School of Public Health

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Evaluation of Exposure to Particulate Air Pollution and Airway Deposition in Cyclists in the Perth Metropolitan Area

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Author's Declaration

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

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

The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conducted in Human Research (2007) –updated March 2014. The proposed research study received human research ethics approval from the Curtin University Human Research Ethics Committee (HREC Ethics A), Approval Number HR 183/2014.

Signature:

Date: 26th August 2019

Abstract

Cycling is often promoted as a means of reducing vehicular congestion, greenhouse gases, noise and air pollutant emissions in urban areas. It is also endorsed as a healthy means of transportation, in terms of reducing the risk of developing a range of physical and psychological conditions. However, people who cycle regularly may not be aware that they can become exposed to high levels of air pollutants such as particulate matters (PM) due to their increased ventilation rate and close proximity to vehicular emissions. As a result, several studies have found an association between personal exposure to traffic air pollution and health impacts among cyclists. PM is a mixture of solid particles and liquid droplets which has an aerodynamic diameter as one of the main features and describes its ability to transport in the atmosphere and be inhaled by organisms. For example, the coarse particles with a size range of 10 µm or less (PM_{10}); respirable PM with an aerodynamic diameter of 4 µm or less is PM_4; fine PM with an aerodynamic diameter of 2.5 µm or less is $PM_{2.5}$; ultrafine particulate with an aerodynamic diameter equal to or less than 0.1 µm is $PM_{0.1}$; total inhalable PM size fractions (PM_{total}). The main aim of this study was to conduct spatial (lung region) assessments to determine exposure to particulate air pollution in the lungs of cyclists commuting in the Perth Metropolitan Area.

The proposed study involved 122 number of cyclists cycling in four routes: two routes within community areas (route 1 and route 2) and two routes near freeways (route 3 and route 4). The participants were and males and females aged between 20 and 55 years recruited randomly from Perth cycling clubs, Curtin University and the general community, with the selection criteria including non-smokers ('never smoked' and 'currently non-smokers' who had quit smoking) who cycle at least 150 km/week – ideally along one of the four study routes. People who reported cardiovascular and other chronic health conditions (not asthma) were excluded from the study. Each cyclist rode the same route at both high and low levels of exertion (self-perceived) during cold and warm seasons. Ambient air quality monitoring was conducted using a DustTrakTM DRX Aerosol Monitor 8533b (TSI Inc., MN, USA) measures PM_1, $PM_{2.5}$, PM_4, PM_{10} and PM_{total}, Testo 350 gas analyser measures oxygen (O_2), carbon monoxide (CO), nitrogen oxide (NO), nitrogen dioxide (NO_2), oxide of nitrogen (NOx), sulphur dioxide (SO_2), hydrogen sulfide (H_2S), carbon

dioxide (CO_2) and total hydrocarbons (CH), P-Trak Model 8525 measures the ultrafine particulate levels, Universal Scanning Mobility Particle Sizer 2700 (USMPS) measures the size and number concentration of ultrafine particles and Anemometer measures temperature, relative humidity and wind speed. Personal exposure monitoring of respirable particulate air pollution was conducted using SKC cyclone samplers. Respiration rate of the participants was monitored using a bioHarness 3.0 heart/breath rate monitor. Lab-based spirometry was also performed to obtain an individual input variable for the Multiple Path Particle Dosimetry (MPPD) Model. This model was then used to determine individual deposition patterns of ultrafine and fine particulate matter in the lungs according to the four cycling routes, level of exertion, gender, season and physical properties of the air particulate matter.

This study established a statistically significant difference of PM_1, $PM_{2.5}$, PM_4, PM_{10} and PM_{total} concentrations between the four cycling routes. Overall, route 1 and route 3 appeared to be the most polluted cycling routes and route 2 was the least polluted in terms of ambient particle concentrations. The PM concentrations were higher during the cold season (only in route 3 and route 4) compared to warm; however, comparison between two seasons in those two routes was not statistically significant. Throughout the sampling period, median pollutant levels of PMs did not exceed the national guideline values. This study showed that the median recorded levels of NO and NO_2 were significantly higher in route 3. The highest median levels of CO and CO_2 were recorded in route 4 but no significant difference was observed between the four routes. Overall, route 3 was found to have higher concentrations of selected gases, among which NO_2 (0.091–0.359 ppm) and SO_2 (0.074–0.241) were above the NEPM standard. Besides, rainfall was significantly (positively) correlated with PM, NO and NO_2 concentrations (likely due to measured vehicle traffic during rain events), but negatively correlated with temperature. The personal exposure assessments demonstrated that the cyclists riding on route 1 were exposed to higher PM_4 concentrations compared to the other three routes and this is consistent with the findings from the background exposure assessment. Also, cyclists were exposed to significantly higher PM_4 concentrations during the cold season compared to the warm season in all four routes.

