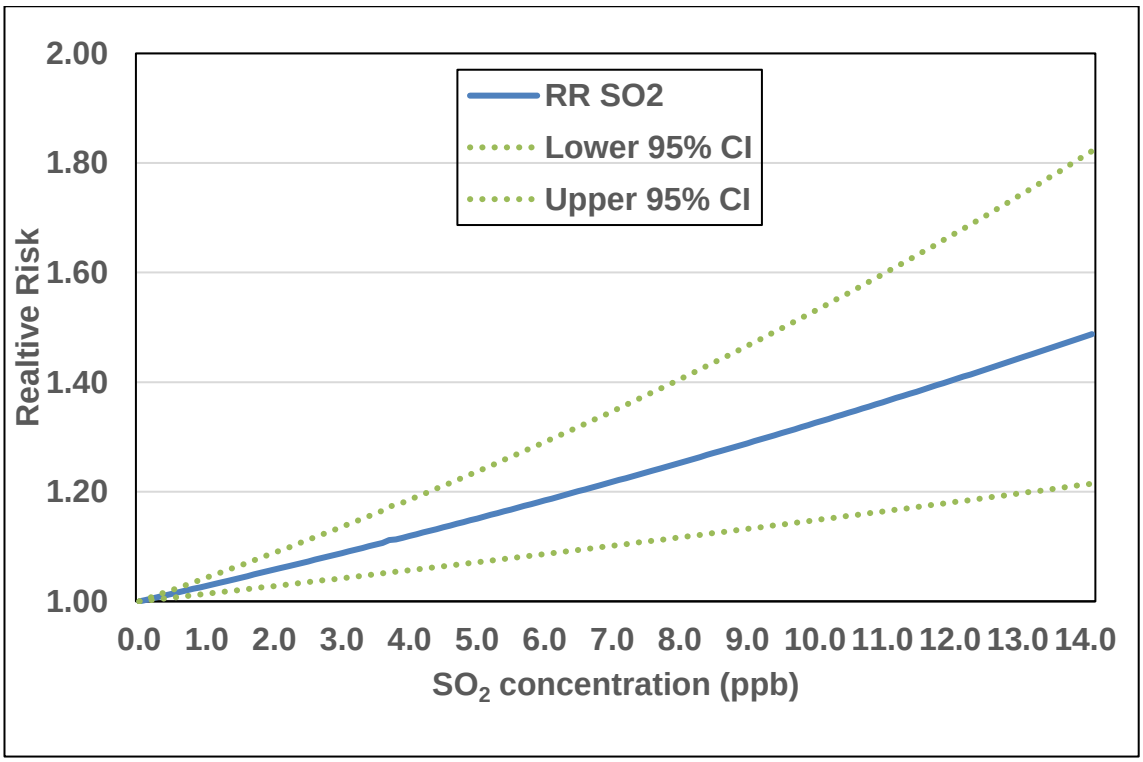


Figure 4.21: Estimates of relative risk of AEDv by SO_2 concentration (the dashed lines are the 95% confidence interval) in the single-pollutant model

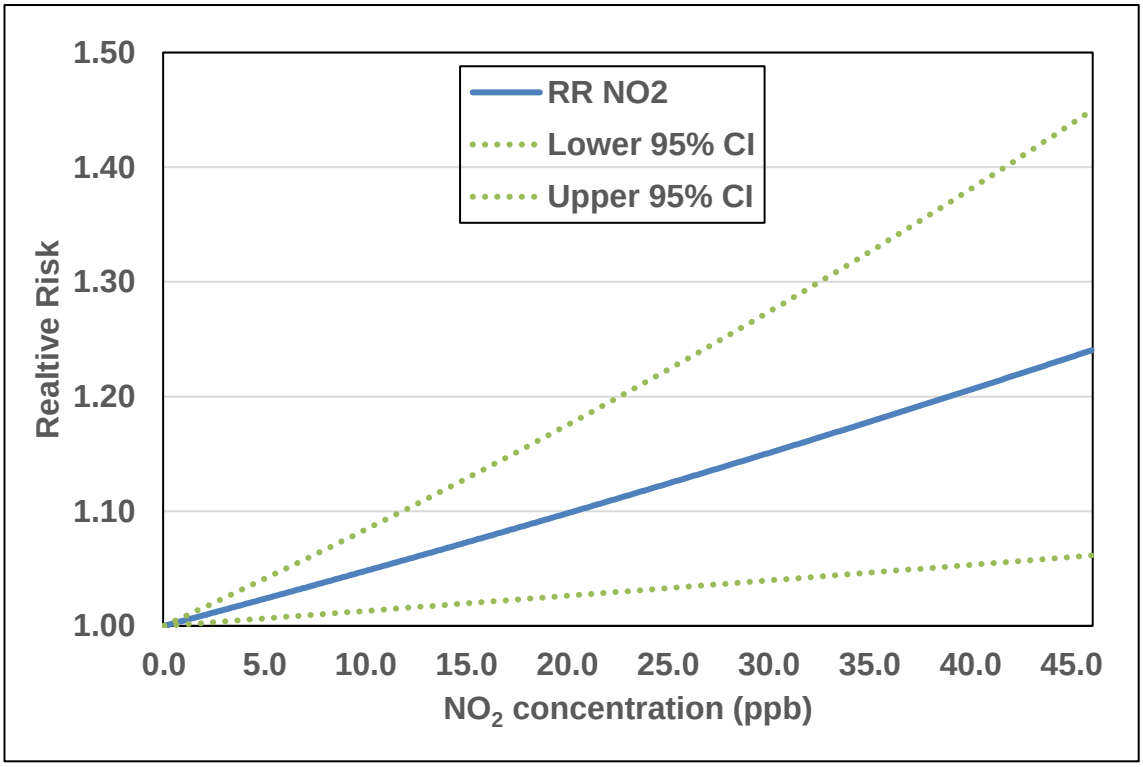


Figure 4.22: Estimates of relative risk of AEDv by NO_2 concentration (the dashed lines are the 95% confidence interval) in the single-pollutant model

4.14 Multi-pollutant model

Multi-pollutant models were used to study the impact on asthma-related emergency department visits of PM_{10}, $PM_{2.5}$, SO_2 and NO_2, adjusting for the other pollutants, as well as relative humidity, temperature and indicator variables for day of the week and holidays. The multi-pollutant models steps described in the method chapter (Step Four: Single and multiple pollutant models, page 82). Pollutants included those which were significant in the single pollutant analysis, and the lag that had the strongest univariate effect was tested using GLM with Poisson regression.

Based on the most significant increase in asthma-related emergency department visits at the single pollutant model, on the same day of admission (lag 0) for PM_{10} and $PM_{2.5}$, on the previous two days of admission (lag 2) for SO_2 and on the previous three days of admission (lag 3) for NO_2 are considered in this multi-pollutant model. The results of relative risks (together with 95% confidence intervals) for asthma-related emergency department visits per IQR increase in pollutant concentration are shown in Table 4.10. The effects of these four pollutants were independent, as the associations remained significant after adjustment for the remaining pollutants which were simultaneously introduced.

The plot of the relative risks of asthma-related emergency department visits against air pollutants concentrations in the multi-pollutant models are shown in Figure 4.23 to Figure 4.26.

Table 4.10: Relative risks (95% Confidence Interval) for AEDv per IQR increase in pollutants concentration for 0-7 lag days in the multi-pollutant models

Pollutant	Lag days	RR per Inter-Quarter Range (IQR) increase			t-statistic	
		RR	95% Confidence Interval			
PM_{10} IQR *(140µg/m^3)*	0	1.022	1.013	1.032	4.50	*
$PM_{2.5}$ IQR *(36µg/m^3)*	0	1.044	1.024	1.066	4.24	*
SO_2 IQR *(2.0ppb)*	2	1.054	1.024	1.085	3.60	*
NO_2 IQR *(7.6ppb)*	3	1.034	1.008	1.061	2.54	*

***** Statistically Significant (**p*-value<0.001) after control for temperature, relative humidity, and indicator variables for day of the week and holidays

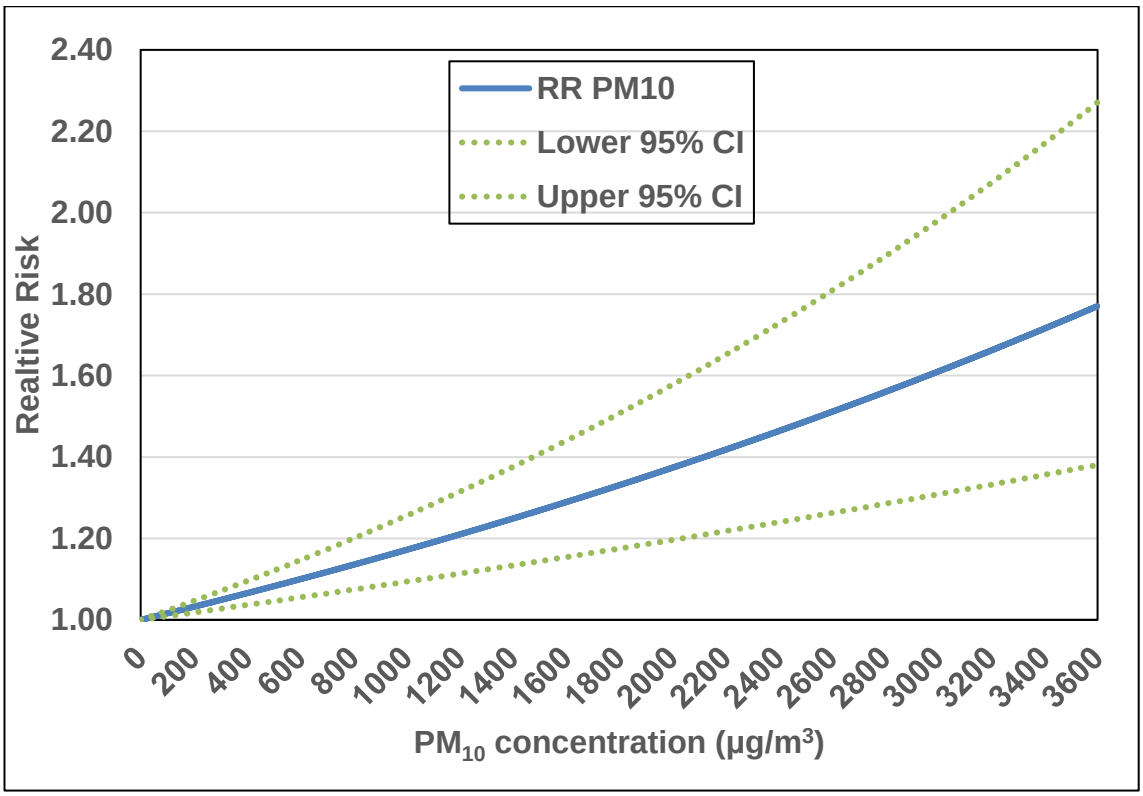


Figure 4.23: Estimates of relative risk of AEDv by PM_{10} concentration (the dashed lines are the 95% confidence interval) in the multi-pollutant model

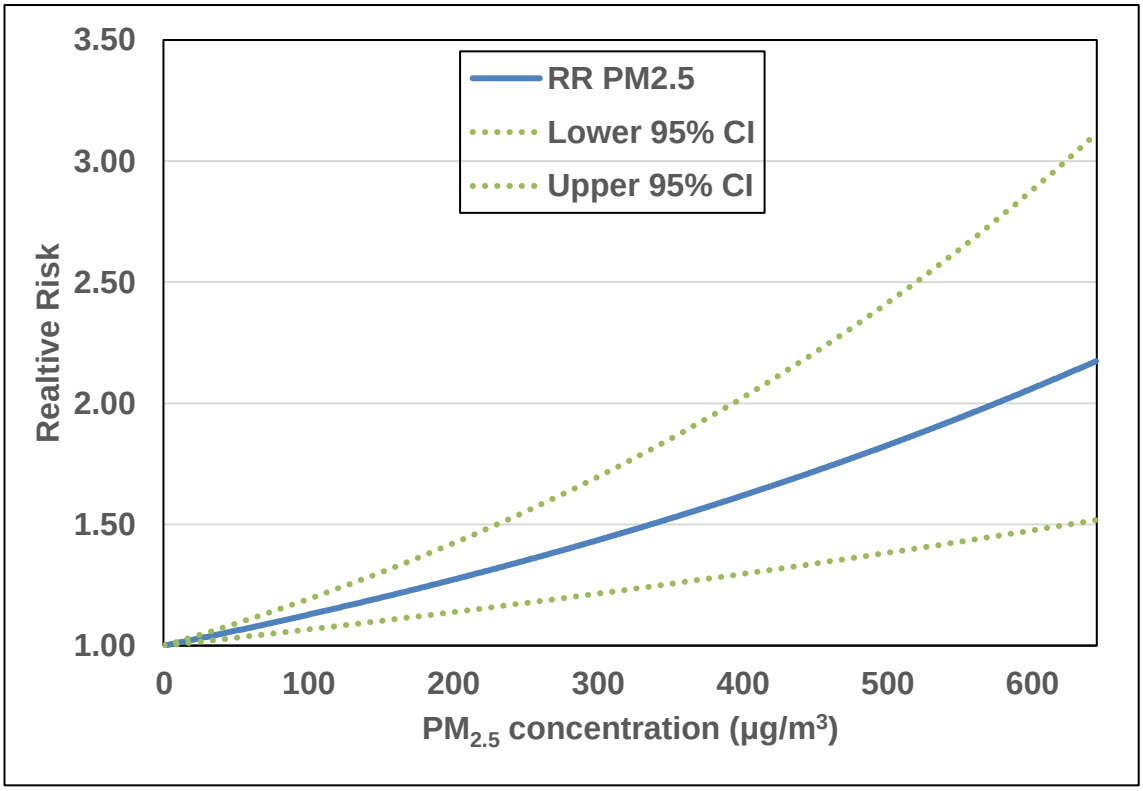


Figure 4.24: Estimates of relative risk of AEDv by $PM_{2.5}$ concentration (the dashed lines are the 95% confidence interval) in the multi-pollutant model

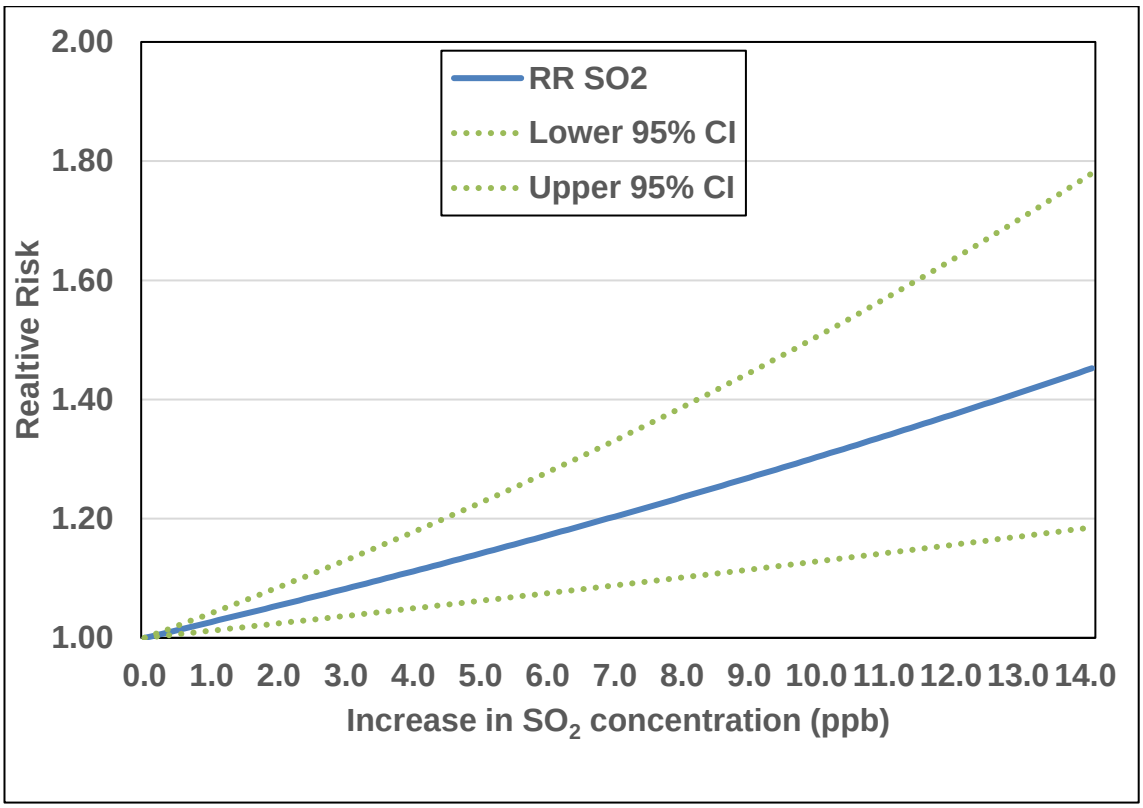


Figure 4.25: Estimates of relative risk of AEDv by SO_2 concentration (the dashed lines are the 95% confidence interval) in the multi-pollutant model

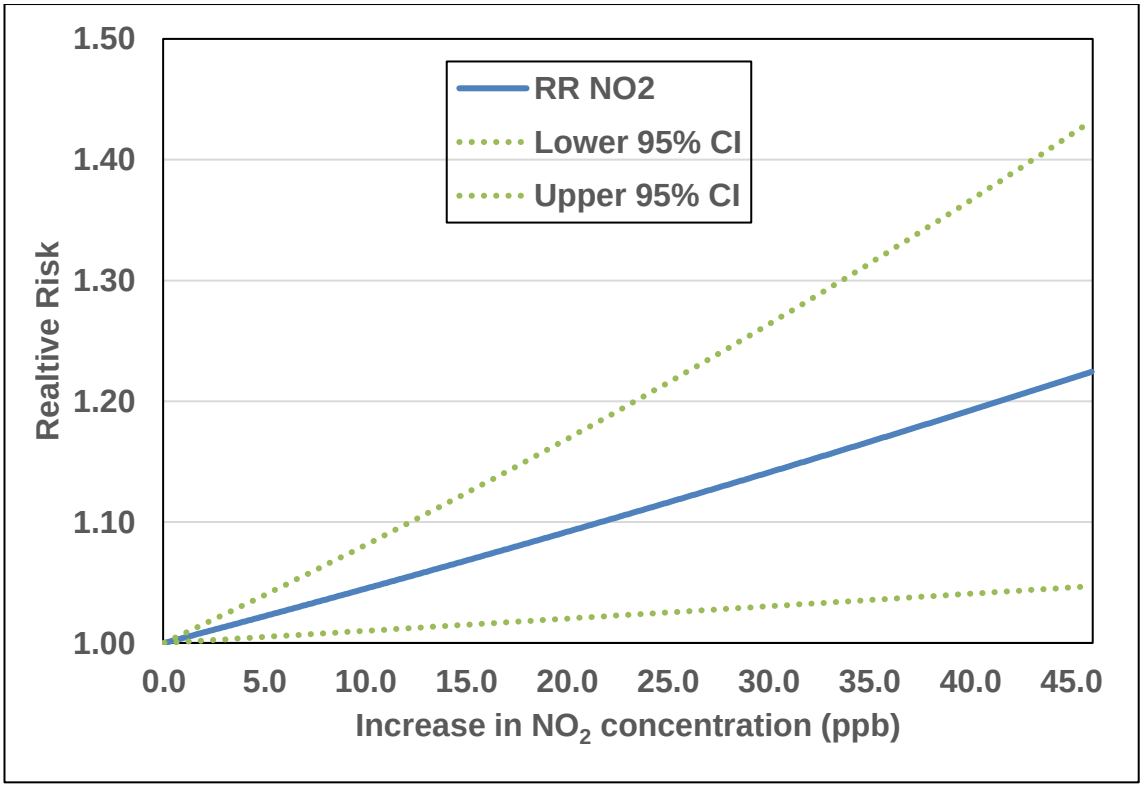


Figure 4.26: Estimates of relative risk of AEDv by NO_2 concentration (the dashed lines are the 95% confidence interval) in the multi-pollutant model

CHAPTER FIVE

Result of Time-Activity Patterns and Microenvironment Exposure

Chapter:5 Result of Time-Activity Patterns and Microenvironment Exposure

5.1 Introduction

This chapter presents the descriptive results of microenvironment exposure including detailed results of students' activities and time spent in each microenvironment. It also presents personal exposure in different microenvironments. The last section includes analysis of the use of fixed-site monitor data as a proxy for personal exposure to $PM_{2.5}$.

5.2 Age, Commuting and Household Members

Table 5.1 shows students characteristics in terms of age, commuting and household members for the 27 students who participated in this study. Their age ranged between 16 and 18 years (mean = 17.1, median = 17, and standard deviation (SD) ± 0.75 years). The majority of the students, 22 (81.5%), used car for transport to and from school. Four (14.8%) students used bus and only one student reported walking to and from school. Total household members ranged from 3 to 10, with mean 7.2, median 7, and SD ± 1.9.

Table 5.1: Age, transportation means and household members for 27 study participants

Variable	No.	Percent %
Age		
16 years	6.0	22.2
17 years	12.0	44.4
18 years	9.0	33.3
Commuting		
Walk	1.0	3.7
Bus	4.0	14.8
Car	22.0	81.5
Household Members		
N ≥7	17.0	63.0
N <7	10.0	37.0

5.3 House Characteristics

The majority of the students 20 (74%) were living in owner-occupied homes, while the other 7 (26%) students were living in a rented home (See Table 5.2). Eighteen (66.7%) students stated that their house type was detached and one student lived in an apartment. Six (22.2%) of the students reported that they had an open fire room at home, and of these, four had open fires located inside the house. The cooking fuel source was electricity in 18 (66.7%) homes, gas/electricity combined in 5 (18.5%) homes, whereas 4 homes used bottled gas. The main ventilation source was air-conditioning in 19 (70.4%) homes, four homes used open windows and one home used fans (See Table 5.2).

Table 5.2: House characteristics of 27 study participants

Variable	No.	Percent %
House Type		
Detached	18.0	66.7
Bungalow	5.0	18.5
Semi-detached	3.0	11.1
Apartments	1.0	3.7
Open Fire Place		
Outside House	2.0	7.4
Inside House	4.0	14.8
Total number	21.0	77.8
Cooking Fuel		
Both Gas/Electric	5.0	18.5
Bottled Gas	4.0	14.8
Electric	18.0	66.7
Ventilation		
Air-conditioning	19.0	70.4
Open windows	5.0	18.5
Fan	1.0	3.7
None	2.0	7.4

5.4 Potential Exposure Sources and Places

Eleven (41.7%) of the students were regularly exposed to incense and seven (24.7%) were exposed to passive smoking and nine (33.6%) were exposed to other sources. Most of those exposures ((66.7%)) occurred at home, and nine (33.3%) in other locations (See Table 5.3).

Table 5.3: Type and place of exposure

Variable	No.	Percent %
Regularly Exposed		
Passive smoking	7.0	24.7
Other sources	9.0	33.6
Incense	11.0	41.7
Place of Exposure		
Home	18.0	66.7
Other locations	9.0	33.3

5.4.1 Time spent in different microenvironments

Based on the data from the time-activity diary, Figure 5.1 shows the time students spent in different microenvironments. Overall, the students spent 58.4% of their time at home-indoors followed by school indoors (25.9%), Transport (6.3%), indoors away from home/school (4.3%), outdoors away from home/school (2.3%), outdoors at home (1.7%) and outdoors at school (1.1%).

Figure 5.2 compares the total amount of time spent in the major microenvironments, which indicates that the majority of the total time spent by students was indoors at 88.6%, followed by transport and outdoor microenvironment at 6.3% and 5.1% respectively.

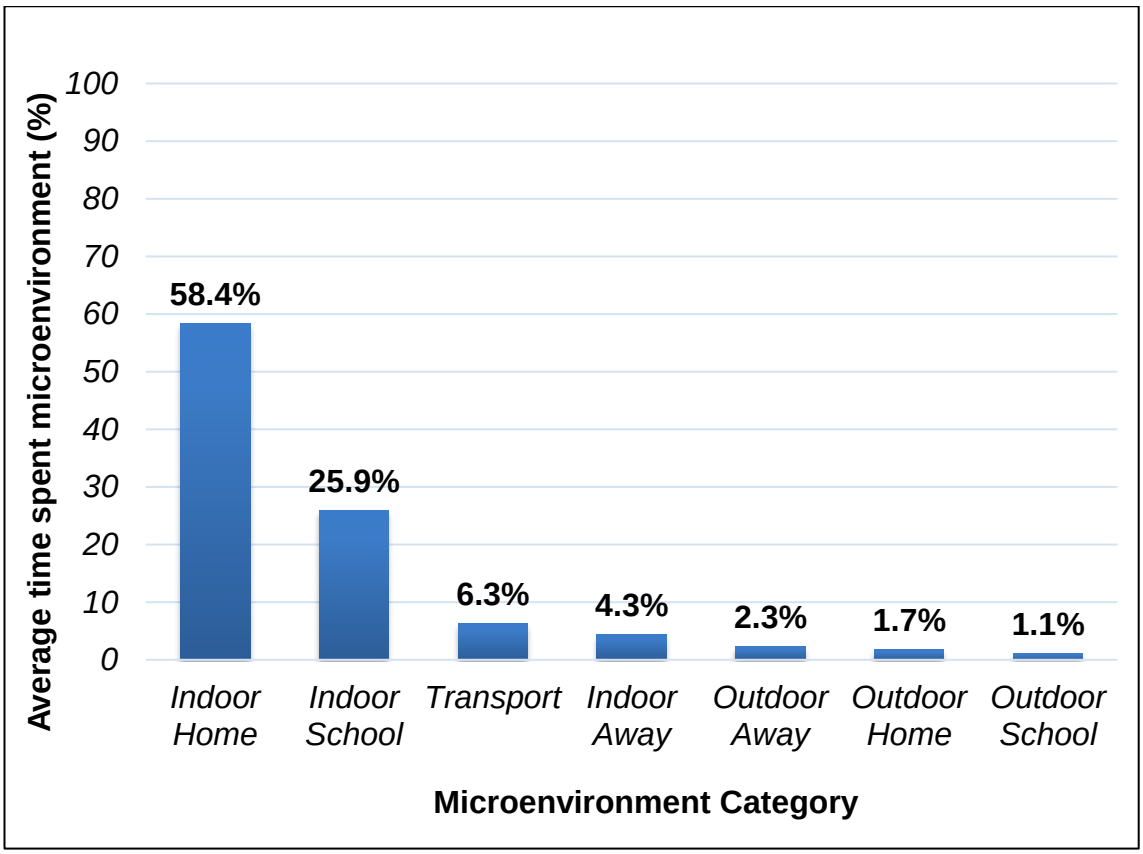


Figure 5.1: Time spent in different microenvironments

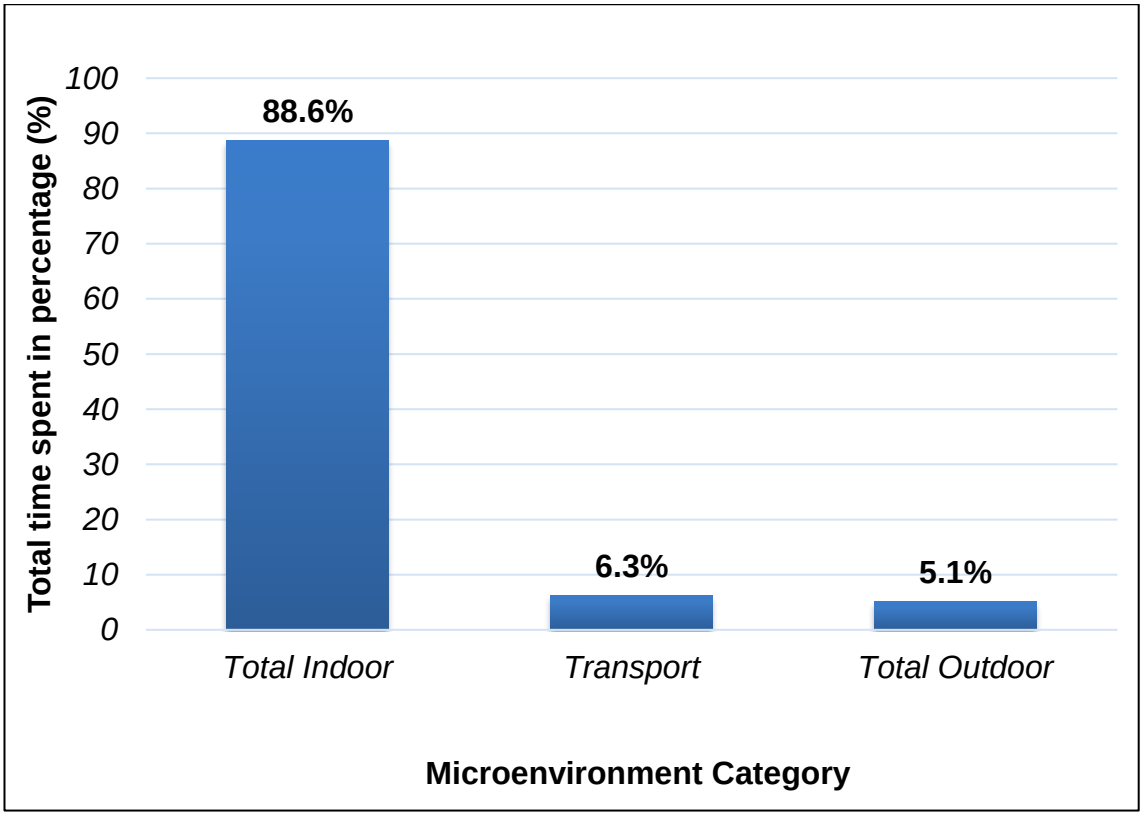


Figure 5.2: Total time spent in major microenvironments

5.5 Descriptive Analysis of All Participants' Personal Exposure to $PM_{2.5}$

Table 5.4 shows the personal $PM_{2.5}$ ($\mu g/m^3$) monitoring at each minute for all 27 participants. The total personal monitoring time measured in this study was 618.2 hours. The total personal $PM_{2.5}$ ($\mu g/m^3$) mean was 37.0 and the median was 29.0. The lowest level of personal $PM_{2.5}$ exposure was 0 ($\mu g/m^3$) while the highest level was 3327 ($\mu g/m^3$). These descriptive statistics clearly suggested that distributions were not normally distributed (Shapiro-Wilk $p < 0.001$) as shown in Figure 5.3.

Table 5.4: Descriptive statistics for all 27 participants

ID	$PM_{2.5}$ (μg/m^3) concentrations measured at 1-minute intervals								Time (hours)
	Mean	StDev	Median	Q1	Q3	IQR	Min	Max	
1	12.7	7.2	10.0	7.0	17.0	10.0	4.0	45.0	21.2
2	44.3	66.4	33.0	16.0	44.3	28.3	10.0	571.0	23.9
3	38.7	22.5	36.0	25.0	50.0	25.0	4.0	154.0	24.0
4	27.3	19.9	22.0	18.0	29.0	11.0	4.0	224.0	24.0
5	51.3	19.3	47.0	38.0	59.0	21.0	9.0	129.0	24.0
6	28.5	23.4	24.0	17.0	29.0	12.0	0.0	214.0	23.5
7	27.8	15.3	22.0	17.0	36.0	19.0	11.0	147.0	19.5
8	54.5	58.9	37.0	28.0	55.0	27.0	14.0	483.0	24.0
9	31.7	27.1	24.0	16.0	41.0	25.0	8.0	495.0	24.0
10	49.1	19.7	45.0	31.0	63.0	32.0	22.0	154.0	23.5
11	34.3	13.9	37.0	26.0	42.8	16.8	5.0	182.0	24.0
12	37.4	16.0	32.0	26.0	47.0	21.0	15.0	110.0	24.0
13	32.6	13.5	29.0	26.0	34.0	8.0	11.0	141.0	22.7
14	17.8	13.9	16.0	12.0	20.0	8.0	5.0	308.0	23.8
15	49.8	93.8	35.0	26.0	61.0	35.0	11.0	1712.0	24.0
16	28.7	16.3	23.0	20.0	32.3	12.3	3.0	152.0	19.1
17	17.6	9.8	18.0	11.0	23.0	12.0	3.0	104.0	11.7
18	38.9	15.1	33.0	29.0	47.0	18.0	20.0	215.0	24.0
19	47.9	184.0	19.0	7.0	37.0	30.0	3.0	3327.0	23.0
20	69.2	173.2	34.0	26.0	45.0	19.0	5.0	1339.0	24.0
21	39.4	100.0	25.0	20.0	32.0	12.0	9.0	1986.0	23.9
22	38.0	15.0	35.0	28.0	43.0	15.0	12.0	198.0	23.7
23	15.6	17.7	7.0	5.0	21.0	16.0	2.0	196.0	23.8
24	43.0	42.5	38.0	32.0	46.0	14.0	0.0	928.0	24.0
25	40.6	16.9	38.0	28.0	51.0	23.0	14.0	287.0	23.9
26	44.9	63.7	27.0	21.0	39.0	18.0	10.0	565.0	23.6
27	21.5	18.8	19.0	15.0	25.0	10.0	11.0	491.0	23.6
Overall Average	**37.0**	**63.7**	**29.0**	**19.0**	**42.0**	**23.0**	**0.0**	**3327.0**	**22.9**

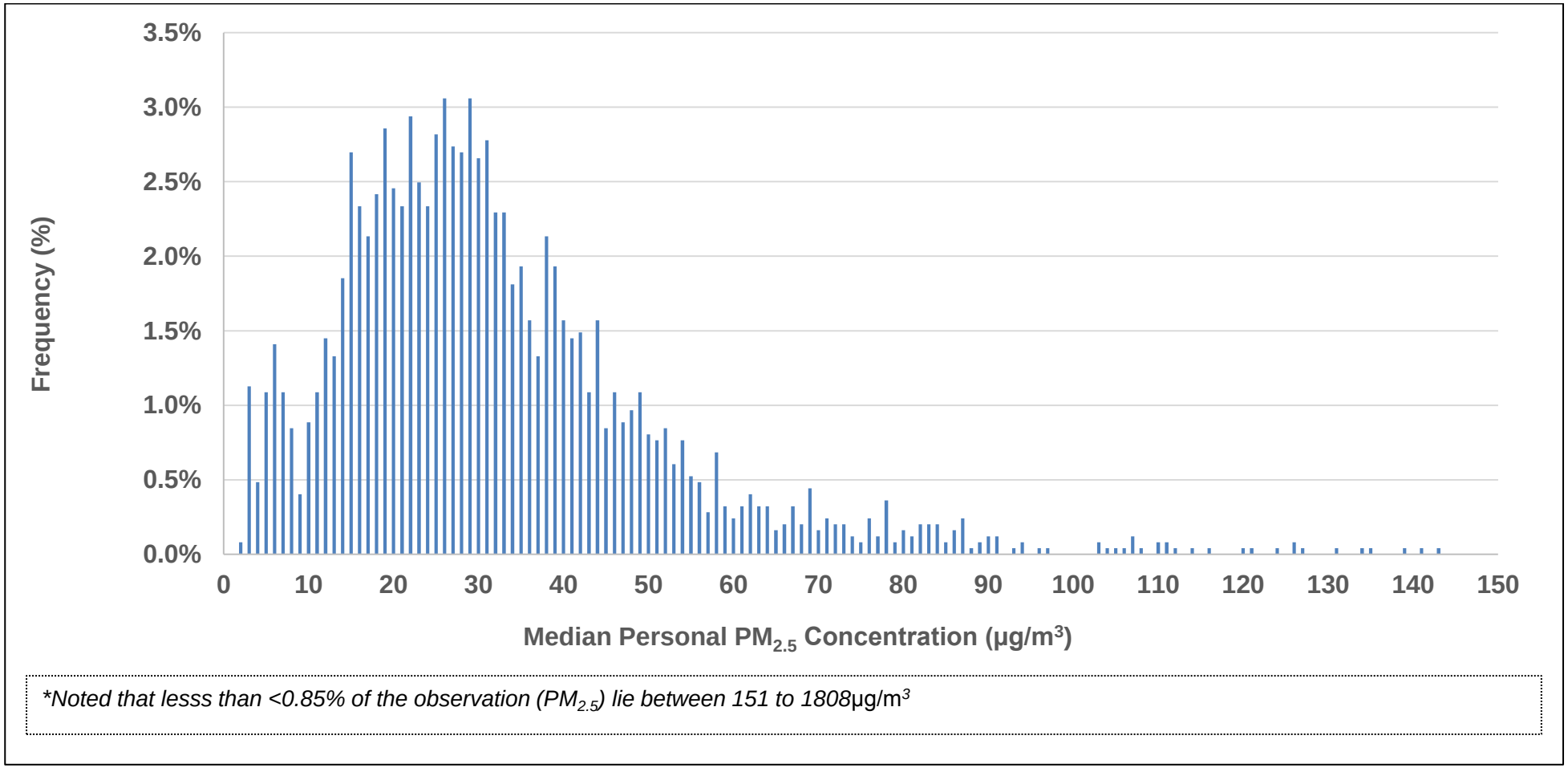


Figure 5.3: Distribution of median personal $PM_{2.5}$ levels for all 27 participants

5.6 Personal Exposure to $PM_{2.5}$ and Time-activity Diary

The activities recorded in the 15-minute resolution time-activity diary were divided into several major categories reflecting key microenvironments (e.g. at home, at school, at other general places), activities (e.g. commuting, physical activity), potential exposure to PM sources (e.g. near to potential exposure sources), and time-periods (e.g. after/before midnight). The personal $PM_{2.5}$ levels were measured at one-minute intervals. Since personal $PM_{2.5}$ concentrations were not normally distributed (Shapiro Wilk's test p-value < 0.001), for each microenvironment, activity, potential exposure source and time period, the associated $PM_{2.5}$ exposure was calculated based on the median of the 15 minutes intervals within that category.

