School of Public Health

Air Pollution in Metropolitan Perth and Cardiovascular and Respiratory Hospital Admissions

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Declaration

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgment has been made.

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

Human Ethics

The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research (2007) – updated March 2014. The proposed research study received human research ethics approval from the Curtin University Human Research Ethics Committee (EC00262), Approval Number #.HR51/2011.

Signature: .....

Date: 1.12.17

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Abstract

This two-stage study analysed historical and recent air quality data and health outcomes in metropolitan Perth. The first stage examined historical ambient air quality (for carbon monoxide, nitrogen dioxide, ozone, coarse particulate matter [PM_{10}] and fine particulate matter [$PM_{2.5}$]) and hospital admission data from 1 January 2001 to the 31 December 2010 to determine whether levels of criteria air pollutants in the Perth metropolitan airshed contributed to the mean number of unscheduled hospital admissions for cardiovascular and respiratory illness. The second stage consisted of monitoring ambient air quality, specifically particulate matter (PM), and collecting samples over a six week period in October / November 2013 and August / September 2014, to determine the size-specific metal composition of the PM.

Generally, Perth air quality is regarded as good with levels of the criteria pollutants only occasionally exceeding the recommended levels stipulated in the Ambient Air Quality National Environmental Protection Measure (AAQ NEPM). There is however a mounting body of knowledge that many of the health effects that result from exposure to air pollution occur when people are exposed to levels below the AAQ NEPM. To examine the effect of low level exposure, mean monitored levels of each pollutant were divided into thirds or tertiles which represented low, medium and high levels of exposure. Logistic regression was performed on all possible combinations of tertile, illness condition, magnitude of any hospitalisation increase (10% or 20%) and whether the number of admissions had been adjusted for seasons. Significant relationships were found between levels of carbon monoxide, nitrogen dioxide and ozone in the second and third tertiles and increases in hospital admissions for various conditions and various combinations of covariates. These findings are consistent with previous local and international studies. Where this study is not consistent is that there were no significant relationships between PM_{10} and hospitalisations for any illness and $PM_{2.5}$ was only found to have a significant relationship with asthma admissions. While this particular study has fewer significant relationships than many other similar studies it is consistent with results from a previous study conducted in the Perth metropolitan area approximately ten years previously.

In the second stage of the study air monitoring was carried out at twelve sites spread over two distinct areas of inner metropolitan Perth. The first area or cluster of sites was zoned industrial and was based around Welshpool and the second was zoned residential/urban (non-industrial) and was based around the Curtin University campus in Bentley. On each occasion ambient air was sampled for five to seven days using a

High Volume sampler to measure Total Suspended Particles (TSP), and also a Nano-MOUDI II™ 125B Multistage Low Pressure Cascade Impactor to measure mass and concentration of PM ranging in size from 10 nm to 10,000 nm (10 µm). Samples collected in the Nano-MOUDI were also analysed by using Inductively Coupled Plasma – Mass Spectrometry (ICP-MS) to characterise metalliferous composition.

The mass concentration of metals found in ambient air in the Perth metropolitan area was generally low, with all metals considered to be hazardous to human health present in concentrations well below any World Health Organisation, United States Environmental Protection Authority, European Commission or Australian Ambient Air Quality guideline or standard values. The mean mass concentration of metals measured in the non-industrial areas was substantially lower than the mean mass concentration for most metals measured at sites in the industrial area. Short-term exposure to airborne metals in either area is not likely to cause any health effects, however further studies would need to be conducted to determine the effect of long-term exposure, especially in the Welshpool industrial area.

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Abbreviations and acronyms

AAQ	Ambient Air Quality
AAQS	Australian air quality standard
AED	Aerodynamic equivalent diameter
AQG	Air Quality Guidelines
BC	Black carbon
BS	Black smoke
B_{sp}	Backscatter co-efficient of light scattering due to particles
CDNP	Combustion derived nano particles
CI	Confidence interval
CO	Carbon monoxide
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
CP	Coarse particles (AED less than 10.0 µm)
CVD	Cardiovascular disease
DALYs	Disability adjusted life-years
DE	Department of Environment, WA
DEC	Department of Environment and Conservation, WA (2006 - 2013)
DEP	Diesel exhaust particles
DEP	Department of Environmental Protection
DER	Department of Environment Regulation, WA (2013 -2017)
DPM	Diesel particulate matter
DWER	Department of Water and Environmental Regulation, WA (2017)
ENP	Engineered nano-particle
EPHC	Environment Protection and Heritage Council
EC	Elemental carbon
EPA	Environment Protection Authority, WA
FEV1	Forced expiratory volume during one second
FP	Fine particles (AED less than 2.5 µm)
HVS	High volume (Hi-Vol) sampler
IARC	International Agency for Research on Cancer
IHD	Ischemic heart disease
ICP	Inductively coupled plasma
ICP- AES	ICP- Atomic emission spectroscopy
ICP- MS	ICP- Mass spectrometry
IQR	Interquartile range

ISO	International Organisation for Standardisation
MOUDI	Micro orifice uniform deposit impactor
NAAQS	National Ambient Air Quality Standard (U.S.A.)
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
N_2O	Nitrous oxide
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_x	Oxides of nitrogen (includes NO, N_2O and NO_2)
NP	Nanoparticle
NPI	National Pollutant Inventory
NSP	Nano-size particle
O_3	Ozone
OR	Odds-ratio
PM	Particulate matter
$PM_{0.1}$	PM with an AED less than 0.1 µm
$PM_{2.5}$	PM with an AED less than 2.5 µm
$PM_{10-2.5}$	PM with an AED less than 10 µm and greater than 2.5 µm
PM_{10}	PM with an AED less than 10 µm
ROS	Reactive oxygen species
SD	Standard deviation
SO_2	Sulphur dioxide
SO_x	Sulphur oxides
TEOM	Tapered element oscillating microbalance
TRAP	Traffic related air pollution
TPS	Town Planning Scheme
TSP	Total suspended particles with an AED less than 100 µm
UFP	Ultrafine particle (AED<0.1µm (100 nm)
U.S. EPA	United States Environmental Protection Agency
VOC	Volatile organic compound
WHO	World Health Organisation

Units of Concentration

mg/m^3	milligrams per cubic meter ($g \times 10^{-3}$)
$\mu g/m^3$	micrograms per cubic meter ($g \times 10^{-6}$)
ng/m^3	nanograms per cubic meter ($g \times 10^{-9}$)
pg/m^3	picograms per cubic meter ($g \times 10^{-12}$)
ppm	parts per million (1,000,000)
pphm	parts per hundred million (100,000,000)
ppb	parts per billion (1,000,000,000)
ppt	parts per trillion (1,000,000,000,000)

Metals (Chemical Symbols)

Ag	Silver	Na	Sodium
As	Arsenic	Ni	Nickel
Ba	Barium	P	Phosphorus
Be	Beryllium	Pb	Lead
Bi	Bismuth	Pd	Palladium
Ca	Calcium	Pr	Praseodymium
Cd	Cadmium	Rb	Rubidium
Ce	Cerium	S	Sulphur
Co	Cobalt	Sc	Scandium
Cr	Chromium	Sm	Samarium
Cs	Caesium	Sr	Strontium
Cu	Copper	Sb	Antimony
Eu	Europium	Th	Thorium
Fe	Iron	Ti	Titanium
Ho	Holmium	Tl	Thallium
K	Potassium	U	Uranium
La	Lanthanum	V	Vanadium
Mg	Magnesium	Y	Yttrium
Mn	Manganese	Zn	Zinc
Mo	Molybdenum	Zr	Zirconium

CHAPTER 1 Introduction

This chapter outlines the background to the study, and also explains the structure and rationale for presenting the study in stages. The aim and objectives are presented and the significance of the study to the field of air pollution research discussed.

1.1 Background of the study

Metropolitan Perth has what is considered to be good to very good air quality with monitored air pollutant levels rarely exceeding the relevant Ambient Air Quality Standards (Department of Environment Regulation [DER] Air Quality Coordinating Committee [AQCC] (WA) 2014, 2017). There are occasional major pollution episodes and these are generally related to combustion; bushfires over summer, and prescribed burning and domestic wood heaters during winter. Previous investigations into levels of pollutants and health effects in Perth conducted in the mid-nineties found little cause for alarm. A comprehensive study by Hinwood et al. (2006) reported only a small number of links between levels of selected air pollutants and cardiovascular and respiratory hospital admissions during the period 1992-1998. In the following decade Western Australia overall and in particular metropolitan Perth, underwent rapid population growth and development with the population increasing from 1.23 million in 1992 to 1.70 million in 2010.

This rapid growth was attributed mainly to increased activity in the resource sector. The Department of Planning (WA Government, 2010) reported that at its peak, 'the resources boom' was attracting approximately 50,000 workers and their families per year from both interstate and overseas and the population of the Perth/Peel region was expected to double from 1.7 million to approximately 3.4 million over the next 30 years. As a consequence of this increasing population there has been significant environmental and social change with rapid increases in vehicle numbers, (specifically kilometres travelled) and industrial /commercial activity. Increased emissions from vehicles and industry are likely to have contributed to increases in levels of air pollutants and potentially to hospitalisations for cardiovascular and respiratory diseases. This study examined whether there were in fact any increases in levels of criteria air pollutants and consequently in the rate of hospital admissions for selected disease conditions.

1.2 Structure of the thesis

The first stage of this study will examine historical statutory air quality and hospitalisation data to determine whether there were any significant associations between pollutant levels and rates of cardiovascular and respiratory hospital admissions over the period 2001-2010. It will also whether there was any change in mean pollutant levels from the previous decade.

The second stage will examine in finer detail the nature of the particulate air pollution that currently exists in the Perth metropolitan area. The chemical composition of particulates has not specifically been investigated in earlier studies and it is not routinely investigated as part of the Department of Water and Environmental Regulation's Statutory Air Quality Management Programme. There is increasing concern regarding the possible health effects of smaller particles in the air as they have been shown to be more harmful than an equivalent mass of larger particles of the same material. In many cases health effects have been shown to be most closely linked to particle number / surface area (Laumbach & Kipen, 2012; HEI, 2013). In this stage field work was conducted to characterise the chemical composition of particulate pollutants in two selected areas that were representative of industrial and non-industrial areas of metropolitan Perth. The sampling took place in the industrial area during October / November 2013 and in the non-industrial area during August / September 2014.

The thesis is structured in 2 main parts as described in Figure 1.1

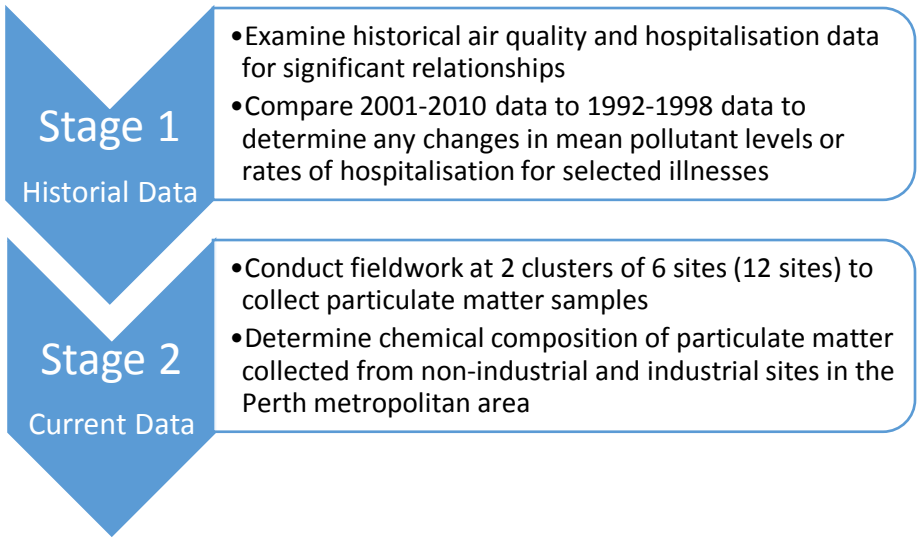


Figure 1.1: Representation of two-stage structure of thesis

1.3 Research aims & specific objectives

This research is split into two main aims which cover the two key foci of the work undertaken.

Aim 1: Evaluation of the relationship between daily levels of selected ambient air pollutants and respiratory and cardiovascular hospital admissions in the Perth metropolitan area from 2001-2010.

Specific Objectives

1. To evaluate the relationship between mean daily levels of selected ambient air pollutant concentrations and daily standardised hospital admissions;
2. To estimate the extent to which the studied air pollutants influence hospital admissions; and
3. To compare the ambient air pollution concentrations and hospital admissions for respiratory and cardio-vascular illnesses between the last two decades.

Aim 2: Characterisation and speciation of fine and coarse particulates at sites representative of non-industrial (background) and light industrial (source) locations within metropolitan Perth.

Specific Objective

1. Compare mass concentration and elemental composition of pollutants between light industrial and non-industrial areas

1.4 Significance of the study

The relationship between air pollutant levels and various health effects has been the subject of many previous studies, both nationally and internationally however this project is significant for several reasons.

This is only the second study, (to the knowledge of the author) and the first in the last 20 years that provides the opportunity to evaluate trends in air quality and associated hospital admissions for respiratory and cardiovascular illness in the Perth metropolitan area over the period 2001 – 2010. Previous similar studies examined air quality and hospitalisations from the previous decade (1992-1998) however there has been a significant growth in population since that time and this study will be able to

ascertain whether there has been a corresponding increase in hospitalisations for selected illnesses.

Previous investigations into metropolitan Perth's air quality have revealed few instances of pollutant levels exceeding statutory guidelines. Rather than look for numbers of hospitalisations when statutory levels were exceeded, this study examined the health effects of exposure to low and medium levels of air pollution, that are below the values stipulated in the Ambient Air Quality National Environmental Protection Measure (AAQ NEPM). This may assist policy makers in providing further evidence to advocate for a reduction in the acceptable levels of air pollutants from emissions.

This study was also the first to undertake a detailed assessment of the chemical composition of particulate matter, specifically metals, in ambient air in two clusters of sites representative of industrial and non-industrial areas of metropolitan Perth. Similar characterisation studies have been conducted in many cities around the world, but we are not aware of any similar studies in any other Australian cities including Perth. This characterisation of metalliferous particles is very important as there is increasing evidence that it is the metals present in particulate matter that may be responsible for any adverse health effects, especially cardiovascular outcomes. If any of the metals present in the sampled air are found to be present at levels that exceed guideline values, there would be justification for further investigation to identify the source of the emissions containing the particular metal/s and also the establishment of control measures and strategies to reduce or even eliminate the emissions.

CHAPTER 2 Literature Review

This chapter outlines Australia's ambient air quality frameworks, the main pollutants of concern, health effects associated with exposure and mechanisms of action, highlighting previous studies on ambient air quality both in Australia and overseas.

2.1 Introduction to ambient air pollution

Clean air is a fundamental determinant for healthy living and exposure to pollutants in the ambient air has the potential to cause significant adverse effects on human health. There is vast scientific evidence regarding the relationships between ambient or outdoor air pollution and various health and disease conditions. The following frequently quoted definition of air pollution (Seinfeld, 1986) highlights that pollutants are substances that are commonly present in our air and it is the concentration that determines whether or not their presence constitutes 'air pollution.'

> *'…an atmospheric condition in which 'substances' are present at concentrations high enough above their own ambient levels to produce a measurable effect on humans, animals, vegetation or materials'*
>
> *(Seinfeld, 1986, 22).*

These 'substances' or pollutants may be classified as either 'primary' pollutants or 'secondary' pollutants, depending on their origin. Primary pollutants are emitted directly into the atmosphere from identifiable sources such as factory chimneys or exhaust pipes, or through the suspension of dusts by the wind. Examples are carbon monoxide, nitric oxide, nitrogen dioxide, sulphur dioxide, carbonaceous and non-carbonaceous particulates and volatile organic compounds (VOCs). Secondary air pollutants are formed within the atmosphere as a result of chemical reactions between primary pollutants and often involve natural atmospheric components such as oxygen and water. Examples include nitrogen oxides, atmospheric sulphuric acid, sulphur trioxide, nitric acid, ammonium, tropospheric ozone and complex secondary particulates (Giorgini et al., 2016; WHO, 2006, 2016).

Ambient air pollution is a mixture of multiple pollutants (gases, liquids and particles) originating from many varied natural and anthropogenic (man-made) sources. The mix of pollutants may demonstrate substantial spatial and temporal variation, depending on sources, weather and other atmospheric processes. The source, concentration and also the chemical composition of the pollution may play a role in

determining biological toxicity and subsequent health effects (Brook, 2008; Loomis et al., 2013). Exposure to air pollution can have both acute and chronic effects on human health, mainly affecting the respiratory and cardiovascular systems. These range from minor upper respiratory irritation to chronic respiratory and heart diseases, asthma, lung cancer, acute respiratory infection in children, chronic bronchitis in adults as well as exacerbation of pre-existing heart and lung disease. It is also linked to premature mortality and reduced life expectancy (Kampa & Castanas, 2008; Keywood, Hibberd, & Emmerson, 2017; Martuzzi, et al., 2002).

In 2017 the World Health Organisation (WHO) stated that 'air pollution currently presents the most significant single environmental risk to public health worldwide' (Keywood, Hibberd, & Emmerson, 2017). Reported mortality figures vary between publications, however in 2012 it was estimated that there were approximately 3.7 million early or premature deaths attributable to the effects of ambient air pollution, and one of every nine deaths reported was the result of air pollution related conditions (Lim et al., 2012; Organization for Economic Co-operation and Development [OECD], 2014; WHO, 2017b). This represents 6.7% of total deaths worldwide, 16% of lung cancer deaths, 11% of COPD related deaths, 29% of ischemic heart disease and stroke and 13% of deaths due to respiratory infection.

More than half of this burden of disease from ambient air pollution is borne by the populations of developing countries, particularly those in Africa, Asia and the Middle East who have much higher levels of air pollution, up to several times higher than that considered safe by the WHO Air Quality Guidelines (AQG) (WHO, 2017b). WHO estimates that worldwide, approximately 90% of people are exposed to air that does not comply with these Guidelines. Global concern about air pollution is embedded in the United Nations Sustainable Development Goals (SDGs) (WHO, 2017a). The UN member states adopted a resolution to 'address the adverse effects of air pollution' through an enhanced global response that includes monitoring and reporting air pollutant levels in cities and air pollution related mortality rates.

2.1.1 International Air Quality Frameworks

The World Health Organisation first published Air Quality Guidelines (AQG) in 1987 which established recommended standards for carbon monoxide (CO), particulate matter (PM), ozone (O_3), nitrogen dioxide (NO_2), sulphur dioxide (SO_2) and lead (Pb). These are commonly known as 'criteria' pollutants' which means that they are regulated and used as indicators of air quality (Keywood, Hibberd & Emmerson, 2017). Since the Guidelines were first introduced, extensive research in many countries has led to ongoing updates and revisions of recommended levels. There is mounting evidence that for many pollutants, there is no clear threshold value below which there are no adverse effects (Department of Environment and Energy [DEE] 2015; Barnett, 2014; Kelly & Fussell, 2015: Keywood, Hibberd & Emmerson, 2017). The 1987 guidelines were first revised in 1997 and then following further extensive reviews of the scientific evidence, a global update was published in 2005. This update revised the guidelines and provided further supporting evidence for particulate matter, ozone, nitrogen dioxide and sulphur dioxide (Morawska, 2010).

The WHO air quality guidelines are applicable across all WHO regions and play an important role in providing guidance and recommendations to other countries to help manage air quality (Keywood, Hibberd & Emmerson, 2017; World Health Organization (WHO), 2017b). The goal of the WHO AQGs is to provide evidence–based recommendations to protect populations worldwide from the adverse effects of air pollution. In 1987, the authors of the guidelines noted that 'compliance with the recommendations regarding guideline values does not guarantee the absolute exclusion of effects at level below such values', especially with respect to sensitive groups that may be more susceptible such as those impaired by concurrent disease or other physiological limitations (WHO, 2017b) The Guidelines also do not consider the uncertainties related to combined exposure to various chemicals or exposure to the same chemical by multiple routes (Spickett, Katscherian, & Harris, 2013; WHO, 2017b).

WHO (2017b) also highlighted that they were providing 'guidelines', not 'standards' and it was up to each particular jurisdiction to consider prevailing exposure levels, as well as environmental, social, economic, and cultural conditions before setting standard values. They acknowledged that there may be circumstances where, in particular jurisdictions, there are justifiable reasons to implement policies which may result in pollutant concentrations higher than 'guideline' values. Most countries, including Australia, have adopted a standards type approach based on the WHO

AQGs to manage levels of pollutants, and conduct some form of air quality monitoring, especially in larger cities and around areas of particular concern such as transport networks and industry.

In the United States, air pollution is regulated by the *Clean Air Act* and its amendments (1970 through 1990), which enables the United States Environmental Protection Authority (U.S. EPA) to set national air quality standards for the same six criteria air pollutants (U.S. EPA, 2011, 2017b) as the WHO Air Quality Guidelines. There are acknowledged air quality problems in many large cities in the U.S. (U.S. EPA, 2017a). In 2012 approximately 74 million people in the U.S. were exposed to levels of $PM_{2.5}$ above the standard and that more than 131 million people were exposed to concentrations of ozone that exceeded maximum allowable levels (Caiazzo, 2013).

2.1.2 Ambient Air Quality Guidelines in Australia

In Australia, ambient air quality guidelines were first introduced in 1998 by the National Environment Protection Council (NEPC) as part of the National Environmental Protection Measure for Ambient Air Quality (the 'Air NEPM' or 'AAQ NEPM'). This NEPM was developed by the Australian Federal Government in consultation with State and Territory Governments, health professionals, environmental groups and the community, and was based on evidence from epidemiological, toxicological and clinical research from all over the world. This evidence provided a foundation for quantifying the risk due to exposure to ambient air pollutants and served to establish national air quality standards as the mechanism to enforce air quality management policies (Morawska, 2010). It also took into account Australian climatic conditions and likely exposures (Keywood, Hibberd & Emmerson, 2017).

Air pollution standards are initially developed based on best evidence available in public health epidemiological, toxicological and clinical studies. Establishing air quality policy is often a compromise between reducing standards sufficiently to minimise possible human health effects while striking a balance with what is economically feasible, pragmatic and achievable (Morawska, 2010). The Air NEPM sets goals or standards for air quality for 'priority' or 'criteria' air pollutants with the desired outcome being ambient air quality that allows for the adequate protection of human health and well-being with all Australians having the same level of air quality protection (Keywood, Hibberd & Emmerson, 2017). There is concern by some researchers however that compliance with the standards may not always achieve the desired level

of ‘adequate protection’ as they do not take into consideration population sub-groups that may have greater susceptibility such as the elderly, the very young and those with compromised immune systems (Barnett, 2014).

There are currently standards for seven ‘criteria’ air pollutants: carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), sulphur dioxide (SO_2), lead (Pb) and two different sizes of particulate matter, PM_{10} and $PM_{2.5}$ (DEE, 2017a) (Table 2.1). As new evidence becomes available, these standards are regularly reviewed and updated (Frangos et al., 2013). The $PM_{2.5}$ standard has only recently been included as an actual ‘Standard’. Until 2016 it was listed as an ‘Advisory Reporting Standard’ while data was being collected to establish appropriate levels. In February 2016 an amendment to the Air NEPM that changed the status of the annual average and 24-hour average for $PM_{2.5}$ from ‘Advisory Reporting Standard’ to ‘Standard’ came into effect.

Each air quality standard is presented as a maximum acceptable concentration and the period of time over which the concentration is averaged. Maximum concentrations are given in ppm for gaseous pollutants and $\mu g/m^3$ for particulate pollutants. For each of these standards occasional exceedances are permitted (see Table 2.1). The final column of Table 2.1 lists the 10 year goal for the maximum number of exceedances that are allowable in a year. In the 2015 ‘Variation to the National Environment Protection (Ambient Air Quality) (National Environmental Protection Council [NEPC], 2015), amendments were made to the number of days that PM_{10} and $PM_{2.5}$ could exceed the maximum concentration allowable per year. For both pollutants this was changed from 5 days per year to ‘None’ with an ‘Exceptional Event’ rule as follows:

> ‘Exceptional event means a fire or dust occurrence that adversely affects air quality at a particular location, and causes an exceedance of 1 day average standards in excess of normal historical fluctuations and background levels, and is directly related to: bushfire; jurisdiction authorised hazard reduction burning; or continental scale windblown dust.’ (NEPC, 2016, p2)

The standards set by the Air NEPM are legally binding on both the state and federal government. The NEPM requires each jurisdiction to monitor air quality in an attempt to identify potential air quality problems and put strategies in place to reduce pollutant emissions to achieve the standards. There is also mandatory reporting for each jurisdiction to report their performance in reducing levels of the criteria pollutants. On a national level, the AAQ NEPM also improves the consistency of air monitoring reporting across Australia (Keywood, Hibberd & Emmerson, 2017). In Western

Australia, the State Government prepares an annual Air Quality Monitoring Report for submission to the National Government to comply with the AAQ NEPM.

Table 2.1: National Air Quality Standards (Australian Government Department of Environment and Energy, 2017; NEPC, 2016).

Pollutant	Averaging Period	Maximum (Ambient) Concentration	Goal within 10 years (Maximum number of allowable exceedances)
Carbon Monoxide	8 hours	9.0 ppm	1 day a year
Nitrogen Dioxide	1 hour	0.12 ppm	1 day a year
	1 year	0.03 ppm	None
Photochemical oxidants (as ozone)	1 hour	0.10 ppm	1 day a year
	4 hours	0.08 ppm	1 day a year
Sulphur Dioxide	1 hour	0.20 ppm	1 day a year
	1 day	0.08 ppm	1 day a year
	1 year	0.02 ppm	None
Lead	1 year	0.50 µg/m^3	None
Particles as PM_{10}	1 day	50 µg/m^3	None [c]
	1 year [a]	25 µg/m^3	None
Particles as $PM_{2.5}$	1 day [b]	25 µg/m^3	None [c]
	1 year [a]	8 µg/m^3	None

[a] Added in the *'Variation to the National Environment Protection (Ambient Air Quality) 2015 (National Environmental Protection Council [NEPC] 2016).*

[b] Status amended in the *'Variation to the National Environment Protection (Ambient Air Quality) 2015 (National Environmental Protection Council [NEPC] 2016).*

[c] In the *'Variation to the National Environment Protection (Ambient Air Quality) 2015 (National Environmental Protection Council [NEPC], 2016),* these values which were previously 5 days per year, were replaced with 'None' and an 'Exceptional Event' rule.

2.1.2.1 National Clean Air Agreement

In the 2016 Australian Federal Government *State of the Environment* Report, it was stated that by world standards, Australia has very clean air which is generally classified as 'good' or 'very good'. Strategies developed to manage air pollution have contributed to reducing the levels of most of the main pollutants, with the levels of carbon monoxide, lead, nitrogen dioxide, sulphur dioxide and PM_{10} all having declined over the last 10 years (Keywood, Hibberd & Emmerson, 2017). There are however, still challenges with levels of particulate matter and ozone frequently exceeding national ambient air quality standards in urban areas and communities exposed to emissions from domestic and industrial combustion sources. There is also the extra challenge of future proofing our air quality to meet the additional stressors or drivers such as population growth and subsequent increases in transport and energy demands that could potentially accelerate air pollution problems (DEE, 2017a).

To ensure that the community continues to enjoy clean air and also to minimise the impact from any ambient pollutants on health and the environment, Australian Environment Ministers from all states and Territories established the '*National Clean Air Agreement'*. The aim of this agreement is to provide a basis to identify, prioritise and focus actions to deliver strategic and consistent approaches to air quality management through cooperation between all levels of government with business and the community. Initial actions from this agreement include strengthening the reporting standards for particulates in the AAQ NEPM and also introducing strategies to reduce air pollution emissions from wood heaters and non-road spark ignition engines and equipment. Future actions that have been flagged include strengthening the sulphur dioxide, nitrogen dioxide and ozone reporting standards in the AAQ NEPM, reviewing Australia's fuel quality standards and introducing initiatives to reduce localised emissions from off-road diesel engines and ships (DEE, 2015).

2.2 Sources of air pollution and mitigators of effects

The main 'naturally' occurring sources of criteria air pollutants in Australia are bushfires and dust storms which are particularly prevalent in urban fringe and regional areas that have large areas of bushland. Dust storms tend to occur in more agricultural areas that have been cleared of natural vegetation (Keywood, Hibberd & Emmerson, 2017). The predominant anthropogenic or man-made sources include vehicle fuel combustion, fossil fuel based power generation, industrial activity,

biomass burning, and domestic heating and cooking (Committee on the Medical Effects of Air Pollutants [COMEAP], 2009; Keywood, Hibberd & Emmerson, 2017; Johnston et al., 2011).

It has been acknowledged by many studies that combustion emissions from various sources are a major contributor of air pollutants. In the United States there are approximately 200,000 early deaths each year due to combustion emissions from several main sectors, mainly transport and energy generation. Caiazzo et.al. (2013) used a multiscale model looking specifically at levels of $PM_{2.5}$ and O_3 emissions to identify which sectors had the greatest impacts on the number of premature or early deaths. They found that emissions from road transport were the most significant contributor to premature death, being responsible for approximately 26.5% of total early deaths due to changes in $PM_{2.5}$ concentrations and half of the early deaths due to changes in ozone concentrations. Power generation was similar, being responsible for 26% of early deaths due to changes in $PM_{2.5}$ but only 20% of early deaths due to changes in ozone. While still significant, industry was responsible for only approximately 20% of premature deaths for both pollutants. The remaining three sectors, commercial and residential sources, marine and rail transportation have less impact and combined were responsible for the residual 27% and 10% of $PM_{2.5}$ and O_3 related early deaths (Caiazzo et al., 2013).

2.2.1 Motor vehicles

Motor vehicles are a major threat to air quality in Australia accounting for 40% of the CO emissions, 41% of VOCs, 18% of NO_x and 17% of the $PM_{2.5}$ registered in the National Pollution Inventory (Keywood, Hibberd & Emmerson, 2017; Department of Sustainability Environment Water Population and Communities [DSEWPC], 2010a). Advances in motor vehicle engine and emission control technology as well as improved fuel standards have driven down emissions of CO and VOCs, and the levels of NO_x are also expected to decline in the future. These reported gains from advances in technology however, are cancelled out by the combination of increasing vehicle numbers, increased kilometres travelled and congestion as well as non-tailpipe particle emissions such as from brake and tyre wear. From 2011-2016 the number of motor vehicle registrations in Australia increased by 12% with passenger vehicles making up 75% of the total Australian fleet. The number of diesel fuelled registered vehicles alone grew 10% between 2010 and 2015 and now make up 20.9 % of all registered vehicles (Keywood, Hibberd & Emmerson, 2017).

Traffic related air pollution (TRAP) is a complex mix of PM derived from combustion (including elemental or black carbon (BC)) and also non-combustion sources such as road dust, tyre wear and brake wear, and also gaseous emissions including NO_x. These primary emissions lead to the generation of secondary pollutants such as ozone, nitrates and organic aerosols (Guarnieri & Balmes, 2014). A study into the long term effects of TRAP in a Dutch cohort (NCLS-AIR Study) found that TRAP and several other variables related to exposure to traffic exposure were associated with mortality although the relative risks were generally small (Beelen et al., 2009). The authors found statistically significant associations between respiratory mortality and exposure to NO_2 and black smoke (BS), and that increased mortality was associated with traffic intensity on the nearest road. They followed the cohort (120,852 subjects) for 10 years and looked at exposures to BS, $PM_{2.5}$, NO_2, and SO_2 at their home address. There was no association with SO_2 however the relative risk for a 10 $\mu g/m^3$ increase in BS was 1.05 (95% CI: 1.00-1.11) for natural causes, 1.04 (95% CI: 0.05-1.13) for cardiovascular disease, 1.22 (95% CI: 0.99-1.5) for respiratory disease, and 1.03 (95% CI: 0.88-1.20) for lung cancer. They found similar results for $PM_{2.5}$ and NO_2 (Beelen et al., 2009).

Künzli et al. (2000, 2005) estimated the impact of outdoor and TRAP on public health in Austria, France and Switzerland by quantifying exposure-response effects for a 10 $\mu g/m^3$ increase in PM_{10}, using PM_{10} as a surrogate or indicator of fossil fuel combustion pollutants. The authors found that air pollution caused 6% of total mortality or more than 40,000 attributable cases per year and about half of these cases were attributed to air pollution from motor vehicles. They also found that TRAP was responsible for more than 2,000 new cases of chronic bronchitis in adults, more than 290,000 episodes of bronchitis in children, more than 500,000 asthma attacks overall and more than 16 million person days of restricted activities. Guarnieri & Balmes, 2014) reported that the concentration of many of the constituent pollutants in TRAP (particularly ultrafine PM and BC particles) diminish quickly with increasing distance from roadways, with a distance of 300-500 metres being the most relevant to human health. In large North American cities, 30-45% of people live within this distance of major roadways, and the burden of near roadway exposure appears to be even higher in many European cities.

Ship exhaust is also an important source of human exposure to $PM_{2.5}$ for those who reside or are employed near shipping lanes such as in the Sydney greater metropolitan region (Broome et al., 2016). In 2008, ship exhaust was the sixth largest

source of $PM_{2.5}$ in the region, responsible for 2.7% of all anthropogenic emissions. The authors noted that in 2010/11, approximately 1.9% of the annual average population weighted-mean concentration of all natural and man-made $PM_{2.5}$ in the region was attributable to ship exhaust, and up to 9.4% at suburbs close to ports. An estimated 220 years of life (95% CI: 140 - 290) were lost during this time as a result of ship-related exhaust exposure. The authors calculated that the population weighted-mean concentration of ship-related $PM_{2.5}$ could be reduced by 25% by switching from standard 2.7% distillate sulphur based fuel to a lower 0.1% sulphur based fuel at berth, which would result in a gain of 390 life years (95% CI: 260 - 520) over a 20-year period. Extending the requirement to use low sulphur fuel to within 300 km of Sydney would reduce the concentration of $PM_{2.5}$ by 56% and result in a gain of 920 life years over 20 years (95% CI: 600 - 1200) (Broome et al., 2016).

2.2.2 Bushfires, prescribed burns and domestic wood heaters

Australian cities are frequently affected by bushfire smoke given their proximity to highly flammable native vegetation and also hot dry weather conditions that favour combustion (Johnston, 2009, 2011; Johnston et al., 2011). In Western Australia, particulate matter from bushfires is the most significant source of 'naturally occurring' or non-industrially related air pollution. Whether these fires are sparked by natural causes such as lightning, caused by acts of vandalism or arson, or are part of a prescribed burning or fuel reduction programme, all have the potential to cause episodes of air pollution across wide geographic areas and frequently affect major population centres. Studies in most states and capital cities (Darwin, Brisbane, Melbourne, Sydney) (Johnston, 2009; Johnston et al., 2011; Morgan, 2011) indicate that bushfire smoke is associated with respiratory morbidity, and the magnitude of the risk may be larger than those for particulate matter from urban sources such as traffic. Morgan (2011) noted that there is less evidence for the effect of bushfire smoke on cardiovascular morbidity, however there is some evidence of increased mortality. Bushfire activity is expected to increase in the future as a result of extreme temperatures associated with climate change and associated changes in controlled vegetation burning practices, both resulting in increased population exposures to pollution (Morgan, 2011). Although it is a broadly accepted bushfire mitigation strategy, there is considerable controversy over the use of controlled burns for fuel reduction due to the potential adverse health effects resulting from resident's exposure to air pollutants (Johnston, 2009; Johnston et.al., 2011).

In Australia, extreme bushfires that last for several days generally occur in regional areas with those that occur in urban areas tending to be shorter and far more frequent. Urban bushfires quite often go unreported as they are extinguished more quickly and tend to do less damage. Depending on the composition of the fuel load, there may also be smouldering for many days after the flames are extinguished. The number of vegetation fires in Perth, whether naturally or deliberately lit, is high relative to other Australian cities (Blake, van Etten, & Horwitz, 2011). They are more frequent in areas with remnant bushland and wetlands, which is generally urban fringe or older suburbs with a high proportion of bushland, and occur all year round, being more common in summer. The high prevalence of these fires close to homes presents a considerable potential for adverse human health effects as a result of exposure to particulates, especially if the exposure is long-term and recurrent (Blake, van Etten, & Horwitz, 2011).

An analysis of the smoke from bushfires in peat containing wetland identified high particulates, irritants and carcinogens (Blake, van Etten, & Horwitz, 2011). Most of the exceedances in particulate levels in Perth reported in the 2012-2013 Air Quality Management Plan Report Card were associated with bushfires (DER AQCC (WA), 2014). A previous study conducted in Darwin during a dry season characterised by minimal rainfall and almost continuous bushfires (Johnston et al., 2011), found a significant increase in hospital emergency presentations with each 10 $\mu g/m^3$ increase in PM_{10} concentration, with the strongest effect when increases were greater than 40 $\mu g/m^3$. In 2007 (Johnston et.al., 2007) examined health effects from biomass fires in Darwin over three fire seasons. During the seasons there were a total of 2,466 admissions, of these 23% were from indigenous populations. They concluded that PM_{10} derived from vegetation fires was predominantly associated with admissions for respiratory illness rather than cardiovascular illness and that indigenous groups were at higher risk.

Johnston et al. (2011) examined the association between air pollution from extreme air pollution events caused by smoke from bushfires and dust storms and mortality in Sydney from 1997 to 2004. During this period there were 52 air pollution event days when the city wide PM_{10} concentration exceeded the 99^{th} percentile. Of the 52 event days, 48 were categorised as being attributable to smoke from bushfires, six were attributable to dust and two were affected by both smoke and dust. The authors (Johnston et al., 2011) found that although the magnitude and temporal patterns (lag periods) differed, both smoke and dust events were significantly associated with

increased mortality. Smoke events were associated with a 5% increase in mortality after a one day lag with an odds ratio of 1.05 (95% CI:1.00–1.10) and dust events were associated with a 15% increase in mortality after a three day lag, with a an odds-ratio of 1.15 (95% CI: 1.03–1.30) (Johnston, 2011; Johnston et al., 2011). They noted that there is still insufficient evidence to determine whether the particulate matter from vegetation fire smoke or dust storm pollution carries a greater or lesser risk of mortality compared with particulate matter from urban sources. They also noted that the episodic nature of large events makes them challenging to predict precisely when and where air quality will be affected. As a result most air pollution studies are retrospective and are dependent upon routinely collected data. They also highlighted that during these extreme events, peak exposures may be too short-lived to detect all but the most sensitive health outcomes. The location of monitoring stations is also crucial in determining whether peak exposures are even recorded.

In addition to smoke from bushfires, domestic wood heaters and prescribed burns are also cited in several studies as sources of particulate matter. In the Huon valley in Tasmania, Reisen, Meyer & Keywood (2013) conducted a study to assess whether prescribed burns rather than domestic wood heaters were responsible for major pollution events that that lead to a number of exceedances of national air quality standards. The authors undertook a 20 month study to quantitatively assess the seasonal atmospheric PM_{10} and $PM_{2.5}$ particle loadings at one rural and one urban site in the area. Their results showed that biomass burning (whether from wood heaters or prescribed burns) was a significant source of $PM_{2.5}$ leading to exceedances of the 24 hr AAQ NEPM standard. They also found that although the intensity of emissions was similar, in terms of the ambient $PM_{2.5}$ load, emissions from wood heaters contributed 77% of the load compared to only 11% from prescribed burns. Air quality at the urban site was impacted more than at the rural site, indicating that localised sources of $PM_{2.5}$ have more effect than regional sources of pollution.

Reisen, Meyer, & Keywood (2013) also found wood heater pollution was more likely to be a night-time issue in contrast to prescribed burn events which always occurred during the day and were of short duration. They suggested that most of the issues with prescribed burns occurred in the late afternoon towards the end of the burn, when there is increased smouldering, reduced vertical mixing and lower ventilation rates, leading to less pollutant dispersal.

In Finland, Kollanus et al. (2016) investigated the association between short-term exposure to long range transported $PM_{2.5}$ from vegetation fires in the south and daily

mortality due to cardiovascular and respiratory causes in the Helsinki metropolitan area, from 2001-2010. They found that on smoke-affected days, a 10 μg/m^3 increase in $PM_{2.5}$ was associated with a borderline statistically significant increase in cardiovascular mortality among the total population at a lag of 3 days (12.4%, 95%CI: -0.2%, 26.5%), and among the elderly (≥65years) following same-day exposure (13.8%, 95% CI: -0.6%, 30.4%), and at a lag of 3 days (11.8%, 95% I: -2.2%, 27.7%). $PM_{2.5}$ levels on these days were not associated with admissions due to respiratory causes. On non-smoke affected days, $PM_{2.5}$ was not associated with any of the studied health outcomes. The authors concluded although most associations were borderline significant, there was suggestive evidence for an association between exposure to $PM_{2.5}$ from vegetation fires that had been transported over a distance of hundreds to thousands of kilometres and increased CVD mortality, and to a lesser extent with increased hospital admission due to respiratory causes. This has serious public health implications as most studies focus only on local pollutants.

2.2.3 Effects of temperature and humidity

Several studies (Barnett et al., 2006; Qiu et al., 2013; Zhao et al., 2017) have examined whether factors such as temperature, season and relative humidity (RH), modify the effects of air pollution and subsequent health effects. Predicted high temperatures and drier weather resulting from climate change are expected to contribute to an increase in the frequency and severity of dust storms and bushfires, resulting in higher photochemical activity and subsequent emissions of ozone precursors. This could lead to more smog and higher ozone and particle concentrations in some areas (DSEWPC, 2011). Strategies developed to mitigate the effects of climate change such as greenhouse gas abatement policies are also likely to reduce air pollution as pollutants and greenhouse gases are often emitted from the same source. Zhao et al. (2017) investigated the relationship between ambient temperature and emergency department visits in China and found that independent of air pollution and humidity, both hot and cold temperatures were associated with increased risk of emergency department presentations.

A number of studies have also noted that lower humidity appears to be associated with a greater association between pollutants and hospital admissions (Barnett et al., 2006). Qiu et al. (2013) conducted a time series study to examine the effects of air pollution on emergency COPD hospital admissions in Hong Kong. They found increases in the adverse effects of air pollution in the cool season and also on low

humidity days suggesting that season and humidity jointly modified the effects of gaseous pollutants, resulting in increased emergency COPD hospitalizations on the cool and dry days.

2.3 Main ambient air pollutants of concern

Urban air pollution is a complex mixture that may contain a mix of pollutants plus other aerosols. The implications of exposure for human health depends on many factors including the types and concentration of pollutants (Williams, 2012). The main ambient air pollutants that have been identified as presenting a risk to human health are nitrogen dioxide (NO_2), ozone (O_3), carbon monoxide (CO), sulphur dioxide (SO_2), lead and both coarse and fine particulate matter (PM_{10} and $PM_{2.5}$) (Keywood, Hibberd & Emmerson, 2017; WHO, 2017b).

Residency time or how long the pollutants stay airborne, is an important consideration for pollutant exposure. Atmospheric residency times are typically only hours and some background levels tend to be low although $PM_{2.5}$ and ozone have residency times of days or even weeks which means they can be transported on a regional scale. Pollutants from urban sources such as NO_x and CO from road traffic tend to be in high concentrations throughout the city and significantly reduced in adjacent rural areas. Sulphates and ozone can readily travel thousands of kilometres in long-range transport and fine aerosols of black carbon (BC) can also be transported over long range (WHO, 2006). Some fine and ultrafine particles are able to stay airborne for up to 100 days which allows for a wide geographic distribution (Geiger & Cooper, 2010).

2.3.1 Carbon monoxide

Carbon monoxide (CO) is a colourless, odourless and tasteless gas that forms during the incomplete combustion of carbon containing fuels such as petrol, gas, oil, coal and tobacco products (Chiew & Buckley, 2014; DSEWPC, 2011). Complete combustion leads to the formation of carbon dioxide (CO_2) however in most combustion systems a proportion of carbon is only oxidised to CO (WHO, 2006). The main source of CO in urban areas is vehicle exhaust with some contribution from other industrial and domestic combustion activities. It is the most common pollutant by mass in the atmosphere. CO levels in Australia tend to be greatest in areas of high traffic density and decreased significantly from 2000 to 2010 due to improved emission

controls on motor vehicles. Ambient levels normally present are unlikely to cause ill effects (Chiew & Buckley, 2014; DSEWPC, 2011).

Unlike most other ambient pollutants, CO doesn't irritate the lungs but rather manifests effects as a result of tissue hypoxia. It is absorbed directly into the bloodstream where it binds with haemoglobin (with an affinity 200 to 240 times that of oxygen) to form carboxyhaemoglobin which has a reduced capacity to transport oxygen and release it to cells (Chiew & Buckley, 2014; Williams, 2012). Toxic effects of CO exposure are first observed in organs that require high levels of oxygen such as the brain, heart and exercising muscle. Exposure to 200 ppm for two to three hours can cause fatigue, headache and dizziness. Exposure to higher concentrations can lead to acute CO poisoning, which causes drowsiness, impaired coordination, reduced cognitive performance, loss of consciousness and death (Chiew & Buckley, 2014).

2.3.2 Nitrogen oxides (NO_x) and nitrogen dioxide

Nitrogen oxides (NO_x) are a group of highly reactive nitrogen and oxygen containing gases, formed during high temperature combustion and found in significant quantities in combustion emissions. NO_x is generally composed of just NO and NO_2 although the group also includes nitrogen trioxide (NO_3), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4), and dinitrogen pentoxide (N_2O_5) (DSEWPC, 2011). In the atmosphere, emitted NO_x are rapidly oxidised by oxidants such as ozone to NO_2 which is a brown coloured, acidic gas that is pungent at high concentrations. It is a precursor of photochemical smog and contributes to its' colour and distinctive odour. NO_2 is a common environmental air pollutant typically associated with on-road vehicle emissions (from internal combustion engines), and burning fossil fuels such as coal, oil and gas for heating and electricity generation (Keywood, Hibberd & Emerson, 2017). Other sources include the intrusion of stratospheric NO_x, bacterial and volcanic action and lightning. In Sydney, motor vehicles account for approximately 70% of NO_x, industrial factories account for 24% and other mobile sources account for the remaining 6% (AIHW, 2010; WHO Regional Office for Europe, 2000). Epidemiological studies have found NO_2 to be highly correlated with other primary and secondary combustion related pollutants and have used it as a marker of combustion related pollution mixtures, particularly traffic emissions or indoor combustion sources (Morawska, 2010; WHO, 2006).

Many studies have provided evidence of associations between NO_2 exposure and a range of adverse health effects including decreased lung function and increased incidence of respiratory illness as well as increases in hospital admissions and emergency room visits for respiratory and cardiovascular diseases and asthma. There is also evidence of increased mortality due to elevated exposure to NO_2 (Hansen, Peng & Nitschke, 2009; Hinwood et.al, 2004; Keywood, Hibberd & Emerson, 2017). Similar findings have been reported by studies in Australian cities, including a 2010 report published by the Australian Institute of Health and Welfare (AIHW) that stated 3% of all hospital admissions to Melbourne hospitals in 2006 were related to NO_2 exposure (AIHW, 2010).

Depending on exposure levels, NO_x can irritate the eyes, nose, throat and lungs, leading to coughing, shortness of breath, tiredness and nausea. Breathing high levels of NO_x can cause rapid burning, spasms and swelling of tissues in the throat and upper respiratory tract. It can lead to the reduced oxygenation of tissues, a build-up of fluid in the lungs and possibly death (DSEWPC, 2010a). NO_2 can also exacerbate pre-existing COPD and asthma, especially in children. On a cellular level, exposure to NO_x increases the risk of lung infections by impairing the function of the alveolar macrophages and epithelial cells (Williams, 2012).

Toxicological studies and clinical trials have provided evidence that exposure to NO_2 between 0.2-0.3 ppm can lead to cell damage in human lung cells and increase airway reactivity in asthma sufferers (California Environmental Protection Air Resources Board, 2009). Several international and Australian studies have also reported significant associations between an increase in NO_2 of 10 $\mu g/m^3$ and increased rates of total, respiratory and cardiovascular mortality (Samoli et al., 2006; Faustini, Rapp, & Forastiere, 2014; Luo et al., 2016). Several large scale studies in Europe and America (California Environmental Protection Air Resources Board, 2009) have reported associations between long-term exposure to NO_2 and lung function in both adults and children. In addition, several epidemiological studies found associations between of NO_2 concentration and lung function and growth in children, symptoms in asthmatic children and premature birth (Larabee & Phipatanakul, 2012; Gaffin et al., 2017).

Although ambient levels of NO_2 in Australian cities are relatively low, it is still a pollutant of concern because as well as being toxic in its present form, it also undergoes photo-chemical reactions with volatile organic compounds (VOCs) and oxygen causing the formation of ozone and other photochemical oxidants (WHO,

2006; DSEWPC, 2011). Concentrations of NO_2 are often strongly correlated with those of other combustion and traffic derived pollutants, and as it is routinely measured in many jurisdictions, is often used as a surrogate for the pollutant mixture as a whole (WHO, 2006).

2.3.3 Tropospheric (ground-level) ozone

Ozone (O_3) is a photochemical oxidant and the main component of smog. It is a secondary pollutant formed naturally in the presence of strong sunlight by the chemical interaction of urban pollutants, principally NO_x and VOCs emitted from motor vehicles, industrial and domestic sources and bushfires (DSEWPC, 2011; Keywood, Hibberd & Emmerson, 2017). Because ozone at ground level forms more readily in warmer weather, it is predominantly a summertime air pollutant and mainly a problem from late spring to early autumn (DSEWPC, 2011). High ozone levels are often associated with regional bushfires (DSEWPC, 2011).

Ozone is a highly corrosive gas and when inhaled can irritate the membranes lining the nose, throat and airways reducing lung function. It has been shown to induce inflammation of mucous membranes and, in some individuals may induce bronchoconstriction (Williams, 2012). Short-term exposure to moderate levels may cause irritation of the eyes and respiratory system and exposure to higher levels may lead to bronchitis and pneumonia. Ozone may also make the lungs more susceptible to infection (Environmental Protection Authority Review Steering Committee, 2007). Vulnerable groups who are more susceptible to the adverse health effects of ozone exposure are children, the elderly, those with pre-existing cardiovascular or respiratory conditions such as asthma sufferers (Johnston, 2009, 2011; Johnston et al., 2011).

Healthy people may also experience symptoms such as chest pain, coughing wheezing and congestion while exercising. Health effects associated with exposure to ozone depend on the concentration and duration of the exposure and range from minor changes in lung function to more serious effects that lead to hospital admissions and emergency room visits for respiratory and cardiovascular disease (Hansen, Peng, & Nitschke, 2009). Symptoms associated with ozone exposure include cough and chest pain on inspiration that, although not life threatening, may affect the quality of life (Jalaludin et al., 2006).

The respiratory health effects of short-term exposure to ozone, start to become evident adverse effect level estimated at 75 ppb. Clinical trials and epidemiological studies have shown small but significant declines in lung function after acute exposure to ozone. Associations between daily mortality and ozone levels have been reported with evidence of a small increase in mortality from respiratory and cardiovascular causes, especially in the elderly. There is also evidence of an association between short-term exposure to ozone and cardiovascular morbidity (Hansen, Peng, & Nitschke, 2009).

Many studies, both Australian and overseas, have demonstrated seasonal variation for a range of health effects associated with ozone levels. Increased adverse effects are generally reported in the warmer months when there is an increased concentration of ozone in the ambient air. Groups that may be exposed to higher levels of ozone and are therefore considered more susceptible are those that spend an increased length of time outside such as outdoor workers, children and athletes (Morawska, 2010; Hinwood et al., 2006). As ozone levels are known to increase in warmer temperatures, it has been suggested by several authors that levels of ozone are likely to rise in response to increased temperatures associated with climate change (Johnston, 2011). Because ozone is formed as a component of a photochemical smog complex, it has also been suggested that its presence is indicative of other toxic oxidants arising from similar sources. There is also evidence that long-term exposure may have chronic health effects but it is not sufficient to recommend an annual NEPM guideline (Morawska, 2010). In Australia ozone currently only has short term exposure standards measured over one and four hour averaging periods. (Table 2.1).

Elevated levels of ground level ozone, together with increased levels $PM_{2.5}$ are frequently associated with the incidence of premature mortality and morbidity outcomes (COMEAP, 2011; Dockery et al. 1993; Jerrett et al., 2005, 2009; Pope et al., 2002; WHO, 2006) These two pollutants are currently considered the most significant known causes of early mortality related to poor outdoor air quality (U.S. EPA, 2011). The U.S. EPA estimated that in 2010 there were approximately 4,700 early deaths related to ozone exposure (Fann et al., 2012). The 2015 Global Burden of Disease Study (Cohen et al., 2017) found that globally, exposure to ambient ozone caused an additional 254,000 deaths (97,000–422,000) and the loss of 4.1 million (1.6 million – 6.8 million) DALYs from COPD in 2015. Jerrett et al (2009) examined the contribution of long-term ozone exposure to air pollution-related mortality using data from 448,850 subjects and 118, 777 deaths over an 18-year follow-up period.

They were able to demonstrate a significant increase in the risk of death from respiratory causes in association with an increase in ozone concentration, however could not show a relationship with death from cardiovascular causes when the concentration of $PM_{2.5}$ was taken into account.

Berman et al. (2012) examined the potential effect of achieving the current NAAQS for ozone (75 ppb) and also lowering it to 70 ppb and 65 ppb as recommended by the U.S. EPA Clean Air Scientific Advisory Committee (CASAC). The authors estimated that the number of ozone-related deaths that could have been avoided in the U.S. during the period 2005-2007 ranged from 1,410 to 2,480 at 75 ppb to 2,450 to 4,130 at 70 ppb and 5,210 to 7,990 at 60 ppb. If the current standard was maintained acute respiratory cases would have been reduced by three million cases and school-days lost by one million cases annually. Significantly improved health outcomes would be achieved if the CASAC recommended range of standards (70-60 ppb) was met. They concluded that reducing the current NAAQS for ozone would significantly reduce ozone-related premature mortality and morbidity.

2.3.4 Sulphur dioxide

Sulphur dioxide (SO_2) is a colourless, irritant gas that has a strong suffocating odour. It is released into the atmosphere by natural processes such as erupting volcanoes and the decay of vegetation in peat bogs, tidal marshes and seaweed. It is also produced anthropogenically by burning fossil fuels such as coal, oil and gas and smelting mineral ores that contain sulphur. Levels in Australia are generally low with the exception of regional towns with smelting operations (DSEWPC, 2011).

SO_2 is non-lethal at usual concentrations of ambient exposure, however exposure for more than a few minutes may cause eye, mucus membrane and throat irritation. People with asthma are particularly susceptible to the adverse effects of exposure and even brief exposure to concentrations as low as 0.25 ppm may cause bronchoconstriction (airway narrowing) with typical asthma symptoms of wheezing, chest tightness and/or shortness of breath. Non-asthmatics are usually not sensitive to concentrations up to 1 ppm (Williams, 2012).

2.3.5 Lead

Lead is a naturally occurring heavy metal that is found in the earth's crust. It can be released into the soil, air and water through natural processes such as erosion, volcanic eruptions, sea sprays and bushfires and can also be emitted from various industrial processes such as waste incinerators, battery recycling, cement, plaster and concrete manufacturing, iron and steel fabrication and petroleum and coal products (DEE, 2017b). Emitted lead stays in the environment and can enter the body by ingestion of dust, food and water as well as through inhalation (DSEWPC, 2011). It is potentially toxic and exposure can affect the health of children, unborn babies and adults. Exposure in babies and children is of particular concern (Laidlaw & Taylor, 2011) as it can permanently damage the brain and impair intellectual development (DEE, 2017b; Laidlaw & Taylor, 2011).

Ambient lead levels in Australia have declined since the introduction of unleaded petrol in 1986 and the subsequent phasing out of leaded petrol which began in 1993 (DSEWPC, 2011). Before the phase-out commenced, the national air quality standard for lead was regularly exceeded in many urban environments. Ambient lead levels above the guidelines are now usually only found in regional towns where smelting occurs (Keywood, Hibberd & Emmerson, 2017). In urban and inner city areas that were subject to dense traffic before leaded petrol was phased out, many homes still have relatively high levels of lead in the soil where airborne particles containing lead have settled out (Laidlaw & Taylor, 2011). Although ambient levels of lead have decreased significantly, it is still considered as a criteria pollutant and is monitored and reported as part of the Ambient Air Quality NEPM. In Perth, lead has not been monitored at any of the Department of Environment Regulation's statutory monitoring stations since 2001 as the mean lead level in Perth is generally less than 5% of the NEPM standard (0.05 µg/m^3 averaged over one year).

2.3.6 Particulate matter

Particulate matter (PM) or Total Suspended Particles (TSP) is an air pollution term for a mixture of organic and inorganic solid particles and liquid droplets found in the air (U.S.EPA, 2010). It comes in a variety of sizes and can be composed of many types of chemicals and materials with the composition varying across time and location (WHO, 2006). PM can be emitted directly from a range of both natural sources such as forest fires, pollen, sea spray, wind erosion and dust storms and anthropogenic or

man-made sources such as agricultural practices, vehicle emissions, iron and steel making, fossil fuel power plants and construction (DSEWPC, 2010b). PM can be either primary pollutants emitted directly or secondary pollutants formed indirectly from the conversion of precursor pollutants through photo-chemical processes (DSEWPC, 2011). Newly formed secondary particles can be as small as 1-2 nm diameter, while crustal dust and sea salt can be as large as 100 µm.

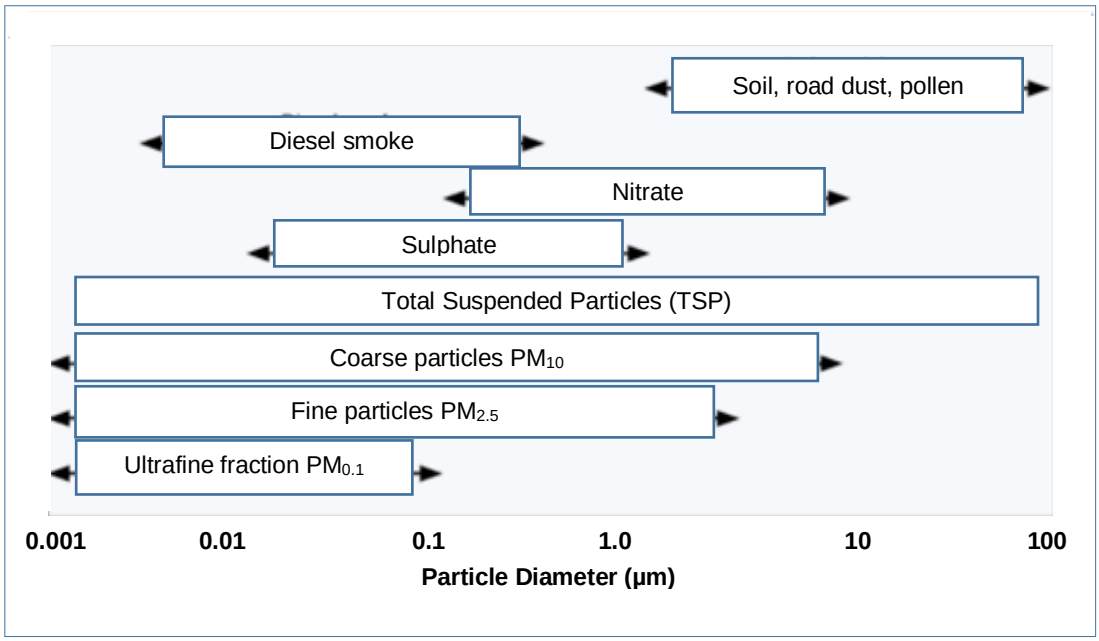


Figure 2.1: Size range of airborne particles, showing the health related ultra-fine, $PM_{2.5}$ and PM fractions and typical size range of some major components (Adapted from WHO, 2006).

PM is typically classified by size and in general, the smaller the particle, the stronger its potential impact on human health because it can be more easily inhaled and penetrate deeper into the respiratory tract (U.S. EPA, 2010; Williams, 2012) (Figure 2.1). Particles larger than PM_{10} are included in the overall definition of PM or TSP and these are usually trapped in the upper respiratory tract so have limited health effects other than irritation (Figure 2.4). Figure 2.2 shows a simplified diagram of the relative occurrence of various sized particles, usual source, composition and also location of deposition in the respiratory system, noting typical bimodal distribution (U.S.EPA, 2010).

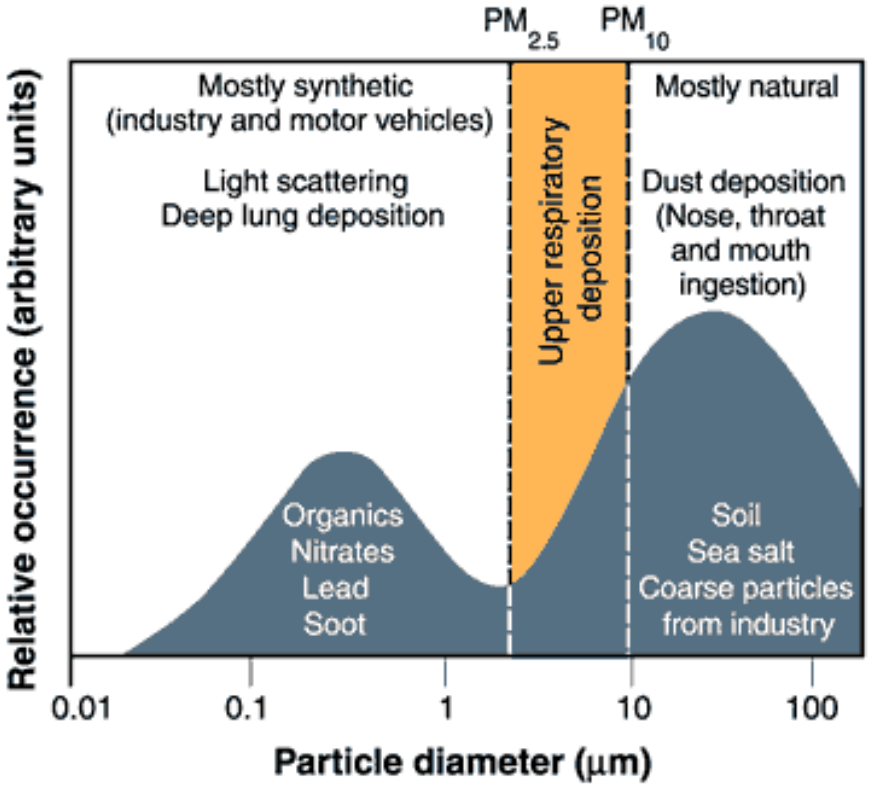


Figure 2.2: Chart showing relative occurrence of various sized particles, usual source, composition and also location of deposition in the respiratory system (U.S. EPA, 2010).