In this study, aerosol particle deposition in human lungs was studied using lung deposition estimates based on the Multiple Path Particle Dosimetry (MPPD) model. In order to determine

the median of the size classes (PM_1, $PM_{2.5}$, PM_4 and PM_{10}), the mass median diameter (MMD) was calculated which is defined as the diameter that divides the graphical representation of the distribution of mass into two segments of equal area. The MMD counter's size classes include $PM_{0.1}$, $PM_{0.562}$, $PM_{1.778}$ µm, $PM_{3.652}$ and $PM_{6.494}$. It is evident from the MPPD model that the overall deposited fraction of smaller particles (sized between 0.1 and 3.652 µm) was higher in the pulmonary region of the respiratory system whereas the deposition of coarse particles ($PM_{6.494}$) was mainly concentrated in the Tracheobronchial (TB) region, and this is consistent for all cycling routes. Regional Cumulative Deposition Curves (a function of particle size in both Pulmonary and TB region) were calculated and averaged for all participants and split variously into four different groups: the four cycling routes, gender, high and low exertion levels, and cold and warm seasons. The general trends indicate that in the pulmonary region, the deposition rate decreased for particles of 4–5 µm (approximately) and no deposition occurred for particles with a size of 7 µm and larger. However, in the TB region, the deposited particles were mainly larger particles, with a deposition rate decreasing above approximately 7–8 µm in particle size. In the pulmonary region, participants cycling along route 1 had the highest deposition of particles (all sized) whereas those cycling along route 3 had the lowest value. With regards to gender, females had higher particle deposition in the pulmonary region compared to males. We also noted higher deposition rates during the cold season compared to the warm season. As discussed above, the trend was reversed in the TB region in each group.

The study outcomes can be applied to developing approaches to determine the deposition of particles in the respiratory tract based on the exertion of cycling (and other populations exposed to environmental or occupational pollutants combined with physical exertion) and ambient PM concentrations. This study also concluded that while developing these kinds of approaches seasonal variation should also be considered while calculating the deposition dose among cyclist to evaluate the impact that the inhalation and uptake of particulate air pollution may have on human health.

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List of Abbreviations

AAQ:	Ambient Air Quality
ADR:	Australian Design Rules
CAFE:	Clean Air for Europe
CFD:	Computational Fluid Dynamics
CO:	Carbon Monoxide
CITT:	Chemical Industry Institute of Toxicology
DEMC:	Differential Electrical Mobility Classifier
DMA:	Differential Mobility Analyser
DWER:	Department of Water and Environmental Regulation
ECG:	Electrocardiography
EPHC:	Environmental Protection and Heritage Council
ENPs:	Engineered Nanoparticles
EU:	European Union
FRC:	Functional Residual Capacity
H_2SO_4:	Sulphuric Acid
H_2O_2:	Hydrogen Peroxide
IGAE:	Intergovernmental Agreement

LRT: The Lower Respiratory Tract

LRI: Long-Range Research Initiative

MACT: Maximum Achievable Control Technology

MPPD: Multiple-Path Particle Dosimetry

NAAQS: National Ambient Air Quality Standard

NEPM: National Environmental Protection Measures

NEPC: The National Environmental Protection Council

NMAPS: National Mortality Air Pollution Study

NO: Nitrogen Oxide

NO_2: Nitrogen Dioxide

NOx: Oxide of Nitrogen

NO_5: Nitrogen Pentoxide

N_2O: Nitrogen Monoxide

NSW: New South Wales

O_3: Ozone

PM: Particulate Matter

PNC: Particle Number Concentration

RIVM: Dutch National Institute for Public Health and the Environment

SO_2: Sulphur Dioxide

SO_3:	Sulphur Trioxide
TB:	Tracheobronchial
TEOMs:	Tapered Element Oscillating Microbalances
UFP:	Ultrafine Particulate
USEPA:	United States Environment Protection Agency
URT:	Upper Respiratory Tract
VAP:	Vehicular Air Pollutant
VOCs:	Volatile Organic Compounds
WA:	Western Australia
WHO:	World Health Organisation

CHAPTER 1 : INTRODUCTION

1.1 Background

Air pollution represents the biggest environmental health risk affecting people around the world. According to the World Health Organisation (WHO), ambient air pollution in both cities and rural areas was estimated to have caused 4.2 million premature deaths worldwide in 2016 (WHO, 2018). This mortality was due to exposure to fine particulate matter ($PM_{2.5}$), which is associated with cancer, cardiovascular and respirable diseases. This inhalable particle ($PM_{2.5}$) which normally made up of sulphate and nitrate particles, organic and elemental carbon is considered as one of the major air quality concerns due to its adverse effect on human health. The WHO estimated that 58% of outdoor air-pollution-related premature deaths were due to strokes and ischaemic heart disease, 18% were due to chronic obstructive pulmonary disease and acute lower respiratory infections, and 6% of deaths were due to lung cancer.