5.6.1 Personal exposure at home

Table 5.5 shows the personal exposure to $PM_{2.5}$ measured within the key microenvironments associated with the home. The summary of descriptive analyses is shown as medians, 25 percentile and 75 percentile, because the distribution of data did not fit a normal pattern (Shapiro Wilk's test P<0.05). The $PM_{2.5}$ levels varied greatly, having a median of 28.0, inter-quartile range (IQR) of 23.0 and range of 2-1808μg/m^3. The personal median $PM_{2.5}$ level differed significantly between the home microenvironments (Kruskal-Wallis p-value <0.001). The highest median $PM_{2.5}$ levels were measured in the living room and home yard (both 40.0μg/m^3) followed by kitchen (35.5μg/m^3), bathroom (34.0 g/m^3) and guest room (30.0μg/m^3). The median levels of $PM_{2.5}$ in the bedroom were significantly lower than other home microenvironments (Mann-Whitney p-value < 0.001).

Table 5.5: Personal exposure to $PM_{2.5}$ measured in home Microenvironments

Home	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Living room	62.0	17.0%	49.6	53.0	40.0	23.3	58.0	34.8	3.0	443.0	<0.001
Home yard	8.0	2.0%	45.3	22.5	40.0	31.3	56.3	25.0	14.0	93.0	
Kitchen	2.5	1.0%	214.8	560.1	35.5	28.3	62.3	34.0	7.0	1808.0	
Bathroom	3.3	1.0%	69.7	96.5	34.0	26.5	76.5	50.0	13.0	381.0	
Guest room	12.3	3.0%	34.1	13.4	30.0	23.0	43.0	20.0	19.0	69.0	
***Bedroom**	285.0	76.0%	31.6	50.8	27.0	17.0	36.0	19.0	2.0	1181.0	
Total	373.0	100.0%	36.5	68.5	28.0	18.0	41.0	23.0	2.0	1808.0	

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.6.2 Personal exposure at school

Personal exposure to $PM_{2.5}$ levels was measured at eight different microenvironments at school which includes science laboratory, schoolyard, common room, library, corridor, class, sport hall and head office (see Table 5.6). The $PM_{2.5}$ levels associated with these exposures varied greatly median = 25.0, inter-quartile range (IQR) = 16.0, and range 5-121µg/m^3. The personal median $PM_{2.5}$ level differed significantly between the school microenvironments (Kruskal-Wallis p-value <0.001). The highest $PM_{2.5}$ median levels were measured in the science laboratory (42.5µg/m^3) followed by schoolyard (36.0µg/m^3), common room (30.0 g/m^3), library (29.5µg/m^3), corridor (25.0µg/m^3), class (24.0µg/m^3), sport hall (23.5µg/m^3). The median level of $PM_{2.5}$ in the head office (where participant interviews took place) was significantly lower, at 18.0µg/m^3, compared to other school microenvironments (Mann-Whitney p-value < 0.001).

Table 5.6: Personal exposure to $PM_{2.5}$ measured at school microenvironments

School	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Science lab	3.0	2.0%	46.3	17.2	42.5	38.0	61.8	23.8	22.0	74.0	<0.001
School yard	8.3	5.0%	39.1	16.4	36.0	29.5	48.0	18.5	14.0	80.0	
Common room	32.5	19.0%	30.7	11.7	30.0	22.8	38.3	15.5	6.0	62.0	
Library	1.0	1.0%	29.0	11.2	29.5	18.3	39.3	21.0	17.0	40.0	
Corridor	13.3	8.0%	27.6	12.9	25.0	18.0	35.5	17.5	9.0	83.0	
Class	99.3	58.0%	27.2	14.6	24.0	18.0	32.0	14.0	8.0	121.0	
Sport hall	9.0	5.0%	27.3	10.9	23.5	19.3	32.5	13.3	15.0	54.0	
***Head office**	5.8	3.0%	18.8	11.3	18.0	7.0	27.0	20.0	5.0	50.0	
Total	172.0	100.0%	28.5	14.3	25.5	19.0	35.0	16.0	5.0	121.0	

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.6.3 Personal exposure at other locations away from home/school

Personal exposure to $PM_{2.5}$ levels was measured at six different microenvironments at other locations away from home and school, including the gym, park, beach, shops, friend house and mosque as shown in Table 5.7. The $PM_{2.5}$ levels across these away from home/school microenvironments varied greatly: median = 41.0, inter-quartile range (IQR) = 22.6, and range 7-539µg/m^3. The highest $PM_{2.5}$ median levels were measured in the gym (47.0µg/m^3) followed by park (44.0µg/m^3), beach (43.0µg/m^3). While the lowest $PM_{2.5}$ median levels were measured in the shops and friend's house (38.5µg/m^3). There was no significant difference in personal exposure to $PM_{2.5}$ between other locations away from home/school as shown in Table 5.7 (Kruskal-Wallis p-value = 0.452).

Table 5.7: Personal exposure to $PM_{2.5}$ measured at other locations away from home/school

Other Locations	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Gym	6.3	19.0%	70.2	104.4	47.0	35.0	52.0	17.0	25.0	539.0	0.452
Park	1.5	4.0%	45.5	5.2	44.0	42.8	47.0	4.3	42.0	56.0	
Beach	9.0	27.0%	46.6	22.3	43.0	26.0	55.5	29.5	18.0	90.0	
Shops	6.0	18.0%	44.2	22.1	38.5	29.3	50.0	20.8	16.0	96.0	
Friend house	4.5	13.0%	54.7	42.8	38.5	23.8	84.9	61.1	18.0	169.0	
Mosque	6.3	19.0%	37.6	15.2	37.0	28.5	47.5	19.0	7.0	67.0	
Total	33.5	100.0%	49.9	50.7	41.0	30.7	53.3	22.6	7.0	539.0	

5.6.4 Personal exposure when commuting

Table 5.8 shows the personal exposure to $PM_{2.5}$ measured within the key microenvironments associated during commuting which includes walking, bus and car. The $PM_{2.5}$ levels across these transport modes varied greatly: median = 38.0, inter-quartile range (IQR) = 27.7, and range 4-371µg/m^3. The highest $PM_{2.5}$ median levels were measured during walking (46.0µg/m^3) followed by bus (42.5µg/m^3). While the lowest $PM_{2.5}$ median levels were measured in the car (35.5µg/m^3). The personal exposure to $PM_{2.5}$ was not statistically different according to commuting mode as shown in Table 5.8 (Kruskal-Wallis p-value = 0.099).

Table 5.8: Personal exposure to $PM_{2.5}$ measured during commuting

Commuting	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Walk	9.5	21.0%	41.7	18.6	46.0	36.0	53.0	17.0	4.0	76.0	0.099
Bus	2.5	5.0%	54.2	27.3	42.5	35.3	76.8	41.5	27.0	111.0	
Car	34.0	74.0%	45.8	44.9	35.5	26.0	51.0	25.0	6.0	371.0	
Total	46.0	100.0%	45.3	40.0	38.0	27.0	51.7	27.7	4.0	371.0	

5.6.5 Personal exposure measured near specific exposure sources

Table 5.9 compares the personal exposure to $PM_{2.5}$ measured when the participant noted exposure to a range of potential exposure sources (incense, dust, chemical, cooking, smoking and perfume). The $PM_{2.5}$ levels associated with these 'exposures' varied greatly: median = 69.0, inter-quartile range (IQR) = 60.3, and range 7-1808μg/m^3. The personal median $PM_{2.5}$ level differed significantly between the potential exposure sources (Kruskal-Wallis p-value <0.001). The highest $PM_{2.5}$ median levels were measured when the participant noted exposure to incense (107.0μg/m^3) followed by dust (78.0μg/m^3), chemical (74.0μg/m^3), cooking (59.0μg/m^3) and smoking (40.0μg/m^3). The median (IQR) levels of $PM_{2.5}$ when exposed to perfume was significantly lower, at 25.5 (53.5), compared to other potential exposure sources (Mann-Whitney p-value < 0.001) as shown in Table 5.9. Some 'exposures' may not have lasted the full 15 minutes, others longer, so this uncertainty should be considered when review these results.

Table 5.9: Personal exposure to $PM_{2.5}$ with the person near potential exposure sources

Variables	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Incense	7.5	19.0%	246.4	395.8	107.0	78.5	149.5	71.0	22.0	1808.0	<0.001
Dust	8.0	20.0%	75.3	20.9	78.0	67.0	86.0	19.0	29.0	121.0	
Chemical	0.5	1.0%	74.0	22.6	74.0	58.0	90.0	32.0	58.0	90.0	
Cooking	17.8	44.0%	73.7	62.6	59.0	41.0	78.0	37.0	9.0	443.0	
Smoking	5.8	14.0%	109.5	134.7	40.0	35.0	167.0	132.0	27.0	539.0	
***Perfume**	1.0	2.0%	33.5	29.8	25.5	10.8	64.3	53.5	7.0	76.0	
Total	40.5	100.0%	110.1	192.2	69.0	42.7	103.0	60.3	7.0	1808.0	

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.6.6 Personal exposure and physical activity

Table 5.10 compares the personal exposure to $PM_{2.5}$ measured according to types/levels of physical activity (exercise, standing, seating and other activities). The total personal concentrations were significantly different across these types/levels of activity (Kruskal-Wallis p-value < 0.001). It can be seen from Table 5.10 that personal median $PM_{2.5}$ levels were significantly different according to types/levels of physical activity (Kruskal-Wallis p-value < 0.001). The highest $PM_{2.5}$ median levels were measured during exercise (42.5μg/m^3) followed by standing (36.0μg/m^3) and other activities (35.0μg/m^3). The median levels of $PM_{2.5}$ during both sleeping and seating were significantly lower than during other activities (Mann-Whitney p-value < 0.001).

Table 5.10: Personal exposure to $PM_{2.5}$ and physical activity

Physical Activity	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Exercise	12.5	2.0%	56.3	77.2	42.5	24.3	55.3	31.0	10.0	539.0	<0.001
Standing	36.8	5.9%	37.1	19.4	36.0	22.0	47.0	25.0	4.0	96.0	
Other	46.3	7.4%	46.7	90.2	35.0	23.0	49.0	26.0	3.0	1181.0	
***Sitting**	310.8	50.0%	39.2	67.6	30.0	21.0	43.0	22.0	3.0	1808.0	
***Sleeping**	214.8	34.6%	26.5	15.1	26.0	16.0	33.0	17.0	2.0	105.0	
Total	621.0	100.0%									

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.6.7 Personal exposure and time slot

The previous results regarding personal exposure levels associated with physical activities (section 5.6.6) showed that the median levels of $PM_{2.5}$ during both sleeping and seating (which, primarily occur between midnight and noon (12am-12pm)), were significantly lower than during other activities as shown in Table 5.11. Therefore, two 'time-slots' were defined as after and before midnight based on the median levels of personal exposure to $PM_{2.5}$. The Table 5.11 compares personal $PM_{2.5}$ levels after and before midnight. The personal median $PM_{2.5}$ levels were significantly higher between noon and midnight than from midnight to noon (Mann-Whitney p-value < 0.001).

Table 5.11: Personal exposure to $PM_{2.5}$ measured after and before midnight

Time Slot	Time		$PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minute intervals								Mann-Whitney p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
00:00-11:59	315.3	51.0%	26.9	15.3	25.0	17.0	34.0	17.0	2.0	121.0	<0.001
12:00-23:59	305.8	49.0%	44.6	77.6	33.0	23.0	49.0	26.0	5.0	1808.0	
Total	621.0	100.0%									

*Mann-Whitney p-value is significant at the 0.001 level

5.7 Personal Home-Indoor Exposure to $PM_{2.5}$ and House Characteristics

House characteristics were identified from the questionnaire completed by all 27 participants. The total home-Indoor concentrations were significantly different across a range of variables (see Table 5.12). Apartments had significantly higher $PM_{2.5}$ levels (34.0μg/m^3) than other home types. Homes with open fires located outside the house had the highest $PM_{2.5}$ levels (42.0μg/m^3). $PM_{2.5}$ levels were higher for those who reported having both gas and electric cooking fuel than those who reported using one source of cooking fuel. $PM_{2.5}$ levels were significantly higher for those who reported having no ventilation and were lowest for those who reported using a fan as ventilation source.

Table 5.12: Descriptive statistics of personal home-indoor exposure to $PM_{2.5}$ levels and house characteristics

Variable	Time		Personal Home-Indoor $PM_{2.5}$ ($\mu g/m^3$) concentrations								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
1. House Type											
***Apartment**	13.8	3.8%	57.8	71.9	34.0	30.0	48.0	18.0	21.0	443.0	<0.001
Detached	247.0	67.7%	31.3	19.1	29.0	17.0	41.4	24.4	2.0	139.0	
Bungalow	62.8	17.2%	36.4	40.2	27.0	21.0	34.0	13.0	13.0	400.0	
Semi-detached	41.5	11.4%	58.9	187.5	23.0	15.0	34.0	19.0	3.0	1808.0	
Total	365.0	100%									
2. Open Fire Room											
***Outside House**	29.5	8.1%	46.9	20.8	42.0	30.0	59.3	29.3	18.0	139.0	<0.001
Inside House	39.0	10.7%	29.5	19.2	26.0	18.0	29.0	11.0	5.0	88.0	
No	296.5	81.2%	36.2	76.0	28.0	17.0	39.0	22.0	2.0	1808.0	
Total	365.0	100%									
3. Cooking Fuel											
Both Gas/Electric	67.0	18.4%	51.3	99.1	35.0	28.0	48.0	20.0	17.0	1181.0	<0.001
Bottled Gas	57.0	15.6%	32.2	16.1	28.0	21.0	36.0	15.0	14.0	110.0	
Electric	241.0	66.0%	33.1	66.2	25.0	15.0	39.0	24.0	2.0	1808.0	
Total	365.0	100%									
4. Ventilation											
***None**	30.0	8.2%	47.8	53.8	34.0	27.0	42.8	15.8	15.0	400.0	<0.001
Open windows	54.0	14.8%	35.0	43.0	29.0	9.0	39.0	30.0	5.0	443.0	
Air-conditioning	265.8	72.8%	33.8	52.8	28.0	19.0	40.0	21.0	2.0	1181.0	
***Fan**	15.3	4.2%	62.5	231.4	14.0	4.0	51.0	47.0	3.0	1808.0	
Total	365.0	100%									
5. Household Members											
N ≥7	225.5	61.8%	39.5	84.2	29.5	20.0	41.0	21.0	3.0	1808.0	<0.001
N <7	139.5	38.2%	31.0	31.6	26.0	15.0	39.0	24.0	2.0	400.0	
Total	365.0	100%									

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.8 Potential Exposure Sources and Locations

Table 5.13 shows the potential exposure sources and locations exposure in relation to personal exposure questionnaire filled by the participants of the current study. The total personal concentrations were significantly different across the potential exposure sources and locations categories (Kruskal-Wallis p-value < 0.001). $PM_{2.5}$ levels were higher for those who reported being regularly exposed to cigarettes (30.0μg/m^3), than those who reported regular exposure to incense and other sources. The lowest median $PM_{2.5}$ levels were measured when the participant noted exposures occurring in the home (27.5μg/m^3), where exposures occurring at other locations were associated with higher median $PM_{2.5}$ levels were measured at other locations (31.0μg/m^3).

Table 5.13: Descriptive statistics of potential exposure sources and locations to $PM_{2.5}$ levels

Variable	Time		Personal $PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minutes interval								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Regularly Exposed											
Cigarette	153.3	24.7%	39.7	76.8	30.0	19.0	39.0	20.0	5.0	1181.0	<0.001
***Other sources**	208.5	33.6%	36.8	27.6	29.0	23.0	44.0	21.0	6.0	400.0	
Incense	259.3	41.7%	32.2	58.8	27.0	17.0	40.0	23.0	2.0	1808.0	
Total	621.0	100.0%									
Exposed Place											
Home	420.0	67.6%	33.9	50.9	27.5	19.0	40.0	21.0	2.0	1808.0	<0.001
Other locations	201.0	32.4%	39.2	65.8	31.0	20.0	43.0	23.0	5.0	1181.0	
Total	621.0	100.0%									

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.9 Personal exposure to $PM_{2.5}$ and Age

Table 5.14 shows personal exposure to $PM_{2.5}$ in relation to age. The total personal concentrations were significantly different across the three age groups (Kruskal-Wallis p-value < 0.001). The highest median $PM_{2.5}$ levels were measured for those aged eighteen years (34.0μg/m^3), and the lowest median $PM_{2.5}$ levels were measured in those aged sixteen years (23.0μg/m^3).

Table 5.14: Descriptive statistics of personal exposure to $PM_{2.5}$ levels

Variable	Time		Personal $PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minutes interval								Kruskal-Wallis p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
16 Years old	129.3	20.8%	30.0	80.6	23.0	16.0	32.0	16.0	3.0	1808.0	<0.001
17 Years old	276.8	44.6%	35.0	56.3	28.0	19.0	39.0	20.0	2.0	1181.0	
18 Years old	215.0	34.6%	39.8	33.3	34.0	24.0	47.0	23.0	5.0	539.0	
Total	621.0	100.0%									

5.10 Personal Exposure in Different Microenvironments

The microenvironments of interest in this study were collapsed into seven main categories (home-indoor, home-outdoor, school-indoor, school-outdoor, indoor away from home or school, outdoor away from home or school, and transport). As stated previously, the personal $PM_{2.5}$ levels were not normally distributed. Therefore, the median $PM_{2.5}$ levels of the 15-minute intervals spent in each microenvironment were calculated.

Table 5.15 shows the descriptive analyses for personal exposure to $PM_{2.5}$ in these main microenvironment categories, including median, 25th and 75th percentiles, and Shapiro Wilk's test p-value). The total number of hours measured across all microenvironments was 621 hours. The majority of those hours were spent at home-indoors (365 hours, 59%) and at school-indoors (165 hours, 26.6%). The least time was spent at school-outdoors (7.3 hours, 1.2%) and at home-outdoors (8.0 hours, 1.3%). The $PM_{2.5}$ levels varied greatly between these microenvironments, with an overall median of 29.0, inter-quartile range (IQR) of 22.0 and range of 2.0-1808μg/m^3. The total median personal concentrations were significantly different across these microenvironments (Kruskal-Wallis p-value < 0.001). Outdoors away from home or school was found to have the highest median level of 44.0μg/m^3, significantly higher than the other microenvironments (Mann-Whitney p-value < 0.001). The lowest median level of $PM_{2.5}$ was found at school-indoors (25.0μg/m^3) and at home-indoors (28.0μg/m^3). Both these microenvironments were significantly different from the other microenvironments (Mann-Whitney p-value < 0.001).

Table 5.15: Descriptive statistics of $PM_{2.5}$ personal exposure levels in different microenvironments

Category		Time		Personal $PM_{2.5}$ ($\mu g/m^3$) concentrations per 15-minutes interval								Kruskal-Wallis p-value
		Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
Outdoors*	Home	8.0	1.3%	45.3	22.5	40.0	31.3	56.3	25.0	14.0	93.0	
	School	7.3	1.2%	39.8	17.4	36.0	30.0	50.5	20.5	14.0	80.0	
	Others	16.3	2.6%	44.4	21.2	44.0	29.0	54.0	25.0	4.0	90.0	
	Total	31.6	5.1%	46.8	21.6	44.4	31.1	59.5	28.4	6.4	531.9	
Indoors	*Home	365.0	58.8%	36.3	69.1	28.0	18.0	40.0	22.0	2.0	1808.0	<0.001
	*School	165.0	26.6%	28.1	14.0	25.0	19.0	34.0	15.0	5.0	121.0	
	Others	23.0	3.7%	51.2	59.7	39.0	31.5	50.0	18.5	7.0	539.0	
	Total	553.0	89.0%	35.5	56.2	28.3	19.2	40.2	21.0	2.3	101.7	
Transport		36.5	5.9%	46.3	43.9	36.5	27.0	51.3	24.3	6.0	371.0	
Total personal exposure		621.1	100.0%	35.6	56.2	29.0	19.0	41.0	22.0	2.0	1808.0	

*Mann-Whitney for all pairwise comparisons, p-value is significant at the 0.001 level

5.11 Comparison between Hourly Personal and Fixed-Site Monitoring $PM_{2.5}$ Exposure Levels

Figure 5.4 shows the distribution of hourly $PM_{2.5}$ levels based on personal and fixed-site monitoring data collected between February and May 2012. As the air pollution levels had a skewed distribution (Shapiro-Wilk $p < 0.001$), non-parametric tests were applied in the descriptive analyses. Wilcoxon-paired-sign-rank tests were performed to test whether personal $PM_{2.5}$ levels and outdoor fixed-site station levels differed significantly.

The summary statistics of hourly $PM_{2.5}$ levels from both personal and fixed-site monitoring are shown in Table 5.16. The fixed-site monitoring median $PM_{2.5}$ level was 51.0μg/m^3, which is significantly higher than the personal monitoring median of 30.0μg/m^3 (Wilcoxon p-value < 0.001). The distributions significantly overlap, but the fixed-site monitoring data is shifted slightly to the right (i.e. tends to record higher $PM_{2.5}$ levels) than the personal monitoring data as shown in Figure 5.4.

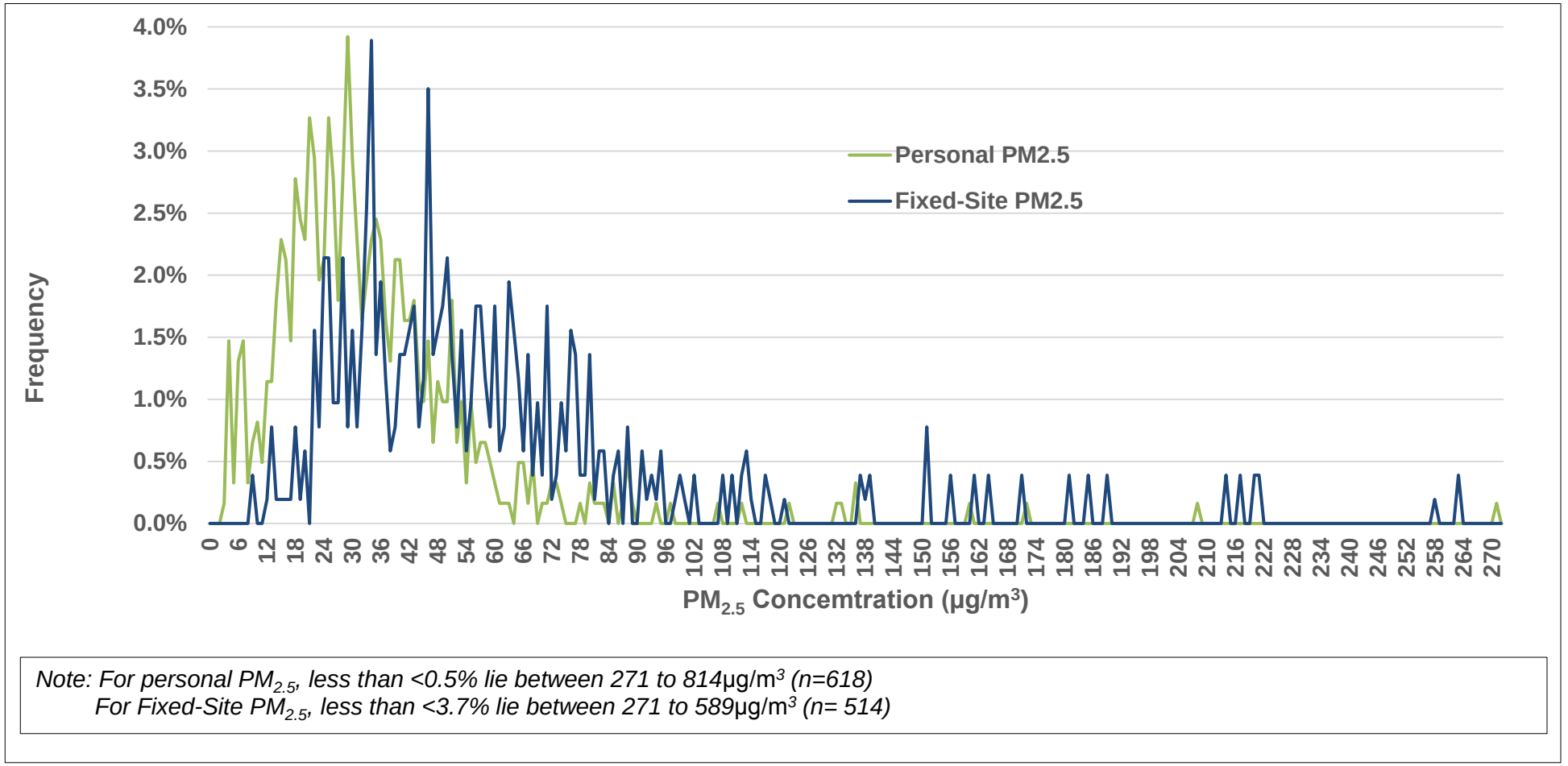


Figure 5.4: Distribution of hourly $PM_{2.5}$ levels for Personal and fixed-site monitoring from February to May 2012

Table 5.16: Summary statistics for comparison between $PM_{2.5}$ measured via personal and fixed-site monitoring from February to May 2012

Category	Time		Hourly $PM_{2.5}$ ($\mu g/m^3$) concentrations								Wilcoxon p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max	
$PM_{2.5}$ Personal Exposure Levels	618.0	95.0%	37.3	44.5	30.0	20.9	42.4	21.4	3.0	814.0	<0.001
Fixed-Site Levels	514.0	79.0%	74.1	83.2	51.0	34.0	74.2	40.2	9.0	589.0	

5.12 Correlation between Hourly $PM_{2.5}$ Levels and Weather Variables

The summary statistics for weather variables are shown in Table 5.17. For the correlation between personal $PM_{2.5}$ and fixed-site, relative humidity (RH) and temperature (Temp), Pearson's correlation was used because it is normally distributed with hourly-log-transformed data for personal and log fixed-site $PM_{2.5}$ monitoring data. The log-transformed hourly personal $PM_{2.5}$ concentration data showed a very weak but significant correlation with temperature (Pearson's correlation coefficient =0.193, p-value <0.001), but not relative humidity (See Table 5.18).

There was a very weak but significant correlation between the log-transformed personal $PM_{2.5}$ and fixed-site $PM_{2.5}$ monitoring data (Pearson's correlation coefficient =0.164, p-value <0.001) (See Table 5.18).

Figure 5.5 shows the association between log-transformed personal and fixed-site monitoring $PM_{2.5}$ data (R^2 = 0.0268).

Table 5.17: Summary statistics for weather variables

Weather Variable	Time		Mean	SD	Median	Q1	Q3	IQR	Min	Max
	Hours	%								
Relative Humidity (%)	648.0	100.0%	64.8	19.2	65.5	53.8	75.5	21.7	11.7	99.6
Temperature (C^{o})	648.0	100.0%	15.0	3.7	15.2	12.2	17.6	5.4	7.1	25.6

Table 5.18: Correlation between $PM_{2.5}$ personal levels, $PM_{2.5}$ Fixed-Site and weather variables

Pearson's Correlation		Log-Fixed-Site $PM_{2.5}$	RH	Temp
Personal $PM_{2.5}$	Pearson's Correlation	0.164**	-0.061	0.193**
	Sig. (2-tailed)	0.001	0.127	0.001
	Total Number	488.0	618.0	618.0
Fixed-Site $PM_{2.5}$	Pearson's Correlation		0.100*	0.052
	Sig. (2-tailed)		0.024	0.241
	Total Number		514.0	514.0
RH	Pearson's Correlation			-0.208**
	Sig. (2-tailed)			<0.001
	Total Number			648.0

**Pearson's Correlation is significant at the 0.01 level
*Pearson's Correlation is significant at the 0.05 level

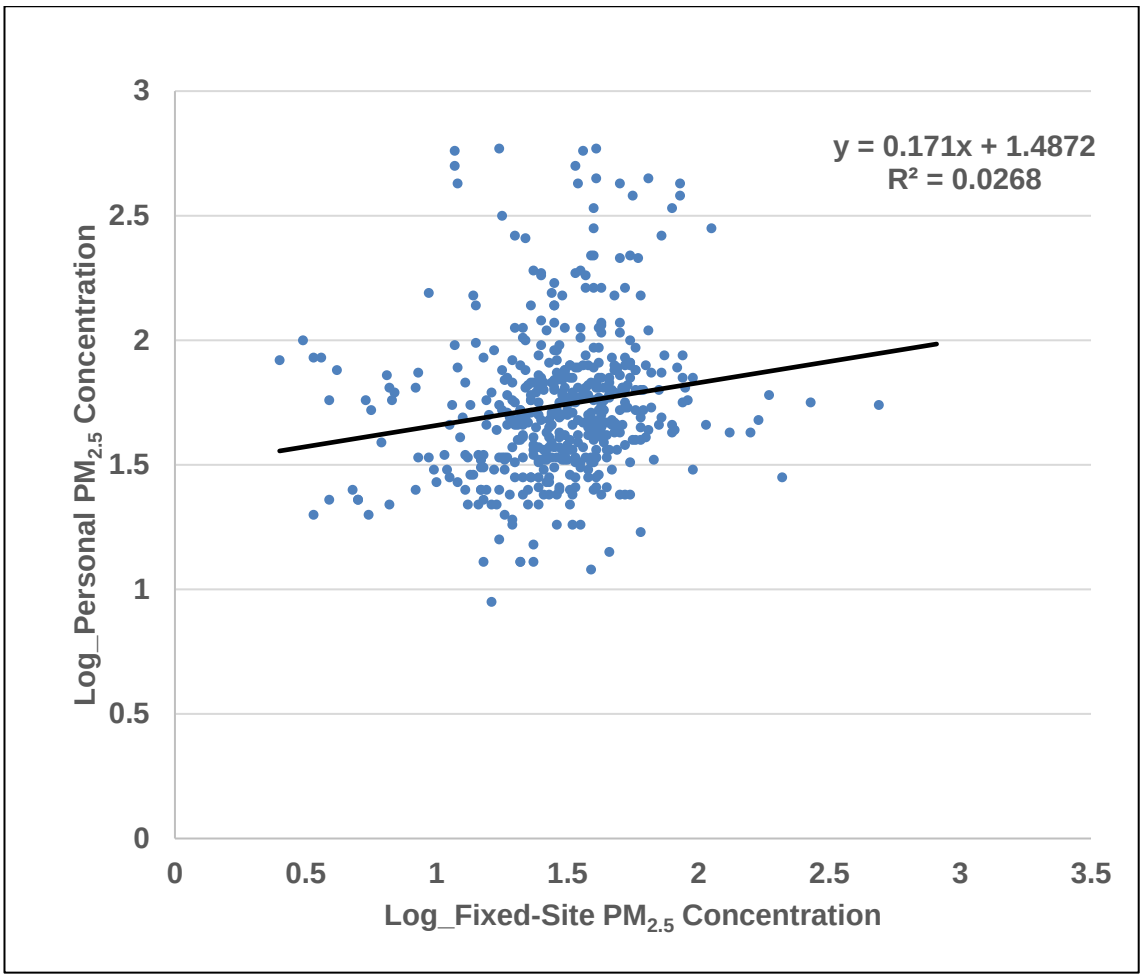


Figure 5.5: Scatter-Plot for Log-transformed Hourly $PM_{2.5}$ Concentration from Personal and Fixed-Site monitoring

5.13 Comparison of Personal and Fixed-Site $PM_{2.5}$ Levels by Major Microenvironments

Table 5.19 shows $PM_{2.5}$ levels measured using fixed-site monitors compared to personal monitoring for the same hours while indoors (home-indoor, school-indoor, indoor away from home/school) and other locations (home-outdoor, school-outdoor, outdoor away from home/school, and transport). The summary statistics are shown as medians and interquartile ranges) because the data were not normally distributed (Shapiro Wilk's test p-value < 0.001). The fixed-site median $PM_{2.5}$ levels were significantly higher than personal level while indoors and at other microenvironments. In addition, there is a significant but weak correlation between fixed-site and personal monitor $PM_{2.5}$ levels indoors (Spearman's rank correlation=0.228, p-value < 0.001 n=544), but not at 'other' microenvironment.