The three main categories of particulate matter are coarse (PM_{10}), fine ($PM_{2.5}$) and ultrafine particles ($PM_{0.1}$). Figure 2.3 illustrates the size differential between the two main categories of particles.

- PM_{10} (coarse or thoracic) particles are less than 10 µm (10,000 nm) in aerodynamic equivalent diameter (AED) and in general arise from natural sources such as dust, soil, agriculture, plants, pollen, mould spores, ocean spray and crustal components from volcanos and surface mining such as metal oxides. They are also associated with bushfires, wood heaters and dust storms (Keywood et al., 2013). Particles in this size range make up a large proportion of the dust that can be drawn into the lungs although they generally do not penetrate beyond the extra-thoracic and upper bronchial regions of the human respiratory system and tend to be trapped in the nose, mouth and throat (Chin, 2015; Williams, 2012). The larger particles tend to have a short atmospheric existence (minutes to hours), dropping out due to gravity and wind driven impaction processes. They only travel one to ten kilometres so are

usually only found close to the emission source (Kim, Kabir, & Kabir, 2015; WHO, 2006).

- $PM_{2.5}$ (fine particles) are less than 2.5 µm (2,500 nm) in AED and generally arise as primary pollutants from combustion (including forest fires), smog, diesel and gasoline sources or are formed as secondary air pollutants when gases react in the air. These particles are of greater concern than PM_{10} as they are able to be remain airborne for longer (days to weeks) and be transported over longer distances (100 to 1000 kilometres) (Kim, Kabir, & Kabir, 2015). Physiologically they can get further into the lungs and penetrate into the small airways (bronchioli) and alveoli (Chin, 2015; Geiger & Cooper, 2010; Williams, 2012) where the adsorption efficiency for trace elements is quite high, varying between 60-80% (Geiger & Cooper, 2010).

- $PM_{0.1}$ (ultrafine particles, also termed 'nano-particles') are less than 0.1 µm (100 nm) in AED and are a direct consequence of man-made activity. They arise from various sources including diesel and gasoline combustion and because of their small size are able to penetrate into the alveoli and possibly the systemic circulation (Chin, 2015; U.S. EPA, 2010). Because of their increased surface-to-volume ration, they have increased solubility compared to larger size particles of the same composition (Geiger & Cooper, 2010). These particles have relatively short residency times in the atmosphere because they accumulate or coagulate to form larger particles (Kim, Kabir, & Kabir, 2015; WHO, 2006).

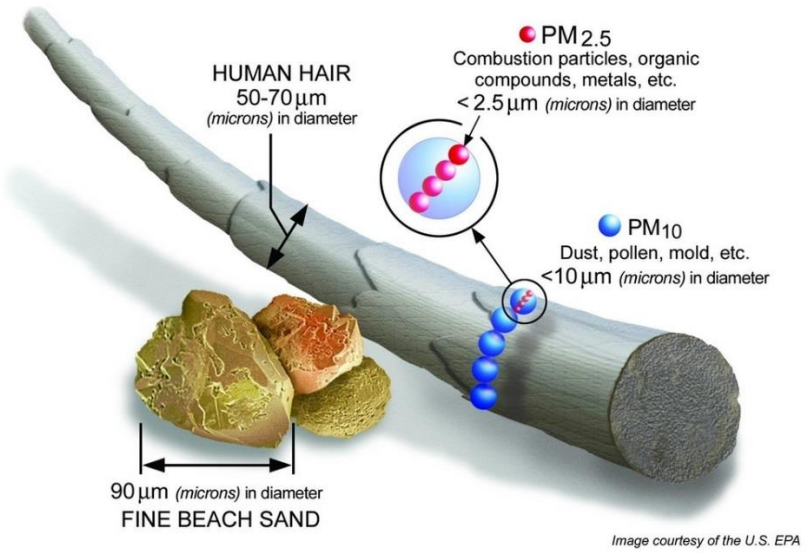


Figure 2.3: Schematic overview of relative size of particulate pollution, PM_{10} and $PM_{2.5}$ (United States Environmental Protection Agency (U.S. EPA), 2010). Retrieved from https://www.epa.gov/pm-pollution/particulate-matter-pm-basics.

Particulate matter has been identified as a significant hazard to human health and is undergoing an increasing level of scrutiny due to the mounting evidence of its potential contribution to health effects resulting from exposure. Studies that have used multi-pollutant models in their analyses in an attempt to distinguish the independent effects of each pollutant have found that estimates for effects of PM are robust and undergo minimal change when other pollutants are added to the model, implying that the majority of the effect is in fact due to the PM (Jalaludin & Cowie, 2012; Kelly & Fussell, 2015).

For both PM_{10} and $PM_{2.5,}$ the risk of negative health outcomes has been shown to increase with an increase in exposure, however $PM_{2.5}$ is being found to be a more significant contributor to a broad range of health effects (Kelly & Fussell, 2015; Keywood, Hibberd & Emmerson, 2017; Risom, Moller, & Loft, 2005). Their small size allows them to get deeper into the lungs and from there they can reach and trigger inflammation in the lung, blood vessels, the heart, and perhaps other organs (U.S. EPA, 2010). There is also a growing body of evidence suggesting that health effects may be better correlated to surface area than to particle size (HEI, 2013; Laumbach & Kipen, 2012). This makes the monitoring of smaller particles (such as $PM_{2.5}$) important, owing to the fact that they have a higher surface area to volume ratio.

Figure 2.4 gives a simplified representation of the interaction between various attributes of particles and their interactions in the human body. For example, as the particle size decreases, surface area, chemical reactivity, and potential toxicity increase. Particle clearance decreases as particles are less able to be cleared and the mechanism of deposition progresses from impaction for larger particles though to diffusion of very small particles. There is a change in the regions of the respiratory system affected and there is also a decrease in the magnitude of directional change and air flow through the system (Bakand, Hayes, & Dechsakulthorn, 2012).

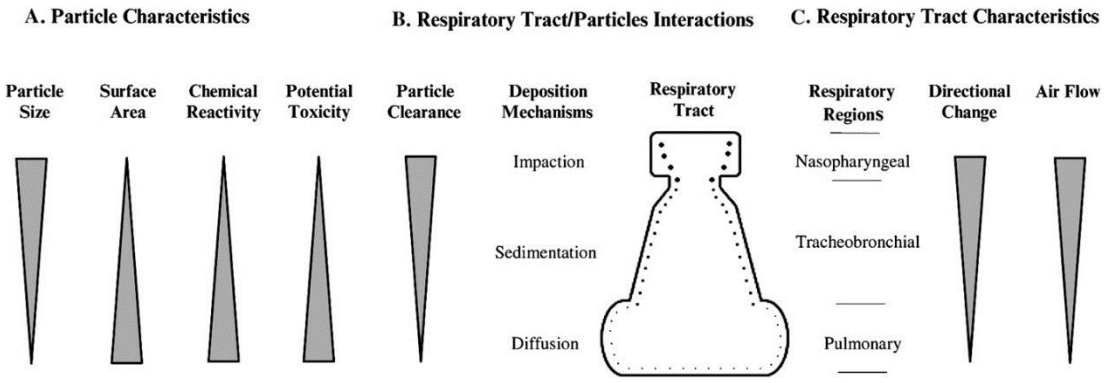


Figure 2.4: The interaction of particles with the human respiratory tract, (A) particle characteristics, (B) respiratory tract/particles interactions, (C) respiratory tract characteristics (Bakand, Hayes, & Dechsakulthorn, 2012, p.127.)

Although the risk to one individual at any single time is small, given the number of people continuously exposed, particulate air pollution imparts a huge burden to global public health and it is ranked as a very significant cause of mortality and morbidity. Although it would be expected that PM would pose a health risk mostly to the lungs and respiratory system, the overall evidence indicates that the majority of adverse effects are upon the cardiovascular system (Brook et al., 2010; Chin, 2015; Lee, Kim, & Lee, 2014). While the association between air pollution and CVD is now well established, the exact causal agents and the pathophysiological mechanisms are incompletely understood (Chin, 2015; Lee, Kim & Lee, 2014).

Many epidemiological studies, both Australian and international, have provided evidence that exposure to particulate matter (PM) in ambient air can lead to increased morbidity and mortality from both cardiovascular and respiratory disease (Loomis et al., 2013). Factors that may influence health effects include the chemical composition and physical properties of the particles as well as the size, mass concentration and duration of exposure.

With the possible exception of those in special filtered environments, all people are continuously exposed to particles to some extent. Exposure may be higher in urban and industrial areas due to an increase in the number of sources (vehicles and industrial emissions), however higher levels may also occur in natural environments following extreme weather events such as bushfires and dust storms. The large arid interior of Australia is a major source of global atmospheric dust. Drought, overgrazing and bushfires all contribute to dust storms that occasionally affect Australian cities (DSEWPC, 2011; Johnston, 2009; Johnston et al., 2011).

In addition to cardiovascular and respiratory impacts, there are many other possible health effects from exposure to particulates. These include toxic effects by absorption of the material into the blood for example metals such as cadmium, lead, and zinc; allergic or hypersensitivity reactions to some woods, pollen, grains, chemicals, bacterial and fungal infections such as Legionellosis, fibrosis from inhalation of asbestos and silica, cancer and irritation of mucus membranes from acids and alkalis (DSEWPC, 2010a, 2010b).

2.3.6.1 Coarse particulate matter (PM_{10})

PM_{10} is both a primary pollutant as emitted with the potential to cause adverse effects and also a secondary pollutant, formed through chemical reactions in the atmosphere. It consists of $PM_{2.5}$ and larger particles of mainly crustal or biological origin, including many aeroallergens such as pollen (Laumbach & Kipen, 2012). Typical sources of PM_{10} are bushfires, domestic wood heaters and dust storms (Keywood, Hibberd & Emmerson, 2017). Several authors have noted that PM_{10} derived from ambient biomass smoke such as from vegetation fires is associated more with respiratory rather than cardiovascular admissions (DWSWEPC, 2011; Johnston et al., 2007, 2011).

The pollutants associated with combustion including NO_2, CO, PM and SO_2, are all correlated so it is difficult for epidemiological studies to allocate observed effects to single pollutants and a pollutant-by-pollutant assessment would grossly overestimate the impact of each. PM_{10} is often used as an indicator of several sources of outdoor air pollution such as fossil-fuel combustion so may be over-represented in the literature (Jalaludin & Cowie, 2012).

2.3.6.2 Fine particulate matter ($PM_{2.5}$)

The finer particulate fraction of $PM_{2.5}$ particles are smaller than PM_{10} so can be transported further and persist in the atmosphere for longer. They can also penetrate deeper into respiratory system and are more hazardous to breathe (Keywood, Hibberd & Emmerson, 2017). Typical background concentrations are about 5 $\mu g/m^3$ but concentrations can increase rapidly under extreme conditions such as during bushfires, smog, dust storms and use of domestic wood heaters (Keywood, Hibberd & Emmerson, 2017).

Research has confirmed the links between $PM_{2.5}$ exposure and both short and long term respiratory conditions such as irritation of the airways, coughing and difficulty breathing, reduced lung function, aggravated asthma and chronic bronchitis as well as circulatory conditions such as irregular heartbeat, nonfatal heart attacks and also some cancers (Martuzzi et al., 2002; Schwartz & Neas, 2000; U.S. EPA, 2010). Those with pre-existing heart and respiratory diseases, children and the elderly are more vulnerable to these effects. Exposure to $PM_{2.5}$ was estimated to have contributed to 3.2 million premature deaths worldwide in 2010, due largely to cardiovascular disease as well as 223,000 deaths from lung cancer. Over half of these deaths occurred in China and East Asia (Arranz et al., 2014; Lee, Kim & Lee, 2014; Lim et al., 2012; Straif, Cohen, & Samet, 2013)

The 2015 Global Burden of Disease Study (Cohen et al., 2017) examined the burden of disease attributable to ambient air pollution from 1990 to 2015 and ranked ambient $PM_{2.5}$ as the fifth-ranking mortality risk factor in 2015. Over the 25 year period, deaths attributable to $PM_{2.5}$ exposure increased from 3.5 million (3.0 million – 4.0 million) in 1990 to 4.2 million (3.7 million – 4.8 million) in 2015. This represented 7.6% of total global mortality. Exposure to $PM_{2.5}$ was the cause of the loss of 103.1 million (90.8 million – 115.1 million) disability adjusted life-years (DALYs) in 2015, representing 4.2% of global DALYs with almost 60% occurring in Asia.

Current epidemiological evidence indicates that $PM_{2.5}$ is more potent for respiratory and cardiovascular disease compared with PM_{10} and that exposure to $PM_{2.5}$, from diesel engines or coal or biomass burning, is more closely related to adverse health effects such as premature mortality (Goldstone, 2015; Jalaludin & Cowie, 2012; Keywood, Hibberd & Emmerson, 2017; Laumbach & Kipen, 2012; Meng, Zhang, Yang, Yang, & Zhou, 2016; Morawska, 2007).

Studies conducted in Melbourne, Sydney and Brisbane have shown adverse health effects associated with exposure to $PM_{2.5}$ (Petroeschevsky et al., 2001; Simpson et al., 2005). There were strong associations between admissions for respiratory and cardiovascular disease, asthma (especially in children less than 14 years of age), chronic obstructive pulmonary disease (COPD) and even increases in daily mortality (all causes, respiratory and cardiovascular causes) and increases in fine particles concentrations with the strongest effects in all studies being found in children (asthma only) and the elderly (Environmental Protection Agency [EPA] Victoria, 2001; Morawska, 2007). Several other Australian and New Zealand studies have also been consistent with the above findings. Increases in the number of visits to emergency departments for children with respiratory illnesses including asthma and the elderly with cardiovascular conditions have been significantly associated with higher exposures of fine particulate matter (Erbas et al., 2005; Jalaludin et al., 2006).

A study of the short-term health effects of air pollution on daily mortality in Brisbane, Melbourne, Perth and Sydney, found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration had significant effects on total mortality. Simpson et al. (2005) estimated an increase in the daily total number of deaths of 0.9% (0.7% to 2.5%). This is quite low compared to a U.K. Department of Health Report by the Committee on the Medical Effects of Air Pollutants (COMEAP), (2009) which estimated that an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ caused an increase in mortality death rates by 6%. Correia et al. (2013) investigated the effect of air pollution control on life expectancy in the US from 2000 to 2007 focussing on levels of $PM_{2.5}$. The authors studied yearly average $PM_{2.5}$ levels and life expectancy in 545 counties and found that a decrease of 10 $\mu g/m^3$ in the concentration of $PM_{2.5}$ was associated with an increase in mean life expectancy of 0.35 years (SD=0.16 years, p=0.0333). This association was found to be stronger in more densely populated counties (Correia et al., 2013).

Elevated levels of $PM_{2.5}$ have long been associated with the incidence of premature mortality and morbidity outcomes (Dockery et al., 1993; Pope et al., 2002; WHO, 2006; Jerrett et al., 2005; COMEAP, 2011). Together with ozone, it is currently considered the most significant known causes of early mortality related to poor outdoor air quality (U.S. EPA, 2011). The U.S. EPA estimated that in 2010 there were approximately 160,000 premature deaths in the U.S. due to $PM_{2.5}$ exposure Fann et al. (2012) estimated there were between 130,000 and 340,000 $PM_{2.5}$-related early deaths in 2005 in the United States.

There is limited scientific evidence to differentiate health effects caused by different components of $PM_{2.5}$ (U.S. EPA, 2016). The 2013 WHO technical report from the Review of the Evidence on Health Aspects of Air Pollution (the REVIHAPP Project) (WHO, 2013) discussed metrics of PM other than just PM_{10} and $PM_{2.5.}$ Their evidence highlighted the health risks associated with other fractions such as black carbon (BC), secondary organic aerosols and secondary inorganic aerosols (Goldstone, 2015; Jansse et al, 2012). Black carbon is a major component of soot formed by the incomplete combustion of fossil fuels including biofuels, and biomass. It is emitted directly into the atmosphere as $PM_{2.5}$ in black smoke and is the most effective form of PM at absorbing solar energy. Per unit of mass in the atmosphere, it is able to absorb a million times more energy than carbon dioxide (U.S. EPA, 2016). The REVIHAAP project reported evidence linking BC with cardiovascular health effects and early mortality, for both short-term and long term exposure (Goldstone, 2015; Kelly & Fussell, 2015).

Most U.S. emissions of gaseous black smoke (BS) (52%) come from mobile sources, with 93% coming from diesel engines (93%). The other major source is biomass burning. Increased BS pollution has been found to be associated with increased mortality rates almost a month after exposure. Researchers Beverland, Carder, & Cohen (2014) studied death rates in relation to pollution concentrations over a 22-year period in the city of Glasgow, U.K., and found significantly higher mortality rates among residents at 13-18 and 19-24 days after increased exposure to BS.

Diesel particulate matter (DPM) (particles from diesel exhaust) is a distinct type of $PM_{2.5}$ that can account for up to approximately 90% of $PM_{2.5}$ in major cities (Chin 2016). It is a prime contributor to air pollution, and consists of both particulate and gaseous components. The particles have a carbonaceous core, surrounded by adsorbed organic compounds, including carcinogenic polycyclic aromatic hydrocarbons (PAHs) and nitro-PAHs, and small amounts of sulphate, nitrate, metals and other trace elements. Diesel emissions have been associated with an increased response to allergens and asthma prevalence (Williams, 2012). Although a significant proportion of ambient $PM_{2.5}$ is derived from diesel engines, the chemical composition of $PM_{2.5}$ can vary greatly depending on proximity to other sources such as power generation, industry, agriculture or aviation (Chin, 2015).

Hoek et al. (2013) conducted a review of air pollution studies focussing on long term exposure to $PM_{2.5}$ and cardio–respiratory mortality. Their summary of the data supported previous cohort studies that found the pooled effect estimate, expressed

as excess risk per 10 µg/m^3 increase in $PM_{2.5}$ exposure, was 6% (95% CI: 4%-8%) for all-cause mortality and 11% (95% CI:5%-16%) for cardiovascular mortality. Long-term exposure to $PM_{2.5}$ was more associated with cardiovascular mortality particularly ischemic heart disease than from other non-malignant respiratory diseases. They found differences in $PM_{2.5}$ health effects were most likely to be related to differences in particle composition, infiltration of particles indoors, population characteristics and methodological differences in exposure assessment and control of confounding factors. All-cause mortality was significantly associated with elemental carbon and NO_2 which are both indicators of combustion. They found little evidence for either an association between long term PM_{10} exposure and mortality, and differences in associations between genders. They also found a larger effect estimate for mortality related to $PM_{2.5}$ in subjects who were obese and those that had lower education (Hoek et al., 2013).

Broome et al. (2015) investigated the effects on health outcomes of reducing air pollution, specifically $PM_{2.5}$ in Sydney, Australia. Ambient levels in Sydney are low compared to levels commonly seen in the U.S.A. and Europe and rarely exceed Australian guideline values. The authors calculated that even a modest 10% decrease in $PM_{2.5}$ exposure would yield significant health effects. Over 10 years there would be approximately 650 (95% CI: 430-850) fewer premature deaths, a gain of 3500 (95% CI: 2300-4600) life years and 700 (95% CI: 450-930) fewer respiratory and cardio-vascular hospital visits.

2.3.6.3 Ultrafine particulate Matter ($PM_{0.1}$)

Ultrafine particles (UFPs) are nano-sized particles (NSPs) of irregular composition and size with an AED of less than 100 nm or 0.1µm (U.S. EPA, 2010). They may occur in single and agglomerate forms in ambient air (Castranova, 2011). The composition of UFPs is dependent on the actual source of particles and also any post-formation processes they may have undergone (Morawska et al., 2009). Properties will vary with the base material, particle size and shape, any surface charge and functionalisation, and rate of dissolution (Castranova, 2011).

UFPs are a ubiquitous component of ambient air from both natural (biogenic) sources including bushfires, sea, volcanic eruptions, viruses and pollen and anthropogenic sources including internal combustion engines, industrial processes such as smelting and welding (fumes), electricity generation and incinerators, cigarettes, wood-heaters and cooking (U.S. EPA, 2010). The main source of ambient UFP exposure by far is

motor vehicles (Kumar et al., 2014). Ultrafine particles are a major component of emissions near fires and tailpipes however may rapidly agglomerate into somewhat larger fine PM within a short distance from the point of release (Laumbach & Kipen, 2012).

UFPs can also be referred to as 'nanoparticles' (NPs) as by definition, they are in the same nano-size range. While both are usually defined as having an aerodynamic diameter of less than 100 nm or 0.1 µm, NPs are also defined by the International Standards Organisation as having three external dimensions measuring less than 100 nm or 0.1 µm (International Standards Organization/ Technical Specification [ISO/TS], 2015; Stanford University Environmental Health and Safety, 2017). NPs are also differentiated from UFPs in that they are precisely manufactured or engineered for industrial purposes, specifically for their material properties with a known shape, size, surface features and chemistry (Debia, et al., 2013; Evelyn, Mannick, & Sermon, 2002; Oberdörster, Oberdörster, & Oberdörster, 2005). The names have evolved historically so that most sources now differentiate the two by describing nano-size particles that have been intentionally engineered as either just 'nanoparticles' (NPs) or 'engineered nanoparticles (ENPs)' and those that are not engineered and have been produced either unintentionally or as an incidental by-product of combustion processes as UFPs (Evelyn et al., 2002; Oberdörster et al., 2005).

Nano-sized particles, such as UFPs can enter the body through absorption following skin contact, inhalation and ingestion from food and water (Oberdorster et al., 2005). They may enter the nervous systems via neuron transfer from the nasal or trachea-bronchial inhalation. Excretion may be through the skin via sweat and exfoliation, or lost through urine or faeces, depending on the exposure and uptake pathway (Kumar et al., 2014; Oberdorster et al., 2005). It is hypothesized that due to their size, once inhaled, UFPs can deposit deep in the respiratory tract in the alveoli of the lung where gas exchange occurs. They are then able to translocate within the body via lymph to blood and then to bone marrow, liver, kidney, spleen and/or heart, avoiding the body's protective phagocytosis process, potentially causing molecular changes in cells. Oxidative DNA damage, which is mutagenic and carcinogenic, has been seen after exposure to UFPs (Kumar et al., 2014; Vinzents et al., 2005).

Ultrafine and particularly nano-sized aerosols are of significant concern to health, as it has been shown that they are up to 1000 times more harmful than an equivalent mass (or dose) of larger particles of the same material (Bakand et al., 2012; Duffin et al., 2002). The 'Ultrafine particulate (UFP) hypothesis' states that UFPs are more toxic

than larger particles of the same composition because of their increased surface area and other characteristics. Although they contribute little to the total mass of PM, UFPs emitted by combustion sources are the major contributor in terms of the actual number of particles (HEI, 2013; Laumbach & Kipen, 2012). There is strong agreement in the literature that mass is not an adequate metric for evaluating exposure to UFPs and properties such as surface area and activity, particle number concentration, or fibre aspect ratio and length, should also be considered (Kumar et al., 2014).

Incomplete combustion emissions, particularly from diesel engines, produce mainly chemically complex UFPs which have carbon centres with surfaces contaminated with various types of metals and organics (including toxins and highly carcinogenic polycyclic aromatic hydrocarbons [PAHs]), usually around a biopersistent core (Morawska, Moore & Rostovski, 2004; Szewczyńska, Pośniak, & Dobrzyńska., 2013). Because of their combustion origin, NSPs such as diesel soot, welding fumes, carbon black and coal fly ash are commonly referred to as 'combustion derived' nanoparticles (CDNP) (Donaldson, 2006).

Because UFPs are so small, they are usually inhaled in large numbers, typically several million particles per breath. There is mounting evidence to support suggestion that CDNP, primarily from traffic, are responsible for a significant proportion of ill health in the population. Donaldson (2006) suggests that these particles are likely to be responsible for many of the adverse health effects that result from exposure to ambient airborne particles. The mechanisms of cell and tissue injury by CDNPs are expected to be diverse as they comprise a wide range of materials however a common mechanism of oxidative stress caused by surfaces, organics and metals associated with the particles has been defined (Stone, Johnston & Clift, 2007). This oxidative stress leads to inflammation which forms the plausible link between exposure and type of health effects. Donaldson (2006) suggests that CDNPs may cause acute effects in individuals already suffering chronic airway and heart disease and also lead to chronic effects that promote the development of cardiac disease and lung cancer as well as exacerbating disease in those already affected.

Ultrafine particles are able to infiltrate indoors through ventilation or other processes that allow the ingress of ambient air. Their concentration can be influenced by indoor activities such as cooking, painting, cleaning, and the number of occupants (HEI, 2013). In addition to ambient exposure, workplace conditions may result in much higher exposure concentrations particularly if workers are close to emissions sources (Cheng et al., 2010).

There has been much previous research into the role of UFPs in inducing oxidative stress leading to inflammation and resulting in exacerbation of pre-existing respiratory and cardiovascular disease. There is mounting evidence for positive correlations between levels of particulates and increased morbidity and mortality rates in both adults and children. Research has also identified a link between respiratory ill health and a number of UFPs (Stone, Johnston & Clift, 2007). Toxicological studies have shown that for low solubility, low toxicity materials such as titanium dioxide (TiO_2), carbon black and polystyrene beads, UFPs are more toxic and inflammogenic than fine particles. They were found to generate reactive oxygen species (ROS) more readily than larger sized particles, which leads to increased transcription of pro-inflammatory mediators via intracellular signalling pathways including calcium and oxidative stress (Stone, Johnston, & Clift, 2007).

2.3.6.4 Engineered nanoparticles (ENP)

Engineered nanoparticles are UFPs that are produced by the intentional manipulation of matter on the near-atomic scale to have very specific properties such as shape, size, strength, chemical reactivity or conductivity (Castranova, 2011). Relative surface area is one of the main factors that increases reactivity, strength and electrical properties and how nanoparticles behave depends on the surface area rather than the actual composition of the particle. ENPs are often designed to be catalytically active, which may increase their potential for causing adverse effects on human health (Castranova, 2011; Holbrook, Kline, & Filliben, 2010).

Industry is exploiting the unique properties of ENPs for commercial use with the development of nano-objects such as nanoparticles, nanotubes or nanoplates (Castranova, 2011; NNI, 2004). There are currently over 1600 commercially available nano-technology products available containing ENPs including food products, cosmetics, pesticides and electronics. In addition, there is increasing development of ENPs for medical applications (Kah & Hofmann, 2014; Project on Emerging Nano-technologies [PEN] Database 2014).

Due to the small size and low density of ENPs, worker exposure via inhalation is anticipated during production, use and disposal of nanoparticles (Maynard & Kuempel, 2005). There is also the potential for ENPs to be released into the environment due to industrial processes or poor handling adding to anthropogenic sources of ambient PM (Holbrook, Kline, & Filliben, 2010). Due to the widespread potential for workplace exposure to nanoparticles there is considerable research

investigating possible exposure through industrial processes routes and any associated health effects (Institute for Occupational Safety Germany (DGUV), 2017; Oberdörster et al., 2005).

2.4 Metals in ambient air

Exposure to metals in ambient air is capable of causing myriad human health effects, ranging from cardiovascular and pulmonary inflammation to cancer and damage of vital organs. Assessing risk for metals is quite difficult as some metals play an important role in human metabolic function. In the body metals undergo various processes and may be bio-transformed from one chemical species to another. As a result the metal ion responsible for the toxicity of a metal may persist in the body regardless of how the metal is metabolised (Geiger & Cooper, 2010).

It has been suggested that the metal components of PM may be more dangerous than other components and are actually responsible for many of the adverse health effects, even when they are present at the low concentrations found in ambient air (Geiger & Cooper, 2010). Chen & Lippman (2009) conducted review of the evidence regarding health effects resulting from inhalation of ambient air PM containing metals at contemporary concentrations. They noted that the most toxic source-mixture was residual oil fly ash which typically contains nickel and vanadium. These two metals were found to be particularly significant in terms of acute cardiac function changes and excess short-term mortality. They also found evidence of health effects of environmental exposure to lead and zinc in ambient air.

The U.S. EPA has designated nine metals as potentially hazardous; antimony, arsenic, beryllium, cadmium, cobalt, manganese, lead, nickel and selenium. These metals are regularly monitored against various U.S. guidelines and international standards. Of these, lead is the only metal that actually has a statutory ambient air standard in Australian Ambient Air Quality NEPM (NEPC, 2016) , the U.S.A. National Ambient Air Quality Standards (U.S. EPA), the WHO Air Quality Guidelines (2006) and the European Commission Air Quality Standards (2003).

As previously discussed, the aerodynamic size and composition of particles determine their behaviour in the respiratory system. Metals emitted by combustion processes generally occur in the fine $PM_{2.5}$ fraction whereas those that result from mechanical disruption of materials, such as crushing, grinding, evaporation of sprays,

or suspensions of dust from construction and agricultural operations are generally found in the larger PM_{10} fraction. These are primarily of crustal origin and include metals such as aluminium, zinc, and iron (Geiger & Cooper, 2010; Taiwo et al., 2014). Studies have shown that most of the toxic metals accumulate in the smaller fine and ultrafine fractions where they are particularly toxic and are considered to be the primary contributors to negative human health. When these metalliferous particles come into contact with lung tissue and cells, metal ions are released into the biological system promoting cellular oxidant generation and subsequent health effects. These possible health implications are one of one of the main reasons it is important to characterise the chemical composition of fine and ultrafine particles (Chin, 2015).

Several studies have conducted metal speciation on airborne PM. Valavanidis et al., (2006) measured trace metals and polyaromatic hydrocarbons (PAHs) adsorbed onto TSP and $PM_{2.5}$ in Athens, which is heavily polluted from dense traffic. The most commonly detected trace metals were iron (Fe), lead (Pb), zinc (Zn), copper (Cu), chromium (Cr), vanadium (V), nickel (Ni) and cadmium (Cd). Analysis showed the metals were traffic-related and higher levels of were found in the finer fraction. Enamorado-Baez et al. (2015) analysed aerosols in Seville, Spain for the 25 most common trace elements. They found significant temporal correlations between metals that had common sources such as crustal elements or anthropogenic metals, and that levels were related to the Atlantic air masses and Sahara Dust Intrusions. Lin, Chen and Huang (2005), characterised metals in PM collected near a busy road in southern Taiwan and found that crustal metals accounted for more than 90% of all of the particulate metals and that the nano-sized or ultrafine fraction contained more traffic-related metals such as Pb, Cd, Cu, Zn, barium (Ba) and Ni than the other size fractions.

Taiwo et al. (2014) conducted a study in the U.K. comparing the mass and size distributions of particulate components in ambient air from an industrial site and an urban background site. Their analysis showed that there was a predominance of fine, mainly traffic related, particles at the urban site whereas the particles collected at the industrial site were mainly in the coarse fraction. They found higher levels of several metals including chlorine (Cl), sodium (Na), potassium (K), calcium (Ca), magnesium (Mg), chromium (Cr), iron (Fe) and zinc (Zn), were higher at the industrial site due to marine influences as well as the presence of industry.

Metal speciation of PM is not frequently reported in Australian air quality studies and the only metal included in the AA NEPM is lead. Almost no measurement or

characterization of nanoparticles has been conducted in Perth or Western Australia, with only two studies in literature (Bertolatti & Rumchev, 2009; Rumchev, Ourangui, Bertolatti, & Spickett, 2007), conducted at a small number of sites, focusing on carbonaceous particles. Several international studies that have undertaken speciation (Enamorado-Baez et al., 2015; Valavanidis et al., 2006) stressed that estimating the airborne PM mass concentration, as well as individual chemical/metal speciation, is critical not only for comparing with recommended values, but also for identifying the major sources that affect a particular area. They noted that such source apportionment could provide an estimation of the PM contribution of various sources at the level of the receptor. This knowledge could help regulators and planners to develop more efficient emission reduction strategies to manage potential air quality issues.

Several authors have observed a high seasonal variability for heavy metal content in ambient air. Melaku, Morris, Raghavan, & Hosten (2008) collected samples of ambient air at a single site in Washington DC, U.S.A. for seven months and analysed the samples for arsenic, cadmium, chromium and lead. The ranges of concentrations were 0.8-15.7 ng/m^3 for arsenic, 1.5-30.0 ng/m^3 for cadmium, 16.8 –112.0 ng/m^3 for chromium and 2.9-123.0 ng/m^3 for lead. Peak values were typically observed during the summer months, except for cadmium and chromium. When compared to meteorological variables, results indicated a strong dependence on temperature.

2.5 Human health effects of exposure to air pollution

It has long been acknowledged that there is a relationship between poor ambient air quality and human health. The first WHO report investigating the potential damaging health effects that may result from exposure to air pollution was published almost 60 years ago (WHO, 1958). Other early reports (Pope, et al., 2002; Pope & Dockery, 2006) focussed on smoke and sulphur dioxide, photochemical generated smog (ozone, peroxyacids and peroxynitrates), secondary aerosols and hydrogen fluoride. At the time health effects were significantly underestimated and reported possible irritant effects ranging from lachrymation to pulmonary oedema Effects were categorised as ‘serious’ when concentrations were unusually high and ‘relatively minor and probably transient’, when there was mainly just irritation of mucus membranes at lower concentrations (Fuks et al., 2017; WHO, 2017b).

Ambient air pollution is a heterogeneous mixture of multiple pollutants originating from myriad natural and anthropogenic sources which may vary substantially over space and time (Loomis et al., 2013; Straif, Cohen & Samet, 2013). Likewise the health effects of exposure to air pollution are many and may vary depending on the concentration of the individual constituent pollutants and any interactions between them (Malmqvist et al., 2013).

The Harvard Six Cites Study (Dockery et al., 1993) is one of the best known early studies into the effects of ambient air pollution on mortality. It was a prospective cohort study that followed 8,111 white cases, aged between 25 and 74 years of age, from 6 cities across the United States. The cases were followed for between 14 and 16 years from 1974 or 1976 to 1991, a total of 111,076 person years. The study initially measured ambient concentrations of total suspended particulate matter (TSP), sulphur dioxide, ozone, and suspended sulphates and from 1984 onwards, data was also collected on fine and coarse particles. After adjusting for other health risk factors, the authors found statistically significant and robust associations between air pollution from fine, inhalable, and sulphate particles and mortality from lung cancer and cardiopulmonary diseases, but not other causes. As a result of this study some of the cities made significant efforts and reduced their levels of $PM_{2.5}$. A follow up report by Laden et al. (2006) found that in cities where PM concentrations had fallen substantially, mortality rates had also fallen substantially and in those cities where there was little improvement in PM concentrations, the mortality rate had stayed the same.

Research has shown that urban air quality is a significant cause of death and illness with statistically significant relationships between higher levels of selected pollutants and respiratory and cardiovascular disease and increased mortality. There is also clear evidence that exposure to even short periods of poor urban air quality can have serious adverse health effects on human health (Keywood, Hibberd & Emmerson, 2017; WHO, 2016). Studies and reports into health effects generally include respiratory, cardiovascular or cancer conditions and endpoints are usually emergency department presentations (Pereira et al., 2010), hospitalisations (Hinwood et al., 2006), or deaths (mortality) (Hinwood et al., 2004; Pope, Ezzati, & Dockery, 2009). It was estimated that urban air pollution was responsible for more than 3000 premature deaths in Australia in 2003, mainly in the elderly. Heart disease was the most common cause of death from long term exposure to air pollution. In addition, the authors also

noted that air pollution also exacerbates asthma and contributes to other respiratory illnesses in children and the elderly (DSEWPC, 2011).

Vulnerability and length of exposure are important factors that need to be considered in determining health outcomes. Many authors have noted that the effects of ambient air pollution are more pronounced among the elderly, children and people with pre-existing cardiovascular and respiratory conditions (Lee, Kim and Lee 2014; Rückerl, et al., 2012). It should be noted however that there is ample evidence that air pollution is also associated with adverse health outcomes in healthy people.

It is often assumed that the onset and exacerbation of respiratory conditions such as asthma is closely linked to daily exposure to pollutants. This does not, however, take into account the effect of chronic exposure to pollution over the previous days, weeks or even months previous to the presentation or admission (AIHW, 2010). While is it not possible to investigate all possible combinations of exposure patterns, many research studies into emergency department presentations and/or hospitalisations resulting from exposure to air pollutants include estimates of a lag effect, or effects that occurs between one to five days following exposure to a pollutant or mixture of pollutants (Pereira, 2010; Hinwood, 2006).

2.5.1 Respiratory health effects

Many studies have reported significant associations between high concentrations of the major air pollutants and respiratory problems such as coughs, bronchitis, asthma, and in severe cases, death. Short term associations between ambient pollutants, particularly PM and respiratory morbidity are already well established (Erbas et al., 2005). Pulmonary effects of PM in particular include increased respiratory symptoms, decreased lung function, increased incidence of chronic cough, bronchitis and conjunctivitis (Lee, Kim & Lee, 2014). Among other environmental factors, air pollution is being investigated as a possible contributor to worldwide increases in rates of asthma and COPD over the past several decades (DEE, 2017a). Evidence also suggests that air pollution may also contribute to the substantial burden from acute lower respiratory tract infections & possibly tuberculosis (Laumbach & Kipen, 2012).

In Chile, Franck, Leitte, & Suppan, (2015) assessed the association between exposure to airborne PM and other gaseous pollutants and hospital admissions due to respiratory illness. They found adverse relationships for CO, NO_2, PM_{10} and $PM_{2.5}$.

Using one-pollutant models, they found the most adverse pollutants were CO and PM_{10} followed by $PM_{2.5}$. Using two-pollutant models, they found that NO_2 persisted in most cases, followed by $PM_{2.5}$. They also found strongest effects appeared to occur either immediately or after a delay or lag period of one day, but effects were still seen for up to seven days. In Ho Chi Min City Phung et al. (2016) reported that 10 µg/m^3 increases in NO_2 and PM_{10} levels were significantly associated with an increases in the risk of respiratory hospital admissions from 0.7% to 8%.

2.5.1.1 Asthma

Over the last few decades childhood asthma has increased quite dramatically in many countries including Australia and New Zealand. During this same time period there has been an increase in urbanisation and exposure to air pollution as people spend more exposed to traffic and consequently traffic associated air pollutants (AIHW, 2010).

Morbidity associated with asthma is often underestimated, as frequently only severe events that result in hospitalisation are included in studies. This is further complicated by the fact that different jurisdictions may also have different policies for managing asthma exacerbations, such as whether or not a case is admitted and so becomes a hospitalisation rather than just an emergency department presentation (AIHW, 2010). It has been suggested that alternative indicators such as an increase in medication use, emergency department and general practitioner visits are probably a reasonable indication of moderate to severe morbidity for this group however this can also be problematic in that asthma medication can be used for other conditions and general practitioner visits may be routine and not asthma related (AIHW, 2010; Erbas et al., 2005).

Various studies have attempted to determine the magnitude of increase in pollutant levels that could lead to an increase in the incidence of asthma. In 2002**,** the National Environmental Protection Council (NEPC) report determined that each 10 µg/m^3 increase in the ambient concentration of $PM_{2.5}$ from various sources was associated with a 3% increase in exacerbations of asthma (Johnston, et al., 2002). A later report by the Australian Institute of Health and Welfare (2010) on monitoring the impact of air pollution on asthma in Australia noted that while there had been improvements in air quality reporting, monitoring and equipment over time there was limited monitoring of the impact of air pollution on the health of asthma sufferers. The Report

recommended increased monitoring of effects and further research to determine the proportion of asthma exacerbations due to air pollution.

Studies into the prevalence of asthma have reported an increase in high-income countries, particularly in the severity of adult asthma (Andersen et al., 2012). The prevalence of asthma in older adults is 6-10% in high-income countries, and the economic burden associated with hospital care, medications and years of lost productivity due to morbidity and mortality is substantial. This burden is expected to escalate due to an increase in the number of older people with asthma and enhanced longevity. Andersen et al. (2012) followed 57,053 cases for 35 years in Denmark and found that long-term exposure to traffic-related air pollution increased the risk for asthma hospitalisation in older people and that people with previous asthma or COPD hospitalisations were the most susceptible.

A U.S. study of 861 children with asthma living in inner city communities in the US found that even though all pollutant levels measured were below the US National Ambient Air Quality Standards, short-term increases in air pollution concentrations were associated with adverse respiratory health effects (O'Connor et al., 2008). Specifically they found that higher 5-day mean concentrations of NO_2, SO2 and $PM_{2.5}$ were associated with significantly decreased lung function and an increase in asthma related missed school days and higher NO_2 symptoms were associated with an increase in asthma symptoms. The association with NO_2 suggests that asthma may be causing excess morbidity (O'Connor et al., 2008). Strickland et al. (2010) investigated pollutant dose-response on children with asthma and found that even at relatively low ambient concentrations, traffic related sources of ozone and primary pollutants independently contributed to the number of ED visits for paediatric asthma.

A study by Silverman & Ito (2010) investigated childhood asthma in New York City and reported severe diverse health effects to air pollutant exposures even below the currently accepted standards. They found warm weather patterns of ozone and $PM_{2.5}$ disproportionately affected children with asthma and appeared to be responsible for severe attacks and hospitalisations and that individuals appeared to be at risk even when pollutants were below the NA AQS. Susceptibility to both pollutants was found to be age-dependent with children at highest risk of non-critical hospitalisations and ICU admissions.

During a period of almost continuous bushfires in Australia, Johnston et al. (2002) found a significant increase in asthma presentations to ED departments with each 10

µg/m^3 increase in PM_{10} concentration. The strongest effect was seen when PM_{10} was greater than 40 µg/m^3 (adjusted relative risk 2.39, 95% CI: 1.46 - 3.90) compared to days when PM_{10} was less than 10 µg/m^3. The authors concluded that airborne particles from bushfires should be considered as harmful to human health and stressed the need to manage smoke from bushfires in urban areas. A NEPC report suggests that each increase of 10 µg/m^3 increase in ambient concentration of respirable particulates is associated with a 3.0% increase in exacerbations of asthma (Johnston et al., 2002).

A study into emergency department (ED) presentations for asthma in children and ambient air pollution levels was conducted in the Perth metropolitan area over a 5 year period from 2002-2006 (Pereira et al., 2010). They found that pollutants showed a clear seasonal cycle with peak NO_2 and CO occurring in the winter months and peak PM_{10} and O_3 occurring in the summer months. They also found significant correlations between daily concentrations of PM_{10} and O_3 as well as between CO and NO_2. PM_{10} was negatively correlated with CO and NO_2. They found there was statistically significant increases in the odds of ED presentations observed only for NO_2 (21% increase per 1 ppb NO_2) and CO (5 fold increase per 1 ppm of CO), with a 1-day lag. They stratified for age and gender and found children 0-4 years of age were most affected, and most cases occurred in boys.

Erbas et al. (2005) investigated the effect of regional ambient air pollutants on ED childhood asthma presentations across four areas of Melbourne over a two year period from 2000-2001. They found consistent associations between childhood ED presentations and regional concentrations of PM_{10}. NO_2 and ozone were only found to be associated in one of the four districts.

While it is generally accepted that ambient air pollution may exacerbate pre-existing asthma, there is increasing evidence that long-term exposure to TRAP, particularly nitrogen dioxide may contribute to the development of new onset asthma in both children and adults by causing oxidative injury to the airways, leading to inflammation, remodelling and increased risk of sensitisation (Guarnieri & Balmes, 2014). This is significant especially for young children growing up in economically disadvantaged areas who are often at increased risk of exposures to high level of TRAP.

2.5.1.2 Chronic obstructive pulmonary disease (COPD)

Chronic obstructive pulmonary disease (COPD) is a respiratory health condition characterized by irreversible airflow obstruction. Exacerbations of COPD contribute greatly to increased morbidity, mortality, diminished quality of life and health care costs. In England, there are an estimated 100,000 COPD related hospital admissions each year and there is an estimated £600 million per year is spent by the U.K. National Health Service to provide care for sufferers (Sarran, Agnew, & Davis, 2010). Patients are alerted in advance of high risk periods (based on air quality and meteorological data) so that they can take appropriate self-care measures to stay out of hospital.

A large U.K. study looked at the role of ambient air pollution on a cohort of 812,063 patients aged from 40-89 without COPD diagnosis. During the following 5 year period, 16,035 developed COPD. The authors (Atkinson et al., 2014) looked at levels of PM_{10}, $PM_{2.5}$, NO_2, SO_2 and O_3 within one kilometre using dispersion models and found limited inconclusive evidence for associations between air pollution and COPD incidence. They concluded however that given the ubiquitous nature of the exposure and the substantial burden of COPD on the healthcare system, further investigation was warranted. In contrast a study carried out on 1,050 hospital admissions in Norfolk, U.K. between January 2006 and February 2007 found that there was a 22% increase in the odds of admission for each 10 $\mu g/m^3$ increase in NO2 and a 2% increase in the odds of admission when CO was increased by 10 $\mu g/m^3$. There were no associations observed for ozone or particulates (Sauerzapf, Jones & Cross, 2009).

Other recent studies report moderately strong positive associations between exposures to several air pollutants and COPD-related emergency department visits and hospital admissions. A study by DeVries, Kriebel & Sama (2016) investigating the effects of air pollutants on a group of COPD patients in the U.S.A. found that even though pollutant levels were below current air quality guidelines, the patients suffered an increased risk of COPD exacerbation following short-term exposures to increased concentrations of SO_2 and NO_2.

2.5.2 Cardiovascular health effects

While each of the six criteria pollutants has been linked to CVDs such as hypertension, myocardial infarction, stroke, heart failure and cardiac arrest, the evidence for an association between PM exposure and CVD far exceeds that for other components. Ambient particulate air pollution was the ninth leading cause of DALYs

worldwide, the fourth leading cause of DALYs in East Asia, and was responsible for 3.2 million deaths worldwide in 2010. The effect estimates of air pollution on cardiovascular complications such as myocardial infarction are higher than the effects on lung disorders such as lung cancer and even on all-cause mortality, resulting in higher population attributable risk (Chin, 2015).

Studies from across the world have shown that both short and long term exposures to particulates are associated with a range of cardiovascular diseases (Brook, 2008; Chin, 2015; Lee et al., 2014; Leiva et al., 2013; Polichetti, 2009). Acute exposure is linked with an increased incidence of myocardial infarction, arrhythmias, strokes, heart failure and all-cause mortality. Chronic exposure is also linked to all-cause mortality to a significantly greater degree than acute exposure (Chin, 2015) and has also been shown to enhance the chronic genesis of atherosclerosis. Although the predominant cardiovascular complication of exposure to air pollution is ischaemic heart disease, statistically significant associations are also seen for arrhythmias, heart failure and cardiac arrest (Lee , Kim & Lee, 2014).

Polichetti et al. (2009) conducted a review of the impacts of the different sizes of PM on CVD and suggested a correlation between the 'short term' and 'long term' effects of PM exposure and the onset of cardiovascular disease. They found that adverse health effects depend not only on the level of PM concentration in the air but also on the composition of the PM (Polichetti, et al., 2009). Lee, Kim and Lee (2014) undertook a review of current evidence and concluded that even though there is a close association between PM concentrations and adverse health effects such as heart disease, stroke, blood pressure, and cardiovascular disease, epidemiological studies consistently exhibited a stronger correlation of adverse health effects with $PM_{2.5}$ than PM_{10}.

Leiva et al. (2013) examined the relationship between $PM_{2.5}$ pollution and stroke, which is the second leading cause of mortality and leading cause of morbidity in Chile and the rest of the world. They examined data for 33,624 stroke admissions between 2002 and 2006 and metropolitan air quality data. They found that for every 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration, the risk of emergency hospital admissions for cerebrovascular cases increased by 1.29% (95% CI: 0.52% - 2.03%). Also in Chile, Franck, Leitte, & Suppan, (2014) assessed the association between exposure to airborne PM and other gaseous pollutants and hospital admissions due to various types of cardiovascular illness. They found adverse relationships for CO, NO_2, PM_{10} and $PM_{2.5}$, but not ozone, and that the strength of the effect and any lag period for the

onset of symptoms depended on the actual pollutant and the disease group. Using one-pollutant models, they found the most adverse pollutants were NO_2, PM_{10} and $PM_{2.5}$, followed by CO. Using two-pollutant models, PM_{10} persisted in most cases and strongest effects appeared to occur after a delay or lag period of two days.

Chen et al. (2014) studied the relationship between $PM_{2.5}$ and four constituents; nitrate, sulphate, organic carbon and elemental carbon and stroke hospitalisations in Taiwan from 2004 to 2008. They investigated 12,982 ischemic stroke and 3,362 haemorrhagic stroke admissions. For haemorrhagic stroke, the strongest relative risks were found with nitrate and elemental carbon, 1.19 (95% CI: 1.07, 1.32) and 1.08 (95% CI: 1.02, 1.15) respectively. For ischemic stroke, increased relative risks were observed for both organic and elemental carbon, 1.21 (95% CI: 1.07, 1.36) and 1.18 (95% CI: 1.06.1.31) respectively. $PM_{2.5}$ and organic carbon were also associated with increased relative risks of emergency room visits for ischemic stroke in female patients and those over 65 years. The authors concluded that it was the constituents of $PM_{2.5}$ rather than the mass that was more closely related to admissions for haemorrhagic stroke and both $PM_{2.5}$ and its constituents are associated with ER visit for ischemic stroke in the over 65 years and women.

Lin et al. (2016) investigated the association of PM pollution with different size particles and chemical constituents with mortality from ischemic and haemorrhagic stroke in Ghangzhou, China. They found significant associations with various PM fractions, with larger magnitude for the smaller particles. For the $PM_{2.5}$ constituents, organic carbon, elemental carbon, sulphate, nitrate and ammonium were significantly associated with stroke mortality. They also found that it was haemorrhagic, rather than ischemic stroke that was significantly associated with PM pollution.

Barnett et al. (2006) examined the effects of CO, NO_2, ozone and PM on hospital admissions for seven different categories of cardiovascular disease – arrhythmia, cardiac disease, cardiac failure, ischaemic heart disease, myocardial infarction, stroke and total CVD. The study concentrated on admissions in the ‘elderly’ (greater than 65 years) in both Australia (Brisbane, Canberra, Melbourne, Perth, Sydney) and New Zealand (Christchurch and Auckland) during the period 1998 – 2001. They found that in both countries, increases in ambient concentrations of all pollutants except ozone, had significant associations with increases in hospitalisations for all categories of CVD, except arrhythmia and stroke. These associations generally occurred in the greater than 65 years age group compared to the younger ‘adult’ age group (15-64 years). CO had the most consistent association with a 0.9 ppm increase in CO giving

a 2.2% increases in admissions for total cardiovascular disease, a 2.8% increase for all cardiac disease, a 6.0% increase for cardiac failure, a 2.3 % increase for ischaemic heart disease and a 2.9% increase for myocardial infarction. Many of these associations were found at concentrations below normal air quality health guidelines. The authors (Barnett et al., 2006) noted that it was difficult to separate the associations for particular pollutants as there were common emission sources for CO, NO_2 and PM such as road traffic emissions. In Ho Chi Min City, Phung et al. (2016) reported that due to heavy traffic, residents were vulnerable to high levels of pollution. For 10 $\mu g/m^3$ increases in the concentrations of NO_2 and $PM_{10,}$ the risk of CVD hospitalisations increased from 0.5% to 4%.

2.5.2.1 Mechanisms of effect

Both acute and chronic exposure to PM can lead to a variety of specific cardiovascular effects (Gonzales-Flecha, 2004). Associations have been reported with exacerbation of ischaemic heart disease, heart failure, cerebrovascular disease, deep venous thrombosis, hypertension, and cardiac arrhythmias, with varying degrees of evidence supporting these associations. Various studies have demonstrated several mechanisms by which particle exposure may both trigger acute events as well as prompt the chronic development of cardiovascular disease Analysis of the specific molecular and cellular mechanisms involved reveals diverse and overlapping effects in which systemic inflammation, oxidative stress, neuro-humoral and epigenetic modifications contribute in a variety of ways (Manke, Wang, & Rojanasakul, 2013; Pope, 2000). There are three distinct hypotheses to explain the association between PM exposure and cardiovascular disease with varying degrees of evidence and consensus (Chin, 2015; Polichetti, 2009) (Figure 2.5).

1. Inflammatory Response: This is the best supported hypothesis and asserts that PM entering the lungs provokes an inflammatory response that promotes localised and systemic oxidative stress and inflammation which may progress to a variety of pathological processes related to cardiovascular disease.

2. Autonomic dysfunction: This hypothesis asserts that pulmonary exposure to PM leads to activation of the lung autonomic nervous system (ANS) causing an imbalance, leading to pathological alterations in vasoconstriction, endothelial dysfunction, hypertension, platelet aggregation, tachycardia, increased heart rate variability and increased arrhythmia potential.

3. Direct entry of toxicant: This hypothesis asserts that PM is inhaled through the lungs and enters the circulation where it may directly interact with tissue components to promote vasoconstriction, endothelial dysfunction, atherosclerosis, hypertension, platelet aggregation, systemic oxidative stress and inflammation. Evidence has also been accumulating that PM exposure can lead to chemical modification of DNA, resulting in epigenetic dysregulation of gene expression.

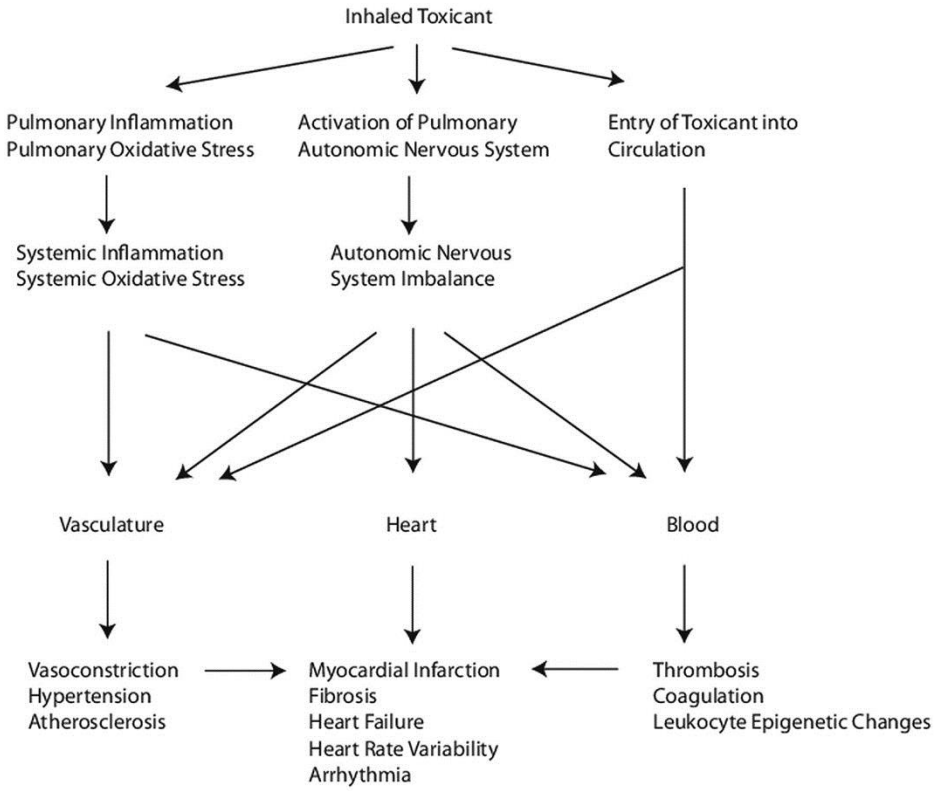


Figure 2.5: Working model of how air pollution exposure promotes adverse cardiovascular effects (Chin et al., 2015, p. 255).

2.5.2.2 Hypertension

Exposure to ambient air pollution is known to be related to a higher risk for several cardiovascular conditions. It has been suggested that it may also be linked to hypertension which is a major risk factor for premature morbidity and mortality (Fuks et al., 2017). It has been shown that short-term increases in air pollution levels can raise blood pressure rapidly although there is little evidence for long-term effects on hypertension. Increases in systolic and/or diastolic blood pressure have been reported in cross-sectional studies, however these finding are not consistent across all studies (Fuks et al., 2017).

Lee, Kim, & Lee (2014) reported a positive association between particulate pollution and a significant rise in blood pressure in selected cities across the United States. In a study conducted in Boston, they reported that for every 10.5 μg/m^3 rise in $PM_{2.5}$ levels, there was a 2.8 mmHg increase in systolic blood pressure (SBP) and a 2.7 mmHg rise in diastolic blood pressure (DBP). They also reported a 5.2 mmHg increase in SBP with increased $PM_{2.5}$ in a study conducted in Detroit.

Giorgini et al. (2016) reviewed recent literature that examined high arterial blood pressure (BP) and elevated levels of $PM_{2.5.}$ They reported epidemiological findings that PM causes small but significant increases in BP in both the short and long term. They also found prolonged exposure could increase both prevalence and incidence of hypertension. The authors looked at several markers and established more robust results with $PM_{2.5}$ and concluded that available evidence suggests causal relationship between $PM_{2.5}$ and hypertension. They noted that differences in results may be due to differences in spatial and temporal variability in sources and composition.

Fuks et al. (2017) investigated whether traffic-related air pollution (and noise) was associated with incident hypertension in seven cohorts of the European Study of Cohorts for Air Pollution Effects (ESCAPE). They found long term residential exposures to air pollution (and noise) are associated with increased incidence of self-reported hypertension but not measured hypertension. Self-reported hypertension was positively associated with increased levels of $PM_{2.5}$ only. Of the 41,072 participants being followed, there were 6,207 incident cases within five to nine years. They estimated an increase of 22% per 5 μg/m^3 increase in $PM_{2.5.}$

2.5.2.3 Ischemic heart disease / Coronary artery disease

Ischemic heart disease (IHD) or coronary artery (heart) disease is a group of diseases that includes atherosclerosis, angina, myocardial infarction and sudden cardiac death (Pope et al., 2006). Recent evidence suggests long-term exposure to particulate air pollution contributes to several of the mechanisms that are considered to lead to CVD such as pulmonary and systemic oxidative stress, inflammation, progression of atherosclerosis, and risk of ischemic heart disease and death (Chin, 2015). Short-term exposure may contribute to complications of atherosclerosis such as plaque, thrombosis and acute ischemic events. Pope et al. (2009) conducted a study and found short-term PM exposure was significantly associated with acute IHD events and found it contributed to acute coronary events, especially among patients with

underlying coronary artery disease. Patients with clean coronaries were less susceptible.

Thurston et al. (2016) investigated the causal characteristics and sources underlying past association between long-term $PM_{2.5}$ and IHD mortality. They found that long-term $PM_{2.5}$ exposures from fossil fuel combustion, especially coal burning and diesel traffic, were associated with an increase in IHD mortality in the US. Results suggest $PM_{2.5}$ related mortality associations can vary greatly by source and that the largest IHD health benefit per $\mu g/m^3$ from $PM_{2.5}$ air pollution control may be achieved via reduction of fossil fuel combustion exposures, especially from coal–burning sources. They found the risk for coal combustion five times higher than particulate matter mass in general. On a mass basis ($\mu g/m^3$) they found diesel traffic-related elemental carbon soot was also significantly associated with IHD mortality. $PM_{2.5}$ from wind-blown soil and biomass combustion were both also associated with IHD mortality but less so.

Chen et al. (2005) reported on a multi-decade study conducted on women who lived in areas of California that had high concentrations of both coarse and fine particulates. It was found that they had a higher risk of developing and dying from coronary heart disease and that PM_{10}, $PM_{10\text{-}2.5}$, and $PM_{2.5}$ were all associated with an increased risk of fatal heart disease, especially in older women, with the effect strongest for $PM_{2.5}$. Several other studies in the US have also reported an association between $PM_{2.5}$ exposure and hardening of the arteries (Künzli et al., 2005), birth weight and infant mortality (Woodruff, Parker, & Schoendorf, 2006) and an increased relative risk for all-cause mortality, deaths from ischemic heart disease, and lung cancer deaths (Jerrett et al., 2005).

There has been suggestion that increases in levels of various pollutants, especially $PM_{2.5}$ may increase the risk of out-of-hospital cardiac arrests (OHCA). A 2013 study conducted in Melbourne investigated the relationship between the incidence of OHCA and ambient levels of $PM_{2.5}$ (Straney et al., 2013, 2014). Using a case-crossover study design the authors found that a 4.26 $\mu g/m^3$ interquartile range (IQR) increase in $PM_{2.5}$ over 2 days (lag 0–1 days) was associated with a 3.6% (95% CI: 1.3% - 6.0%) increase in risk for an OCHA. A similar study was then conducted in Perth using pollutant data from the DER and OCHA data from incidents attended by St John Ambulance (WA) paramedics (Straney et al., 2013, 2014). In Perth they found that an IQR increase in the 24-h mean level of $PM_{2.5}$ was associated with an even higher 9.8% (95% CI: 3.4% –16.2%) increase in the risk of OHCA. They also found that in the

same period, an IQR increase in the 12-h concentration of CO was associated with a 4.0% (0.6–7.4%) increase in the risk of OHCA.

Dennekamp et al. (2015) measured association between OCHA and forest fire exposures in a large city (Melbourne) during a severe forest fire season 2006-2007. The authors found there was an association between exposure to forest fire smoke and an increase in the rate of OCHA in men only. They also found that over 12 days of the 2006/2007 fire season period there were 174 fire hours during which Melbourne's air quality was affected by forest fire smoke. During this time there were 23.9 (95% CI: 3.1 – 40.2) excess OCHAs estimated to have occurred due to elevations in $PM_{2.5}$ with a 48 hour lag period most common.

A study conducted in England & Wales examined over 400,000 myocardial infarction events from the Myocardial Ischemic National Audit Project (MINAP) database which contains data on two million CVD emergency department admissions and over 600,000 cardiovascular related deaths (Milojevic et al., 2014). The data was linked with daily mean concentration of CO, NO_2, $PM_{10,}$ $PM_{2.5}$ and SO_2.and O_3 using lags up to 4 days and adjusted for temperature and day of the week. They found short term exposure to NO_2 and $PM_{2.5}$ had adverse effects on non-MI outcomes, such as arrhythmias. There was no clear evidence for pollution effects on STEMIS (ST-Elevated myocardial infarction) (very serious heart attack where major arteries carrying oxygen to the heart are blocked) and stroke. The strongest associations with air pollution were observed with selected non-MI outcomes. Experimental studies with known controlled pollution mixes are needed to help elucidate mechanistic pathways (Milojevic et al., 2014).