To reduce the harmful impacts of PM on human health, WHO has developed a series of air quality guidelines since 1987 for some pollutants, such as particulate matter (PM), sulphur dioxide (SO_2), nitrogen dioxide (NO_2) and ozone (O_3). These pollutants, along with carbon monoxide (CO), are classified as 'criteria air pollutants' by the WHO (2005), the United States Environmental Protection Agency (2011) and the Department of the Environment and Heritage Australia (2005). In Australia, there has been significant progress in controlling the emission of air pollutants to comply with the National Environmental Protection Measures (NEPM) standard; however, the recommended PM concentration levels are yet to be achieved as levels often exceed the national standard in most large cities in Australia, including Perth, Western Australia (Department of Environment and Conservation, 2010). Since particles have been significantly associated with human mortality and morbidity, it is necessary to monitor and control the emission of PMs in various micro-environments.

Traffic-related emissions are commonly recognised as a significant and increasing source of air pollutants, representing 23% of greenhouse gases globally (Leipzig, 2010). Traffic emissions

mainly consist of CO, hydrocarbon, NOx and PM (Choudhary & Gokhale, 2016), the majority of which result from incomplete combustion of fuel. The concentrations of these pollutants are significantly higher in urban areas, especially near busy roads and freeways. People living and/or working in urban areas are therefore exposed to high levels of air pollution, especially when they commute on highly trafficked roads. It is widely acknowledged that air pollution emission from vehicles has adverse impacts on human health, including on mortality, morbidity and hospital admissions (Dons et al., 2012; Okokon et al., 2017). Several studies have tried to explain the association between pollutant concentrations, traffic intensity and human exposure; however, the inhalation intensity appeared to vary greatly between commuters – such as car or bus passengers – and cyclists (Peters et al., 2014; Ragettli et al., 2013). Most studies to date have shown limited consistency, likely due to sample size and methodology problems (de Nazelle et al., 2012; Kingham et al., 2013; Panis et al., 2010). The inconsistency in the results of the above studies may be due to differences in research design, trip mode, choice of route and type of equipment used to measure air pollution (Kendrick et al., 2011; Xu et al., 2016). The majority of these studies were conducted in large urban settings (Peters et al., 2014; Strak et al., 2010) where commuters were in close vicinity to vehicle emissions (Hatzopoulou, Weichenthal, Dugum, Pickett, Miranda-Moreno, & Kulka, 2013). However, the general trends from previous studies showed that cyclists were likely to receive higher exposures to traffic air pollution due to their close proximity to vehicle emissions and their increased minute ventilation (MacNaughton et al., 2014; Panis et al., 2010). Even though there were conflicting outcomes regarding vehicular pollution and its exposure, several epidemiological studies have already proved the association between traffic emission and adverse health outcomes among cyclists, including respirable-related diseases (Barkovich et al., 2012; Nwokoro et al., 2012). To evaluate the impact of air pollution inhalation on human health, better estimates of spatial lung deposition in the human respiratory system is highly recommended.

The chemical composition of inhaled particles and their deposition within different regions of the respiratory system may play a significant role in causing harm to human health. Therefore, it is necessary to understand the mechanism of how and where particle deposition occurs in the lungs to assess the proper impact of aerosols on human wellbeing. The human respiratory system

consists of three regions: the head region (nose, mouth, pharynx and larynx), where inhaled air gets warmed and humidified; the tracheobronchial region (TB), the airways from the trachea to the terminal bronchioles; and the pulmonary region (alveolar) where the exchange of carbon dioxide and oxygen through the process of respiration takes place (Sánchez-Soberón et al., 2015). Once particles get deposited, they are retained in the lungs for varying times depending on their physicochemical properties, the region where they are deposited and the type of mechanism involved. To integrate more realistic asymmetries in the lung structure and calculate the particle deposition at individual airway levels, the Multiple-Path Particle Dosimetry (MPPD) model was developed. The degree of particle deposition depends on particle size, shape and density, airway geometry and the breathing pattern of the individual (either nose or mouth breathing). In spite of this, the deposition could be different for different groups of people, such as adults, children or people with respiratory diseases.