Figure 5.6 is a scatter plot showing the correlation between the log-transformed hourly fixed-site compared to personal monitoring $PM_{2.5}$ levels at indoor and other microenvironments.

Table 5.19: Descriptive statistics for hourly $PM_{2.5}$ levels from fixed-site station and personal exposure levels measured at indoors and other microenvironments

Category	Time		Hourly $PM_{2.5}$ ($\mu g/m^3$) levels								Spearman's Correlation	Wilcoxon p-value
	Hours	%	Mean	SD	Median	Q1	Q3	IQR	Min	Max		
Fixed-Site	427.0	66.0%	76.1	89.0	51.0	34.0	75.0	41.0	9.0	589.0	0.288*	0.001
Personal Indoors	427.0	66.0%	34.8	32.0	29.8	20.6	42.0	21.4	2.0	487.0		
Fixed-Site	61.0	9.0%	59.1	32.9	56.0	39.5	68.5	29	13.0	189.0	0.171	0.005
Personal Others	61.0	9.0%	47.7	33.4	41.0	28.1	52.9	24.8	7.0	208.0		

*Spearman's correlation is significant at the 0.001 level.

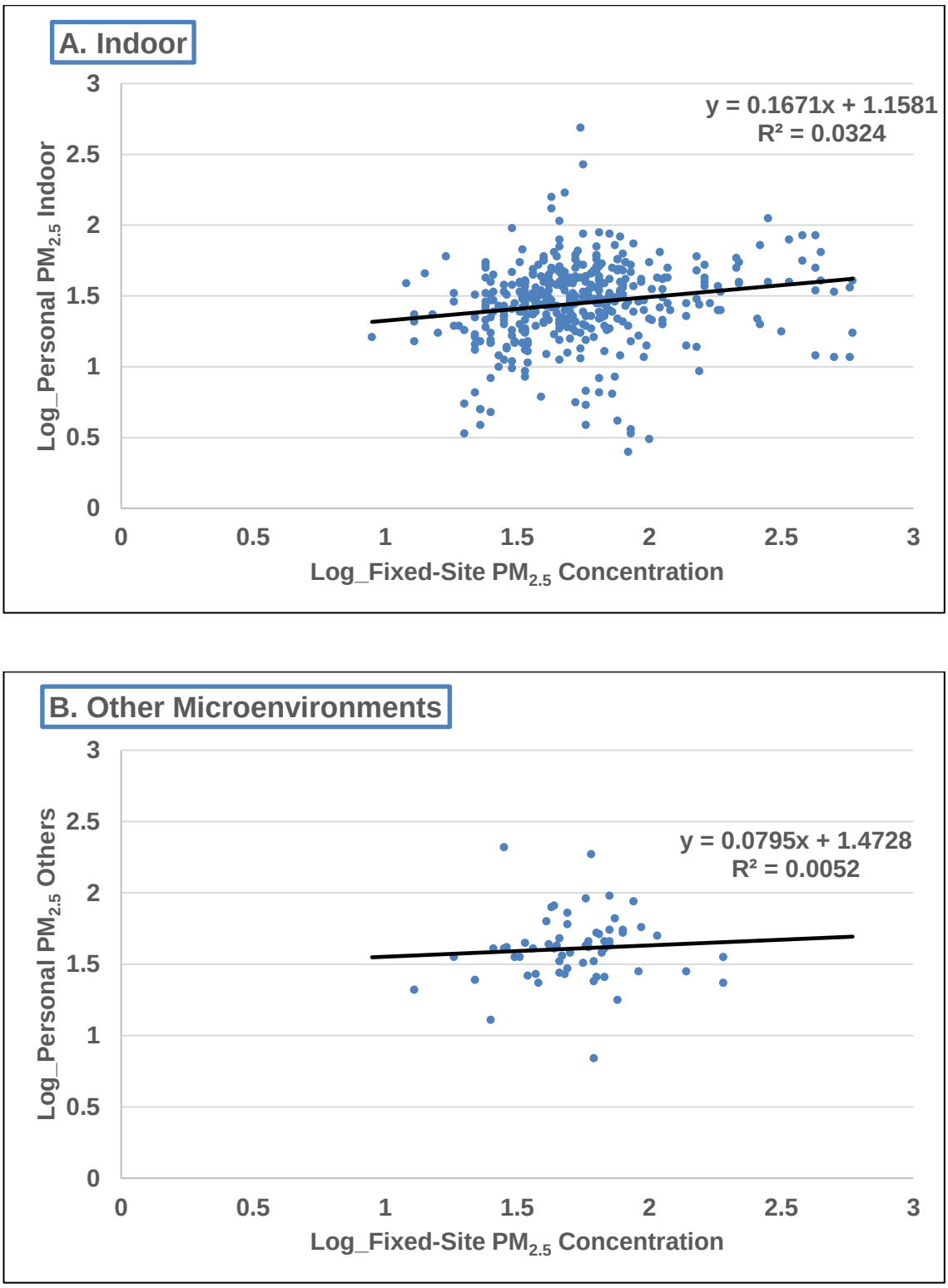


Figure 5.6: Scatter-Plot for Hourly $PM_{2.5}$ Concentration from Fixed-Site Station and Personal concentration measured at A. Indoor and B. Other Microenvironment

5.14 Variation in Personal Exposure to $PM_{2.5}$

A multiple regression model was run to try to predict personal $PM_{2.5}$ exposure levels from the residential fixed-site monitor $PM_{2.5}$ levels. Variables included in the model were those that showed a significant difference in personal exposure to $PM_{2.5}$ by using non-parametric tests (Kruskal-Wallis test, to compare more than two groups and the Mann Whitney U test, to compare two groups). A stepwise selection method was used to exclude or include variables in a sequential process, with a statistical significance level of 95% ($p<0.05$) required for inclusion. The variables included in the final model were residential fixed-site monitor, time slot, participant age, home characteristics (cooking fuel, ventilation), and microenvironment (indoor versus 'other' locations) (see Table 5.20). This model explained only 28% of the variability in personal $PM_{2.5}$ levels. The results indicated that individual variable making the largest contribution to the total R^2 was the time-slot (R^2 change=0.070) followed by residential fixed-site (R^2 change=0.060), age of the participant (R^2 change=0.057), cooking fuel (R^2 change=0.056), ventilation (R^2 change=0.033) and indoor/other location microenvironments (R^2 change=0.017). All these variables statistically significantly contributed to the model, $F = 30.5$, degree of freedom = 487, p-value < 0.001, $R^2 = 0.275$.

Other variables were eligible for inclusion in the model, including home ownership, house type, open fireplace, exposure place, household members, and meteorological variables (temperature and relative humidity) but did not contribute to the model. The variable 'day of the week' could not be included, as the personal $PM_{2.5}$ exposure data was collected only during the school days and not during the weekend. Like other studies, wind speed or wind direction were not considered as potential explanatory variables; the students spent 85% of their time indoors where wind speed and direction would have no likely impact.

Figure 5.7 shows the association between predicted and personal hourly $PM_{2.5}$ level.

Table 5.20: Multiple linear regression analysis of predicted personal $PM_{2.5}$

Variable	Coefficients[1] *B*	95% CI for *B*		p-value	R^2 Changed
		Lower Bound	Upper Bound		
Residential $PM_{2.5}$ Fixed-Site	0.189	0.114	0.264	<0.001	0.060
Time Slot	0.156	0.111	0.201	<0.001	0.070
Age	0.097	0.066	0.127	<0.001	0.057
Cooking Fuel	0.088	0.060	0.116	<0.001	0.056
Ventilation	0.080	0.046	0.113	<0.001	0.033
Indoor and other locations	-0.118	-0.186	-0.050	<0.001	0.017

[1]Dependent Variable: Personal $PM_{2.5}$ exposure levels

Note: Other variables (homeowner, house type, open fireplace, exposure place, household members and temperature) were eligible for inclusion in the model, but were found not to contribute to the model

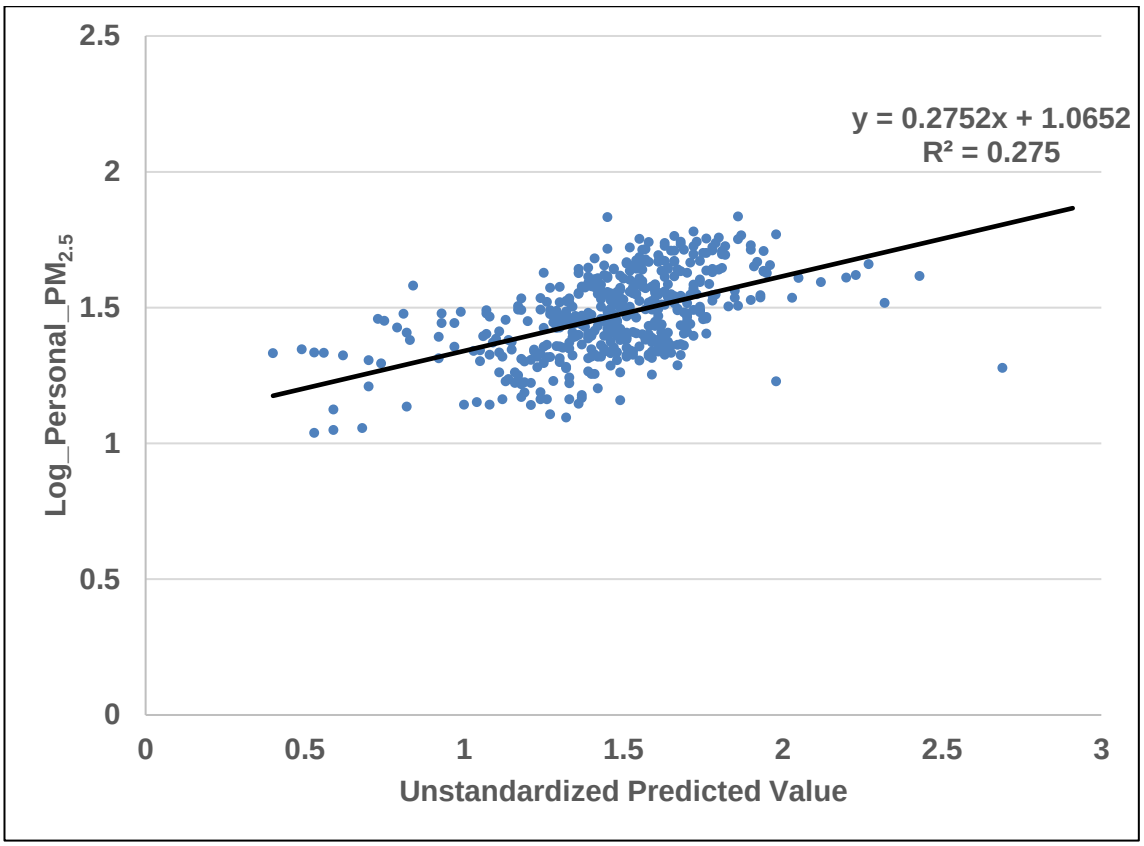


Figure 5.7: Scatter plot for measured log-personal $PM_{2.5}$ versus predicted $PM_{2.5}$

5.15 Spatial Distribution of Personal $PM_{2.5}$ Levels

Figure **5.8** shows hourly mean personal $PM_{2.5}$ levels plotted in GIS according to the mean GPS location across the hour on an Al Jubail base map. $PM_{2.5}$ exposures were distributed across the locations visited by the students, with no discernible pattern to indicate exposure hot or cold spots.

Figure 5.9 shows the residuals from the model described in section 5.14 (see Table 5.20 above) again plotted on the Al Jubail base map. The residuals are classified into three categories based on the standard deviation. If the fixed-site monitoring data was a good proxy for local personal exposure, I might expect to see larger residuals further from the monitor location. While residuals are used to give some indication of the “exposure error” from using the proxy measure, this measure is not based on just fixed-site data but also other covariates so is not error due to using fixed-site data. There is no clear spatial pattern in residuals shown in the Figure 5.9.

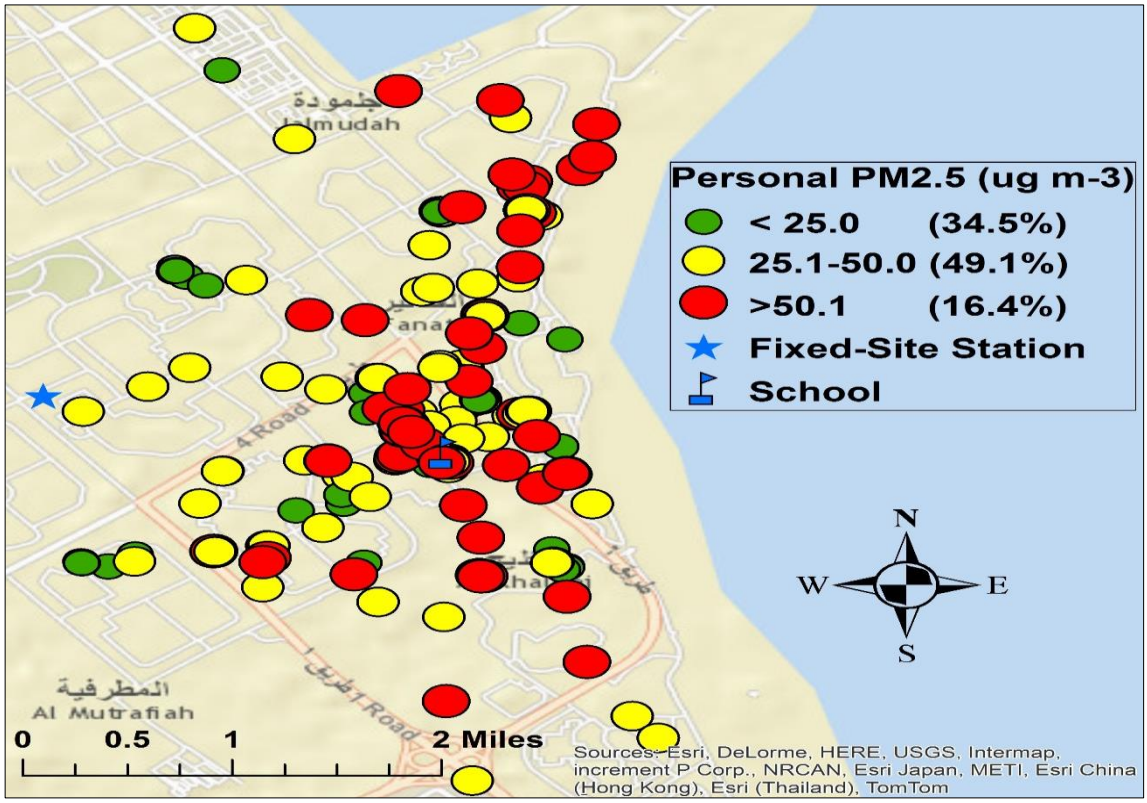


Figure 5.8: Location-based personal exposure showing hourly personal mean $PM_{2.5}$

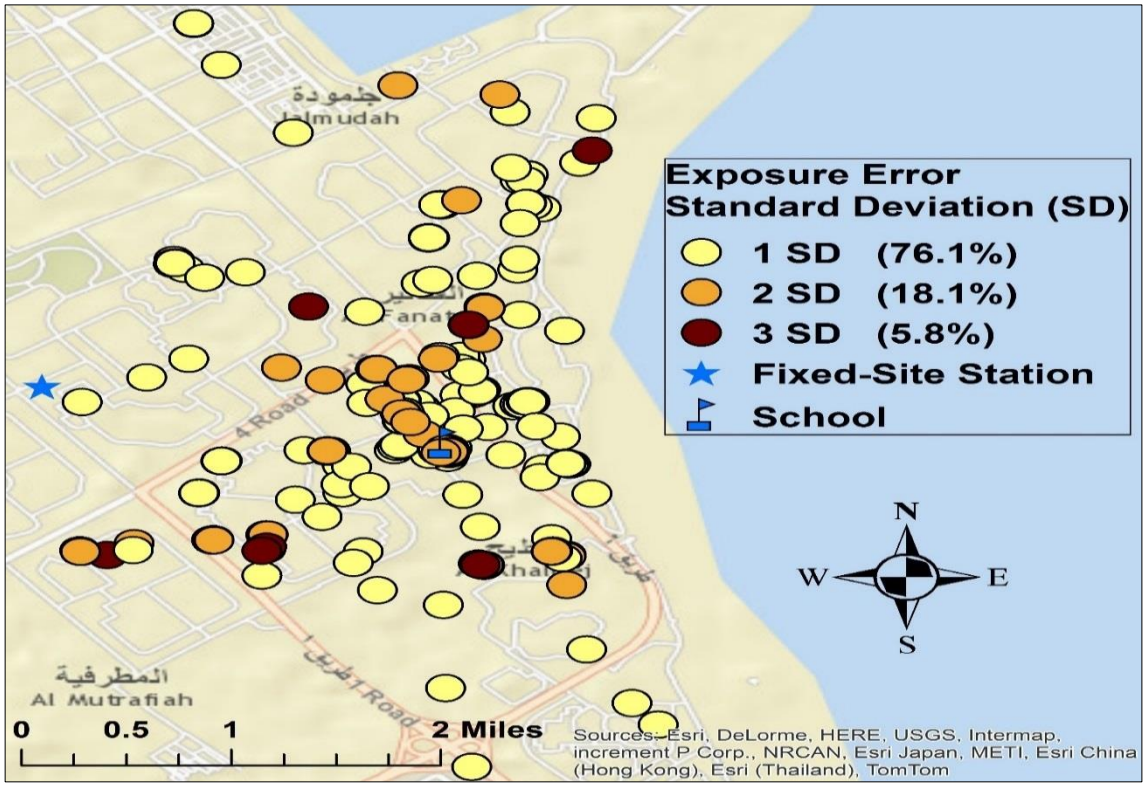


Figure 5.9: Location-based exposure error in predicted personal exposure (based on standard deviation or residuals)

5.16 The relationship between AEDv and $PM_{2.5}$ before and after conversion ambient levels

The relationship of AEDv for school age group (6-18 years old) and air pollution was re-examined to investigate how it would change if personal air pollution exposures (from objective two) were incorporated into the exposure variable, instead of data from fixed-site monitor (from objective one). In order to investigate this relationship, a valid strategy was developed by using the insights gained of $PM_{2.5}$ levels from the personal monitoring campaign to refine the ambient concentrations derived from the fixed-site monitor for the period 2007-2012, which can then be used to re-run the main model of time-series analysis.

5.16.1 Conversion ambient $PM_{2.5}$ levels to the new scale ($PM_{2.5}C$)

The converted ambient $PM_{2.5}$ levels ($PM_{2.5}C$) were calculated using a correction factor based on the median ratio of the fixed site to personal monitoring $PM_{2.5}$ values. (total personal exposure monitoring median $PM_{2.5}$ / total fixed site monitoring median $PM_{2.5}$). As described in section3.4.3 (Phase three: TSA of AEDv and ambient PM2.5 levels corrected from personal monitoring campaign), the $PM_{2.5}C$ levels were calculated by applying a correction factor of 1.70 (total personal exposure monitoring median $PM_{2.5}$ / total fixed site monitoring median $PM_{2.5}$). A lower correction factor based on the ratio of the 25^{th} centile of total personal exposure monitoring $PM_{2.5}$ / 25^{th} centile of total fixed site monitoring $PM_{2.5}$ ($PM_{2.5}C$-L) and upper correction factor based on the ratio of the 75^{th} centile of total personal exposure monitoring $PM_{2.5}$ / 75^{th} centile of total fixed site monitoring $PM_{2.5}$ ($PM_{2.5}C$-H) was also applied to reflect the whole distribution of exposures, and assess the sensitivity of the risk estimates to these correction values. The correction factors for $PM_{2.5}C$-L and $PM_{2.5}C$-H were 1.63 and 1.75 respectively.

Table 5.21 shows the descriptive statistics of daily ambient $PM_{2.5}$ levels before and after conversion for the period 2007-2012. The summary shows medians, 25 and 75 percentiles, because the distribution of data did not fit a normal pattern (Shapiro Wilk's test $P<0.05$).

Table 5.21: Descriptive statistics of daily ambient $PM_{2.5}$ levels before and after conversion to the new scale

	Mean	SD	Median	Min	Max	Q1	Q3	IQR
$PM_{2.5}$ unconverted	64.61	64.52	45.63	9.96	643.70	32.13	68.41	36.28
$PM_{2.5}$C *CF=1.70	37.72	37.72	26.68	5.82	376.39	18.79	39.73	20.94
$PM_{2.5}$C-H *CF=1.63	39.82	39.82	28.17	6.15	397.35	19.83	41.94	22.11
$PM_{2.5}$C-L *CF=1.75	36.87	36.86	26.08	5.69	367.83	18.36	38.82	20.46

*CF= Correction Factor

5.16.2 Relative risk of AEDv for school age group (6-18 years old) using ambient $PM_{2.5}$ levels before and after conversion to the new scales

Table 5.22 summarises the output from the sensitivity analysis, re-evaluating the of risk of AEDv for school age admissions (aged 6-18 years old) per 10μg/m^3 increase in $PM_{2.5}$ levels before and after re-scaling. A lower ($PM_{2.5}$C-L) and upper ($PM_{2.5}$C-H) quartiles correction factor was also applied for 0-7 lag days. The RRs of AEDv for this school age group showed a very similar effect when using $PM_{2.5}$C, $PM_{2.5}$C-H or $PM_{2.5}$C-L. An increase in daily AEDv on the same day (lag 0) and the following day (lag 1) positively and significantly associated with an increase in 10μg/m^3 of $PM_{2.5}$C, $PM_{2.5}$C-H and $PM_{2.5}$C-L. The most statistically significant increase in AEDv was found on the same day (lag 0); RRs = 1.017 for $PM_{2.5}$C and $PM_{2.5}$C-L, RR = 1.016 for $PM_{2.5}$C-H. The results of this sensitivity analysis show that using the corrected ambient $PM_{2.5}$ levels in the time-series analysis for AEDv in children aged 6-18 years did not dramatically affect the interpretation of this analysis. The relative risk of AEDv per 10μg/m^3 increase in $PM_{2.5}$ concentration increased by 1.0% (95% CI: 0.7-1.3) when using the unconverted ambient $PM_{2.5}$ levels and 1.7% (95% CI: 0.7-2.7) when using the converted ambient $PM_{2.5}$ ($PM_{2.5}$C).

The relative risks of AEDv for school age group (6-18 years old) are graphically represented for a range of $PM_{2.5}$C concentrations in the time-series model as shown in Figure 5.10.

Table 5.22: Sensitivity analysis of relative risks (95% CI) for AEDv from 6 to 18 years old per 10µg/m^3 increase in $PM_{2.5}$ levels before and after conversion to the new scales ($PM_{2.5}$C, $PM_{2.5}$C-H and $PM_{2.5}$C-L) for 0-7 lag days

Pollutant	Lag days	RR per 10µg/m^3 increase			t-value	
		RR	95% Confidence Interval			
AEDv 6-18 years $PM_{2.5}$ unconverted	**0#**	**1.010**	**1.004**	**1.016**	**3.3**	*
	1	1.007	1.002	1.013	2.5	*
	2	1.000	0.994	1.007	0.1	
	3	1.001	0.994	1.008	0.3	
	4	1.001	0.994	1.008	0.3	
	5	1.005	0.998	1.011	1.4	
	6	0.999	0.992	1.006	-0.4	
	7	0.995	0.988	1.002	-1.4	
AEDv 6-18 years $PM_{2.5}$C	**0#**	**1.017**	**1.007**	**1.027**	**3.4**	*
	1	1.013	1.007	1.027	2.6	*
	2	1.001	0.990	1.012	0.1	
	3	1.002	0.990	1.013	0.3	
	4	1.002	0.991	1.014	0.4	
	5	1.009	0.997	1.020	1.5	
	6	0.998	0.986	1.010	-0.3	
	7	0.992	0.980	1.004	-1.4	
AEDv 6-18 years $PM_{2.5}$C-H	**0#**	**1.016**	**1.007**	**1.025**	**3.3**	*
	1	1.012	1.003	1.022	2.5	*
	2	1.000	0.990	1.011	0.1	
	3	1.001	0.991	1.012	0.3	
	4	1.002	0.991	1.013	0.3	
	5	1.008	0.997	1.019	1.4	
	6	0.998	0.987	1.009	-0.4	
	7	0.992	0.981	1.003	-1.4	
AEDv 6-18 years $PM_{2.5}$C-L	**0#**	**1.017**	**1.007**	**1.028**	**3.3**	*
	1	1.013	1.003	1.024	2.5	*
	2	1.000	0.989	1.012	0.1	
	3	1.002	0.990	1.013	0.3	
	4	1.002	0.990	1.014	0.3	
	5	1.008	0.997	1.020	1.4	
	6	0.998	0.986	1.010	-0.4	
	7	0.991	0.980	1.003	-1.4	

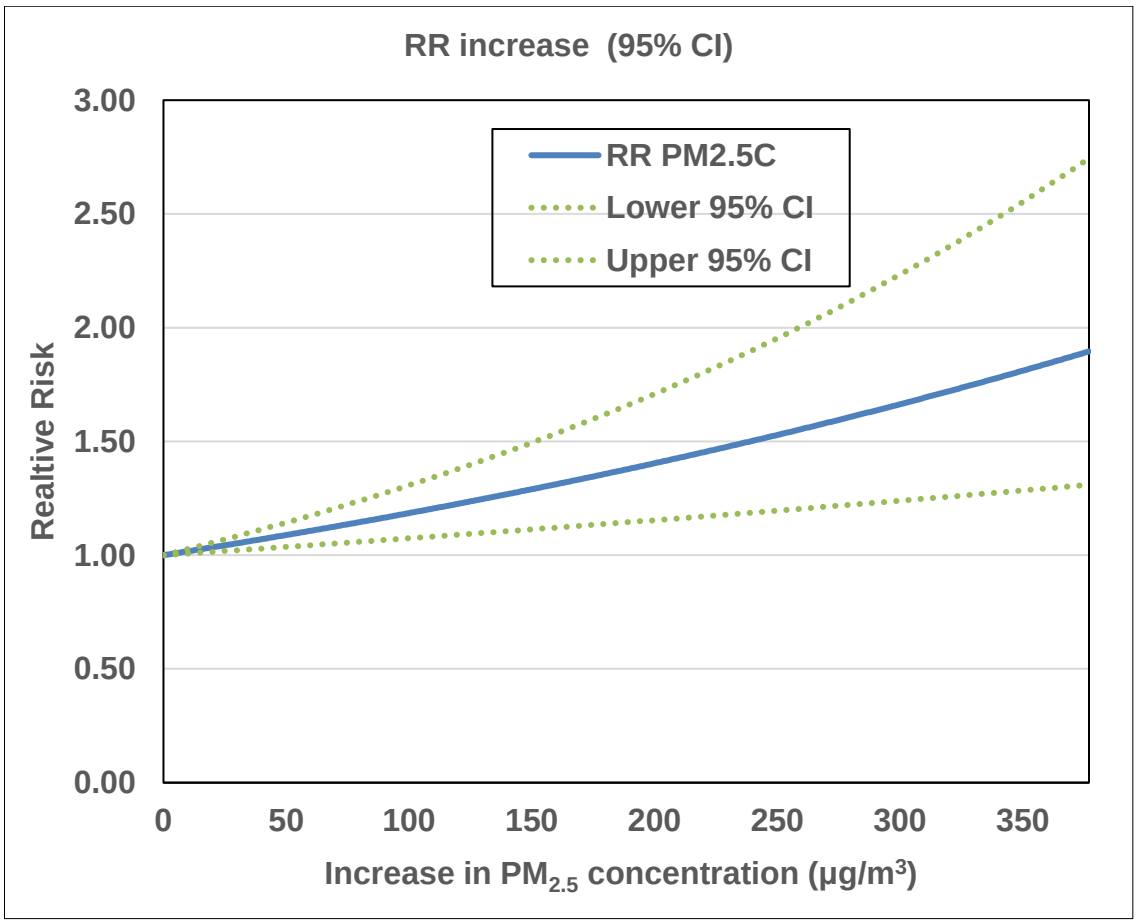


Figure 5.10: Estimates of relative risk of AEDv by $PM_{2.5}$C concentration (the dashed lines are the 95% confidence interval)

CHAPTER SIX

Discussion of Time-Series Analysis

Chapter:6 Discussion of Time-Series Analysis

6.1 Introduction

A large number of epidemiological studies have found an association between air pollution and asthma-related hospital visits. However, most of these studies were conducted in Europe and North America where there is a temperate climate with distinct seasons. To the best of my knowledge, no study exploring this issue has been conducted in a hot and dry industrial city in the Middle East. This study used time-series analysis to investigate the statistical association between exposure to air pollution and asthma-related emergency department visits in Al Jubail Industrial City in Saudi Arabia.

In this chapter, the findings are discussed under three main headings: First, the main key findings of this study. Second, air quality exceedance in Al Jubail industrial City. In addition, the relative risk of asthma-related emergency department visits based on the time-series analysis have been discussed, followed by a discussion of the limitations and strengths of this aspect of the study.

6.2 Key findings

Table 6.1: Key findings for the Relative Risk in Multi-Pollutant Model

Variables	Relative Risk (risk ratio) in the Multi-Pollutant Model
PM_{10} µg/m^3	Positive significant association At lag day0 RR increased 2.2% (95% CI: 1.3 – 3.2) per IQR increase (140µg/m^3)
$PM_{2.5}$ µg/m^3	Positive significant association At lag day0 RR increased 4.4% (95% CI: 2.4 – 6.6) per IQR increase (36µg/m^3)
SO_2 ppb	Positive significant association At lag day2 RR% increased 5.4% (95% CI: 2.4, 8.5) per IQR increase (2.0ppb).
NO_2 ppb	Positive significant association At lag day3 RR% increased 3.4% (95% CI: 0.8, 6.1) per IQR increase (7.6ppb).

6.3 Relative Risk of Time-Series Analysis

The main results yielded by this study suggest that relative risks (RRs) of asthma-related emergency department visits increased positively and with statistical significance with increasing ambient levels of $PM_{2.5}$, PM_{10}, SO_2 and NO_2. The effects of these four pollutants were independent, as the associations remained significant in the multi-pollutant model, where the remaining pollutants are simultaneously introduced.

6.3.1 Particulate matter of 10 microns in diameter (PM_{10})

In this study, I have found a small positive association between increase in PM_{10} levels and increase in daily asthma-related emergency department visits. This positive association corroborates the findings of many of the previous studies of PM_{10} and asthma-related emergency department visits or hospital admissions for all ages (Schwartz *et al.*, 1993; Wong *et al.*, 1999; Galan *et al.*, 2003; Ko *et al.*, 2007; Bell *et al.*, 2008; Tadano *et al.*, 2012) and for the younger age groups (Norris *et al.*, 1999; Lee *et al.*, 2006b; Chimonas and Gessner, 2007; Nastos *et al.*, 2010; Samoli *et al.*, 2011). This study shows a significantly positive association between PM_{10}, on the same day of measurement and after 24 hours, and asthma-related to emergency department visits. The most significant increase in daily asthma visits was a 2.2% increase (95% CI: 1.3, 3.2) associated with an inter-quartile range (IQR) increase in PM_{10} levels (140μg/m^3) on the same day. The estimated effect in the current study is lower than those reported in Sao Paulo, Brazil, at 5.0% (Tadano et al., 2012), in Taipei, Taiwan, at 4.5% (Bell et al., 2008), in Madrid, Spain, at 3.9% (Galan et al., 2003) and in Seattle, USA, at 3.7% (Schwartz et al., 1993). Two previous studies conducted in Hong Kong in 1999 (Wong et al., 1999) and in 2007 (Ko *et al.*, 2007) have a similar estimated risk to the current study, which ranged between 1.5% and 1.9% respectively. In the Air Pollution and Health: A European Approach project 2 (APHEA-2), asthma admissions for younger (0-14) and older (15-64) age groups in eight European cities were examined (Atkinson, 2004). The study showed that asthma admissions increased by 1.5% for the younger age group and 1.0% for the older age group for each 10μg/m^3 increase in PM_{10} level. Furthermore, most of the studies on asthma visits in the younger age group showed a higher estimated risk

(2.5% to 14.0%) (Norris *et al.*, 1999; Lee *et al.*, 2006b; Chimonas and Gessner, 2007; Nastos *et al.*, 2010; Samoli *et al.*, 2011).

Another study conducted in Milan, Italy, failed to observe any association between PM_{10} and asthma-related emergency department visits in a multi-pollutant model (Giovannini *et al.*, 2010). A possible explanation may be the higher correlation between PM_{10} and other pollutants such as CO, SO_2 and NO_2 reported in some of the previous studies (Wong *et al.*, 1999; Galan *et al.*, 2003; Ko *et al.*, 2007; Bell *et al.*, 2008; Mar *et al.*, 2010), which may reduce the influence of PM_{10}. However, the results presented in this study did not show a correlation between PM_{10} and CO, SO_2 and NO_2, and the estimated effect remained significant after further adjustment for $PM_{2.5}$, SO_2 and NO_2 in the multi-pollutant model. This indicates that PM_{10} is not acting as a proxy for other pollutants, but rather points to an independent association.

6.3.2 Particulate matter of 2.5 microns in diameter ($PM_{2.5}$)

The current study found positive associations between increase in daily $PM_{2.5}$ levels and increase in daily asthma-related emergency department visits. This finding is consistent with those of other studies, which reported a positive association between $PM_{2.5}$ levels and asthma visits for all ages (Ko *et al.*, 2007; Bell *et al.*, 2008; Mar *et al.*, 2010; Silverman and Ito, 2010) and for a younger age group (Lee *et al.*, 2006b; Li *et al.*, 2011). The single-pollutant model had a significantly positive association for the current day (lag 0) and lag day 1 of $PM_{2.5}$ with asthma-related emergency department visits. The most significant increase in asthma-related emergency department visits was 4.4% (95% CI: 2.4, 6.6) per inter-quartile range (IQR) change of $PM_{2.5}$ level (36µg/m^3) on the current day. This effect remained significant after further adjustment for PM_{10}, SO_2, NO_2 and CO in the multi-pollutant model. The size effect in the current study is similar to that reported in Tacoma, USA, which was 4.0% (Mar et al., 2010). Lower estimated risks have been reported in Hong Kong 2.1% (Ko *et al.*, 2007) for all ages and in Detroit, USA, at 3.6% (Li et al., 2011) and in Hong Kong, at 3.2% (Lee *et al.*, 2006b) for a younger age group (less than 18 years), while a higher risk has been observed in New York, USA, at 7.0% (Silverman and Ito, 2010). In contrast, a study conducted in Taipei, Taiwan, did not observe an association with $PM_{2.5}$ (Bell et al., 2008).