Meng et al. (2016) investigated the harmful effects of $PM_{2.5}$ and its association with acute coronary syndrome (ACS) (including myocardial infarction). ACS is one of the most common forms of coronary heart disease and is a clinical syndrome caused by acute ischaemia and commonly includes unstable angina and different types of myocardial infarction. $PM_{2.5}$ induced health effects and ACS arise through multiple mechanisms as discussed in 2.5.2.1.

In 2013, Shah et al. (2013) conducted a meta analysis examining associations between air pollution and heart failure and concluded that air pollution has a close temporal association with heart failure hospital admission and heart failure mortality. They reviewed 195 articles in-depth and found that overall heart failure hospitalisation or death was associated with increases in CO (3.52% per 1 ppm: 95% CI: 2.52, 4.54),

sulphur dioxide (2.36% per 10 ppb; 95% CI: 1.35, 3.38) and nitrogen dioxide (1.70% per 10 ppb; 95% CI: 1.26, 2,16), $PM_{2.5}$ (2.12% per 10 µg/m^3, 95% CI: 1.42, 2.82) and $PM_{10,}$ 1.63% per 10 µg/m^3, 95% CI: 1.20, 2.07). There were no associations observed with ozone (0.46% per 10 ppb; 95% CI: -0.10, 1.02). The strongest associations were found on the day of exposure, rather than after a lag period. The authors estimated that a mean reduction in $PM_{2.5}$ of 3.9 µg/m^3 would prevent 7,978 heart failure hospitalisations per year.

2.5.3 Carcinogenic effects

While ambient air pollution has long been known to increase risks for a range of diseases, primarily respiratory and cardiovascular, there is mounting evidence that ambient air pollution may contain carcinogenic chemicals (Loomis et al., 2013; Straif et al., 2013) and exposure can also lead to cancer in humans (IARC, 2013). In the WHO Global Burden of Disease Project it was estimated that urban air pollution (measured as PM) was responsible for mortality attributable to cancers of the trachea, bronchus and lungs (Cohen et al., 2017). WHO data indicates that in 2010 there were 223,000 deaths worldwide from lung cancer that resulted from exposure to ambient air pollutants (Straif et al., 2013).

In 2013 the International Agency for Research on Cancer (IARC) working group classified both outdoor air pollution and PM as Group 1 carcinogens. This means that there is sufficient evidence available from human and experimental animal studies as well as strong mechanistic evidence to be able to state that exposure to outdoor air pollution and PM causes cancer in humans. This was consistent with the increased risk of lung cancer consistently observed in cohort and case-control studies including millions of people and many thousands of lung cancer cases from Europe, North America and Asia. Notably many of these studies were done in areas where the annual mean level of $PM_{2.5}$ range from 10-30 µg/m^3 which represents the lower third of exposures worldwide (IARC, 2013).

In terms of types of cancer, they specifically noted that outdoor air pollution caused lung cancer and that there is an increased risk of bladder cancer. There was also an increased risk of lung cancer observed in areas where $PM_{2.5}$ was less than current health-based guidelines (IARC, 2013). In the past the IARC has also evaluated many individual chemicals and mixtures that occur in air pollution such as diesel and

solvents but this is the first time that outdoor air has been classified as carcinogenic (Loomis et al., 2013; Straif et al., 2013).

There is evidence that real-world exposures to ambient air pollution, in several species are associated with increases in genetic damage, including cytogenetic abnormalities, mutations in both somatic and germ cells, and altered gene expression; all which may be linked to increased cancer risk in humans. Exposure to polluted air in occupational settings or urban and industrial areas is also associated with changes in expression of genes involved in DNA damage and repair, inflammation, immune and oxidative stress response (Loomis et al., 2013; Straif et al., 2013).

2.5.4 Health effects in children

There are many studies on the respiratory effects of exposure to air pollutants in children as they are a particularly vulnerable group. Their lungs are immature, their immune system is still developing and they have a higher ventilation rate so inhale more pollutants per surface area than adults. They also tend to spend more time outdoors and are usually be involved in more vigorous activity, further increasing their ventilation rate (Rückerl et al., 2011).

The Australian Child Health and Air Pollution Study (ACHAPS) was a cross-sectional study undertaken to examine the respiratory health of 2,860 Australian schoolchildren in response to concerns that exposure to air pollutants may reduce their lung volume which may have longer term consequences for respiratory health (Williams, 2012). The study showed consistent evidence of adverse effects of short and long term exposure to NO_2 such as increased asthma like symptoms (especially wheezing), increased airway inflammation and reduced lung volumes. There was no evidence of heightened response in atopic subjects (except for SO_2). The results also demonstrate that the consequences of NO_2 exposure is not typically asthma but is more likely to be more non-specific PM exposure showed varied results, depending on gender and atopic status. There was no overall increase in current respiratory symptoms with elevated PM_{10}, however $PM_{2.5}$ showed not only an increase in current symptoms but also the likelihood of an increased risk of lifetime wheezing, asthma, and asthma medication. There were no adverse effects associated with increased ozone levels. Elevated SO_2 levels were associated with adverse effects on lung function measures, which was worse if the children were atopic (Williams, 2012).

Recent studies have shown that efforts to improve air quality can have a positive effect on children's respiratory health which will have long-term public health impacts. Gauderman et al. (2015) followed 2,120 children in three separate cohorts in California for four years each (from 11-15 years of age) and found that long term improvements in air quality (declining NO_2, $PM_{2.5}$ and PM_{10}) were associated with statistically and clinically significant positive effects on lung-function growth in both asthmatic and non-asthmatic children.

2.5.5 Health effects of exposure to low levels of air pollution

There is increasing evidence that shows adverse human impacts occurring from exposure to lower concentrations air pollution than previously regarded as hazardous. Many of these studies demonstrate health effects well below current standard levels or regulatory limits for air pollutants (Andersen et al., 2012; Desqueyroux et al., 2002; DeVries, Kriebel, & Sama, 2016; Keywood, Hibberd & Emmerson, 2017; Malmqvist et al., 2013; Moshammer et al., 2006; Schelegle et al., 2009; Shi et al., 2016; Strickland et al., 2010).

Strickland et al. (2010) found that even at relatively low ambient concentrations, ozone and primary pollutants from traffic sources such as NO_2, SO_2, $PM_{2.5}$ and PM_{10} were independently associated with emergency department (ED) visits for childhood asthma in metropolitan Atlanta, U.S.A. These associations tended to be greater for concentrations on the day of the ED visit and reinforced the need for continued evaluation of EPA's national ambient air quality standards. Desqueyroux et al. (2002) investigated the short-term effects of low-level air pollution on respiratory health of adults who were suffering moderate to severe asthma in Paris. They found significant health effects despite low ozone and PM_{10} concentrations never reaching standard values.

Andersen et al. (2012), Kim et al. (2011) and Schelegle et al. (2009), all conducted similar studies on healthy young adults investigating whether airway effects occurred after prolonged exposure to levels to ozone below the current standard. Schelegle et al. (2009) found that exposure to 0.07 ppm ozone for five 6.6 hour sessions (in a chamber undertaking variable patterns of moderate exercise) caused a significant decrement of lung function FEV_1 and an increase in airway inflammation in the airways. Kim et al. (2011) and Anderson et al. (2012) found similar decreases in FEV_1 after exposing subjects to six 6.6 hour sessions of an even lesser exposure of 0.06

ppm ozone. They also found there had been significant effects on pulmonary function and airway inflammation in healthy young adults following exposures below the current National Ambient ASQ standard for ground-level ozone of 75 ppb (0.075 ppm) (U.S. EPA).

Schwartz, Bind, & Koutrakis, (2017) conducted a study in Boston, U.S.A., estimating the effects of low–level local air pollution on daily deaths. They concluded that there was a causal association of $PM_{2.5}$, BC and NO_2 with daily deaths at concentrations below U.S. EPA standards. They also noted that the association for $PM_{2.5}$ still existed when all the days that the $PM_{2.5}$ concentration was greater than 30 $\mu g/m^3$ were removed from the model. They estimated that the attributable risk exceeded 1,800 deaths from 2000-2009, indicating that important public health gains can be achieved from increased traffic related emission control. Shi et.al (2016) also investigated whether any associations existed between short and long term exposure to low-concentration $PM_{2.5}$ and mortality. Their study was conducted in the New England area of the U.S.A. from 2003 to 2008 and concentrated on the over 65 year age bracket. They found exposure to $PM_{2.5}$ overall was associated with increased mortality with a 2.14% increase (95% CI: 1.38, 2.89%) and 7.5% increase (95% CI: 1.95, 13.40%) for each 10 $\mu g/m^3$ increase in short (two-day) and long-term (one year) exposure respectively. They also found that the associations were significant even when analyses were restricted to exposures below the current U.S. EPA standards and that there was a larger effect for mortality with exposures ≥ 6 $\mu g/m^3$ than for exposures ≤ 6 $\mu g/m^3$.

Crouse et al. (2012) also investigated the risk of non-accidental and cardiovascular mortality from long-term exposure to low concentrations of $PM_{2.5.}$ Their study followed a cohort of 2.1 million non-immigrant Canadian adults, identifying deaths that occurred from 1991 – 2001. They found hazard ratios of 1.15 (95% CI: 1.13, 1.16) from non-accidental causes and 1.31 (95% CI: 1.27, 1.35) from ischaemic heart disease for each 10 $\mu g/m^3$ increase in concentration of $PM_{2.5}$. Associations were observed with exposures to $PM_{2.5}$ at concentrations of 8.7 $\mu g/m^3$ (interquartile range 6.2 $\mu g/m^3$) which is lower than the NAAQS.

Several studies have also looked at the effects of low level exposure to air pollutants on pregnancy complications and birth outcomes. Maroziene & Grazuleviciene (2002) investigated maternal exposure to low-level air pollution and pregnancy outcomes in Lithuania. They found there may be a relationship between maternal exposure to ambient formaldehyde and the risk of low birth weight (LBW), as well as between NO_2

exposure and the risk of preterm birth. They found the risk of preterm birth increased by 25% (adjusted odds ratio 1.68 (95% CI: 1.15-2.46) per 10 µg/m^3 increase in NO_2 concentrations. The adjusted odds-ratios increased but only in medium and high tertiles of NO_2 concentration associated with exposure to pollutants during the first trimester. The incidence of LBW increased with increasing formaldehyde levels (in the second and third tertiles). In a Swedish study, Malmqvist et al. (2013) found low level exposure to NO_2 below current air quality guidelines was significantly associated with both gestational diabetes and preeclampsia.

DeVries et al. (2016) examined documented exacerbations of COPD (rather than emergency department visits or hospital admissions) and low level air pollution and found that despite living in an area with air pollution concentrations below current U.S. EPA NAAQs for SO_2 independently and NO_2 in association with $PM_{2.5}$, COPD patients appeared to suffer increased risk of COPD exacerbation following short-term exposures to increased concentrations of SO_2 and NO_2. They also found an unexpected negative association with $PM_{2.5}$ may which they suggested may have been due to the complex air chemistry of low level PM in the region. With regard specifically to PM, epidemiological evidence indicates that the adverse health effects of short-term $PM_{2.5}$ exposure are due more to frequent mid-range exposure days rather than less frequent peak exposure days (Morgan, Broome, & Jalaludin, 2013).

Barnett (2014) highlighted the concerns of several authors that air quality standards, including the AAQ NEPMs, are often incorrectly interpreted to imply levels above a given standard are ‘potentially dangerous’ and those below the standard are ‘safe’. Previous studies (Andersen et al. 2012; DeVries et al., 2016; Kim et al., 2011; Schwartz, Bind, & Koutrakis, 2017; Malmqvist et al., 2013; Morgan, Broome, & Jalaludin, 2013; Schelegle et al., 2009) show that this is not the case, particularly for vulnerable groups being exposed to levels just below the standard. The 2005 update to the WHO air quality guidelines (WHO, 2006) noted that there was little evidence to suggest a threshold below which no adverse health effects would be anticipated. Similarly an international air pollution expert group that convened in 2009 stated ‘there are no established thresholds of exposure below which population health impacts are absent’ (Giles et al., 2011, para 1). Barnett (2014) has called for changes to be made to the AQ NEPM highlighting that the standards should not be used to judge whether individual projects are safe or dangerous. As air pollution standards are developed based on best evidence available in public health epidemiological, toxicological and

clinical studies; given the evidence now available, it appears there may be justification for lowering current Australian standards, particularly for CO, NO_2 and ozone.

2.6 Air quality in Perth (Western Australia)

The Perth Air Quality Management Plan (AQMP) was released in December 2000 in response to the reporting requirements of the AAQ NEPM (DEP (WA) 2000). Its aim is to 'to maintain and improve Perth's air quality so that people of Western Australia have clean air to a level that will not harm the environment or affect the health of our community' (DEP (WA) 2000 ,p.3). The WA Environmental Protection Agency (EPA) set up a Select Committee (now the Air Quality Coordinating Committee (AQCC) with representatives from State Government, industry, business and the community to monitor the implementation of the AQMP (Environmental Protection Authority Review Steering Committee, 2007; DER AQCC (WA), 2014). The short-term and long-term actions within the Perth AQMP address a variety of issues through eight key initiatives comprising: land use and transport planning; vehicle emissions management; health effects research and indoor air quality; monitoring and modelling; industrial emissions management; small to medium enterprise emissions management; and haze reduction and smoke management.

The initial AQMP report acknowledged that for most of the year and in most years metropolitan Perth has good air quality, but also warned that complacency in planning and implementing air quality management strategies could mean unacceptable air quality in the future. Photochemical smog and particulate haze were identified as two of several areas requiring further investigation and action. Since that time an AQMP Report Card giving details of monitoring and achievements in each of the eight key initiative areas has been published annually by the AQCC (DER AQCC (WA), 2017).

2.6.1 Ambient air monitoring in Perth

Perth has a network of monitoring stations scattered throughout the metropolitan region, with additional stations in several regional areas such as Bunbury and Collie. The metropolitan stations were established in response to the Perth Photochemical Smog Study (1992-1996) which concluded that at that time Perth experienced photo-chemical smog levels which exceeded goals set by the National Health and Medical Research Council (NHMRC) and other bodies, and also identified the potential for the problem to grow with increases in population and associated vehicle use (DE (WA),

2004). At that time Perth experienced photochemical smog to a similar extent as Brisbane, greater than Adelaide and less than Sydney and Melbourne (DE (WA), 2004). It should be noted that many of the current monitoring stations in Perth are located away from the more densely populated, high traffic areas. It is possible that they may not give a representative picture of the concentrations of pollutants that inner city residents are likely to be exposed to. In late 2017 the DWER is commissioning a new monitoring station in a central location in the city that will provide much needed data regarding pollutant levels in areas of dense traffic (DER AQCC (WA), 2017).

In terms of ambient air quality, Perth is uniquely situated in that it is somewhat protected from the influence of air pollution entering from other urban airsheds by its location on a coastal plain between the Indian Ocean to the west and a 400 metre high escarpment to the east. It is over 2000 km from the nearest major city (Pereira et al., 2010). Generally ambient air quality in the Perth metropolitan area is classified as 'good' to 'very good' with occasional instances of photochemical smog in summer and particle haze during winter. Major sources of pollution have been identified as motor vehicles, bushfires, domestic sources (principally wood heaters) and industry (DEC (WA), 2011; DER (WA) 2014, 2017)

According to the 2015-2016 AQMP Report Card, the results of DER air quality monitoring in Perth from 2003-2016 indicated that the AAQ NEPM goals for CO_2, NO_2, and SO_2 were met every year. There were a number of exceedances for PM_{10}, $PM_{2.5}$ and ozone, however the number was consistently less than the maximum number stipulated under the Standards and in most cases the exceedances only occurred on one or two occasions over the 10 year period. Exceedances were generally associated with bushfires or controlled burns (DER AQCC (WA), 2014, 2017).

Figures 2.6(a)-2.6(d) demonstrate the monthly maximum levels of selected pollutants over the last 3 years as reported in the 2015-2016 AQMP Report Card (DER AQCC (WA), 2016). Figure 2.6 (a) illustrates the levels of fine particulates over the 3 year period. Exceedances over the 2015/2016 season were generally a result of prescribed burns in the south-west.

It can be seen from Figure 2.6 (b) that there was one major exceedance of PM_{10} levels over the three-year period. This occurred due to dust created during the clean-up of a local drainage pit in close proximity to the Duncraig monitoring station. Figure 2.6 (c) shows ozone levels hovering around the NEPM standard over the warmer months, with borderline exceedances most likely due to bushfires and high temperatures. The

warmer summer temperatures are likely to have contributed to the production of ozone. Figure 2.6 (d) shows that NO_2 level are consistently low and at no point over the last three years have come close to the NEPM standard value.

2.6.2 Previous air quality and health studies in Perth

Several studies examining the health impacts of exposure to air pollutants have been conducted in metropolitan Perth, Western Australia. Hinwood et al. (2006) conducted a case-crossover study investigating the relationship between daily air pollutant concentrations and daily hospitalisations for cardiovascular and various respiratory illnesses during the period 1992-1998. A large number of analyses were performed however only a small number of significant associations were found between daily concentrations of PM, NO_2 and CO for COPD, pneumonia, asthma and CVD hospitalisations. For each 1 ppb of increase in NO_2, respiratory disease hospitalisations increased by 0.6%, there was an estimated 0.4% increase in cardiovascular (CVD) hospitalisations in the 65 and over age group and an estimated 0.4% increase in CVD hospitalisations for all groups. Changes in daily $PM_{2.5}$ concentrations were significantly associated with respiratory disease, pneumonia and asthma hospitalisations for specific time lags. There were no significant associations between changes in ozone concentrations and any disease outcomes.

Hinwood et al. (2004) also investigated relationships between air pollution and mortality from cardiovascular disease, respiratory disease and 'other mortality'' in the Perth metropolitan region. The results showed significant associations between cardiovascular mortality and increased NO_2 and ozone concentrations. Respiratory mortality was significantly associated with increased. 8-hour ozone concentrations. There was also a significant association found between increases in CO concentrations and 'other mortality'.

Pereira et al. (2010) conducted a case-crossover analysis of traffic-related air pollution in Perth, specifically investigating emergency department presentations for childhood asthma. They found there were statistically significant increases in the odds of ED presentations observed only for NO_2 (21% increase per 1 ppb NO_2) and CO (5 fold increase per 1 ppm of CO) with a 1-day lag. After stratifying for age and gender, they found those that were 0-4 years of age were most affected, and most cases occurred in boys.

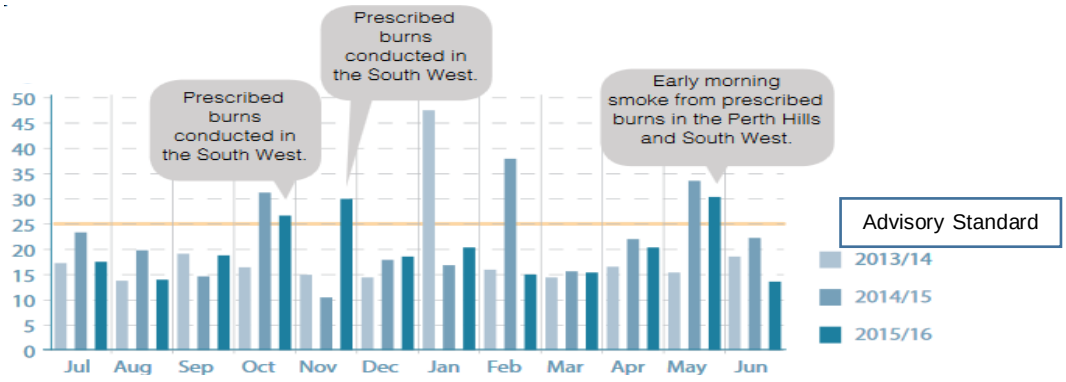


Figure 2.6 (a) Monthly maximum 24-hr levels of $PM_{2.5}$ ($\mu g/m^3$) from 2013-2016

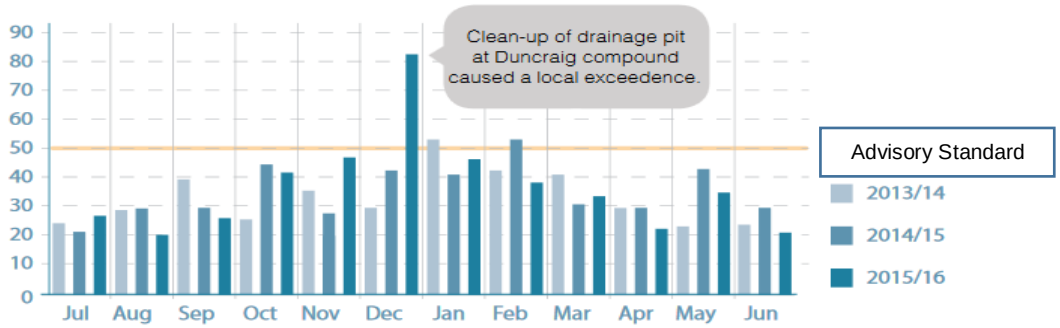


Figure 2.6 (b) Monthly maximum 24-hr levels of PM_{10} ($\mu g/m^3$) from 2013-2016

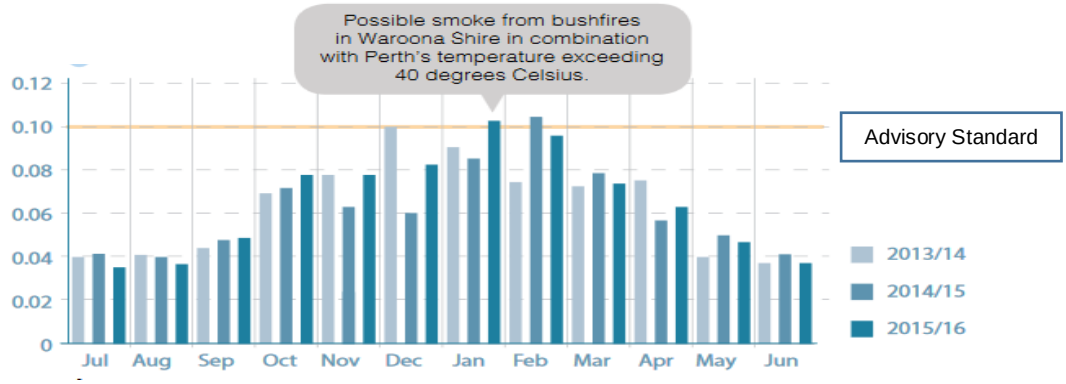


Figure 2.6 (c) Monthly maximum 24-hr levels of ozone (ppm) from 2013-2016

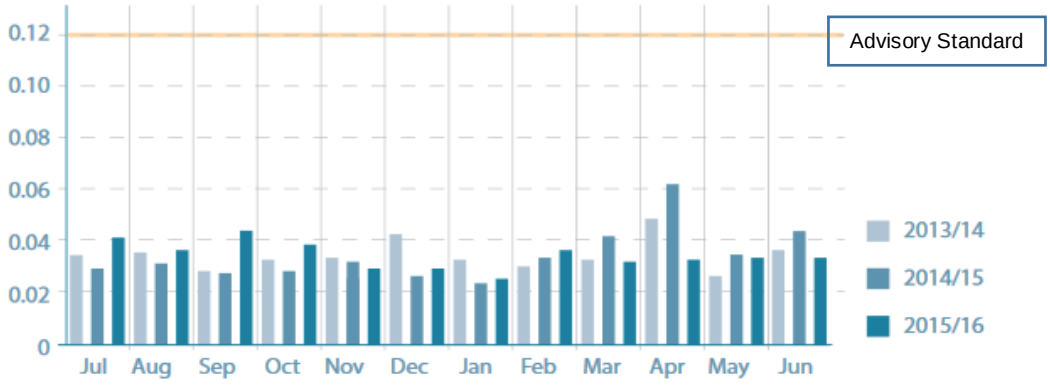


Figure 2.6 (d) Monthly maximum 24-hr levels of nitrogen dioxide (ppm) from 2013-2016

2.7 Conclusion

It is clear that the quality of the air we breathe affects our health. Many studies have demonstrated the relationship between air pollutants and various health conditions, from self-reported symptoms to emergency department presentations, hospitalisation and death. Air pollution is a very complex phenomena, involving many different types of organic and inorganic contaminants and influenced by chemical interactions and weather and energy processes. What has become evident is that increasing levels of pollutants are associated with increased levels of health effects but even more importantly, that health effects are occurring at levels well below the standards that are designed to protect our heath. Identification of individual pollutants, down to the level of speciation of metals, may help to identify sources, however what needs to be kept in mind is that pollutants rarely occur individually but rather as part of a complex mix of aerosols.

With regard to air quality in the Perth metropolitan area, there is little information available about any current associations between various pollutants and health effects. While a previously discussed study by Hinwood et.al (2006) investigated these associations, this study provides a more recent analysis of associations one decade later. In addition, there is no published data available that identifies and quantifies levels of airborne metals present in ambient air. This is particularly important in industrial zones that are located upwind or in close proximity to areas that are zoned residential as residents may be exposed to emissions containing concentrations of metals capable of contributing to adverse health outcomes.

As Perth, Australia and urbanised areas of the world undergo population growth, there is likely to be a corresponding increase in the level of pollutants caused by various activities, including power generation, development and construction and more particularly traffic. It is important that governments act now to translate scientific evidence into policy and move to adopt and implement strategies to achieve a balance between anthropogenic pollutants resulting from these activities and protecting our air quality into the future. It has been shown that even small improvements in air quality can have positive effects on human health and wellbeing, in particular on respiratory and cardiovascular health. Improving the health of the population has obvious economic benefits by reducing productivity losses in the workplace and even more importantly by reducing the burden of disease and associated health care costs (DEE, 2016; Kelly & Fussell, 2015; Keywood, Hibberd & Emmerson, 2017).

CHAPTER 3 Research methodology

This chapter will outline the methodology used to achieve the aims of the study.

This study was conducted in 2 stages. In the first stage, profiles of ambient air pollutants were established and examined in relation to hospital admissions for selected conditions to determine whether any relationships existed between levels of pollutants and frequency of emergency admissions.

The overall aim for the first stage was:

- To evaluate the relationship between daily levels of selected ambient air pollutants and respiratory and cardiovascular hospital admissions in the Perth metropolitan area from 2001-2010.

In the second stage monitoring was undertaken in two selected areas of metropolitan Perth to determine the composition of particulates being emitted, since these data were found to be sparse in the first stage.

The overall aim for the second stage was:

- To characterise and speciate fine and coarse particulates at sites representative of residential / background and light industrial (source) areas in metropolitan Perth.

3.1 Stage 1: Determining relationships between ambient air quality and hospitalisations

Using existing data, historical profiles were established for levels of selected pollutants and also for hospital admissions for respiratory and cardiovascular conditions, over a 10 year period (2001-2010) in the Perth metropolitan area. Descriptive analyses of all profiles were conducted. Profiles were then analysed to determine whether any significant relationships existed between mean daily levels of individual pollutants and hospital admissions for selected conditions. These results were compared to similar studies conducted by over the period 1992-1998 (Hinwood et al., 2006).

3.1.1 Hospital admission data

Hospital morbidity data for patients admitted with a principal or co-diagnosis of diseases of the circulatory system (ICD10-CM codes I00 – I99) and respiratory system (ICD10-CM codes J00 – J99) between 1 January 2001 and 31 December 2010 were obtained from the Hospital Morbidity Data Custodian at the Western Australian Department of Health. Diagnosis was based on primary discharge obtained from the patients' charts (Table 3.1).

Table 3.1: Data obtained from the Hospital Morbidity Data Custodian at the WA Department of Health.

Variables	Definition & Categories
Demographic Data	
Admission Age	Age in years on admission
Gender	Male / Female
Postcode	Postcode of Residence
Hospital Data	
Patient ID Number	Hospital Identification Number
Admission Date	Date patient was admitted
Separation Date	Date patient was discharged
Length of Stay	Length of stay minus days on leave
Admission Status	Waitlist / Non-waitlist / Emergency
Principal / Co- Diagnosis	ICD10 code
Hospital category	Tertiary, Public / Private Metro, Rural

The following inclusion and exclusion criteria were applied to the hospital admission cases:

- Wait-listed admissions were removed so that only unscheduled, 'emergency' admissions, i.e. only those that could potentially be related to environmental conditions were considered;
- Cases in non-metropolitan hospitals were removed as there was insufficient environmental data covering these areas to consider these exposures.
- Cases were also excluded if their residential postcode was outside the metropolitan area. Only postcodes between 6000 – 6199, 6800-6999 were included with the exception of those between 6200 and 6799 as these areas are also outside the metropolitan area.

The ‘metropolitan’ region is defined by the *Planning and Development Act 2005* to include 30 local government areas with the outer extent being the Cities of Wanneroo and Swan to the north, the Shires of Mundaring and Kalamunda and the City of Armadale to the east, the Shire of Serpentine-Jarrahdale to the southeast and the City of Rockingham to the southwest, and includes Rottnest and Garden Islands off the west coast. This extent correlates with the Metropolitan Region Scheme (Department of Planning [DP] (WA), 2015).

Age was obtained in years only, date of birth was not given. For descriptive analysis, cases were grouped according to age to determine if any particular group in the community was more likely to be admitted to hospital for respiratory or cardiovascular conditions: The groups were as follows:

- Less than 15 years of age (<15) : children & teenagers;
- Between 15 and 65 years of age (15- 65): adults; and
- Over 65 years of age (>65): elderly.

These particular groupings were also chosen to enable comparison with other research as these groups are most commonly employed in air pollution studies investigating health outcomes (Hinwood et al., 2006).

3.1.1.1 Comparison with historical data

To determine whether there had been any notable increase in hospital admissions since the previous decade, the mean daily number of admissions for each selected illness conditions was determined by averaging the number of daily admissions for each month for the ten year period (2001-2010) and compared to corresponding data from 1992-1998. The mean number of admissions was determined for three categories, the whole year, the warmer months from November to April and also the cooler months from May to October. These time periods were selected to enable direct comparison with data from a similar previous study conducted 20 years ago in Perth (Hinwood et al., 2006). As there had been substantial population increases between the two decades, the mean values were standardised by dividing the number of admissions by the mean population of the Perth metropolitan area during the corresponding time period.

3.1.2 Statutory air quality monitoring data

Environmental air quality data was obtained from the Department of Environment Regulation who have an established network of ambient air quality monitoring stations throughout the Perth metropolitan area as well as in some regional areas to assess the state of air quality against Australian standards (Figure 3.1). The monitoring stations test for various air pollutants such as carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), particulate matter <10 µm (PM_{10}) and <2.5 µm ($PM_{2.5}$) and visibility. Not all pollutants are measured at all locations and not all are measured continuously (Table 3.2). In industrial areas such as Kwinana, Wattleup, Hope Valley and North Rockingham, monitoring is also carried out for sulphur dioxide (SO_2) and Volatile Organic Compounds (VOCs). Monitoring is frequently the result of a particular campaign or project and is not long term or continuous (DEC (WA), 2011).

The Perth metropolitan stations that were operational during the study period (2001-2010) are located at Caversham, Duncraig, Quinn's Rock, Rockingham, South Lake and Swanbourne. Until 2007 there was also a monitoring station located in the Perth Central Business District (CBD) on top of the Queen's Building in William Street. The monitoring station was removed as part of a refurbishment of the building and was not relocated, and as such there is currently no DER monitoring conducted in the Perth central business district (CBD).

The locations of these monitoring stations were originally chosen for the purposes of conducting the Perth Photochemical Smog and Perth Haze studies and to investigate sulphur dioxide (SO_2) emissions in the Kwinana area. The network design was based on prior knowledge of emission sources, pollutant chemistry and important features of the local meteorology (DEC (WA), 2004) as well as advice provided by the Atmospheric Research Branch of the Commonwealth Scientific Industrial Research Organisation (CSIRO) (EPA 2007).

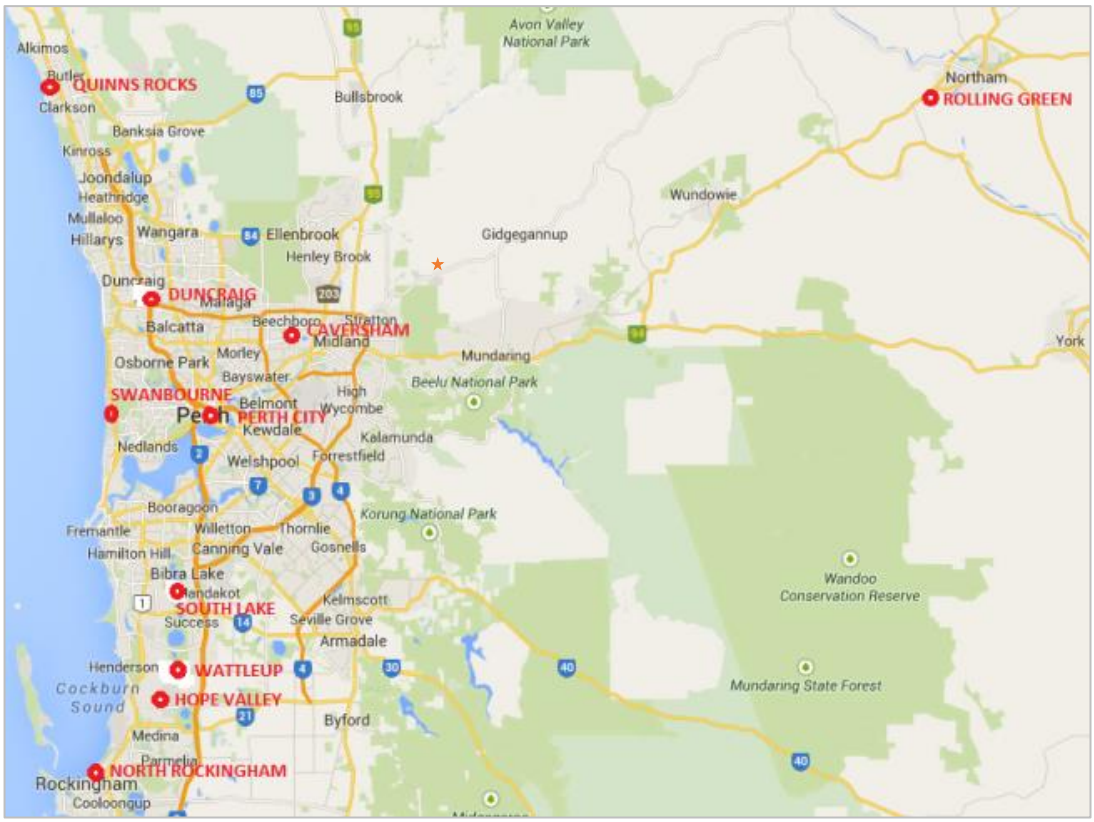


Figure 3.1: Perth metropolitan air quality data map showing locations of monitoring stations (Red dots). Perth City monitoring station currently decommissioned. (Department of Environment Regulation, 2016).

For this study 24-hour averaged data for CO, NO_2, O_3, PM_{10} and $PM_{2.5}$, collected between the 1st January 2001 and the 31st December 2010, was obtained from the Caversham, Duncraig and South Lake monitoring stations as these particular stations had the most complete data sets and have been shown in previous studies (Hinwood et al., 2006) to be representative of the Perth metropolitan area. These particular pollutants were selected for the study as they are considered 'criteria' air pollutants in Australia and are included in most studies on ambient air pollution and health impacts (Hinwood et al., 2004, 2006; Pereira et al., 2010). Although sulphur dioxide and lead are also 'criteria pollutants', they were not included in this study as monitoring data is scarce and they are not regularly investigated in general air quality studies (Hinwood et al., 2004, 2006; Pereira et al., 2010). During 2001-2010 sulphur dioxide was only monitored at the Wattleup, South Lake and Rockingham sites which are all situated in the south of the metropolitan area, and lead was not monitored at any of the DER monitoring stations.

Table 3.2: Location of monitoring stations and pollutants monitored in the Perth metropolitan area 2001 – 2010 during the period of this study.

	NO_2 (pphm)	CO (ppm)	O_3 (pphm)	$PM_{2.5}$ ($\mu g/m^3$)	PM_{10} ($\mu g/m^3$)
Caversham	✓	✓	✓	✓	✓
Duncraig	✓	✓	✓	✓	✓
Quinn's Rocks	✓	X	✓	✓	X
South Lake	✓	✓	✓	✓	✓
Rockingham	✓	X	✓	✓	X
Swanbourne	✓	X	✓	X	✓

In the Perth metropolitan area ambient CO, PM_{10} and NO_2 levels have been measured continuously over many years. NO_2 and O_3 were first measure in 1990, CO in 1993 and PM_{10} in 1998. Although $PM_{2.5}$ had been monitored at some stations as early as 1995, direct measurement was not available at Caversham, Quinn's Rock, Rockingham and South Lake before 2006. Previously $PM_{2.5}$ values were estimated from an optical backscatter coefficient measured by nephelometry (measure of visibility) (Hinwood et al., 2004, 2006; Petroeschevsky et al., 2001; Simpson et al., 2000).

During the period of this data collection, PM_{10} and $PM_{2.5}$ were measured at most sites using a Rupprecht and Patashnick 1400AB Tapered Element Oscillating Microbalance (TEOM) Continuous Ambient Particulate Monitor (Thermo Scientific). At both Caversham and Swanbourne, PM_{10} was measured using a HiVolume Sampler. CO, NO_2 and O_3 were all measured using Thermo Environmental Instruments (TEI), model 48C for CO, model 42C for NO_2 and model 49C for O_3 (Table 3.3).

In a 2003 Department of Environment report on air pollution in Perth, the authors noted that due to the limited number of monitoring sites in the greater Perth area and the large variations in population density and subsequent particle concentrations between sites, no single site value was considered likely to provide an accurate estimate of the mean exposure of the city's population (DER, 2003). For this study all available pollutant levels for a particular day from the selected monitoring stations were averaged to use as a surrogate value for metropolitan exposure. The number of values included in this calculation depended on the number of stations monitoring for

a given pollutant at any given time. This approach has been used for other studies (Hinwood et al., 2004, 2006) and is necessary to account for frequent gaps in the monitoring programme and also incidents of equipment malfunction or non-recording.

Daily temperature and relative humidity were also obtained from the monitoring stations where available. Daily values were averaged to give a representative value across the metropolitan area. Rainfall data was obtained from the Bureau of Meteorology at Perth Airport.

Table 3.3: Metadata for Ambient Air Quality Monitoring Stations. Table includes dates (month/year) of commissioning (and decommissioning), surrounding land use, emission sources and pollutants monitored. A dash '(-)' indicates monitoring for pollutant not conducted during the period of this study.

Date sampling for individual pollutants commenced and ceased (M/YY). If decommission date not given assume data still being collected.	**Metropolitan Air Quality Monitoring Stations**							
	Caversham De Burgh Rd	**Duncraig** Doveridge Dr.	**Gosnells** 81 Station St.	**Quinn's Rocks** Quinn's Rd.	**Rockingham** Governor Rd. E	**South Lake** Barrine Gdns.	**Swanbourne** W. Coast Hwy.	**Wattleup** Tomislav Pl
CO	8/93	8/95	-	-	-	3/00	1/93 – 6/95	-
NO_2	9/90	8/95	-	1/93	1/96	3/00	1/93	-
O_3	1/90	-	-	1/93	1/96	3/00	1/93	-
SO_2	-	-	-	-	8/88	3/00	-	1/88
PM_{10}	5/06	6/95	12/07 – 9/09	-	-	3/00	3/94 – 4/06	-
$PM_{2.5}$	5/06	1/95	-	6/06	-	3/00	-	-
Visibility	1/90 – 1/94	3/94 – 8/05	-	1/96 – 6/06	-	3/00 – 9/05	6/94 – 6/03	-
Surrounding land use (residential/industrial/ rural & density)	Semi-rural (low density)	Residential (hi density)	Residential (hi-density)	Residential coastal (low density)	Residential coastal (low density)	Residential (hi density)	Coastal (med density)	Industrial buffer (med density)
Nearby emission sources (industrial, domestic & mobile; e.g., distances, direction to roads with estimated traffic density)	Vineyards, orchards and paddocks	Low density residential traffic flow. Freeway 200m East	Low density residential. 500m from Hwy	Low density residential traffic flow. 100m from beach.	Low density residential traffic flow. 100m from beach.	Low density residential traffic flow	Coastal bushland	Low density residential traffic flow. Industry 2km West.

3.1.3 Statistical analysis of hospital and air quality data

Descriptive and inferential statistical analysis of the hospital admission and air quality data was undertaken using IBM SPSS Version 23 Software and p-values less than 0.05 were considered as statistically significant.

3.1.3.1 Descriptive analysis of hospital admission data

Hospital admissions were categorised into 3 main categories based on 'Diagnosis at admission' namely diseases of the respiratory, cardiovascular (circulatory) and gastrointestinal (digestive) systems, according to ICD-10 (Centres for Disease Control [CDC], 2010). Daily numbers of admissions were calculated for each category and sub-category. Cardiovascular and gastrointestinal admissions were analysed as an intact group however respiratory admissions were further sub-categorised into four distinct groups -'asthma', 'chronic obstructive pulmonary disease' (COPD), 'pneumonia/ influenza/ acute bronchitis', and 'other respiratory'. These categories and sub-categories have been used by previous studies conducted in the Perth metropolitan area (Hinwood et al., 2006). Descriptive analysis was performed to calculate the distribution between gender, age group and length of stay across each season for each disease condition.

Initially plots were constructed demonstrating the mean number of daily admissions for each condition over the ten year period (2001-2010) (Figures 4.1-4.7). These charts were used to identify whether there were any seasonal trends in hospital admissions for particular conditions or age groups and also whether there were any seasonal trends in levels of pollutants. To account for population changes (generally increases) over the 10 year period, the number of admissions were standardised before computing the ratio of respiratory and cardiovascular admissions by the gastrointestinal conditions groups. The gastrointestinal conditions group was used as a control as there was no indication that gastrointestinal admissions were affected by month or season.

The four respiratory sub-categories of 'asthma', 'COPD', 'pneumonia / influenza/acute bronchitis' and 'other respiratory' were further standardised to account for the smaller number of admissions in comparison to the number of gastrointestinal admissions. In these instances, the standardised number of admissions for each sub-category was

divided by the 'median' number of admissions for each condition each month. This was then divided by the standardised number of gastrointestinal admissions.

3.1.3.2 Descriptive analysis of air quality data

Analysis was also undertaken to determine if there were any temporal relationships between hospital admissions and levels of various ambient pollutants for the same day. Although not all pollutants were measured at all sites, all data for sites where there were measurements for over 50% of the days were included to give the most complete representation of air quality at that particular time. As an example, although $PM_{2.5}$ was monitored at the Kwinana and Rockingham stations, it was only for a 12 month period from the beginning of September 2005 to mid-September 2006 so was not included.

Historically, air pollutant levels in the Perth metropolitan area have rarely exceeded the Australian Air Quality standards and when they have, it has usually been associated with events such as bushfires and usually only for an individual site or closely located sites and for a limited period. In this study rather than concentrating on the number of individual instances of exceedances above the standards, mean daily values for each pollutant were divided into tertiles with the lowest tertile containing the lowest 33.3% of monitored values, the middle tertile containing the middle 33.3% of values and the third tertile containing the highest 33.4% of values. Once the data had been allocated into tertiles, descriptive analysis was performed to determine the mean values, standard deviation and range within each tertile for each pollutant.

Tertiles have frequently been used in air quality research to categorise levels of air pollutants into low, medium and high relative exposures. This method was used as the level of pollutants rarely exceeded any established guidelines or standards. Maroziene and Grazuleviciene (2002) investigated low-level exposures of NO_2 and formaldehyde pollution and pregnancy outcomes; Yang, et al. (2010) used tertiles in their investigation into the relationship between petrochemical air pollutants and cancer in females in Taiwan; Zmirou et al. (2005) used tertiles in their analysis of the relationship between traffic related air pollution and incidence of childhood asthma and Lacasaña, Esplugues and Ballester (2005) used tertiles in their in their investigation of the effect of PM_{10} levels on health effects in early childhood. Similarly, a study by Malmqvist et al. (2013) used quartiles to investigate whether exposure to

levels of NO_2 affected pregnancy complications in an area of Sweden with low air pollution levels.

To allow for analysis to determine whether there was any seasonal influence on admissions, months were also categorised into 'seasons' as per the Australian Government Bureau of Meteorology (BOM) (2017) definition:

- Summer – December, January, February
- Autumn – March, April, May
- Winter – June, July, August
- Spring – September, October, November

3.1.3.3 Inferential analysis of hospital and air quality data

Cross tabulations were utilised to evaluate the associations between air quality data and hospital admissions. The air quality data was categorised into tertiles, signifying low, medium and higher pollutant levels within the monitored range. Hospital admissions were categorised in 2 ways:

- If the ratio of the standardised number of admissions was greater than10% for each condition compared to gastrointestinal conditions (≥1.10); and
- If the ratio of the standardised number of admissions was greater than 20% for each condition compared to gastrointestinal conditions (≥1.20).

The categorisation of the standardised hospital admission reflects changes that are generally considered to be clinically significant. Considering both a 10% change and a 20% change, allows us to conduct a sensitivity analysis to explore the effect on associations with air quality by varying the cut-point (Baird, 1989).

Logistic regression analysis was used to assess the associations between selected air pollutants and hospital admissions, before adjustment and after adjustment by season. The effects of the air pollutants are represented as odds-ratios comparing higher tertiles with the lowest tertile (reference tertile), and associated 95% confidence intervals were computed. These odds-ratios represent the magnitude of influence of the pollutant levels on admissions.

3.2 Stage 2: Fine-scale air quality monitoring and metals speciation

The overall aim of this stage is to evaluate and compare exposure levels of particulate matter between residential and industrial areas in metropolitan Perth.

Stage 2 of the study involved a determination of the concentration and elemental composition of ambient PM_{10} and $PM_{2.5}$ air from two (2) selected areas of Perth to establish current air quality profiles, focusing specifically on metalliferous particulates.

3.2.1 Monitoring areas and site selection

Monitoring was conducted in two distinct areas of metropolitan Perth representing industrial and non-industrial areas. Each area contains a different mix of zonings under their respective Local Government Town Planning Schemes (TPS).

The two areas were:

1. Welshpool, (zoned mixed-use) industrial / commercial / residential/ parks and recreation. This area lies within the Local Government areas of the City of Canning (TPS 40), Belmont (TPS 15) and the Town of Victoria Park (TPS 1). This cluster of sites is representative of a light industrial setting.

2. Curtin University campus, Bentley which is situated within the Town of Victoria Park and is zoned 'Public purposes – University'. Although the site in Technology Park is technically part of Curtin, under the Town of Victoria Park TPS 1 it is zoned 'Urban'. This cluster of sites is representative of a non-industrial setting.

3.2.1.1 Area 1: Welshpool

The Welshpool area was chosen as it is an older established area of metropolitan Perth and has mixed industrial, commercial and residential zoning. In addition, previous monitoring conducted as part of a broader study commissioned by DEC (now DWER), had identified the potential for significant emissions of lead from this area (Mullins, Personal Communication 2017). For the present study, monitoring was conducted at six (6) sites across both industrial and residential zones within three different Local Government jurisdictions, the City of Belmont (CoB), the City of

Canning (CoC) and the Town of Victoria Park (ToVP) (Figure 3.2). These sites were chosen as they are downwind of the industrial area.

1. Domestic Residence – Oats St, KEWDALE (CoB)
2. High School, President Ave, KEWDALE (CoB)
3. Department of Mines & Petroleum Core Library, Harris St, CARLISLE (ToVP)
4. Town of Victoria Park Works Depot, Briggs St, CARLISLE (ToVP)
5. Museum storehouse, Cnr Welshpool Rd & Leach Hwy, WELSHPOOL (CoC)
6. Domestic Residence, Gemini Way, CARLISLE (ToVP)

Figure 3.2: Map of Welshpool (industrial) area showing cluster of sampling sites (red squares). Source: City of Canning (TPS 40), Belmont (TPS 15) and the Town of Victoria Park (TPS 1).

These locations were selected as they:

(a) gave a representative spread across both the residential and semi-industrial pockets within the area;

(b) were reasonably secure and had access to AC power; and

(c) were generally downwind of likely sources of pollutants from the industrial area

Generally, sites provided little opportunity for public interference. At each site, equipment was left in place for seven days to obtain sufficient spatial and temporal resolution of the emissions. It also allowed for the capture of weekend as well as weekday readings as many of the industries located in this area operate seven days per week.

3.2.1.2 Area 2: Curtin/Bentley

This area was chosen to represent residential, non-industrial areas. It has a high population density and includes a tertiary institution which has approximately 15,000 adults and children attending each day (Curtin University, 2016). The area includes several multi-story buildings, lecture and tutorial rooms, offices, shops, 2 childcare centres, laboratories and recreation areas including a gymnasium and open air sports fields (Figure 3.3). With the exception of Site 5 at Technology Park, Curtin University is not specifically recognised as a source of particulates. It does, however, provide background levels of pollutants and also give an indication of potential long range transport of emissions from the previous monitoring area in Welshpool.

Sampling was conducted at 6 sites selected to give a representative spread over the area. Each site was monitored over 5 working days from 8.00 am Monday to 4.00pm Friday each week (104 hours). Monitoring was not conducted on the weekends in this location as there is a significant decrease in population over these days and likely sources of emissions were not functioning which would misrepresent the potential for exposure. These sites were all located within the Town of Victoria Park.

The sites monitored were

1. Building 400, School of Public Health (south-west corner of balcony on third floor)
2. Building 003, Childcare Centre (utilities area on south-west corner of building, ground level)
3. Building 402, School of Business (rooftop, Level 10)
4. Building 215, Engineering Pavilion (north-east corner of roof Level 4)
5. Building 611 (Technology Park), School of Environment and Agriculture (adjacent to plant room, ground level)
6. Building 105, Library - (North-east corner of walkway, level 3)

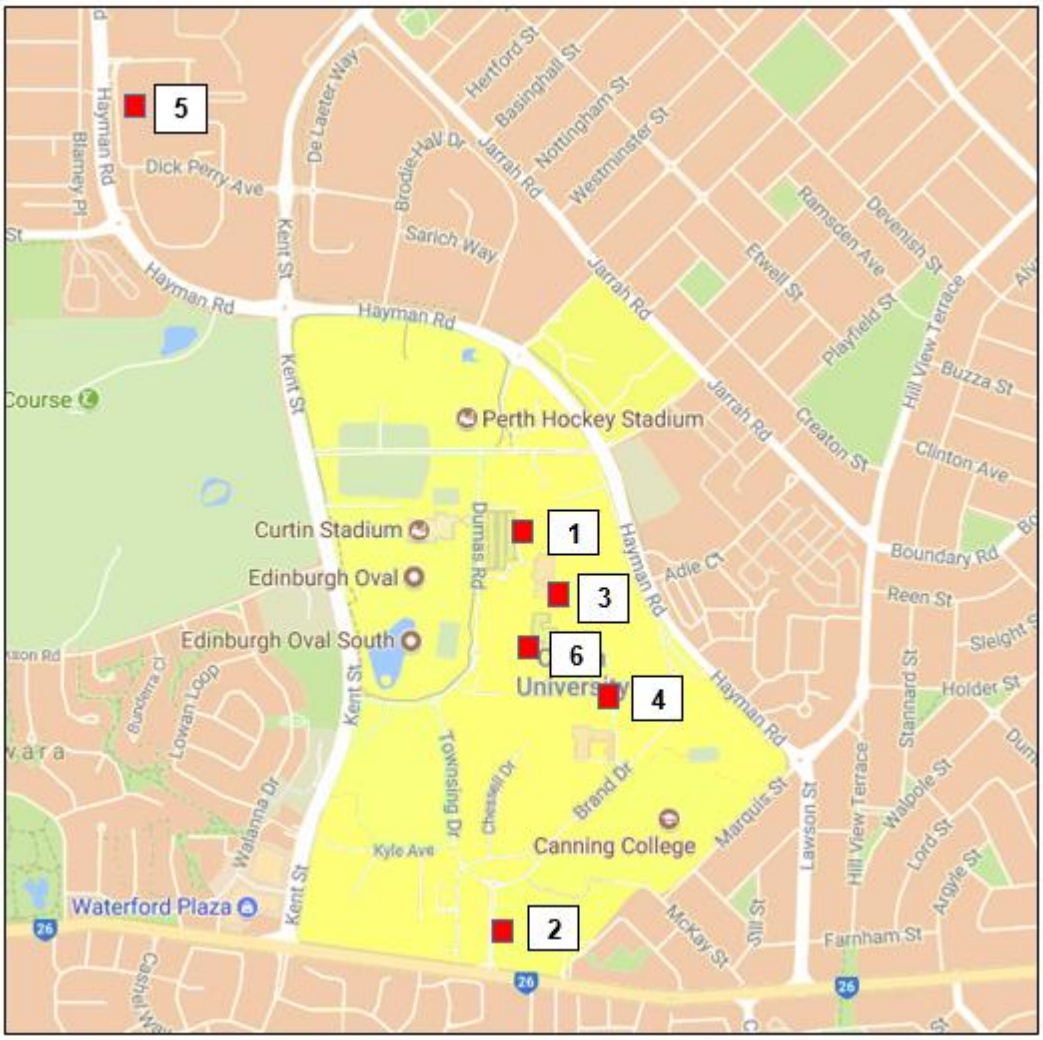
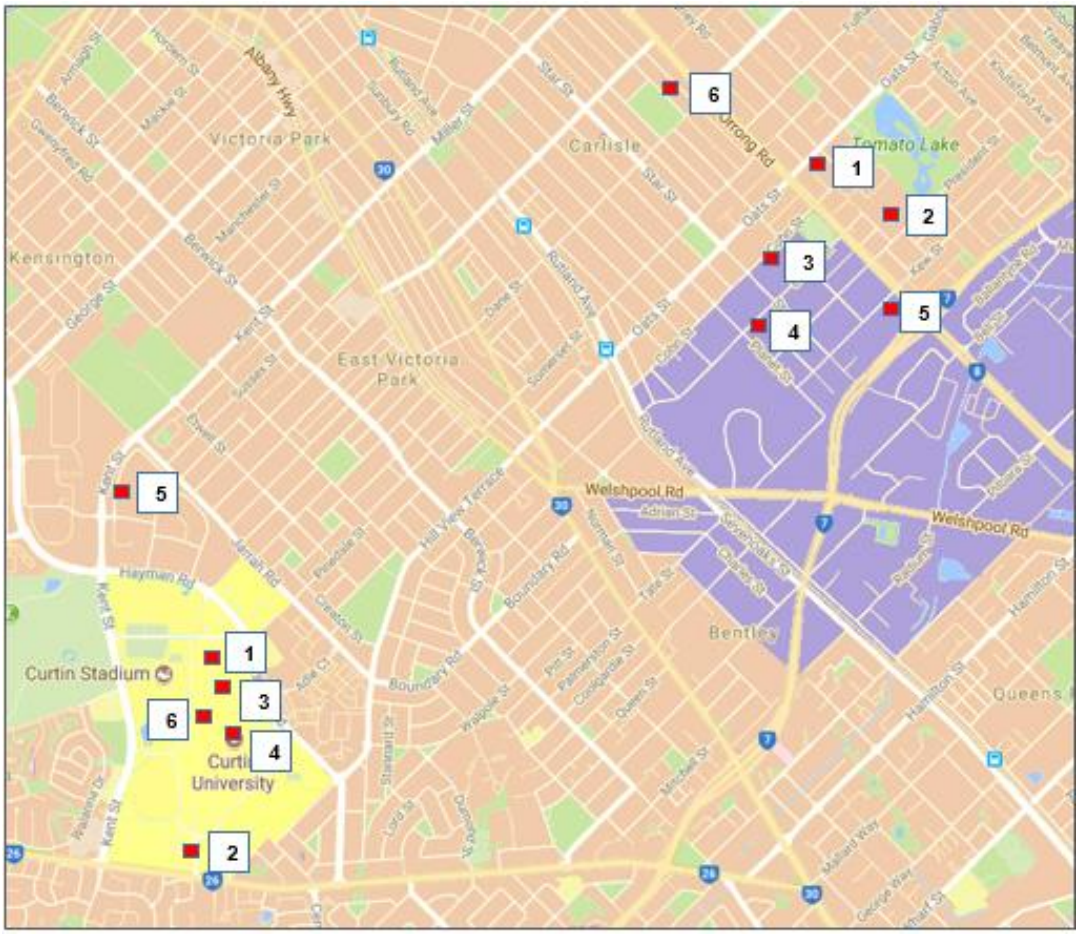


Figure 3.3: Map showing cluster of Curtin/Bentley (Non-industrial) sampling sites (red squares). Source: Town of Victoria Park (TPS 1).

Figure 3.4 illustrates the proximity of the industrial Welshpool cluster of sites to the non-industrial Curtin/Bentley sites. The Welshpool cluster of sites were approximately seven kilometres north-east of the Curtin/Bentley cluster of sites.



Legend: Special Purpose – University Residential / Urban Light / Industrial

Figure 3.4: Map of Welshpool / Curtin area showing location of Curtin/Bentley (non-industrial) cluster of sampling sites in relation to Welshpool (industrial) cluster sites. Source: City of Canning (TPS 40), Belmont (TPS 15) and the Town of Victoria Park (TPS 1).

3.2.2 Sampling & equipment

Sampling methods were selected to comply with AS/NZS 3580.1.1:2007, *Methods for sampling and analysis of ambient air. Part 1.1 – Guide to siting air monitoring equipment* (Standards Australia, 2007) and US-EPA standard methods where possible. Gravimetric sampling was conducted in both locations. Sampling methodologies were selected to comply with AS/NZS 3580.10.1.2003 *Methods for sampling and analysis of ambient air* (*Determination of particulate matter - Deposited matter - Gravimetric method)* (Standards Australia, 2003) where possible, as well as to allow comparison with previous studies.

The sampling equipment used for this study included a High Volume Sampler (Control Engineering, WA) and a Nano-MOUDI II™ 125B Multistage Low Pressure Cascade Impactor (MSP. Corporation, MN, U.S.A.) A summary of the equipment characteristics is given in Table 3.4.

Table 3.4: Summary of characteristics for equipment used to monitor the Welshpool and Curtin/Bentley clusters of sites.

	High Volume Sampler	**Nano-MOUDI II™ 125B**
Manufacturer	Control Engineering WA	MSP Corp., Shoreview, MN, Minnesota, U.S.A.
Method	Filtration	Impaction
Particle size captured	Up to 50 µm, Total Suspended Particles (TSP)	10 nm →10 µm
Parameters measured	Total mass concentration	Total mass concentration
Suitability for qualitative analysis	Suitable for qualitative analysis	Suitable for qualitative analysis

Sampling equipment was transported to each site and placed in a secure location with access to AC power. The Nano-MOUDI II™ 125B was placed inside a well ventilated cabinet to protect the equipment from weather and possible interference. The cabinet was custom modified to allow for sample inlet tubing and also power cables. The High Volume Sampler was placed approximately one meter away from the cabinet. The pump for the Nano-MOUDI II™ 125B was located nearby within 0.5 metres (Figure 3.5). Once the equipment was set up at each site, all power switches and all external points of entry for water on the equipment were sealed with weather proof tape.

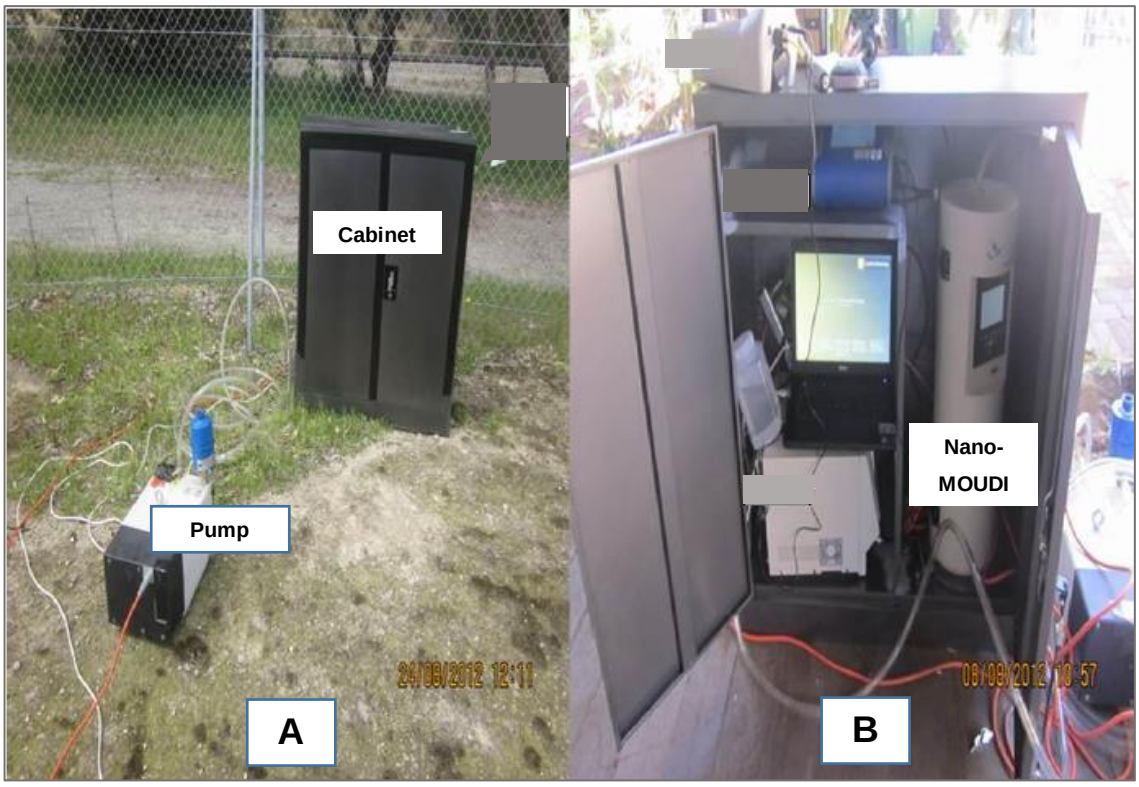


Figure 3.5: Custom made cabinet for air monitoring equipment. A: Exterior of cabinet and pump for Nano-MOUDI II™ 125B. B: Interior of cabinet showing placement of Nano-MOUDI. Remaining equipment not included in this study.

Wind speed and direction data from the South Lake, Caversham and Swanbourne monitoring stations was obtained from the Bureau of Meteorology (BOM), and averaged for the monitoring period. These three monitoring stations were used as they are the closest to the monitored sites and are most likely to represent the ambient conditions in the study area. Rainfall data was also obtained from the BOM at Perth Airport as it is not monitored at individual monitoring stations.