To date, no study has been conducted to properly quantify spatial and temporal lung deposition of particulates among cyclists in Perth, Western Australia. A limited number of studies have been conducted in Australia that compares the exposure level of air pollutants among different modes of commuters – such as car, bus or ferry commuters, bicycle riders, and pedestrians – with inconsistent results (Badland & Duncan, 2009; Chertok et al., 2004; Farrar, Dingle, & Tan, 2001; Knibbs & de Dear, 2010). One of the studies conducted in Australia (Hunter et al. (2012) measured exposure to particle-number concentrations among cyclists in Brisbane, finding higher particulate-number (and hence surface-area) concentrations near high-traffic roads during morning peak hour; however, again the work was somewhat limited in scope and methodology.

This study assessed exposures to PM among cyclists in Perth, providing vital insights for evaluating the toxicological dose and the associated respiratory health effects of exposure to PM (through the literature review) in urban areas.

1.2 Aims and objectives of the study

The main aim of the study is to conduct spatial (lung region) assessments to determine exposure to particulate air pollution in the lungs of cyclists commuting in Perth, Western Australia.

The specific objectives of the study are:

- To determine exposure levels to particulate air pollution (ultrafine, fine, respirable, coarse and PM_{total}) and other selected air pollutants, including NO_2, NO, NOx, SO_2 and CO at different cycling routes;
- To compare personal exposure levels of respirable (PM_4) particulate air pollution between cyclists at high and low levels of exertion along the same routes;
- To model the deposition of particulate air pollution in the lungs of each cyclist according to the duration of exposure, level of exertion and physical properties of the air particulate matter;
- To propose suggestions and recommendations to governments setting public health policies regarding cycling near major roads.

1.3 Hypotheses of the study

As per the above aims and objectives, it is hypothesised that:

- Ambient levels of particulate and other selected air pollutants will be higher near major routes compared to community routes;
- Cyclists' personal exposure levels of respirable particulate air pollution will vary significantly according to the level of ambient particulate concentrations, gender, level of exertion of cycling, location and season;
- The lung deposition pattern of particulates will depend on the exposure level of inhaled particulates, level of exertion, gender, season and type of cycling route.

1.4 Significance of the study

This research gives insight into the potential risks that cyclists may face from exposure to particulate air pollution during riding. Cycling in cities can be of particular concern because more people choose to cycle to work or for pleasure as a healthy option; however, cyclists may not be aware of the extent to which particulate matter may impact their health. The findings from this

study highlight the present scenario of ambient air pollution levels in different cycling routes in Perth. In addition, it develops concepts about personal exposure levels of particulate air pollution among cyclists, which are crucial in assessing and managing environmental health risks for them. The application of the MPPD model enhances our understanding of the deposition pattern of particulate matter of different size throughout the respiratory system and also offers further insight into the impact of certain personal and environmental characteristics on the deposition patterns.

To the best of our knowledge, this study is the first to assess the spatial lung deposition of ultrafine particles in cyclists that incorporates (real-world) temporal exposure and particle morphology data. The study outcome will prove vital for assessing the toxicological dose and potential health effects due to exposure to particulates. Also, the study could be useful for policymakers in developing strategies for new cycling paths that consider the potential adverse health impacts due to their close proximity to busy roads.

CHAPTER 2 : LITERATURE REVIEW

2.1 Introduction to air pollution

Air pollution, both indoor and outdoor, is recognised as a threat to human health, even at low doses, and is strongly associated with increased mortality and morbidity worldwide (WHO, 2016). The WHO estimates that around 4.2 million people died prematurely in 2016 due to the effects of ambient air pollution both in developed and developing countries (WHO, 2018). Numerous studies have been conducted regarding air pollution (Chan & Yao, 2008; Fransen et al., 2012; Ohara et al., 2007; Zou et al., 2018), its associated health impacts (Buteau & Goldberg, 2016; Chen, Hu, et al., 2016; Di, Dai, et al., 2017; Wheida et al., 2018) and potential control measures (Kumar & Katoria, 2013; Wang et al., 2014). The WHO reported that these premature deaths were due to a range of causes, including stroke, lung cancer, heart disease, and both chronic and acute respiratory diseases – including asthma, which ranked among the top ten causes of death in the world in 2012. These estimates are based not only on additional knowledge about the diseases caused by air pollution but also upon better assessment of human exposure to air pollutants through the use of enhanced measurement and technology (WHO, 2018). Rather than decreasing, the studies showed that the worldwide burden of disease due to ambient air pollution has increased steadily since 1990. The global risk factor associated with ambient particulate matter in terms of these diseases increased by 6% between 2000 and 2013 (Forouzanfar et al., 2016).