Nevertheless, systematic review of panel studies showed an adverse effect of $PM_{2.5}$ on lung functioning in asthmatic subjects (Lee *et al.*, 2006a). Therefore, the result observed in the present study adds to the evidence showing positive associations between $PM_{2.5}$ and asthma-related emergency department visits in Al Jubail Industrial City, Saudi Arabia.

6.3.3 Sulphur dioxide (SO_2)

The present study found a significant positive association between increase in SO_2 levels and increase in asthma-related emergency department visits. This finding supports previous research which has observed this positive association with younger age groups (Sunyer et al., 1997; Sunyer et al., 2003; Li et al., 2011; Samoli et al., 2011) and all ages (Cirera *et al.*, 2012). The current study indicated a positive and statistically significant association between SO_2 levels and asthma-related emergency department visits for all day lags (0-7). The most significant increase in asthma-related emergency department visits was 5.4% (95% CI: 2.4, 8.5) for all ages associated with an inter-quartile range (IQR) change in SO_2 (2.0ppb) at lag day 2. The result of the present study is similar to a recent study conducted in Cartagena, Spain, which found a 5.2% (95% CI: 1.4, 11.0) increase in asthma visits for all ages per 10μg/m^3 increase in SO_2 levels at lag day 4 (Cirera *et al.*, 2012). In addition, the estimated risk presented in this study falls within those reported by APHEA-1 in four cities (Sunyer et al., 1997) and APHEA-2 (Sunyer et al., 2003) in seven cities in Europe for asthma visits in children, which ranged between 1.3% and 7.5%. Other studies have reported no significant effect of SO_2 on asthma visits (Castellsague *et al.*, 1995; Sunyer *et al.*, 1997; Wong *et al.*, 1999; Galan *et al.*, 2003; Sunyer *et al.*, 2003; Ko *et al.*, 2007; Bell *et al.*, 2008). Most of these studies have reported an interaction between SO_2 levels and other pollutants such as PM, CO and NO_2 due to collinearity among pollutants generated by the same sources (Galan *et al.*, 2003; Sunyer *et al.*, 2003; Ko *et al.*, 2007; Bell *et al.*, 2008; Li *et al.*, 2011). This can result in removal of statistical significance of SO_2 in the multi-pollutant model. The estimated effect in the current study remained significant after adjustment for PM_{10}, $PM_{2.5}$ and NO_2 in the multi-pollutant model, which suggests that SO_2 may not simply act as a proxy for other pollutants, but has an independent effect. In addition, the results of most controlled-chamber experiments with asthmatics have consistently shown that they are more sensitive to SO_2 than non-asthmatics (Castellsague et al., 1995; WHO, 2006a).

6.3.4 Nitrogen dioxide (NO_2)

Another important finding was a significant positive association between increase in daily NO_2 levels and increase in daily asthma-related emergency department visits. This result agrees with other studies that observed these associations for all ages (Castellsague *et al.*, 1995; Sunyer *et al.*, 1997; Wong *et al.*, 1999; Galan *et al.*, 2003; Ko *et al.*, 2007; Cirera *et al.*, 2012) and for a younger age group (Sunyer *et al.*, 1997; Lee *et al.*, 2006b; Li *et al.*, 2011). In the current study, a significantly positive association between NO_2 levels and asthma-related emergency department visits was observed at lag day 1 and 3. The most significant increase in asthma-related emergency department visits at lag day 3 was 3.4% (95% CI: 0.8, 6.1) associated with inter-quartile range (IQR) change (7.6ppb). This estimated risk is similar to a previous study conducted in Madrid, Spain, which indicated a 3.3% increase of asthma visits at lag day 3 for all ages per 10μg/m^3 increase in NO_2 level (Galan et al., 2003). Another study in Detroit, USA, also showed a similar effect, with a 3.8% increase of asthma visits at lag day 5 for a younger age group (2-18 years) per IQR change in NO_2 (9.65ppb) (Li et al., 2011). A lower risk effect was reported in Cartagena, Spain, which was 2.6% at lag day 4 (Cirera et al., 2012), and in Hong Kong in 2007 (Ko *et al.*, 2007) and in 1999 (Wong et al., 1999), at 2.8% for lag day 0-4 and 2.6% for lag day 0-3 per 10μg/m^3 increase of NO_2 level. Furthermore, the APHEA-1 project observed a similar increase of 2.6% for asthma visits in younger age group (0-14 years) and 2.9% in older age group (15-64 years) (Sunyer et al., 1997). A higher increase was found in Barcelona, Spain, at 4.5% in summer and 5.6% in winter (Castellsague et al., 1995) per 25μg/m^3 increase in NO_2 level. Also, a higher risk was reported in Hong Kong with a 5.6% increase of asthma visits at lag day 3 in the younger age group (0-18 years) per IQR change in NO_2 (27.1ppb) (Lee *et al.*, 2006b). Conversely, a study in Taipei, Taiwan, failed to observe a statistically significant association of asthma visits for all ages (Bell et al., 2008). Likewise, studies of asthmatic children visits and NO_2 levels in Athens, -Greece, (Samoli et al., 2011), Milan-Italy (Giovannini et al., 2010) and Seattle, USA, (Norris et al., 1999) found no significant associations. These inconsistent results may be due to a higher correlation between NO_2 and other pollutants, such as PM, CO and SO_2 reported in previous studies (Norris et al., 1999; Bell et al., 2008; Giovannini et al., 2010; Samoli et al., 2011). Hence, NO_2 could be a marker to other pollutants

generated by traffic-related sources, such as PM (Sunyer et al., 1997; Atkinson et al., 2001). However, the estimated risk shown in the presented study remained unaltered on inclusion of the other pollutants in the multi-pollutant model. It can therefore be suggested that NO_2 is independently associated with asthma-related emergency department visits. Furthermore, panel studies among asthmatic subjects found that exposure to NO_2 levels was related to reduced pulmonary function, which suggests that the observed association may be plausible (Sunyer et al., 1997; WHO, 2006a).

6.3.5 Carbon monoxide (CO)

This study did not find a statistically significant association between daily CO levels and asthma-related emergency department visits. The lack of association observed on this study supports previous works conducted in Detroit, USA, (Li et al., 2011), Milan-Italy (Giovannini et al., 2010) and Taipei, Taiwan (Bell et al., 2008). In contrast to earlier findings, however, a significant positive effect of CO levels on asthma visits was found in two previous studies conducted in two cities within USA, Tacoma (Mar et al., 2010) and Seattle (Norris et al., 1999). They found that an inter-quartile range (IQR) in CO levels of 0.6 and 0.7ppm resulted in 10% increase (95% CI: 2.0, 19.0) in Tacoma and 3.0% increase (95% CI: 0.1, 6.0) in Seattle, in asthma-related emergency department visits respectively). These results might be related to the high correlations between CO and particulates, which range between 0.74 in Tacoma and 0.82 in Seattle. CO has no biological plausible mechanism for exacerbation of asthma, so this effect is interpreted as being related to traffic air pollution and not to CO itself (Norris et al., 1999; Mar et al., 2010).

6.4 Contribution of Different Sources on Pollutants and Health

Air pollution is a complex mixture of a number of individual pollutants, including particulates, gases, bioaerosols, and toxic substances (Samet and Krewski, 2007). Both 'mobile' sources (i.e. cars) and 'stationary' sources (i.e. smoke stacks) make significant contributions to urban ambient (outdoor) air pollution (WHO, 2016). In most urban areas, and increasingly in suburban areas, traffic related emissions are a major source of air pollution. Some of the major sources include emissions from manufacturing facilities (e.g. factories), power generation (e.g. coal-fired power plants) and natural sources, such as PM from dust and wildfires (Trasande and Thurston, 2005; WHO, 2016)

There is a lack of air quality assessment data from power plant generation in Saudi Arabia (Al-Jeelani, 2008). Only few investigations have been carried out to evaluate urban air pollution in Saudi Arabia. Most studies conducted in the capital city, Riyadh, were limited in scope and in many cases were not enough to evaluate the whole picture of air quality in the city (Al-Rehaili, 2002). In fact, most studies were conducted for only a few hours, in a limited number of locations, and mostly for one or only few air quality parameters, other were spot check type studies (Al-Rehaili, 2002).

However, in Al Jubail, as elsewhere, there is a mix of primary pollutants (particles, carbon monoxide, sulphur dioxide, and nitrogen dioxide) and of secondary pollutants (particulate matter and ozone). Additionally, sulphur dioxide and nitrogen dioxide contribute to the formation of secondary particles and nitrogen dioxide is critical in the formation of ozone. Given these interrelationships and the not surprising correlations among concentrations of these pollutants, separating their effects may prove difficult (Samet and Krewski, 2007). The emphasis on identifying toxicity determining characteristics of particulate matter is even more challenging, as particulate matter itself is a mixture having multiple sources (Samet and Krewski, 2007).

The WHO concluded in 2007 that current knowledge does not allow specific quantification of the health effects of emissions from different sources, or of individual components. In 2009, the EPA integrated science assessment concluded that “there are many components contributing to the health effects of

$PM_{2.5}$, but not sufficient evidence to differentiate those sources (or constituents) that are more closely related to specific health outcomes". The integrated science assessment further noted that a number of source types – including motor vehicle emissions, coal combustion, oil burning, and vegetative burning– are associated with health effects and went on to include crustal material as another potentially toxic component (WHO, 2013).

A recent published study by WHO used a total of 419 source apportionment records from 51 countries to calculate regional averages of sources of ambient particulate matter (Karagulian *et al.*, 2015). This study shows, based on the available information, that traffic (25%), combustion and agriculture (22%), domestic fuel burning (20%), natural dust and salt (18%), and industrial activities (15%) are the main sources of particulate matter contributing to cities' air pollution (Karagulian *et al.*, 2015). The relative importance of different sources contributions varies among pollutants over time and from one community to another (Trasande and Thurston, 2005; Karagulian *et al.*, 2015).

Atmospheric processes that lead to the formation of particles as a result of gaseous traffic, heating and agriculture emissions appear to be most considerable in North America, Western Europe, Turkey and the Republic of Korea (Karagulian *et al.*, 2015). Domestic fuel burning dominates the contributions to particulate matter in Eastern Europe and in many developing countries in Africa (Karagulian *et al.*, 2015). In the developing countries, this source is likely to be associated with cooking, while in Eastern Europe the use of coal for heating seems to be the most probable reason. Natural dust is the main source of PM10 in the Middle-East and Northern African countries, likely due to their vicinity to arid areas. Sea salt is the most important natural source of PM10 in north-western Europe (Karagulian *et al.*, 2015).

The few studies based on source apportionment provide an opportunity to compare the short-term health effects of particles from biomass combustion with particles from traffic – the source with the most evidence on health effects (Krewski and Rainham, 2007; WHO, 2013). In a study conducted in Copenhagen (Andersen *et al.*, 2007), particles from biomass combustion were associated with cardiovascular and respiratory hospital admissions, whereas particles from traffic were not (Andersen *et al.*, 2007; WHO, 2013). Some studies also found that

particles from locations with high traffic areas induce more effects than those from lower traffic areas (Andersen *et al.*, 2007; Krewski and Rainham, 2007).

Only two recent studies have looked at the associations between desert dust days and hospital admissions (WHO, 2013). A study conducted in Hong Kong (Tam *et al.*, 2012), reported an increased rate of hospitalization for chronic pulmonary disease, but not for pneumonia or influenza, during desert dust days (Tam *et al.*, 2012). In contrast, in a study in Nicosia, Cyprus (Middleton *et al.*, 2008), desert dust days were associated with an increased rate of hospitalization for cardiovascular, but not respiratory, causes (Middleton *et al.*, 2008; WHO, 2013). Evidence for an effect of desert dust on human health is increasing, but at the moment it is not clear whether crustal, anthropogenic, or biological components of dust are most strongly associated with the effects (WHO, 2013). Evidence presented in this thesis from Al Jubail confirms the previously observed association between air pollution and asthma admissions in a Middle East setting. In the Al Jubail industrial city it is likely that industrial, mobile and desert dusts contribute to the PM pollution.

Epidemiological studies have not extensively examined the potential for statistical interactions among pollutants. This is likely due to the moderate to high correlation among pollutants in some locations, and the existence of pollutant mixtures, making it difficult to identify the effects of individual pollutants in observational studies (Samet and Krewski, 2007; WHO, 2013). However, the observed effects of the major air pollutants on asthma hospitalization reported here and elsewhere are biologically plausible (WHO, 2013). The evidence for a biological mechanism, derived from both epidemiological and toxicological studies, has also increased and indicates that exposure to those pollutants is associated with systemic inflammation, oxidative stress and alteration of the electrical processes of the heart (WHO, 2013; Zhang *et al.*, 2015).

Finally, the focus of the current study is on the concentration-response of pollutants and not to the sources-response. Therefore, the health estimates reported here are not linked to specific sources. However, in a region with at least three important sources of air pollution, biomass burning and industrial and automotive emissions, it can be seen that asthma aggravation measured by increased emergency department visits is clearly associated with outdoor

concentrations of common air pollutants in Jubail Industrial City. Identification of the source of pollutant most clearly related to the asthma visits was not intended in the current study, but this represents an important area for future research.

6.5 Strengths and Limitations of Time-Series Analysis

The strength of this analysis is that to the best of my knowledge, this study is the first to show a relationship between air pollution and asthma-related emergency department visits in an industrial city in Saudi Arabia. The data were recent and collected over a considerably long study period (from January 2007 to December 2011) from a reliable central-computerised system of the Medical Records Department and Environmental Department from the Royal Commission of Al Jubail Industrial City, Saudi Arabia.

Similar to other ecological time-series studies, this study was limited by the fact that precise individual level of exposure to a specific pollutant could not be assessed. The interaction between pollutants introduces possible collinearity issues, and limitations of the time series design also imposed limits when assessing which particular pollutant had a direct adverse effect on asthmatics. Nevertheless, the correlation between the air pollutants in the model was weak, which increases the validity of the model. Another inherent limitation in this time-series study may potentially occur with regard to exposure misclassification. The fixed-site ambient monitoring station may not reflect the true exposure of the people living in Al Jubail industrial city.

Data on hospital emergency department visits capture only the most severe cases of asthma and are not representative of mild or less severe cases. However, the extended period (2007-2011) and the detailed day-to-day variability for asthma-related emergency department visits and for air pollutant levels considered in this study should give enough reliability to the data presented in this study.

Although many important confounding variables have been controlled in the analysis, further adjustment of other confounders such as pollens and aeroallergens which may be alter the associations between asthma-related emergency department visits and air pollution, would be desirable. However, some studies have observed that pollen and aeroallergens could precipitate the exacerbation of asthma, whereas other studies have not (Anderson *et al.*, 1998; Galan *et al.*, 2003; Cirera *et al.*, 2012). It was not possible to include pollen in the present study, as this might be the focus of further works.

6.6 Discussion Summary

The main results found in this study suggest that $PM_{2.5}$, PM_{10}, SO_2 and NO_2 are positively associated with asthma-related emergency department visits, and have statistical significance on the same. The effects of these four pollutants were independent as the associations remained significant in the multi-pollutant model. The results presented in this study were consistent with other studies and suggested that the current outdoor air pollution levels were associated with increased asthma-related emergency department visits. Therefore, current air quality standards might not be sufficient to protect public health in this particular city. Further control of air pollution is likely to result in health benefits in Al Jubail Industrial City.

CHAPTER SEVEN

Discussion of Time-Activity Patterns and Microenvironment Exposure

Chapter:7 Discussion of Time-Activity Patterns and Microenvironment Exposure

7.1 Introduction

In this chapter, the findings of the time-activity patterns and microenvironment exposure have been discussed under four main headings: total time-spent at indoors, outdoors and commuting; personal exposure in different microenvironments; comparison between personal and fixed-site monitoring of $PM_{2.5}$ exposure levels; and limitations and strengths of this part of the study.

7.2 Time Spent in Different Microenvironments

The current study found that the students spent the majority of their total time indoors (88.6%), followed by transport (6.3%) and outdoor (5.1%). The total time spent indoors is consistent with what other studies reported in Europe and North America, which ranged between 81.5% to 94.5% (Jantunen *et al.*, 1998; Burke *et al.*, 2001; Lai *et al.*, 2004; Wu *et al.*, 2005; Kim *et al.*, 2006; Johannesson *et al.*, 2007; Braniš and Kolomazníková, 2010; Mohammadyan, 2011; Michikawa *et al.*, 2014; Wang *et al.*, 2014). The current study results indicate that most of the time spent indoors was at home (58.4%), which is similar to previous studies conducted in Bradford, UK, at 55.9% (Mohammadyan, 2011) and in Alpine, USA at 62.1% (Wu *et al.*, 2005). However, the time spent at school-indoors by students in the present study (25.9%) was higher than that reported in two cities in the USA, which is 13.1% in Alpine (Wu *et al.*, 2005) and 19.2% in Philadelphia (Burke *et al.*, 2001).

The total time spent outdoors in the present study was 5.1%, which is similar to what other studies reported in Toronto, Canada (5.3%) (Kim *et al.*, 2006), Gothenburg, Sweden (4.0%) (Johannesson *et al.*, 2007), the study on Air Pollution Exposure Distributions within Adult Urban Populations in six European cities (EXPOLIS) 4.0%(Marino M, 2002), Oxford, UK, (3.8%) (Lai *et al.*, 2004) and Bradford, UK (3.4%) (Mohammadyan, 2011). However, two studies in the USA have reported that more time was spent outdoors than the present study, standing at 14.1% in Alpine (Wu *et al.*, 2005) and at 10.8% in Philadelphia (Burke *et al.*, 2001).

The current study found that the amount of time spent in transportation was 6.3%. This is similar to the findings of other studies, which showed that the time spent in transport for the general population ranged from 4.7% to 8.0% (Jantunen *et al.*, 1998; Burke *et al.*, 2001; Marino M, 2002; Lai *et al.*, 2004; Kim *et al.*, 2006; Braniš and Kolomazníková, 2010; Mohammadyan, 2011). In contrast, two studies have reported lower proportions, 3.3% in Alpine, USA (Wu *et al.*, 2005) and 2.5% in Gothenburg, Sweden (Johannesson *et al.*, 2007).

7.3 Personal Exposure and Time-Activity Patterns

7.3.1 Personal exposure at home

The results of the present study indicate that the personal exposure median level of $PM_{2.5}$ differed statistically significantly across the various at home microenvironments. The lowest median personal exposure to $PM_{2.5}$ was found in the bedroom (27.0µg/m^3) which was significantly lower than other home microenvironments. A possible explanation for this might be that the students spent most of their time sleeping in their bedrooms, which means no physical activity occurred during this time. In addition, as shown earlier, the lowest median level of personal exposure to $PM_{2.5}$ was measured during sleep (26.0µg/m^3), and it is significantly lower than other physical activities such as standing (36.0µg/m^3), sitting (30.0µg/m^3) or exercising (42.5µg/m^3). The highest median levels were recorded in the living room and home yard (40.0µg/m^3) followed by kitchen (35.5µg/m^3). These microenvironments contain potential air pollution sources, which can influence the personal exposure to $PM_{2.5}$, such as cooking in the kitchen, incense in the living room and dust from outside the home. The mean personal exposure to $PM_{2.5}$ levels while indoors at home exceed the daily WHO Air Quality Guidelines (AQG), which are set at 25µg/m^3, but did not exceed the daily Al Jubail Air Quality Standard (AQS) of 50µg/m^3.

When comparing indoor and outdoor home microenvironments, the median personal exposure level was significantly lower while indoors at home (28.0µg/m^3) than outdoors (40.0µg/m^3). These results agree with the findings of the Air Pollution Exposure Distributions within Adult Urban Populations study (EXPOLIS), undertaken in six European cities, which showed that personal home-indoor levels were lower than outdoor home levels in Milan-Italy, Athens-Greece, Basel-Switzerland and Prague-Czech (Marino M, 2002). Similarly, studies conducted in Guangzhou-China (Wang *et al.*, 2014), Alpine-USA (Wu *et al.*, 2005) and Mexico City (Vallejo *et al.*, 2004) observed the same finding. However, one city in the EXPOLIS study, which was conducted in Helsinki-Finland (Marino M, 2002) and another study in Gothenburg-Sweden (Johannesson *et al.*, 2007) did not show this result.

In comparison to other studies, the present $PM_{2.5}$ levels while indoors at home (28.0μg/m^3) seem to be consistent with the EXPOLIS study, which found similar median $PM_{2.5}$ levels at home-indoor in Athens, Greece (26.6μg/m^3) and in Prague, Czech (24.8μg/m^3) (Marino M, 2002). However, studies conducted in Guangzhou, China (median=117.4μg/m^3) (Wang *et al.*, 2014), Mexico City (median=54.5μg/m^3) (Vallejo *et al.*, 2004) and Milan, Italy (median=34.7μg/m^3) (Marino M, 2002) found higher median $PM_{2.5}$ levels indoor at home than in the present study. Whereas studies conducted in Seoul, Korea (median=11.6μg/m^3) (Lim *et al.*, 2012), Stockholm, Sweden (median=10.0μg/m^3) (Wichmann *et al.*, 2010), Gothenburg, Sweden (median=8.6μg/m^3) (Johannesson *et al.*, 2007) and in two cities of EXPOLIS study, which are Basel, Switzerland and Helsinki, Finland (median=13.5μg/m^3) (Marino M, 2002), showed lower $PM_{2.5}$ levels. A possible explanation for the difference between these results may be related to different age groups among those studies, as the current study only focused on the male high school students aged between 16-18 years old while others looked at older age groups (> 21 years old) (Jantunen *et al.*, 1998; Marino M, 2002; Vallejo *et al.*, 2004; Johannesson *et al.*, 2007; Mohammadyan, 2011) or younger age groups (between 6-18 years old) (Wu *et al.*, 2005; Wichmann *et al.*, 2010; Wang *et al.*, 2014). A more likely explanation may be related to the ambient air quality, which is very high in China and Mexico City and low in Sweden, Basel and Finland.

House characteristics were identified from the questionnaire. The personal exposure to $PM_{2.5}$ levels while indoors at home was significantly different across a range of variables as shown in the results chapter 6. Only one participant indicated that he lived in an apartment, which had significantly higher median $PM_{2.5}$ levels (34.0μg/m^3) than detached (29.0μg/m^3), bungalow (27.0μg/m^3) or semi-detached (23.0μg/m^3) home types. Furthermore, homes with open fires located outside the house showed the highest $PM_{2.5}$ levels. The reason for this is not clear, but it may have something to do with the type of coal used for open fires located outside the house. It may also be related to the ventilation sources, since $PM_{2.5}$ levels were significantly higher for those who reported having no ventilation and were lowest for those who reported using a fan as ventilation source.

7.3.2 Personal exposure at school

In the current study, the median personal exposure to $PM_{2.5}$ differed significantly across school microenvironments. The lowest median levels were found in the head office (18.0µg/m^3), while the highest median level was recorded in the science lab (42.5µg/m^3) although this difference has not been found elsewhere in the literature. Students only spent about 3% of their time at school in the head office or in the science lab (as shown in the results chapter). The Majority of the time spent by students at school was in class (58%), where the median personal exposure to $PM_{2.5}$ was 24.0%. These levels did not exceed the daily WHO (AQG) of 25µg/m^3. Nevertheless, the median personal exposure across all school microenvironments was 25.5µg/m^3, which slightly exceeds daily WHO (AQG) limit values.

When comparing school-indoor and -outdoor microenvironments, the median personal exposure to $PM_{2.5}$ was significantly lower at school-indoors than -outdoors. These results agree with the findings of other studies in Guangzhou, China (Wang *et al.*, 2014), Stockholm, Sweden (Wichmann *et al.*, 2010) and Alpine, USA (Wu *et al.*, 2005), which also showed that personal school-indoor levels were lower than outdoor levels. In contrast to earlier findings, however, the median personal exposure level at school-indoor microenvironments presented in the present study (25.0µg/m^3) was lower than those reported in Guangzhou-China (57.5µg/m^3) (Wang *et al.*, 2014) and in Mexico City (93.3µg/m^3) (Vallejo *et al.*, 2004), but higher than levels reported in Stockholm, Sweden (Wichmann *et al.*, 2010) (8.3µg/m^3). None of these studies reported the amount of time spent in school-indoor microenvironments during the measuring period.

7.3.3 Personal exposure when commuting

The results of the present study did not show any statistically significant difference in personal exposure to $PM_{2.5}$ according to the commuting mode used by the participating students. The findings of the current study are consistent with other researchers', who also observed this result (Janssen *et al.*, 1997; Janssen *et al.*, 1999; Gauvin *et al.*, 2002). It seems possible that these results are due to the young population spending less time commuting than their adult counterparts (Leech *et al.*, 1996; Echols *et al.*, 1999; Gauvin *et al.*, 2002). This was confirmed by Echols *et al.* (1999) in Maryland, USA, and by Leech *et al.* (1996) in the Canadian Human Activity Pattern Survey. In the Canadian study, young population under the age of 12 spend less time commuting (3.6%) than adults do (6.0%) (Leech *et al.*, 1996).

The importance of the mode of transport in influencing the exposure of people is likely to be due to features and characteristics of the different modes. For example, in the present study, the lowest median personal exposure to $PM_{2.5}$ level was reported in car, while the highest was whilst walking. This finding is in agreement with findings of Gulliver and Briggs (2007), which showed that personal exposure to $PM_{2.5}$ in the car is significantly lower than during walking. The authors have speculated that personal exposure inside is less because the vehicle acts as a semi sealed environment. Therefore, this may result in lower personal exposure (Gulliver and Briggs, 2007). Another possible explanation for this is that inhalation rates for walkers may be higher than for the vehicle's occupants, and may therefore result in higher inhaled doses and exposure to $PM_{2.5}$ levels (Rank *et al.*, 2001; McNabola *et al.*, 2008; Kaur and Nieuwenhuijsen, 2009).

In comparison to other studies, the median personal exposure to $PM_{2.5}$ whilst walking in the present study was 46.0 $\mu g/m^3$, higher than in Leicester, UK (11.1$\mu g/m^3$) (Gulliver and Briggs, 2007), and in London, UK (25.3$\mu g/m^3$) (Kaur and Nieuwenhuijsen, 2009). In addition, the median personal exposure to $PM_{2.5}$ levels in car and bus found in the current study (35.5$\mu g/m^3$ and 42.5$\mu g/m^3$ respectively), were lower than those reported in Mexico City (64.2$\mu g/m^3$ and 101.7$\mu g/m^3$ respectively) (Vallejo *et al.*, 2004) and higher than those reported in London, UK (32.4$\mu g/m^3$ and 34.1$\mu g/m^3$ respectively) (Kaur and Nieuwenhuijsen,

2009), and in Leicester, UK for car (7.2μg/m^3) (Gulliver and Briggs, 2007). Furthermore, the personal exposure to $PM_{2.5}$ level in transport (car and bus) presented in the present study was 36.5μg/m^3. This is higher than those reported in Seoul, Korea (16.8μg/m^3) (Lim *et al.*, 2012) and in Prague, Czech Republic (11.7μg/m^3) (Braniš and Kolomazníková, 2010).

Comparison of results from different studies is not always possible, or indeed wise, due to the differences in study designs and monitoring equipment used. Furthermore, there are a range of additional factors which can influence results from these studies such as, for walking-based studies, proximity to other people, passing smokers in the street, position on the pavement/road; for motorised vehicles factors include the number of passengers, fuel type, vehicle upholstery, use of air conditioning/vents, and whether windows were open or closed. Direct comparison of results between many of these studies is almost always impossible, and notwithstanding attempts to replicate study designs in different areas is still problematic due to not being able to control for certain environmental factors (e.g. background air pollution, street characteristics, meteorology etc.). All in all, there are many factors which should be considered when comparing personal exposure levels obtained in different studies (Johannesson, 2013). Some comments can however be made by way of summarizing the general messages coming from these studies.

7.4 Comparison between Personal Exposure in Different Microenvironments

The microenvironments of interest in this study were divided into seven categories (home-indoors, home-outdoors, school-indoors, school-outdoors, indoors away from home or school, outdoors away from home or school, and transport). It is interesting to note that the $PM_{2.5}$ levels were found to significantly differ between each of these microenvironments. The highest median level of personal exposure to $PM_{2.5}$ was found outdoors away from home/school (44.0μg/m^3) followed by home-outdoors (40.0μg/m^3). The median level of $PM_{2.5}$ was significantly lower school-indoors (25.0μg/m^3) and home-indoors (28.0μg/m^3). Interestingly, all seven microenvironment categories presented in this study, with the exception of school-indoors, exceeded the daily WHO (AQG) of 25.0μg/m^3.

When comparing total indoors (home-indoors, school-indoors and indoors away from home/school) and total outdoors (home-indoors, school-indoors and away from home/school), the total outdoor median personal $PM_{2.5}$ exposure level (44.4μg/m^3) was significantly higher than across the indoor microenvironments (28.8μg/m^3). Although these results differ from some published studies (Rojas-Bracho *et al.*, 2002; Pekey *et al.*, 2010; Borgini *et al.*, 2011), they are consistent with other studies, which also suggest that personal exposure outdoors is higher than personal exposure indoors (Vallejo *et al.*, 2004; Wu *et al.*, 2005; Braniš and Kolomazníková, 2010; Wichmann *et al.*, 2010; Lim *et al.*, 2012). A possible explanation for this might be that personal exposure to $PM_{2.5}$ levels when outdoors is influenced by the level of atmospheric variations.

Previous studies conducted in Mexico City (Vallejo *et al.*, 2004), in Santiago, Chile (Rojas-Bracho *et al.*, 2002) and in Milan, Italy (Borgini *et al.*, 2011) have reported higher median levels of personal exposure for outdoor and indoor microenvironments than found in the present study. The higher PM2.5 levels observed from these previous studies may result from these being substantial exposure sources in both indoor and outdoor microenvironments. In contrast to earlier findings, however, the indoor and outdoor $PM_{2.5}$ levels obtained in the present study are higher than the levels obtained in previous studies conducted in Seoul, Korea (Lim *et al.*, 2012), Kocaeli, Turkey (Pekey *et al.*, 2010), Prague,

Czech (Braniš and Kolomazníková, 2010), Stockholm, Sweden (Wichmann *et al.*, 2010) and Alpine, USA (Wu *et al.*, 2005).

7.5 Total Personal Monitoring Exposures to $PM_{2.5}$

When comparing total personal monitoring exposures to $PM_{2.5}$ levels in the present study to other studies, this study has higher personal exposure levels than those reported by the EXPOLIS study in six European cities (with the exception of in Athens, Greece, which showed a similar result to the present study) (Marino M, 2002). In addition, previous studies conducted in Seoul, Korea (Lim *et al.*, 2012), Bradford, UK (Mohammadyan, 2011), Prague, Czech Republic (Braniš and Kolomazníková, 2010), Gothenburg, Sweden (Johannesson *et al.*, 2007), Toronto, Canada (Kim *et al.*, 2006), Alpine, USA (Wu *et al.*, 2005) and Oxford, UK (Lai *et al.*, 2004) have shown lower $PM_{2.5}$ personal exposure levels than the present study. In contrast, two previous studies conducted in Milan, Italy (Borgini *et al.*, 2011) and in Santiago, Chile (Rojas-Bracho *et al.*, 2002) have reported higher personal exposure levels to $PM_{2.5}$ than the present study. This discrepancy could be attributed to the study design; the present study measured personal exposure to $PM_{2.5}$ levels by the students themselves by carrying personal monitoring device in their backpack with inlet near to the breathing zone, whilst other studies (Marino M, 2002; Lai *et al.*, 2004; Braniš and Kolomazníková, 2010; Lim *et al.*, 2012; Michikawa *et al.*, 2014) relied on equipment carried by hand by volunteers. Therefore, this suggests that those studies may systematically underestimate or overestimate true personal exposure.

7.6 Comparison between Hourly Personal and Fixed-Site Monitoring of $PM_{2.5}$ Exposure Levels

The results of the present study showed that the fixed-site monitoring median $PM_{2.5}$ level (51.0µg/m3) was significantly higher than the personal monitoring median $PM_{2.5}$ level (30.0µg/m^3). In contrast to previous studies, which showed that the personal exposure to $PM_{2.5}$ levels were higher than the fixed-site station (Wu *et al.*, 2005; Kim *et al.*, 2006; Borgini *et al.*, 2011; Michikawa *et al.*, 2014). This result may be explained by the fact that the levels of ambient $PM_{2.5}$ measured by fixed-site monitoring station in the current study were much higher than those observed in earlier studies, with the exception of Borgini *et al.* (2011), who reported a slightly higher $PM_{2.5}$ level than the present study. The time series of ambient $PM_{2.5}$ levels from 2007 to 2011 (results chapter 4 of time series analysis) shows that the average ambient $PM_{2.5}$ levels in Al Jubail (64.6µg/m^3) exceeded the world average $PM_{2.5}$ levels reported by WHO from 2008 to 2013 (mean = 28.0µg/m^3) (WHO, 2014b).

In contrast to earlier findings, other studies reported relatively strong correlations between personal $PM_{2.5}$ and fixed-site $PM_{2.5}$ monitoring data (correlation coefficient ranged from 0.62 to 0.69) (Kim *et al.*, 2006; Borgini *et al.*, 2011; Michikawa *et al.*, 2014). The present study did not find a strong correlation between personal $PM_{2.5}$ and fixed-site $PM_{2.5}$ monitoring data. The log-transformed hourly personal $PM_{2.5}$ data showed a very weak but significant correlation with fixed-site $PM_{2.5}$ monitoring data (Pearson's correlation coefficient =0.164, p-value <0.001, n=488). The correlations between fixed-site and personal monitoring data were found to be significant at indoor microenvironments (Spearman's rank correlation=0.228, p-value < 0.001, n=427), but not at 'other' microenvironments (outdoor and transport). Given the high significant amount of time spent by students at indoor microenvironments, it seems probable that fixed-site monitoring data would not correlate well with personal measurements.