3.2.2.1 High volume sampler

A High Volume Sampler (Hi-Vol) (Control Engineering, WA) was used to sample Total Suspended Particulates (TSP) in accordance with AS/NZ 3580.9.6:2003. *Methods for sampling and analysis of ambient air - Determination of suspended particulate matter - PM_{10} high volume sampler with size-selective inlet - Gravimetric method* (Standards Australia, 2003) and AS3580.9.3: 2003 *Methods for sampling and analysis of ambient air, Determination of suspended particulates matter—Total suspended particulate matter (TSP) High Volume sampler gravimetric method (Standards Australia, 2003).*

High volume air samplers (HVS) are used to collect samples of air to measure the total mass of Total Suspended Particulate matter (TSP). The HVS draws a large known volume of air, typically more than 1500 m^3, through a pre-weighed filter which traps the TSP as air passes through the instrument (Figure 3.6). This type of sampling usually takes place over 5-7 day intervals, depending on the location of sampling. The filter is reweighed once sampling is complete and the PM trapped on the filter can be analysed to determine the composition and concentration of pollutants (Tisch Environmental, 2015).

Prior to sampling for this study a 20 cm X 25 cm Whatman's ashless cellulose filter was weighed and fitted and the flow rate was set to 1000 L/minute using a TSI Anemometer. After sampling, the filter was re-weighed and the difference in filter weight recorded as the collected particulate matter mass. TSP was then calculated by dividing the mass by the volume of air sampled.

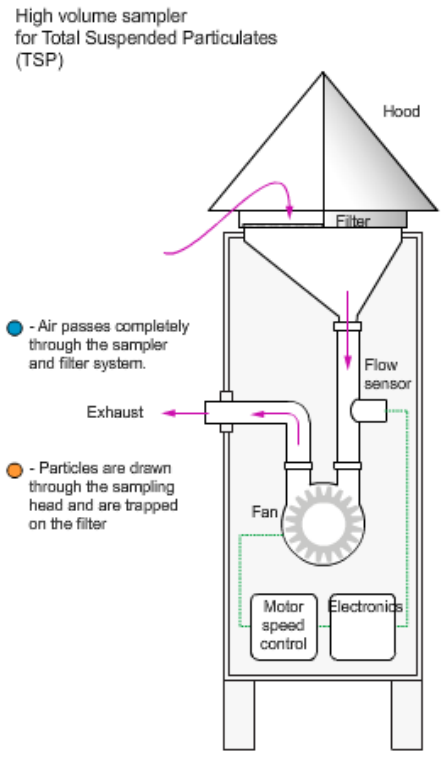


Figure 3.6: Cross section of internal workings of High Volume Sampler (Tisch Environmental, 2015).

3.2.2.2 Nano-MOUDI II™ 125 cascade impactor

The Nano-MOUDI (Micro-Orifice Uniform Deposit Impactor) II™ 125 is a low pressure multi-stage cascade impactor designed for sampling and size-fractionation of airborne particles for gravimetric and/or chemical analysis. It has a series of 13 stages (Figure 3.7 & 3.8) or impactor plates which means that once air samples are drawn in by way of a pump, particles that are not collected by a particular stage flow to the stage below where they are collected (based on their aerodynamic equivalent diameter) with smaller and smaller particles being taken out as the air stream progresses through the stack. The specific sharp nozzle cut off sizes are 10,000 nm, 5,600 nm, 3,200 nm, 1,800 nm, 1,000 nm, 560 nm, 320 nm, 180 nm, 100 nm, 56 nm, 32 nm, 18 nm and 10 nm. There is also an additional stage with a final filter for particles smaller than 10 nm. There are typically low inter-stage losses during sampling (MSP Corporation, 2017).

The Nano-MOUDI II™ 125B uses up to 6000 micro-orifice nozzles for jet acceleration and each stage consists of an impaction plate (with some type of substrate inserted to collect the dust deposit), a motor and a nozzle plate (Figure 3.7). The motor rotates to prevent heavy particle build-up under each nozzle, reduce particle bounce and blow-off and also to give a more uniform deposit of sample on the collecting substrate (in this instance 47 mm diameter aluminium foils) (Figure 3.8).The nozzle plates reduce the jet velocity, pressure drop, particle bounce, and re-entrainment. They also reduce evaporative loss and ensure that sucked air flows in the desired direction (MSP Corporation, 2017). Air flows in through the nozzle plates from the above stage to the below stage and particles are collected on the foil on the impaction place due to kinetic inertia (MSP Corporation, 2017).

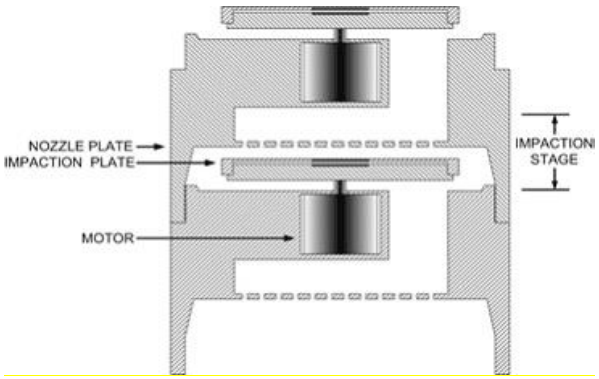


Figure 3.7: Typical Stepper Motor Operated Impaction Stage (MSP Corporation, 2017).

The Nano-MOUDI II™ 125B has built-in sensors that continuously monitor and record the ambient temperature and pressure at pre-determined time intervals during sampling. Absolute pressure sensors monitor the stability of flow and the pressure in the micro-orifice stages. If the micro orifice nozzles of a stage become partially blocked during sampling, the drop in pressure will be recorded. Integrated electronics oversee all aspects of the instrument's operation and also record environmental and pressure data.

Figure 3.8: Typical Rotating Stage with Uniform Deposit in centre of aluminium foil substrate (MSP Corporation, 2017).

Previous studies utilising the Nano-MOUDI II™ 125B have used a range of different impaction substrates to collect the samples The most common substrates used were mixed cellulose ester (MCE) (Fang, 2014; Pekney et al., 2006), Teflon (Polytetrafluoroethylene (PTFE)) (Lin, Chen & Huang, 2005; Gugamsetty et al., 2012; Lough et al., 2005; Taiwo, 2014), polyvinyl chloride (PVC) (Cena, 2014), poly-propylene (Enamorado-Baez et al., 2015), quartz (Moreno et al., 2013; Qi et al., 2016; Sanchez de la Campa et al., 2010) and aluminium (Bertolatti & Rumchev, 2009). Many studies have added silicon grease to their substrates to reduce the likelihood of particle 'rebound' (Cena et al., 2014; Fang et al., 2014; Gugamsetty et al., 2012; Lin, Chen, & Huang, 2005).

In this study, samples were collected on uncoated aluminium foils as previous studies using aluminium substrates found that they actually achieved better elemental characterisation on foils without silicon coating (Bertolatti & Rumchev 2009). The flow rate was set to 10 L/minute using a TSI Flowmeter 4140.

3.2.3 Sample analysis

Previous studies that have established metal profiles have used a variety of chemical and non-chemical methods. Non-chemical metals analysis included Scanning Electron Microscopy (SEM) combined with Energy Dispersive X-ray Spectrometer (EDS) (Bertolatti & Rumchev, 2009). Chemical analysis was most commonly conducted by digesting samples while on the substrates and then subjecting the digest to Inductively Coupled Plasma (ICP) combined with either Atomic Emission Spectroscopy (AES) (Cena et al., 2014; Fang et al., 2014; Moreno et al., 2013; Swami et al., 2001; Wang, Chang, Tsai, & Chiang, 2005) or Mass Spectrometry (MS) (Cena, 2014; Enamorado-Baez et al., 2015; Fang, 2014; Gugamsetty et al., 2012; Lin et al., 2005; Lough et al., 2005; Moreno et al., 2013; Pekney et al., 2006; Qi et al., 2016; Sanchez de la Campa et al., 2010; Swami et al., 2001; Taiwo, 2014; U.S Department of the Interior Geological Survey, 2013; Wang et al., 2005).

Several authors (Cena, 2014; Fang, 2014; Swami et al., 2001) noted that compared to ICP-MS, ICP-AES lacks sensitivity and often selectivity to determine accurately many of the less abundant but more important elements in environmental samples. ICP-MS is therefore more suited to the determination of trace and ultra-trace elements in environmental samples, where concentrations of trace elements are generally lower than workplace levels as pollutants tend to be diluted when emitted into the ambient atmosphere. It also has a lower limit of detection, and a wide linear dynamic range (U.S. Dept of Interior US Geological Survey 2013.

For this study ICP-MS was used for analysis and the samples were prepared as outlined in 3.2.3.1.

3.2.3.1 Preparation of samples for metal analysis

Once the sampling was completed at each site, the Nano-MOUDI II™ 125B stack was disconnected and the foils were removed from each stage, re-desiccated, weighed as per standard methods to determine gravimetric particulate masses, and placed into individual 50 mm petri-dishes (Figure 3.9).

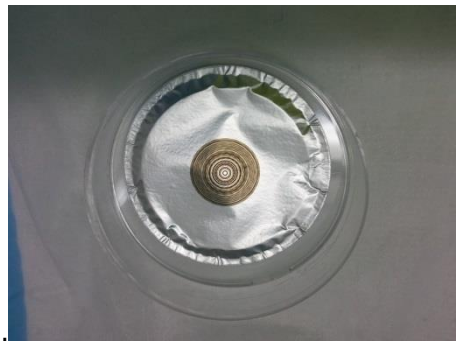


Figure 3.9: Nano-MOUDI II™ 125B foil showing annular pattern of sample distribution.

Dust samples were removed from the foils before digestion as in previous studies where the samples were digested while still on the aluminium foils, the researchers (Personal communication, West 2010) noted high levels of aluminium in the digest and suggested that this may have interfered with the levels of other metals detected through the development of aluminium and other metal complexes.

Sample removal was conducted using an adaption of NIOSH method 7302 *Elements by ICP (Microwave Digestion)* (NIOSH, 2014) as follows:

- 4 mL of ammonium hydroxide (NH_4OH) was decanted for each sample
- Using acid-washed plastic forceps, a small quantity of acid-washed quartz gauze was dipped into the NH_4OH and rubbed over the foil until all visible matter was removed.
- The gauze was then dropped into a *Digi*TUBE and the remaining NH_4OH was poured over the foil to rinse off any residual sample.
- 4 mL (5.652 g) of 70% ultra-pure (99.999% trace metal basis) nitric acid (HNO_3) (Sigma Aldrich) was added to each tube.
- 200 µL (0.222 g) of hydrogen peroxide (H_2O_2) was added drop wise to each tube until any reaction (fizzing) ceased.
- Samples were digested by placing tubes in a water bath (with lids on) at 70˚C for 24 hours.
- Blanks (unused foils) were processed and digested with the samples to derive a ‘foil blank’.

All tubes, forceps and quartz wool were acid washed with 70% ultra-pure (99.999% trace metal basis) nitric acid (HNO_3) before they came into contact with the samples.

3.2.3.2 Inductively coupled plasma – Mass spectrometry

Digested samples were quantitatively and qualitatively analysed in the Trace Research Advanced Clean Environment (TRACE) laboratory which is located in the National Association of Testing Authorities (NATA) accredited Physics Department in the Faculty of Science and Engineering at Curtin University. All 156 samples (12 sites x 13 size fractions) plus blanks and standards for all elements were analysed for the forty (40) metals listed in Table 3.5 using a Thermo Fisher ELEMENT 2 Inductively Coupled Plasma – Mass Spectrometry (ICP-MS) with an ESI SC auto sampler using the standard method for metals analysis *AS 4873.1-2005 Recommended practice for inductively coupled plasma-mass spectrometry (ICP-MS).* Values for aluminium were disregarded due to the potential for contamination of the sample from the aluminium foils used for collection. The parameter and sensitivity of the instrument were calibrated daily according to the standard protocols. External standards (2 ppb, 5 ppb, 10 ppb, and 18 ppb) and internal standard (2 ppb, ln) were applied to calibrate the concentrations of trace elements (Qi et al., 2016).

ICP-MS can measure most of the elements in the periodic table with detection limits at or below the parts per trillion (ppt) range (10^{-12}) range (Table 3.5). For some metals and several non-metals it is able to detect concentrations as low as one part in 10^{-15} (part per quadrillion, (ppq)) on non-interfered low-background isotopes. While other methods such as atomic absorption (AA) and inductively coupled plasma-optical emission (ICP-OES) are also capable of measuring concentrations of various elements, ICP-MS has the advantage of being able to detect extremely low levels and it is also the only method that is able to isotopic analysis which is often important in human health studies (PerkinElmer, 2011).

Samples are introduced as aerosolised droplets into an argon plasma. The plasma dries the aerosol, dissociates the molecules, and removes an electron to form singly-charged ions, which are separated and directed into the MS which sort ions by their mass-to-charge ratio. The ratio can be set such that only one specific mass-to-charge ratio will be allowed to pass through the MS from the entrance to the exit and then upon exiting, the individual ions are detected and counted. The ions cause the release of a cascade of electrons, which is converted into a measureable pulse. The intensity of the pulse is then compared to those from standard calibration curves to determine the concentration of the element (PerkinElmer, 2011).

Table 3.5: Metals and non-metals tested for using the ICP-MS.

Alkali Metals

- Sodium (Na)
- Potassium (K)
- Rubidium (Rb)
- Caesium (Cs)

Alkaline Earth Metals

- Beryllium (Be)
- Magnesium (Mg)
- Calcium (Ca)
- Strontium (Sr)
- Barium (Ba)

Transition Metals

- Scandium (Sc)
- Titanium (Ti)
- Vanadium (V)
- Chromium (Cr)
- Manganese (Mn)
- Iron (Fe)
- Cobalt (Co)
- Nickel (Ni)
- Copper (Cu)
- Zinc (Zn)
- Yttrium (Y)
- Zirconium (Zr)
- Molybdenum (Mo)
- Palladium (Pd)
- Silver (Ag)
- Cadmium (Cd)

Lanthanoids (Metals)

- Lanthanum (La)
- Cerium (Ce)
- Praseodymium (Pr)
- Samarium (Sm)
- Europium (Eu)
- Holmium (Ho)

Other Metals

- Aluminium (Al) ***
- Antimony (Sb)
- Thallium (Tl)
- Lead (Pb)
- Bismuth (Bi)

Actinoids (Metals)

- Thorium (Th)
- Uranium (U)

Non-Metals

- Phosphorus (P)
- Sulphur (S)
- Arsenic (As)

*** Excluded due to possible contamination from the aluminium foils

3.2.3.3 ICP-MS output calculations

For analysis, results for all samples were corrected for dilution and sample blanks. Values in ppt (parts per trillion) were converted to µg/m^3 for comparison with standards and other studies.

Output from the ICP-MS gave concentrations of each metal for each of the 13 stages. To calculate PM_{10}, the mass values for all of the stages were summed. As there is no stage which cuts off precisely at 2.5 µm, levels of $PM_{2.5}$ were calculated by combining the values from the smaller stages (1,800 nm down to less than 10 nm) with one-third of the value of the adjacent stage which collects particles between 1,800 nm and 3,200 nm. This calculation used one-third rather than one-half of the value as the proportion to be included, as the relative mass of the larger particles would skew the mean particle size towards the larger particles. The initial 2 stages which collect particles larger than 3,200 nm (with cut-offs at 10,000 nm and 5,600 nm) were not included in the calculation. (Figure 3.10). To calculate $PM_{0.1}$ the mass values of all of the stages below and including 100 nm (100 nm, 56 nm, 32 nm, 18 nm and 10 nm) were summed. Final concentrations were then compared to published values from previous studies to determine the level of risk to the local community.

Stage #	Stage cut off size	PM_{10}	$PM_{2.5}$	$PM_{0.1}$
1	10,000 nm (10.0 µm)			
2	5,600 nm (5.6 µm)			
3	3,200 nm (3.2 µm)			
4	1,800 nm (1.8 µm)			
5	1,000 nm (1.0 um)			
6	560 nm (0.56 µm)			
7	320 nm (0.32 µm)			
8	180 nm (0.18 µm)			
9	100 nm (0.10 µm)			
10	56 nm (0.056 µm)			
11	32 nm (0.032 µm)			
12	18 nm (0.018 µm)			
13	10 nm (0.010 µm)			

Figure 3.10: Representation of stages included in PM_{10}, $PM_{2.5}$ and $PM_{0.1}$ calculations.

CHAPTER 4: Results

This chapter presents the results for both stages of the study, including the descriptive and inferential analysis of the hospital and air quality data. It also includes presentation and interpretation of the monitoring data collected in two areas of metropolitan Perth. Tables within this chapter generally show only relevant data. More comprehensive tables of results are located in the Appendices.

4.1 Stage 1: Hospital admissions data

Data received from the Western Australian Department of Health was screened to exclude admissions that did not fit the inclusion criteria described in the method (Section 3.1.1) and summarised below.

4.1.1 Inclusion criteria

The inclusion criteria as given in 3.1.1 stipulated that:

- Case must be admitted from 1 January 2001 to 31 December 2010, inclusive of both dates
- Admission must be an emergency presentation
- Admission must be to a Perth metropolitan hospital
- Patients must be residents of the WA metropolitan area

From the original 717,004 respiratory and cardiovascular admissions, 446,425 were removed which left a total of 270,579 admissions (130,936 respiratory cases and 139,973 cardiovascular cases). An additional 116,519 admissions for gastrointestinal / digestive conditions (that met the inclusion criteria) were used as controls.

The complete data set used for this study contained 387,428 non-scheduled admissions to Perth metropolitan hospitals for respiratory, cardiovascular or gastrointestinal conditions between 1 January 2001 and 31 December 2010. There were 216,564 individual patients, many with multiple admissions. Nine patients were admitted on more than 60 occasions during the 10 year time period. Table 4.1 summarises the number of cardiovascular and respiratory cases removed from the original data set.

Table 4.1: Summary of cases removed that did not fit the inclusion criteria for cardiovascular and respiratory hospital admissions (1 January 2001 - 31 December 2010).

Criteria for inclusion / exclusion	Number Removed	Remaining
Total number of respiratory and cardiovascular admissions		717,004
Outside specified date	406	716,598
Admission type		
'Elective', 'elective (from waitlist & not from waitlist)'	292,374	424,224
Rural hospital admissions	112,735	311,489
Non-metropolitan residences		
• Rural WA	37,041	274,448
• Overseas	1,317	273,131
• Other states & Territories	2,387	270,744
• Errors, other codes	165	270,579
Remaining cases		**270,579**

4.1.1.1 Admission type

Of the 387, 428 non-scheduled admissions, the majority were categorised simply as 'emergency' (351,375) (90.7%), with an additional 8% (30,942) classified as 'emergency, with admission through the emergency department' and the remaining 1.3% (5,111) classified as 'emergency with direct admission'. All three descriptors met the inclusion criteria of non-waitlisted admissions. All elective and wait-listed admissions were removed.

4.1.1.2 Hospital classifications

The majority of admissions were to hospitals that were classified as 'tertiary' hospitals (61.7%), almost a quarter (23.7%) were to hospitals classified as 'private metropolitan' and the remaining 14.7% were admitted to hospitals classified as 'public metropolitan'.

4.1.2 Demographics of the admissions

Descriptive analysis of the available demographic data was performed and the following results obtained.

4.1.2.1 Gender

Just over half of the group, (204,996) (52.9%) were male and 47.1% female (182,432). In the gastrointestinal and asthma categories there was little variation in gender distribution however in the cardiovascular, COPD, pneumonia/ influenza/acute bronchitis and 'other respiratory' groups, the proportion of admissions were overwhelmingly male, with up to 12.2%, 7.6%, 11.2% and 16% difference in admissions respectively. (See Table 4.2 for gender distribution for all conditions).

4.1.2.2 Age

Almost half of admissions for all conditions were in the over 65 years age group (183,056) (47.2%), followed by the 15-65 years age group, (151,647) (39.1%), with fewest admissions in the less than 15 years age group (52,725) (13.6%). The mean age for all admissions was 55.5 years ± 28.0 years (range 0-108 years). Table 4.2 presents the mean ages for all conditions overall, by gender, and seasonally. The lowest unadjusted mean age for any condition was the asthma admissions 20.1 years ± 23.1 years. After seasonally adjusting asthma admissions, the youngest sub-category was found to have occurred in autumn (17.8 ± 21.7 years). The highest unadjusted mean age for any condition was the COPD admissions (72.9 ± 12.1 years), which when seasonally adjusted, were highest in spring (73 ± 12.1 years).

Table 4.3 presents the summary statistics for number of admissions for each illness condition on the basis of age group. For gastrointestinal admissions, which were the control condition, the majority of cases (57.0%) were in the middle age group, 15-65 years. There were substantial more admissions in the less than 15 years age group for asthma (60.5%), and 'other respiratory' illness (46.1%) and in the over 65 years age group, there were more admissions for cardiovascular disease (65.5%), COPD (77.2%) and Influenza/ pneumonia/ acute bronchitis (48.6%) as well as overall (47.2%).

Table 4.2: Summary of hospital admission data showing mean age in years (± SD), mean length of stay in hospital in days (± SD) and gender distribution for each disease condition, overall and per season.

			Gastrointestinal	Cardiovascular	Asthma	COPD	Flu / Pneum/ Acute Bronchitis	Other Respiratory	TOTAL
Summer	**Age**		51.1 ± 24.9	68.9 ± 16.9	19.6 ± 23.0	72.7 ± 11.6	57.8 ± 28.5	36.2 ± 31.6	57 ± 26.3
	Length of stay		4.7 ± 7.3	5.8 ± 8.8	2.2 ± 2.9	7.4 ± 9.0	6.1 ± 7.6	4.3 ± 7.6	5.2 ± 8.0
	Gender	***male***	14,278 (48.8%)	17,970 (56.1%)	1,655 (52.4%)	2,886 (53.3%)	3,085 (55.6%)	4,972 (57.5%)	44,846 (53.3%)
		female	14,985 (51.2%)	14,069 (43.9%)	1,505 (47.6%)	2,532 (46.7%)	2,461 (44.4%)	3,677 (42.5%)	39,229 (46.7%)
Autumn	**Age**		50.9 ± 25.1	69.2 ± 17.0	17.8 ± 21.7	72.8 ± 12.0	57.9 ± 28.8	32.3 ± 32.3	55.8 ± 27.5
	Length of stay		4.9 ± 8.0	6.0 ± 8.9	2.2 ± 2.6	7.7 ± 9.2	6.3 ± 7.7	4.0 ± 6.7	5.3 ± 8.2
	Gender	***male***	14,593 (49.2%)	18,685 (54.3%)	2,532 (52.6%)	3,185 (53.8%)	3,093 (54.2%)	6,123 (58.0%)	48,211 (52.9%)
		female	15,069 (50.8%)	15,721 (45.7%)	2,286 (47.4%)	2,733 (46.2%)	2,609 (45.8%)	4438 (42.0%)	42,856 (47.1%)
Winter	**Age**		51.3 ± 25.1	70.3 ± 16.9	21.8 ± 23.7	72.8 ± 12.4	51.4 ± 32.4	26.5 ± 32.5	54.1 ± 29.7
	Length of stay		4.8 ± 7.5	6.2 ± 8.9	2.5 ± 3.0	7.6 ± 8.5	5.9 ± 9.1	3.9 ± 7.4	5.3 ± 8.2
	Gender	***male***	14,057 (49.7%)	20,461 (54.4%)	3,063 (47.8%)	4,960 (52.2%)	5,750 (51.9%)	9,695 (55.9%)	57,986 (52.6%)
		female	14,211 (50.3%)	17,182 (45.6%)	3,339 (52.2%)	4,547 (47.8%)	5,334 (48.1%)	7,641 (44.1%)	52,254 (47.4%)
Spring	**Age**		50.7 ± 24.9	69.5 ± 17.0	20.5 ± 23.4	73.0 ± 12.1	54.4 ± 31.1	33.3 ± 33.2	55.7 ± 28.0
	Length of stay		4.7 ± 7.9	5.9 ± 8.6	2.3 ± 2.6	7.2 ± 8.4	5.8 ± 7.2	4.1 ± 6.5	5.2 ± 7.9
	Gender	***male***	14,557 (49.6%)	19,848 (55.3%)	2,623 (49.8%)	4,479 (53.1%)	5,176 (51.4%)	7,270 (55.6%)	53,953 (52.9%)
		female	14,769 (50.4%)	16,037 (44.7%)	2,642 (50.2%)	3,951 (46.9%)	4,900 (48.6%)	5,794 (44.4%)	48,093 (47.1%)
Total	**Age**		51.0 ± 25.0	69.5 ± 17.0	20.1 ± 23.1	72.9 ± 12.1	54.6 ± 30.8	31.2 ± 32.7	55.5 ± 28.0
	Length of stay		4.8 ± 7.7	5.9 ± 8.8	2.3 ± 2.8	7.5 ± 8.7	6.0 ± 8.1	4.0 ± 7.1	5.3 ± 8.1
	Gender	***male***	57,485 (49.3%)	76,964 (55.0%)	9,873 (50.3%)	15,510 (53.0%)	17,104 (52.8%)	28,060 (56.6%)	204,996 (52.9%)
		female	59,034 (50.7%)	63,009 (45.0%)	9,772 (49.7%)	13,763 (47.0%)	15,304 (47.2%)	21,550 (43.4%)	182,432 (47.1%)

Table 4.3: Summary statistics for individual age groups and all ages (± SD) for admissions for each illness. The age group with the highest proportion of admissions for each illness condition is bolded.

Age group (years)	Parameter	Gastrointestinal	Cardiovascular	Asthma	COPD	Influenza / Pneumonia/ Acute Bronchitis	Other Respiratory	TOTAL
<15	No. of admissions (% of admissions)	10,314 (8.9%)	1,571 (1.1%)	11,826 **(60.2%)**	75 (0.2%)	6,092 (18.8%)	22,847 **(46.1%)**	52,725 (13.6%)
	Mean age ± SD	6.9 ± 4.8	7.9 ± 4.3	4.5 ± 3.4	5.9 ± 4.8	3.4 ± 3.4	1.8 ± 2.9	3.8 ± 4.09
	Median	7.0	9.0	3.0	4.0	2.0	1.0	2.0
	IQR	8	6	4	10	4	2	5
	Min - Max	0 - 14	0 - 14	0 - 14	0 - 14	0 - 14	0 - 14	0 - 14
15-65	No. of admissions (% of admissions)	66,431 **(57.0%)**	46,620 (33.3%)	6,543 (33.3%)	6,604 (22.6%)	10,555 (32.6%)	14,894 (30.0%)	151,647 (39.1%)
	Mean age ± SD	41.3 ± 14.3	51.9 ± 10.9	37.45 ± 14.1	56.3 ± 8.6	45.0 ± 13.9	37.4 ± 15.5	44.9 ± 14.5
	Median	42	55	37	59	47	36	48
	IQR	25	14	24	10	22	28	23
	Min - Max	15 - 65	15 - 65	15 - 65	15 - 65	15 - 65	15 - 65	15 - 65
>65	No. of admissions (% of admissions)	39,774 (34.1%)	91,782 **(65.6%)**	1,276 (6.5%)	22,594 **(77.2%)**	15,761 **(48.6%)**	11,869 (23.9%)	183,056 **(47.2%)**
	Mean age ± SD	80.77 ± 7.9	79.5 ± 7.5	76.7 ± 7.6	77.9 ± 6.9	80.7 ± 7.9	80.0 ± 7.7	79.24 ± 7.6
	Median	78	79	76	78	81	80	79
	IQR	12	11	12	11	12	12	12
	Min - Max	66 - 106	66 - 106	66 - 100	66 - 102	66 - 108	66 - 105	66 - 108
All	No. of admissions	116,519	139,973	19,645	29,273	32,408	49,610	387,428
	Mean age ± SD	51.0 ± 25.0	69.5 ± 17.0	20.1 ± 23.1	72.9 ± 12.1	54.6 ± 30.8	31.2 ± 32.7	55.5 ± 28.0
	Median	52.0	73.0	8.0	75.0	64.0	19.0	63
	IQR	42	22	32	15	50	63	43
	Min - Max	0-106	0-106	0-100	0-102	0-108	1-105	0 - 108

4.1.2.3 Country of birth

The majority of cases were listed as being born in Western Australia (40.4%), with a further 8.6% listed in 'other Australian states and territories', 5% were listed as being born in 'Australia' and a further 9.2% listed as being born in Oceania Antarctica. Twenty-eight point two per-cent were born in Europe and the U.K., 2.5% in Africa, 4.5% in Asia and 0.7% in the Americas.

4.1.2.4 Indigenous Status

The majority of admissions (96.9%) identified as neither Aboriginal nor Torres Strait Islanders (TSI), 3.1% identified as Aboriginal, and less than 1% identified as either TSI or TSI and Aboriginal.

4.1.2.5 Employment status

Just over two-fifths of the admissions were categorised as 'pensioners' (41.5%), with the remainder being categorised as 'employed' (16.6%), 'child not at school' (9.6%), 'retired' (9.1%), 'student' (5.5%), 'home duties' (4.3%), 'unemployed' (2%), and 'other' (11.4%).

4.1.2.6 Marital Status

Almost half of the admissions (46%) were either married or in a 'de facto' relationship, 27.8% had never married, 17.6% were widowed, and 7.8% were either divorced or separated.

4.1.3 Diagnosis classifications of admissions

Admissions were included based on primary diagnosis as recorded on file at separation. The primary diagnoses were classified according to the ICD-10 codes (International Statistical Classification of Diseases and Related Health Problems Revision 10) and were as follows:

- K00-K93: Diseases of the digestive / gastrointestinal system (116,519) (30.1%);

- I00-I99: Diseases of the circulatory / cardiovascular system (139,973), (36.1%); and
- J00-J99: Diseases of the respiratory system (130,936) (33.8%).

Of the 130,936 admissions for diseases of the respiratory system, the cases were further categorised into the following ICD sub-categories:

- J45 & J46: Asthma (19,645) (5.1%);
- J40-J44 & J47: Chronic Obstructive Pulmonary Disease (COPD) (29,273) (7.6%);
- J09-J18 & J20: Influenza, Pneumonia & Acute Bronchitis (32,408) (8.4%); and
- All other respiratory admissions were grouped into 'Other respiratory' (49,610) (12.8%)

During the data collection period (2001-2010), the Department of Health changed from using ICD-9 to ICD-10. This did not affect the categorisation of cardiovascular or gastrointestinal conditions however there were changes in the respiratory sub categories. The primary diagnosis for all cases initially categorised under ICD-9 were re-categorised under the ICD-10 Revision of the Code.

4.1.4 Admissions for disease categories (2001-2010)

Plots were constructed of the number of admissions in each category to determine whether there were any patterns in temporal distribution over the ten year period 2001-2010 (Figures 4.1 – 4.7) (Figures should be viewed in colour). All plots showed a fairly consistent pattern of the monthly number of admissions over the ten year period. There were however overall yearly increases which correlated with the increase in Perth's population over this time period (ABS, 2011).

As can be seen from Figure 4.1, mean daily gastrointestinal admissions on a month to month basis were fairly stable with little indication of monthly or seasonal variation. This category was therefore used as the control group which is consistent with many other studies where gastrointestinal admissions have used as the control group in air pollution studies (Hinwood et al., 2006).

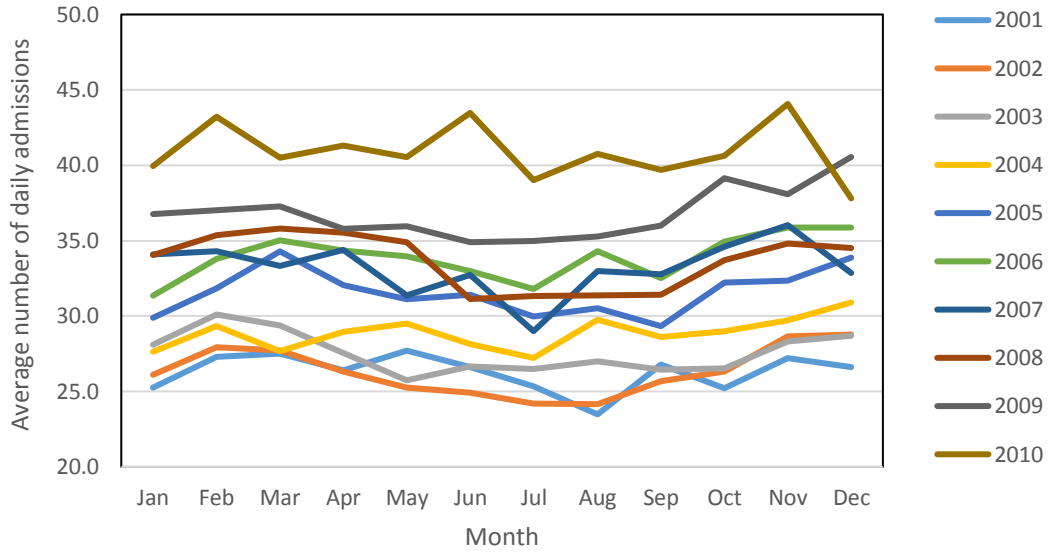


Figure 4.1: Mean number of daily hospital admissions for gastrointestinal conditions (2001-2010).

To account for the population increase over time, the mean daily number of admissions for cardiovascular and respiratory conditions (total as well as sub-categories) (Figures 4.2-4.7) were standardised by dividing by the mean daily number of gastrointestinal conditions for the corresponding time period (the control group).

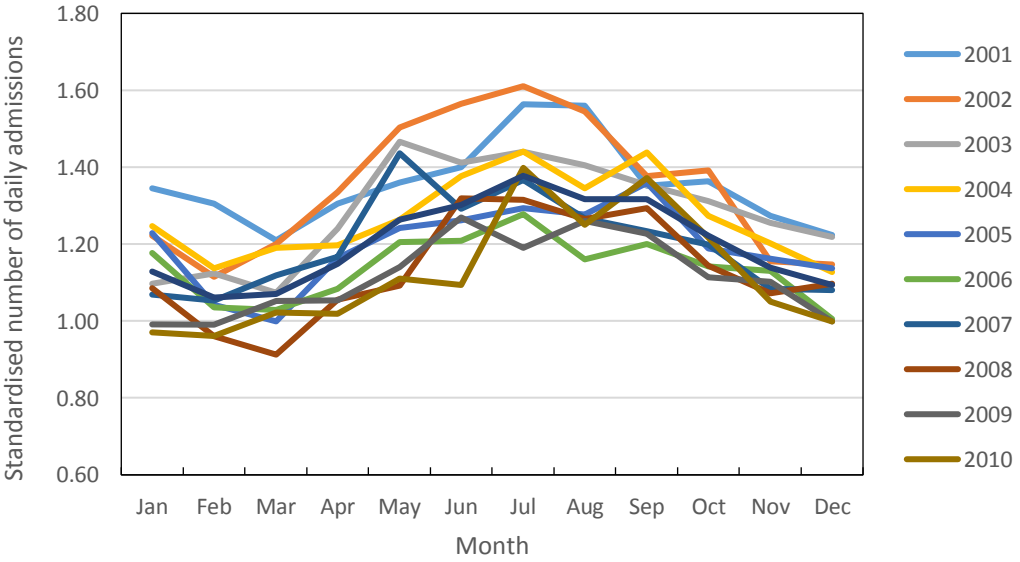


Figure 4.2: Standardised number of hospital admissions for cardiovascular conditions (2001-2010).

Figure 4.2 illustrates the distribution of CVD admissions with increases over the cooler months from April/May to September. The fewest admissions consistently occur around February and March each year.

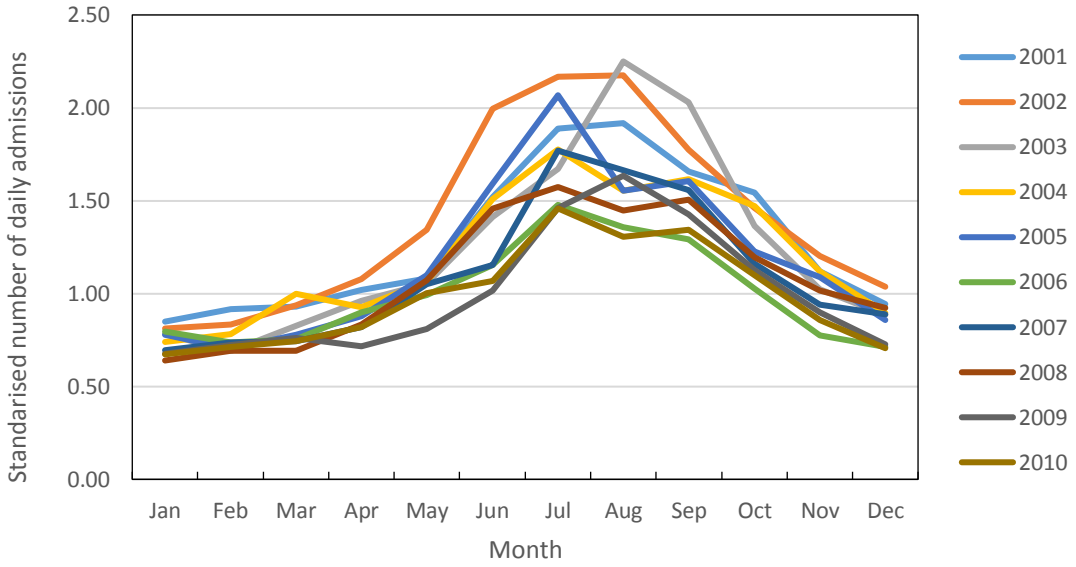


Figure 4.3: Standardised number of daily hospital admissions for total respiratory conditions (2001-2010).

Fig 4.3 shows total respiratory admissions (all respiratory conditions combined) peaked between the cooler months of July and September with the fewest admissions consistently occurring in the warmer months of January & February.

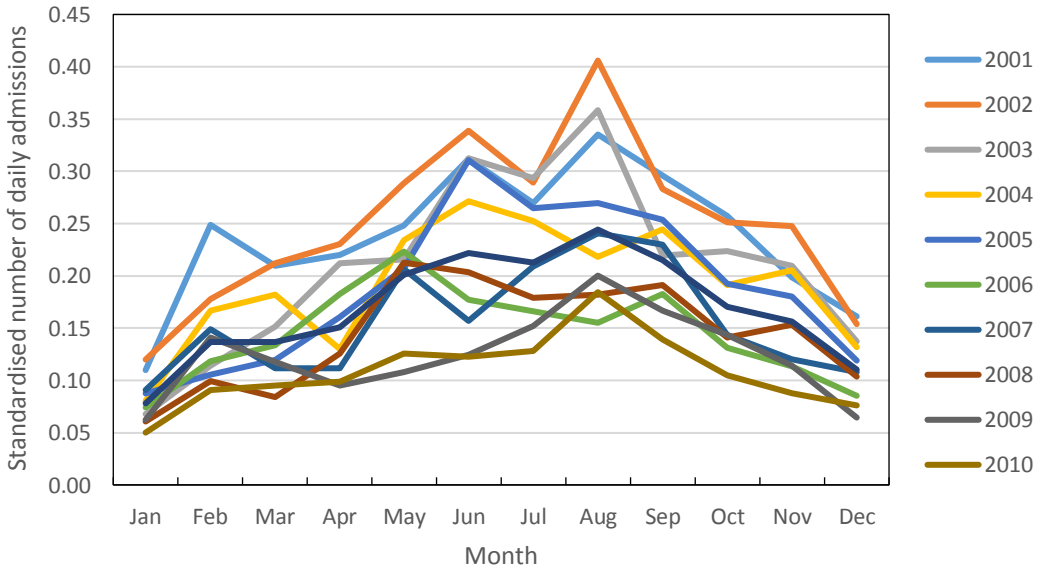


Figure 4.4: Standardised number of asthma admissions (2001-2010).

The number of asthma admissions (Figure 4.4) showed an increasing trend over the cooler months from April/May and peaking in August/September with fewest admissions consistently lowest during the warmer months of December and January.

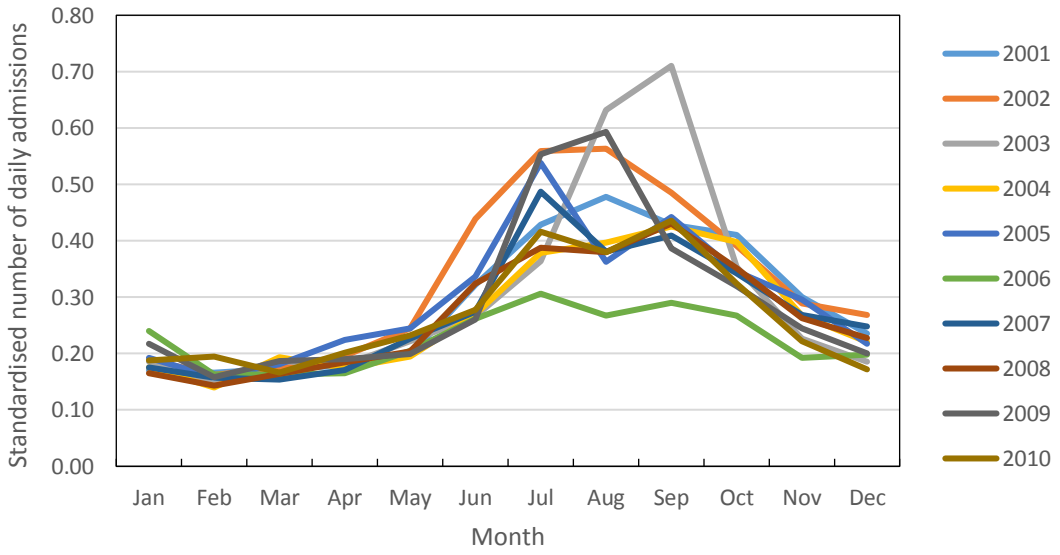


Figure 4.5: Standardised number of daily influenza / pneumonia / acute bronchitis admissions (2001-2010).

The number of admissions for lower respiratory conditions, influenza, pneumonia, and acute bronchitis (Figure 4.5) peaked during winter to early spring, July to September with the fewest admissions in the warmer months around February.

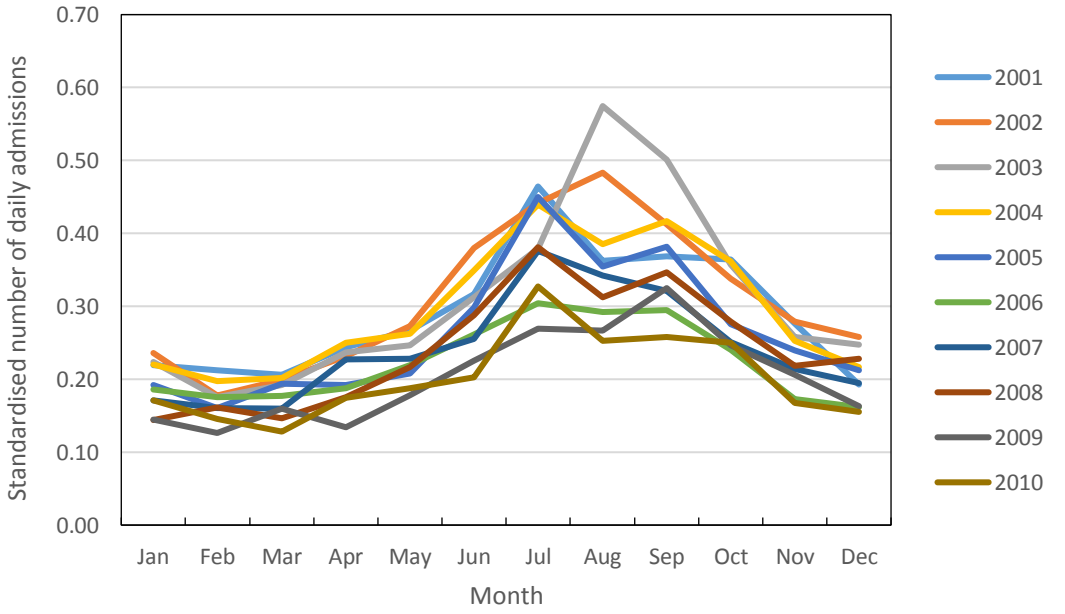


Figure 4.6: Standardised number of daily chronic obstructive pulmonary disease (COPD) admissions (2001-2010).

The number of admissions for COPD (Figure 4.6) peaked during winter to early spring, July to September with the fewest admissions in the warmer months around February and March.

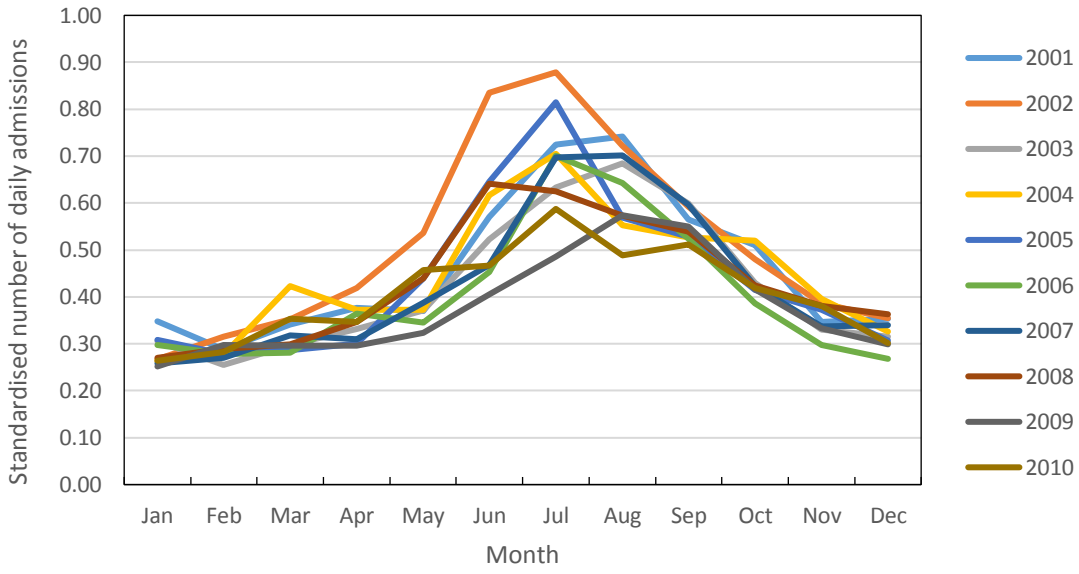


Figure 4.7: Standardised Number of 'Other respiratory' admissions (non-Asthma, non-COPD, non-influenza/pneumonia/acute bronchitis (2001-2010).

The number of admissions for 'Other Respiratory 'conditions (Figure 4.7) peaked during winter to early spring, June to August with the fewest admissions in the warmer months from December to February.

4.1.5 Length of stay

The mean length of stay for all admissions include in this study was 5.3 days ± 8.1 days. This varied between different disease conditions and seasons. Overall the shortest mean length of stay was for asthma admissions which was 2.3 days ± 2.8 days and the longest mean length of stay was 7.5 days ± 8.7 days for COPD admissions. When this is examined seasonally, by condition, asthma admissions are consistently the shortest mean length of stay and COPD is consistently the longest mean length of stay. See Table 4.2 for length of stay results for all health conditions, overall and seasonally.

4.1.6 Comparison of the number of admissions between decades

The mean daily numbers of hospital admissions for each selected disease conditions from 2001-2010 were compared to the corresponding figures from 1992-1998 as published by Hinwood et al. (2006). Mean admissions were calculated over the whole year and also for the warmer months of November to April and cooler months of May to October. Although these months do not align with the standard Bureau of Meteorology definition of season they were used so that comparisons could be made with the previous published data.

The mean number of admissions for each time period were standardised by the mean population of metropolitan Perth over the corresponding time period (Australian Bureau of Statistics. Regional Population Growth, Australia Cat. No. 3218.0, 2017) to take into account the substantial increase in the population of the Perth metropolitan area from one decade to the next.

- 1 January 1992 – 31 December 1998, Mean population 1.282 mil ± 0.042 mil (Range 1.23 mil – 1.33 mil)

- 1 January 2001 – 31 December 2010, Mean Population 1.484 million ± 0.144mil (Range 1.33 mil – 1.70 mil)

There was a marked increase in admissions for cardiovascular and all respiratory conditions except asthma, overall and across warmer and cooler months. This increase was observed even after standardisation. Within each decade there was little seasonal variation for either gastrointestinal or cardiovascular illnesses, however there was consistent variation with seasonal distribution for respiratory admissions. During both decades there is a marked increase in the mean number of admissions during the cooler months of May-October for all categories of respiratory illnesses, including total respiratory (Table 4.4).

Table 4.4: Comparison of mean daily admissions (± SD) and standardised mean for selected illness conditions between 1992-1998 and 2001 – 2010.

	Mean Number of Daily Hospitalisations (± SD)					
	1 January 1992 – 31 December 1998 ^			1 January 2001 – 31 December 2010		
	Nov-April	**May-October**	**All Year**	**Nov-April**	**May-October**	**All Year**
Gastrointestinal	17.2 ± 5.0	16.3 ± 4.8	16.8 ± 4.9	32.7 ± 4.7	31.2 ± 4.8	31.9 ± 4.8
Cardiovascular	24.9 ± 6.4	28.1 ± 6.4	26.5 ± 6.6	36.2 ± 3.1	40.5 ± 4.2	38.3 ± 4.2
Total Respiratory	19.3 ± 6.0	31.0 ± 8.1	25.3 ± 9.5	27.3 ± 4.0	44.3 ± 7.9	35.9 ± 10.6
Asthma	6.9 ± 3.7	10.7 ± 4.1	8.8 ± 4.3	4.2 ± 1.3	6.6 ± 1.4	5.4 ± 1.8
COPD	2.4 ± 1.7	4.1 ± 2.3	3.3 ± 2.2	6.3 ± 0.9	9.7 ±1.8	8.0 ± 2.3
Pneumonia / influenza/ acute bronchitis	3.6 ± 2.1	5.7 ± 3.1	4.6 ± 2.9	6.4 ± 1.5	11.3 ± 3.4	8.9 ± 3.6
	Standardised mean number of daily hospitalisations (± SD)					
Gastrointestinal	13.4 ± 3.9	12.7 ± 3.7	13.1 ± 3.8	22.0 ± 3.2	21.0 ± 3.2	21.5 ± 3.2
Cardiovascular	19.4 ± 5.0	21.9 ± 5.0	20.7 ± 5.1	24.4 ± 2.1	27.3 ± 2.8	25.8 ± 2.8
Total Respiratory	15.1 ± 4.7	24.2 ± 6.3	19.7 ± 7.4	18.4 ± 2.7	29.9 ± 5.3	24.0 ± 7.1
Asthma	5.4 ± 2.9	8.3 ± 3.2	6.9 ± 3.4	2.8 ± 0.9	4.4 ± 0.9	3.7 ± 1.2
COPD	1.9 ± 1.5	3.2 ± 1.8	2.6 ± 1.7	4.2 ± 0.6	6.5 ± 1.2	5.4 ± 1.5
Pneumonia / influenza/ acute bronchitis	2.8 ± 1.7	4.4 ± 2.4	3.6 ± 2.3	4.3 ± 1.0	7.6 ± 2.3	6.0 ± 2.4

^ Figures as presented in Hinwood, et al. (2006)

4.2 Ambient air pollutant levels and meteorological parameters 2001-2010

Table 4.5: Descriptive statistics for overall and seasonal pollutant levels and meteorological parameters in the Perth metropolitan areas from 2001 – 2010.

Pollutant	Mean	Median	IQR	Min	Max
CO, 8 hr avg (ppm)	0.22 ± 0.2	0.2	0.2	0.0	3.0
Summer	0.12 ± 0.1	0.1	0.1	0.0	1.1
Autumn	0.25 ± 0.3	0.2	0.2	0.0	2.4
Winter	0.39 ± 0.4	0.3	0.4	0.0	3.0
Spring	0.15 ± 0.1	0.1	0.1	0.0	1.7
NO_2, 24 hr avg (ppb)	6.1 ± 3.3	5.6	4.8	0.3	19.2
Summer	4.1 ± 1.8	3.9	2.4	0.8	11.6
Autumn	7.2 ± 3.3	6.9	4.7	0.5	19.2
Winter	8.1 ± 3.4	8.6	5.1	0.0	16.0
Spring	5.0 ± 2.5	4.8	3.4	0.5	16.0
Ozone, 24 hr avg (ppb)	19.1 ± 5.0	18.7	7.1	7.5	37.4
Summer	19.2 ± 4.3	18.7	5.3	8.8	36.0
Autumn	17.3 ± 4.1	17.1	5.6	7.5	31.7
Winter	17.9 ± 6.0	16.4	8.7	7.7	37.4
Spring	22.2 ± 4.0	21.9	5.3	10.0	34.9
PM_{10}, 24 hr avg (µg/m^3)	17.0 ± 6.5	15.8	7.8	0	68.3
Summer	21.3 ± 7.4	20.3	9.2	3.9	68.3
Autumn	17.1 ± 6.0	16.3	7.1	2.9	57.1
Winter	14.0 ± 4.3	13.7	5.1	5.0	39.0
Spring	15.5 ± 5.5	14.5	6.7	5.6	45.6
$PM_{2.5}$, 24 hr avg (µg/m^3)	8.0 ± 3.1	7.4	3.2	2.8	40.8
Summer	8.6 ± 3.3	7.9	3.4	3.2	40.5
Autumn	8.1 ± 3.2	7.4	3.2	3.0	40.8
Winter	8.0 ± 2.9	7.5	3.5	2.8	26.7
Spring	7.4 ± 2.7	6.9	2.6	3.5	29.3
Temperature (°C) 24 hr	18.1 ± 4.5	17.5	7	8	33
Summer	22.5 ± 3.3	22.2	5	15	33
Autumn	19.3 ± 3.6	18.8	5	11	33
Winter	13.7 ± 2.0	13.7	3	8	19
Spring	16.9 ± 3.2	16.3	4	10	29
Relative Humidity (%)	63.1 ± 14.8	65.3	21	16	100
Summer	54.2 ± 13.1	55.0	19	19	84
Autumn	61.3 ± 15.6	63.7	24	16	72
Winter	72.4 ± 11.0	73.5	13	32	100
Spring	64.4 ± 13.0	66.6	15	20	95

Table 4.5 shows descriptive statistics for overall and seasonal pollutant levels and meteorological parameters in the Perth metropolitan areas from 2001 – 2010. These levels were all well below the National Air Quality Standards for the corresponding time periods.

Figures 4.8 – 4.12 were produced of the mean daily levels for each of the studied pollutants, for each month over the 10 year period of the study, to determine whether there were any temporal or seasonal patterns present in monitored levels.

4.2.1 Carbon monoxide

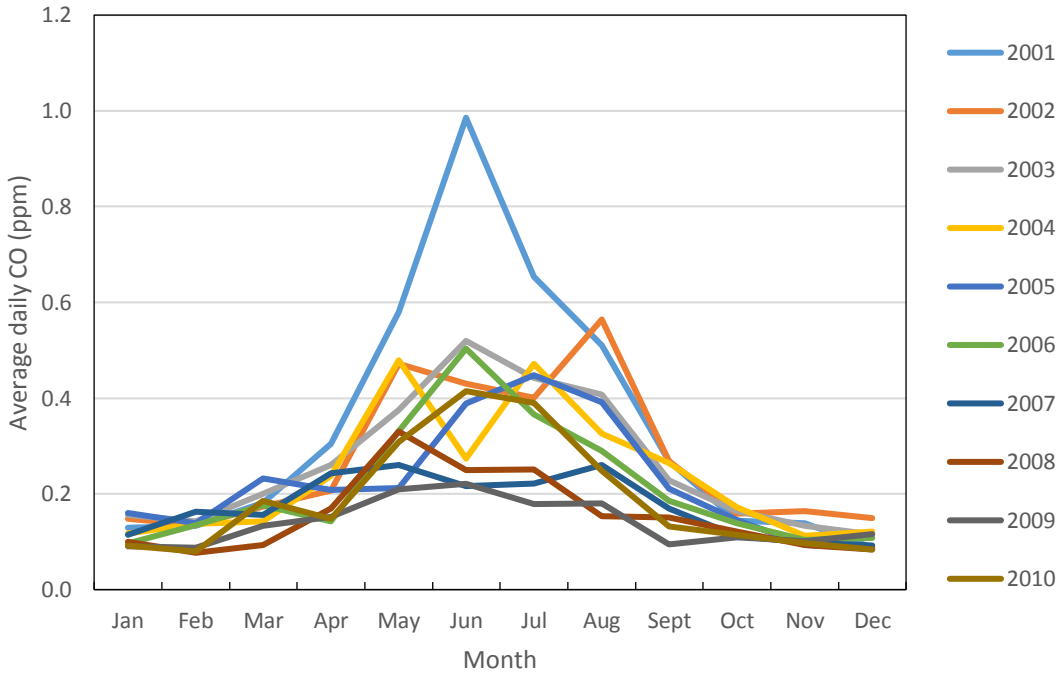


Figure 4.8: Mean daily levels of carbon monoxide from 2001 – 2010

There is a pattern in the CO levels with a slight increase in CO levels over the cooler months, with a higher than average peak in 2001. This peak was caused by higher that usual results at the Duncraig monitoring station only on the 22nd and 23rd June, which is indicative of a localised combustion issue. Levels were at their lowest during the warmer months of November through to February

4.2.2 Nitrogen dioxide

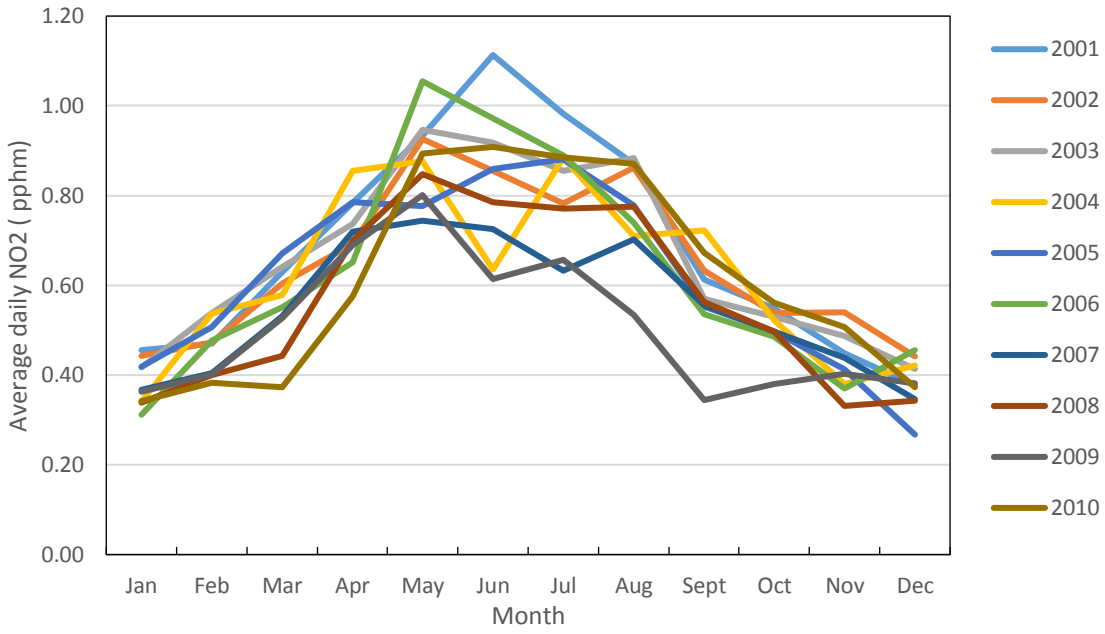


Figure 4.9: Mean daily levels of nitrogen dioxide from 2001 – 2010

Nitrogen dioxide levels show a fairly consistent pattern across all years with levels at their lowest in the warmer months of December & January and increasing in the cooler months, peaking in the cooler months around June.

Mean CO and NO_2 levels both peaked in the cooler months of June and July and were at their lowest in the summer months of December and January. These two pollutants are both associated with combustion, including vehicle emissions, bushfires and domestic wood heaters. As the level of vehicle emissions is fairly consistent throughout the year, the winter peaks are likely to be due to domestic wood heaters. Although in warmer periods the Perth metropolitan area is subject to bushfires, these are frequently only very small and managed locally so are unlikely to affect air quality over the whole metropolitan region. In many areas across the metropolitan area, particularly the outer suburbs, a substantial number of domestic wood heaters burn or at least smoulder constantly for long periods.

4.2.3 Ozone

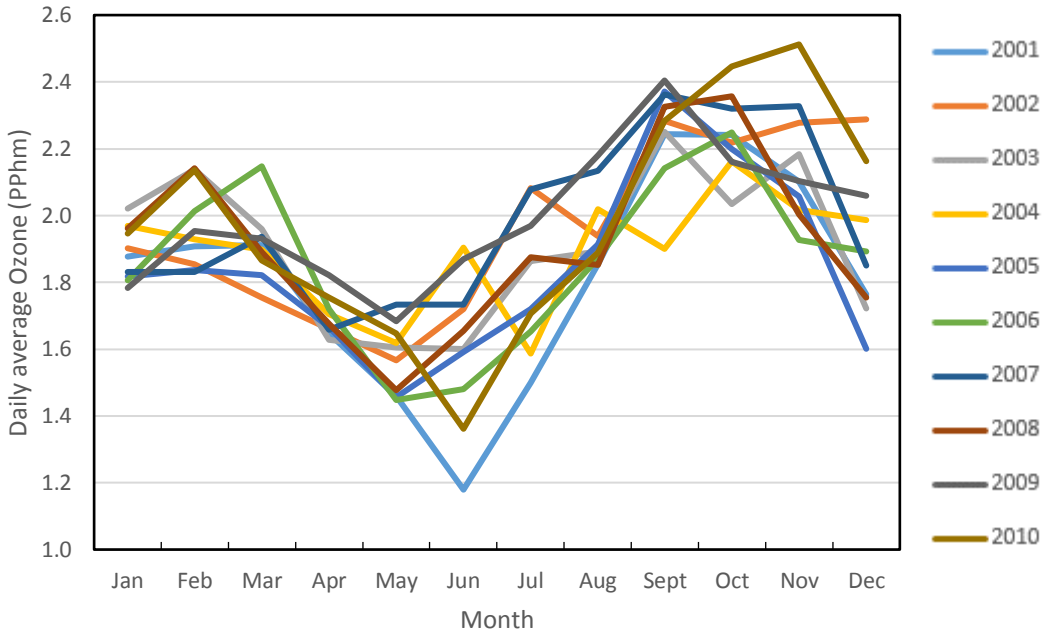


Figure 4.10: Mean daily levels of ozone from 2001 – 2010.