The observed differences between $PM_{2.5}$ levels measured via the personal monitor (SidePak) and the fixed-site monitor data must be interpreted with caution because co-location was not undertaken to establish that these devices would measure similar levels at the same location. This is as a limitation of the current

study design. Even if co-location is incorporated into the study, differences in the co-located measurement can be observed between the personal monitor and fixed-site monitor data due to vertical and horizontal differences in distance between the personal monitor and fixed-site monitor inlets. Even where a personal monitor is located within the same space occupied by fixed-site monitor there will generally still be a 1.5–2m difference in the vertical height of the personal monitor and fixed-site monitor inlets (Gulliver and Mosler, 2010).

Another factor that might contribute to the observed differences in personal and fixed site monitor values relates to measurements methods. The SidePak nephelometer has been shown in previous studies to overestimate the particle concentration by a factor of approximately 3.4 compared with the fixed site gravimetric method (Zhu *et al.*, 2007; Lee *et al.*, 2008). Because no co-location was undertaken, the data measured with the SidePak during the present study was not rescaled to account for any systematic overestimation, which may add additional uncertainty to the data. This limitation should be kept in mind throughout this thesis.

In addition to different measurement technologies, other important factor which may explain the differences that are observed between the fixed site and personal exposures is the fact that this present study (as most others dealing with personal exposure) did not measure exposures to $PM_{2.5}$ of only outdoor origin, but rather total $PM_{2.5}$ exposures. Thus, it the personal measure comprises a mixture of PM generated outdoors and from indoor sources, whereas the fixed site monitor data measures ambient air pollution concentrations, i.e. outdoor sources only. The failure to measure source-specific indicators is a weakness of the current study design.

7.7 Variation in Personal Exposure to $PM_{2.5}$

The second sub-objective of phase two was to estimate exposure error introduced by using fixed-site monitoring stations as a proxy for personal exposure. A multiple regression model was run to try to understand the variability in personal $PM_{2.5}$ exposure levels explained by the following predictor variables: the hourly fixed-site monitor $PM_{2.5}$ levels, time slot, participant age, home characteristics (including cooking fuel and ventilation), and microenvironment (indoor versus 'other' locations), as well as home ownership, house type, open fire place, exposure place, household members and temperature, although these latter variables did not to contribute to the model. This model explained only 28% of the variability in personal $PM_{2.5}$ exposure, on the one hand, by outdoor factors such as ambient levels of $PM_{2.5}$ from fixed-site monitor (19%) and on the other hand by indoor factors such as cooking fuel (9%) and ventilation (8%) as well as the other factors such as age of students and time slot (10% and 16% respectively). All these statistically significantly variables contributed to the model. These results are in agreement with other published studies that modelled $PM_{2.5}$ exposure among children (Gauvin *et al.*, 2002; Wu *et al.*, 2005). One study by Gauvin *et al.* (2002) developed a regression model for children aged 8-14 years to predict their 48-hours of personal exposure to $PM_{2.5}$ with an R^2 of 0.36. Similarly, Wu *et al.* (2005) reported an R^2 value of 0.41 for modelling 24-hour personal exposure among children aged 9-17 years. However, some studies reported a small R^2 value among asthmatic children, which ranged from 0.09 (Liu *et al.*, 2003) to 0.11 (Wu *et al.*, 2005). The researchers have speculated that the low explained variance is due to the lack of monitoring data at their school microenvironments (Wu *et al.*, 2005).

7.8 Comparison between RRs of AEDv for school age group before and after conversion ambient $PM_{2.5}$ levels in time-series analysis

Table 7.1: Key findings for RR of AEDv for school age group in time-series analysis before and after conversion ambient $PM_{2.5}$ levels for the period 2007-2012

Variable	Relative risk of AEDv for school age group (6-18 years old) per 10μg/m^3 increase
$PM_{2.5}$	At lag 0, RR increased 1.0% (95% CI: 0.7-1.3)
$PM_{2.5}$C	At lag 0, RR increased 1.7% (95% CI: 0.7-2.7)

The key findings from the time series analysis of AEDv for the school age group (6-18 years old) per 10μg/m^3 increase in $PM_{2.5}$ levels before and after conversion to the new scale presented in Table 5.22 (page 195) are summarised above in Table 7.1. The most statistically significant increases in AEDv were found on the same day of admission (lag 0) where RR per 10μg/m^3 increase in $PM_{2.5}$ concentration increased by 1.0% (95% CI: 0.7-1.3) for unconverted $PM_{2.5}$ levels and 1.7% (95% CI: 0.7-2.7) for converted $PM_{2.5}$ levels ($PM_{2.5}$C). The results presented in Chapter 5/Section 5.11 (Figure 5.4 page 179) show that $PM_{2.5}$ exposures assessed via personal monitoring are lower than those assigned using fixed site monitoring data, and the results in Table 5.22 (page 195) show that the time-series analysis risk estimates for AEDv in those aged 6-18 years for the period 2007-2012 based on the unconverted fixed-site monitoring station data may underestimate the risk when compared to the converted ambient $PM_{2.5}$ levels. This result may be explained by the fact that the risk associated with the converted 10μg/m^3 increase of ambient $PM_{2.5}$ levels is approximately equivalent to the risk associated with an unconverted 17μg/m^3 increase of fixed-site monitoring station; as a consequence, the risk for AEDv appears greater when using the converted versus unconverted exposure estimate. However, risk associated with the converted $PM_{2.5}$ level better reflects the association between personal exposure to $PM_{2.5}$ and AEDv. Furthermore, the results for both the converted and unconverted $PM_{2.5}$ levels are consistent with those of other studies, which reported a positive association between $PM_{2.5}$ levels and asthma

visits amongst children (0-18 years old) (Lee *et al.*, 2006b; Li *et al.*, 2011). The effect size in the current study adds to the evidence suggesting this personal monitoring and AEDv association is also present in this industrial city setting in Saudi Arabia.

7.9 Strengths and Limitations of Time-Activity Patterns and Microenvironment Exposure

The strength of this analysis is that, based on available evidence, this study is the first to identify factors that influence personal exposure to air pollution in an industrial city in Saudi Arabia.

In addition, this study used a personal air monitor device placed in the backpack and the height of the sample inlet on top of the backpack was close to the breathing height. Therefore, the backpack measurement reflected the personal exposure to $PM_{2.5}$ better than fixed-site measurement stations.

It is evident that the $PM_{2.5}$ measured by the personal air monitor does not reflect the exact dose that an individual is exposed to. Inhalation of $PM_{2.5}$ is dependent on the rate and pattern of breathing and for inhaled particles, on the time the air stays in the lungs, on personal activities and the level of exertion during an activity (for example, a number of factors are altered during exercise: cardiac output; ventilatory rate; tidal volume; thickness of mucous layer of the lung; possibly gas diffusion patterns (Carlisle and Sharp, 2001; Rom and Markowitz, 2007). However, this study setup provides a very detailed picture of the daily personal exposure to $PM_{2.5}$ and allows identification of activities leading to high exposure levels.

Another strength of this study was the various statistical methods that were used to evaluate the air pollution levels in different microenvironments.

A further strength of this research was that the sample size is large compared to efforts from other authors but is still limited as this sample cannot be regarded as representative for a complete population.

In addition, it was possible to record the detailed movements of students and the time spent in important microenvironments through the use of the GPS logging device. The GPS logger data can be used to verify whether the information about activities (including travel, outdoor, at school/work, at home, and at other locations), reported by the participant, is consistent with the actually recorded trip. The GPS was not used to its full extent in this study, but further work could be undertaken in future studies.

Finally, the tools, devices and methodology applied in this thesis provide a starting point to address issues and challenges regarding the quantification of human exposure to air pollution in everyday environments. They are not immediately applicable or transferable to other study areas and aims without further development and adjustments.

As with any study, there were also some limitations associated with this study. One limitation was that the results of the diaries differed in precision. Furthermore, since 27 different test students participated in this study, each of them had to familiarize themselves with the measurement equipment for the measuring period. As the measurement period was only 24 hours per student, the conclusions regarding long-term exposure cannot be drawn from these results, and would, for instance, require repeat samples distributed over the year. In addition, a generalization of these results is only feasible to a very limited degree. Only a sufficiently large database will allow us to draw quantitative conclusions concerning the influence of time-activity patterns and microenvironments on personal $PM_{2.5}$ exposure. Although students were encouraged to undergo their normal daily routine, leisure activities such as football training were not easy to accomplish with the backpack.

The SidePak measurement based on nephelometer measurement estimated the mass based on the Arizona Test Dust calibration factor (default factory calibration factor left at 1.00). For aerosols with different physical and/or optical properties than Arizona Test Dust, including mixtures from different aerosol sources, leaving the SidePak calibration factor set at 1.00 may lead to inaccuracies to the $PM_{2.5}$ levels measured at gravimetric sampler. (Lee *et al.*, 2008) and (Zhu *et al.*, 2007) concluded that the SidePak nephelometer overestimated the particle concentration by a factor of approximately 3.4 compared with the gravimetric method. Therefore, the $PM_{2.5}$ data measured with the SidePak during the present study was not modified by introducing rescaling factor, which may add additional uncertainty to the data. This is the limitation of the SidePak, which has to be kept in mind since throughout this thesis.

Over recent years many novel sensors have become available measuring body functions (such as, heart rate, ventilation, galvanic skin response, etc.). In

this current study, no proxy for physical activity was measured, making it impossible to truthfully report inhaled doses.

Another limitation of this study relates to participants carrying the backpack at home. Participants were educated on the proper handling of the monitoring pack, however we cannot be certain how long the monitoring pack was worn at home (although GPS data confirmed locations during home, transit, and school periods).

Previous studies have shown that seasonality can affect air pollutant concentrations (Kornartit *et al.*, 2010). The current study sampling campaign, conducted between Feb and May, did not allow assessment during both a winter and a summer season. It is anticipated that season may influence time-activity patterns, as well as meteorological circumstances, and daylight hours, thus potentially leading to different exposure profiles for individuals. Additionally, over the summer period, time-allocations would possibly have included more time spent outdoors due to school holidays, fieldwork and leisure activities. On the other hand, the summer time would have been even more challenging with respect to recruiting volunteers due to holidays, and summer time activities that may not be conducive to wearing the monitoring pack. Therefore, the current study could not be used to assess seasonality of exposure.

Finally, the tools, devices and methodology applied in this thesis provide a starting point to address issues and challenges regarding the quantification of human exposure to air pollution in everyday environments, and demonstrate their application in a unique setting – an industrial city in the Middle East. However, these approaches are not immediately applicable or transferable to other study areas or other study aims without further development and adjustments.

7.10 Discussion Summary

The findings of this chapter showed that although there appear to be similarities between time activity patterns in this small population sample from the Middle East and in Europe/USA, the levels of personal monitoring exposure to $PM_{2.5}$ in this industrial city appear high with most microenvironments categories presented in this study exceeding the daily WHO air quality guideline limits. In addition, the validity of fixed-site air monitoring data as a proxy for personal exposure to PM needs to be characterised so as to better understand the exposure error associated with this proxy measure.

The next chapter provides an overall discussion and conclusion of the three phases of my PhD and implications for researchers and practitioners of my findings.

CHAPTER EIGHT

Conclusion and Recommendations

Chapter:8 Conclusion and Recommendations

8.1 Introduction

In this chapter, an overall discussion and conclusion of the research conducted and a number of implications are highlighted. Recommendations for future research are also presented.

8.2 Overall Discussion and Conclusion

Summary of the main findings

In Al Jubail Industrial City, investigate the association between air pollution and asthma-related hospital visits, identify factors that influence personal exposure, and assess how the relationship between air pollution and asthma-related hospital visits varies when using different types of exposure estimates.

Most previous studies of this kind have been conducted in Europe and North America, where there is a temperate climate with distinct seasons. Based on the collected evidence, no study exploring these issues has been conducted in a hot and dry industrial city in the Middle East. This study therefore fills important gaps in our understanding of the influence of time-activity patterns and microenvironments on personal exposure in an industrial city in Saudi Arabia.

To achieve these aims, the thesis comprised of three phases:

Phase One: Time-series analysis

Phase one of the thesis aimed to undertake a time-series analysis to investigate the statistical association between air pollution and asthma-related emergency department visits (AEDv) in Al Jubail Industrial City, Saudi Arabia. This was achieved by using existing data from medical records of asthmatic patients visiting the Emergency Department at Royal Commission Hospital in Al Jubail from 2007 – 2011, linked to exposure data from fixed-site monitoring stations.

The residential community studied is located in the north of the industrial zone as well as the prevailing wind direction blowing from the north-west. The residential fixed-site monitoring station is classed as representing the community

area as shown in the city map in Chapter 3. This fixed-site station is not under the influence of any nearby direct source. Accordingly, the selected residential fixed-site fulfilled the aim of the current study and reflected the air pollutants of the community area in Jubail Industrial City.

Wind direction analysis was also used to look at the variability in air pollution levels and AEDv over time in relation to wind direction as well as to assess the choice of fixed-site monitoring station. The result indicated that the weighted mean of air pollutants is dominated by north-westerly winds. This significant contribution to the overall mean was due to the prevailing wind direction which is dominated by north-westerly winds. This would explain why there is no contribution from south and east winds. In addition, this proves the selected residential fixed-site satisfied the aim of this study and represented the air pollutants of the community area in Jubail Industrial City.

The main results yielded by this study (phase one) suggest that RRs of AEDv increased positively and with statistical significance with increasing ambient levels of $PM_{2.5}$, PM_{10}, SO_2 and NO_2. The effects of these four pollutants were independent, as the associations remained significant in the multi-pollutant model, where the pollutants are simultaneously introduced. In contrast, CO showed a non-statistically significant negative association with asthma AEDv. These findings are consistent with those of other studies undertaken elsewhere, and add to the growing body of literature concerning adverse health effects of air pollution on AEDv (Ko *et al.*, 2007; Bell *et al.*, 2008; Mar *et al.*, 2010; Silverman and Ito, 2010).

In addition, the results of the current study show that both $PM_{2.5}$ and PM_{10} were in excess of the daily and annual limit values for every year in the study period. The validity of this current study is supported by long study period, reliable central-computerised source of hospital admission data and good air-quality monitoring system of international standard for over five years.

Phase Two: Collection and analysis of microenvironment and time-activity pattern data

Phase two involved a collection and analysis of microenvironment and time-activity pattern data of personal exposure. This phase aimed to investigate two sub-objectives:

The first sub-objective was to identify factors that influence personal exposure to $PM_{2.5}$. The time-activity diary provided details to categorise the participants' time over the study period into broad groupings to reflect indoor, outdoor and travel related exposures, as well as time spent at specific locations and undertaking specific activities. For this phase, data were collected from 27 students in Al Jubail Industrial City. The specific population selected for sampling in this study survey consisted of male non-smoking students aged between 16 and 18 years who attend public high school in the middle of Al Jubail Industrial City, Saudi Arabia.

The current study found that the students spent the majority (88.6%) of their total time at indoors while 5.1% and 6.3% of their time was spent outdoors and in transportation, respectively. This result is consistent with those of other studies reported, mostly undertaken in Europe and North America (Jantunen *et al.*, 1998; Burke *et al.*, 2001; Lai *et al.*, 2004; Wu *et al.*, 2005; Kim *et al.*, 2006; Johannesson *et al.*, 2007; Braniš and Kolomazníková, 2010; Mohammadyan, 2011; Michikawa *et al.*, 2014; Wang *et al.*, 2014). When the microenvironments of interest in this study were aggregate into seven categories (home-indoors, home-outdoors, school-indoors, school-outdoors, indoors away from home or school, outdoors away from home or school, and transport), the $PM_{2.5}$ levels were found to significantly differ between each of these microenvironments. Interestingly, all seven microenvironment categories presented in this study, with the exception of school-indoors, exceeded the daily limit values of WHO air quality guidelines of 25.0μg/m^3.

When comparing total indoors (home-indoors, school-indoors and indoors away from home/school) and total outdoors (home-indoors, school-indoors and away from home/school), the total outdoor median personal $PM_{2.5}$ exposure level (44.4μg/m^3) was significantly higher than across the indoor microenvironments

(28.8µg/m^3). These results are consistent with other studies, which also suggest that personal exposure outdoors is higher than personal exposure indoors (Vallejo *et al.*, 2004; Wu *et al.*, 2005; Braniš and Kolomazníková, 2010; Wichmann *et al.*, 2010; Lim *et al.*, 2012). A possible explanation for this might be that the personal exposure to $PM_{2.5}$ levels when outdoors is influenced by high background levels of ambient particulates.

The second sub-objective of phase two was to estimate exposure error introduced by using fixed-site monitoring stations as proxy for personal exposure. The current study did not find a strong correlation between personal $PM_{2.5}$ and fixed-site $PM_{2.5}$ monitoring data collected over the same time period.

The observed differences between $PM_{2.5}$ levels measured via the personal monitor and the fixed-site monitor data must be interpreted with caution because co-location was not undertaken to establish that these devices would measure similar levels at the same location. This is a limitation of the current study design. Even if co-location is incorporated into the study, differences in the co-located measurement can be observed between the personal monitor and fixed-site monitor data due to vertical and horizontal differences in distance between the personal monitor and fixed-site monitor inlets. In addition, another important factor which may explain the differences that are observed between the fixed site and personal exposures is the fact that this present study (as most others dealing with personal exposure) did not measure exposures to $PM_{2.5}$ of only outdoor origin, but rather total $PM_{2.5}$ exposures. Thus, it the personal measure comprises a mixture of PM generated outdoors and from indoor sources, whereas the fixed site monitor data measures ambient air pollution concentrations, i.e. outdoor sources only. The failure to measure source-specific indicators is a weakness of the current study design.

The multiple regression model was run to try to understand the variability in personal $PM_{2.5}$ exposure levels. This model explained only 28% of the variability in personal $PM_{2.5}$ exposure, on the one hand, by outdoor factors such as ambient levels of $PM_{2.5}$ from fixed-site monitor (19%) and, on the other hand, by indoor factors such as cooking fuel (9%) and ventilation (8%) as well as the other factors such as age of students and time slot (10% and 16% respectively). All these statistically significantly variables contributed to the model. These findings

suggest that both the indoor and outdoor environments have sources that elevate the $PM_{2.5}$ concentrations to different extents, which in turn lead to higher personal exposures. An important finding of this study is that personal exposure to $PM_{2.5}$ can be significantly influenced by time-activity patterns, type of microenvironment as well as the time of the day.

Phase three: TSA of AEDv and ambient $PM_{2.5}$ levels converted from personal monitoring campaign

In this phase, the relationship of AEDv and air pollution was re-examined to investigate how it would change if personal air pollution exposures (from objective two) were incorporated into the exposure variable, instead of data from fixed-site monitor (from objective one). In order to investigate this relationship, a valid strategy was developed by using the insights gained of $PM_{2.5}$ levels from the personal monitoring campaign to refine the ambient concentrations derived from the fixed-site monitor, which can then be used to re-run the main model of time-series analysis.

The risk estimates for AEDv in those aged 6-18 years in time-series analysis based on unconverted ambient $PM_{2.5}$ levels measured at the fixed-site station data may slightly underestimate the risk when compared to the converted ambient $PM_{2.5}$ levels associated with personal exposure for the period 2007-2012. However, the results of $PM_{2.5}$ levels before and after conversion to the new scale are consistent with those of other studies, which reported a positive association between $PM_{2.5}$ levels and asthma visits amongst children (0-18 years old) (Lee *et al.*, 2006b; Li *et al.*, 2011). The effect size in the current study adds to the evidence suggesting this personal monitoring and AEDv association is also present in this industrial city setting in Saudi Arabia.

The result observed in the present study indicates that the ambient $PM_{2.5}$ levels measured at fixed-site station would be a good choice of exposure to estimate population exposure to ambient air pollution instead of personal exposure. In time-series studies, only population-level exposure estimates are needed, as the focus is entirely on quantifying a temporal effect. Whereas in individual-level studies, such as cohort studies exposure estimates for individual are required (Oezkaynak et al., 2013).

Conclusion

In conclusion, this thesis has revealed that relative risks of asthma-related emergency department visits increased positively and with statistical significance with increasing ambient levels of $PM_{2.5}$, PM_{10}, SO_2 and NO_2. The effects of these four pollutants were independent, as the associations remained significant in the multi-pollutant model. These results were consistent with other studies and suggested that the current outdoor air pollution levels were associated with increased asthma-related emergency department visits. In addition, the results of the current study show that both $PM_{2.5}$ and PM_{10} were in excess of the daily and annual limit values for every year in the study period. The study benefitted from a long study period, reliable central-computerized source of hospital admission data and good air-quality monitoring system operating to international standards for over five years.

Although there appear to be similarities between time activity patterns in this small population sample from the Middle East and in Europe/USA, the levels of personal exposure to $PM_{2.5}$ in this industrial city appear high in most microenvironments, exceeding the daily WHO air quality guideline limits. In addition, the validity of fixed-site air monitoring data as a proxy for personal exposure to PM needs to be characterised so as to better understand the exposure error associated with this proxy measure.

In general, the findings of this study improve the current understanding of the characterisation between personal, indoor and outdoor pollution levels among schoolchildren in Al Jubail Industrial City.

8.3 Recommendation for Further Studies and Policy Makers

Some recommendations can be drawn from the results and lessons learnt from this study. The results of this work have revealed the need for continued studies in this field, especially in the Middle East setting. Examples are given below.

Although many important confounding variables have been controlled in the time-series analysis, further adjustment of other confounders such as pollens and aeroallergens which may alter the associations between asthma-related emergency department visits and air pollution, would be desirable. It was not possible to include pollen in the present study, as this might be the focus of further works.

Further research is needed to help in better understanding the mechanisms involved in the exacerbations of asthmatic symptoms, as the chemical composition of the air pollutants appears to play an important role in the inflammatory process of the airways.

The use of multi-city air pollution studies for Saudi Arabia, such as those conducted in Europe and North America, would minimise sample size concerns and potentially lower uncertainty surrounding effect estimates.

Personal exposure monitoring is a viable method for improving the knowledge about individual level exposures and the contribution of different microenvironments, especially when compared to the use of fixed-site monitor data currently in the literature. For future research, it would be helpful to expand the study to include a larger sample size, from different age groups and to allow investigation of other factors such as socio-economic status for example. The current study sampling campaign, which conducted between Feb and May, did not allow assessment during both a winter and a summer season. Future work should examine personal exposures during different seasons.

Additional research is needed to measure the extent to which the health consequences of other pollutants in this area differ from the health impacts of air pollution in other regions of the world.

The findings of the current study showed that current air quality standards in this setting might not be sufficient to protect public health in Al Jubail Industrial City. This calls for greater awareness of environmental protection and the implementation of effective measures to improve the quality of air. It is important for policy makers to facilitate investigation of air pollution and health effects, as there is now sufficient compelling evidence describing the devastating health and socioeconomic impacts of air pollution on both individuals and the community. There is experience around the world, especially from developed countries, that can be drawn on regarding effective health policy.

Until air quality issues are resolved, the levels of air pollutants should be incorporated into weather forecasts, as is done elsewhere, so as to issue alerts to populations at risk, which may enable individuals to reduce their risks from outdoor air pollution. Finally, the tools, devices and methodology applied in this thesis provide a starting point to address issues and challenges regarding the quantification of human exposure to air pollution in everyday environments.

CHAPTER NINE

Appendixes

Chapter:9 Appendixes

9.1 Appendix A, Conferences Publications

International Epidemiology Association (IEA), 20th World Congress on Epidemiology (WCE) 2014 USA

International Society of Environmental Epidemiology (ISEE) 2014 USA.

Association between daily air pollution levels and asthma emergency department visits in Al Jubail Industrial City, Saudi Arabia

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Background: In the last few years, epidemiological studies have provided evidence that exposure to air pollution can aggravate symptoms in asthmatic patients. To the best of our knowledge, no study exploring this issue has been conducted in an industrial city in Saudi Arabia.

Objective: To investigate the statistical association between ambient air pollution and asthma emergency department visits in Al Jubail Industrial City in Saudi Arabia.

Method: Daily asthma emergency department visits (n = 1826), Air pollution levels (particulate matter ($PM_{2.5}$ and PM_{10}), sulphur dioxide (SO_2), carbon monoxide (CO) nitrogen oxides (NO_x) and weather variables (temperature and relative humidity) were constructed from data obtained from the Royal Commission of Al Jubail Industrial City for the period between 2007 and 2011. Data were analysed using a time series approach, which involved application of a generalised linear model (GLM). Relative risks (RRs) were estimated using

Poisson regression, controlling for weather variables, day of the week and holiday indicator, for lag times of 0 – 7 days.

Result: The association between AEDv and change in the quantity of SO_2, NO_2, $PM_{2.5}$ and PM_{10} remained positive and statistically significantly after adjustment in the multi-pollutants model. The RR (in percent %) of AEDv increased by 5.4% (95% CI: 2.4, 8.5) at lag 2 for SO_2 , 3.4% (95% CI: 0.8, 6.1) at lag 3 for NO_2, 4.4% (95% CI: 2.4, 6.6) for $PM_{2.5}$ and 2.2% (95% CI: 1.3, 3.2) for PM_{10} at lag 0 per IQR change in pollutants, 2.0ppb, 7.6ppb, 36μg/m^3 and 140μg/m^3 respectively. No significant associations between AEDv and CO were found.

Conclusion: Current levels of ambient air pollution were associated with asthma emergency department visits in Al Jubail. Reductions in air pollution levels are necessary to protect the health of the community.

Keywords: Air pollution, Asthma, Emergency Department visits and time-series analysis

Inaugural UK & Ireland Exposure Science Meeting (ESM), Edinburgh, 2013

The 7th UK & Ireland Occupational & Environmental Epidemiology (OEE), Edinburgh, March 2013

International Society of Environmental Epidemiology (ISEE), Basel, 2013

Exploring the Influence of Time-Activity Patterns on Personal Exposure to $PM_{2.5}$ in Different Microenvironments

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Introduction: Many epidemiological studies use outdoor concentrations of air pollutants as a proxy for personal exposure. However, exposure will result not only to these outdoor concentrations, but also to the concentrations in the different microenvironments in which a person spends time. In the recent past, technology has greatly improved, making it possible to do personal monitoring of indoor and outdoor microenvironments. While there is a growing literature on time-activity patterns and microenvironment exposures for populations in the USA and Europe, little research on this topic has been carried out in the Middle East.

Objective: This study aims to explore the influence of Time-Activity Patterns on personal exposure to $PM_{2.5}$ in different microenvironments in Al Jubail, an Industrial City in Saudi Arabia.

Method: 28 students aged between 16-18 years old were recruited and asked to record their detailed movements on a time-activity diary, at 15-minute intervals over a period of 24 hours, and to carry a small backpack containing a personal air monitor (SidePak AM510) to measure personal exposure to $PM_{2.5}$,

as well as a GPS device to help identify microenvironments, including travelling, outdoors, at school, at home, and in other locations.

Results: Majority of the total time was spent indoors (88.7%), similar to figures reported in studies in EU and North America. Indoor away from home microenvironments (shops, restaurants & gyms) had the highest $PM_{2.5}$ concentrations (86µg/m^3) followed by transport (car, bus, taxi) (65µg/m^3) and outdoor away from home (park, beach) (52µg/m^3). Lowest $PM_{2.5}$ concentrations were found at school-indoors and at home-indoors (29µg/m^3) and (36µg/m^3) respectively. All microenvironment categories exceed the reference concentration (25µg/m^3) of WHO air quality guideline.

Conclusion: The time activity patterns and microenvironment PM concentrations in the Middle East are not well characterised. While there appear to be similarities between time activity patterns in this small population sample from the Middle East and Europe/USA, the exposure levels in this industrial city appear very high.

International Society of Environmental Epidemiology (ISEE) Basel, August 2013

International Society of Exposure Sciences (ISES) Basel, August 2013

Personal versus fixed-site monitoring for assessing $PM_{2.5}$ exposure in an industrial city, Saudi Arabia

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Background: Air pollution is a known risk factor for adverse health effects. Many epidemiological studies use outdoor air pollution levels based on fixed-site monitoring data as a surrogate for human exposure. However, individuals spend, on average, 85% of their time indoors, where exposure sources differ from those outdoors. Personal monitoring allows more appropriate exposure estimation, but to date, little has been done to compare exposure based on fixed-site versus personal monitoring, and no such comparisons have been carried out in the Middle East.

Aims: To investigate the validity of fixed-site versus personal monitoring of $PM_{2.5}$ in an industrial city in Saudi Arabia.

Methods: We collected 24-hour personal monitoring data of $PM_{2.5}$ exposure from 28 students aged 16-18 years, using a SidePak AM510 set to record $PM_{2.5}$ levels every minute. Students completed a time-activity diary to identify the time spent in key microenvironments, especially outdoors and indoors. Students also carried a GPS device to log their geographic position. Hourly ambient $PM_{2.5}$ levels for the same 24-hour periods were extracted for the nearest fixed-site monitoring station. The GPS data and home locations were used to plot the geographic distribution of residuals in GIS in order to explore the spatial variation in the fixed-site versus personal monitor data.

Results: Hourly mean $PM_{2.5}$ exposures were higher when assessed via personal monitoring (Mean=39.3µg/m^3, Median=30.5, SD=52.2, Range=816.3) than fixed-site monitoring (Mean=20.4µg/m^3, Median=12.2, SD=27.1, Range=257.6). There was a non-significant correlation between log-transformed personal and fixed-site monitor $PM_{2.5}$ levels when subjects were predominantly outdoors (Correlation=0.31, p=0.07, n=39hrs), and a weak, but significant correlation when subjects were indoors (Correlation=0.14, p<0.00, n=605hrs).

Conclusion: The validity of fixed-site air monitoring data as a proxy for personal exposure to PM needs to be characterised so that the exposure error associated with this proxy measure is better understood.

Saudi Scientific International Conference (SIC) London, 2012
The North East Postgraduate Conference (NEPG), Newcastle University 2012

Daily Air Pollution Levels and Asthma; Exploring the Influence of Time-Activity Patterns on Exposure in Al Jubail Industrial City, Saudi Arabia

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There is robust scientific evidence showing that exposure to air pollutants is associated with both acute and chronic health effects (WHO, 2014a). There is also evidence that exposure to air pollution can aggravate symptoms in asthmatic patients. Some of these studies have evaluated the short-term effects of particulate matter on asthma attacks and emergency department visits.

Many epidemiological studies have used outdoor concentrations of air pollutants as a surrogate for human exposure (Avery et al., 2010). A common feature of such studies is their reliance on ambient fixed-site measurement stations. In the recent past, the technology has greatly improved, making it possible to do personal monitoring to cover both indoor and outdoor environments in detail.

Little is known about the relationship between personal or indoor and outdoor air pollutants concentrations in developing countries, especially in countries within the Middle East. To the best of our knowledge, no study of this kind has been conducted in an industrial city in Saudi Arabia. This study will investigate the association between exposure to air pollution and asthma-related hospital visits and identify factors that influence personal exposure in Al Jubail Industrial City. Such a study will fill important gaps in our understanding of the influence of time-activity patterns and microenvironments on personal exposure in this setting.

9.2 Appendix B, Questionnaire, Time-Activity Diary, Information Sheet & Consent Form in English and Arabic Forms

Subject ID: .....

Newcastle University

Questionnaire Form

Daily Air Pollution Levels and Asthma; Exploring the Influence of Time-Activity Patterns on Exposure in Al Jubail Industrial City, Saudi Arabia

Salem AlBalawi

PhD student, Institute of Health & Society

Newcastle University

Thank you for filling in this questionnaire. This will help us learn about aspects of your lifestyle and your home so that we understand more about the types of air pollutants that you might be exposed to on a regular basis. We will use the information you provide here to help us interpret the time-activity data, the personal air monitor data, and the GPS data that we are also collecting. Together we hope this data will allow us to better understand the influence of time-activity patterns and microenvironments on personal exposure.

The contents of this questionnaire will be confidential. Under no circumstances will the information provided by you be disclosed.

Version 1.1 July 2011

Subject ID: .....

Lifestyle & Activity

1. Are you? (Please tick one box)	
a) A student	☐
b) Employed	☐
c) Non employed	☐
d) Other (Please specify):	☐

2. If you are student, write the name of school/college you are currently attending.
(Please write here): .....

3. How old are you?
(Please write here): ..... Years

4. How do you usually travel to school/college or work or other location on a daily basis? (Please tick one box)	
a) Car	☐
b) Bus	☐
c) Taxi	☐
d) Walk	☐
e) Other (Please specify):	☐

Building Characteristics

5. Is your home: (Please tick on box)	
a) Owned by you or someone in this household	☐
b) Rented from a private landlord	☐
c) Other (Please specify):	☐

6. What type of house do you live in (Please tick on box)	
a) Detached house	☐
b) Semi-detached house	☐
c) Bungalow	☐
d) Apartment	☐
e) Room in hall of residence	☐
f) Other (Please specify):	☐

7. Do you have in your home an open fire?	
a) Yes, How many?	☐/.....
b) No	☐

Subject ID: .....

8. What type of cooking fuel is used? (please tick one box or check all that apply)	
a) Mains Gas	☐
b) Electric	☐
c) Bottled Gas	☐
d) Gas/Electric Combination	☐
e) Other (Please specify):	☐

Ventilation

9. In the warm weather, do you usually use? (Please tick all that apply)	
a) Fan	☐
b) Air-conditioning	☐
c) Open windows	☐
d) None	☐
e) Other (Please specify):	☐

Potential Exposures

10. Which of the below you are regularly exposed to either yourself or from other people around you? (Please tick all that apply)	
a) Tobacco Smoke	☐
b) Water-Pipe Smoke	☐
c) Candles	☐
d) Incense	☐
e) Open fire rooms	☐
f) Other (Please specify):	☐

11. Are you smoker?	
a) Yes, How many cigarette per day	☐ / .....
b) No	☐

12. If you are smoker. Which type of smoke do you have?	
a) Tobacco Smoke	☐
b) Water-Pipe Smoke	☐
c) Both	☐

Subject ID: .....

13. If you chose any of the above, where you are regularly exposed? (Please tick all that apply)	
d) At home	☐
e) At school/college	☐
f) At work	☐
g) Coffee shops	☐
h) Other (Please specify):	☐

14. Please provide the number of persons in your household, including yourself, who are in each of the following age categories. (Please write your answer)	
a) 16 years old or younger	..... ☐
b) Above 16 years	..... ☐

15. Could you please mark on the map the usual places within AlJubail that you frequently visit?

Thank you very much; you have now completed this questionnaire.