There is a marked increase in ozone levels over the spring months, peaking from September to November, remaining quite high over December and January and reaching their lowest levels from April to June. Ozone production is associated with warmer temperatures and generally peaks in either spring or summer, depending on other localised meteorological factors, such as humidity, wind speed and direction, cloud cover and stratospheric-tropospheric ozone exchange (Diem, 2013). Generally, warm dry weather is more conducive to ozone formation than cool, wet weather. (U.S.EPA 2014)

4.2.4 PM_{10}

Levels of PM_{10} show a consistent pattern of lower levels in the cooler months and increasing in the warmer months, peaking in December – February. This is consistent with the peak period for bushfires and dust storms in WA. PM_{10} levels are at their lowest in the cooler months of July to September which also coincides with the period of most rainfall which would reduce the concentration of PM in the ambient air.

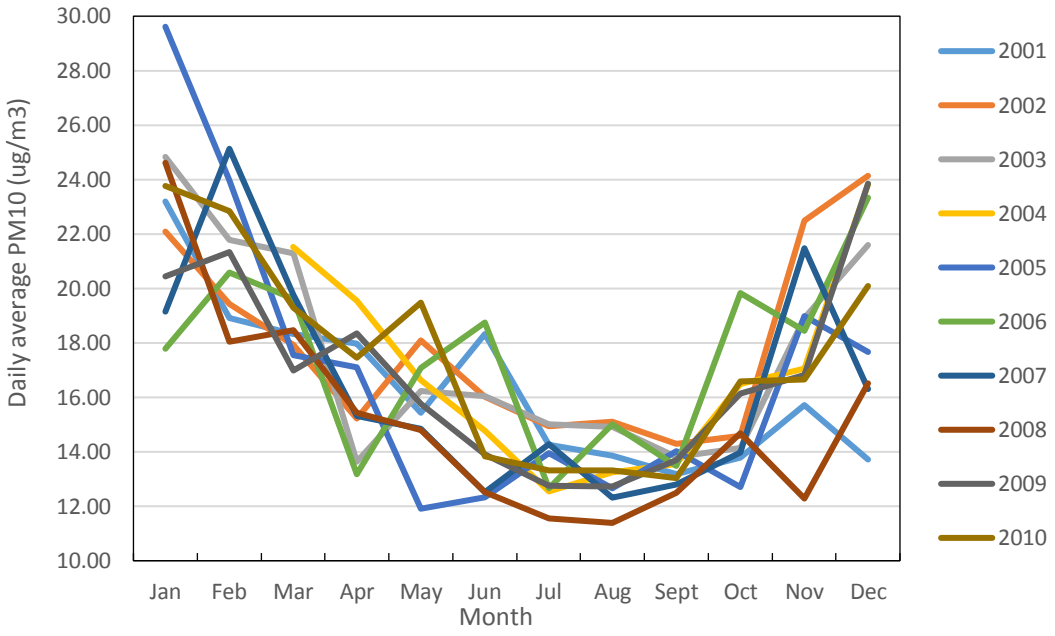


Figure 4.11: Mean daily levels of PM_{10} from 2001 – 2010

4.2.5 $PM_{2.5}$

There does not appear to be a consistent trend in $PM_{2.5}$ levels across the 10 year period. $PM_{2.5}$ levels are closely associated with traffic levels which tend not to vary seasonally.

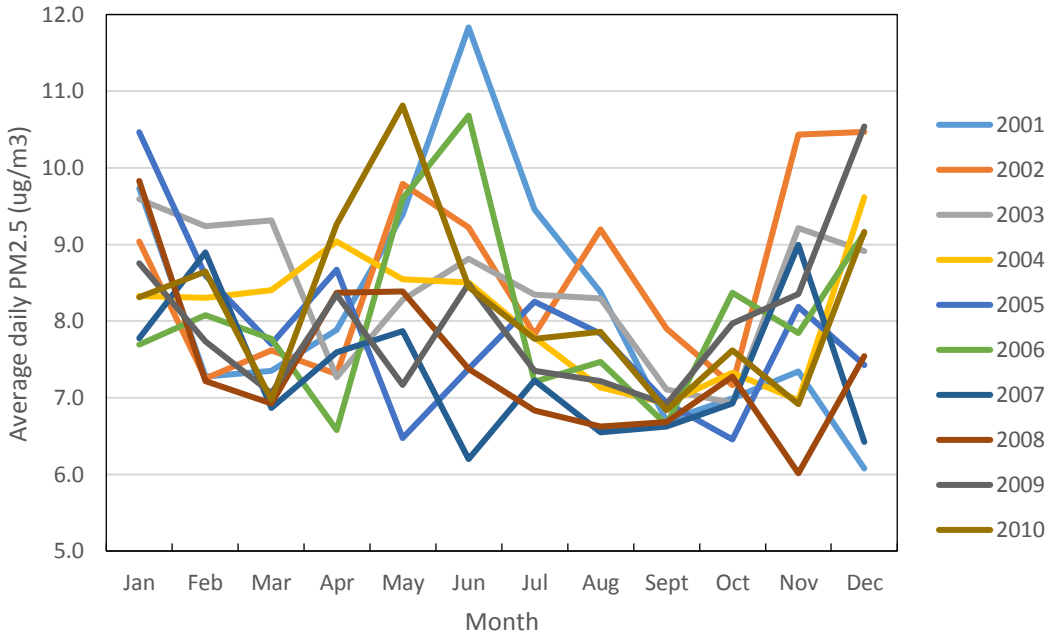


Figure 4.12: Mean daily levels of $PM_{2.5}$ from 2001 – 2010

Table 4.5 shows the overall yearly and seasonal mean, median, interquartile range, maximum and minimum for all pollutants and meteorological parameters.

Table 4.5 is consistent with the plotted values and shows for the Perth metropolitan area (Figures 4.8 - 4.12), that mean temperatures are highest in summer and lowest in winter. Humidity is highest in winter and lowest in summer and autumn. Nitrogen dioxide and carbon monoxide concentrations are highest in the cooler months, particularly winter while ozone and PM_{10} concentrations are highest in the warmer months, particularly summer. $PM_{2.5}$ demonstrates little seasonal variation, showing only a slight dip in spring, which in the context of the averaging smoothing process cannot be interpreted as significant.

4.2.6 Temperature and humidity

Mean temperature levels over the ten year period show that temperatures consistently peaked in the summer months and were lowest in the winter months. Humidity is the reverse and was highest in winter and lowest in summer (Table 4.5). This is to be expected as Perth has a temperate climate and receives most of its' annual rainfall in the cooler winter months.

4.2.7 Comparison of pollutant levels between decades

Mean levels of each of the pollutants of concern were calculated and compared to levels reported in a similar study investigating the association between pollutant levels and hospital admissions conducted by Hinwood et al. (2006) (Table 4.6). It should be noted that these figures are indicative only as due to the averaging process over such a large data set, any peaks or exceedances are smoothed out. Generally the number of exceedances is a better indication of air quality over a given period however this information is not available for the 1992-1998 data.

Table 4.6 shows that there has been a substantial increase in mean levels of NO_2 for a 1-hour averaging period from 1992-1998 to 2001-2010. These mean levels increased for both the warmer and cooler months separately (24.7 ppb to 41.0 ppb and 24.9 ppb to 40.7 ppb, respectively) and also over the whole year from 24.8 ppb to 41.0 ppb. In contrast there was a decrease in mean NO_2 levels over a 24-hour averaging period for warmer and cooler months (9.6 ppb to 4.9 ppb and 11.1 ppb to 7.3 ppb respectively) and the whole year (10.3 ppb to 6.1 ppb).

Mean ozone levels increased substantially for both the 1-hour and 4-hour averaging periods. For the 1-hour period, in the warmer and cooler months they increased from 35.0 ppb to 68.2 ppb and 28.3 ppb to 45.3 ppb respectively. Over the whole year they increased from 31.6 ppb to 75.3 ppb. For the 4-hour averaging period the increases for warmer and cooler months were from 31.1 ppb to 71.9 ppb and 26.6 ppb to 43.9, respectively. For the whole year the increase was from 28.8 ppb to 71.9 ppb.

Mean CO levels decreased slightly over the warmer months (2.2 ppm to 1.3 ppm) and increased slightly over the cooler months and whole year (2.4 ppm to 3.0 ppm and 2.3 ppm to 3.0, respectively). There was very little change in either PM_{10} or $PM_{2.5}$ mean levels between the decades. Likewise there was very little change in temperature or humidity.

Table 4.6: Comparison of mean air pollutant levels and meteorological parameters between 1992-1998 and 2001-2010.

		Mean Pollutant Levels (1 Jan 1992 – 31 Dec 1998)			**Mean Pollutant Levels** (1 Jan 2001 – 31 Dec 2010)		
Pollutant Averaging period	**AAQ NEPM**	**November – April** **Mean ± SD** (10^{th}-90^{th} centile)	**May-October** **Mean ± SD** (10^{th}-90^{th} centile)	**All year** **Mean ± SD** (10^{th}-90^{th} centile)	**November-April** **Mean ± SD** (10^{th}-90^{th} centile)	**May-October** **Mean ± SD** (10^{th}-90^{th} centile)	**All year** **Mean ± SD** (10^{th}-90^{th} centile)
Nitrogen dioxide	120 ppb / hour	9.6 ± 4.8	11.1 ± µ5.1	10.3 ± 5.0	4.9 ± 2.5	7.3 ± 3.5	6.1 ± 3.3
24 hr avg (ppb)	3 ppb/ year	(4.3 – 15.7)	(4.8 – 18.0)	(4.4 – 17.1)	(2.1 – 8.4)	(2.5 – 11.9)	(2.3 - 10.9)
Nitrogen dioxide	120 ppb	24.7 ± 11.1	24.9 ± 8.9	24.8 ± 10.1	41.0 ± 4.6	40.7 ± 6.2	41.0 ± 5.6
1 hr max (ppb)	(0.12 ppm)	(12.4 - 39.2)	(14.4 - 35.7)	(13.3 - 37.5)	(1.2 – 11.8)	(1.5 – 17.1)	(1.3 – 15.0)
Ozone	80 ppb	31.1 ± 9.4	26.6 ± 5.0	28.8 ± 7.8	71.9 ± 10.0	43.9 ± 10.1	71.9 ± 10.1
4 hr max (ppb)	(0.8 ppm)	(20.8 - 45.0)	(21.1 - 32.3)	(21.0 - 39.5)	(7.3 – 32.0)	(3.7 – 30.5)	(5.3 – 30.8)
Ozone	100 ppb	35.0 ± 12.4	28.3 ± 5.6	31.6 ± 10.2	68.2 ± 9.7	45.3 ± 10.5	75.3 ± 10.2
1 hr max (ppb)	0.10 ppm	(22.1 - 53.7)	(22.4 - 34.0)	(22.2 - 46.1)	(6.6 - 30.4)	(2.4 – 30.2)	(3.8 – 30.3)
Carbon monoxide	9.0 ppm	2.2 ± 1.3	2.4 ± 1.2	2.3 ± 1.3	1.3 ± 0.1	3.0 ± 0.4	3.0 ± 0.3
8 hr max (ppm)		(0.8 - 4.2)	(1.1 - 4.2)	(0.9 - 4.2)	(0.1 – 0.3)	(0.1 – 0.7)	(0.1 – 0.5)
PM_{10}	50 µg/m^3	20.6 ± 7.7	18.8 ± 7.8	19.6 ± 7.8	19.5 ± 7.1	14.4 ± 4.6	17.0 ± 6.5
24 hr avg (µg/m^3)		(12.6 - 29.7)	(12.7 - 29.0)	(11.3 - 29.5)	(11.5 – 28.7)	(9.2 – 20.1)	(10.0 – 25.5)
$PM_{2.5}$	25 µg/m^3	8.6 ± 3.8	9.7 ± 4.7	9.2 ± 4.3	8.2 ± 3.3	7.8 ± 2.8	8.0 ± 3.1
24 hr avg (µg/m^3)		(4.9 - 13.1)	(5.1 - 16.2)	(5.0 - 14.5)	(5.2 – 12.0)	(5.1 – 11.1)	(5.1 – 11.6)
Temperature	-	21.8 ± 3.4	15.1 ± 2.5	18.4 ± 4.5	21.4 ± 3.5	14.8 ± 2.5	18.1 ± 4.5
24 hr avg (°C)		(17.8 - 26.7)	(12.0 - 18.2)	(13.0 - 24.7)	(17.2 – 26.0)	(11.8 – 18.0)	(12.8 – 24.3)
Humidity	-	56.2 ± 15.3	71.4 ± 12.	64.0 ± 15.8	56.1 ± 14.2	69.9 ± 11.9	63.1 ± 14.8
24 hr avg (R.H. %)		(35.0 - 75.5)	(53.5 - 85.0)	(40.5 - 82.5)	(36.8 – 73.8)	(53.3 – 83.7)	(41.8 – 80.8)

4.3 Statistical analysis of hospital admissions and levels of pollutants

The following tables (4.7(a)–4.7(e)) summarise the relationship between levels of pollutants and hospital admissions for each illness category. Average daily pollutant levels were categorised into low, medium and high tertiles. The low level of pollutants (tertile 1) was used as the baseline against which the second (medium) and third (high) tertiles were compared. Logistic regression was used to assess the association between pollutants (CO, NO_2, ozone, PM_{10} and $PM_{2.5}$) with a 10% increase in standardised admissions relative to gastrointestinal admissions for cardiovascular illness, total respiratory illness, asthma, COPD, pneumonia/influenza/acute bronchitis and 'other respiratory' illness, before adjusting and after adjusting for seasons. The admissions were standardised (see Section 3.1.3.1 for method). The effect of pollutants on admissions was expressed as an odds-ratio with the associated 95% confidence interval.

For example, in table 4.7 (a) the unadjusted odds-ratio for cardiovascular admissions and NO_2, when NO_2 levels are in the medium tertile (tertile 2) is 1.2 (95% CI: 1.01-1.43). This is interpreted as the risk of hospitalisation is 1.2 times more likely (or an increase of 20%) when NO_2 levels are in the medium tertile (tertile 2) of the pollutant, than when they are in the low tertile (tertile 1), with respect to the 10% increase in standardised cardiovascular admissions. When NO_2 levels are in the high tertile (tertile 3) the odds-ratio for is increased to 2.0 (95% CI: 1.69 – 2.37). This is interpreted as the risk of hospitalisation when NO_2 levels are in the high level (tertile 3) of the pollutant being twice as likely (or an increase of 100%) relative to the low level (tertile 1), with respect to the 10% increase in standardised cardiovascular admissions.

As the cut-point of a 10% increase in standardised admissions was arbitrarily chosen, a sensitivity analysis was undertaken to assess the effect of the pollutants on a 20% increase in standardised admissions before and after adjusting for seasons (Tables 4.7(a) – 4.7(e)). A comparison of the results from both sets of analyses allows us to investigate the effect of the choice of cut-point on conclusions drawn the statistical analyses (Baird, 1989). Still using NO_2 as an example, the odds-ratio for a 20% increase in the number of cardiovascular admissions when NO_2 levels are in the high tertile (tertile 3) is 1.99 (95% CI: 1.65-2.40), which is very similar to the odds-ratio for a 10% increase in admissions. The odds ratio after adjusting for season decreased to 1.47 (95%CI: 1.19 – 1.83)

Some pollutants appear to be protective with significant p-values (<0.005) and odds-ratios less than one, but this effect should be interpreted with caution, as it usually becomes detrimental or non-significant after adjusting for seasons. This anomaly is also reported in DeVries, Kriebel, & Sama (2016).

In summary, notwithstanding tertile classification, seasonal adjustment and magnitude of any increase in the odds-ratio:

- Carbon monoxide levels, and to a lesser extent nitrogen dioxide levels, were found to have significantly increased the likelihood of an increase in hospital admissions for all disease conditions tested - cardiovascular disease, total respiratory disease, asthma, COPD, pneumonia/influenza/ acute bronchitis and other respiratory conditions.

- Ozone levels were found to have increased the likelihood admissions for cardiovascular disease, pneumonia/influenza/acute bronchitis and COPD.

- PM_{10} was not associated with increases in hospital admission for any of the disease conditions studied and $PM_{2.5}$ was only found to be associated with an increase in hospital admissions for asthma.

Table 4.7 (a): Odds ratios and P-values for 10% and 20% increase in 'Cardiovascular' hospital admissions for medium (tertile 2) and high tertiles (tertile 3) of pollutant level compared to the low tertile (tertile 1), before and after adjusting for seasons. Significant values (P<0.05 and OR>1.00) are highlighted in red.

Pollutant (Reported Range) Tertile 1 range		Cardiovascular Admissions (standardised) exceeding Gastrointestinal admissions by ≥ 10%		Cardiovascular Admissions (standardised) exceeding Gastrointestinal admissions by ≥ 20%	
	Tertile Range	Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) Adjusted for season	Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) Adjusted for season
Carbon Monoxide (CO) (0.00-3.04) ppm Tertile 1(0.00 -≤0.11)	2 (middle) (>0.11 - ≤0.21)	1.49 (1.25-1.78) P<0.0005	1.41 (1.18-1.69) p<0.0005	1.59 (1.29-1.95) p<0.0005	1.52 (1.24-1.87) p<0.0005
	3 (high) (>0.21 - ≤3.04)	2.24 (1.88-2.65) P<0.0005	1.63 (1.34-1.98) p<0.0005	2.37 (1.95-2.88) p<0.0005	1.77 (1.42-2.20) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day) Tertile 1 (0.00 - ≤0.042)	2 (middle) (>0.042- ≤0.73)	1.20 (1.01-1.43) p=0.040	1.11 (0.93-1.33) p=0.254	1.10 (0.84 – 1.34) p=0.381	1.024 (0.83-1.26) p=0.821
	3 (high) (>0.73 – ≤1.92)	2.00 (1.69-2.37) p<0.0005	1.45 (1.19-1.76) p<0.0005	1.99 (1.65 – 2.40) p<0 0005	1.47 (1.19-1.83) p<0.0005
Ozone (O_3) (0.75 – 3.74) pphm (1 day) Tertile 1 (0.75 - ≤1.67)	2 (middle) (>1.67 - ≤2.10)	0.85 (0.72-1.01) p=0.061	1.06 (0.88-1.27) p=0.550	0.81 (0.67-0.97) p=0.024	1.46 (1.14-1.88) p=0.003
	3 (high) (>2.10 - ≤3.74)	0.94 (0.79-1.11) p=0.452	1.00 (0.83-1.20) p=0.977	0.84 (0.70-1.02) p=0.073	3.31 (2.63-4.18) p<0.0005
Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day) Tertile 1(2.92 - ≤13.55)	2 (middle) (>13.55 - ≤18.50)	0.98 (0.83-1.12) p=0.801	1.11 (0.94-1.32) p=0.202	0.94 (0.78-1.12) p=0.487	1.05 (0.87-1.26) p=0.637
	3 (high) (>18.50 -≤68.30)	0.66 (0.56-0.78) p<0.0005	1.01 (0.84-1.22) p=0.898	0.61 (0.51-0.75) p<0.0005	0.89 (0.64- 1.10.) p=0.237
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day) Tertile 1 (0.00 – ≤6.45)	2 (middle) (>6.45 - ≤8.51)	1.04 (0.88-1.23) p=0.647	1.11 (0.94-1.32) p=0.223	1.01 (0.84-1.22) p=0.910	1.07 (0.88-1.29) p=0.500
	3 (high) (>8.51 - ≤40.78)	1.02 (0.87-1.21) p=0.785	1.11 (0.93-1.32) p=0.237	0.93 (0.77-1.13) p=0.475	0.98 (0.81-1.19) p=0.852

Table 4.7 (b): Odds ratios and P-values for 10% and 20% increase in 'Total Respiratory' hospital admissions for medium (tertile 2) and high (tertile 3) tertiles of pollutant level compared to the low tertile (tertile 1), before and after adjusting for seasons. Significant values (P<0.05 and OR>1.00) are highlighted in red.

Pollutant (Reported Range) Tertile 1 Range	Tertile Range	Total Respiratory Admissions (standardised) exceeding Gastrointestinal admissions by ≥ 10%		Total Respiratory Admissions (standardised) exceeding Gastrointestinal admissions by ≥ 20%	
		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) Adjusted for season	Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) Adjusted for season
Carbon Monoxide (CO) (0.00-3.04) ppm Tertile 1 (0.00 -≤0.11)	2 (middle) (>0.11 - ≤0.21)	1.44 (1.21-1.71) p<0.0005	1.39 (1.13-1.71) p=0.002	1.45 (1.20-1.74) p<0.0005	1.38 (1.11-1.71) p=0.003
	3 (high) (>0.21 - ≤3.04)	3.71 (3.13-4.40) p<0.0005	2.31 (1.85-2.88) p<0.0005	3.72 (3.12-4.44) p<0.0005	2.24 (1.79-2.81) P<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day) Tertile 1 (0.00 - ≤0.042)	2 (middle) (>0.042- ≤0.73)	1.25 (1.05-1.47) p=0.011	1.09 (0.89-1.33) p=0.431	1.20 (1.00 – 1.44) p=0.047	1.04 (0.84-1.28) p=0.749
	3 (high) (>0.73 – ≤1.92)	2.40 (1.40-3.34) p<0.0005	1.50 (1.21-1.88) p<0.0005	2.75 (2.31 – 3.27) p<0.0005	1.42 (1.13-1.77) p=0.002
Ozone (O_3) (0.75 – 3.74) pphm (1 day) Tertile 1 (0.75 - ≤1.67)	2 (middle) (>1.67 - ≤2.10)	0.63 (0.54-0.75) p<0.0005	0.89 (0.71-1.10) p=0.272	0.60 (0.51-0.72) p<0.0005	0.86 (0.68-1.08) p=0.191
	3 (high) (>2.10 - ≤3.74)	1.16 (0.99-1.40) p=0.064	1.03 (0.83-1.29) p=0.103	1.10 (0.931-1.294) p=0.268	1.00 (0.79-1.25) p=0.964
Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day) Tertile 1(2.92 - ≤13.55)	2 (middle) (>13.55 - ≤18.50)	0.66 (0.56-0.77) p<0.0005	0.92 (0.76-1.10) p=0.363	0.72(0.61-0.84) p<0.0005	1.01 (0.83-1.22) p=0.938
	3 (high) (>18.50 -≤68.30)	0.24 (0.20-0.28) p<0.0005	0.61 (0.49-0.76) p<0.0005	0.24 (0.-0.29) p<0.0005	0.63 (0.50-0.79) p=<0.0005
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day) Tertile 1 (0.00 – ≤6.45)	2 (middle) (>6.45 - ≤8.51)	0.94 (0.80-1.10) p=0.0431	1.17 (0.96-1.43) p=0.113	0.92 (0.78-1.09) p=0.320	1.12 (0.92-1.37) p=0.267
	3 (high) (>8.51 - ≤40.78)	0.79 (0.67-0.93) p=0.004	0.99 (0.81-1.21) p=0.901	0.80 0.67-0.94 p=0.008	0.97 (0.79-1.19) p=0.763

Table 4.7 (c): Odds ratios and P-values for 10% and 20% increase in 'Asthma' hospital admissions for medium (tertile 2) and high (tertile 3) tertiles of pollutant level compared to the low tertile (tertile 1), before and after adjusting for seasons. Significant values (P<0.05 and OR>1.00) are highlighted in red.

Pollutant Reported Range Tertile 1 Range		**Asthma Admissions (standardised) exceeding Gastrointestinal admissions by ≥ 10%**		**Asthma Admissions (standardised) exceeding Gastro-intestinal admissions by ≥ 20%**	
	Tertile Range	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**
Carbon Monoxide (CO) (0.00-3.04) ppm Tertile 1 (0.00 -≤0.11)	2 (medium) (>0.11 - ≤0.21)	1.70 (1.44-2.00) p<0.0005	1.55 (1.30-1.45) p<0.0005	1.61 (1.35-1.91) p<0.0005	1.47 (1.22-1.76) p<0.005
	3 (high) (>0.21 - ≤3.04)	3.20 (2.71-3.78) p<0.0005	2.03 (1.68-2.45) p<0.0005	3.00 (2.54-3.56) p<0.0005	1.89 (1.56-2.29) p<0.005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day) Tertile 1 (0.00 - ≤0.042)	2 (medium) (>0.042- ≤0.73)	1.32 (1.11-1.55) p=0.001	1.11 (0.93-1.30) p=0.237	1.25 (1.06 – 1.48) p=0.010	1.05 (0.88-1.26) p=0.065
	3 (high) (>0.73 – ≤1.92)	2.41 (2.04-2.83) P<0.0005	1.39 (1.15-1.68) p=0.001	2.18 (1.85 – 2.57) p<0.0005	1.23 (1.02-1.49) p=0.34
Ozone (O_3) (0.75 – 3.74) pphm (1 day) Tertile 1 (0.75 - ≤1.67)	2 (medium) (>1.67 - ≤2.10)	0.77 (0.62-0.85) p<0.0005	0.95 (0.79-1.13) p= 0.543	0.71 (0.61-0.84) p<0.0005	0.94 (0.78-1.13) p=0.494
	3 (high) (>2.10 - ≤3.74)	0.87 (0.74-1.02) p=0.090	0.94 (0.78-1.12) p=0.474	0.92 (0.78-1.08) p=0.290	0.99 (0.82-1.19) p=0.895
Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day) Tertile 1 (2.92 - ≤13.55)	2 (medium) (>13.55 - ≤18.50)	0.80 (0.68-0.94) p=0.006	0.94 (0.80-1.11) p=0.467	0.80 (0.68-0.94) p=0.0006	0.94 (0.80-1.11) p=0.477
	3 (high) (>18.50 -≤68.30)	0.43 (0.37-0.51) p<0.0005	0.76 (0.64-0.91) p=0.0004	0.44 (0.37-0.57) p<0.0005	0.78 (0.65-0.95) p=0.011
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day) Tertile 1 (0.00 – ≤6.45)	2 (medium) (>6.45 - ≤8.51)	1.09 (0.93-1.28) p=0.291	1.22 (1.03-1.45) p=0.019	1.09 (0.93-1.28) p=0.303	1.22 (1.03-1.45) p=0.022
	3 (high) (>8.51 - ≤40.78)	0.89 (0.76-1.05) p=0.168	1.02 (0.86-1.21) p=0.812	0.39 (0.75-1.05) p=0.157	1.01 (0.89-1.21) p=0.893

Table 4.7 (d): Odds ratios and P-values for 10% and 20% increase in 'COPD' hospital admissions for medium (tertile 2) and high (tertile 3) tertiles of pollutant level compared to the low tertile (tertile 1), before and after adjusting for seasons. Significant values (P<0.05 and OR>1.00) are highlighted in red.

Pollutant Reported Range Tertile 1 Range		**COPD Admissions (standardised) exceeding Gastrointestinal admissions by ≥ 10%**		**COPD Admissions (standardised) exceeding Gastro-intestinal admissions by ≥ 20%**	
	Tertile Range	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**
Carbon Monoxide (CO) (0.00-3.04) ppm Tertile1 (0.00 -≤0.11)	2 (medium) (>0.11 - ≤0.21)	1.40 (1.18-1.66) p<0.0005	1.37 (1.14-1.65) p=0.001	1.47 (1.22-1.77) p<0.0005	1.45 (1.19-1.76) p<0.0005
	3 (high) (>0.21 - ≤3.04)	2.50 (2.12-2.96) p<0.0005	1.88 (1.54-2.30) p<0.0005	2.57 (2.15-3.06) p<0.0005	1.93 (1.57-2.37) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day) Tertile1 (0.00 - ≤0.042)	2 (medium) (>0.042- ≤0.73)	1.23 (1.04-1.45) p=0.018	1.17 (0.97-1.40) p=0.104	1.21 (1.01 – 1.44) p=0.040	1.15 (0.94-1.39) p=0.168
	3 (high) (>0.73 – ≤1.92)	1.95 (1.65-2.30) p<0.0005	1.35 (1.11-1.66) p=0.003	1.92 (1.61 – 2.28) p<0.0005	1.33 (1.08-1.63) p=0.008
Ozone (O_3) (0.75 – 3.74) pphm (1 day) Tertile1 (0.75 - ≤1.67)	2 (medium) (>1.67 - ≤2.10)	0.74 (0.63-0.87) p<0.0005	0.88 (0.72-1.06) p=0.174	0.75 (0.630-0.896 p=0.001	0.91 (0.75-1.12) p=0.375
	3 (high) (>2.10 - ≤3.74)	1.19 (1.01-1.40) p=0.037	0.99 (0.82-1.21) p=0.947	1.19 (1.01-1.41) p=0.038	1.02 (0.84-1.26) p=0.770
Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day) Tertile1 (2.92 - ≤13.55)	2 (medium) (>13.55 - ≤18.50)	0.81 (0.69-0.95) p=0.010	1.04 (0.87-1.24) p=0.060	0.87 (0.74-1.03) p=0.100	1.12 (0.94-1.10) p=0.210
	3 (high) (>18.50 -≤68.30)	0.41 (0.35-0.49) p<0.0005	0.82 (0.68-1.00) p=0.046	0.45 (0.37-0.54) p<0.0005	0.90 (0.73-1.10) p=0.315
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day) Tertile1 (0.00 – ≤6.45)	2 (medium) (>6.45 - ≤8.51)	0.86 (0.73-1.01) p=0.065	0.96 (0.80-1.15) p=0.644	0 .90 (0.75-1.06) p=0.021	1.00 (0.84-1.21) p=0.972
	3 (high) (>8.51 - ≤40.78)	0.83 (0.070-0.097) p=0.023	0.98 (0.82-1.17) p=0.792	0.86 (0.73-1.02) p=0.091	1.01 (0.84-1.22) p=0.883

Table 4.7 (e): Odds ratios and P-values for 10% and 20% increase in 'Pneumonia/ Influenza/ Acute Bronchitis' hospital admissions for medium (tertile 2) and high (tertile 3) tertiles of pollutant level compared to the low tertile (tertile 1), before and after adjusting for seasons. Significant values (P<0.05 and OR>1.00) are highlighted in red.

Pollutant Reported Range Tertile 1 Range		**Pneumonia/ Influenza/ Acute Bronchitis Admissions (standardised) exceeding Gastro by ≥ 10%**		**Pneumonia/ Influenza/ Acute Bronchitis Admissions (standardised) exceeding Gastro by ≥ 20%**	
	Tertile Range	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**
Carbon Monoxide (CO) (0.00-3.04) ppm Tertile 1 (0.00 -≤0.11)	2 (medium) (>0.11 - ≤0.21)	1.00 (0.85-1.18) p=0.994	0.91 (0.75-1.10) p=0.337	1.08 (0.91-1.28) p=0.397	1.00 (0.82-1.22) p=0.990
	3 (high) (>0.21 - ≤3.04)	1.92 (1.63-2.25) p<0.0005	1.33 (1.08-1.64) p=0.008	2.05 (1.74-2.42) p<0.0005	1.43 (1.16-1.77) p=0.001
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day) Tertile 1 (0.00 - ≤0.042)	2 (medium) (>0.042- ≤0.73)	1.01 (0.86-1.19) p=0.901	0.91 (0.75-1.11) p=0.358	1.02 (0.86 – 1.21) p=0.829	0 .92 (0.76-1.12) p=0.425
	3 (high) (>0.73 – ≤1.92)	1.66 (1.41-1.95) p<0.0005	1.08 (0.87-1.33) p=0.498	1.77 (1.50 – 2.08) p<0.0005	1.16 (0.94-1.44) p=0.176
Ozone (O_3) (0.75 – 3.74) pphm (1 day) Tertile 1 (0.75 - ≤1.67)	2 (medium) (>1.67 - ≤2.10)	0.80 (0.68-0.94) p=0.007	0.92 (0.75-1.13) p=0.420	0.76 (0.64-0.90) p=0.001	0.89 (0.72-1.10) p=0.286
	3 (high) (>2.10 - ≤3.74)	1.51 (1.29-1.77) p<0.0005	1.10 (0.90-1.34) p=0.324	1.57 (1.34-1.85) p<0.0005	1.21 (0.98-1.49) p=0.075
Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day) Tertile 1 (2.92 - ≤13.55)	2 (medium) (>13.55 - ≤18.50)	0.63 (0.54-0.74) p<0.0005	0.83 (0.69-1.00) p=0.054	0.65 (0.55=0.76) p<0.0005	0.86 (0.71-1.03) p=0.095
	3 (high) (>18.50 -≤68.30)	0.31 (0.26-0.36) p<0.0005	0.67 (0.69-0.82) p<0.0005	0.30 (0.25-0.35) p<0.0005	0.87 (0.54-0.82) p<0.0005
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day) Tertile 1 (0.00 – ≤6.45)	2 (medium) (>6.45 - ≤8.51)	0.94 (0.80-1.10) p=0.437	1.13 (0.94-1.37) p=0.191	0 .936 (0.80-1.10) p=0.425	1.13 (0.93-1.36) p=0.215
	3 (high) (>8.51 - ≤40.78)	0.76 (0.65-0.89) p=0.001	0.95 (0.78-1.15) p=0.608	0.754 (0.64-0.89) p=0.001	0.94 (0.77-1.14) p=0.523

Table 4.7 (f): Odds ratios and P-values for 10% and 20% increase in 'Other respiratory' hospital admissions for medium (tertile 2) and high (tertile 3) tertiles of pollutant level compared to the low tertile (tertile 1), before and after adjusting for seasons. Significant values (P<0.05 and OR>1.00) are highlighted in red.

Pollutant Reported Range Tertile 1 range		**Other Respiratory Admissions (standardised) exceeding Gastro by ≥ 10%**		**Other Respiratory Admissions (standardised) exceeding Gastro by ≥ 20%**	
	Tertile Range	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**	**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**
Carbon Monoxide (CO) (0.00-3.04) ppm Tertile 1 (0.00 -≤0.11)	2 (middle) (>0.11 - ≤0.21)	1.21 (1.02-1.43) p=0.031	1.08 (0.89-1.31) p=0.439	1.24 (1.02-1.49) p=0.027	1.10 (0.89-1.35) p=0.042
	3 (high) (>0.21 - ≤3.04)	3.06 (2.59-3.62) p<0.0005	1.59 (1.29-1.96) p<0.0005	3.29 (2.76-3.92) p<0.0005	1.61 (1.29-1.70) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day) Tertile 1 (0.00 - ≤0.042)	2 (middle) (>0.042- ≤0.73)	1.20 (1.01-1.42) p=0.038	1.03 (0.84-1.25) p=0.808	1.2 (1.00-1.44) p=0.050	1.01 (0.82-1.20) p=0.923
	3 (high) (>0.73 – ≤1.92)	2.60 (2.21-3.07) p<0.0005	1.26 (1.02-1.55) p=0.036	2.67 (2.25-3.18) p<0.0005	1.19 (0.95-1.48) p=0.131
Ozone (O_3) (0.75 – 3.74) pphm (1 day) Tertile 1 (0.75 - ≤1.67)	2(middle) (>1.67 - ≤2.10)	0.62 (0.53-0.74) p<0.0005	0.96 (0.78-1.20) p=0.692	0.59 (0.50-0.71) p<0.0005	0.97 (0.78-1.20) p=0.800
	3 (high) (>2.10 - ≤3.74)	1.03 (0.87-1.20) p=0.761	1.10 (0.89-1.40) p=0.363	0.92 (0.72-1.09) p=0.315	1.02 (0.82-1.27) p=0.850
Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day) Tertile 1 (2.92 - ≤13.55)	2 (middle) (>13.55 - ≤18.50)	0.71 (0.60-0.83) p<0.0005	0.93 (0.77-1.12) p=0.443	0.72 (0.61-0.84) p<0.0005	0.95 (0.78-1.14) p=0.565
	3 (high) (>18.50 -≤68.30)	0.27 (0.23-0.32) p<0.0005	0.61 (0.77-1.12) p<0.0005	0.25 (0.20-0.3) p<0.0005	0.58 (0.46-0.72) p<0.0005
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day) Tertile 1 (0.00 – ≤6.45)	2 (middle) (>6.45 - ≤8.51)	0.95 (0.81-1.12) p=0.559	1.13 (0.94-1.34) p=0.203	0.87 (0.74-1.03) p=0.116	1.00 (0.82-1.20) p=0.990
	3 (high) (>8.51 - ≤40.78)	0.79 (0.67-0.93) p=0.004	0.90 (0.74-1.08) p=0.263	0.82 (0.69-0.97) p=0.020	0.92 (0.75-1.13) p=0.410

4.3.1 Summary of associations for combinations of covariates

Associations were tested for statistical significance ($p < 0.05$) for both second and third tertiles (medium and high levels of pollutants) against the first tertile (low level of pollutants), with both a 10% and 20% increase in standardised admissions (see Section 3.1.3.1). All four of these combinations were also tested before and after adjusting for the covariate season. Tables 4.8 (a) – (d) summarise the statistically significant associations for each combination separately. Also presented are the odds-ratios to indicate the magnitude of the association. The following legend was used for each table:

'a' denotes effect is statistically significant ($p<0.05$) at 10% before adjusting by season
'b' denotes effect is statistically significant ($p<0.05$) at 10% after adjusting by season
'c' denotes effect is statistically significant ($p<0.05$) at 20% before adjusting by season
'd' denotes effect is statistically significant ($p<0.05$) at 20% after adjusting by season

Subscript '$_2$' indicates the comparison between second (medium) tertile to first tertile
Subscript '$_3$' indicates comparison between third (high) tertile to first tertile

Table 4.8 (a): Summary of statistically significant ($p < 0.05$) relationships where pollutant levels are associated with a 10% increase in hospital admissions, before adjusting for seasons. Odds-ratios are indicated in parenthesis.

	Pollutant				
Selected Illness	**Carbon monoxide** (OR)	**Nitrogen dioxide** (OR)	**Ozone** (OR)	**PM_{10}** (OR)	**$PM_{2.5}$** (OR)
Cardiovascular disease	a_2 (1.49) a_3 (2.24)	a_2 (1.20) a_3 (2.00)	-	-	-
Total respiratory disease	a_2 (1.44) a_3 (3.71)	a_2 (1.25) a_3 (2.40)	-	-	-
Asthma	a_2 (1.70) a_3 (3.20)	a_2 (1.32) a_3 (2.41)	-	-	-
Chronic obstructive pulmonary disease	a_2 (1.40) a_3 (2.50)	a_2 (1.23) a_3 (1.95)	a_3 (1.19)	-	-
Pneumonia / influenza/ acute bronchitis	a_3 (1.92)	a_3 (1.66)	a_3 (1.51)	-	-
Other respiratory diseases	a_2 (1.21) a_3 (3.06)	a_2 (1.20) a_3 (2.60)	-	-	-

Table 4.8 (b): Summary of statistically significant relationships where pollutant levels are associated with a 10% increase in hospital admissions, after adjusting for seasons. Odds-ratios are indicated in parenthesis.

	Pollutant				
Selected Illness	**Carbon monoxide** (OR)	**Nitrogen dioxide** (OR)	**Ozone** (OR)	**PM_{10}** (OR)	**$PM_{2.5}$** (OR)
Cardiovascular disease	b_2 (1.41) b_3 (1.63)	b_3 (1.45)	-	-	-
Total respiratory disease	b_2 (1.39) b_3 (2.31)	b_3 (1.50)	-	-	-
Asthma	b_2 (1.55) b_3 (2.03)	b_3 (1.39)	-	-	b_2 (1.22)
Chronic obstructive pulmonary disease	b_2 (1.37) b_3 (1.88)	b_3 (1.35)	-	-	-
Pneumonia / influenza/ acute bronchitis	b_3 (1.33)	-	-	-	-
Other respiratory diseases	b_3 (1.59)	b_3 (1.26)		-	-

Table 4.8 (c): Summary of statistically significant relationships where pollutant levels are associated with a 20% increase in hospital admissions, before adjusting for seasons. Odds-ratios are indicated in parenthesis.

	Pollutant				
Selected Illness	**Carbon monoxide** (OR)	**Nitrogen dioxide** (OR)	**Ozone** (OR)	**PM_{10}** (OR)	**$PM_{2.5}$** (OR)
Cardiovascular disease	c_2 (1.59) c_3 (2.37)	c_3 (1.99)	-	-	-
Total respiratory disease	c_2 (1.45) c_3 (3.72)	c_2 (1.20) c_3 (2.75)	-	-	-
Asthma	c_2 (1.61) c_3 (3.00)	c_2 (1.25) c_3 (2.18)	-	-	-
Chronic obstructive pulmonary disease	c_2 (1.47) c_3 (2.57)	c_2 (1.21) c_3 (1.92)	c_3 (1.19)	-	-
Pneumonia / influenza/ acute bronchitis	c_3 (2.05)	c_3 (1.77)	c_3 (1.57)	-	-
Other respiratory diseases	c_2 (1.24) c_3 (3.29)	c_2 (1.20) c_3 (2.67)	-	-	-

Table 4.8 (d): Summary of statistically significant relationships where pollutant levels are associated with a 20% increase in hospital admissions, after adjusting for seasons. Odds-ratios are indicated in parenthesis.

	Pollutant				
Selected Illness	**Carbon monoxide** (OR)	**Nitrogen dioxide** (OR)	**Ozone** (OR)	**PM_{10}** (OR)	**$PM_{2.5}$** (OR)
Cardiovascular disease	d_2 (1.52) d_3 (1.77)	d_3 (1.47)	d_2 (1.46) d_3 (3.31)	-	-
Total respiratory disease	d_2 (1.38) d_3 (2.24)	d_3 (1.42)	-	-	-
Asthma	d_2 (1.47) d_3 (1.89)	-	-	-	d_3 (1.22)
Chronic obstructive pulmonary disease	d_2 (1.45) d_3 (1.93)	d_3 (1.33)	-	-	-
Pneumonia / influenza/ acute bronchitis	d_3 (1.43)	-	-	-	-
Other respiratory diseases	d_3 (1.61)	-	-	-	-

Table 4.8 (e) summarises where any associations existed between the various pollutants and hospital admissions for each of the selected illnesses for all four combinations of covariates, a, b, c and d.

Table 4.8 (e): Summary of statistically significant associations ($p<0.0005$) between pollutants and hospitalisations for various diseases conditions, unadjusted and adjusted for season.

	Pollutant				
Selected Illness	**Carbon monoxide** (OR)	**Nitrogen dioxide** (OR)	**Ozone** (OR)	**PM_{10}** (OR)	**$PM_{2.5}$** (OR)
Cardiovascular disease	a_2,b_2,c_2,d_2 a_3,b_3,c_3,d_3	a_2 a_3,b_3,c_3,d_3	d_2 d_3	-	-
Total respiratory disease	a_2,b_2,c_2,d_2 a_3,b_3,c_3,d_3	a_2,c_2 a_3,b_3,c_3,d_3	-	-	-
Asthma	a_2,b_2,c_2,d_2 a_3,b_3,c_3,d_3	a_2,c_2 $a_3,b_3,c_3,$	-	-	b_2,d_2
Chronic obstructive pulmonary disease	a_2,b_2,c_2,d_2 a_3,b_3,c_3,d_3	a_2,c_2 a_3,b_3,c_3,d_3	a_3,c_3	-	-
Pneumonia / influenza/ acute bronchitis	a_3,b_3,c_3,d_3	a_3,c_3	a_3,c_3	-	-
Other respiratory diseases	a_2,c_2 a_3,b_3,c_3,d_3	a_2,c_2 a_3,b_3,c_3	-	-	-

4.3.2 Summary of significant associations

The following tables summarise the strength of the statistically significant relationships that existed between levels of pollutants and hospitalisations for selected illnesses for each possible combination of conditions. Using two of the values in Table 4.9 (a) as an example, the following interpretation can be made:

^ When carbon monoxide levels are in the second (middle) tertile compared to the first (lower) tertile, regardless of season, it is 1.49 times more likely that there will be a 10% increase in hospitalisations for cardiovascular illness (an increase of 49%).

^^ When carbon monoxide levels are in the third (high) tertile compared to the first (lower) tertile, after adjusting for season, it is 1.77 times more likely that there will be a 20% increase in hospitalisations for cardiovascular illness (an increase of 77%).

4.3.2.1 Cardiovascular disease admissions

The association between CO levels and hospitalisations for 'Cardiovascular' illness was statistically significant ($p < 0.0005$) for all of the comparisons.

Table 4.9 (a): Summary of odds-rations and p-values for statistically significant associations between cardiovascular hospitalisations and CO. Significant results extracted from Table 4.7 (a).

Comparisons of tertiles of CO	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.49 (1.25-1.78)^	p<0.0005
		after	1.41 (1.18-1.69)	P<0.0005
	20%	before	1.59 (1.29-1.95)	p<0.0005
		after	1.52 (1.24-1.87)	p<0.0005
High vs Low	10%	before	2.24 (1.88-2.65)	p<0.0005
		after	1.63 (1.34-1.98)	p<0.0005
	20%	before	2.37 (1.95-2.88)	p<0.0005
		after	1.77 (1.42-2.20)^^	p<0.0005

The association between NO_2 levels and hospitalisations for 'Cardiovascular' illness was statistically significant ($p < 0.0005$) for the following comparisons:

Table 4.9 (b): Summary of odds-ratios and p-values for statistically significant associations between cardiovascular hospitalisations and NO_2. Significant results extracted from Table 4.7 (a).

Comparisons of tertiles of NO_2	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.20 (1.01-1.43)	p=0.040
High vs Low	10%	before	2.00 (1.69-2.37)	p<0.0005
		after	1.45 (1.19-1.76)	p<0.0005
	20%	before	1.99 (1.65-2.40)	p<0.0005
		after	1.47 (1.19-1.83)	p<0.0005

The association between O_3 levels and hospitalisations for 'Cardiovascular' illness was statistically significant ($p < 0.0005$) for the following comparisons:

Table 4.9 (c): Summary of odds-ratios and p-values for statistically significant associations between cardiovascular hospitalisations and O_3. Significant results extracted from Table 4.7 (a).

Comparisons of tertiles of O_3	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	20%	after	1.46 (1.14-1.88)	p=0.003
High vs Low	20%	after	3.31 (2.63-4.18)	p<0.0005

The associations between the pollutants, PM_{10}, and $PM_{2.5}$, and hospitalisations for 'Cardiovascular' admissions were not statistically significant ($p > 0.05$), either before or after adjusting for seasons.

4.3.2.2 Total respiratory admissions

The association between CO levels and hospitalisations for 'Total respiratory' illness was statistically significant ($p < 0.0005$) for all of the comparisons:

Table 4.10 (a): Summary of odds-ratios and p-values for statistically significant associations between 'Total respiratory' hospitalisations and CO. Significant results extracted from Table 4.7 (b).

Comparisons of tertiles of CO	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.44 (1.21-1.71)	p<0.0005
		after	1.39 (1.13-1.71)	p=0.002
	20%	before	1.45 (1.20-1.74)	p<0.0005
		after	1.38 (1.11-1.71)	p=0.003
High vs Low	10%	before	3.71 (3.13-4.40)	p<0.0005
		after	2.31 (1.85-2.88)	p<0.0005
	20%	before	3.72 (3.12-4.44)	p<0.0005
		after	2.24 (1.79-2.81)	p<0.0005

The association between NO_2 levels and hospitalisations for 'Total respiratory' illness was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.10 (b): Summary of odds-ratios and p-values for statistically significant associations between 'Total respiratory' hospitalisations and NO_2. Significant results extracted from Table 4.7 (b).

Comparisons of tertiles of NO_2	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.25 (1.05-1.47)	p=0.011
	20%	before	1.20 (1.00-1.44)	p=0.047
High vs Low	10%	before	2.40 (1.40-3.34)	p<0.0005
		after	1.50 (1.21-1.88)	p<0.0005
	20%	before	2.75 (2.31-3.27)	p<0.0005
		after	1.42 (1.13-1.77)	p=0.002

The association between the pollutants, PM_{10}, $PM_{2.5}$ and ozone and hospitalisations for 'Total Respiratory' illness were not statistically significant ($p > 0.05$), either before or after adjusting for seasons.

4.3.2.3 Asthma admissions

The association between CO levels and hospitalisations for 'Asthma' was statistically significant ($p < 0.05$) for all the comparisons:

Table 4.11 (a): Summary of odds-ratios and p-values for statistically significant associations between 'asthma' hospitalisations and CO. Significant results extracted from Table 4.7 (c).

Comparisons of tertiles of CO	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.70 (1.44-2.00)	p<0.0005
		after	1.55 (1.30-1.45)	p<0.0005
	20%	before	1.61 (1.35-1.91)	p<0.0005
		after	1.47 (1.22-1.76)	p<0.0005
High vs Low	10%	before	3.20 (2.71-3.78)	p<0.0005
		after	2.03 (1.68-2.45)	p<0.0005
	20%	before	3.00 (2.54-3.56)	p<0.0005
		after	1.89 (1.56-2.29)	p<0.0005

The association between NO_2 levels and hospitalisations for 'Asthma' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.11 (b): Summary of odds-ratios and p-values for statistically significant associations between 'asthma' hospitalisations and NO_2. Significant results extracted from Table 4.7 (c).

Comparisons of tertiles of NO_2	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.32 (1.11-1.55)	p=0.001
	20%	before	1.25 (1.06-1.48)	p=0.010
High vs Low	10%	before	2.41 (2.04-2.83)	p<0.0005
		after	1.39 (1.15-1.68)	p=0.001
	20%	before	2.18 (1.85-2.57)	p<0.0005

The association between $PM_{2.5}$ levels and hospitalisations for 'Asthma' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.11 (c): Summary of odds-ratios and p-values for statistically significant associations between 'asthma' hospitalisations and $PM_{2.5}$.

Comparisons of tertiles of $PM_{2.5}$	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	after	1.22 (1.03-1.45)	p=0.019
	20%	after	1.22(1.03-1.45)	p=0.022

The associations between PM_{10} and ozone, and hospitalisations for 'Asthma' were not statistically significant ($p > 0.05$), either before or after adjusting for seasons.

4.3.2.4 Chronic obstructive pulmonary disease

The association between CO levels and hospitalisations for 'COPD' was statistically significant ($p < 0.05$) for all the comparisons:

Table 4.12 (a): Summary of odds-ratios and p-values for statistically significant associations between 'COPD' hospitalisations and CO. Significant results extracted from Table 4.7 (d).

Comparisons of tertiles of CO	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.40 (1.18-1.66)	p<0.0005
		after	1.37 (1.14-1.65)	p=0.001
	20%	before	1.47 (1.22-1.77)	p<0.0005
		after	1.45 (1.19-1.76)	p<0.0005
High vs Low	10%	before	2.50 (2.12-2.96)	p<0.0005
		after	1.88 (1.54-2.30)	p<0.0005
	20%	before	2.57 (2.15-3.06)	p<0.0005
		after	1.93 (1.57-2.37)	p<0.0005

The association between NO_2 levels and hospitalisations for 'COPD' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.12 (b): Summary of odds-ratios and p-values for statistically significant associations between 'COPD' hospitalisations and NO_2. Significant results extracted from Table 4.7 (d).

Comparisons of tertiles of NO_2	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.23 (1.04-1.45)	p=0.018
	20%	before	1.21 (1.01-1.44)	p=0.040
High vs Low	10%	before	1.95 (1.65-2.30)	p<0.0005
		after	1.35 (1.11-1.66)	p=0.003
	20%	before	1.92 (1.61-2.28)	p<0.0005
		after	1.33 (1.08-1.63)	p=0.008

The association between ozone levels and hospitalisations for 'COPD' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.12 (c): Summary of odds-ratios and hospitalisations and p-values for statistically significant associations between 'COPD' and O_3. Significant results extracted from Table 4.7 (d).

Comparisons of tertiles of O_3	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
High vs Low	10%	before	1.19 (1.01-1.40)	p=0.037
	20%	before	1.19 (1.01-1.41)	p=0.038

The association between PM_{10} and $PM_{2.5}$ levels and hospitalisations for 'COPD' were not statistically significant ($p > 0.05$), before or after adjusting for seasons.

4.3.2.5 Pneumonia / influenza / acute bronchitis admissions

The association between CO levels and hospitalisations for 'Pneumonia/ influenza /acute bronchitis' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.13 (a): Summary of odds-ratios and p-values for statistically significant associations between 'Pneumonia / influenza / acute bronchitis' hospitalisations and CO. Significant results extracted from Table 4.7 (e).

Comparisons of tertiles of CO	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
High vs Low	10%	before	1.92 (1.63-2.25)	p<0.0005
		after	1.33 (1.08-1.64)	p=0.008
	20%	before	2.05 (1.74-2.42)	p<0.0005
		after	1.43 (1.16-1.77)	p=0.001

The association between NO_2 levels and hospitalisations for 'Pneumonia/ influenza/ acute bronchitis' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.13 (b): Summary of odds-ratios and p-values for statistically significant associations between 'Pneumonia / influenza / acute bronchitis' hospitalisations and NO_2. Significant results extracted from Table 4.7 (e).

Comparisons of tertiles of NO_2	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
High vs Low	10%	before	1.66 (1.41-1.95)	p<0.0005
	20%	before	1.77 (1.50-2.08)	p<0.0005

The association between O_3 levels and hospitalisations for 'Pneumonia/ influenza/ acute bronchitis' was statistically significant ($p < 0.05$) for the following comparisons:

Table 4.13 (c): Summary of odds-ratios and p-values for statistically significant associations between 'Pneumonia/ influenza/ acute/ bronchitis' hospitalisations and O_3. Significant results extracted from Table 4.7 (e).

Comparisons of tertiles of O_3	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
High vs Low	10%	before	1.51 (1.29-1.77)	p<0.0005
	20%	before	1.57 (1.34-1.85)	p<0.0005

The associations between PM_{10} and $PM_{2.5,}$ and hospitalisations for 'Pneumonia / influenza / acute bronchitis' were not statistically significant (p >0.05), before or after adjusting for seasons.

4.3.2.6 Other respiratory illness admissions

The association between CO levels and hospitalisations for 'Other respiratory' illness was statistically significant (p <0.05) for the following comparisons:

Table 4.14 (a): Summary of odds-ratios and p-values for statistically significant associations between 'Other respiratory' hospitalisations and CO. Significant results extracted from Table 4.7 (f).

Comparisons of tertiles of CO	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.21 (1.02-1.43)	p=0.031
	20%	before	1.24 (1.02-1.49)	p=0.027
High vs Low	10%	before	3.06 (2.59-3.62)	p<0.0005
		after	1.59 (1.29-1.96)	p<0.0005
	20%	before	3.29 (2.76-3.92)	p<0.0005
		after	1.61 (1.29-1.70)	p<0.0005

The association between NO_2 levels and hospitalisations for 'Other respiratory' was statistically significant (p <0.05) for the following comparisons:

Table 4.14 (b): Summary of odds-ratios and p-values for statistically significant associations between 'Other respiratory' hospitalisations and NO_2. Significant results extracted from Table 4.7 (f).

Comparisons of tertiles of NO_2	Increase in standardised admissions	Seasonal adjustment	Odds-ratio (95% confidence interval)	p-value
Middle vs Low	10%	before	1.20 (1.01-1.42)	p=0.038
	20%	before	1.20 (1.00-1.44)	p=0.050
High vs Low	10%	before	2.60 (2.21-3.07)	p<0.0005
		after	1.26 (1.02-1.55)	p=0.036
	20%	before	2.67 (2.25-3.18)	p<0.0005

The association between the pollutants, PM_{10}, $PM_{2.5}$ or ozone, and hospitalisations for 'Other respiratory' illnesses were not statistically significant ($p > 0.05$), before and after adjusting for seasons.

4.4 Stage Two: Industrial and non-industrial site monitoring

This section presents the results of sampling at the Welshpool and Curtin/Bentley clusters of sites, representing an industrial and non-industrial area respectively.

4.4.1 Meteorological and gravimetric results for Welshpool area

Figure 4.13 shows mean wind speed and direction for each week of the sampling period (16/10/13 to 27/11/13). These values were obtained by averaging the meteorological data from the Caversham, South Lake and Swanbourne monitoring stations as these are the closest and most likely to reflect the prevailing weather conditions in Welshpool. Data for individual monitoring stations is given in Table 4.15. Winds during the sampling period were predominantly light and south / south-east, blowing pollutants from the industrial area towards the residential area. Although there were substantial changes in wind direction throughout each day these values indicate the overall prevailing direction over the week. There was light rainfall recorded while sampling at sites 1, 2 and 6, however it would not be expected that this would have a significant effect on monitored pollutant levels.

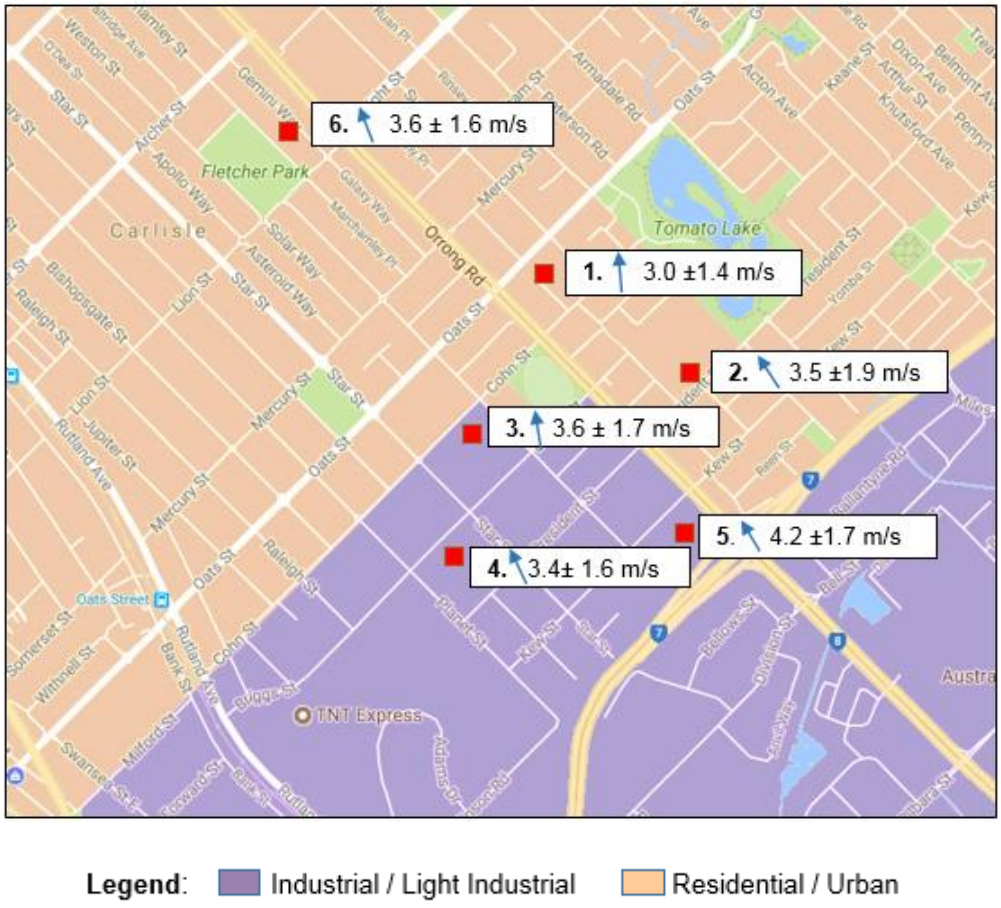


Figure 4.13: Map of Welshpool area showing sampling sites (red squares), predominant wind direction (blue arrows) and speed for each sampling period.

Table 4.15: Details of the sample locations, date and time of sampling, mean wind speed and direction and total rainfall during sampling period. Meteorological data are taken from published BOM measurements.

Site No.	Date Start / Finish	Time Start / Finish	Location of Monitoring	Mean Wind Speed (m/s) / Mean Wind Direction (Degrees)				Total Rain (mm)
				South Lake	Caversham	Swanbourne	AVERAGE (3 sites)	Perth Airport
1	16/10/13 23/10/13	12.00md 11.00am	Domestic Residence – 8 Oats St, KEWDALE	2.5 ± 1.1 180.1° ± 82.0°	2.6 ± 1.1 168.1° ± 84.3°	4.1 ± 1.5 187.9° ± 77.7°	**3.0 ± 1.4** **177.9° ± 82.1°**	17.8
2	23/10/13 30/10/13	12.00md 11.00am	High School, President Ave, KEWDALE	2.9 ± 1.6 145.0° ± 62.4°	3.2 ± 1.9 150.5° ± 69.7°	4.4 ± 1.8 144.4° ± 59.9°	**3.5 ± 1.9** **147.0° ± 64.1°**	0.2
3	30/10/13 6/11/13	12.00md 11.30am	Dept of Mines and Petroleum Core Library, Harris St, CARLISLE	3.0 ± 1.4 171.2° ± 76.1°	3.2 ± 1.6 166.8° ± 79.2°	4.5 ± 1.7 175.2° ± 66.7°	**3.6 ± 1.7** **170.6° ± 74.2°**	0.0
4	06/11/13 13/11/13	12.30pm 9.30am	Town of Victoria Park Works Depot, Briggs St, CARLISLE	2.7 ± 1.1 157.3° ± 60.8°	2.8 ± 1.2 153.7° ± 65.6°	4.7 ± 1.6 154.5° ± 50.0°	**3.4 ± 1.6** **154.9° ± 59.1°**	0.0
5	13/11/13 20/11/13	10.10am 10.30am	Museum storehouse, Cnr Orrong Rd & Leach Hwy, WELSHPOOL	3.5 ± 1.3 154.1° ± 75.0°	3.9 ± 1.6 138.4° ± 67.4°	5.2 ± 1.5 155.4° ± 78.4°	**4.2 ± 1.7** **149.1° ± 76.3°**	0.0
6	20/11/13 27/11/13	11.15am 10.00am	Domestic Residence, 42 Gemini Way, CARLISLE	3.1 ± 1.4 157.3° ± 82.1°	3.3 ± 1.7 157.8° ± 83.7°	4.3 ±1.6 170.1° ± 80.6°	**3.6 ± 1.6** **161.7° ± 82.0°**	0.2

Table 4.16 shows the results of the Department of Environmental Regulation monitoring over the corresponding time period. To obtain the mean value, data was collected from the Caversham and South Lake monitoring stations as these are the closest stations that have comprehensive PM monitoring and are most likely to reflect the pollutant levels in the Welshpool area. At all sites the DWER mean values for $PM_{2.5}$ and PM_{10} were below their respective AAQS maximum values of 25 $\mu g/m^3$ and 50 $\mu g/m^3$.

Table 4.16: Results of DWER PM_{10} and $PM_{2.5}$ monitoring at Caversham (CA) and South Lake (SL) during the corresponding time periods.

Site No.	**Date Week**	**Location of Monitoring**	**Mean PM_{10} ± SD ($\mu g/m^3$)** Caversham	**Mean PM_{10} ± SD ($\mu g/m^{3)}$** South Lake	**MEAN PM_{10} ± SD ($\mu g/m^3$) Both sites**	**Mean $PM_{2.5}$ ± SD ($\mu g/m^3$)** Caversham	**Mean $PM_{2.5}$ ± SD ($\mu g/m^3$)** South Lake	**MEAN $PM_{2.5}$ ± SD ($\mu g/m^3$) Both sites**	**RATIO $PM_{2.5}$:PM_{10}**
1	16/10/13	Domestic Residence – 8 Oats St KEWDALE	9.2 ± 5.4	10.8 ± 5.3	**10.1 ± 5.3**	5.7 ± 2.5	5.8 ± 2.4	**5.7 ± 2.5**	**0.56**
2	23/10/13	High School, President Ave, KEWDALE	16.1 ± 8.8	18.4 ± 11.0	**17.3 ± 10.0**	8.7 ± 5.3	9.5 ± 6.0	**9.1 ± 5.7**	**0.53**
3	30/10/13	Dep Mines & Petroleum Core Library, Harris St, CARLISLE	17.4 ± 8.5	18.8 ± 8.8	**18.1 ± 8.6**	7.9 ± 4.1	8.5 ± 4.6	**8.2 ± 4.4**	**0.45**
4	06/11/13	Town of Victoria Park Works Depot, Briggs St, CARLISLE	18.2 ± 13.8	21.4 ± 16.6	**19.8 ± 15.3**	9.0 ± 7.7	9.8 ± 8.9	**9.4 ± 8.4**	**0.47**
5	13/11/13	Museum storehouse, Cnr Orrong Rd & Leach Hwy, WELSHPOOL	17.3 ± 10.4	21.7 ± 10.3	**20.1 ± 10.5**	6.8 ± 2.5	8.7 ± 4.2	**8.0 ± 3.8**	**0.40**
6	20/11/13	Domestic Residence, 42 Gemini Way, CARLISLE	14.8 ± 6.7	16.1 ± 7.5	**15.4 ± 7.2**	7.8 ± 3.6	7.4 ± 3.1	**7.6 ± 3.3**	**0.49**

Table 4.17 gives the results for monitoring conducted at the Welshpool sites using both Nano-MOUDI and the Hi-Vol Sampler (HVS). Results are given for PM_{10} and $PM_{2.5}$ to enable comparison to the BOM results for the same time period (Table 4.13). Results were also calculated for $PM_{0.1}$ as although this is not monitored by the BOM, the values indicate the proportion of ultrafine size particles present in the ambient air. The HVS results indicates how much particulate matter present in the sampled air has an AED greater than 10 µg as the HVS is able to capture particles up to 50 µm AED.

Table 4.17: Nano-MOUDI and High Volume Sampler Results for the Welshpool industrial cluster.

			Nano-MOUDI II™ 125B			Hi Vol Sampler
Site No.	**Date Sampled**	**Site**	**PM_{10} (µg/m^3)**	**$PM_{2.5}$ (µg/m^3)**	**$PM_{0.1}$ (µg/m^3)**	**TSP (µg/m^3)**
1	16-23/10/13	Domestic Residence 8 Oats St KEWDALE	13.5	7.5	4.7	10.1 *
2	23-30/10/13	High School President Ave, KEWDALE	18.6	10.2	0.7	41.9
3	30-6/11/13	Dep Mines & Petroleum Harris St, CARLISLE	18.3	11.0	3.1	35.6
4	6-13/11/13	ToVP Works Depot, Briggs St, CARLISLE	33.0	7.7	2.0	33.2
5	13-20/11/13	Museum store, Orrong Rd & Leach Hwy, WELSHPOOL	15.9	8.2	1.2	42.6
6	20-27/11/13	Domestic Residence 42 Gemini Way, CARLISLE	37.2	30.3	3.6	19.2*

* HVS stopped working for an unknown period. It is expected that this result would be at least equal to or greater than 13.5 µg/m^3 at Site 1 and 37.2 µg/m^3 at Site 6.

4.4.2 Meteorological and gravimetric results for Curtin/Bentley area

Figure 4.14 shows mean wind speed and direction for each week of the sampling period (4/8/14 to 12/9/14). These values were obtained by averaging the meteorological data from the Caversham, South Lake and Swanbourne monitoring stations as these are the closest and most likely to reflect the prevailing weather conditions in the Curtin/Bentley area. Data for individual monitoring stations is given in Table 4.3.

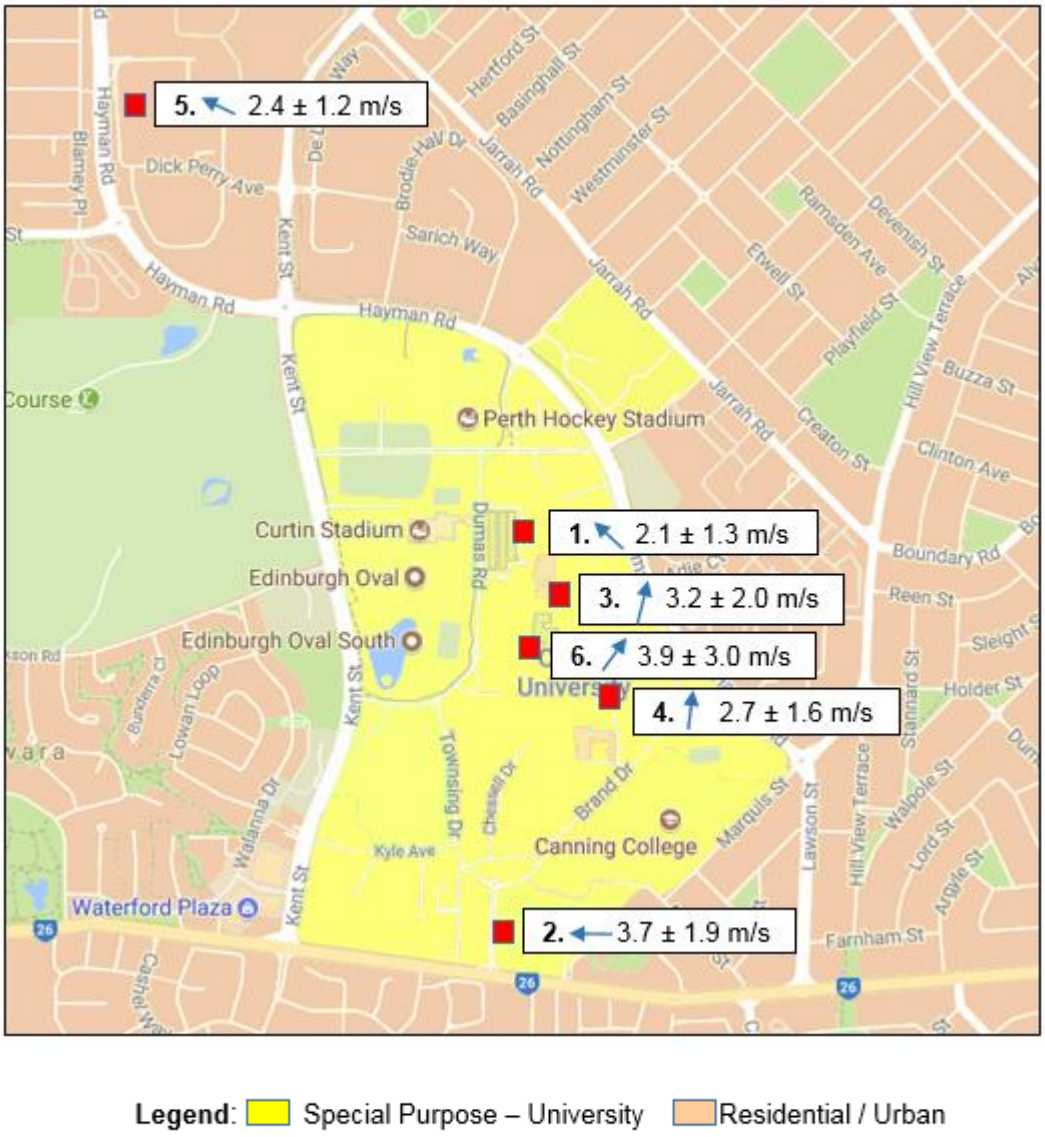


Figure 4.14: Map of Curtin/Bentley area showing sampling sites (red squares), predominant wind speed and direction (blue arrows).

A summary of meteorology data is given in Table 4.18. Significant rainfall occurred during weeks 1, 3 and 6 of the study (B400, B215, B105), which would be expected

to reduce the levels of some pollutants, particularly PM and light rain was recorded in week 4. Winds were predominantly light during the whole sampling period and tended to be southerly and from the east while monitoring sites1, 2 and 5 and more westerly while monitoring sites 3, 4 and 6. All winds were directly towards residential areas, away from the Welshpool sites.

Table 4.19 shows the results of the Department of Environmental Regulation monitoring over the corresponding time period. To obtain the mean value, data was collected from the Caversham and South Lake monitoring stations as these are the closest stations that have comprehensive PM monitoring and are most likely to reflect the pollutant levels in the Curtin/Bentley area. At all sites the DWER mean values for $PM_{2.5}$ and PM_{10} were below their respective AAQS maximum values of 25 µg/m^3 and 50 µg/m^3. The ratio of $PM_{2.5}$ to PM_{10} in this cluster was 0.44-0.58 which is similar to the ratio for the Welshpool area.

Table 4.20 gives the results for the gravimetric monitoring conducted at the Curtin/Bentley sites using both Nano-MOUDI and the high volume sampler (HVS). Results are presented for PM_{10} and $PM_{2.5}$ to enable comparison to the BOM results for the same time period (Table 4.16). Results were also calculated for $PM_{0.1}$ as although this is not monitored by the BOM, the values give a good indication of the proportion of the ultrafine fraction present in ambient air. The HVS results indicates of how much particulate matter present in the sampled air has an AED greater than 10 µg as the HVS is able to capture particles up to 50 µm AED.

Table 4.18: Details of the sample locations, date and time of sampling, mean wind speed and direction and total rainfall during sampling. Meteorological data are taken from published BOM measurements.

Date Start Finish	Time Start Finish	Location of Monitoring B=Building Number	Mean Wind Speed (± SD) (m/s) Mean Direction (± SD) (Degrees) South Lake	Caversham	Swanbourne	MEAN All sites	Total Rain (mm) Perth Airport
4-8/8/14	8.45am 3.00pm	B400, School of Public Health (south-west corner of verandah, 3rd floor)	1.4 ±1.0 134.3° ±117.7	1.7 ± 1.0 121.2° ± 95.3	3.1 ± 1.3 142.8° ± 108.9	**2.1 ± 1.3** **132.8 ±° 107.7**	5.2
11-15/8/14	8.40am 2.40pm	B003, Childcare Centre (utilities area, south side, ground level)	2.7 ± 1.2 94.2° ± 72.6	3.7 ± 1.9 82.9° ± 52.1	4.8 ± 1.8 96.1° ± 59.0	**3.7 ± 1.9** **91.1° ± 62.7**	0.0
18-22/8/14	8.50am 2.50pm	B215, Engineering Pavilion (north-east corner roof, Level 4)	2.5 ± 1.3 201.9° ± 120.2	2.3 ± 1.5 163.0° ± 129.8	4.7 ± 2.2 215.9° ± 108.8	**3.2 ± 2.0** **193.6°± 121.7**	56.2
25-29/8/14	10.30am 2.45pm	B402, School of Business (roof, Level 10)	2.3 ± 1.0 166.5° ± 140.2	1.9 ± 1.2 169.8° ± 125.0	3.9 ± 1.6 219.7° ± 114.3	**2.7 ± 1.6** **187.1° ± 127.6**	0.4
1-5/9/14	10.10am 3.20pm	B611, (Technology Park), School of Environment and Agriculture (ground level)	1.9 ± 1.1 115.6° ± 75.0	1.9 ± 1.1 112.5°± 70.4	3.1 ± 1.0 119.5 °± 68.3	**2.4 ± 1.2** **115.9° ± 71.1**	0.0
8-12/9/14	9.40am 2.50pm	B105, Library, (North-east corner of walkway, level 3)	3.1 ± 2.3 214.1° ± 92.5	3.1 ± 2.5 218.0° ± 95.6	5.4 ± 3.5 214.2° ± 80.5	**3.9 ± 3.0** **215.5°± 89.5**	17.4

Table 4.19: Results of Bureau of Meteorology PM_{10} and $PM_{2.5}$ monitoring at Caversham (CA) and South Lake (SL) during the corresponding time periods.

Site No.	**Date** Start Finish	**Location of Monitoring B=Building number**	**Mean PM_{10} ± SD ($\mu g/m^3$)** Caversham	**Mean PM_{10} ± SD ($\mu g/m^{3)}$** South Lake	**MEAN PM_{10} ± SD ($\mu g/m^3$)**	**Mean $PM_{2.5}$ ± SD ($\mu g/m^3$)** Caversham	**Mean $PM_{2.5}$ ± SD ($\mu g/m^3$)** South Lake	**MEAN $PM_{2.5}$ ± SD ($\mu g/m^3$)**	**RATIO $PM_{2.5}$:PM_{10}**
1	4/8/14 8/8/14	B400 L3, School of Public Health (south-west corner of verandah	15.0 ± 4.4	13.3 ± 2.6	**14.2 ± 3.5**	8.8 ± 2.1	7.7 ± 0.9	**8.3 ± 1.6**	**0.58**
2	11/8/14 15/8/14	B003, Childcare Centre (utilities area, south side, ground level)	13.9 ± 5.9	13.8 ± 8.7	**13.9 ± 7.0**	7.5 ± 4.2	8.2 ± 6.5	**7.9 ± 5.2**	**0.57**
3	18/8/14 22/8/14	B215, Engineering Pavilion (north-east corner roof, Level 4)	13.0 ± 5.8	14.0 ± 5.6	**13.5 ± 6.4**	5.7 ± 3.5	6.3 ± 3.8	**6.0 ± 2.2**	**0.44**
4	25/8/14 29/8/14	B402, Business (roof, Level 10)	16.2 ± 3.6	13.0 ± 2.2	**14.6 ± 3.3**	7.6 ± 1.5	7.0 ± 1.2	**7.3 ± 1.3**	**0.50**
5	1/9/14 5/9/14	B611, (Technology Park), School of Environment and Agriculture	12.5 ± 3.8	10.4 ± 3.5	**11.5 ± 3.6**	6.8 ± 2.2	6.3 ± 1.7	**6.6 ± 1.8**	**0.57**
6	8/9/14 12/9/14	B105, Library, (North-east corner of walkway, level 3)	17.2 ± 8.3	17.0 ± 9.1	**17.1 ± 8.2**	8.7 ± 2.6	8.5 ± 2.8	**8.6 ± 2.5**	**0.50**

Table 4.20: Nano-MOUDI II™ 125B and Hi-Volume Sampler Results for the Curtin/Bentley non-industrial cluster.

			Nano-MOUDI II™ 125B			Hi Vol Sampler
Site No.	**Date Sampled**	**Site**	**PM_{10} ($\mu g/m^3$)**	**$PM_{2.5}$ ($\mu g/m^3$)**	**$PM_{0.1}$ ($\mu g/m^3$)**	**TSP ($\mu g/m^3$)**
1	4-8/8/14	B400 – School of Public Health	12.4	9.9	5.0	14.8
2	11-15/8/14	B003 – Childcare Centre	8.8	6.9	4.7	20.6
3	18-22/8/14	B216 – Engineering Pavilion	5.3	2.5	0.6	24.9
4	25-29/8/14	B402 – School of Business	14.0	11.8	6.7	18.5
5	1-5/9/14	B611 – Technology Park	11.9	7.8	1.0	17.5
6	8-12 / 14	B105 – Library	13.1	8.2	0.3	17.9

4.5 Nano-MOUDI II™ 125B gravimetric results

The following charts illustrate the distribution of different size particles for each of the monitoring sites in the light industrial Welshpool area and the non-industrial Curtin/Bentley area. The charts display the mass concentration and mass of particles collected on each stage charted against the cut off for each stage of the Nano-MOUDI II™ 125B. The x–axis is not to scale to accommodate the large differential in the sizes of the particles.

4.5.1 Welshpool (industrial) area

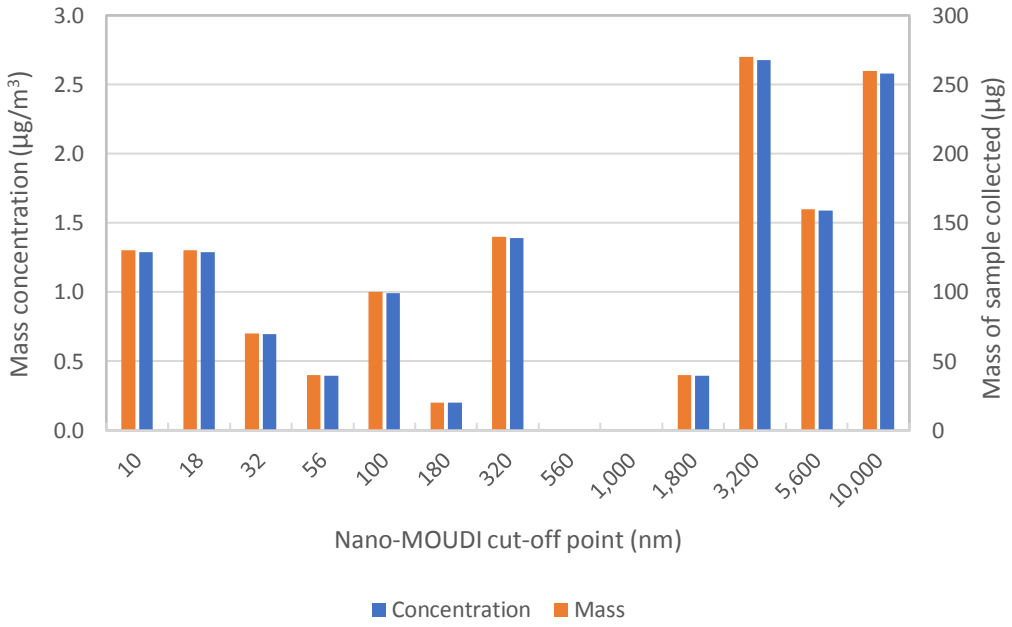


Figure 4.15 (a): Seven-day mean airborne mass concentration and mass of sample collected using the Nano-MOUDI II 125B from Site 1.

Figure 4.15 (a) shows that while there are particles distributed over the range of size fractions, the highest concentration is distributed over the larger particle sizes. This sample was collected at a domestic residence located, approximately 100 metres from a very busy intersection with a major road.

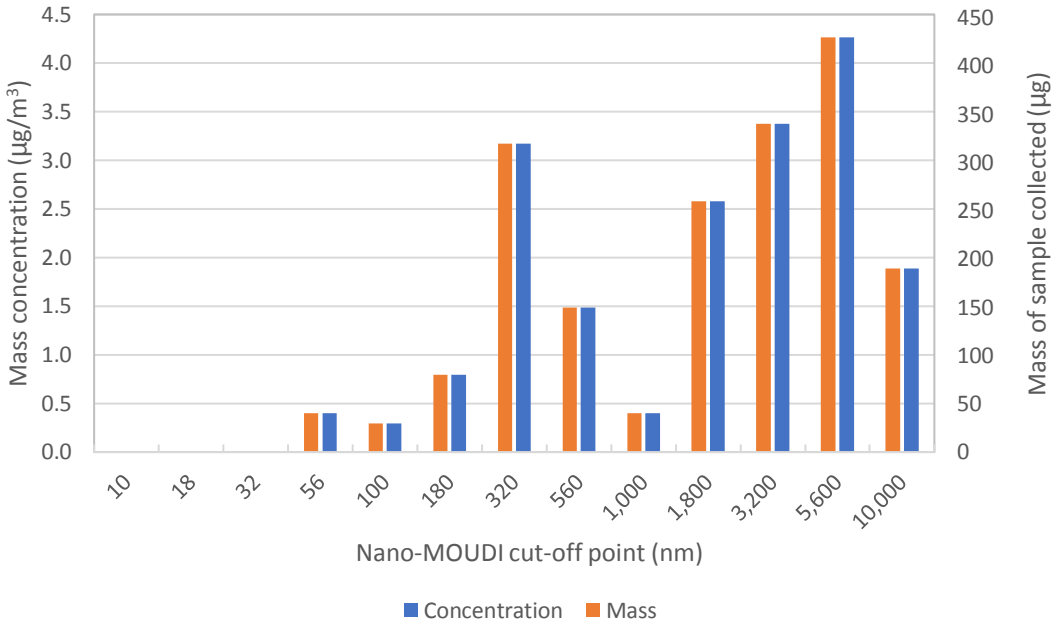


Figure 4.15 (b): Seven-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Site 2.

At this site, which was a secondary school located approximately 300 metres from a major road, there was a greater proportion of particles collected in the larger fractions.

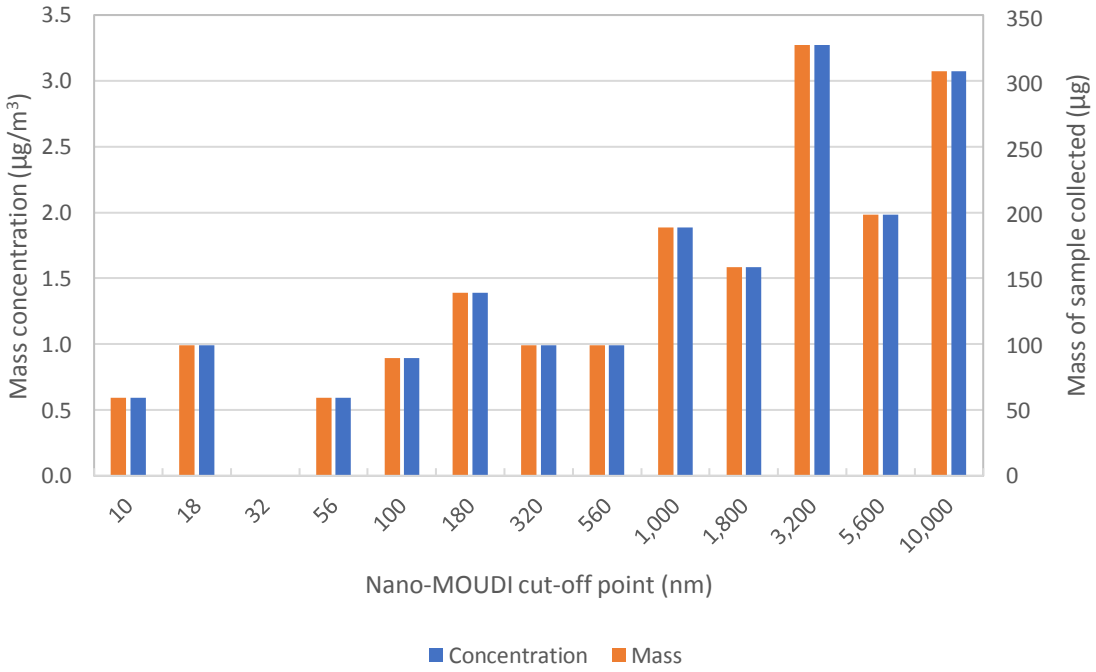


Figure 4.15 (c): Seven-day mean airborne mass concentration and mass collected using the Nano-MOUDI from Site 3.

Site 3 is the Department of Minerals and Petroleum Core Library which is used to store core samples of rock which have been collected during geological expeditions. The cores are processed (including grinding) which emits large quantity of rock dust. Figure 4.15 (c) demonstrates that there was a greater proportion of collected particles in the larger fractions.

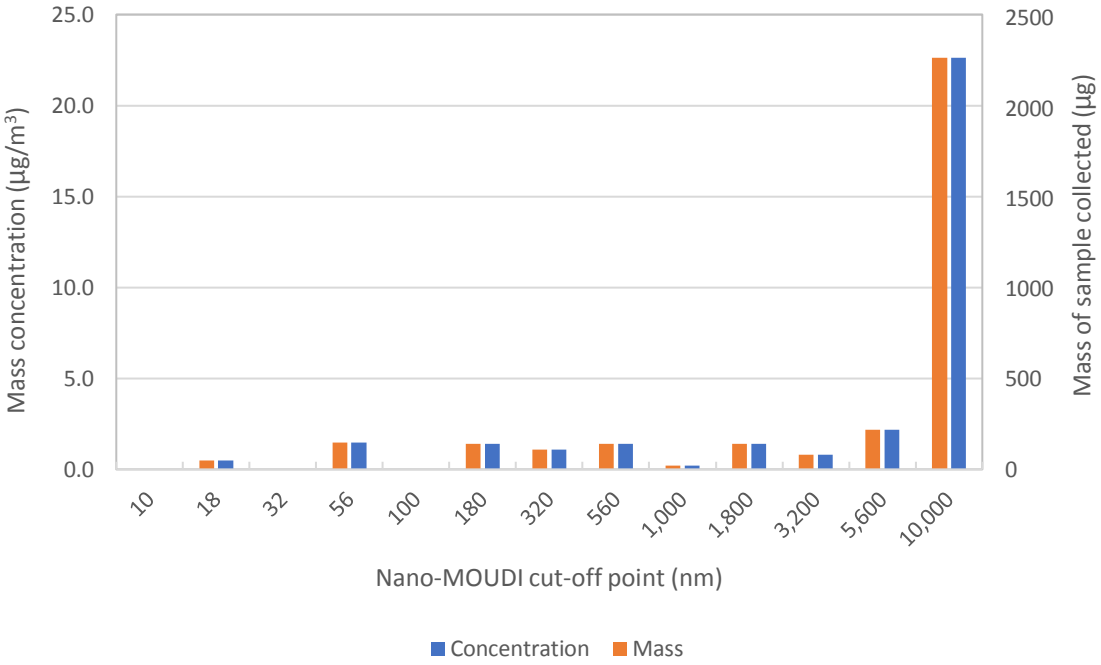


Figure 4.15 (d): Seven-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Site 4.

In Figure 4.15 (d) which illustrates the size distribution of particles collected from the Town of Victoria Park Works Depot, the result for 10,000 nm stage is extremely high, up to 10 times the maximum value of most other sites in this cluster. This anomaly may be due to contamination or possible equipment malfunction. The chart was redrawn without the 10,000 nm value to better illustrate the distribution across the smaller fractions (Figure 4.15 (e)). In this chart most particles were found in the fractions above 56 nm. This site was located approximately 500 metres from a major road and approximately 100 metres from the Town of Victoria park engineering workshop.

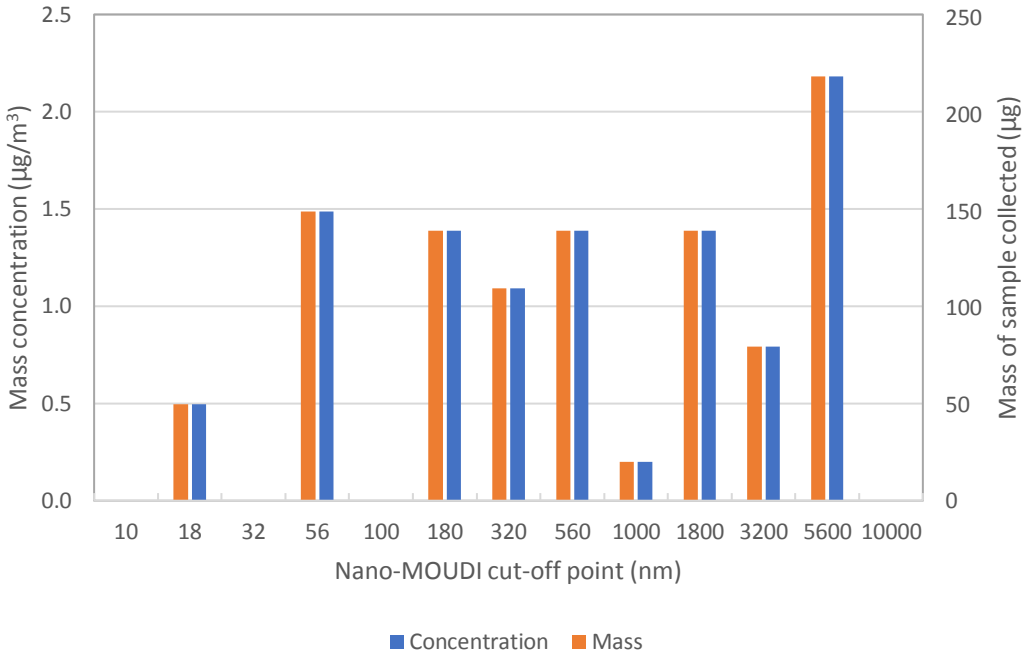


Figure 4.15 (e): Seven-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Site 4 with the mass of fraction collected from 10,000 nm stage omitted.

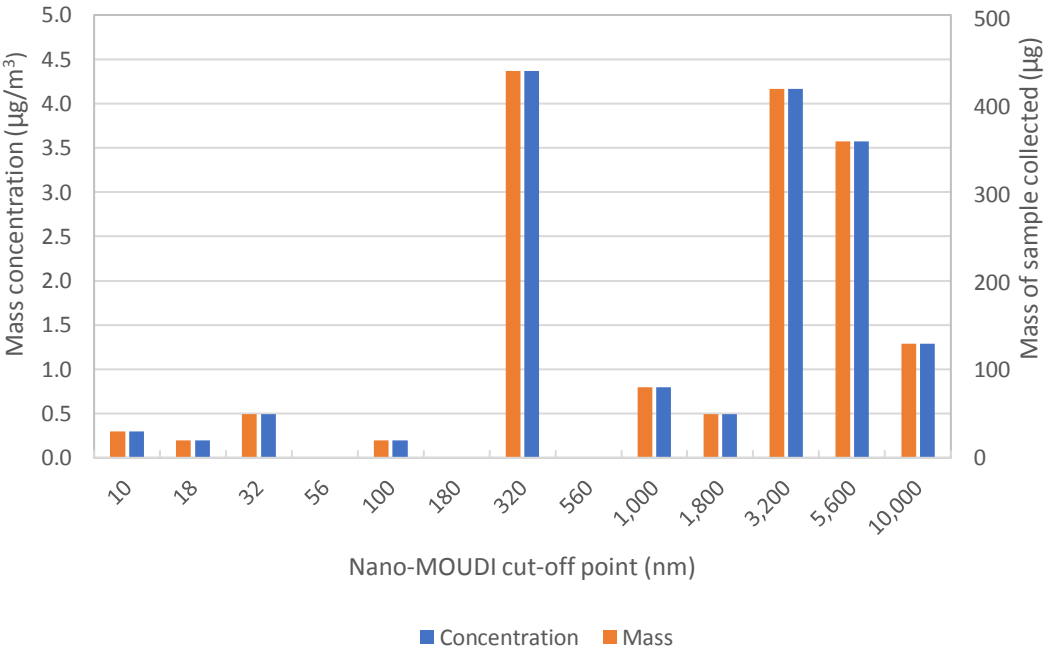


Figure 4.15 (f): Seven-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Site 5.

Site 5 was a large industrial store used by the Western Australian Museum and is located approximately 100 metres from a heavily trafficked intersection between a major road and a Highway overpass. Figure 4.15 (f) illustrates that even although

there was a peak at the 320 nm stage, most particles were found in the larger fractions, from 3200nm onwards

At the final site which was a domestic residence there was a peak at 1800 nm with particles being widely spread across the range from 180 nm to 10,000 nm. This site was located approximately 200 metres from a major road.

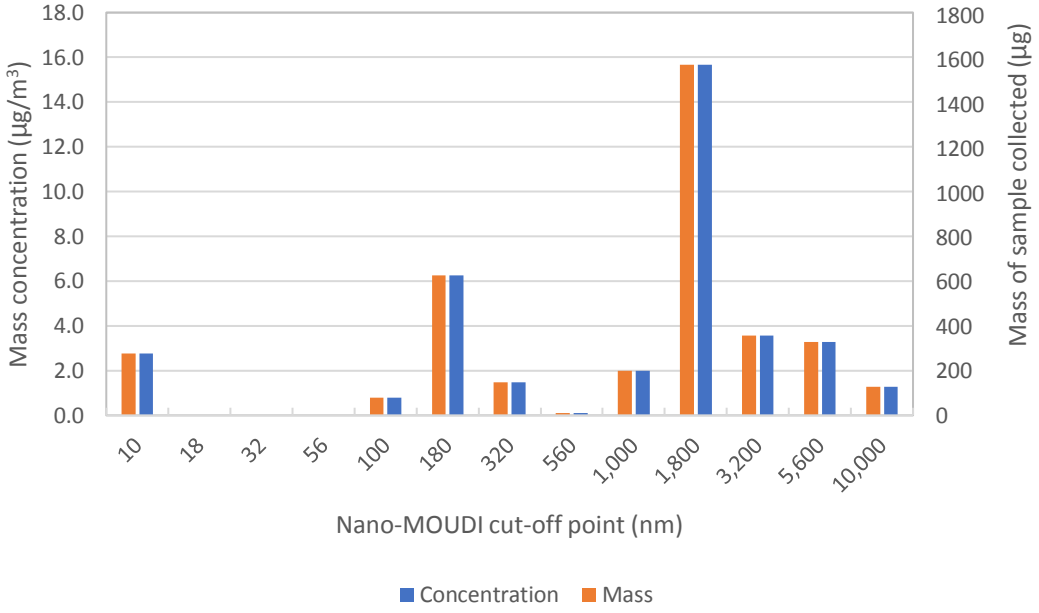


Figure 4.15 (g): Seven-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Site 6.

4.5.2 Curtin/Bentley (non-industrial) area

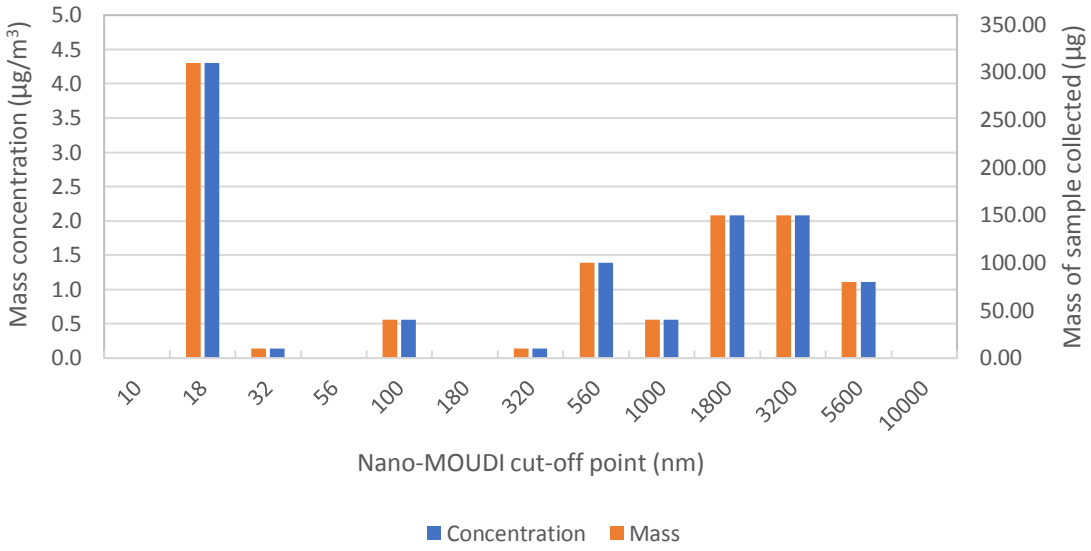


Figure 4.16 (a): Five-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Curtin Site 1.

The first Curtin/Bentley site was located on the eastern aspect of the School of Public Health. At this site there was an unexplained peak at 18 nm, although most particles were distributed towards the larger fractions (Figure 4.16 (a)). At site 2, which was the childcare centre (Figure 4.16(b)), most particles were found in the smaller fractions. This site is closer to traffic than the remaining sites in this cluster.

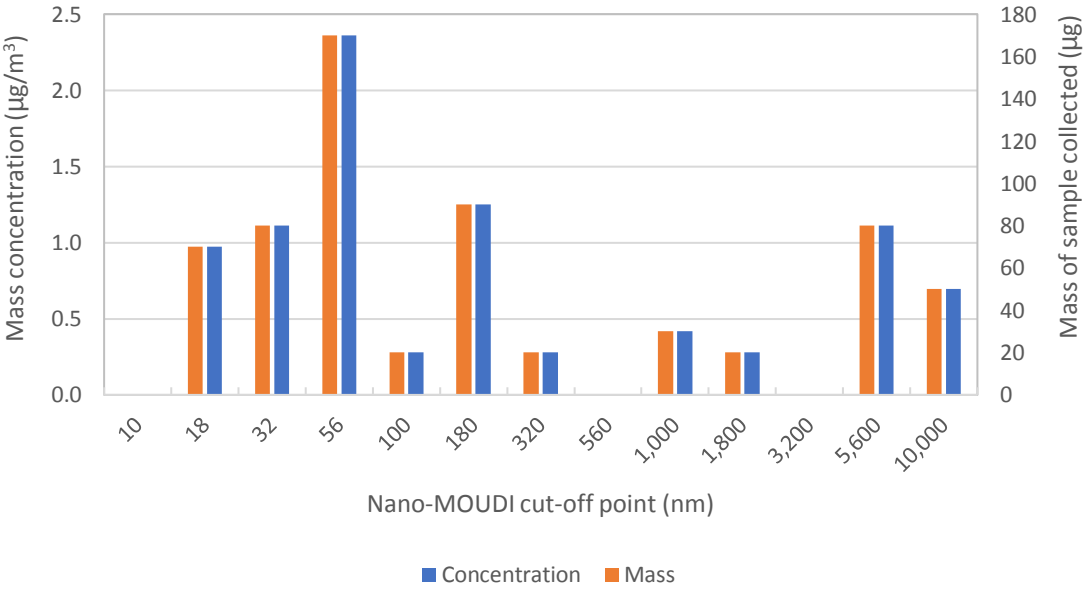


Figure 4.16 (b): Five-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Curtin Site 2.

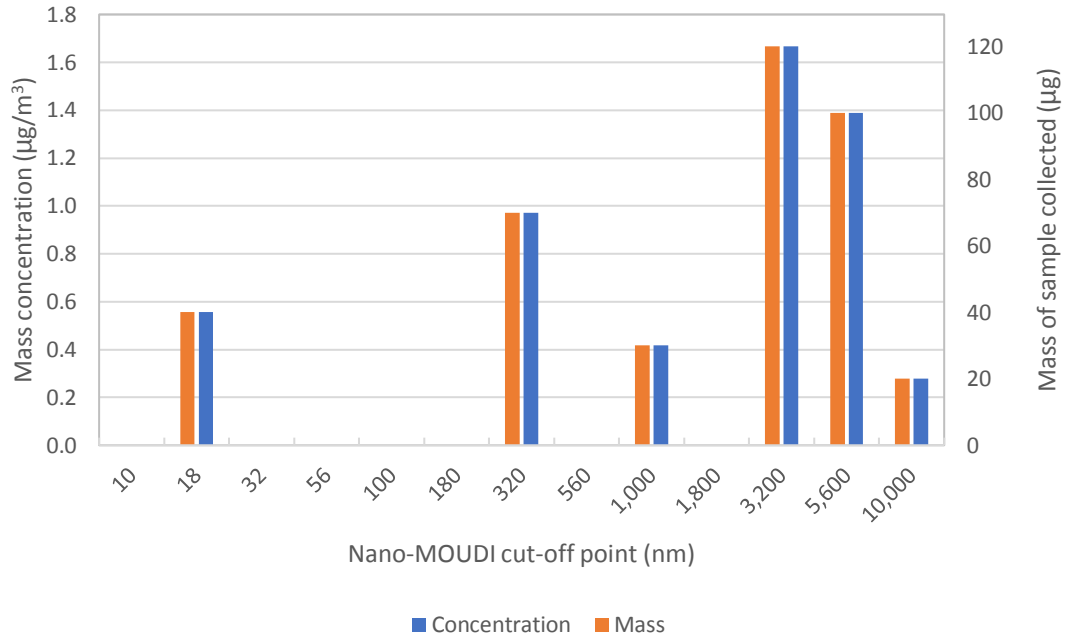


Figure 4.16 (c): Five-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Curtin Site 3.

At site 3, which was the roof of the multistorey School of Business building (Figure 4.16 (c)) most particles were found towards the larger stages, particularly from 3,200 nm onwards. At site 4, also on the roof of a multistorey building (Engineering pavilion) (Figure 4.16 (d)) there were two peaks of distribution, one around 32-56 nm and the other between the 1,000 and 10,000 nm fractions.

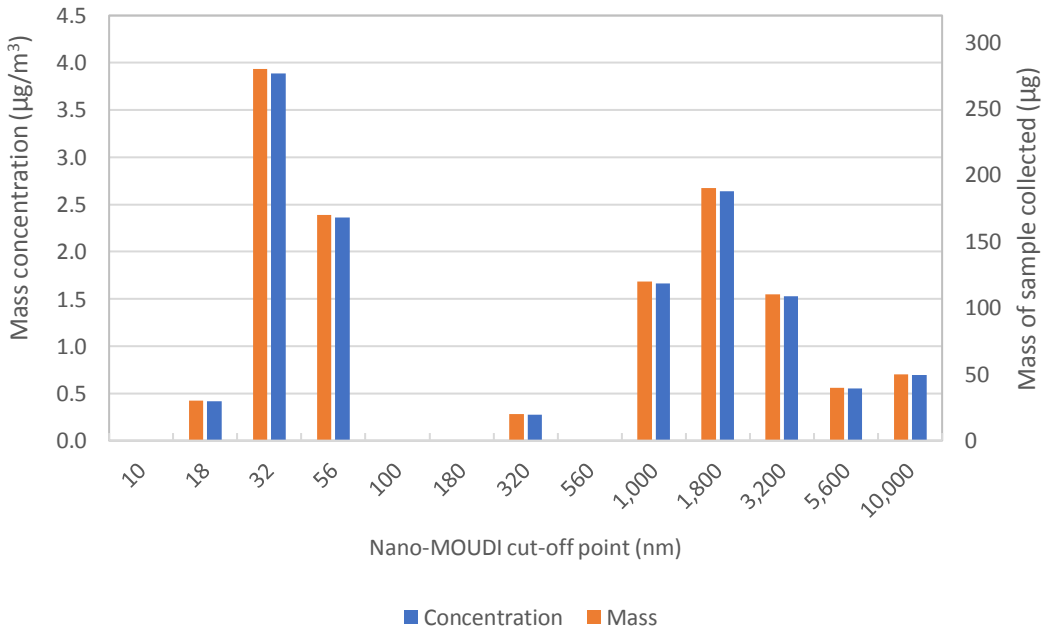


Figure 4.16 (d): Five-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Curtin Site 4.

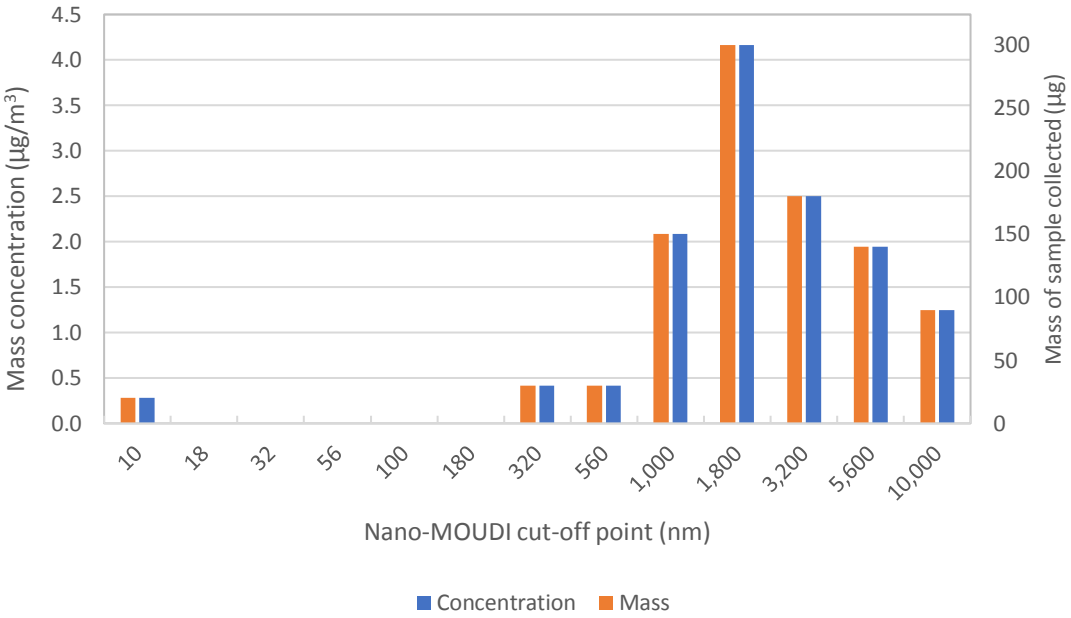


Figure 4.16 (e): Five-day mean airborne mass concentration and mass collected using the Nano-MOUDI from Curtin Site 5.

At both sites 5 (Figure 4.16 (e) and 6 (Figure 4.16 (f)) most particles were found in the larger fractions. Site 5 was located at the School of Environment and Agriculture, research facility in Technology Park. There may be emissions from the facility but there is very little traffic. Site 6 was on the veranda on the third floor of the library which has a very little vehicular traffic however a large volume of foot traffic.

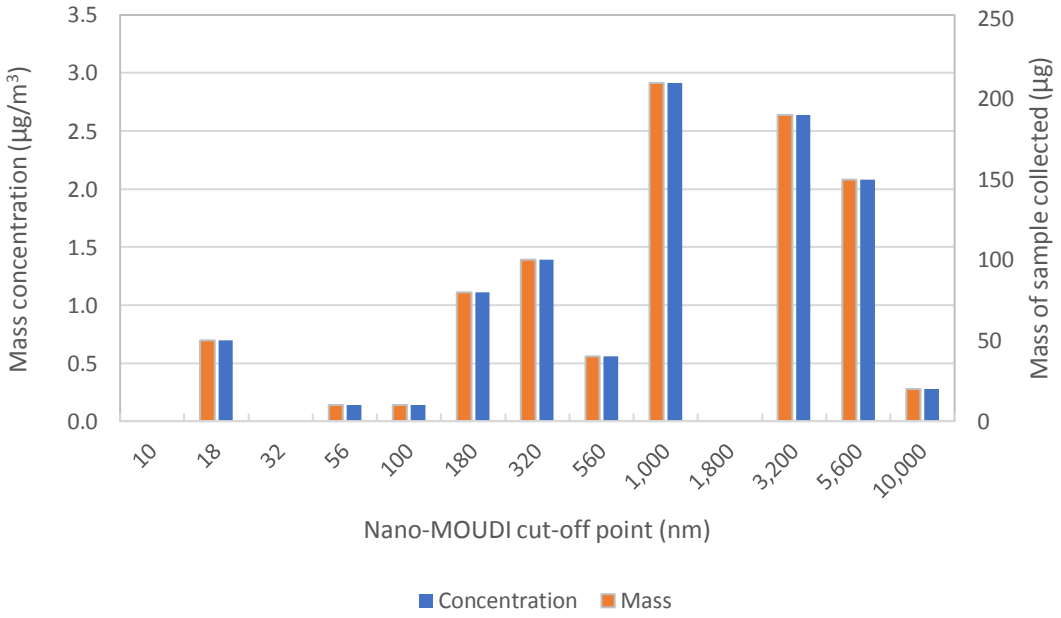


Figure 4.16 (f): Five-day mean airborne mass concentration and mass collected using the Nano-MOUDI II 125B from Curtin Site 6.

4.5.3 Comparison of particulate size distribution between sites

To compare the distribution of particle sizes across both areas, the individual masses for each fraction from all 6 sites in each were summed and charted (Figure 4.17). The Welshpool light industrial site produced a significantly greater mass of particles than the Curtin/Bentley non-industrial site, 13.76 mg compared to 4.71 mg.

In order to better compare size distribution between the two clusters of sites, the data was normalised such that both groups have the same total area, by dividing the mass of each stage by the total mass for that site cluster. (Figure 4.18). The normalised chart showed that the Curtin/Bentley cluster of sites had a greater proportion of the distribution in the sub 100 nm range however the Welshpool cluster of sites showed a greater proportion of the distribution on the far right side of the chart at the PM_{10} stage. Additionally, the Welshpool data is trending up at this point, suggesting an additional coarse particle mode beyond the range of measurement, whereas the Curtin data is tending downwards.

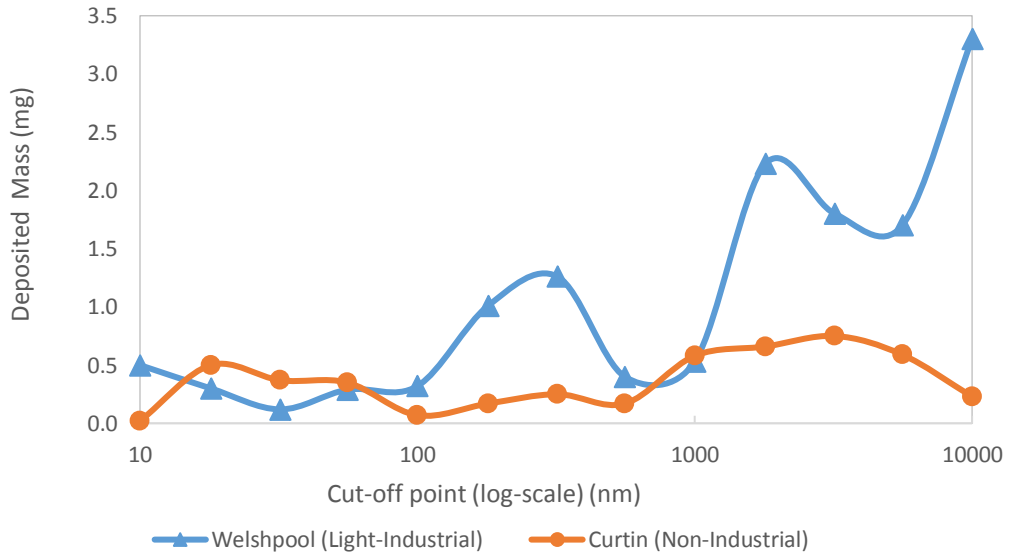


Figure 4.17: Comparison of summed masses of deposits collected from each stage of the Nano-MOUDI II™ 125B.

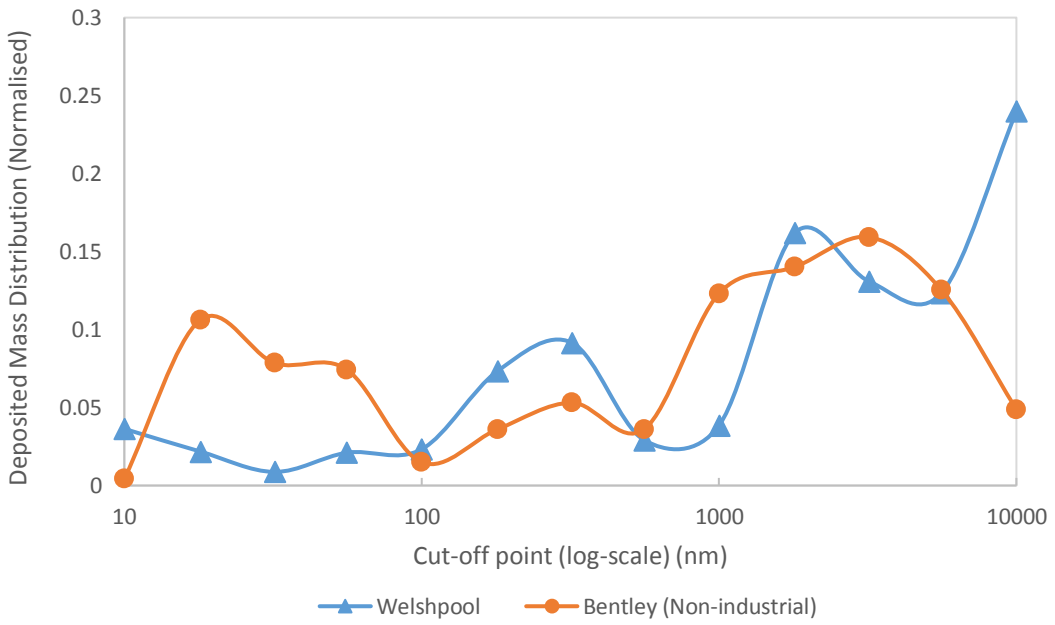


Figure 4.18: Comparison of 'Normalised' masses for the Nano-MOUDI II™ 125B data between the two clusters of sites.

4.6 Inductively coupled plasma – Mass spectrometry results

Tables 4.21 and 4.22 summarise the metal speciation results performed using the ICP-MS on deposits collected. Results for each site were grouped into $PM_{2.5}$ and PM_{10}. Metals that were 'below the detectable limit' (BDL) for both size fractions at all sites within the cluster were omitted from the tables. Aluminium was also omitted from both tables due to possible contamination from the aluminium foils used for sample collection. Levels were generally low so are presented as picograms ($\mu g \times 10^{-6}$) per cubic meter (10^{-12} pg/m^3) **to** make the values easier to compare.

In most cases metals were detected predominantly in the $PM_{2.5}$ fraction, with little or no additional metal in the PM_{10} fraction. This is of note since health effects for ultrafine particles have been shown to correspond most closely with particle number or surface area meaning that a given mass of one micron particles would have a greater health effect than the same mass of 10 µm particles.

Results for the Welshpool industrial area are presented in table 4.21. Calcium and arsenic were not detected in any samples within the limits of detection used and were removed from the table. Results for the Curtin/Bentley non-industrial area are presented in table 4.22. For this cluster, calcium and caesium were not detected in any samples within the limits of detection used and were removed from the table.

Table 4.21: Summary of mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Welshpool industrial area.

Metals (pg)/m^3 (pg=µg x 10^{-6})	Site 1 Domestic Residence Kewdale		Site 2 High School Kewdale		Site 3 DMP Core Library Carlisle		Site 4 Town of Vic Park Depot, Carlisle		Site 5 WA Museum Store Welshpool		Site 6 Domestic Residence, Carlisle	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Antimony (Sb)	285.4	143.8	626.3	318.2	389.6	240.2	552.1	226.0	664.1	274.8	1,481.5	1,193.9
Barium (Ba)	3,723.8	1,548.6	6,852.5	2,649.4	8,990.5	3,972.3	10,011.4	1,730.5	10,612.2	3,302.6	8,007.9	5,383.6
Beryllium (Be)	17.6	BDL	BDL	BDL	34.2	34.2	BDL	BDL	BDL	BDL	BDL	BDL
Bismuth (Bi)	101.3	101.3	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	1893.3	BDL
Cadmium (Cd)	44.1	27.1	290.9	73.9	106.4	79.1	28.6	28.6	159.0	64.7	66.1	66.1
Cerium (Ce)	107.0	78.0	466.5	174.1	330.3	62.4	434.6	60.7	988.4	284.3	419.8	207.1
Cesium (Cs)	126.2	50.3	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL
Chromium (Cr)	915.6	915.6	710.7	236.9	BDL	BDL	BDL	BDL	180.9	180.9	**8,205.7**	**6,587.0**
Cobalt (Co)	3.2	3.2	23.8	7.9	BDL	BDL	*2552.0	*BDL	74.9	18.1	37.1	BDL
Copper (Cu)	4,393.5	1,599.4	7,189.8	2,803.7	7,888.1	5,713.2	6,581.7	1,773.2	7,750.9	2,771.2	9,945.4	7,547.2
Europium (Eu)	5.3	2.3	8.9	2.1	7.1	1.4	10.9	0.8	15.6	2.8	14.1	10.2
Holmium (Ho)	1.1	0.3	3.8	0.8	3.4	0.7	5.6	0.5	6.6	1.3	7.6	5.7

Table 4.21: Summary of mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Welshpool industrial area (cont'd).

Metals (pg)/m^3 (pg=µg x 10^{-6})	Site 1 Domestic Residence Kewdale		Site 2 High School Kewdale		Site 3 DMP Core Library Carlisle		Site 4 Town of Vic Park Depot, Carlisle		Site 5 WA Museum Store Welshpool		Site 6 Domestic Residence, Carlisle	
	PM_{10}	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**
Iron (Fe)	117,833.5	47,770.4	407,370.0	140,687.1	611,574.9	381,821.6	576,869.4	74,086.3	673,374.7	162,308.3	254,668.7	109,477.9
Lanthanum (La)	**161.1**	**97.3**	271.1	98.3	266.1	86.3	497.2	84.7	466.1	139.7	434.5	272.4
Lead (Pb)	33.0	33.0	29,353.1	3,349.0	1,570.7	1,405.0	398.0	398.0	7,890.9	1,808.3	8,137.6	8137.6
Magnesium (Mg)	118,832.1	46,249.7	304,727.9	130,277.6	337,631.9	143,420.2	230,730.9	64,173.8	496,077.2	167,621.6	852,874.7	622,599.3
Manganese (Mn)	1,988.3	1,235.7	9,209.8	6,147.9	6,754.9	3,936.7	41,809.0	11,599.1	1,7461.6	6,294.1	4,724.7	3,029.7
Molybdenum (Mo)	11.1	11.1	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL
Nickel (Ni)	821.3	821.3	831.8	277.5	BDL	BDL	BDL	BDL	227.7	227.7	10,861.5	8,861.8
Palladium (Pd)	429.7	231.6	488.4	158.5	86.6	BDL	BDL	BDL	436.2	135.1	1,585.2	1,235.2
Phosphorous (P)	132,334.6	89,481.6	288,988.9	137,212.6	323,720.0	168,180.0	294,642.5	104,367.7	205,136.3	111,496.3	138,597.6	110,031.8
Potassium (K)	76,824.2	41,256.1	252,468.5	112,663.9	193,507.5	101,116.4	266,548.2	67,599.0	288,486.6	146,815.8	436,802.9	328,250.3
Praseodymium (Pr)	9.0	3.0	38.7	10.3	38.6	8.6	72.7	7.6	69.6	15.8	48.6	23.9
Rubidium (Rb)	350.2	188.9	538.6	201.1	417.4	149.2	648.6	106.5	539.5	184.7	183.4	26.7

Table 4.21: Summary of mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Welshpool industrial area (cont'd).

Metals (pg)/m^3 (pg=µg x 10^{-6})	Site 1 Domestic Residence Kewdale		Site 2 High School Kewdale		Site 3 DMP Core Library Carlisle		Site 4 Town of Vic Park Depot, Carlisle		Site 5 WA Museum Store Welshpool		Site 6 Domestic Residence, Carlisle	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Samarium (Sm)	9.6	4.1	31.8	9.1	30.3	7.6	48.9	5.5	45.7	9.7	41.5	24.4
Scandium (Sc)	BDL	BDL	18.3	BDL	11.2	BDL	12.3	BDL	81.8	17.9	15.8	BDL
Silver (Ag)	152.4	152.4	1,493.6	1,493.6	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL
Sodium (Na)	1105,666.8	465,939.3	2343,654.5	1052,466.3	629,870.5	1230,305.3	2046,175.3	623,390.8	3707,958.1	1369,202.6	7873,654.9	5965,740.1
Strontium (Sr)	1697.9	687.4	5590.4	1,977.8	5,556.5	2,127.2	6,952.9	1,490.6	7,043.8	2,373.5	8,168.9	5,695.1
Sulphur (S)	305,259.4	305,259.4	318,994.7	106,331.6	BDL	BDL	BDL	BDL	71,083.7	71,083.7	28,313,71.5	2,176,843.7
Thallium (Tl)	2,767.3	1,042.9	101,77.5	3,158.8	8,923.9	2,813.4	16,750.0	2,249.8	13,929.2	4,668.1	4,321.6	1,603.4
Thorium (Th)	31.5	9.0	153.0	36.4	137.9	31.8	241.2	25.4	268.5	61.5	203.6	105.5
Titanium (Ti)	7.1	4.6	15.5	11.4	12.2	8.8	18.2	6.8	10.7	5.5	29.5	25.6
Uranium (U)	13.1	5.2	46.6	15.6	50.2	16.7	59.8	11.7	75.9	19.8	41.0	22.7
Vanadium (V)	507.6	329.6	1,375.2	721.6	1172.3	633.6	1560.8	465.5	1500.9	683.8	2,241.1	1,752.2
Yttrium (Y)	28.3	9.5	131.2	35.5	113.1	27.6	206.6	21.6	263.4	54.1	128.4	67.2

Table 4.21: Summary of mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Welshpool industrial area (cont'd).

Metals (pg)/m^3 (pg=µg x 10^{-6})	Site 1 Domestic Residence Kewdale		Site 2 High School Kewdale		Site 3 DMP Core Library Carlisle		Site 4 Town of Vic Park Depot, Carlisle		Site 5 WA Museum Store Welshpool		Site 6 Domestic Residence, Carlisle	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Zinc (Zn)	227,063.0	97,917.8	2986,802.5	581,384.0	828,964.2	591,298.4	575,083.9	204,054.6	1020,304.4	434,388.0	574,994.3	555,206.5
Zirconium (Zr)	1,069.6	278.3	1,204.8	494.6	1,036.6	637.9	6,332.4	741.9	1,933.9	813.9	2,360.2	2,101.5

* The high cobalt level recorded in the PM_{10} fraction at Site 4, (Town of Victoria Park Depot), is likely to be an error as this result is inconsistent with the BDL result obtained for the corresponding $PM_{2.5}$ fraction and also the low results obtained from other sites in the Welshpool cluster.

Note: There were no ICP-MS results recorded for any metals for the 5.6 µm stage at Site 1, domestic residence in Kewdale. This is likely to result in a lower PM_{10} value only as this size fraction is not included in $PM_{2.5}$ estimations

Table 4.22: Mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Curtin/Bentley non-industrial area.

Metals (pg/m^3) (pg=µg x 10^{-6})	Site 1 B400: Public Health		Site 2 B003: Childcare		Site 3 B402: Business		Site 4 B215: Engineering		Site 5 B611: Tech park		Site 6 B105: Library	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Antimony (Sb)	299.4	184.9	80.0	57.7	105.8	38.6	155.8	121.0	198.9	105.6	225.6	139.7
Barium (Ba)	3,339.5	2,0452.2	805.5	445.5	1,136.8	346.1	1,817.9	1,317.1	2,027.6	932.3	3,512.7	1,936.3
Beryllium (Be)	BDL	BDL	BDL	BDL	229.6	229.6	663.6	127.1	BDL	BDL	BDL	BDL
Bismuth (Bi)	BDL	BDL	BDL	BDL	BDL	BDL	1152.8	1152.8	139.6	139.6	1,033.7	468.9
Cadmium (Cd)	45.9	39.4	112.2	105.9	414.4	178.3	233.6	178.6	73.6	32.9	183.8	67.2
Cerium (Ce)	64.7	30.7	67.1	45.3	8.4	5.8	1.3	0.4	43.6	26.2	165.7	82.1
Chromium (Cr)	1,575.8	1,128.0	678.2	394.4	338.9	258.1	1,129.4	806.8	5,171.8	4,854.0	1,030.3	488.2
Cobalt (Co)	8.3	2.7	11.2	1.2	BDL	BDL	13.2	13.2	13.0	13.0	BDL	BDL
Copper (Cu)	2,929.8	1,871.0	1,708.3	1,432.2	701.5	194.6	3,729.2	3,268.3	1,647.4	806.0	2,991.0	1,438.5
Europium (Eu)	1.2	0.7	0.4	0.1	0.9	0.3	0.2	0.1	BDL	BDL	1.3	0.2
Holmium (Ho)	0.2	0.1	0.2	0.1	0.1	0.1	0.1	BDL	BDL	BDL	0.6	0.1
Iron (Fe)	51,161.3	20,868.3	13,729.6	2,665.9	16,876.5	3,211.8	11,663.7	3,612.4	32,252.8	17,104.2	58,995.9	14,119.3

Table 4.22: Mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Curtin/Bentley non-industrial area (cont'd).