Please enter the date you completed this questionnaire:			
Date	Day	month	Year

If there is anything you like to add or comment on, please do so in the space provided below:

4

Time-Activity Diary Form English Version

PARTICIPANT ID:_____ DATE: _____ Day of Week: _____

Newcastle University

TIME-ACTIVITY DIARY

Time	Activity	Location (please specify)	Indoor at Home	Indoors at School	Indoors away from Home	Outdoors. If yes, how many min.	In transit. If yes, how many min.	Smoker/Near Smoker If yes, how many min.
6:00 - 6:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:15 - 6:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:30 - 6:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:45 - 7:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:00 - 7:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:15 - 7:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:30 - 7:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:45 - 8:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:00 - 8:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:15 - 8:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:30 - 8:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:45 - 9:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:00 - 9:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:15 - 9:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:30 - 9:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:45 - 10:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:00 - 10:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:15 - 10:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:30 - 10:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:45 - 11:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:00 - 11:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:15 - 11:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:30 - 11:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:45 - 12:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:00 - 12:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:15 - 12:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:30 - 12:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:45 - 1:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
1:00 - 1:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
1:15 - 1:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
1:30 - 1:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
1:45 - 2:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
2:00 - 2:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
2:15 - 2:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
2:30 - 2:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
2:45 - 3:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
3:00 - 3:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
3:15 - 3:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
3:30 - 3:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
3:45 - 4:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
4:00 - 4:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
4:15 - 4:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
4:30 - 4:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
4:45 - 5:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___

Time	Activity	Location (please specify)	Indoor at Home	Indoors at School	Indoors away from Home	Outdoors: If yes, how many min.	In transit: If yes, how many min.	Smoker/Non Smoker If yes, how many min.
8:00 - 8:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:15 - 8:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:30 - 8:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:45 - 6:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:00 - 6:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:15 - 6:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:30 - 6:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
6:45 - 7:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:00 - 7:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:15 - 7:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:30 - 7:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
7:45 - 8:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:00 - 8:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:15 - 8:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:30 - 8:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
8:45 - 9:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:00 - 9:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:15 - 9:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:30 - 9:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
9:45 - 10:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:00 - 10:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:15 - 10:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:30 - 10:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
10:45 - 11:00 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:00 - 11:15 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:15 - 11:30 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:30 - 11:45 pm			❑	❑	❑	❑ /___	❑ /___	❑ /___
11:45 - 12:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:00 - 12:15 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:15 - 12:30 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:30 - 12:45 am			❑	❑	❑	❑ /___	❑ /___	❑ /___
12:45 - 1:00 am			❑	❑	❑	❑ /___	❑ /___	❑ /___

* At night time the monitor will be next to the bed and no time activity will be required.

The following are examples of locations:

Indoors at Home	Indoors Away from Home	In Transit	Outdoors
Kitchen	Grocery store/Pharmacy	Car	School/College
Bedroom	Hospital/Doctor's office	Bus	Park/ Beach
Bathroom	Exercise Group	Train	Gas station
Living room	Shopping mall/Bank	Taxi	Someone else's yard

Activities Example List:

Leisure	Cleaning	Office
Sleeping	Washing	Watching TV
Computer Work	Laundry	Reading
Shower/bath	Cooking	Eating
Shopping	Ironing	Walking

Participant Information Sheet English Version

Subject ID: .....

Newcastle University

PARTICIPANT INFORMATION SHEET

Daily Air Pollution Levels and Asthma; Exploring the Influence of Time-Activity Patterns on Exposure in Al Jubail Industrial City, Saudi Arabia

Introduction

Greetings! This form contains information about a study on time-activity patterns/microenvironments and drivers of personal exposure being undertaken by Newcastle University with the approval of the Al Jubail Royal Commission. To be sure you understand the study we ask you to read this form (or have it read to you). If this form contains some words that you do not understand, please ask us to explain.

Reason for the research

This study is being conducted in order to identify factors that influence personal exposure to air pollution to allow us to better understand why some people are exposed to higher levels of air pollution than others, and potentially why some people's health is more adversely impacted by this exposure.

General Information about the study

We are inviting students between ages of 16 – 24 years who attend a secondary school or a university in Al Jubail industrial city to participate in this study.

Your part in the research

If you agree to participate in this study:

1. You will be asked to carry a small backpack for 24 hours which contains a small personal air monitor, which may be emitted a low level background noise, and a GPS device. We ask that you carry this backpack with you wherever you go over the 24 hours, including when travelling, outdoors, at school, at home, and inside other locations.
2. You will be asked to complete a time-activity diary noting your location and activity every 15 minutes throughout the 24 hours, at night time the monitor will be next to the bed and no time activity will be required.
3. You will be asked to complete a questionnaire to assess potential sources of exposure to air pollution that might relate to your usual travel habits, home or lifestyle.
4. We will need to collect contact information from you so that we can contact you to arrange a convenient time to provide you with the study equipment, however this information will be kept confidential
5. If you complete the study you will be given a £10 voucher to thank you for your time.

Subject ID: .....

Confidentiality

I assure you that all the information collected from you will be kept confidential. Only people working in this research study will have access to the information. We will be compiling a project report based on the data collected for this study, but no individuals will be able to be identified from these data.

Who to contact

If you have questions about this study, you should contact the Principal Investigator Mr. Salem AlBalawi, PhD student at IHS, Newcastle University. Telephone Number: (UK) +44 (0) 785 910 4196. (SA) +966 (0) 559 556 899. Email: salem.albalawi@ncl.ac.uk

I want to take part!

Great news! Please complete the consent form and hand it back to Mr. AlBalawi. Remember that you are under no obligation to take part and you can withdraw your participation at any point in the study without giving a reason.

Under 18 years

If you are under 18 years old you and you want to take part at this study you need to complete these details below with your parent and hand it back to your school.

Parent (or Guardian) Name:

Phone Number:

Address:

Signature

Date

....

Email:

Study Consent Form English Version

Subject ID: .....

Newcastle University

Study Consent Form

Title of project: Daily Air Pollution Levels and Asthma; Exploring the Influence of Time-Activity Patterns on Exposure in Al Jubail Industrial City, Saudi Arabia

Please tick all of the boxes that you agree with:	
1. I confirm that I have read and understood the information sheet for the above study and have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.	
2. I understand that my participation is voluntary and that I am free to decide not to participate in any aspect of the study, without giving any reason, at any time.	
3. I am aware that my movements will be recorded by GPS over 24 hrs data collection period.	
4. I confirm that I would like to take part in this study.	

Name of Participant:

Phone Number:

Address:

Date

..... Day ..... month ..... year

Signature

Email

Name of Researcher:

Date

..... Day ..... month ..... year

Phone Number:

Signature

Questionnaire Form Arabic Version

الرقم التعريفي

نموذج الإستبيان

عنوان البحث :إستكشاف تأثير أنماط النشاط-الزمني على مراقبة جودة الهواء في مدينة الجبيل الصناعية, المملكة العربية السعودية

الباحث سالم البلوي

طالب دكتوراه في الصحة العامة والبيئة

جامعة نيوكاسل – بريطانيا

أشكرك على تفضلك بملء هذا الإستبيان. فهذا سوف يساعدنا على معرفة بعض الجوانب من نمط حياتك وكذلك بعض خصائص منزلك. حتى نتمكن من معرفة المزيد حول أنواع ملوثات الهواء التي قد تتعرض لها بشكل منتظم. سوف تستخدم هذه المعلومات من أجل تفسير النشاط الزمني المصاحب للتعرض الشخصي لتلوث الهواء وربطها مع بيانات تحديد المواقع (جي بي إس). بتعاونك معنا نأمل جميعاً نحو فهم أفضل لتأثير النشاط الزمني في البيئات الصغيرة على التعرض الشخصي لتلوث الهواء.

جميع محتويات هذا الإستبيان سيتم التعامل معها بشكل سري. ولن يتم الكشف عن هذه المعلومات التي قدمتها لأي جهة أخرى تحت أي ظرف من الظروف.

الإصدار 1.1 يناير 2012

Newcastle University
جامعة نيوكاسل

الرقم التعريفي ☐

نمط الحياة والنشاط

1. هل أنت؟ (الرجاء إختيار صندوق واحد)	
أ) طالب	☑
ب) موظف	☐
ج) غير موظف	☐
د) اخرى (الرجاء حدد):	☐

2. إذا كنت طالب الرجاء كتابة إسم المدرسة/الكلية الملتحق بها حاليا
(الرجاء الكتابة هنا): مدرسة الاحساء الثانوية

3. كم عمرك؟
(الرجاء الكتابة هنا): ..... (سنة)

4. كيف تذهب يومياً إلى المدرسة/الكلية أو العمل أو أي مكان آخر بشكل منتظم؟ (الرجاء إختيار صندوق واحد)	
أ) سيارة	☐
ب) باص	☐
ج) تاكسي	☐
د) سيراً على الأقدام	☐
ه) اخرى (الرجاء حدد):	☐

خصائص بناء المنزل

5. هل المنزل الذى تسكن به حالياً؟ (الرجاء إختيار صندوق واحد)	
أ) مُلك لك أو لأحد أفراد أسرتك	☐
ب) مؤجر من قبل مالك آخر	☐
ج) اخرى (الرجاء حدد):	☐

6. ماهو نوع المنزل الذى تسكن به؟ (الرجاء إختيار صندوق واحد)	
أ) فيلا مقصولة	☐
ب) فيلا مشتركة (دبيلوكس)	☐
ج) منزل من دور واحد	☐
د) شقة	☐
ه) غرفة في مجمع سكني	☐
و) اخرى (الرجاء حدد):	☐

7. هل يوجد في منزلك غرفة موقد حطب (مشبّ)؟ (الرجاء إختيار صندوق واحد)	
أ) نعم. كم عددها؟ مكانها داخل المنزل؟	☐...../.....
ب) لا.	☐

Newcastle University
جامعة نيوكاسل

الرقم التعريفي ☐

8. ماتوع وقود فرن الطبخ المستخدم فى منزلك؟ (الرجاء إختيار كل مايتطبق عليك)	
أ) غاز مركزي	☐
ب) كهرباء	☐
ج) اسطوانة غاز	☐
د) مشترك (غاز/كهرباء)	☐
ه) اخرى (الرجاء حدد):	☐

التهوية

9. فى الطقس الدافئ, هل تستخدم غالباً؟ (الرجاء إختيار كل مايتطبق عليك)	
أ) المروحة	☐
ب) مكيف هواء	☐
ج) فتح النوافذ	☐
د) لاشيء	☐
ه) اخرى (الرجاء حدد):	☐

التعرض الشخصي

10. أى من الخيارات التالية تجد أنك تتعرض لها بشكل منتظم سواء من قبل نفسك أو من قبل المتواجدين حولك؟ (الرجاء إختيار كل مايتطبق عليك)	
أ) دخان السجائر	☐
ب) دخان الأرجيلة	☐
ج) الشموع	☐
د) البخور	☐
ه) دخان وقود الحطب	☐
و) اخرى (الرجاء حدد):	☐

11. إذا إخترت شيئاً من السؤال السابق, أين عادةً المكان المنتظم الذى تتعرض فيه؟ (الرجاء إختيار كل مايتطبق عليك)	
أ) في المنزل	☐
ب) في المدرسة/الكلية	☐
ج) في العمل	☐
د) في المقهى	☐
ه) اخرى (الرجاء حدد):	☐

12. هل أنت مدخن؟ (الرجاء إختيار صندوق واحد)	
أ) نعم.	☐
ب) لا.	☐

Newcastle University
جامعة نيوكاسل

الرقم التعريفي ☐

13. إذا كنت مدخن.. ماهو نوع التدخين الذى تمارسة؟	
أ) تدخين السجائر	☐
ب) الأرجيلة (الشيشه)	☐
ج) الاثنين معاً	☐

14. كم شخص يسكن معك فى نفس المنزل بما فيهم أنت, مع تحديد الفئة العمرية كما فى الأسفل؟ (الرجاء كتابة الإجابة)	
أ) 16 سنة فما دون	☐.....
ب) أكبر من 16 سنة	☐.....

15. الرجاء توضيح الأماكن على الخريطة إدناه التي تزورها بشكل متكرر داخل مدينة الجبيل الصناعية.

الرجاء كتابة تاريخ اليوم		
.....	.....	.....
اليوم	الشهر	السنة

إذا كنت ترغب بإضافة أي شئ أو تعليق حول هذا الإستبيان, الرجاء كتابتها في المساحة المخصصة أدناه:

Time-Activity Diary Form Arabic Version

Newcastle University
جامعة نيوكاسل

تاريخ اليوم ..... اليوم من الأسبوع .....

الرقم التعريفي

مذكرة النشاط الزمني

الوقت	النشاط	المكان (الرجاء حدد)	داخل المنزل	داخل المدرسة	مكان مغلق بعيد عن المنزل	مكان مفتوح (كم دقيقة مكثت فيه)	وسيلة انتقال (كم دقيقة مكثت فيها)	تدخين أو قريب من مدخن (كم دقيقة)
6:00 - 6:15 am			❑	❑	❑	❑/___	❑/___	❑/___
6:15 - 6:30 am			❑	❑	❑	❑/___	❑/___	❑/___
6:30 - 6:45 am			❑	❑	❑	❑/___	❑/___	❑/___
6:45 - 7:00 am			❑	❑	❑	❑/___	❑/___	❑/___
7:00 - 7:15 am			❑	❑	❑	❑/___	❑/___	❑/___
7:15 - 7:30 am			❑	❑	❑	❑/___	❑/___	❑/___
7:30 – 7:45 am			❑	❑	❑	❑/___	❑/___	❑/___
7:45 - 8:00 am			❑	❑	❑	❑/___	❑/___	❑/___
8:00 - 8:15 am			❑	❑	❑	❑/___	❑/___	❑/___
8:15 - 8:30 am			❑	❑	❑	❑/___	❑/___	❑/___
8:30 - 8:45 am			❑	❑	❑	❑/___	❑/___	❑/___
8:45 -9:00 am			❑	❑	❑	❑/___	❑/___	❑/___
9:00 - 9:15 am			❑	❑	❑	❑/___	❑/___	❑/___
9:15 - 9:30 am			❑	❑	❑	❑/___	❑/___	❑/___
9:30 - 9:45 am			❑	❑	❑	❑/___	❑/___	❑/___
9:45 - 10:00 am			❑	❑	❑	❑/___	❑/___	❑/___
10:00 - 10:15 am			❑	❑	❑	❑/___	❑/___	❑/___
10:15 - 10:30 am			❑	❑	❑	❑/___	❑/___	❑/___
10:30 - 10:45 am			❑	❑	❑	❑/___	❑/___	❑/___
10:45 - 11:00 am			❑	❑	❑	❑/___	❑/___	❑/___
11:00 - 11:15 am			❑	❑	❑	❑/___	❑/___	❑/___
11:15 - 11:30 am			❑	❑	❑	❑/___	❑/___	❑/___
11:30 - 11:45 am			❑	❑	❑	❑/___	❑/___	❑/___
11:45 - 12:00 pm			❑	❑	❑	❑/___	❑/___	❑/___
12:00 - 12:15 pm			❑	❑	❑	❑/___	❑/___	❑/___
12:15 - 12:30 pm			❑	❑	❑	❑/___	❑/___	❑/___
12:30 - 12:45 pm			❑	❑	❑	❑/___	❑/___	❑/___
12:45 - 1:00 pm			❑	❑	❑	❑/___	❑/___	❑/___
1:00 - 1:15 pm			❑	❑	❑	❑/___	❑/___	❑/___
1:15 - 1:30 pm			❑	❑	❑	❑/___	❑/___	❑/___
1:30 – 1:45 pm			❑	❑	❑	❑/___	❑/___	❑/___
1:45 - 2:00 pm			❑	❑	❑	❑/___	❑/___	❑/___
2:00 - 2:15 pm			❑	❑	❑	❑/___	❑/___	❑/___
2:15 - 2:30 pm			❑	❑	❑	❑/___	❑/___	❑/___
2:30 - 2:45 pm			❑	❑	❑	❑/___	❑/___	❑/___
2:45 – 3:00 pm			❑	❑	❑	❑/___	❑/___	❑/___
3:00 - 3:15 pm			❑	❑	❑	❑/___	❑/___	❑/___
3:15 - 3:30 pm			❑	❑	❑	❑/___	❑/___	❑/___
3:30 – 3:45 pm			❑	❑	❑	❑/___	❑/___	❑/___
3:45 - 4:00 pm			❑	❑	❑	❑/___	❑/___	❑/___
4:00 - 4:15 pm			❑	❑	❑	❑/___	❑/___	❑/___
4:15 - 4:30 pm			❑	❑	❑	❑/___	❑/___	❑/___
4:30 - 4:45 pm			❑	❑	❑	❑/___	❑/___	❑/___
4:45 – 5:00 pm			❑	❑	❑	❑/___	❑/___	❑/___

الوقت	النشاط	المكان (الرجاء حدد)	داخل المنزل	داخل المدرسة	مكان مغلق بعيد عن المنزل	مكان مفتوح (كم دقيقة مشيت فيها)	وسيلة تنقل (كم دقيقة مشيت فيها)	تدخين أو قريب من مدخن (كم دقيقة)
5:00 - 5:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
5:15 - 5:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
5:30 – 5:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
5:45 - 6:00 pm			☐	☐	☐	☐/___	☐/___	☐/___
6:00 - 6:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
6:15 - 6:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
6:30 - 6:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
6:45 – 7:00 pm			☐	☐	☐	☐/___	☐/___	☐/___
7:00 - 7:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
7:15 - 7:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
7:30 – 7:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
7:45 - 8:00 pm			☐	☐	☐	☐/___	☐/___	☐/___
8:00 - 8:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
8:15 - 8:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
8:30 - 8:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
8:45 -9:00 pm			☐	☐	☐	☐/___	☐/___	☐/___
9:00 - 9:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
9:15 - 9:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
9:30 - 9:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
9:45 - 10:00 pm			☐	☐	☐	☐/___	☐/___	☐/___
10:00 - 10:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
10:15 - 10:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
10:30 - 10:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
10:45 - 11:00 pm			☐	☐	☐	☐/___	☐/___	☐/___
11:00 - 11:15 pm			☐	☐	☐	☐/___	☐/___	☐/___
11:15 - 11:30 pm			☐	☐	☐	☐/___	☐/___	☐/___
11:30 - 11:45 pm			☐	☐	☐	☐/___	☐/___	☐/___
11:45 – 12:00 am			☐	☐	☐	☐/___	☐/___	☐/___
12:00 - 12:15 am			☐	☐	☐	☐/___	☐/___	☐/___
12:15 - 12:30 am			☐	☐	☐	☐/___	☐/___	☐/___
12:30 - 12:45 am			☐	☐	☐	☐/___	☐/___	☐/___
12:45 - 1:00 am			☐	☐	☐	☐/___	☐/___	☐/___

ملاحظة: عند وقت النوم الرجاء التوقف عن كتابة مذكرة النشاط اليومي وضع الحقيبة في مكان قريب من مكان نومك

أمثلة للأنشطة التي يمكن أن تقوم بها خلال اليوم:

مشاهدة التلفاز	التنظيف	التسوق
القراءة / الكتابة	الغسيل	عمل على الانترنت
الأكل	الكوي	مكالمة هاتف
المشي	الإستحمام	إسترخاء
رياضة	الطبخ	النوم

أمثلة للأماكن التي من ممكن زيارتها خلال اليوم:

في مكان مفتوح	مكان مغلق	داخل المنزل	وسيلة تنقل
تسوق	سوبرماركت صيدلية /	الصالة	سيارة
حديقة	/ مستشفى مستوصف	المجلس	باص
كورنيش	نادي رياضي	المشب	تاكسي أجره
محطة وقود	سوق مغلق بنك /	غرفة النوم	

Participant Information Sheet Arabic Version

Newcastle University
جامعة نيوكاسل

الرقم التعريفي ☐

معلومات عن الدراسة

عنوان البحث :إستكشاف تأثير أنماط النشاط-الزمني على مراقبة جودة الهواء في مدينة الجبيل الصناعية, المملكة العربية السعودية

المقدمة

السلام عليكم! هذه الورقة تحتوي على معلومات حول دراسة أنماط النشاط-الزمني في البيئات الصغيرة وأثرها على التعرض الشخصي. هذه الدراسة تحت إشراف جامعة نيوكاسل وتم أخذ الموافقة عليها من قبل الهيئة الملكية في الجبيل. نطلب منك أن تقرأ هذه المعلومات بنفسك وإذا كانت هناك أي كلمات غير واضحة، يرجى الطلب منا لشرحها وتوضيح معناها لك.

أسباب هذا البحث

تقوم هذه الدراسة من أجل تحديد العوامل التي تؤثر على مراقبة جودة الهواء وذلك من أجل إتاحة الفرصة لنا لفهم اسباب تعرض بعض الناس لمستويات عالية من ملوثات الهواء أكثر من غيرهم. وكذلك إحتمالية لماذا بعض الناس صحتهم تتأثر بشكل أكثر سلبي نتيجة لهذا التعرض.

معلومات عامة حول الدراسة

هذه الدراسة تستهدف الطلاب الذين تتراوح أعمارهم مابين 16 – 18 سنة. الذين يلتحقون بالمرحلة الثانوية في مدينة الجبيل الصناعية . وذلك من أجل المشاركة في هذه الدراسة.

الدور المطلوب منك أثناء هذه الدراسة

إذا وافقت على المشاركة في هذه الدراسة:

1. سوف يطلب منك أن تحمل حقيبة صغيرة لمدة 24 ساعة. تحتوي هذه الحقيبة على جهازين صغيرين: الأول جهاز شخصي لمراقبة الهواء، والذي يمكن أن يصدر صوت منخفض جداً من الضجيج أثناء العمل. والثاني جهاز صغير جداً لتحديد المواقع (جي بي اس). سوف نطلب منك أن تحمل هذه الحقيبة معك طيلة اليوم, بما في ذلك عند التنقل ، وفي الأماكن المفتوحة, في المدرسة، في البيت، وغيرها من الأماكن المغلقة.
2. سوف يطلب منك ان تكمل مذكرة النشاط اليومي لكل 15 دقيقة مع الإشارة إلى موقعك الحالي وذلك طيلة اليوم. وعند النوم سوف نطلب منك وضع الجهاز قريبا من مكان نومك.
3. سوف يطلب منك ملء إستبيان (لايتجاوز عشر دقائق) لتقييم المصادر المحتملة للتعرض الى تلوث الهواء والتي قد ترتبط بعادات التنقل المعتادة أو في المنزل أو نمط الحياة.
4. سوف نحتاج لجمع معلومات الإتصال بك وذلك حتى نتمكن من ترتيب موعد مناسب لإستلام الحقيبة. مع العلم أن تلك المعلومات سوف تحفظ بمكان آمن وتكون محاطة بسرية تامة.
5. عند إكتمال الدراسة سوف نقوم بمنحك قسيمة شرائية للتعبير عن شكرك نظير جهدك ووقتك في هذه الدراسة.

الرقم التعريفي ☐

السرية

أؤكد لك أنه سيتم الحفاظ بشكل سري على كافة المعلومات التي سوف يتم جمعها من خلال هذه الدراسة. ولن يستطيع الإطلاع عليها إلا الباحثين العاملين فقط في هذه الدراسة. ولن تستخدم إلا في نطاق البحث العلمي وذلك لإكمال مشروع بحث الدكتوراة. مع العلم أنه لن يتم التعرف بشكل فردي على المشاركين في هذه الدراسة من خلال تلك المعلومات.

بمن سوف تتواصل

إذا كان لديك سؤال حول هذه الدراسة. الرجاء التواصل مع الباحث المسؤول: سالم بن محمد البلوي, طالب دكتوراة في الصحة العامة والبيئة في جامعة نيوكاسل من خلال رقم الجوال 0559556899. ويمكنك كذلك التواصل معي فيما بعد في بريطانيا عن طريق نفس الرقم السابق (خدمة التجوال) أو الجوال البريطاني 00447859104196. أو من خلال البريد الإلكتروني salem.albalawi@ncl.ac.uk

أرغب بالمشاركة!

خير جميل. الرجاء إكمال المعلومات المطلوبة في الأسفل مع ولي الأمر. بالإضافة إلى إكمال نموذج الموافقة المرفق بنفسك ومن ثم تسليمها إلى الباحث المسؤول. وتستطيع الإنسحاب من هذه المشاركة في أي وقت خلال الدراسة وبدون إعطاء أي سبب.

إسم ولي الأمر:

رقم التليفون:

التوقيع:

العنوان:

التاريخ:

اليوم ..... الشهر ..... السنة .....

البريد الإلكتروني (إذا توفر):

Study Consent Form Arabic Version

الرقم التعريفي ☐

نموذج الموافقة على الدراسة

عنوان البحث :إستكشاف تأثير أنماط النشاط-الزمني على مراقبة جودة الهواء في مدينة الجبيل الصناعية, المملكة العربية السعودية

الرجاء وضع علامة (√) على كل المربعات التي توافق عليها:	
1. أؤكد أنني قد قرأت وفهمت ورقة المعلومات عن الدراسة المشار إليها أعلاه، وأتيحت لي الفرصة للنظر في المعلومات، وطرح الأسئلة وأُجبت عليها بشكل مُرضي.	
2. أفهم أن مشاركتي طوعية وأنني لي الحرية في أن أقرر عدم المشاركة في أي جانب من جوانب هذه الدراسة في أي وقت، ومن دون إعطاء أي سبب.	
3. أدرك أن تنقلاتي سوف تسجل بواسطة نظام تحديد المواقع (جي بي إس) على مدار 24 ساعة, خلال فترة جمع البيانات.	
4. أؤكد أنني أرغب بالمشاركة في هذه الدراسة.	
5. أوافق على إستلام هدية عبارة عن قسيمة شرائية بمبلغ 100 ريال كعرفان وشكر لتطوعك بهذا البحث	

إسم المشترك:

رقم التليفون:

التاريخ:

اليوم ..... الشهر ..... السنة .....

العنوان:

التوقيع:

الايميل:

إسم الباحث:

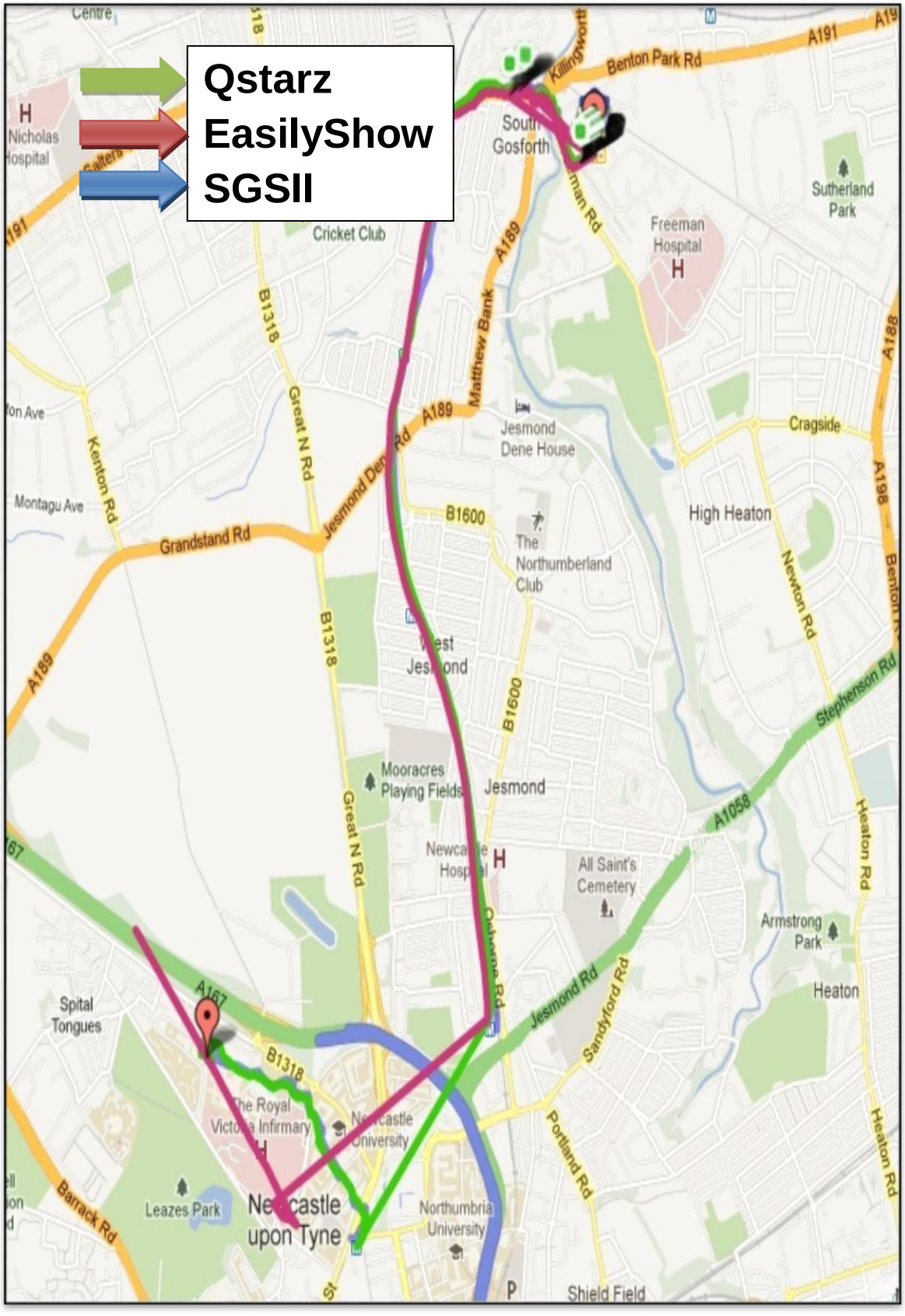
رقم الهاتف:

التاريخ:

اليوم ..... الشهر ..... السنة .....

التوقيع:

9.3 Appendix C, Comparing three GPS devices (EasilyShow, Qstarz and SGSII)



9.4 Appendix D, Ethical Approval Letter from FMS Ethics Committee at Newcastle University

Newcastle University

17 October 2011

Salem Al Balawi
PhD room
Ground Floor
Institute of Health and Society
Baddiley Clark Building

Faculty of Medical Sciences

Newcastle University
The Medical School
Framlington Place
Newcastle upon Tyne
NE2 4HH United Kingdom

Professor Michael Whitaker
FBiol FMed Sci

FACULTY OF MEDICAL SCIENCES: ETHICS COMMITTEE

Dear Salem

Title: Daily air pollution levels and asthma: exploring the influence of time-activity patterns on exposure
Application No: 00505/2011
Start date to end date: September 2011 to December 2013

On behalf of the Faculty of Medical Sciences Ethics Committee, I am writing to confirm that the ethical aspects of your proposal have been considered and your study has been given ethical approval.

The approval is limited to this project: **00505/2011**. If you wish for a further approval to extend this project, please submit a re-application to the FMS Ethics Committee and this will be considered.

During the course of your research project you may find it necessary to revise your protocol. Substantial changes in methodology, or changes that impact on the interface between the researcher and the participants must be considered by the FMS Ethics Committee, prior to implementation.*

At the close of your research project, please report any adverse events that have occurred and the actions that were taken to the FMS Ethics Committee.*

Best wishes,

Yours sincerely

M. Holbrough

Marjorie Holbrough
On behalf of Faculty Ethics Committee

cc.
Professor Michael Whitaker, Dean of Research & Innovation
Ms Lois Neal, Assistant Registrar (Research Strategy)

*Please refer to the latest guidance available on the internal Newcastle Biomedicine web-site.

tel: +44 (0)191 222 5264
fax: +44 (0)191 222 5164
e-mail: Michael.Whitaker@ncl.ac.uk
www.ncl.ac.uk

The Queen's Anniversary Prizes
2005

9.5 Appendix E, Approval Letter from Royal Commission in Al Jubail, Saudi Arabia

بسم الله الرحمن الرحيم

KINGDOM OF SAUDI ARABIA
Royal Commission for Jubail & Yanbu
Royal Commission in Jubail

المملكة العربية السعودية
الهيئة الملكية للجبيل وينبع
الهيئة الملكية بالجبيل

12th Jan. 2011

Dr. Susan Hogson
Institute of Health & Society
Baddiley-Clark Building
Richardson Road, NE2 4AX
Newcasle University, UK.

Dear Susan,

Mr. Salem M. Al-Balawi, request approval of a trip to a scientific study and research in Jubail Industrial City for the purpose of obtaining a doctoral degree from the UK.

Accordingly, we report to you our approval in doing the required scientific journey in Jubail Industrial City.

Regards,

Dr. Mosleh H. Al-Otaibi
CEO, Royal Commission in Jubail

P.O. BOX 10001 JUBAIL INDUSTRIAL CITY 31961
TEL.: (03) 341-3000 FAX.: (03) 341-9891
Website: WWW.RCJY.GOV.SA

ص.ب. ١٠٠٠١ مدينة الجبيل الصناعية ٣١٩٦١
هاتف: ٣٤١-٣٠٠٠ (٠٣) فاكس: ٣٤١-٩٨٩١ (٠٣)
الموقع على الانترنت: WWW.RCJY.GOV.SA

9.6 Appendix F, Insurance Indemnity Letter

ZURICH
MUNICIPAL

To Whom It May Concern

Our ref: NK/IND 11 July, 2011

Zurich Municipal Customer: University of Newcastle

This is to confirm that University of Newcastle have in force with this Company until the policy expiry on 31 July 2012 Insurance incorporating the following essential features:

Policy Number: NHE-08CA03-0013

Limit of Indemnity:

Public Liability:	£ 25,000,000	any one event
Products Liability:	£ 25,000,000	for all claims in the
Pollution:		aggregate during any one period of insurance
Employers' Liability:	£ 25,000,000	any one event inclusive of costs

Excess:

Public Liability/Products Liability/Pollution:	£ 2,500 any one event
Employers' Liability:	Nil any one claim

Indemnity to Principals:
Covers include a standard Indemnity to Principals Clause in respect of contractual obligations.

Full Policy:
The policy documents should be referred to for details of full cover.

Yours faithfully

Underwriting Services
Zurich Municipal
Farnborough

Zurich Municipal
Zurich House
2 Gladiator Way
Farnborough
Hampshire
GU14 6GB

Telephone 0870 2418050
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9.7 Appendix G, Results of time series analysis

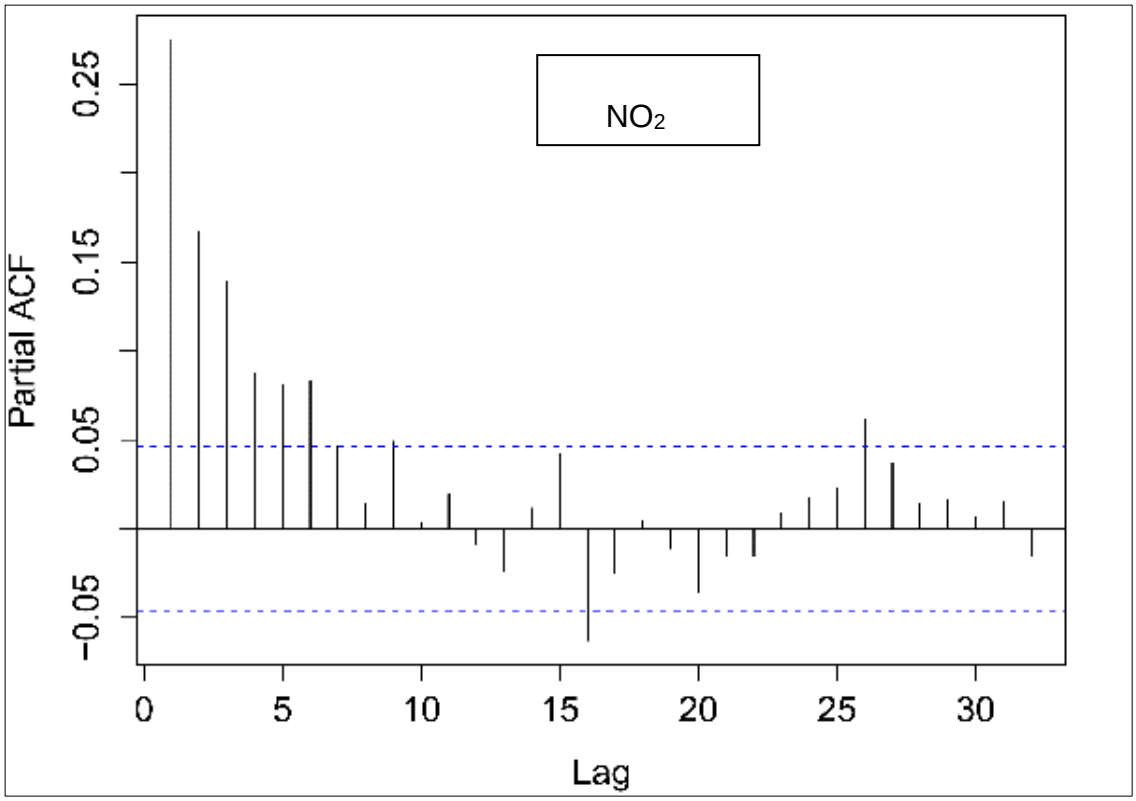


Figure 9.1: Partial ACF plot for NO_2 against lag days with no residuals included

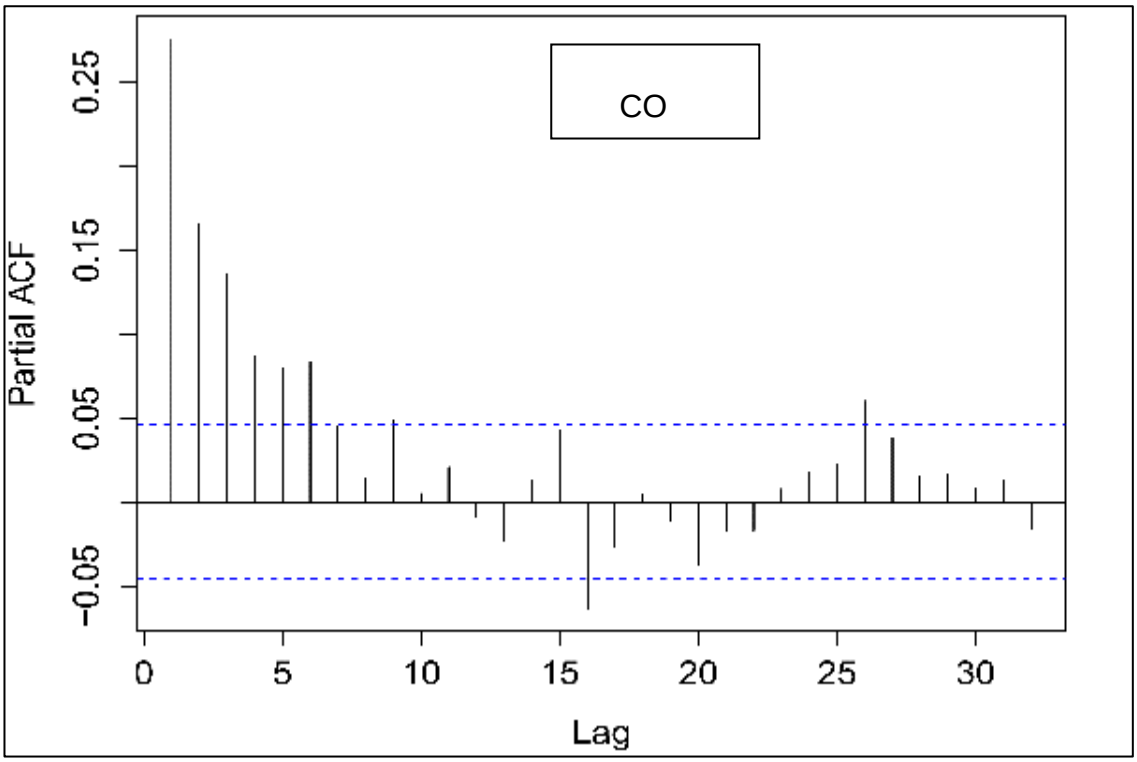


Figure 9.2: Partial ACF plot for CO against lag days with no residuals included

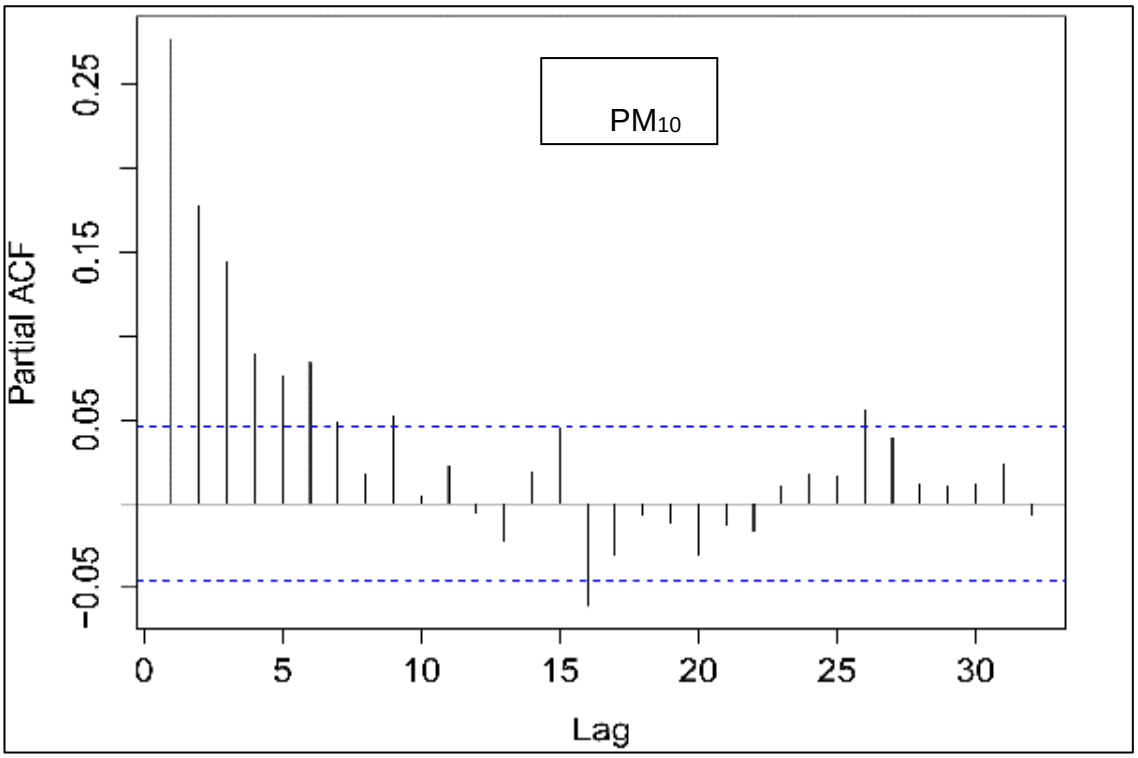


Figure 9.3: Partial ACF plot for PM_{10} against lag days with no residuals included

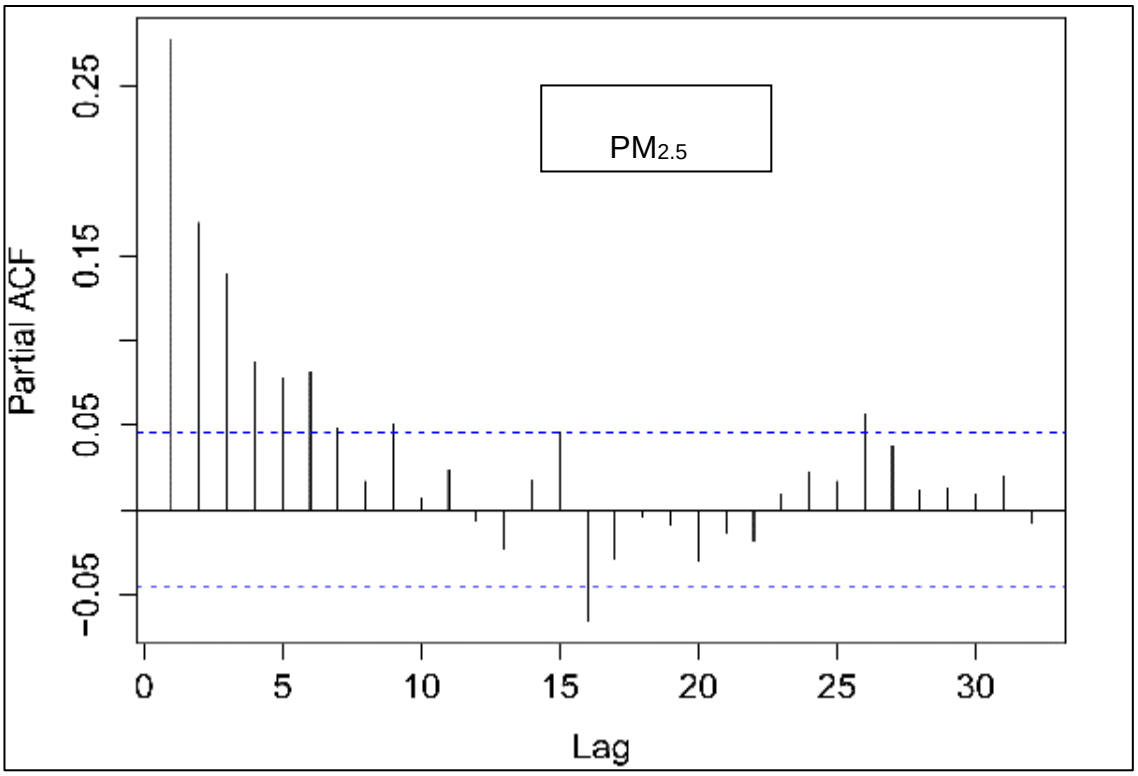


Figure 9.4: Partial ACF plot for $PM_{2.5}$ against lag days with no residuals included

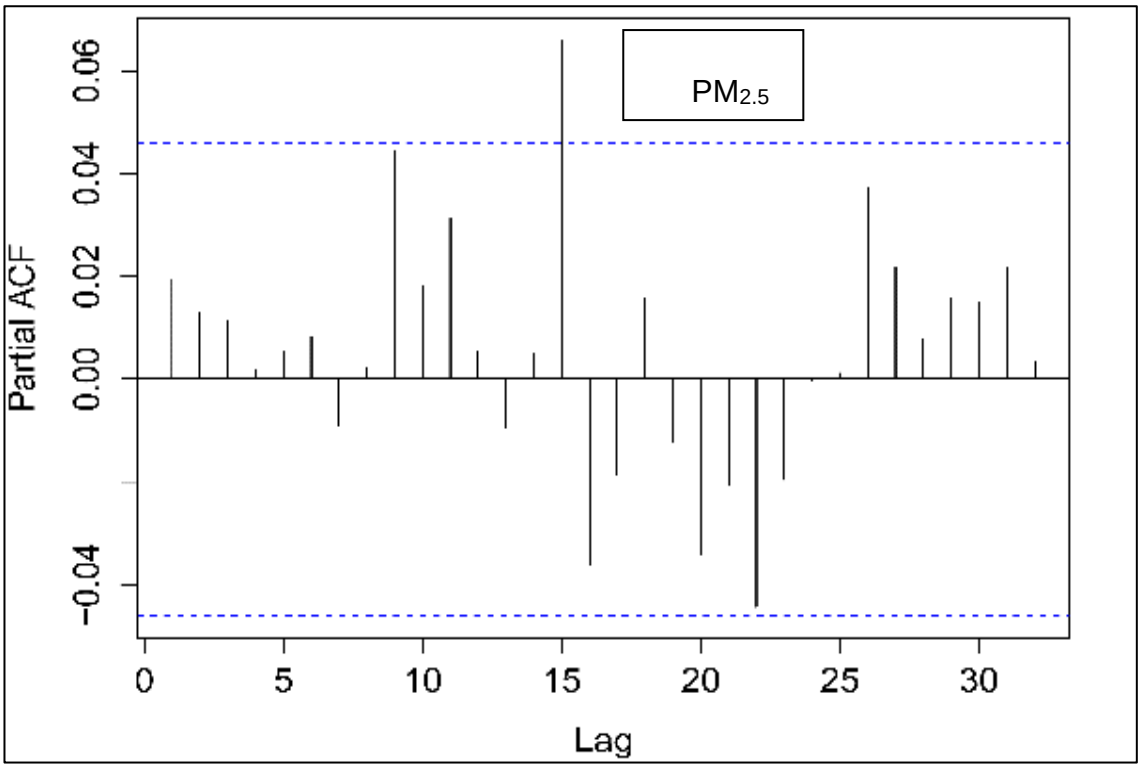


Figure 9.5: Partial ACF plot for $PM_{2.5}$ against lag days including residuals

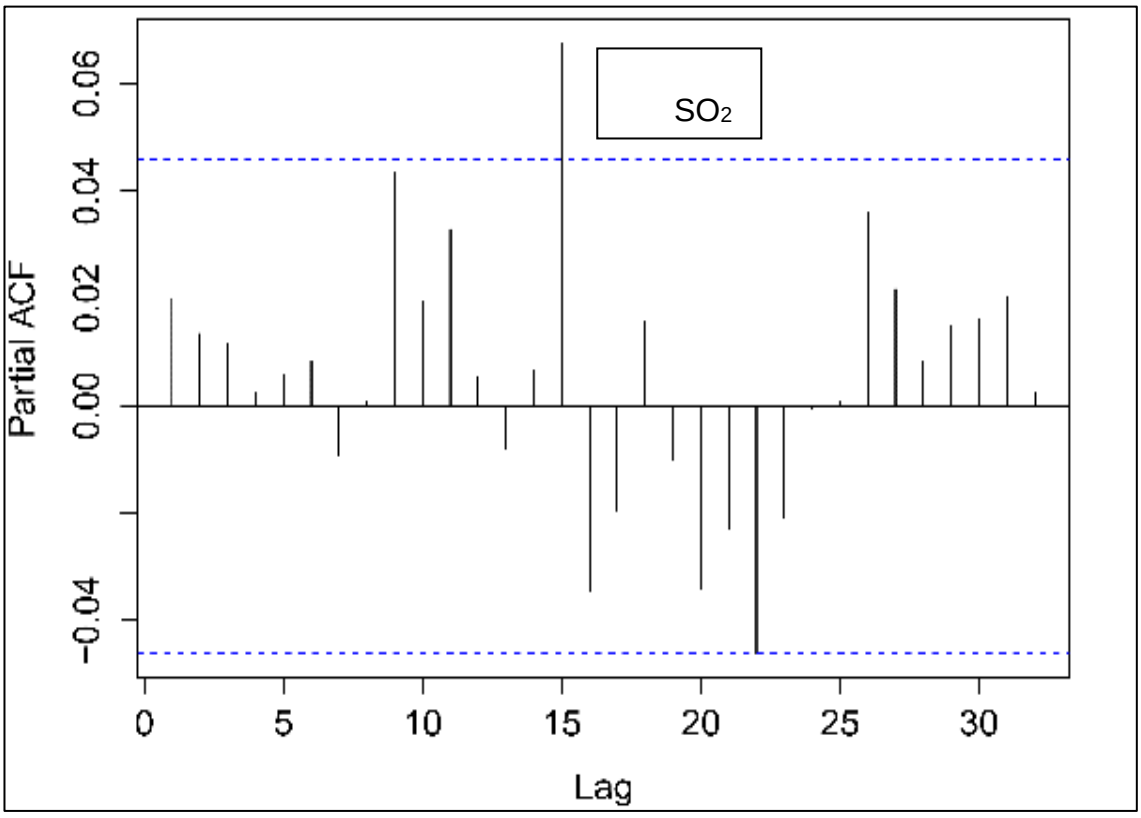


Figure 9.6: Partial ACF plot for SO_2 against lag days including residuals

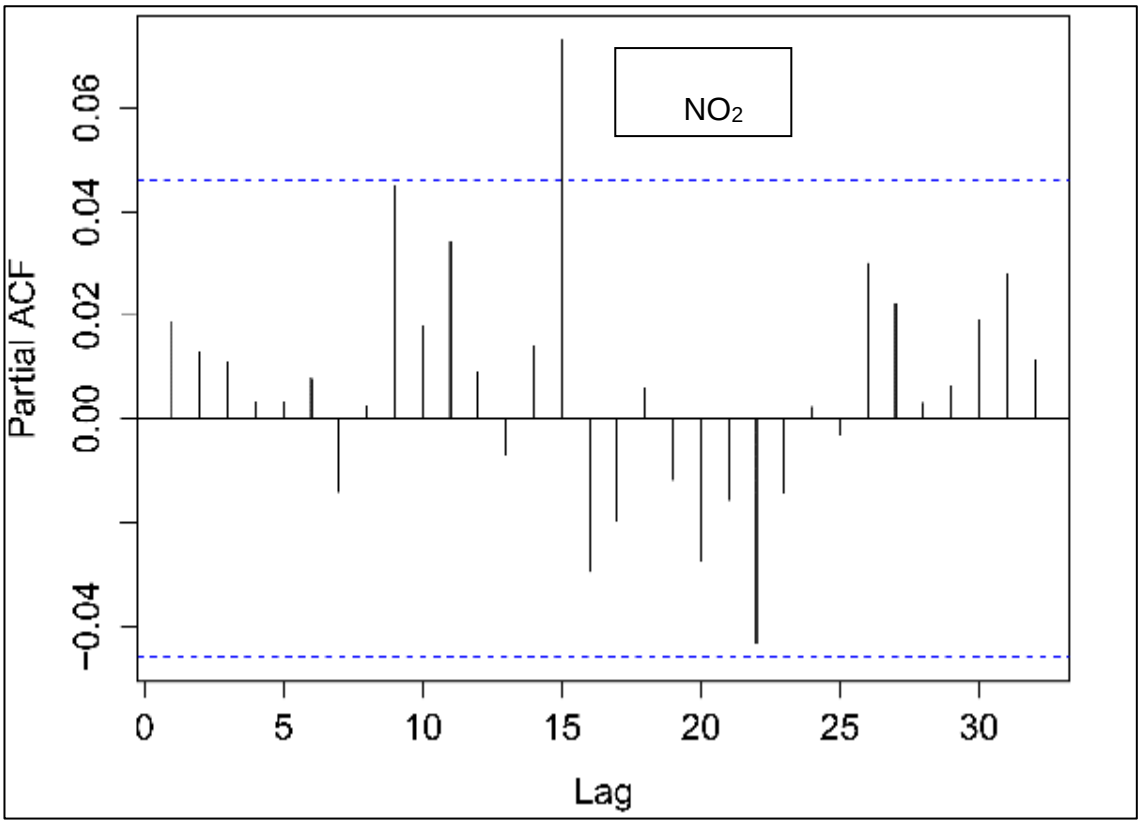


Figure 9.7: Partial ACF plot for NO_2 against lag days including residuals

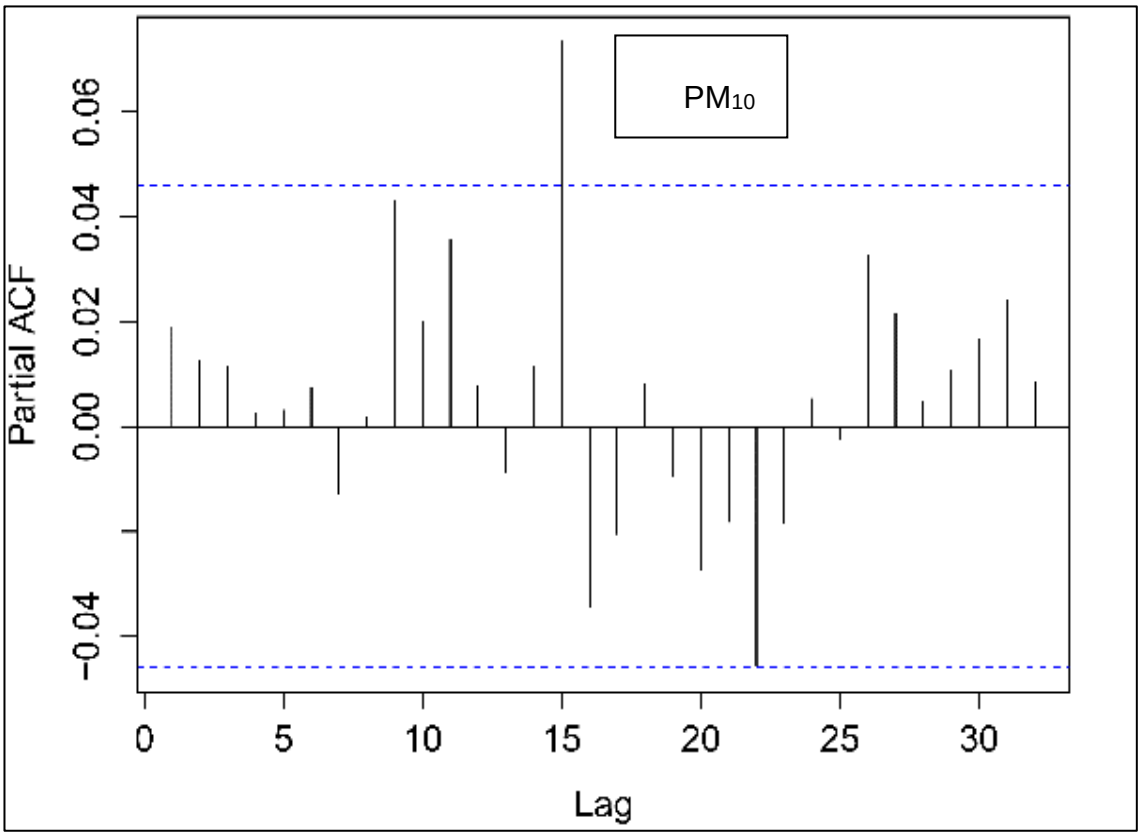


Figure 9.8: Partial ACF plot for PM_{10} against lag days including residuals

Table 9.1: Goodness of fit results for the analyses of $PM_{2.5}$ from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

$PM_{2.5}$	**without residuals**				**with residuals**				
Lag days	***R^2***	***X^2***	***df***	***X^2/df***	***Lag days***	***R^2***	***X^2***	***df***	***X^2/df***
0	0.44621	2943.73	1825	1.61	**0**	0.57038	2532.91	1818	1.39
1	0.49248	2783.71	1806	1.54	**1**	0.56386	2576.01	1818	1.42
2	0.51418	2707.43	1804	1.50	**2**	0.56171	2584.69	1818	1.42
3	0.52835	2666.17	1802	1.48	**3**	0.56231	2580.08	1818	1.42
4	0.53464	2648.29	1800	1.47	**4**	0.56179	2584.60	1818	1.42
5	0.54154	2625.15	1798	1.46	**5**	0.56187	2583.23	1818	1.42
6	0.54714	2606.72	1796	1.45	**6**	0.56179	2586.02	1818	1.42
7	0.55502	2573.55	1794	1.43	**7**	0.56471	2572.70	1818	1.42

Table 9.2: Goodness of fit results for the analyses of PM_{10} from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

PM_{10}	**without residuals**				**with residuals**				
Lag days	***R^2***	***X^2***	***df***	***X^2/df***	***Lag days***	***R^2***	***X^2***	***df***	***X^2/df***
0	0.44406	2959.59	1825	1.62	**0**	0.56616	2558.13	1818	1.41
1	0.49110	2795.27	1806	1.55	**1**	0.56236	2581.29	1818	1.42
2	0.51174	2727.05	1804	1.51	**2**	0.56131	2585.48	1818	1.42
3	0.52514	2690.42	1802	1.49	**3**	0.56136	2583.49	1818	1.42
4	0.53131	2673.47	1800	1.49	**4**	0.56133	2584.33	1818	1.42
5	0.53831	2651.61	1798	1.47	**5**	0.56137	2584.25	1818	1.42
6	0.54402	2632.80	1796	1.47	**6**	0.56131	2584.92	1818	1.42
7	0.55202	2598.29	1794	1.45	**7**	0.56303	2575.53	1818	1.42

Table 9.3: Goodness of fit results for the analyses of CO from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

CO	***without residuals***				***with residuals***				
Lag days	***R^2***	***X^2***	***df***	***X^2/df***	***Lag days***	***R^2***	***X^2***	***df***	***X^2/df***
0	0.43856	2988.66	1825	1.64	**0**	0.56256	2580.36	1818	1.42
1	0.48509	2824.49	1806	1.56	**1**	0.56261	2581.14	1818	1.42
2	0.50529	2760.59	1804	1.53	**2**	0.56149	2585.98	1818	1.42
3	0.51884	2725.47	1802	1.51	**3**	0.56123	2586.74	1818	1.42
4	0.52522	2708.80	1800	1.50	**4**	0.56147	2586.42	1818	1.42
5	0.53198	2688.76	1798	1.50	**5**	0.56119	2587.02	1818	1.42
6	0.53778	2669.58	1796	1.49	**6**	0.56128	2586.43	1818	1.42
7	0.54624	2630.50	1794	1.47	**7**	0.56141	2586.30	1818	1.42

Table 9.4: Goodness of fit results for the analyses of SO_2 from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

SO_2	***without residuals***				***with residuals***				
Lag days	***R^2***	***X^2***	***df***	***X^2/df***	***Lag days***	***R^2***	***X^2***	***df***	***X^2/df***
0	0.44211	2991.15	1825	1.64	**0**	0.56079	2592.93	1818	1.43
1	0.48535	2825.95	1806	1.56	**1**	0.56196	2586.92	1818	1.42
2	0.50551	2762.94	1804	1.53	**2**	0.56264	2588.41	1818	1.42
3	0.51881	2728.0	1802	1.51	**3**	0.56070	2590.10	1818	1.42
4	0.52508	2711.29	1800	1.51	**4**	0.56024	2590.52	1818	1.42
5	0.53181	2691.33	1798	1.50	**5**	0.56052	2590.42	1818	1.42
6	0.53757	2672.15	1796	1.49	**6**	0.56160	2585.46	1818	1.42
7	0.54597	2633.14	1794	1.47	**7**	0.56022	2588.18	1818	1.42

Table 9.5: Goodness of fit results for the analyses of NO_2 from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

NO_2	without residuals				with residuals				
Lag days	**R^2**	**X^2**	**df**	**X^2/df**	**Lag days**	**R^2**	**X^2**	**df**	**X^2/df**
0	0.43825	2991.28	1825	1.64	**0**	0.56105	2586.87	1818	1.42
1	0.48512	2824.98	1806	1.56	**1**	0.56240	2584.44	1818	1.42
2	0.50520	2760.59	1804	1.53	**2**	0.56138	2586.92	1818	1.42
3	0.51881	2723.59	1802	1.51	**3**	0.56281	2576.81	1818	1.42
4	0.52533	2705.46	1800	1.50	**4**	0.56133	2584.96	1818	1.42
5	0.53215	2684.41	1798	1.49	**5**	0.56123	2586.06	1818	1.42
6	0.53805	2664.28	1796	1.48	**6**	0.56114	2586.22	1818	1.42
7	0.54651	2625.40	1794	1.46	**7**	0.56119	2584.96	1818	1.42

Table 9.6: The analysis of the regression coefficients (β) for $PM_{2.5}$ from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

$PM_{2.5}$	**without residuals**				**with residuals**				
Lag days	**AIC**	**β**	**ε**	**t-value**	**Lag days**	**AIC**	**β**	**ε**	**t-value**
0	8779.6	8.72E-04	1.63E-04	5.3 *	**#0**	**8335.9**	**1.02E-03**	**1.62E-04**	**6.3 ***
1	8624.6	8.69E-04	1.63E-04	5.3 *	**1**	8363.3	5.16E-04	1.67E-04	3.1 *
2	8547.6	9.82E-04	1.63E-04	6	**2**	8372.2	-7.59E-05	1.82E-04	-0.4
3	8496.3	1.04E-03	1.63E-04	6.4	**3**	8369.7	-3.11E-04	1.92E-04	-1.6
4	8474.6	1.04E-03	1.63E-04	6.4	**4**	8371.9	-1.35E-04	1.91E-04	-0.7
5	8450.2	1.05E-03	1.62E-04	6.5	**5**	8371.6	1.73E-04	1.89E-04	0.9
6	8430.6	1.05E-03	1.62E-04	6.5	**6**	8371.9	-1.32E-04	1.88E-04	-0.7
7	8401.5	1.03E-03	1.62E-04	6.3	**7**	8359.7	-6.89E-04	1.99E-04	-3.5

*****Statistical Significance level – α (0.001)
 # = the better model

Table 9.7: The analysis of the regression coefficients (β) for PM_{10} from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

PM_{10}	**without residuals**				**with residuals**				
Lag days	**AIC**	**β**	**ε**	**t-value**	**Lag days**	**AIC**	**β**	**ε**	**t-value**
0	8786.6	1.63E-04	3.57E-05	4.6 *	**#0**	**8353.7**	**1.66E-04**	**3.52E-05**	**4.7 ***
1	8629.5	1.74E-04	3.62E-05	4.8 *	**1**	8369.5	8.13E-05	3.71E-05	2.2 *
2	8556.7	1.85E-04	3.60E-05	5.1	**2**	8373.9	1.99E-05	3.90E-05	0.5
3	8508.7	1.86E-04	3.57E-05	5.2	**3**	8373.7	-2.74E-05	4.06E-05	-0.7
4	8487.6	1.84E-04	3.56E-05	5.2	**4**	8373.8	-2.46E-05	4.13E-05	-0.6
5	8463.0	1.89E-04	3.56E-05	5.3	**5**	8373.6	3.00E-05	4.12E-05	0.7
6	8443.1	1.88E-04	3.54E-05	5.3	**6**	8373.9	2.13E-05	3.98E-05	0.5
7	8413.7	1.84E-04	3.56E-05	5.2	**7**	8366.7	-1.13E-04	4.24E-05	-2.7

*****Statistical Significance level – α (0.001)
 # = the better model

Table 9.8: The analysis of the regression coefficients (*β*) for CO from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B)

CO	without residuals				with residuals				
Lag days	**AIC**	**β**	**ε**	**t-value**	**Lag days**	**AIC**	**β**	**ε**	**t-value**
0	8804.6	-7.62E-02	6.35E-02	-1.2	**0**	8368.7	-1.52E-01	6.39E-02	-2.4
1	8651.0	-1.61E-02	5.45E-02	-0.3	**1**	8368.5	-1.56E-01	6.42E-02	-2.4
2	8580.6	-3.12E-02	5.45E-02	-0.6	**2**	8373.1	-7.21E-02	6.46E-02	-1.1
3	8532.7	-4.89E-02	5.46E-02	-0.9	**3**	8374.2	2.64E-02	6.43E-02	0.4
4	8511.1	-5.46E-02	5.46E-02	-1.0	**4**	8373.2	-6.97E-02	6.43E-02	-1.1
5	8487.8	-5.54E-02	5.47E-02	-1.0	**5**	8374.4	-3.18E-03	6.42E-02	0.0
6	8467.8	-5.64E-02	5.48E-02	-1.0	**6**	8374.0	-3.97E-02	6.41E-02	-0.6
7	8437.0	-6.07E-02	5.48E-02	-1.1	**7**	8373.5	-6.04E-02	6.37E-02	-0.9

Table 9.9: The analysis of the regression coefficients (*β*) for SO_2 from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B).

SO_2	without residuals				with residuals				
Lag days	**AIC**	**β**	**ε**	**t-value**	**Lag days**	**AIC**	**β**	**ε**	**t-value**
0	8793.0	2.73E-02	7.46E-03	3.7 *	**0**	8376.0	1.97E-02	7.54E-03	2.6 *
1	8651.0	-1.61E-02	5.45E-02	-0.3	**1**	8371.2	2.53E-02	7.35E-03	3.4 *
2	8580.6	-3.12E-02	5.45E-02	-0.6	**#2**	**8368.4**	**2.82E-02**	**7.33E-03**	**3.8** *
3	8532.7	-4.89E-02	5.46E-02	-0.9	**3**	8376.4	1.92E-02	7.56E-03	2.5 *
4	8511.1	-5.46E-02	5.46E-02	-1.0	**4**	8378.3	1.61E-02	7.61E-03	2.1
5	8487.8	-5.54E-02	5.47E-02	-1.0	**5**	8377.1	1.78E-02	7.47E-03	2.4
6	8467.8	-5.64E-02	5.48E-02	-1.0	**6**	8372.7	2.34E-02	7.30E-03	3.2
7	8437.0	-6.07E-02	5.48E-02	-1.1	**7**	8378.4	1.56E-02	7.44E-03	2.1

*****Statistical Significance level – α (0.001)
 # = the better model

Table 9.10: The analysis of the regression coefficients (β) for NO_2 from no lag to seven lag days for models with no residual inclusion (A) and including residuals in the model (B).