Metals (pg/m^3) (pg=µg x 10^{-6})	Site 1 B400: Public Health		Site 2 B003: Childcare		Site 3 B402: Business		Site 4 B215: Engineering		Site 5 B611: Tech park		Site 6 B105: Library	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Lanthanum (La)	47.8	25.0	25.3	16.7	25.2	11.7	65.3	60.6	50.8	19.7	93.7	52.4
Lead (Pb)	1,231.5	1,231.5	5637.7	5,637.7	21,911.5	8,958.7	7,870.2	6,164.3	1,558.9	719.1	929.5	929.5
Magnesium (Mg)	53,507.3	29,980.1	37,220.0	32,055.3	56,817.6	13,959.5	116,473.3	75,702.8	183,439.5	86,051.7	134,253.2	80,695.3
Manganese (Mn)	1,697.2	1,269.2	535.7	348.0	513.9	228.3	571.3	345.3	694.0	426.7	1,375.9	695.3
Molybdenum (Mo)	24.4	13.8	89.6	89.6	BDL	BDL	BDL	BDL	BDL	BDL	7.0	2.3
Nickel (Ni)	2,485.6	1,835.9	1,382.4	1,015.6	831.0	442.7	2,456.1	2,037.2	2,940.8	2,205.0	2,767.4	1,553.1
Palladium (Pd)	379.6	284.6	266.3	207.3	168.2	73.7	343.0	271.4	267.7	126.7	423.2	279.3
Phosphorous (P)	26,731.1	19,857.6	20,930.1	16,528.2	9,826.6	4,349.5	3,075.9	1,174.8	5,454.5	3,082.0	22,706.6	11,914.7
Potassium (K)	46,636.8	33,316.8	34,401.6	29,041.2	39,398.9	18,317.1	82,861.6	63,639.0	101,130.4	57,536.8	84,419.7	48,969.3
Praseodymium (Pr)	5.5	3.3	2.6	0.8	2.6	0.7	1.7	1.0	3.0	0.6	9.0	3.8
Rubidium (Rb)	BDL	BDL	BDL	BDL	14.9	14.9	BDL	BDL	BDL	BDL	BDL	BDL
Samarium (Sm)	5.0	3.1	2.3	0.8	2.6	0.8	2.0	0.4	2.5	0.6	7.4	3.0

Table 4.22: Mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Curtin/Bentley non-industrial area (cont'd).

Metals (pg/m^3) (pg=µg x 10^{-6})	Site 1 B400: Public Health		Site 2 B003: Childcare		Site 3 B402: Business		Site 4 B215: Engineering		Site 5 B611: Tech park		Site 6 B105: Library	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Scandium (Sc)	BDL	BDL	BDL	BDL	BDL	BDL	8.7	2.3	BDL	BDL	BDL	BDL
Silver (Ag)	399.3	399.3	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL	BDL
Sodium (Na)	341,377.3	188,888.6	138,440.7	115,109.6	429,809.2	104,757.1	894,655.8	566,661.9	1471,377.4	687,252.7	963,910.0	599,131.5
Strontium (Sr)	798.0	488.7	235.8	126.1	588.4	147.4	784.1	508.6	1321.3	581.7	1060.3	602.5
Sulphur (S)	346,919.6	231,515.2	80,143.9	80,143.9	187,211.2	125,949.2	BDL **	BDL**	111,542.5	111,542.5	832,118.5	520,050.2
Thallium (Tl)	2,839.7	2,416.8	704.9	385.1	232.0	44.8	95.8	31.6	126.9	BDL	1117.9	371.9
Thorium (Th)	17.8	9.6	10.0	3.3	7.8	1.7	7.9	6.0	6.8	1.8	35.0	15.7
Titanium (Ti)	6.2	5.4	4.8	4.0	2.0	1.4	3.3	2.8	4.8	3.3	1.6	1.2
Uranium (U)	5.1	3.1	3.9	1.6	2.3	0.5	2.8	2.0	3.9	1.6	10.7	5.2
Vanadium (V)	187.5	132.8	114.5	77.3	116.3	66.2	189.8	157.2	355.8	273.0	259.0	150.6
Yttrium (Y)	20.2	11.3	9.9	4.0	9.5	2.1	8.3	5.5	12.4	3.8	33.7	13.5
Zinc (Zn)	69,841.3	54,481.3	35,328.5	28,217.8	19,208.3	7,568.0	304,221.3	298,087.4	48,964.3	16,452.3	22,695.2	14,533.8

Table 4.22: Mass concentrations (pg/m^3) of individual metals in PM_{10} and $PM_{2.5}$ fractions of dust samples collected on the Nano-MOUDI II™ 125B from individual sites in the Curtin/Bentley non-industrial area (cont'd).

Metals (pg/m^3) (pg=µg x 10^{-6})	Site 1 B400: Public Health		Site 2 B003: Childcare		Site 3 B402: Business		Site 4 B215: Engineering		Site 5 B611: Tech park		Site 6 B105: Library	
	PM_{10}	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{2.5}$**
cZirconium (Zr)	490.6	400.1	375.9	259.0	19.8	19.8	848.6	812.8	266.0	198.2	583.0	535.4

** It appears that there was an error in processing this sample (B402 Engineering Pavilion, 18-22 August 2014) as it is unlikely that there would be BDL for sulphur given the proximity to other sites with recorded high levels of airborne sulphur and also the high levels of other PM collected during that week.

4.6.1 Comparison of concentration of metals between areas

To compare the combined level of metals in the ambient air sampled in industrial Welshpool to non-industrial Curtin/Bentley, the total mass of all 40 metals analysed was calculated as a proportion of the total mass of particulate matter collected for that particular area. Table 4.23 shows that the Welshpool area had a higher percentage of metals in all three size fractions, of the deposit collected than the Curtin/Bentley cluster. This would be expected in an industrial zone, particularly in areas where there are gaseous emissions being released. For PM_{10}, the proportion of metals in the industrial Welshpool cluster (2,742.94 µg metal/g PM**)** was 68.2% higher than in the non-industrial Curtin/Bentley cluster (1,630.67 µg metal/g PM**)** and for $PM_{2.5}$ the level of metals was 48.3% higher (1,428.30 µg metal/g PM compared to 963.18 µg metal/g PM). The proportions of metals in the smaller $PM_{0.1}$ fraction was 37.18% higher in the Welshpool samples (133.27µg metal/g PM) than the Curtin/Bentley samples (97.15 µg metal/g PM).

Table 4.23: Comparison of quantity of metal collected in different size fractions of air collected from Welshpool and Curtin/Bentley clusters given as a mass proportion of the total mass of the sample collected (µg/g).

Particle Size	**Mass proportion of metals in total mass collected (µg metal/g PM)**	
	Welshpool (Industrial)	Curtin/Bentley (Non-industrial)
PM_{10}	2742.94	1630.67
$PM_{2.5}$	1428.30	963.18
$PM_{0.1}$	133.27	97.15

Ratios of PM_{10} to $PM_{2.5}$ and $PM_{0.1}$, and $PM_{2.5}$ to $PM_{0.1}$ were calculated to see whether the proportions of metals were consistent between the Welshpool and Curtin/Bentley cluster of sites (Table 4.24). The PM_{10} to $PM_{2.5}$ ratios were also compared to those presented in Tables 4.16 and 4.19 that were calculated using data from DWER monitored stations.

Table 4.24: Comparison of ratio of mass of metals collected in each fraction size from Welshpool and Curtin/Bentley clusters.

Ratio	Ratio of metals in different size fractions	
	Welshpool (Industrial)	Curtin/Bentley (Non-industrial)
$PM_{2.5}$: PM_{10}	0.52	0.59
$PM_{0.1}$: PM_{10}	0.05	0.06
$PM_{0.1}$: $PM_{2.5}$	0.09	0.10

It was found that the ratios between clusters of sites were very similar with $PM_{2.5}$ being approximately 50%-59% of PM_{10} values and $PM_{0.1}$ being approximately 5% of PM_{10} values and 10% of $PM_{2.5}$ levels. The $PM_{2.5}$:PM_{10} ratios are slightly higher than ratios found for the DWER data where the ratios for the individual Welshpool sites ranged from 0.40-0.56 and for the individual sites in the Curtin/Bentley area ranged from 0.44-0.58. These ratios are fairly typical for urban areas. Sykes (2016) found that the average ratio in Scotland typically varied between 0.51 – 0.69, with a mean of 0.63, although it did vary with sites and years. The WHO Ambient Air Pollution Database (2014) uses city-specific conversion factors to estimate ratios for the same year when either $PM_{2.5}$ or PM_{10} data is not available. City-specific conversion factors are estimated as the mean ratio of $PM_{2.5}$ to PM_{10} of stations for the same year, and alternatively as the ratio of city values if the values by station were not provided. If national conversion factors are not available, regional ones are used, which are obtained by averaging country-specific conversion factors Australia, the U.S. and Singapore have a conversion factor of 0.6 whereas Canada is only 0.3-0.4, indicating that typically a smaller proportion of their PM is $PM_{2.5}$ (WHO, 2014).

Generally mean levels of metals for the Welshpool area were higher than levels obtained from the Curtin/Bentley cluster of sites. Higher levels were detected for antimony, beryllium, cobalt, copper, lead, manganese, vanadium, zinc, iron and sulphur. There was no arsenic detected in either area and the levels of nickel were similar across both areas, although the wider range for the Welshpool sites indicates that there were high levels in few sites. Although selenium is considered a hazardous metal, it is not routinely included in the suite of metals analysed by the ICP-MS. Cadmium levels were slightly higher in the Curtin/Bentley area although the mean value for both areas was well below the European Commission standard.

4.6.2 Comparison of ambient metal concentrations with standards

With exception of lead, there are no ambient air quality standards for metals in Australia. Lead is included in the AAQ NEPM and the maximum concentration is 0.50 µg/m^3 over a one year averaging period with the goal being no allowable exceedances in ten years. In comparison the NAAQS for lead in the U.S. is lower at 0.15 µg/m^3 as a rolling three month average (Standard concentrations generally decrease with an increased averaging period).

As there are no standards for comparison (with the exception of lead) the metal concentrations obtained in this study were benchmarked firstly to levels presented in a 2003 Internal Working Paper on 'Ambient concentrations of heavy metals in NSW' (Table 4.25). In this NSW project, ambient concentrations of selected metals were measured and compared to various standards including those from the European Commission, WHO and the U.S. EPA. Metals tested included the nine metals that the U.S. EPA has nominated as 'Hazardous Air Pollutants'– antimony, arsenic, beryllium, cadmium, cobalt, lead, manganese, nickel and selenium. Although not on the hazardous list, copper, vanadium and zinc were also included as exposure can have significant health effects in the workplace. Results from the current study were also compared to levels for typical rural, urban and industrial ambient air concentrations in the U.S presented in an overview of airborne metals regulations published by the U.S. EPA (Geiger & Cooper, 2010). As expected, for all metals, levels for rural areas were the lowest, followed by levels for urban areas with levels in industrial areas consistently being the highest.

For the metals presented in Table 4.25, the following comparisons were made with standard and guideline values as well as typical mean concentrations found in NSW and U.S. rural, urban and industrial areas:

- Mean antimony concentrations in both Perth metropolitan sites (0.66 ng/m^3 in the Welshpool sites and 0.18 ng/m^3 in the Curtin/Bentley sites) were lower than mean NSW levels (1.2 ng/m^3) and also rural US levels (<1 ng/m^3).

- Mean beryllium concentrations for both Welshpool (0.01 ng/m^3) and Curtin/ Bentley areas were very low (<BDL), less than the U.S. EPA standard (0.4 ng/m^3) and also less than typical levels recorded in U.S rural areas (0.1 ng/m^3). Beryllium was not detected in the NSW study.

- Mean cadmium concentrations were quite low in both areas with the Curtin/ Bentley level (0.18 ng/m^3) slightly higher than the Welshpool level (0.12 ng/m^3). Mean levels in both areas were lower than the European commission standard of 5.0 ng/m^3 as well as the typical level in U.S. rural areas (1.0 ng/m^3). The Curtin/Bentley value was similar to the NSW mean level of 0.17 ng/m^3.

- Mean cobalt concentrations for the Welshpool area (0.45 ng/m^3) were higher than levels obtained in NSW (0.19 ng/m^3) and typical U.S. rural areas (0.1 ng/m^3), although less than U.S. urban areas (0.5 ng/m^3). Mean levels for the Curtin Bentley site were very low (0.01 ng/m^3), less than both NSW values and typical U.S. rural values. There is no standard for ambient cobalt.

- Mean copper concentrations for both the Welshpool and Curtin/Bentley areas, 7.29 ng/m^3 and 2.28 ng/m^3 respectively, were below the mean concentration in NSW (8.2 ng/m^3), and the typical urban level in the U.S. (290.00 ng/m^3). They were higher than the typical U.S. rural level (0.10 ng/m^3). There are no standards for ambient copper.

- Mean lead concentrations in both areas were very low, 7.90 ng/m^3 in the Welshpool area and 6.52 ng/m^3 in the Curtin/Bentley area. This is less than mean levels detected in NSW (30.0 ng/m^3), typical U.S. rural areas (20.0 ng/m^3), and well below the WHO guideline and Australian AAQ NEPM (500.0 ng/m^3).

- Mean manganese concentrations for both the Welshpool and Curtin/Bentley areas, 13.66 ng/m^3 and 0.90 ng/m^3 respectively, are below the WHO guideline of 150 ng/m^3, the NSW average (18.0 ng/m^3) and the typical U.S. urban level (20.0 ng/m^3). The Curtin/Bentley mean concentration (0.90 ng/m^3) is also slightly lower than the typical U.S. rural concentration (1.0 ng/m^3).

- Mean nickel concentrations were very low and similar for both sites, 2.12 ng/m^3 for the Welshpool area and 2.14 ng/m^3 for the Curtin/Bentley area. Mean levels for both were less than the European Commission standard of 20.0 ng/m^3, the NSW mean (3.5 ng/m^3) and typical U.S rural areas (6.0 ng/m^3).

- Mean vanadium concentrations in both areas were quite low, (1.39 ng/m^3 for the Welshpool area and 0.16 ng/m^3 for the Curtin/Bentley area) which is well below the WHO standard of 1000 ng/m^3 and also below the mean level detected in NSW (2.6 ng/m^3). The Welshpool level is greater than the U.S.

rural (0.8 ng/m^3) level but less than the U.S urban level (65.0 ng/m^3) while the Curtin/Welshpool level is less than the U.S rural level.

- Mean zinc concentrations were higher in both the Welshpool and Curtin Bentley areas (1035.54 ng/m^3 and 79.67 ng/m^3 respectively) than levels recorded in NSW (33.0 ng/m^3). The concentrations recorded in Welshpool were greater that U.S. urban levels (103 ng/m^3) but less than U.S. industrial levels (5000 ng/m^3). Monitored levels at the Curtin/Bentley site (79.67 ng/m^3) were greater that levels in U.S. rural areas (6.0 ng/m^3) but less than U.S. urban areas (103.0 ng/m^3).

Several other 'non-hazardous metals' were detected in elevated levels compared to the majority of the metals. In both cluster of sites there were elevated levels of sulphur, sodium and magnesium. Welshpool also had elevated levels of iron, phosphorous and potassium. As there are no ambient standards for any of these metals, no inference can be made from the monitored concentrations.

Table 4.25: Comparison of concentration of selected metals detected in industrial Welshpool and non-industrial Curtin/Bentley areas with levels detected in NSW study and internationally recognised goals / standards (Department of Environment and Conservation [DEC] (NSW), 2003). Typical U.S. national ambient concentrations are also given for comparison (Geiger & Cooper, 2010). For ease of reading and interpreting, all values are given as µg/m^3 x 10^{-3} (nanograms).

Metal	**Ambient Air Standard** µg/m^3 x 10^{-3}	**Source of Standard**	**Welshpool** Industrial µg/m^3 x 10^{-3} ± SD (Range)	**Curtin/Bentley** Non-industrial µg/m^3 x 10^{-3} ± SD (Range)	**Mean Ambient NSW ***** µg/m^3 x 10^{-3} (Range)	**Typical U.S. National Ambient Air Concentrations**		
						Rural µg/m^3 x 10^{-3}	**Urban** µg/m^3 x 10^{-3}	**Industrial**** µg/m^3 x 10^{-3}
***Antimony**	–	None available	0.66 ± 0.42 (0.29-1.48)	0.18 ± 0.08 (0.08-0.30)	1.2 (0.04-4.6)	<1.0	32.0	550.0
***Arsenic**	6.0	EC ^	BDL	BDL	0.6 (0.09–2.5)	2.0	20.0	7600.0
***Beryllium**	0.4	U.S. EPA^^^	0.01 ± 0.01 (0.00-0.03)	BDL	Nil Detected (BDL)	0.1	2.0	10.0
***Cadmium**	5.0	EC^	0.12 ± 0.10 (0.03-0.29)	0.18 ± 0.14 (0.05-0.41)	0.17 (0.03-1.0)	1.0	8.0	600.0
***Cobalt**	–	None available	0.45 ± 1.03 (0.00-2.55)	0.01 ± 0.01 (0.00-0.01)	0.19 (0.10-0.39)	0.1	0.5	400.0
Copper	–	None available	7.29 ± 1.82 (4.39-9.95)	2.28 ± 1.11 (0.70-3.73)	8.2 (2.4-28)	0.1	290.0	870.0
***Lead**	500.0	EC^ NEPM^^ USEPA^^^ WHO^^^^	7.90 ± 11.12 (0.03-29.35)	6.52 ± 8.04 (0.93-21.91)	30.0 (2.4-99)	20.0	40.0	760.0

***Manganese**	150.0	WHO^^^^	13.66 ± 14.77 (1.99-41.80)	0.90 ± 0.51 (0.51-1.70)	18.0 (3.7-119)	1.0	20.0	300.0
***Nickel**	20.0	EC^	2.12 ± 4.30 (0.00-10.86)	2.14 ± 0.84 (0.83-2.94)	3.5 (0.86-20)	6.0	20.0	170.0
Vanadium	1000.0	WHO^^^^	1.39 ± 0.56 (0.51-2.24)	0.16 ± 0.12 (0.00-0.36)	2.6 (0.16-49)	0.8	65.0	500.0
Zinc	–	None available	1035.54 ± 992.78 (227.06-2986.80)	79.67 ± 112.57 (0.46-304.22)	33.0 (11-71)	6.0	103.0	5000.0

* Designated as ‘Hazardous Air Pollutants’ by the U.S.EPA

** Concentrations found in ambient air in the U.S. in the vicinity of industries known to emit metals (Geiger & Cooper, 2010).

*** Mean results from New South Wales study (DEC (NSW) 2003)

^ European Commission 2003

^^ NEPC 1998 – Australian goal based on AAQ NEPM (based on TSP)

^^^ U.S. EPA

^^^^ World Health Organisation 2000 Guidelines (WHO, 2006)

Chapter 5 Discussion

This chapter discusses the main findings as presented in the previous chapter and the implications and potential health effects of these findings for human health.

5.1 Outcomes from Stage One

Results from the first stage of this study found that changes in daily air quality in Perth are likely to be associated with increases in hospitalisations for selected morbidity. Statistically significant associations were found between pollutants and mean numbers of admissions, in many cases when the pollutant levels were well below the current recommended standard found in the Ambient Air Quality National Environmental Protection Measure. This is consistent with results found in other Perth, Australian and international studies (Hinwood et al., 2006, Barnett, 2014; Crouse et al., 2012; Malmquist et al., 2013), although there was some variation in the actual pollutants that were found to be significant.

This study is unique in Perth and Australian air quality studies as the focus is on low level exposure. The main pollutants of concern overall were found to be carbon monoxide and nitrogen dioxide. Both were significantly associated with increases in admissions for all illness hospitalisations investigated, for most combinations of covariates (Table 4.8(e)). This is despite the mean monitored CO levels (0.00 ppm – 3.04 ppm) being well below the CO Australian Air Quality Standard (AAQS) of 9.0 ppm (8 hours) and the mean NO_2 levels monitored between 0.000 – 0.019 ppm (1.92 pphm) which is well below the NO_2 AAQS of 0.12 ppm (1hr). The maximum increase in risk of hospitalisation for total respiratory conditions following exposure to levels of CO above baseline levels was 3.72 times, or a 272% increase in the likelihood of being hospitalised following exposure (Table 4.10 (a)). The maximum increase in risk of hospitalisation following exposure to nitrogen dioxide above baseline levels was also for 'total respiratory' conditions. This risk increased 2.75 times or a 175% increase in the likelihood of being hospitalised following exposure (Table 4.10 (b)).

Elevated mean ozone levels were only found to be significantly associated with increases in hospital admissions for cardiovascular disease, COPD and pneumonia/ influenza / acute bronchitis (Table 4.8 (e)). The maximum increase in risk with ozone exposure occurred with for cardiovascular disease and the risk increase up to 3.31

times the risk for the lowest level of exposure (Table 4.9 (c)). The average ozone levels monitored ranged from 0.0075 – 0.004 ppm (3.74 pphm) which is well below the ozone AAQS of 0.1 ppm (1hr) and 0.08 ppm (4 hrs). Previous studies in metropolitan Perth by Hinwood et al. (2006) did not find any significant associations between average levels of ozone and increased hospitalisation for any of the studied illnesses.

Increased mean $PM_{2.5}$ levels were found to be associated with a 22% increase in the likelihood of both a 10% and 20% increase in hospitalisations for asthma, after adjusting for seasons (Table 4.11(c)). Increased mean PM_{10} levels were not found to be associated with increases in hospitalisations for any of the illness conditions studied. The ranges of the levels for both $PM_{2.5}$ and PM_{10} levels over the ten-year period were large and the upper limit for both exceeded the relevant AAQSs. The maximum PM_{10} level was 68.30 µg/m^3 which exceeded the AAQS of 50 µg/m^3 (24hrs) and the maximum $PM_{2.5}$ levels was 40.78 µg/m^3 which exceeded the AAQS of 25 µg/m^3 (24hrs) (Table 4.7(c), Table 2.1). Despite these exceedances, the mean levels of PM_{10} and $PM_{2.5}$ over this time period were 17.0 ± 6.5 µg/m^3 and 8.0 ± 3.1 µg/m^3 respectively (Table 4.6). These findings indicate that there were isolated instances of high levels as would be expected with bushfires, prescribed burns and dust storms which may have impacted hospitalisations although any increases would have been smoothed out in the averaging process over the ten-year time period.

Based on previous published literature, (Goldstone, 2015; Jalaludin & Cowie, 2012; Keywood, Hibberd & Emmerson, 2017; Laumbach & Kipen, 2012; Meng et al., 2016; Morawska, 2007), it would be expected that there would be more significant associations found between PM, in particular $PM_{2.5}$, and increases in admissions for cardiovascular and respiratory illnesses as the literature review provided many examples of such relationships (see Section 2.3.4.2). Studies in Melbourne, Sydney and Brisbane have all found strong associations between admissions for respiratory and cardiovascular disease, asthma COPD and daily mortality (all causes, respiratory and cardiovascular causes) and increases in fine particles concentrations (EPA (Victoria), 2001; Morawska, 2007; Petroeschevsky et al., 2001; Simpson et al., 2005).

Overall, significant associations were found between all disease categories and selected pollutants, although not for all combinations of covariates. These associations were all influenced by whether the elevated levels were causing an increase of 10 or 20% in admissions and whether or not the relationship had been adjusted for seasons. Cardiovascular illness, pneumonia/ influenza/ acute bronchitis

and COPD admissions were all increased with elevated CO, NO_2 and ozone. Total respiratory illness and 'other' respiratory admissions were increased with elevated CO and NO_2. Asthma admissions also increased with elevated CO and NO_2, and was the only illness category affected by $PM_{2.5}$ levels.

The evidence from this study demonstrates statistically significant associations between hospitalisations for cardiovascular and respiratory conditions and exposure to ambient pollutants well below the current Australian standards. Given the direct and indirect health care costs incurred with these hospitalisations, as well as the associated decrease in well-being and loss of productivity, there is justification for further reductions in the ambient air NEPM for selected pollutants. A reduction in the maximum concentration for both short and long term exposure carbon monoxide, nitrogen dioxide and ozone is likely to provide economic and health benefits, not only to those who risk hospitalisation but also the public in general.

Of particular concern is those hospitalisations that occurred when pollutant levels were in the second or middle tertile as these levels are not only lower than the relevant standard but are also lower than the top third of recorded values for that pollutant over the time period. The main pollutants that were responsible for an increase in hospitalisations following exposure to these levels were carbon monoxide and nitrogen dioxide. People who are more sensitive to these levels of pollutants and require hospitalisation following exposure are likely to be those groups recognised as being vulnerable, the very young, the elderly, and those with a previous history of cardiovascular or respiratory disease. The implications here are significant in terms of the provision of health care and associated costs as the demographic profile of the metropolitan Perth population is skewing towards an increase in the number of people in the older age group, including those who have survived previous respiratory or cardiovascular disease. Not only is this group more vulnerable to developing respiratory and cardiovascular disease in the first place, they are more likely to be admitted to hospital and from the 2001-2010 data it can be seen they are also likely to stay in hospital longer.

Comparison of the admissions data from 2001-2010 with mean daily numbers of hospitalisations during a seven year period the previous decade(1992-1998) (Hinwood et al., 2006), revealed a substantial increase in the mean daily number of hospitalisations for the various categories of illness (Table 4.4). There was a marked increase in the population of metropolitan Perth over the time period however even though population figures were standardised to account for this increase, there was

still a marked overall increase in the mean number of daily admissions in all categories. The increases in standardised mean admissions for most illness categories may have been due to factors such as changes in case definitions, criteria for admission, the age profile and the health status of the population. This decrease in asthma admissions is likely to be due to improved home management of asthma cases and changes in admission criteria.

Comparing pollutant levels between the two decades showed that there had been some changes in mean levels although it should be noted that even though there were some increases, all mean pollutant levels for both decades were below the standards set in the Australian Air Quality NEPM (Table 4.6). Mean NO_2 levels had increased for 1-hour average (from 24.8 to 41.0 ppb), however had decreased for 24-hour average (10.3 to 6.1 ppb). Mean ozone levels for all averaging periods, 1-hour, 4-hour and 8-hour, had increased although the increase was far greater in the warmer months than the cooler months. There was little change in CO or PM levels from 1992-1998 to 2001-2010. These changes in pollutant levels do not appear to be able to explain the increase in hospital admissions, especially as the main pollutant increases have occurred with ozone which showed very few associations with hospital admissions.

In comparing the mean values for temperature and humidity obtained from 2001 to 2010 to values reported by Hinwood et al. (2004, 2006) the previous decade (1992-1998), it was noted that there had been no significant change in either humidly or temperature. In both decades the mean temperature from November to April was higher (21.8°, 21.4°) than the mean temperature from May to October (15.1°C, 14.8°C) respectively. Humidly levels for both decades were also very similar with levels for May to October (71.4%, 69.9%) being higher than levels for November to April (56.2%, 56.1%) respectively. From this it can be seen that temperature and humidity are not likely to have contributed to the changes in the number of admissions over the decade.

The overall increase in hospitalisations may be due to a number of factors including changes in case definition, hospital diagnosis procedures and variations in criteria for admission as well as changes in the age profile and health status of the population. It could also be due to external factors such as an increase in the capacity of metropolitan hospitals at the expense of smaller regional hospitals. There may also have been anomalies introduced with the population increases as much of the growth has occurred in the outer metropolitan areas which may not have been included in

the previous study. In comparing the 2001 – 2010 data to data from the previous decade (Hinwood et al, 2006), the inclusion criteria and variables such as illness categories and residential and demographic data were purposely consistent although internal processes in cleaning the data and categorising cases may have varied. The only illness category that showed a decrease in admissions from 1992-1998 to 2001-2010 was asthma. This is likely to be due to the improved home management of asthma cases and a subsequent change in admission criteria.

This increase in admissions is likely to have significant financial implications for the health care sector in terms of costs for not only hospitalisations but also after care upon discharge. In addition to the health care costs, if this increase in hospitalisation is reflective of the overall health status of the population, there may also be consequential productivity losses associated with unwell workers either not participating in the workforce or not performing to their usual standard.

The results from the first stage of this study indicate that policy-makers and planners should be mindful that even if NEPMs are being met the potential impact that planning decisions can have on population exposure to air pollutants should be considered. In Perth there are calls to curb urban expansion by increasing population density in the metropolitan area. This would have the unintended consequence of increasing the number of people exposed to ambient pollutants present in concentrations capable of causing and exacerbating health effects. In planning for future developments, climate change projections should also be considered as increased temperatures cause the increased production in ozone.

5.2 Outcomes from Stage Two

The second stage of this study investigated concentration and metalliferous composition of particulate matter in ambient air in industrial and non-industrial areas of metropolitan Perth. It was found that levels of metals in both areas are well below any relevant standards and that levels in industrial area were almost consistently greater than in the non-industrial area.

Mass concentrations of metals showed that although levels were generally low, there were far greater levels detected from the industrial cluster of sites than the non-industrial sites. In particular, there were high levels of zinc detected in the Welshpool cluster as would be expected given the nature of industry based in the area. It is believed that at the time of the study a galvanising plant was in operation in the area.

Although zinc is considered by the U.S. EPA to be a 'hazardous pollutant', there are currently no ambient air standards for zinc in Australia, the US or the European Commission. Given the U.S. EPA designation as a 'hazardous pollutant' and the high levels present in ambient air in industrial zones, consideration should be given to developing a standard for maximum zinc concentration in ambient air, particularly when industrial areas are proximate to residential areas.

It should be noted that due to the lack of ambient air standards for most of the metals reported in Tables 4.21 and 4.22, it is difficult to interpret the results. While some levels appear high, it is difficult to justify whether this is likely to present a health concern. Table 4.25 outlines the metals that are considered by the U.S. EPA to be 'hazardous pollutants' however several of these (antimony, cobalt, copper) do not have ambient standard values in Australia or the U.S.A, and are not listed in the European Commission or WHO Air Quality Guidelines.

In the past, the Welshpool area was predominantly industrial / light industrial with many small-medium enterprises within the Local Government area. This particular area is quite close to the central business district and there has been a gradual change in land use with residential development increasingly encroaching on the Welshpool industrial area. As there is still a substantial amount of light industrial activity including metal fabrication, smelting and chrome plating there is some concern about resident exposure to metal and metal oxide nanoparticles. Due to the nature of the industries, it is possible that lead, titanium and a range of other nano-metals may be emitted at significant rates. Lead emissions from a radiator manufacturer located in this area have previously been reported (Mullins and Latunij, personal communication, 2017), however were not detected on this occasion. For this study both the Welshpool and Curtin/Bentley areas had ambient lead levels well below the AAQS. Previous ambient air monitoring of the non-industrial Curtin/Bentley site in 2009-2010 did not find any pollutant levels of concern however given the number of staff, students and visitors on the campus and nearby sporting and recreation facilities on a daily basis, the area, including new sites was selected for monitoring on this occasion.

The PM estimates for the Welshpool area correlated quite well with the values obtained from the Nano-MOUDI with the exception of site 6 where a concentration of 37.2 µg/m^3 was obtained for PM_{10} and 30.3 µg/m^3 was obtained for $PM_{2.5}$. The estimated PM_{10} concentration for that week was 15.4 µg/m^3 and the estimated $PM_{2.5}$ concentration was only 7.4 µg/m^3. Apart from this one instance all other values for

PM_{10} and $PM_{2.5}$ were below the air quality standards of 50 µg/m^3 and 25 µg/m^3 respectively. This is to be expected as the measurements were performed during the winter months and there were several instances of light rainfall which provides air cleaning. The results for total suspended particles from the High Volume Sampler indicated that at all sites (where there were no technical issues with the HVS) there was a significant component of larger particles that had an AED greater that 10 µm so was not collected by the Nano-MOUDI and less than 50 µm which is the cut-off size for the HVS.

The plots produced for the Welshpool area show that for most sites, the particles tend to be mainly clustered around the larger fractions, with several of the sites having very low concentrations of particles in the smallest fractions. This is fairly typical of industrial areas where there are high concentrations of dust and particles from grinding and other mechanical processes. At many of the sites within the Curtin / Bentley cluster, particles were spread more across the size range however they were mainly in the fine to ultrafine size fraction. This is also typical of a non-industrial area as most of the particulates would be from vehicular traffic on campus.

The design and results from this current study have similarities with a study conducted in the U.K. by Taiwo et al. (2014). This previous study also conducted speciation and characterisation of particulate matter in ambient air from an industrial area and an urban (non-industrial) background site. Similar to this study, they found predominantly fine, mainly traffic related, particles at the urban site whereas the particles collected at the industrial site were mainly in the coarse fraction. Overall they found higher levels of metals at the industrial site due to marine influences as well as the presence of industry.

5.3 Limitations of the study

Although there were significant findings from this study, there were several limitations that impacted on the completeness of the data and subsequent outcomes, in both stages of the study.

In the first stage of the study, the lack of Department of Water and Environmental Regulation (DWER) statutory monitoring stations within the central business districts of either the City of Perth or the City of Fremantle, meant that the mean pollutant levels calculated across the metropolitan area were not truly representative as they did not include the levels people are exposed to in the more densely populated areas of the city. In addition the short term nature of many of the DWER air quality monitoring programs, particularly in outer metropolitan areas, meant that there were often gaps in the data. Several sites did not have continuous monitoring making long range analysis and forecasting difficult. There were also frequent technical interruptions in statutory DWER monitoring which also led to large gaps in many data sets.

In terms of the hospital data, available admission data does not indicate pre-existing respiratory or cardiovascular illness, unless it resulted in hospitalisation in the given time frame. Likewise the data does not indicate the presence of any related conditions that may make the person more vulnerable if it was treated out of hospital, or if it was not one of the underlying reasons for admission. It is therefore not possible to determine what influence these factors may have had on the analysis.

A further limitation also noted in many other studies (Hinwood et al., 2004, 2006) is the inability of this type of study to distinguish between mixtures of pollutants and the difficulty in determining independent effects from a range of interrelated, correlated factors and the confounding of co-pollutant effects. These studies are also unable to assess the interaction effects between age, season and pollutant levels on admission due to each condition. These effects can best be studied when individual patients are followed prospectively as a cohort. Lag periods were not determined in this study as they are most relevant to acute pollution events which tend to be diluted by averaging concentrations over serval days and sites.

For Stage two monitoring there was only one set of monitoring equipment, so only one site was monitored at a time. This meant there was up to six weeks between sampling at the first and last sites at each location which could give a marked variation in climatic conditions. It also meant that there was a 10 month delay between sampling

in the Welshpool and Curtin/Bentley areas so that weather conditions were comparable. The monitoring in Stage Two was undertaken in late winter and spring when there was substantial rainfall. As has been discussed PM levels in the Perth metropolitan area are frequently elevated during the warmer months as a result of natural and deliberately lit bushfires. By monitoring in the cooler months these elevated levels were not observed.

Chapter 6 Conclusions and recommendations

This chapter brings together the discussion and addresses how the aim and objectives of the study were met. It also lists possible recommendations from the study that would improve the design and conduct of further research in this area.

6.1 Conclusions

This study has met the aims and objectives as set out in Chapter 1.

Aim 1: Evaluation of the relationship between daily levels of selected ambient air pollutants and respiratory and cardiovascular hospital admissions in the Perth metropolitan area from 2001-2010.

- *To evaluate the relationship between mean daily levels of selected ambient air pollutant concentrations and daily hospital admissions*
- *To estimate the extent to which the studied air pollutants influence the number of hospital admissions*
- *To compare the ambient air pollution concentrations and hospital admissions for respiratory and cardio-vascular illnesses between the last two decades*

This study evaluated the relationship between daily levels of carbon monoxide, nitrogen dioxide, ozone, PM_{10} and $PM_{2.5}$ and daily hospital admissions for cardio-vascular and respiratory illnesses in metropolitan Perth from 2001-2010. Historical hospital admission data from the Department of Health and air quality monitoring data the Department of Water, and Environmental Regulation was obtained and statistically analysed to determine significant associations.

This study found that changes in daily air quality in Perth are likely to be associated with increases in hospitalisations for selected illness conditions, in many cases when the pollutant levels are well below the current recommended standard found in the Ambient Air Quality National Environmental Protection Measure. There were significant associations between all illness categories and selected pollutants which were all influenced by various combination of covariates. Cardiovascular illness, Pneumonia/ influenza/ acute bronchitis and COPD admissions were all increased with elevated CO, NO_2 and ozone. Total Respiratory illness and 'Other' respiratory admissions were increased with elevated CO and NO_2. Asthma admissions also increased with elevated CO and NO_2, and was the only illness category affected by

$PM_{2.5}$ levels. There were no significant associations found with elevated concentrations of PM_{10}.

In comparing hospital admission and air quality monitoring data between decades, it was found that although there had been some small increases in overall and seasonal pollutant levels from 1992-1998 to 2001-2010, there was a marked increase in admissions (standardised to account for population increases) for all disease conditions, excepting asthma which had decreased. NO_2 and ozone levels had increased slightly although were still well below the levels set in the Australian air quality standards (Table 4.6). There was little change in CO or PM levels. These changes in pollutant levels do not appear to be able to explain the increase in hospital admissions, especially as the largest increases have occurred with ozone (in warmer months) which showed very few associations with hospital admissions.

Aim 2: Characterisation and speciation of fine and coarse particulates at sites representative of non-industrial (background) and light industrial (source) locations within metropolitan Perth.

- *Compare mass concentration and elemental composition of PM between light industrial and non-industrial areas*

Air quality monitoring and analysis in the second stage of the study was able to show that ambient levels of most metals (except cadmium and nickel) were significantly higher in industrial areas (Welshpool cluster of sites) than in non-industrial areas (Curtin/Bentley cluster of sites). Metals that are considered to be 'hazardous' by the United States Environmental Protection Authority were detected across both sites at levels well below any recognised standard or guideline including the World Health Organisation guidelines, U.S. EPA standards, European Commission standards and the Australian Ambient Air Quality National Environmental Protection Measure.

6.2 Recommendations

Based on the discussion and conclusion the following recommendations are made:

- It is recommended that more comprehensive air quality monitoring data is collected and analysed to determine trends in pollutant levels and health outcomes, focusing on low level exposure. Historical data contains frequent gaps due to equipment malfunction or the cessation of sampling campaigns. It is also recommended that the DWER upgrade their monitoring protocol and equipment so that more reliable, representative data is available.

- It is evident that higher spatial resolution of air quality data would be beneficial. New monitoring stations are being commissioned in the City of Perth and the City of Mandurah in the latter half of 2017 (DER, 2017) and it is recommended that these sites be included in any future metropolitan and regional air quality studies to overcome the lack of relevant spatial data, particularly relating to heavily populated, high traffic exposures.

- It is recommended that future studies include collection and analysis of more detailed patient data, including history of previous respiratory and cardiovascular conditions. Following patients prospectively would enable evaluation of the cause and effect relationship between ambient pollution levels and hospital admissions.

- The results of this study and many others indicate that there is a need to revisit the current Australian Ambient Air Quality NEPM, particularly the standards for carbon monoxide and nitrogen dioxide and consider reducing the maximum concentration allowable.

- With the exception of cadmium and nickel that were detected in similar concentrations across both areas, levels of airborne metals were consistently higher in the Welshpool industrial areas than the Curtin/Bentley non-industrial area. It is recommended that authorities responsible for local planning and policy decisions are made aware of the potential for resident exposure to airborne metals in areas surrounding and downwind of industrial emissions.

- Given the U.S. EPA designation of ambient zinc as a hazardous pollutant and the high levels measured in the Welshpool industrial area, it is recommended that a guideline or ambient standard be considered. This is particularly

important in the Welshpool area as there are residential areas downwind of the industrial zone.

- It is recommended that for future air monitoring studies, sampling is conducted over an extended period and includes both warmer and cooler months to enable comparisons of pollutants levels to be made across seasons. It would also be beneficial to use multiple sets of equipment to obtain simultaneous results across sites, which would reduce the influence of climatic variations.

- It is recommended that future air quality studies at the Curtin/Bentley site include Site 3 which is a child care centre. While this site did not have higher levels than other sites tested, given the age and vulnerability of the children who attend the centre, there is the potential for significant low level exposure. Monitoring would need to cover drop off and pick up peak periods and include qualitative data such as whether vehicles are idling for extended periods.

References

Andersen, Z. J., Bønnelykke, K., Hvidberg, M., Jensen, S. S., Ketzel, M., Loft, S., . . . Raaschou-Nielsen, O. (2012). Long-term exposure to air pollution and asthma hospitalisations in older adults: a cohort study. *Thorax, 67*(1), 6-11. doi:10.1136/thoraxjnl-2011-200711

Arranz, M. A., Moreno, M. F., Medina, A. A., Capitan, M. A., Vaquer, F. C., & Gomez, A. A. (2014). Health impact assessment of air pollution in Valladolid, Spain. *BMJ Open, 4*(e005999), 1-12. doi:10.1136/bmjopen-2014-005999

Atkinson, R. W., Carey, I. M., Kent, A. J., van Staa, T. P., Anderson, H. R., & Cook, D. G. (2014). Long-term exposure to outdoor air pollution and the incidence of chronic obstructive pulmonary disease in a national English cohort. *Occupational and Environmental Medicine*. doi:10.1136/oemed-2014-102266

Australian Bureau of Statistics (ABS). (2017). 3218.0 Regional Population Growth, Australia 2015-16. Retrieved from http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3218.02015-16?OpenDocument

Australian Institute of Health and Welfare (AIHW). (2010). *Monitoring the impact of air pollution on asthma in Australia*. Canberra: Australian Government. Retrieved from https://www.aihw.gov.au/reports/asthma-other-chronic-respiratory-conditions/monitoring-the-impact-of-air-pollution-on-asthma-i/contents/table-of-contents

Bakand, S., Hayes, A., & Dechsakulthorn, F. (2012). Nanoparticles: a review of particle toxicology following inhalation exposure. *Inhalation Toxicology, 24*(2), 125-135. doi:10.3109/08958378.2010.642021

Barnett, A. (2014). It's safe to say there is no safe level of air pollution. *Australian and New Zealand Journal of Public Health, 38*(5), 407-408. doi:10.1111/1753-6405.12264

Barnett, A., Williams, G., Schwartz, J., Best, T., Neller, A., Petroeschevsky, A., & Simpson, R. (2006). The effects of air pollution on hospitalizations for cardiovascular disease in elderly people in Australian and New Zealand cities. *Environmental Health Perspectives, 114*(7), 1018-1023. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/16835053

Beelen, R., Hoek, G., Houthuijs, D., van den Brandt, P. A., Goldbohm, R. A., Fischer, P., . . . Brunekreef, B. (2009). The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. *Occup Environ Med, 66*. doi:10.1136/oem.2008.042358

Berman, J., Fann, N., Hollingsworth, J., Pinkerton, K., Ronm, W., Szema, A., . . . Curriero, F. (2012). Health benefits from large-scale ozone reduction in the United States. *Environmental Health Perspectives, 120*(10), 1404-1410. doi: 10.1289/ehp.1104851

Bertolatti, D., & Rumchev, K. (2009). Size distribution and elemental composition of ultrafine and nanoparticles. In C. A. Brebbia (Ed.), *Environmental Health Risk V* (Vol. 14, pp. 47-54). doi: 10.2495/EHR090051

Beverland, I. J., Carder, M., & Cohen, G. (2014). Associations between short/mediumterm variations in blacksmoke air pollution and mortality in the Glasgow conurbation, U.K. *Environment International., 62*, 126-132. doi:10.1016/j.envint.2013.01.001.

Blake, D., van Etten, E., & Horwitz, P. (2011). *Smoke and fire in the urban context: Remnant vegetation and wetlands in Perth.* Paper presented at the ECU/FESA Bushfire Smoke Issues Research Symposium, ECU Perth, WA.

Brook, R. (2008). Cardiovascular effects of air pollution. *Clinical Science, 115*, 175-187. doi: 10.1042/CS20070444

Brook, R., Rajagopalan, S., Pope, C., Brook, J., Bhatnagar, A., Diez-Roux, A., . . . Kaufman, J. (2010). Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation, 121*(21), 2331-78. doi:10.1161/CIR.0b013e3181dbece1

Broome, R., Cope, M., Goldsworthy, B., Goldsworthy, L., Emmerson, K., Jegasothy, E. & Morgan, G. (2016). The mortality effect of ship-related fine particulate matter in the Sydney greater metropolitan region of NSW, Australia. *Environment International, 87*, 85-93. doi: 10.1016/j.envint.2015.11.012

Broome, R., Fann,. N., Cristina, T., Fulcher. C., Duc, H. & Morgan, G. (2015) The health benefits of reducing air pollution in Sydney, Australia. *Environmental Research 143*, 19-25. doi: htt://dx.doi/10.1016/j.envres2015.09.007

Caiazzo, F., Ashok, A., Waitz, I., Yim, S., & Barrett, S. (2013). Air pollution and early deaths in the United States. Part I: Quantifying the impact of major sectors in 2005. *Atmospheric Environment, 79*, 98-208. doi: https://doi.org/10.1016/j.atmosenv.2013.05.081

California Environmental Protection Air Resources Board. (2009). Review of the California ambient air quality standard for nitrogen dioxide. Retrieved from https://www.arb.ca.gov/research/aaqs/no2-rs/no2-doc.htm

Castranova, V. (2011). Overview of Current Toxicological Knowledge of Engineered Nanoparticles. *Journal of Occupational and Environmental Medicine, 53*(6 Supplement), S14-S17. doi: 10.1097/JOM.0b013e31821b1e5a.

Cena, L., Chisholm, W., Keane, M., Cumpston, A., & Chen, B. (2014). Size distribution and estimated respiratory deposition of toal chromium, hexavalent chromium, manganese and nickel in gas metal arc welding fume aerosols. *Aerosol Science and Technology, 48*(12), 1254-1263. doi:10.1080/02786826.2014.980883

Centres for Disease Control (CDC). (2010). International Statistical Classification of Diseases and Related Health Problems Revision 10 (ICD-10). Retrieved from https://www.cdc.gov/nchs/icd/icd10cm.htm

Chen, L., Knutsen, S., Shavlik, D., Beeson, W., Petersen, F., Ghamsary, M., & Abbey, D. (2005). The association between fatal coronary heart disease and ambient particulate air pollution: Are females at greater risk? . *Environmental Health Perspectives, 113*, 1723-1729. Retrieved from

http://ehp03.niehs.nih.gov/article/fetchArticle.action?articleURI=info:doi/10.1289/ehp.8190

Chen, L. & Lippman, M. Effects of metals within ambient air particulate matter (PM) on human health. (2009). Inhalation Toxicology 21(1), 1-31. doi: 10.1080/08958370802105405

Chen, S.-Y., Lin, Y.-L., Chang, W.-T., Lee, C.-T., & Chan, C.-C. (2014). Increasing emergency room visits for stroke by elevated levels of fine particulate constituents. *Science of The Total Environment, 473*(Supplement C), 446-450. doi:https://, doi.org/10.1016/j.scitotenv.2013.12.035

Cheng, Y.-H., Huang, C.-H., Huang, H.-L., & Tsai, C.-J. (2010). Concentrations of ultrafine particles at a highway toll collection booth and exposure implications for toll collectors. *Science of The Total Environment, 409*(2), 364-369. doi: 10.1016/j.scitotenv.2010.10.023

Chiew, A. & Buckley, N. 2014. Carbon monoxide poisonong in the 21st century. *Critical Care 18* (221). doi: https://doi.org/10.1186/cc13846

Chin, M. (2015). Basic mechanisms for adverse cardiovascular events associated with air pollution. *Heart, 101*(4), 253-256. doi: 10.1136/heartjnl-2014-306379

Cohen, A., Brauer, M., Burnett, R., Anderson, H., Frostad, J., Estep, K., . . . Forouzanfar, M. (2017). Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. *The Lancet, 389*(10082), 1907-1918. doi:10.1016/S0140-6736(17)30505-6

Committee on the Medical Effects of Air Pollutants (COMEAP). (2009). Long-term exposure to air pollution: Effects on mortality. Retrieved from http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@ab/documents/digitalasset/dh_108152.pdf

Correia, A., Pope, C., Dockery, D., Wang, Y., Ezzati, M., & Dominici, F. (2013). Effect of air pollution control on life expectancy in the United States: An analysis of 545 U.S. counties for the period from 2000 to 2007 *Epidemiology, 24*(1), 23-31. doi: 10.1097/EDE.0b013e3182770237

Crouse, D., Peters, P., van Donkelaar, A., Goldberg, M., Villeneuve, P., Brion, O., . . . Burnett, R. (2012). Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. *Environmental Health Perspectives, 120*. doi:10.1289/ehp.1104049

Debia, M., Beaudry, C., Weichenthal, S., Tardif, R., & Dufresne, A. (2013). Characterization and control of occupational exposure to nanoparticles and ultrafine particles. *Chemical Substances and Biological Agents, Report R-777*. Retrieved from http://www.irsst.qc.ca/media/documents/pubirsst/r-777.pdf

Dennekamp, M., Straney, L., Erbas, B., Abramson, M., Keywood, M., Smith, K., . . . Tonkin, A. (2015). Forest fire smoke exposures and out-of-hospital cardiac arrests in Melbourne, Australia: A case-crossover study. *Environmental Health Perspectives, 123*(10), 959-964. doi: 10.1289/ehp.1408436

Department of Environment (DE) (WA). (2004). 2003 Western Australian Air Monitoring Report. (Technical Series 121), 11. Government of Western Australia, Perth.

Department of Environment and Conservation (DEC) (NSW). (2003). Internal Working paper no. 4: Ambient concentrations of heavy metals in NSW. Retrieved from http://www.environment.nsw.gov.au/resources/air/heavymetals.pdf

Department of Environment and Conservation (DEC) (WA). (2011). *2010 Western Australia Air Monitoring Report*. Government of Western Australia. Retrieved from http://www.nepc.gov.au/system/files/resources/7c4e85af-5d11-9074-d171-0184e06fc243/files/wa-aaq-nepm-air-monitoring-report-2011.pdf

Department of Environment and Energy (DEE). (2015). National Clean Air Agreement; Towards a clean air future for all Australians. Retrieved from https://www.environment.gov.au/system/files/resources/188756ab-ed94-4a3c-9552-62763ca86a7f/files/national-clean-air-agreement.pdf

Department of Environment and Energy (DEE). (2017a). Ambient air quality standards. Rtrieved from http://www.environment.gov.au/protection/air-quality/air-quality-standards

Department of Environment and Energy (DEE). (2017b). Lead. Retrieved from http://www.environment.gov.au/protection/chemicals-management/lead

Department of Environmental Protection (DEP) (WA) (2000). Perth Air Qualty Management Plan, Government of Western Australia, Perth.

Department of Environment Regulation (WA) (2017) Perth Air Quality Coordinating Committee (DER AQCC) Report Card 2015-2016. Retrieved from https://www.der.wa.gov.au/images/documents/about/committees/AQ-reportcard15-16.pdf

Department of Environment Regulation Air Quality Coordinating Committee (DER AQCC) (WA). (2014). Perth Air Quality Coordinating Committee Report Card 2012-2013. Retrieved from https://www.der.wa.gov.au/images/documents/about/committees/2012-13_WA_AQMP_Report_Card.pdfdo

Department of Planning (DP) (WA). (2015). Metropolitan Region Scheme. Retrieved from https://www.planning.wa.gov.au/1222.aspx

Department of Sustainability, Environment, Water, Population and Communities (DSEWPC). (2010a). National Pollution Inventory (NPI), Particulate Matter (PM_{10} and $PM_{2.5}$) Retrieved from http://www.npi.gov.au/substances/particulate-matter/index.html

Department of Sustainability, Environment, Water, Population and Communities (DSEWPC). (2010b). *National Pollution Inventory (NPI), Particulate Matter (PM_{10} and $PM_{2.5}$) & NO_x (Oxides of Nitrogen)*. Retrieved from http://www.npi.gov.au/substances/particulate-matter/index.html

Department of Sustainability, Environment, Water, Population and Communities (DSEWPC). (2011). *State of the air in Australia 1999-2008*. Canberra, ACT:

Australian Government. Retrieved from https://www.environment.gov.au/system/files/resources/655f7df8-1454-461b-a061-f84d37225cb4/files/state-air.pdf

Desqueyroux, H., Pujet, J.-C., Prosper, M., Squinazi, F., & Momas, I. (2002). Short-term effects of low-level air pollution on respiratory health of adults suffering from moderate to severe asthma. *Environmental Research, 89*(1), 29-37. doi:http://dx.doi.org/10.1006/enrs.2002.4357

DeVries, R., Kriebel, D., & Sama, S. (2016). Low level air pollution and exacerbation of existing COPD: a case crossover analysis. *Environmental Health, 15*(1), 98. doi:10.1186/s12940-016-0179-z

Diem, J. (2013) Explanations for the spring peak in ground ozone-level ozone in ht Southwester United staes. Physical Geography 25(2). doi: https://doi.org/10.2747/0272-3646.25.2.105

Dockery , D., Pope , C., Xu , X., Spengler , J., Ware , J., Fay , M., . . . Speizer , F. (1993). An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine, 329*(24), 1753-1759. doi:10.1056/nejm199312093292401

Donaldson, K. (2006). Resolving the nanoparticles paradox. *Nanomedicine, 1*(2), 229-234. doi:10.2217/17435889.1.2.229

Duffin, R., Clouter, A., Brown, D., Tran, C., MacNee, W., Stone, V., & Donaldson, K. (2002). The importance of surface area and specific reactivity in the acute pulmonary inflammatory response to particles. *Annals of Occupational Hygiene, 46*(S1), 242-245. doi: http://doi.org/10.1093/annhyg/46.suppl_1.242

Enamorado-Baez, S., Gomez-Guzman, J., Chamizo, E., & Abril, J. (2015). Levels of 25 trace elements in high-volume air filter samples from Seville (2001-2002): Sources, enrichment factors and temporal variations. *Atmospheric Research, 155*, 118-129. doi:10.1016/j.atmosres.2014.12.005

Environmental Protection Agency (EPA) Victoria. (2001). *Ambient air pollution and daily hospital admissions in Melbourne 1994-1997*. Melbourne: Environmental Protection Agency (EPA) Victoria. Retrieved from http://www.epa.vic.gov.au/~/media/Publications/789.pdf

Environmental Protection Authority (EPA) Review Steering Committee. (2007). *Perth air quality management plan (AQMP) five-year review*. Retrieved from https://www.der.wa.gov.au/images/documents/about/committees/Perth_Air_Quality_Management_Plan.pdf

European Commission. (2003) Air Quality Standards. Retrieved from http://ec.europa.eu/environment/air/quality/standards.htm

Erbas, B., Kelly, A., Physick, B., Code, C., & Edwards, M. (2005). Air-pollution and childhood asthma emergency hospital admissions: Estimating intra-city regional variations. *International Journal of Environmental Health Research, 15*(1), 11-20. doi:10.1080/09603120400018717

Evelyn, A., Mannick, S., & Sermon, P. A. (2002). Unusual Carbon-Based Nanofibers and Chains among Diesel-Emitted Particles. *Nano Letters, 3*(1), 63-64. doi:10.1021/nl025803u

Fang, G.-C., Chang, C.-Y., Tsai, J.-H., & Lin, C.-C. (2014). The size distribution of ambient air metallic pollutants by using a multi-stage MOUDI sampler. *Aerosol and Air Quality Research, 14*, 970-980. doi:10.4209/aaqr.2013.05.0139

Fann, N., Lamson, A., Anenberg, S., Wesson, K., Risley, D., & Hubbell, B. (2012). Estimating the national public health burden associated with exposure to ambient $PM_{2.5}$ and ozone. *Risk Analysis, 32*(1), 81-95. doi:10.1111/j.1539-6924.2011.01630.x

Faustini, A., Rapp, R., & Forastiere, F. (2014). Nitrogen dioxide and mortality: review and meta-analysis of long-term studies. *European Respiratory Journal, 44*(3), 744-753. doi:10.1183/09031936.0011471

Franck, U., Leitte, A. M., & Suppan, P. (2014). Multiple exposures to airborne pollutants and hospital admissions due to diseases of the circulatory system in Santiago de Chile. *Science of The Total Environment, 468*(Supplement C), 746-756. doi:https://doi.org/10.1016/j.scitotenv.2013.08.088

Franck, U., Leitte, A. M., & Suppan, P. (2015). Multifactorial airborne exposures and respiratory hospital admissions — The example of Santiago de Chile. *Science of The Total Environment, 502*(Supplement C), 114-121. doi:https://doi.org/10.1016/j.scitotenv.2014.08.093

Frangos, J., Di Marco, P., Mikonnen, H., Mikonnen, A., Barker, A., Papadopoulos, C., & McKiernan, S. (2013). Exposure assessment and risk characterisation to inform recommendations for updating ambient air quality standards for $PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2 (127643066-001-R-Rev0). Retrieved from https://www.researchgate.net/publication/299968729_Exposure_Assessment_and_Risk_Characterisation_to_Inform_Recommendations_for_Updating_Ambient_Air_Quality_Standaards_for_PM25_PM10_O3_NO2_SO2

Fuks, K. B., Weinmayr, G., Basagaña, X., Gruzieva, O., Hampel, R., Oftedal, B., . . . Hoffmann, B. (2017). Long-term exposure to ambient air pollution and traffic noise and incident hypertension in seven cohorts of the European study of cohorts for air pollution effects (ESCAPE). *European Heart Journal, 38*(13), 983-990. doi:10.1093/eurheartj/ehw413

Gaffin, J. M., Hauptman, M., Petty, C. R., Sheehan, W. J., Lai, P. S., Wolfson, J. M., . . . Phipatanakul, W. (2017). Nitrogen dioxide exposure in school classrooms of inner-city children with asthma. *Journal of Allergy and Clinical Immunology*. doi:https://doi.org/10.1016/j.jaci.2017.08.028

Gauderman, W. J., Urman, R., Avol, E., Berhane, K., McConnell, R., Rappaport, E., . . . Gilliland, F. (2015). Association of improved air quality with lung development in children. *New England Journal of Medicine, 372*(10), 905-913. doi:10.1056/NEJMoa1414123

Geiger, A. & Cooper, J. (2010). *Overview of airborne metals regulations, exposure limits, health effects and contemporary research (Appendix C)*.: Retrieved from https://www3.epa.gov/ttnemc01/prelim/otm31appC.pdf

Giles, L. V., Barn, P., Künzli, N., Romieu, I., Mittleman, M. A., van Eeden, S., . . . Brauer, M. (2011). From good intentions to proven interventions: Effectiveness of actions to reduce the health impacts of air pollution. *Environmental Health Perspectives, 119*(1), 29-36. doi:10.1289/ehp.1002246

Giorgini, P., Di Giosia, P., Grassi, D., Rubenfire, M., Brook, R., & Ferri, C. (2016). Air pollution exposure and blood pressure: An updated review of the literature. *Current Pharmaceutical Design, 22*(1), 28-51. doi:10.2174/1381612822666151109111712

Goldstone, M. E. (2015). Review of evidence on health aspects of air pollution – REVIHAAP Project *Air Quality and Climate Change, 49*(2), 35-41. Retrieved from <http://search.informit.com.au.dbgw.lis.curtin.edu.au/documentSummary;dn=351123840805252;res=IELENG> ISSN: 1836-5884.