NO_2	without residuals				with residuals				
Lag days	**AIC**	**β**	**ε**	**t-value**	**Lag days**	**AIC**	**β**	**ε**	**t-value**
0	8805.6	1.17E-03	1.78E-03	0.7	**0**	8375.0	1.72E-04	1.81E-03	0.1
1	8650.9	4.96E-04	1.09E-03	0.5	**1**	8369.3	4.11E-03	1.73E-03	2.4 *
2	8581.0	-9.00E-05	1.09E-03	-0.1	**2**	8373.6	2.04E-03	1.73E-03	1.2
3	8532.8	-8.99E-04	1.10E-03	-0.8	**#3**	**8367.7**	**4.68E-03**	**1.73E-03**	**2.7 ***
4	8510.7	-1.31E-03	1.10E-03	-1.2	**4**	8373.8	1.86E-03	1.74E-03	1.1
5	8487.1	-1.44E-03	1.10E-03	-1.3	**5**	8374.2	1.53E-03	1.75E-03	0.9
6	8466.7	-1.62E-03	1.11E-03	-1.5	**6**	8374.6	1.08E-03	1.75E-03	0.6
7	8435.9	-1.69E-03	1.11E-03	-1.5	**7**	8374.4	1.37E-03	1.77E-03	0.8

*****Statistical Significance level – α (0.001)
 # = the better model

9.8 Appendix H: Fieldwork Procedure

Part One: Prepare SidePak

Step One: Charging the monitor

Make sure you have an adaptor for your cord

Charge the Battery Pack, 2700 mAH for at least 5.5 hours prior to taking measurements for an extended period or by using Six AA-size Battery cells in the Battery Pack.

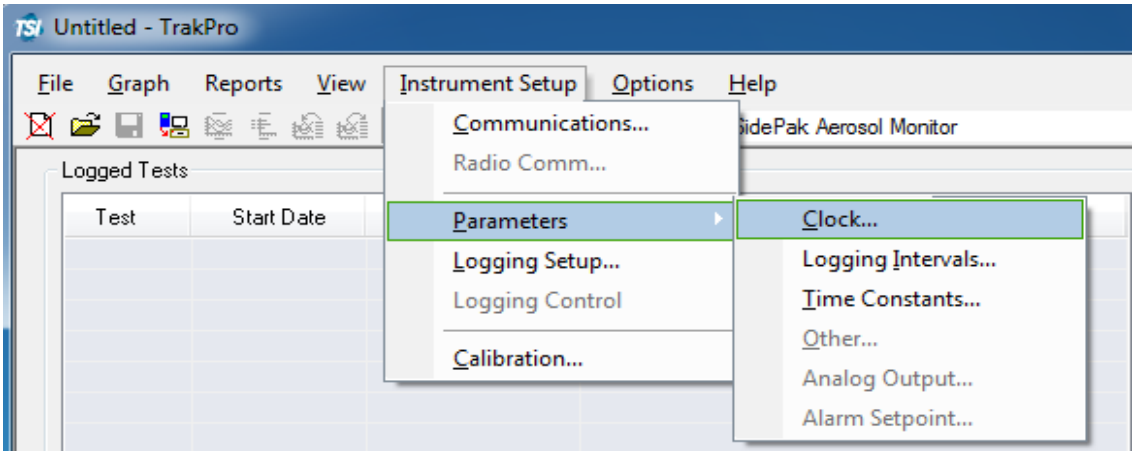
Typically Battery Life

Battery Pack	Cell Type	Hours @ 0.7 lpm	Hours @ 1.7 lpm
1650 mAH NiMH Pack (P/N 801724)	NiMH (4.8 V, 1650 mAH)	11.5	9.2
2700 mAH NiMH Pack (P/N 801722)	NiMH (4.8 V, 2700 mAH)	19.8	15.6
6-Cell AA-size Pack (P/N 801708, with six user-supplied AA cells)	AA Alkaline Cells	29.6	22.5

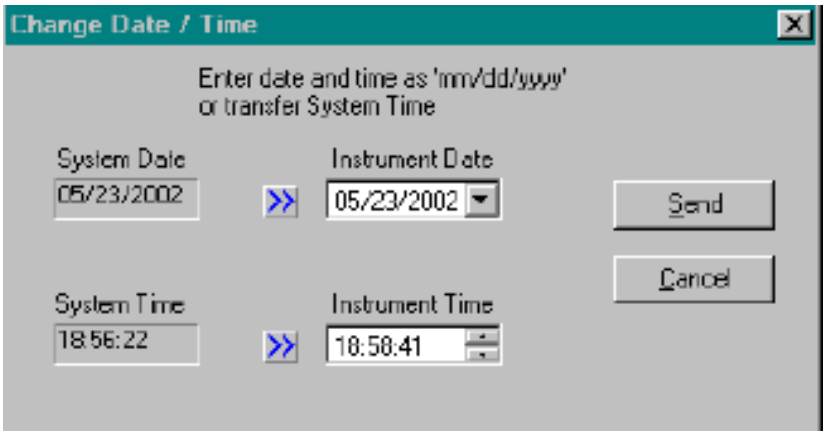
Step Two: Setting the Time & Date

Using TrakPro software:

1. The date and time are read from the instrument along with the current system time as read from the computer. The instrument date/time is displayed using Windows Date/Time Picker controls.
2. The current system time as read from the PC may also be transferred directly into the date/time controls. Go to Instrument Setup, then Parameters and Select Clock.

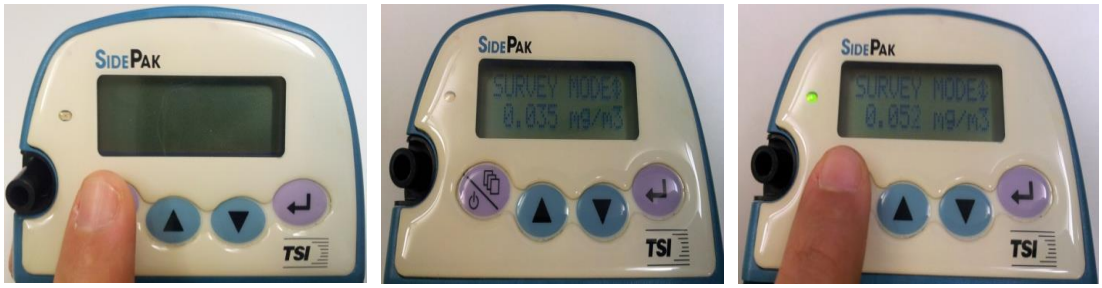


3. By using the “>>” buttons. Pressing the Send button sends the new clock data to the currently connected instrument.



Manually setting time and date using SidePak device:

1. Press PAGE key to turn monitor on. Then, wait for about 1 minute for pump to start up and readings to stabilize. Display will read “SURVEY MODE”. Then press PAGE key.



2. At “Data Log” Press the down arrow once - it should display “setup” Press ENTER key. . Then press down arrows until “Time/Date” is displayed, then press ENTER key.

3. At “Time” Press ENTER key . Setup the Time then press again ENTER key

4. Press the down arrow once – it should say “Date” then press ENTER key . Setup the date then press ENTERS key Press the page key to return to “survey mode”.


SIDEPAK
TIME/DATE
Date
SIDEPAK
DATE
/01/14
SIDEPAK
SURVEY MODE
0.035 mg/m3
TSI

Step Three: Cleaning and greasing the impactor

The SidePak is fitted with a 2.5 µm impactor that requires cleaning & greasing prior to use.

Remove the inlet assembly:

1. Use the supplied screwdriver to loosen screws that attach the inlet assembly.


2. Hold the inlet assembly and gently wiggle it until it dislodges from the SidePak unit.


3. Next, remove the impactor from the inlet assembly.

The impactor is located inside the inlet assembly.

Remove the impactor from the inlet assembly by gently pushing it out with the screwdriver.



4. Once the impactor is removed, clean and greases the impactor:

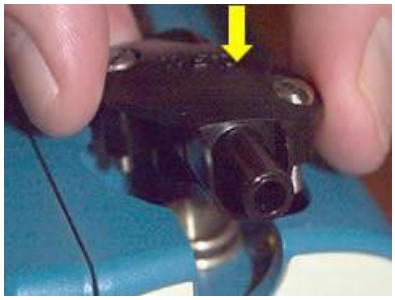
A. Wipe off the end of the impactor with a tissue paper or towel. Using the High Vacuum Grease supplied with your kit, smear a *very small* amount of the supplied grease on the end (tip) of the impactor.

B. Use your finger to remove excess grease, leaving only a thin layer of grease on the tip of the impactor. Leave just a thin layer of grease on the impactor.

5. Next, reassemble the impactor:

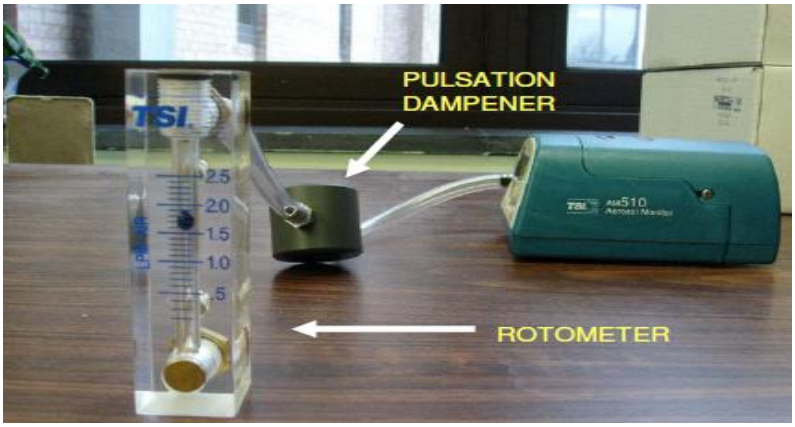
A. Carefully slide the cleaned and greased impactor back into the inlet assembly. Gently push the impactor in with your finger until it is fully seated.

B. Reattach the inlet assembly to the SidePak with the two previously loosened screws.



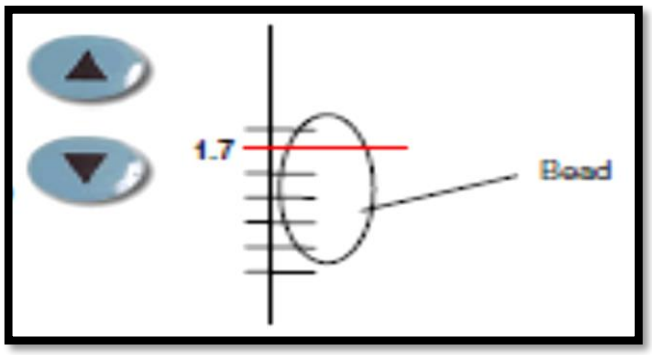
Step Four: Calibrate Flow Rate

1. Attach the tube on the Rotometer (the clear plastic piece) to the dampener (the black cylinder shaped piece)
2. Attach the tube on the dampener to the machine
3. After the tubes are connected to the machine, turn on the machine and wait until it says “Survey Mode” 



4. Press the on/off button again. It should display “main menu” and “data log”. 
5. Press the down arrow once – it should say “setup” 
6. Hit the enter key to arrive at the “setup menu” 
7. Press the up arrow once  – it should say “adjust flow” and hit the enter key again 
8. When it displays “adjust flow,” adjust the airflow with the up and down arrows on the machine. As you press the up or down arrows, the bead in the rotometer will rise or fall.

9. Keep pressing the arrows until the middle of the top half of the bead aligns with the 1.7 line (see diagram at right).



10. Once the bead is in the proper place, hit the “enter” key again. 

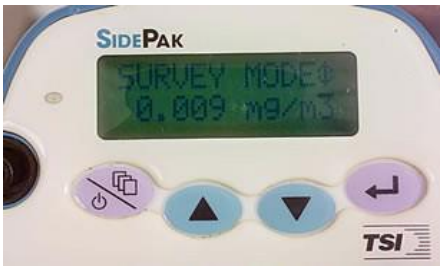
Step Five: Set Log Interval

1. From the setup menu, hit the down arrow twice until it displays “log interval” 
2. Press the enter key  . Use the arrows until you arrive at the1-minute Log interval. When it display one minute, press the enter key. 

Step Six: Zero-Calibrate the SidePak

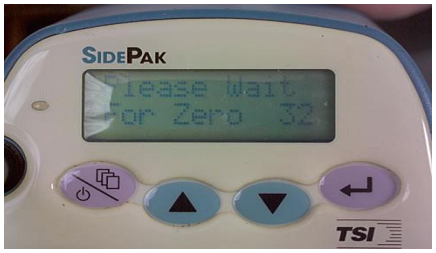
To zero-calibrate your unit:

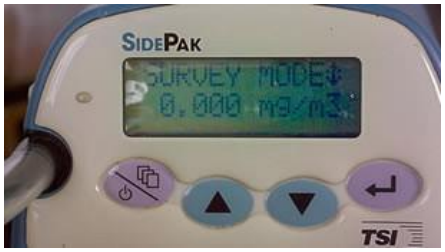
1. Press PAGE key to turn monitor on. . Wait about 1 minute for pump to start up and readings to stabilize. Display will read “SURVEY MODE”.



2. Press PAGE key . Press down arrow until “Zero Cal” is displayed. . Then Press ENTER . Attach Zero Filter. With the Zero Filter attached, Press ENTER again.

3. Wait until zero calibration I s finished, this will take 60 seconds. After the countdown is complete, you may need to press the page key one or more times to return to survey mode.





4. With the Zero Filter still attached, verify that the reading stays near 0.000 mg/m 3 or below 0.003 mg/m^3. If the reading is not near 0.00 mg/m^3, repeat the zero calibration until it is.

5. Remove the Zero Filter.

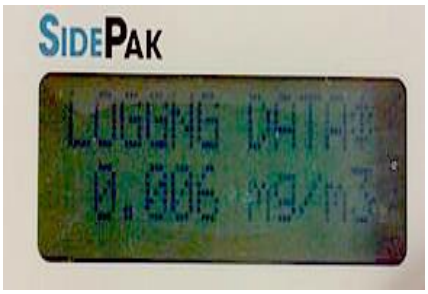
Step Seven: Operating the side pack

Setting up the SidePak to log (record into memory) measurements

1. After zero calibrating the machine, the screen should display survey mode

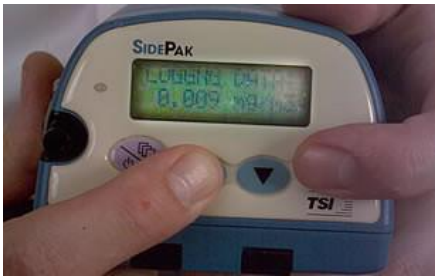
1. Press PAGE key.  At “Data Log” press the ENTER key.  . Press down arrows until “Run Manual” is displayed. 

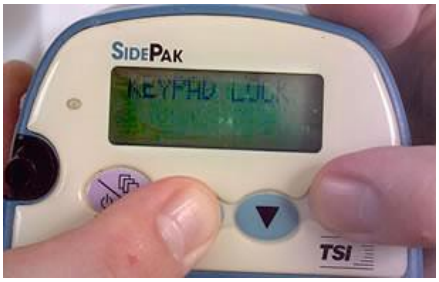
2. Press ENTER key. 
3. The screen should now display “Logging Data”. It is now recording measurements to memory.



2. Lock the SidePak

Press and hold the UP ARROW key and then press ENTER while holding the UP ARROW key.  . This will lock the device and prevent accidentally pressing any of the buttons during logging.



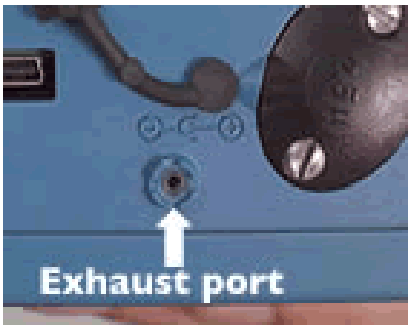


4. Attach tubing

Attach the clear plastic tubing to the inlet. If the tubing is loose on the inlet, you may need to cut no more than 2 cm from the end of the tubing for a tighter seal.

5. Place in a Backpack

A. Place SidePak in a backpack with the end of the plastic tubing protruding from backpack. Make sure that the exhaust port, the small outlet hole on the left side of the SidePak, is unobstructed.



B. Make sure that the small outlet hole on the side of the machine is also unobstructed and near the breath zone.

Part Two: Prepare Qstarz GPS

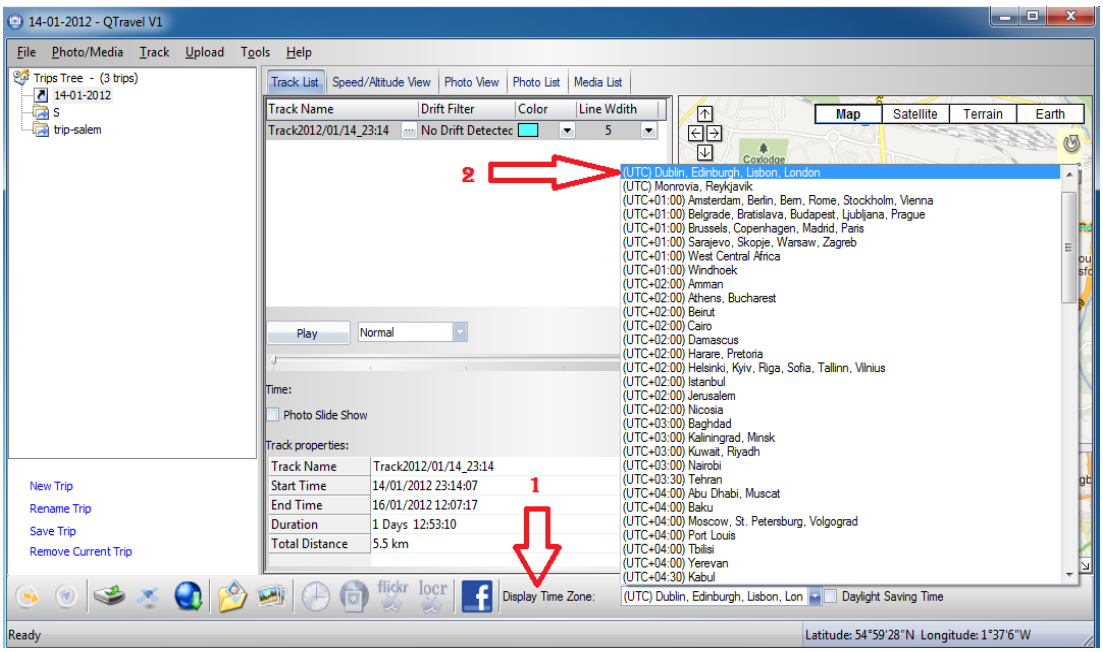
1. Charging battery

Take the power cable and connect it to the power jack (mini USB type) and recharge through USB cable. Charging time is 3 hours typically.

2. Setting Time & Date

The Qstarz time is sync with satellite and it uses the GMT/UTC time.

If you want to select your local time zone you need to use Qtravel software to sync with your local time. To do this, open Qtravel, then click Display Time Zone > Select, your local time zone as shown below.



3. Start recording and save POI locations

When switched to “LOG” mode, Qstarz can be used to record participants’ movements. Push the POI button (Red button) to log favourite Point of Interest.

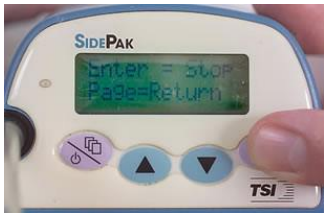
4. Place in a backpack

Part Three: After completing 24 hours

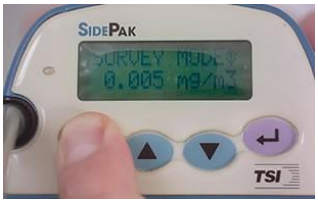
Step One: SidePak Aerosol monitor

1. Remove the SidePak from the backpack.
2. Unlock the device: Press and hold the UP ARROW key (these are the same steps used to lock the device) and press “Enter” key together.  

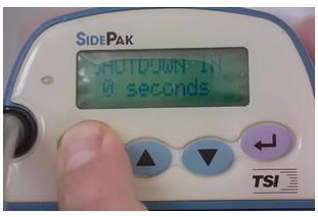
3. Press the “ENTER” key 2 times to stop data logging.  



4. Press the “PAGE” repeatedly until the device returns to SURVEY MODE. 



5. To turn off the SidePak, hold down the “PAGE” key for 3 seconds to shut down.  Shutting off the SidePak will not delete the data that you have collected. Your data remains in the memory until the user has intentionally deleted it.



Step Two: Qstarz GPS Logger

1. Remove the Qstarz from the backpack.
2. To turn OFF the device, as shown below, to stop recording data.

Part Four: Preparing your data for Analysis

Step One: SidePak Aerosol monitor

A. Install TRAKPRO software version 3.40 or later in your PC. A CD of the software is available with SidePak kit.
B. Connect SidePak to PC:
   Press PAGE key to turn the SidePak on.

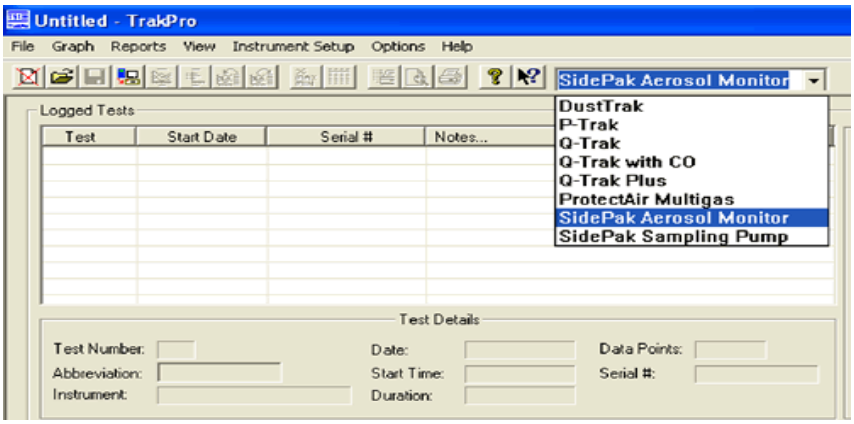
C. Take the larger end of the USB cable and insert it into an available USB port on the PC. Take the small (mini USB) end of the USB cable and insert it into the USB port on the side of the SidePak.

D. Your system is now connected

E. Transfer Data to PC:

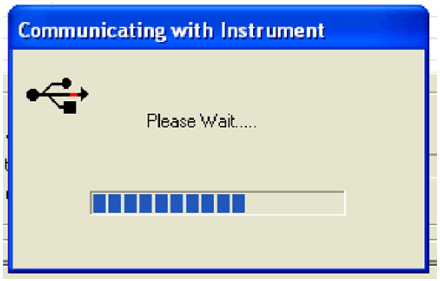
1. Open TrakPro software 

2. Make sure “SidePak Aerosol Monitor” is selected from the drop-down menu.



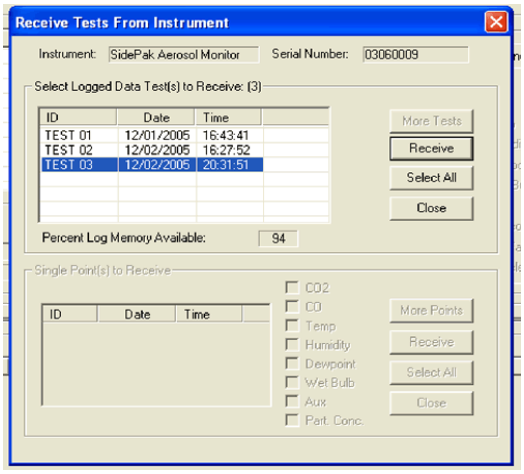
3. Select the “Receive Data” Icon

4. Once you select Receive Data, the PC will communicate with the SidePak. A dialog box will appear “Communicating with Instrument”.

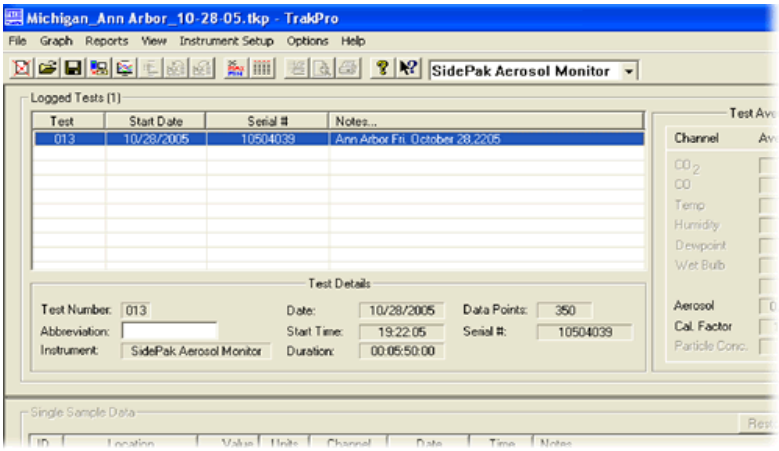


5. If receive an error message, check your cable connections and check that the SidePak is turned on. We recommend that you consult the User Manual for support, make recommended changes, and then try to Receive Data again.
6. Select the test or tests that you wish to download and press Receive.

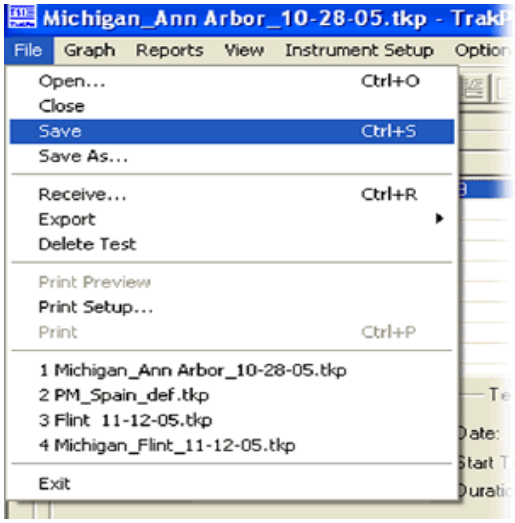
7. If there are a large number of tests (approximately 6 or more), Select “More Tests” one or more times to see and download all of the necessary tests.



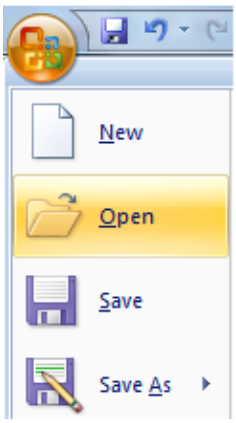
8. The test or tests that you have downloaded will appear in the Logged Tests window.



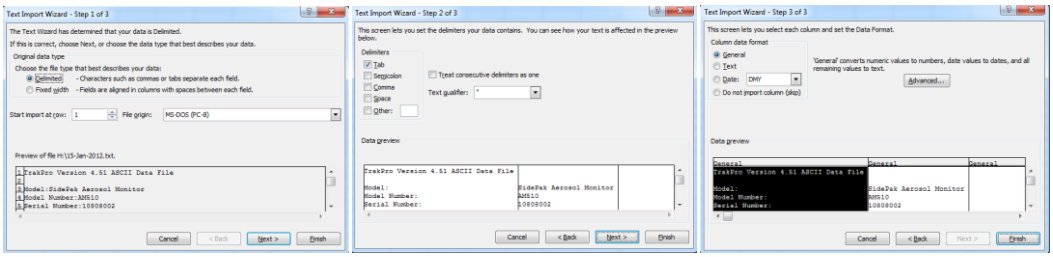
9. Save new data file. The file will be saved as a TrakPro file with a (.tkp) extension.



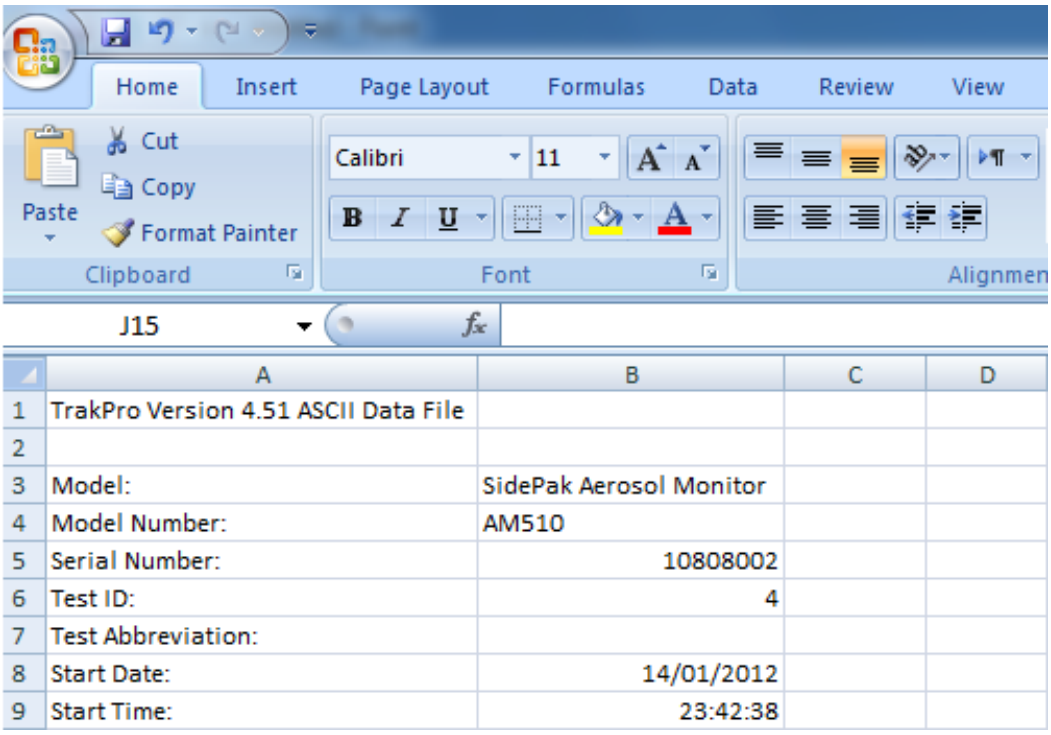
10. Open Excel, from the menu select open file



11. Select the TrakPro file (.tkp) and click open
12. Will open dialog box click next and next again



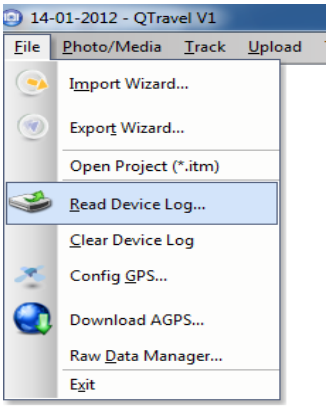
13. Now you complete transfer the data on Excel sheet and ready for analysis.



	A	B	C	D
1	TrakPro Version 4.51 ASCII Data File			
2				
3	Model:	SidePak Aerosol Monitor		
4	Model Number:	AM510		
5	Serial Number:	10808002		
6	Test ID:	4		
7	Test Abbreviation:			
8	Start Date:	14/01/2012		
9	Start Time:	23:42:38		

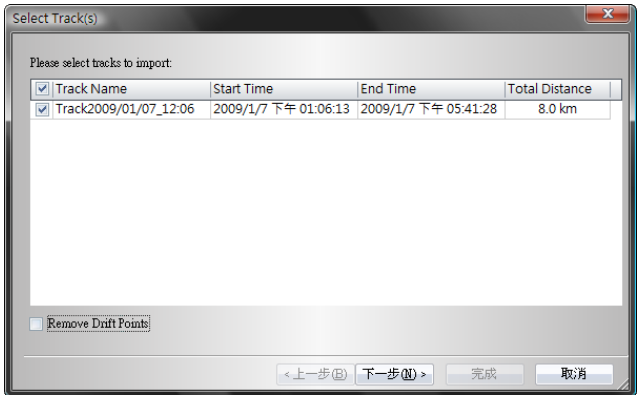
14. Step Two: GPS Logger
15. Power on Qstarz and connect it to your PC through a mini USB cable.

16. Start Qtravel software on your PC.
17. On the menu select File and from the list choice, Read Device Log

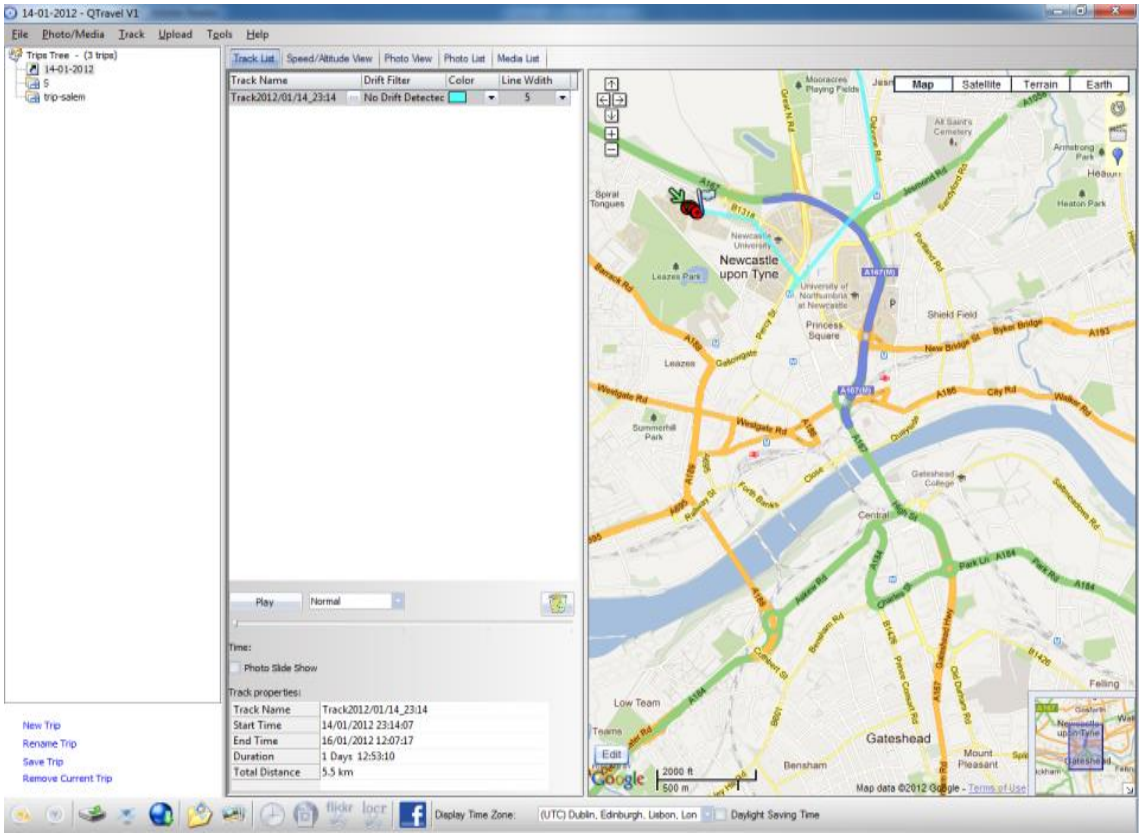


18. Your PC will start read the data from Qstarz

19. Once done reading the data, a track list window will pop up. Select the tracks to import and click finish.



20. Tracks will be shown on the Google Map window.



CHAPTER TEN

References

Chapter:10 References

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