Gonzales-Flecha, B. (2004). Oxidant mechanisms in response to ambient air particles. *Molecular Aspects of Medicine, 25*, 169-182. doi:10.1016/j.mam.2004.02.017

Guarnieri, M., & Balmes, J. R. (2014). Outdoor air pollution and asthma. *The Lancet, 383*(9928), 1581-1592. doi:https://doi.org/10.1016/S0140-6736(14)60617-6

Gugamsetty, B., Wei, H., Liu, C.-N., Awasthi, A., Hsu, S.-C., Tsai, C.-J., . . . Chen, C.-F. (2012). Source characterization and apportionment of PM_{10}, $PM_{2.5}$ and $PM_{0.1}$ by using positive matrix fatorization. *Aerosol and Air Quality Research, 12*, 476-491. doi: 10.4209/aaqr.2012.04.0084

Hansen, A., Peng, B., & Nitschke, M. (2009). Air pollution and cardiorespiratory health in Australia: The impact of climate change. *Environmental Health Perspectives, 9*(1 & 2), 17-37. Retrieved from <http://search.informit.com.au/documentSummary;dn=112270319634810;res=IELHEA> ISSN: 1444-5212

Health Effects Institute (HEI). (2013). *Understanding the Health Effects of Ambient Ultrafine Particles. Review Panel on Ultrafine Particles*. Retrieved from https://www.healtheffects.org/publication/understanding-health-effects-ambient-ultrafine-particles

Hinwood, A., de Klerk, N., Rodriguez, C., Jacoby, P., Runnion, T., Rye, P., . . . Spickett, J. (2006). The relationship between changes in daily air pollution and hospitalizations in Perth, Australia 1992 - 1998: A case-crossover study. *International Journal of Environmental Health Research 16*(1), 27-46. Retrieved from http://dx.doi.org/10.1080/09603120500397680

Hinwood, A., de Klerk, N., Rodriguez, C., Runnion, T., Jacoby, P., Landau, L., . . . Spickett, J. (2004). Changes in daily air pollution and mortality in Perth: A case crossover study. *Environmental Health, 4*(4), 13-23. Retrieved from http://search.informit.com.au/documentSummary;dn=203136059870062;res=IELHSS

Hoek, G., Krishnan, R. M., Beelen, R., Peters, A., Ostro, B., Brunekreef, B., & Kaufman, J. D. (2013). Long-term air pollution exposure and cardio-respiratory mortality: a review. *Environmental Health, 12*(1), 43. doi:10.1186/1476-069x-12-43

Holbrook, R. D., Kline, C. N., & Filliben, J. J. (2010). Impact of Source Water Quality on Multiwall Carbon Nanotube Coagulation. *Environmental Science & Technology, 44*(4), 1386-1391. doi:10.1021/es902946j

Institute for Occupational Safety Germany (DGUV). (2017). Ultrafine aerosols and nanoparticles at the workplace. Retrieved from http://www.dguv.de/ifa/fachinfos/nanopartikel-am-arbeitsplatz/index-2.jsp

International Agency for Research on Cancer (IARC). (2013). Outdoor air pollution a leading environmental cause of cancer deaths [Press release]. Retrieved from https://www.iarc.fr/en/media-centre/iarcnews/pdf/pr221_E.pdf

International Standards Organization/ Technical Specification (ISO/TS). (2015). 80004-2:2015 Nanotechnologies — Vocabulary — Part 2: Nano-objects. Retrieved from https://www.iso.org/standard/54440.html

Jalaludin, B., & Cowie, C. (2012). *Health Risk Assessment – Preliminary Work to Identify Concentration-Response Functions for Selected Ambient Air Pollutants*. Retrieved from Respiratory and Environmental Epidemiology Woolcock Institute of Medical Research: http://www.nepc.gov.au/system/files/pages/18ae5913-2e17-4746-a5d6-ffa972cf4fdb/files/health-report.pdf

Jalaludin, B., Morgan, G., Lincoln, D., Sheppeard, V., Simpson, R., & Corbett, S. (2006). Associations between ambient air pollution and daily emergency department attendances for cardiovascular disease in the elderly (65+ years), Sydney, Australia. *Journal of Exposure Science & Environmental Epidemiology, 16*(3), 225-237. doi:10.1038/sj.jea.7500451

Jansse, N., Gerlofs-Nijland, M., Lanki, T., Salonen, R., Cassee, F., Hoek, G., Fischer, P., Brunekreef, B and Krzyzanowski, M. (2012). *Health effects of black carbon.* World Health Organization (WHO). Regional Office for Europe.Copenhagen. Retrieved from http://www.euro.who.int/__data/assets/pdf_file/0004/162535/e96541.pdf

Jerrett, M., Burnett, R., Ma, R., Pope, C., Krewski, D., Newbold, K., . . . Thun, M. (2005). Spatial analysis of air pollution and mortality in Los Angeles. *Epidemiology, 16*, 727-736. doi:10.1097/01.ede.0000181630.15826.7d

Jerrett, M., Burnett, R.T., Pope, C.A., Ito, K., Thurston, G., Krewski, D., et al., (2009). Long-term ozone exposure and mortality. N. Engl. J. Med. 360. 1085–95 doi: 10.1056/NEJMoa0803894

Johnston, F. (2009). Bushfires and human health in a changing environment. *Australian Family Physician, 38*(9) 720-724. Retrieved from https://search.informit.com.au/documentSummary;dn=254067230909712;res=IELHEA

Johnston, F. (2011). *Bushfire smoke and the wildland urban interface: Issues and challenges*. Paper presented at the ECU/FESA Bushfire Smoke Issues Research Symposium, ECU Perth, WA.

Johnston, F., Bailie, R., Pilotto, L., & Hanigan, I. (2007). Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin, Australia. *BioMed Central Public Health, 7*, 240. doi: https://doi.org/10.1186/1471-2458-7-240

Johnston, F., Hanigan, I., Henderson, S., Morgan, G., & Bowman, D. (2011). Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994–2007. *Environmental Research, 111*, 811-816. doi: 10.1016/j.envres.2011.05.007

Johnston, F., Kavanagh, A., Bowman, D., & Scott, R. (2002). Exposure to bushfire smoke and asthma: an ecological study. *MJA, 176*, 535-538. Retrieved from https://www.mja.com.au/journal/2002/176/11/exposure-bushfire-smoke-and-asthma-ecological-study

Kah, M., & Hofmann, T. (2014). Nanopesticide research: Current trends and future priorities. *Environment International, 63*(0), 224-235. Retrieved from http://www.sciencedirect.com/science/article/pii/S0160412013002754. doi:10.1016/j.envint.2013.11.015

Kampa, M., & Castanas, E. (2008). Human health effects of air pollution. *Environmental Pollution, 151*(2), 362-367. doi:https://doi.org/10.1016/j.envpol.2007.06.012

Kelly, F. & Fussell, J. (2015). Air pollution and public health: Emerging hazards and improved understanding of risk. Environmental Geochemistry and Health, 37, 631-649. doi: 10.1007/s10653-015-9720-1

Keywood, M., Hibberd, M., & Emmerson, K. (2017). *Australia state of the environment 2016: atmosphere, Independent report to the Australian Government Minister for the Environment and Energy* Australian Government Department of the Environment and Energy Canberra. Retrieved from https://soe.environment.gov.au/theme/atmosphere

Keywood, M., Kanakidou, M., Stohl, A., Dentener, F., Grassi, G., Meyer, C., . . . Burrows, J. (2013). Fire in the air: biomass burning impacts in a changing climate. *Critical Reviews in Environmental Science and Technology, 43*(1), 40-83. doi: http://dx.doi.org/10.1080/10643389.2011.604248

Kim, C. S., Alexis, N. E., Rappold, A. G., Kehrl, H., Hazucha, M. J., Lay, J. C., . . . Diaz-Sanchez, D. (2011). Lung function and inflammatory responses in healthy young adults exposed to 0.06 ppm ozone for 6.6 Hours. *American Journal of Respiratory and Critical Care Medicine, 183*(9), 1215-1221. doi:10.1164/rccm.201011-1813OC

Kim, K.-H., Kabir, E., & Kabir, S. (2015). A review on the human health impact of airborne particulate matter. *Environment International, 74*(Supplement C), 136-143. doi:https://doi.org/10.1016/j.envint.2014.10.005

Kollanus, V., Tiittanen, P., Niemi, J. V., & Lanki, T. (2016). Effects of long-range transported air pollution from vegetation fires on daily mortality and hospital admissions in the Helsinki metropolitan area, Finland. *Environmental Research, 151*(Supplement C), 351-358. doi:https://doi.org/10.1016/j.envres.2016.08.003

Kumar, P., Morawska, L., Birmili, W., Paasonen, P., Hu, M., Kulmala, M., . . . Britter, R. (2014). Ultrafine particles in cities. *Environment International, 66*(0), 1-10. doi: https://doi.org/10.1016/j.envint.2014.01.013

Künzli, N., Jerrett, M., Mack, W., Beckerman, B., LaBree, L., Gilliland, F., . . . Hodis, H. (2005). Ambient air pollution and atherosclerosis in Los Angeles. *Environmental Health Perspectives, 113*, 201-206. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1277865/

Künzli, N., Kaiser, R., Medina, S., Studnicka, M., Chanel, O., Filliger, P., . . . Schnider, J. (2000). Public-health impact of outdoor and traffic-related air pollution: a European assessment. *The Lancet, 356*(9232), 795-801. doi:10.1016/S0140-6736(00)02653-2

Laden, F., Schwartz, J., Speizer, F. E., & Dockery, D. W. (2006). Reduction in fine particulate air pollution and mortality: Extended follow-up of the Harvard six cities study. *American Journal of Respiratory and Critical Care Medicine, 173*(6), 667-672. doi: 10.1164/rccm.200503-443OC

Laidlaw, M., & Taylor, M. (2011). Potential for childhood lead poisoning in the inner cities of Australia due to exposure to lead in soil dust. *Environmental Pollution, 159*(1), 1-9. doi: 10.1016/j.envpol.2010.08.020

Larabee, K., & Phipatanakul, W. (2012). The respiratory health effects of nitrogen dioxide in children with asthma. *Pediatrics, 130*(Supplement 1), S10-S10. doi:10.1542/peds.2012-2183M

Laumbach, R., & Kipen, H. (2012). Respiratory health effects of air pollution: Update on biomass smoke and traffic pollution. *Clinical Reviews in Allergy and Immunology, 129*(1), 3-12. doi: 10.1016/j.jaci.2011.11.021

Lee, B.-J., Kim, B., & Lee, K. (2014). Air pollution exposure and cardiovascular disease. *Toxicological Research, 30*(2), 71-75. doi:10.5487/TR.2014.30.2.071

Leiva G, M. A., Santibañez, D. A., Ibarra E, S., Matus C, P., & Seguel, R. (2013). A five-year study of particulate matter (PM2.5) and cerebrovascular diseases. *Environmental Pollution, 181*(Supplement C), 1-6. doi:https://doi.org/10.1016/j.envpol.2013.05.057

Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya , K., Adair-Rohani, H., . . . Balmes, J. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet, 380*(9859), 2224-2260. doi:http://dx.doi.org/10.1016/S0140-6736(12)61766-8

Lin, C.-C., Chen, S.-J., & Huang, K.-L. (2005). Characteistics of metals in nano/ultrafine/fine/coarse particles collected beside a heavily trafficked road. *Environmental Science & Technology, 39*(21), 8113-8122**.** doi:10.1021/es048182a

Lin, H., Tao, J., Yadong, D., Liu, T., Qian, Z., Tian, L., . . . Ma, W. (2016). Differentiating the effects of charactersitics of PM pollution on mortlity from ischemic and hemorrhagic strokes. *Interantional Journal of Hygiene and Environmental Health, 219*, 204-211. doi: 10.1016/j.ijheh.2015.11.002

Loomis, D., Grossea, Y., Lauby-Secretana, B., El Ghissassia, F., Bouvarda, V., Benbrahim-Tallaaa, L., . . . Straifa, K. (2013). The carcinogenicity of outdoor air pollution. *The Lancet: Oncology, 14*, 1262-1263. doi: http://dx.doi.org/10.1016/S1470-2045(13)70487-X

Lough, G., Schauer, J., Park, J.-S., Shafer, M., Deminter, J., & Weinstein, J. (2005). Emissions of metals associated with motor vehicle roadways. *Environmental Science & Technology, 39*, 826-836. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/15757346

Luo, K., Li, R., Li, W., Wang, Z., Ma, X., Zhang, R., . . . Xu, Q. (2016). Acute effects of nitrogen dioxide on cardiovascular mortality in Beijing: An exploration of spatial heterogeneity and the district-specific predictors. *6*, 38328. doi:10.1038/srep38328

Malmqvist, E., Jakobsson, K., Tinnerberg, H., Rignell-Hydbom, A., & Rylander, L. (2013). Gestational diabetes and preeclampsia in association with air pollution at levels below current air quality guidelines. *Environmental Health Perspectives (Online), 121*(4), 488. doi:http://dx.doi.org/10.1289/ehp.1205736

Manke, A., Wang, L., & Rojanasakul, Y. (2013). Mechanisms of nanoparticle-induced oxidative stress and toxicity. *Biomed Research International*. doi:http://dx.doi.org/10.1155/2013/942916

Maroziene, L., & Grazuleviciene, R. (2002). Maternal exposure to low-level air pollution and pregnancy outcomes: a population-based study. Environmental Health: A Global Access Science Source, 1(6), 1-7. doi:https://doi.org/10.1186/1476-069X-1-6

Martuzzi, M., Galassi, C., Ostro, B., Forastiere, F., & Bertollini, R. (2002). *Health impact assessment of air pollution in the eight major Italian cities*. Retrieved from World Health Organization (WHO) Europe. Retrieved from http://www.euro.who.int/__data/assets/pdf_file/0013/91111/E75492.pdf

Maynard, A., & Kuempel, E. (2005). Airborne nanostructured particles and occupational health. *Journal of Nanoparticle Research, 7*(6), 587-614. doi:10.1007/s11051-005-6770-9

Melaku, S., Morris, V., Raghavan, D., & Hosten, C. (2008). Seasonal variation of heavy metals in ambient air and precipitation at a single site in Washington, DC. *Environmental Pollution, 155*(1), 88-98. doi:https://doi.org/10.1016/j.envpol.2007.10.038

Meng, X., Zhang, Y., Yang, K.-Q., Yang, Y.-K., & Zhou, X.-L. (2016). Potential Harmful Effects of PM2.5 on Occurrence and progression of acute coronary syndrome: Epidemiology, mechanisms, and prevention measures. *International Journal of Environmental Research and Public Health, 13*(748), 1-16. doi:10.3390/ijerph13080748

Milojevic, A., Wilkinson, P., Armstrong, B., Bhaskaran, K., Smeeth, L., & Hajat, S. (2014). Short-term effects of air pollution on a range of cardiovascular events in England and Wales: case-crossover analysis of the MINAP database, hospital admissions and mortality. *Heart, 100*(14), 1093-1098. doi:10.1136/heartjnl-2013-304963

Morawska, L. (2007). Air quality and its impact on health: Focus on particulate matter. *Environmental Health, 7*(3), 52-57. Retrieved from http://search.informit.com.au/documentSummary;dn=833396312213015;res=IELHEA;type=pdf

Morawska, L. (2010). Adverse health effects to air pollution and guidelines to prevent them. *Air Quality and Climate Change, 44*(1), 16-18. Retrieved from http://search.informit.com.au/documentSummary;dn=308805214276169;res=IELENG

Morawska, L., Wang, H., Ritovski, Z., Jayaratane, R., Johnson, G., Cheung, H., . . . He, C. (2009). Environmental monitoring of airborne nanoparticles [Review]. *The Royal Society of Chemistry*. doi: 10.1039/b912589m

Moreno, T., Kojima, T., Amato, F., Lucarelli, F., de la Rosa, J., Calzolai, G., . . . Gibbons, W. (2013). Daily and hourly chemical impact of springtime transboundary aerosols on Japanese air quality. *Atmospheric Chemistry and Physics, 13*, 1411-1424. doi: https://doi.org/10.5194/acp-13-1411-2013

Morgan, G. (2011). *The health effects of bushfire smoke in Australia*. Paper presented at the ECU/FESA Bushfire Smoke Issues Researech Symposium, ECU Perth, WA.

Morgan, G., Broome, R., & Jalaludin, B. (2013). *Summary for policy makers of the health risk assessment on air pollution in Australia*. Retrieved from https://www.environment.gov.au/system/files/pages/dfe7ed5d-1eaf-4ff2-bfe7-dbb7ebaf21a9/files/summary-policy-makers-hra-air-pollution-australia.pdf

Moshammer, H., Hutter, H.-P., Hauck, H., & Neuberger, M. (2006). Low levels of air pollution induce changes of lung function in a panel of schoolchildren. *European Respiratory Journal, 27*(6), 1138-1143. doi:10.1183/09031936.06.00089605

MSP Corporation. (2017). Aerosol Model 120R, 122R and 125R MOUDI II™ Impactors.Retrieved from https://www.mspcorp.com/resources/msp-pi-120-revc-us-moudi-ii-impactors-120r-122r-125r.pdf

National Environmental Protection Council (NEPC). (2016). National Environmental Protection (Ambient Air Quality) Measure. Canberra.: Department of Environment. Retrieved from http://www.nepc.gov.au/nepms/ambient-air-quality

National Institute of Occupational Health and Safety (NIOSH) Manual of Analytical Methods (NNAM) (2014) *Method 7302 Elements by ICP (Microwave Digestion).* Retrieved from https://www.cdc.gov/niosh/docs/2003-154/pdfs/7302.pdf

National Nanotechnology Initiative (NNI). (2004). What is Nanotechnology? Retrieved from http://www.nano.gov/nanotech-101/what/definition

O'Connor, G. T., Neas, L., Vaughn, B., Kattan, M., Mitchell, H., Crain, E. F., . . . Lippmann, M. (2008). Acute respiratory health effects of air pollution on children with asthma in US inner cities. *Journal of Allergy and Clinical Immunology, 121*(5), 1133-1139.e1131. doi:https://doi.org/10.1016/j.jaci.2008.02.020

Oberdorster, G., Maynard, A., Donaldson, K., Castranova, V., Fitzpatrick, J., Ausman, K., . . . Yang, H. (2005). *Principles for characterizing the potential human health effects from exposure to nanomaterials: elements of a screening strategy* (1743-8977). Retrieved from http://www.particleandfibretoxicology.com/content/2/1/8

Oberdörster, G., Oberdörster, E., & Oberdörster, J. (2005). Nanotoxicology: An emerging discipline evolving from studies of ultrafine particles. *Environmental Health Perspectives, 113*(7), 823-839. doi:10.2307/3436201

Organization for Economic Co-operation and Development (OECD). (2014). *The Cost of Air Pollution*. Paris: OECD Publishing. Retrieved from http://www.oecd.org/env/the-cost-of-air-pollution-9789264210448-en.htm

Pekney, N., Davidson, C., Bein, K., Wexler, A., & Johnston, M. (2006). Identification of sources of atmospheric PM at the Pittsburgh supersite, Part 1: SIngle particle analysis and filter-based positive matrix factorization. *Atmospheric Environment, 40*, S411-S423. doi: 10.1016/j.atmosenv.2005.12.072

Pereira, G., Cook, A., De Vos, A., & Holman, D. (2010). A case-crossover analysis of traffic-related air pollution and emergency department presentations for asthma in Perth, Western Australia. *Medical Journal of Australia, 193*(09), 511-514. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/21034384

PerkinElmer. (2011). The 30-Minute Guide to ICP-MS. Retrieved from http://www.perkinelmer.com/PDFs/Downloads/tch_icpmsthirtyminuteguide.pdf

Petroeschevsky, A., Simpson, R., Thalib, L., & Rutherford, S. (2001). Associations between outdoor air pollution and hospital admissions in Brisbane, Australia. *Archives of Environmental Health, 56*, 37-52. doi:10.1080/00039890109604053

Phung, D., Hien, T, Linh, H., Luong, L., Morawska, L., Chu, C., . . . Thai, P. (2016). Air pollution and risk of respiratory and cardiovascular hospitalizations in the most populous city in Vietnam. *Science of The Total Environment, 557-558*, 322-330. doi: 10.1016/j.scitotenv.2016.03.070

Polichetti, G., Cocco, S., Spinali, A., Trimarco, V., & Nunziata, A. (2009). Effects of particulate matter (PM_{10}, $PM_{2.5}$ and PM_1) on the cardiovascular system. *Toxicology, 261*(1–2), 1-8. doi:http://dx.doi.org/10.1016/j.tox.2009.04.035

Pope, C. (2000). Epidemiology of fine particualte air pollution and human health: Biologic mechanisms and who's at risk? *Environmental Health Perspectives, 108*(S4), 713-723. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1637679/

Pope, C., & Dockery, D. (2006). Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manag Assoc, 56*. doi:10.1080/10473289.2006.10464485

Pope, C., Ezzati, M., & Dockery, D. (2009). Fine-particle air pollution and life-expectancy in the United States. *New England Journal of Medicine, 360*(4), 376-386. doi: 10.1056/NEJMsa0805646

Pope, C., Muhlestein, J., May, H., Renlund, D., Naderson, J., & Horne, B. (2006). Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. *Circulation: Journal of the American Heart Association, 114*, 2443-2448. Retrieved from http://circ.ahajournals.org/cgi/content/full/114/23/2443

Pope, C., Thun, M., Calle, E., Krewski, D., Ito, K., & Thurston, G. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA, 287*. doi:10.1001/jama.287.9.1132

Project on Emerging Nanotechnologies (PEN) Database. (2014). The project on nanotechnologies. Woodrow Wilson International Centre for Scholars. Retrieved from http://www.nanotechproject.org/inventories/medicine/apps/

Qi, L., Zhang, Y., Ma, Y., Chen, M., Ge, X., Ma, Y., . . . Li, S. (2016). Source identification of trace elements in the atmosphere during the second Asian Youth Games in Nanjing, China: Influence of control measures on air quality. *Atmospheric Pollution Research, 7*, 547-556. doi: 10.1016/j.apr.2016.01.003

Qiu, H., Yu, I. T. S., Wang, X., Tian, L., Tse, L. A., & Wong, T. W. (2013). Season and humidity dependence of the effects of air pollution on COPD hospitalizations in Hong Kong. *Atmos Environ, 76*. doi:10.1016/j.atmosenv.2012.07.026

Reisen, F., Meyer, C., & Keywood, M. (2013). Impact of biomass burning sources on seasonal aerosol air quality. *Atmospheric Environment, 67*, 437-447. https://doi.org/10.1016/j.atmosenv.2012.11.004.

Risom, L., Moller, P., & Loft, S. (2005). Oxidative stress-induced DNA damage by particulate air pollution. *Mutation Research, 592*, 119-137. doi:10.1016/j.mrfmmm.2005.06.012.

Rückerl, R., Schneider, A., Breitner, S., Cyrys, J. & Peters, A. 2011. Health effects of particulate air pollution: A review of epidemiological evidence. *Inhalation Toxicology, 23*(10), 555-592. doi: 10.3109/08058378.2011.593587.

Rumchev, K., Ourangui, R., Bertolatti, D., & Spickett, J. (2007). Indoor air quality in old and new schools. In C. A. Brebbia (Ed.), *Environmental Health Risk IV* (Vol. 11, pp. 25-32). doi: 10.2495/EHR070031.

Samoli, E., Aga, E., Touloumi, G., Nisiotis, K., Forsberg, B., Lefranc, A., . . . Katsouyanni, K. (2006). Short-term effects of nitrogen dioxide on mortality: an analysis within the APHEA project. *European Respiratory Journal, 27*(6), 1129-1138. doi:10.1183/09031936.06.00143905.

Sanchez de la Campa, A., de la Rosa, J., Gonzalez-Castanedo, Y., Fernandez-Camacho, R., Alustuey, A., Querol, X., & Pio, C. (2010). High concentrations of heavy metals in PM from ceramic factories of Southern Spain. *Atmospheric Research, 96*, 633-644. https://doi.org/10.1016/j.atmosres.2010.02.011

Sarran, C., Agnew, P., & Davis, L. (2010). *The influence of air quality on COPD hospital admissions*. Met Office, U.K. Retrieved from *gmes-atmosphere.eu/documents/deliverables/o-int/MACC_COPD.pdf*

Sauerzapf, V., Jones, A., & Cross, J. (2009). Environmental factors and hospitalisation for chronic obstructive pulmonary disease in a rural county of England. *Journal of Epidemiology and Community Health, 63*, 324-328. doi: 10.1136/jech.2008.077024

Schelegle, E. S., Morales, C. A., Walby, W. F., Marion, S., & Allen, R. P. (2009). 6.6-Hour Inhalation of Ozone Concentrations from 60 to 87 Parts per Billion in Healthy Humans. *American Journal of Respiratory and Critical Care Medicine, 180*(3), 265-272. doi: 10.1164/rccm.200809-1484OC.

Schwartz, J., Bind, M. A., & Koutrakis, P. (2017). Estimating causal effects of local air pollution on daily deaths: Effect of low levels. *Environmental Health Perspectives, 125*(1), 23-29. doi: 10.1289/EHP232

Schwartz, J., & Neas, L. (2000). Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology, 11*(1), 6-10. Retrieved from http://www.jstor.org/stable/3703646

Seinfeld, J. (1986). *Atmospheric chemistry and physics of air pollution*. California: Wiley-Interscience. doi: 10.1021/es00151a602

Shah, A. S. V., Langrish, J. P., Nair, H., McAllister, D. A., Hunter, A. L., Donaldson, K., . . . Mills, N. L. (2013). Global association of air pollution and heart failure: a systematic review and meta-analysis. *The Lancet, 382*(9897), 1039-1048. doi:10.1016/S0140-6736(13)60898-3

Shi, L., Zanobetti, A., Kloog, I., Coull, B., Koutrakis, P., Melly, S., & Schwartz, J. (2016). Low-concentration PM2.5 and mortality: estimating acute and chronic effects in a population-based study. *Environmental Health Perspectives, 124*, 46-52. doi:http://dx.doi.org/10.1289/ehp.1409111

Silverman, R. A., & Ito, K. (2010). Age-related association of fine particles and ozone with severe acute asthma in New York City. *Journal of Allergy and Clinical Immunology, 125*(2), 367-373.e365. doi:http://dx.doi.org/10.1016/j.jaci.2009.10.061

Simpson, R., Williams, G., Petroeschevsky, A., Best, T., Morgan, G., Denison, L., . . . Neller, A. (2005). The short-term effects of air pollution on daily mortality in four Australian cities. *The Australian and New Zealand Journal of Public Health, 29* (3), 205-212. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/15991767

Spickett, J., Katscherian, D., & Harris, P. (2013). The role of Health Impact Assessment in the setting of air quality standards: An Australian perspective. *Environmental Impact Assessment Review, 43*, 97-103. doi:http://dx.doi.org/10.1016/j.eiar.2013.06.001

Standards Australia (2003). *Methods for sampling and analysis of ambient air - Determination of particulate matter - Deposited matter - Gravimetric method.(*AS/NZS 3580.10.1.2003). Sydney, NSW: SAI Global.

Standards Australia (2003) *Methods for sampling and analysis of ambient air - Determination of suspended particulate matter - PM_{10} high volume sampler with size-selective inlet - Gravimetric method* (AS/NZ 3580.9.6:2003). Sydney, NSW: SAI Global.

Standards Australia (2003). *Methods for sampling and analysis of ambient air - Determination of suspended particulates matter—Total suspended particulate matter (TSP) High Volume sampler gravimetric method* (AS3580.9.3: 2003). Sydney, NSW: SAI Global.

Standards Australia (2007) *Methods for sampling and analysis of ambient air - Guide to siting air monitoring equipment*. (AS 3580.1.1: 2007). Sydney, NSW: SAI Global.

Standards Australia (2005) *Recommended practice for inductively coupled plasma-mass spectrometry (ICP-MS).* (AS 4873.1-2005). Sydney, NSW: SAI Global.

Stanford University Environmental Health and Safety. (2017). Nanomaterials. Retrieved from https://ehs.stanford.edu/topic/hazardous-materials/nanomaterials

Stone, V., Johnston, H., & Clift, M. J. (2007). Air pollution, ultrafine and nanoparticle toxicology: cellular and molecular interactions. *IEEE Trans Nanobioscience, 6*(4), 331-340.

Straif, K., Cohen, A., & Samet, J. (2013). *Air Pollution and Cancer Report no. 161*. Retrieved from International Agency for Research on Cancer (IARC):

Straney, L., Bremner, A., Tonkin, A., Dennekamp, M., Jacobs, I., & Finn, J. (2013). Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth (Western Australia) metropolitan area: 2000–2010. *Resuscitation, 84S*, S59. doi: http://dx.doi.org/10.1016/j.resuscitation.2013.08.151

Straney, L., Finn, J., Dennekamp, M., Bremner, A., Tonkin, A. & Jacobs, I. (2014). Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth) metropolitan region: 2000–2010*. Journal of Epidemiology & Community Health. 68*(1). doi: http://dx.doi.org/10.1136/jech-2013-202955

Strickland, M. J., Darrow, L. A., Klein, M., Flanders, W. D., Sarnat, J. A., Waller, L. A., . . . Tolbert, P. E. (2010). Short-term associations between ambient air pollutants and pediatric asthma emergency department visits. *American Journal of Respiratory and Critical Care Medicine, 182*(3), 307-316. doi: 10.1164/rccm.200908-1201OC

Swami, K., Judd, C., Orsini, J., Yang, K., & Husain, L. (2001). Microwave assisted digestion of atmospheric aerosol samples followed by inductively coupled plasma mass spectrometry determination of trace elements. *Fresenius Journal of Analytical Chemistry, 369*, 63-70. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/11210233

Sykes, D. (2016). *$PM_{2.5}$ and PM_{10} in Scotland: Report for the Scottish Government*. Glasgow: Scottish Government. Retrieved from http://www.scottishairquality.co.uk/assets/documents/technical%20reports/pm2.5-pm10ratio_29Mar2016-FINAL_Version_Approved.pdf

Szewczyńska, M., Pośniak, M. & Dobrzyńska, E. (2013). Study on individual PAHs content in ultrafine particles from solid fractions of diesel and biodiesel exhaust fumes, *Journal of Chemistry, (2013*), doi:10.1155/2013/528471

Taiwo, A., Beddows, D., Shi, Z. & Harrison, R. (2014). Mass and number size distributions of particualte matter components: comparison of an industrial site and an urban background site. *Science of the Total Environment 475*, 29-38. https://doi.org/10.1016/j.scitotenv.2013.12.076

Thurston, G., Burnett, R., Turner, M., Shi, Y., Krewski, D., Lall, R., . . . Pope, C. (2016). Ischemic heart disease mortality and long-term exposure to source-related components of U.S. fine particle air pollution. *Environmental Health Perspectives (Online), 124*(6), 785. doi: 10.1289/ehp.1509777.

Tisch Environmental. (2015).TSP High Volume Air Sampler. Rtrieved from https://tisch-env.com/high-volume-air-sampler/TSP.

U.S Department of the Interior Geological Survey. (2013). What is ICP-MS? ... and more importantly, what can it do? *Crustal Geophysics and Geochemistry Science Centre.* Retrieved from https://crustal.usgs.gov/laboratories/icpms/

United States Environmental Protection Agency (U.S. EPA). (2010). *Particulate matter*. Retrieved from https://www.epa.gov/pm-pollution/particulate-matter-pm-basics

United States Environmental Protection Agency (U.S. EPA). (2011). *Policy assessment for the review of the particulate matter national ambient air quality standards, 452/R-11-003.* Retrieved from https://www3.epa.gov/ttn/naaqs/standards/pm/data/20110419pmpafinal.pdf

United States Environmental Protection Agency (U.S. EPA). (2014). *Report on the Environment, Ozone concentrations*. Retrieved from *https://cfpub.epa.gov/roe/indicator_pdf.cfm?i=8*

United States Environmental Protection Agency (U.S. EPA). (2016). *Black carbon*. Retrieved from https://www3.epa.gov/airquality/blackcarbon/basic.html

United States Environmental Protection Agency (U.S. EPA). (2017a). National ambient air quality standards. Retrieved from https://www.epa.gov/criteria-air-pollutants/naaqs-table

United States Environmental Protection Agency (U.S. EPA). (2017b). Nonattainable areas for citeria pollutants. Retrieved from https://www.epa.gov/green-book

Valavanidis, A., Fiotakis, K., Vlahogianni, T., Bakeas, E., Triantafillaki, S., Paraskevopoulou, V., & Dassenakis, M. (2006). Characterization of atmospheric particulates, particle-bound transition metals and polycyclic aromatic hydrocarbons of urban air in the centre of Athens (Greece). *Chemosphere, 65*(5), 760-768. doi:10.1016/j.chemosphere.2006.03.052

Vinzents, P. S., Møller, P., Sørensen, M., Knudsen, L. E., Hertel, O., Jensen, F. P., . . . Loft, S. (2005). Personal exposure to ultrafine particles and oxidative DNA damage. *Environmental Health Perspectives, 113*(11), 1485-1490. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/16263500

Wang, C.-F., Chang, C.-Y., Tsai, S.-F., & Chiang, H.-L. (2005). Characteristics of road dust from different sampling sites in northern Taiwan. *Journal of the Air & Waste Management Association 55*, 1236-1244. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/16187593

Williams, G. (2012). *National Environmental Protection Council (NEPC) Australian child health and air pollution Study (ACHAPS)*. Retrieved from University of Queensland: http://www.nepc.gov.au/resource/australian-child-health-and-air-pollution-study-achaps-final-report

Woodruff, T., Parker, J., & Schoendorf, K. (2006). Fine particulate matter ($PM_{2.5}$) air pollution and selected causes of post neonatal infant mortality in California. *Environmental Health Perspectives, 114*(5), 786-790. Retrieved from http://ehp.niehs.nih.gov/members/2006/8484/8484.pdf

World Health Organisation (WHO) Regional Office for Europe. (2016). *Health risk assessment of air pollution: General principles*. Retrieved from http://www.euro.who.int/__data/assets/pdf_file/0006/298482/Health-risk-assessment-air-pollution-General-principles-en.pdf?ua=1

World Health Organization (WHO). (2006). WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. Summary of risk assessment. Retrieved from: http://apps.who.int/iris/bitstream/10665/69477/1/WHO_SDE_PHE_OEH_06.02_eng.pdf

World Health Organization (WHO). (2013). *Review of evidence on health aspects of air pollution-REVIHAAP project: technical report*. Retrieved from http://www.euro.who.int/__data/assets/pdf_file/0020/182432/e96762-final.pdf

World Health Organization (WHO). (2017a). *Ambient air pollution: A global assessment of exposure and burden of disease* (9789241511353). Retrieved from http://who.int/phe/publications/air-pollution-global-assessment/en/

World Health Organization (WHO). (2017b). *Evolution of WHO air quality guidelines: past, present and future*. Retrieved from http://www.euro.who.int/__data/assets/pdf_file/0019/331660/Evolution-air-quality.pdf

World Health Organization (WHO) Regional Office for Europe. (2000). *Air Quality Guidelines (2nd edn:) Chapter 7.1 Nitrogen Dioxide*. Retrieved from http://www.euro.who.int/__data/assets/pdf_file/0017/123083/AQG2ndEd_7_1nitrogendioxide.pdf

World Health Organization (WHO). (1958) *Air pollution; Fifth Report of the Expert Committee on Environmental Sanitation*: Retrieved from http://apps.who.int/iris/handle/10665/40416

World Health Organization (WHO). (2014). Ambient (outdoor) air pollution in cities database. Retrieved from http://www.who.int/phe/health_topics/outdoorair/databases/cities-2014/en/

Zhao, Q., Zhang, Y., Zhang, W., Li, S., Chen, G., Wu, Y., . . . Guo, Y. (2017). Ambient temperature and emergency department visits: Time-series analysis in 12 Chinese cities *Environmental Pollution, 224*, 310-316. doi:10.1016/j.envpol.2017.02.010

Appendices

A: Associations between air pollutants and a 10% increase in hospital admissions

B: Associations between air pollutants and a 20% increase in hospital admissions

Appendix A: Associations between air pollutants and a 10% increase in hospital admissions

Table A1 (i): Associations between air pollutants and a 10% increase in 'Cardiovascular' hospital admissions based on, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Cardiovascular Admissions (standardised) exceeding Gastro by ≥ 10%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	885 (72.8%)	331 (27.2%)	1	1
	2 (>0.042- ≤0.73)	839 (69.0%)	377 (31.0%)	1.20 (1.01-1.43) p=0.040	1.11 (0.93-1.33) p=0.254
	3 (>0.73 – ≤1.92)	698 (57.2%)	522 (42.8%)	2.00 (1.69-2.37) p<0.0005	1.45 (1.19-1.76) p<0.0005
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	786 (64.6%)	430 (35.4%)	1	1
	2 (>1.67 - ≤2.10)	833 (68.2%)	388 (31.8%)	0.85 (0.72-1.01) p=0.061	1.06 (0.88-1.27) p=0.550
	3 (>2.10 - ≤3.74)	803 (66.1%)	412 (33.9%)	0.94 (0.79-1.11) p=0.452	1.00 (0.83-1.20) p=0.977
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	924 (74.9%)	309 (25.1%)	1	1
	2 (>0.11 - ≤0.21)	797 (66.8%)	397 (33.2%)	1.49 (1.25-1.78) p<0.0005	1.41 (1.18-1.69) p<0.0005
	3 (>0.21 - ≤3.04)	701 (57.2%)	524 (42.8%)	2.24 (1.88-2.65) p<0.0005	1.63 (1.34-1.98) p<0.0005

Particulate Matter $(PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	769 (63.1%)	449 (36.9%)	1	1
	2 (>13.55 - ≤18.50)	775 (63.6%)	443 (36.4%)	0.98 (0.83-1.12) p=0.801	1.11 (0.94-1.32) p=0.202
	3 (<18.50 - ≤68.3)	876 (72.2%)	338 (27.8%)	0.66 (0.56-0.78) p<0.0005	1.01 (0.84-1.22) p=0.898
Particulate Matter $(PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	812 (66.8%)	404 (33.2%)	1	1
	2 (>6.45 - ≤8.51)	800 (65.9%)	414 (34.1%)	1.04 (0.88-1.23) p=0.647	1.11 (0.94-1.32) p=0.223
	3 (>8.51 - ≤40.78)	805 (66.3%)	410 (33.7%)	1.02 (0.87-1.21) p=0.785	1.11 (0.93-1.32) p=0.237

Table A1 (ii): Associations between air pollutants and a 10% increase in 'Total Respiratory' hospital admissions based on low, medium and high levels of pollutants. Significant values (p<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range Average)	**Tertile Range**	Total Respiratory Admissions (standardised) exceeding Gastro by ≥ 10%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	844 (69.4%)	372 (30.6%)	1	1
	2 (>0.042- ≤0.73)	785 (64.6%)	431 (35.4%)	1.25 (1.05-1.47) p=0.011	1.09 (0.89-1.33) p=0.431
	3 (>0.73 – ≤1.92)	543 (44.5%)	677 (55.5%)	2.40 (1.40-3.34) p<0.0005	1.50 (1.21-1.88) p<0.0005
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	695 (57.2%)	521 (42.8%)	1	1
	2 (>1.67 - ≤2.10)	828 (67.8%)	393 (32.2%)	0.63 (0.54-0.75) p<0.0005	0.89 (0.71-1.10) p=0.272
	3 (>2.10 - ≤3.74)	649 (53.4%)	566 (46.6%)	1.16 (0.99-1.40) p=0.064	1.03 (0.83-1.29) p=0.103
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	893 (72.4%)	340 (27.6%)	1	1
	2 (>0.11 - ≤0.21)	771 (64.6%)	423 (35.4%)	1.44 (1.21-1.71) p<0.0005	1.39 (1.13-1.71) p=0.002
	3 (>0.21 - ≤3.04)	508 (41.5%)	717 (58.5%)	3.71 (3.13-4.40) p<0.0005	2.31 (1.85-2.88) p<0.0005

Particulate Matter (PM)$_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	550 (45.2%)	668 (54.8%)	1	1
	2 (>13.55 - ≤18.50)	677 (55.6%)	541 (44.4%)	0.66 (0.56-0.77) p<0.0005	0.92 (0.76-1.10) p=0.363
	3 <18.50 - ≤68.3)	943 (77.7%)	271 (22.3%)	0.24 (0.20-0.28) p<0.0005	0.61 (0.49-0.76) p<0.0005
Particulate Matter (PM)$_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	693 (57.0%)	523 (43.0%)	1	1
	2 (>6.45 - ≤8.51)	711 (58.6%)	503 (41.4%)	0.94 (0.80-1.10) p=0.0431	1.17 (0.96-1.43) p=0.113
	3 (>8.51 - ≤40.78)	762 (62.7%)	453 (37.3%)	0.79 (0.67-0.93) p=0.004	0.99 (0.81-1.21) p=0.901

Table A1 (iii): Associations between air pollutants and a 10% increase in 'Asthma' hospital admissions based on low, medium and high levels of pollutants. Significant values ($p<0.05$ and $OR>1.0$) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Asthma Admissions (standardised) exceeding Gastro by ≥ 10%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	799 (65.7%)	417 (34.3%)	1	1
	2 (>0.042- ≤0.73)	721 (59.3%)	495 (40.7%)	1.32 (1.11-1.55) p=0.001	1.11 (0.93-1.30) p=0.237
	3 (>0.73 – ≤1.92)	541 (44.3%)	679 (55.7%)	2.41 (2.04-2.83) p<0.0005	1.39 (1.15-1.68) p=0.001
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	640 (52.6%)	576 (47.4%)	1	1
	2 (>1.67 - ≤2.10)	740 (60.6%)	481 (39.4%)	0.77 (0.62-0.85) p<0.0005	0.95 (0.79-1.13) p=0.543
	3 (>2.10 - ≤3.74)	681 (56.0%)	534 (44.0%)	0.87 (0.74-1.02) p=0.090	0.94 (0.78-1.12) p=0.474
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	860 (69.7%)	373 (30.3%)	1	1
	2 (>0.11 - ≤0.21)	688 (57.6%)	506 (42.4%)	1.70 (1.44-2.00) p<0.0005	1.55 (1.30-1.45) p<0.0005
	3 (>0.21 - ≤3.04)	513 (41.9%)	712 (58.1%)	3.20 (2.71-3.78) p<0.0005	2.03 (1.68-2.45) p<0.0005

Particulate Matter (PM)$_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	584 (47.9%)	634 (52.1%)	1	1
	2 (>13.55 - ≤18.50)	652 (53.5%)	566 (46.5%)	0.80 (0.68-0.94) p=0.006	0.94 (0.80-1.11) p=0.467
	3 <18.50 - ≤68.3)	825 (68.0%)	389 (32.0%)	0.43 (0.37-0.51) p<0.0005	0.76 (0.64-0.91) p=0.0004
Particulate Matter (PM)$_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	684 (56.3%)	532 (43.7%)	1	1
	2 (>6.45 - ≤8.51)	657 (54.1%)	557 (45.9%)	1.09 (0.93-1.28) p=0.291	1.22 (1.03-1.45) p=0.019
	3 (>8.51 - ≤40.78)	717 (59.0%)	498 (41.0%)	0.89 (0.76-1.05) p=0.168	1.02 (0.86-1.21) p=0.812

Table A1 (iv): Associations between air pollutants and a 10% increase in 'COPD' hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	COPD Admissions (standardised) exceeding Gastro by ≥ 10%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	841 (69.2%)	375 (30.8%)	1	1
	2 (>0.042- ≤0.73)	786 (64.6%)	430 (35.4%)	1.23 (1.04-1.45) p=0.018	1.17 (0.97-1.40) P=0.104
	3 (>0.73 – ≤1.92)	653 (53.5%)	567 (46.5%)	1.95 (1.65-2.30) p<0.0005	1.35 (1.11-1.66) p=0.003
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	748 (61.5%)	468 (38.5%)	1	1
	2 (>1.67 - ≤2.10)	835 (68.4%)	386 (31.6%)	0.74 (0.63-0.87) P<0.0005	0.88 (0.72-1.06) p=0.174
	3 (>2.10 - ≤3.74)	697 (57.4%)	518 (42.6%)	1.19 (1.01-1.40) p=0.037	0.99 (0.82-1.21) p=0.947
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	887 (71.9%)	346 (28.1%)	1	1
	2 (>0.11 - ≤0.21)	773 (64.7%)	421 (35.3%)	1.40 (1.18-1.66) p<0.0005	1.37 (1.14-1.65) p=0.001
	3 (>0.21 - ≤3.04)	620 (50.6%)	605 (49.4%)	2.50 (2.12-2.96) p<0.0005	1.88 (1.54-2.30) p<0.0005

Particulate Matter (PM)$_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	659 (54.1%)	559 (45.9%)	1	1
	2 (>13.55 - ≤18.50)	722 (59.3%)	496 (40.7%)	0.81 (0.69-0.95) p=0.010	1.04 (0.87-1.24) p=0.060
	3 <18.50 - ≤68.3)	899 (74.1%)	315 (25.9%)	0.41 (0.35-0.49) p<0.0005	0.82 (0.68-1.00) p=0.046
Particulate Matter (PM)$_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	726 (59.7%)	490 (40.3%)	1	1
	2 (>6.45 - ≤8.51)	769 (63.3%)	445 (36.7%)	0.86 (0.73-1.01) p=0.065	0.96 (0.80-1.15) p=0.644
	3 (>8.51 - ≤40.78)	780 (64.2%)	435 (35.8%)	0.83 (0.070-0.097) p=0.023	0.98 (0.82-1.17) p=0.792

Table A1 (v): Associations between air pollutants and a 10% increase in Pneumonia / Influenza/Acute Bronchitis hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Pneumonia/ Influenza/ Acute Bronchitis Admissions (standardised) exceeding Gastro by ≥ 10%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	740 (60.9%)	476 (39.1%)	1	1
	2 (>0.042- ≤0.73)	737 (60.6%)	479 (39.4%)	1.01 (0.86-1.19) p=0.901	0.91 (0.75-1.11) p=0.358
	3 (>0.73 – ≤1.92)	590 (48.4%)	630 (51.6%)	1.66 (1.41-1.95) p<0.0005	1.08 (0.87-1.33) p=0.498
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	708 (58.2%)	508 (41.8%)	1	1
	2 (>1.67 - ≤2.10)	776 (63.6%)	445 (36.4%)	0.80 (0.68-0.94) p=0.007	0.92 (0.75-1.13) p=0.420
	3 (>2.10 - ≤3.74)	583 (48.0%)	632 (52.0%)	1.51 (1.29-1.77) p<0.0005	1.10 (0.90-1.34) p=0.324
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	764 (62.0%)	469 (38.0%)	1	1
	2 (>0.11 - ≤0.21)	740 (62.0%)	454 (38.0%)	1.00 (0.85-1.18) p=0.994	0.91 (0.75-1.10) p=0.337
	3 (>0.21 - ≤3.04)	563 (46.0%)	662 (54.0%)	1.92 (1.63-2.25) p<0.0005	1.33 (1.08-1.64) p=0.008

Particulate Matter (PM)$_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	529 (43.4%)	689 (56.6%)	1	1
	2 (>13.55 - ≤18.50)	668 (54.8%)	550 (45.2%)	0.63 (0.54-0.74) p<0.0005	0.83 (0.69-1.00) p=0.054
	3 <18.50 - ≤68.3)	868 (71.5%)	346 (28.5%)	0.31 (0.26-0.36) p<0.0005	0.67 (0.69-0.82) p<0.0005
Particulate Matter (PM)$_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	654 (53.8%)	562 (46.2%)	1	1
	2 (>6.45 - ≤8.51)	672 (55.4%)	542 (44.6%)	0.94 (0.80-1.10) p=0.437	1.13 (0.94-1.37) p=0.191
	3 (>8.51 - ≤40.78)	735 (60.5%)	480 (39.5%)	0.76 (0.65-0.89) p=0.001	0.95 (0.78-1.15) p=0.608

Table A1 (vi): Associations between air pollutants and a 10% increase in 'Other Respiratory' admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile range**	Other Respiratory Admissions (standardised) exceeding Gastro by ≥ 10%		**Odds-ratio (95% CI) (p-value)**	**Odds-ratio (95% CI) (p-value) Adjusted for season**
		No	**Yes**		
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	844 (69.4%)	372 (30.6%)	1	1
	2 (>0.042- ≤0.73)	796 (65.5%)	420 (34.5%)	1.20 (1.01-1.42) p=0.038	1.03 (0.84-1.25) p=0.808
	3 (>0.73 – ≤1.92)	568 (46.6%)	652 (53.4%)	2.60 (2.21-3.07) p<0.0005	1.26 (1.02-1.55) p=0.036
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	693 (57.0%)	523 (43.0%)	1	1
	2 (>1.67 - ≤2.10)	830 (68.0%)	391 (32.0%)	0.62 (0.53-0.74) p<0.0005	0.96 (0.78-1.20) p=0.692
	3 (>2.10 - ≤3.74)	685 (56.4%)	530 (436%)	1.03 (0.87-1.20) p=0.761	1.10 (0.89-1.40) p=0.363
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	872 (70.7%)	361 (293%)	1	1
	2 (>0.11 - ≤0.21)	796 (66.7%)	398 (333%)	1.21 (1.02-1.43) p=0.031	1.08 (0.89-1.31) p=0.439
	3 (>0.21 - ≤3.04)	540 (44.1%)	685 (55.9%)	3.06 (2.59-3.62) p<0.0005	1.59 (1.29-1.96) p<0.0005

Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	581 (47.7%)	637 (52.3%)	1	1
	2 (>13.55 - ≤18.50)	687 (56.4%)	531 (43.6%)	0.71 (0.60-0.83) p<0.0005	0.93 (0.77-1.12) p=0.443
	3 <18.50 - ≤68.3)	938 (77.3%)	276 (22.7%)	0.27 (0.23-0.32) p<0.0005	0.61 (0.77-1.12) p<0.0005
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	707 (58.1%)	509 (41.9%)	1	1
	2 (>6.45 - ≤8.51)	720 (59.3%)	494 (40.7%)	0.95 (0.81-1.12) p=0.559	1.13 (0.94-1.34) p=0.203
	3 (>8.51 - ≤40.78)	775 (63.8%)	440 (36.2%)	0.79 (0.67-0.93) p=0.004	0.90 (0.74-1.08) p=0.263

Appendix B: Associations between air pollutants and a 20% increase in hospital admissions

Table A2 (i): Associations between air pollutants and a 20% increase in 'Cardiovascular' hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Cardiovascular Admissions (standardised) exceeding Gastro by ≥ 20%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	1039 (84.3%)	194 (15.7%)	1	1
	2 (>0.11 - ≤0.21)	921 (77.1%)	273 (22.9%)	1.59 (1.29-1.95) p<0.0005	1.52 (1.24-1.87) p<0.0005
	3 (>0.21 - ≤3.04)	849 (69.3%)	376 (30.7%)	2.37 (1.95-2.88) p<0.0005	1.77 (1.42-2.20) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	992 (81.6%)	224 (18.4%)	1	1
	2 (>0.042- ≤0.73)	975 (80.2%)	241 (19.8%)	1.10 (0.84 – 1.34) p=0.381	1.024 (0.83-1.26) p=0.821
	3 (>0.73 – ≤1.92)	842 (69.0%)	378 (31.0%)	1.99 (1.65 – 2.40) p<0 0005	1.472 (1.19-1.83) p<0.0005
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	907 (74.6%)	309 (25.4%)	1	1
	2 (>1.67 - ≤2.10)	958 (78.5%)	263 (21.5%)	0.81 (0.67-0.97) p=0.024	1.46 (1.14-1.88) p=0.003
	3 (>2.10 - ≤3.74)	944 (77.7%)	271 (22.3%)	0.84 (0.70-1.02) p=0.073	3.31 (2.63-4.18) p<0.0005

Particulate Matter $(PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	898 (73.7%)	320 (26.3%)	1	1
	2 (>13.55 - ≤18.50)	913 (75.0%)	305 (25.0%)	0.94 (0.78-1.12) p=0.487	1.05 (0.87-1.26) p=0.637
	3 <18.50 - ≤68.3)	996 (82.0%)	218 (18.0%)	0.61 (0.51-0.75) p<0.0005	0.89 (0.637) p=0.237
Particulate Matter $(PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	931 (76.6%)	285 (23.4%)	1	1
	2 (>6.45 - ≤8.51)	927 (76.4%)	287 (23.6%)	1.03 (0.84-1.22) p=0.910	1.07 (0.88-1.29) p=0.500
	3 (>8.51 - ≤40.78)	945 (77.8%)	270 (22.2%)	0.93 (0.77-1.13) p=0.475	0.98 (0.81-1.19) p=0.852

Table A2 (ii) : Associations between air pollutants and a 20% increase in 'Total Respiratory' hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Total Respiratory Admissions (standardised) exceeding Gastro by ≥ 20%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	971 (78.8%)	262 (21.2%)	1	1
	2 (>0.11 - ≤0.21)	859 (71.9%)	335 (28.1%)	1.45 (1.20-1.74) p<0.0005	1.38 (1.11-1.71) p=0.003
	3 (>0.21 - ≤3.04)	611 (49.9%)	614 (50.1%)	3.72 (3.12-4.44) p<0.0005	2.24 (1.79-2.81) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	919 (75.6%)	297(24.4%)	1	1
	2 (>0.042- ≤0.73)	876 (72.0%)	340 (28.0%)	1.20 (1.00 – 1.44) p=0.047	1.04 (0.84-1.28) p=0.749
	3 (>0.73 – ≤1.92)	646 (53.0%)	574 (47.0%)	2.75 (2.31 – 3.27) p<0.0005	1.42 (1.13-1.77) p=0.002
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	778 (64.0%)	438 (36.0%)	1	1
	2 (>1.67 - ≤2.10)	912 (74.7%)	309 (25.3%)	0.60 (0.51-0.72) p<0.0005	0.86 (0.68-1.08) p=0.191
	3 (>2.10 - ≤3.74)	751 (61.8%)	464 (38.2%)	1.10 (0.931-1.294) p=0.268	1.00 (0.79-1.25) p=0.964

Particulate Matter $(PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	664 (54.5%)	554 (45.5%)	1	1
	2 (>13.55 - ≤18.50)	763 (62.6%)	455 (37.4%)	0.72(0.61-0.84) p<0.0005	1.01 (0.83-1.22) p=0.938
	3 <18.50 - ≤68.3)	1012 (84.3%)	202 (16.6%)	0.24 (0.-0.29) p<0.0005	0.63 (0.50-0.79) p=<0.0005
Particulate Matter $(PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	784 (64.5%)	432 (35.5%)	1	1
	2 (>6.45 - ≤8.51)	806 (66.4%)	408 (33.6%)	0.92 (0.78-1.09) p=0.320	1.12 (0.92-1.37) p=0.267
	3 (>8.51 - ≤40.78)	845 (69.5%)	370 (30.5%)	0.80 0.67-0.94 p=0.008	0.97 (0.79-1.19) p=0.763

Table A2 (iii) : Associations between air pollutants and a 20% increase in 'Asthma' hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Asthma Admissions (standardised) exceeding Gastro by ≥ 20%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	910 (73.8%)	323 (26.2%)	1	1
	2 (>0.11 - ≤0.21)	760 (63.7%)	434 (36.3%)	1.61 (1.35-1.91) p<0.0005	1.47 (1.22-1.76) p<0.005
	3 (>0.21 - ≤3.04)	593 (48.4%)	632 (51.6%)	3.00 (2.54-3.56) p<0.0005	1.89 (1.56-2.29) p<0.005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	848 (69.7%)	368 (30.3%)	1	1
	2 (>0.042- ≤0.73)	788 (64.8%)	428 (35.2%)	1.25 (1.06 – 1.48) p=0.01	1.05 (0.88-1.26) p=0.065
	3 (>0.73 – ≤1.92)	627 (51.4%)	593 (48.6%)	2.18 (1.85 – 2.57) p<0.0005	1.23 (1.02-1.49) p=0.34
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	713 (58.6%)	503 (41.4%)	1	1
	2 (>1.67 - ≤2.10)	812 (66.5%)	409 (33.5%)	0.71 (0.61-0.84) p<0.0005	0.94 (0.78-1.13) p=0.494
	3 (>2.10 - ≤3.74)	738 (60.7%)	477 (39.3%)	0.92 (0.78-1.08) p=0.290	0.99 (0.82-1.19) p=0.895

Particulate Matter ($PM)_{10}$ ($2.93-68.30) µg/$m^3$ (1 day)	1 ($2.92 - ≤$13.55)	657 (53.9%)	561 (46.1%)	1	1
	2 (>$13.55 - ≤$18.50)	724 (59.4%)	494 (40.6%)	0.80 (0.68-0.94) p=0.0006	0.94 (0.80-1.11) p=0.477
	3 <$18.50 - ≤$68.3)	882 (72.7%)	332 (27.3%)	0.44 (0.37-0.57) p<0.0005	0.78 (0.65-0.95) p=0.011
Particulate Matter ($PM)_{2.5}$ ($0.00-40.78) µg/$m^3$ (1 day)	1 ($0.00 – ≤$6.45)	751 (61.8%)	465 (38.2%)	1	1
	2 (>$6.45 - ≤$8.51)	725 (59.7%)	489 (40.3%)	1.09 (0.93-1.28) p=0.303	1.22 (1.03-1.45) p=0.022
	3 (>$8.51 - ≤$40.78)	784 (64.5%)	431 (35.5%)	0.39 (0.75-1.05) p=0.157	1.01 (0.89-1.21) p=0.893

Table A2 (iv): Associations between air pollutants and a 20% increase in 'Chronic Obstructive Pulmonary Disease' (COPD) hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	COPD Admissions (standardised) exceeding Gastro by ≥ 20%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	960 (77.9%)	273 (22.1%)	1	1
	2 (>0.11 - ≤0.21)	842 (70.5%)	352 (29.5%)	1.47 (1.22-1.77) p<0.0005	1.45 (1.19-1.76) p<0.0005
	3 (>0.21 - ≤3.04)	708 (57.8%)	517 (42.2%)	2.57 (2.15-3.06) p<0.0005	1.93 (1.57-2.37) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	908 (74.7%)	308 (25.3%)	1	1
	2 (>0.042- ≤0.73)	863 (71%)	353 (29.0%)	1.21 (1.01 – 1.44) p=0.040	1.15 (0.94-1.39) p=0.168
	3 (>0.73 – ≤1.92)	739 (60.6%)	481 (39.4%)	1.92 (1.61 – 2.28) p<0.0005	1.33 (1.08-1.63) p=0.008
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	828 (68.1%)	388 (31.9%)	1	1
	2 (>1.67 - ≤2.10)	903 (74.0%)	318 (26.0%)	0.75 (0.630-0.896 p=0.001	0.91 (0.75-1.12) p=0.375
	3 (>2.10 - ≤3.74)	739 (64.1%)	436 (35.9%)	1.19 (1.01-1.41) p=0.038	1.04 (0.84-1.26) p=0.770

Particulate Matter ($PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	758 (%)	460 (%)	1	1
	2 (>13.55 - ≤18.50)	797 (%)	421 (%)	0.87 (0.74-1.03) p=0.10	1.12 (0.94-1.10) p=0.210
	3 <18.50 - ≤68.3)	955 (%)	259 (%)	0.45 (0.37-0.54) p<0.0005	0.90 (0.73-1.10) p=0.315
Particulate Matter ($PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	813 (%)	403 (%)	1	1
	2 (>6.45 - ≤8.51)	841 (%)	373 (%)	0 .90 (0.75-1.06) p=0.021	1.00 (0.84-1.21) p=0.972
	3 (>8.51 - ≤40.78)	851 (%)	364 (%)	0.86 (0.73-1.02) p=0.091	1.01 (0.84-1.22) p=0.883

Table A2 (v) : Associations between air pollutants and a 20% increase in 'Pneumonia / Influenza / Acute Bronchitis' hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Pneumonia/ Influenza/ Acute Bronchitis Admissions (standardised) exceeding Gastro by ≥ 20%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	849 (68.9%)	384 (31.1%)	1	1
	2 (>0.11 - ≤0.21)	803 (67.3%)	395 (32.7%)	1.08 (0.91-1.28) p=0.397	1.00 (0.82-1.22) p=0.99
	3 (>0.21 - ≤3.04)	635 (51.8%)	590 (48.2%)	2.05 (1.74-2.42) p<0.0005	1.43 (1.16-1.77) p=0.001
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	818 (67.3%)	398 (32.7%)	1	1
	2 (>0.042- ≤0.73)	813 (66.9%)	403 (33.1%)	1.02 (0.86 – 1.21) p=0.829	0 .92 (0.76-1.12) p=0.425
	3 (>0.73 – ≤1.92)	656 (53.8%)	564 (46.2%)	1.77 (1.50 – 2.08) p<0.0005	1.16 (0.94-1.44) p=0.176
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	781 (64.2%)	435 (35.8%)	1	1
	2 (>1.67 - ≤2.10)	858 (70.3%)	363 (29.7%)	0.76 (0.64-0.90) p=0.001	0.89 (0.72-1.10) p=0.286
	3 (>2.10 - ≤3.74)	648 (53.3%)	567 (46.7%)	1.57 (1.34-1.85) p<0.0005	1.21 (0.98-1.49) p=0.075

Particulate Matter $(PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	609 (50.0%)	609 (50.0%)	1	1
	2 (>13.55 - ≤18.50)	740 (60.8%)	478 (39.2%)	0.65 (0.55=0.76) p<0.0005	0.86 (0.71-1.03) p=0.095
	3 <18.50 - ≤68.3)	936 (77.1%)	278 (22.9%)	0.30 (0.25-0.35) p=<0.0005	0.87 (0.54-0.82) p<0.0005
Particulate Matter $(PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	728 (59.9%)	488 (40.1%)	1	1
	2 (>6.45 - ≤8.51)	746 (61.4%)	468 (38.6%)	0 .936 (0.80-1.10) p=0.425	1.13 (0.93-1.36) p=0.215
	3 (>8.51 - ≤40.78)	807 (66.4%)	408 (33.6%)	0.754 (0.64-0.89) p=0.0001	0.94 (0.77-1.14) p=0.523

Table A2 (vi) : Associations between air pollutants and a 20% increase in 'Other Respiratory' hospital admissions based on low, medium and high levels of pollutants. Significant values (P<0.05 and OR>1.0) are highlighted in red.

Pollutant (Range)	**Tertile Range**	Other Respiratory Admissions (standardised) exceeding Gastro by ≥ 20%		Odds-ratio (95% CI) (p-value)	Odds-ratio (95% CI) (p-value) **Adjusted for season**
		No	Yes		
Carbon Monoxide (CO) (0.00-3.04) ppm	1 (0.00 -≤0.11)	964 (78.2%)	269 (21.8%)	1	1
	2 (>0.11 - ≤0.21)	888 (74.4%)	306 (25.6%)	1.24 (1.02-1.49) p=0.027	1.10 (0.89-1.35) p=0.042
	3 (>0.21 - ≤3.04)	639 (52.2%)	580 (47.8%)	3.29 (2.76-3.92) p<0.0005	1.61 (1.29-1.70) p<0.0005
Nitrogen Dioxide ($N0_2$) (0.00-1.92) pphm (1 day)	1 (0.00 - ≤0.042)	931 (76.6%)	285 (23.4%)	1	1
	2 (>0.042- ≤0.73)	889 (73.1%)	327 (26.9%)	1.2 (1.00-1.44) p=0.050	1.01 (0.82-1.20) p=0.923
	3 (>0.73 – ≤1.92)	671 (55.0%)	549 (45.0%)	2.67 (2.25-3.18) p<0.0005	1.19 (0.95-1.48) p=0.131
Ozone (O_3) (0.75 – 3.74) pphm (1 day)	1 (0.75 - ≤1.67)	777 (63.9%)	439 (36.1%)	1	1
	2 (>1.67 - ≤2.10)	914 (74.9%)	307 (25.1%)	0.59 (0.50-0.71) p<0.0005	0.97 (0.78-1.20) p=0.800
	3 (>2.10 - ≤3.74)	800 (65.8%)	415 (34.2%)	0.92 (0.72-1.09) p=0.315	1.02 (0.82-1.27) p=0.850

Particulate Matter $(PM)_{10}$ (2.93-68.30) µg/m^3 (1 day)	1 (2.92 - ≤13.55)	686 (56.3%)	532 (43.7%)	1	1
	2 (>13.55 - ≤18.50)	783 (64.3%)	435 (35.7%)	0.72 (0.61-0.84) p<0.0005	1.00 (0.82-1.20) p=0.565
	3 <18.50 - ≤68.3)	1020 (84.0%)	194 (16.0%)	0.25 (0.20-0.30) p<0.0005	0.92 (0.75-1.13) p<0.0005
Particulate Matter $(PM)_{2.5}$ (0.00-40.78) µg/m^3 (1 day)	1 (0.00 – ≤6.45)	799 (65.7%)	417 (34.3%)	1	1
	2 (>6.45 - ≤8.51)	834 (68.7%)	380 (31.3%)	0.87 (0.74-1.03) p=0.116	1.00 (0.82-1.20) p=0.990
	3 (>8.51 - ≤40.78)	852 (70.1%)	363 (29.9%)	0.82 (0.69-0.97) p=0.02	0.92 (0.75-1.13) p=0.410