Air pollution normally consists of unwanted gaseous, liquid and solid substances suspended in the air. When present in sufficient concentrations for sufficient time and under certain conditions, air pollution may interfere with human health (Arden P & Dockery, 2012; Brook, Rajagopalan, Pope, Brook, Bhatnagar, Diez-Roux, et al., 2010). These agents are produced by both anthropogenic and natural sources. They may occur directly (primary pollutant) or be formed in the atmosphere from other emitted substances (secondary pollutants). Air pollutants can have several impacts on health, ecosystems, climate and the built environment. Further, they can be transported over long distances and hence may affect large areas. Impacts are generally

proportional to the concentration (Crouse et al., 2015; Huang et al., 2012; Kingham et al., 2013), the exposed population (Fakhri et al., 2009; James et al., 2010b; Kunzli et al., 2009; Nyhan, McNabola, & Misstear, 2014) and duration of exposure (Ichinose et al., 1998; Roberts, 2013; Shields et al., 2013). Health effects could be both acute and chronic depending upon exposure to the pollutants. These effects range from minor upper respiratory irritation to chronic respiratory and heart diseases, lung cancer, acute respiratory infection and asthmatic attacks (Horemans et al., 2012; Raaschou-Nielsen et al., 2016a; Tam, Wong, & Wong, 2015). Long-term exposure to air pollution has also been linked to premature mortality and reduced life expectancy (Lelieveld et al., 2015a). In 2016, around 91% of the world's population was living in a location where WHO air quality guideline levels were not met (WHO, 2018). As a consequence, it is estimated that the average life expectancy is 8.6 months lower than it would be without air pollution (European Commission, 2017).

Human activities are significantly raising the concentration of air pollutants in both urban and rural regions around the world. Among them, one of the main causes of air pollution is emissions from vehicular traffic in urban areas (Chen, Bekhor, et al., 2016; Nyhan et al., 2016). Numerous studies in the literature demonstrated substantial health impacts due to exposure to vehicular air pollution (Gonzalez et al., 2017; Kahr et al., 2016; York Bigazzi & Rouleau, 2017). The effects on human health range from minor concerns to major disorders, such as cardiovascular disease, asthma, leukaemia, lung cancer, stroke, and premature births and deaths (Adar et al., 2007; Bowatte et al., 2016; Kahr et al., 2016). In spite of this literature, knowledge about the health impacts of exposure to certain components of motor vehicle emissions – especially for small particles and toxic compounds – is still developing. The impact of these air pollutants on human health, the environment, vegetation and climate have been in the spotlight for research communities, environmental protection and regulatory agencies.

Therefore, further study is needed to monitor the emission sources and the type of pollutant it generates to clarify the impact it may cause to the human being.

2.2 Types of air pollutants

Air pollutants enter the atmosphere in two different forms: primary and secondary**.** Primary air pollutants are emitted directly from anthropogenic air pollution sources (e.g., chimneys, exhaust pipes and burning fuel) and natural sources (e.g., ash from volcanic eruptions). These pollutants enter the atmosphere and react further with other components to form harmful substances. Examples of primary pollutants include particulate matter (PM), carbon monoxide (CO), sulphur dioxide (SO_2), nitrogen oxide (NO) and nitrogen dioxide (NO_2).

Secondary air pollutants are those harmful substances that are formed due to the reaction between primary pollutants and other natural substances found in the atmosphere, especially oxygen and water. The most common examples include ozone (O_3), sulphur trioxide (SO_3), sulphuric acid (H_2SO_4) and hydrogen peroxide (H_2O_2). More information on air pollutants is provided below.

2.2.1 Particulate matter

Particulate matter is a mixture of solid particles and liquid droplets found in the air that vary in size, number, shape, surface area, chemical composition, solubility and origin (Kunzli et al., 2009; Peters et al., 2014; Pope et al., 2004). Although PM can be defined or classified in different ways, an aerodynamic diameter is one main feature and describes its ability to transport in the atmosphere and be inhaled by organisms (Esworthy, 2013). The United States Environment Protection Agency (US-EPA) mainly categorises particles into four

Figure 2.1: Comparison of the relative size of PM (Source US-EPA)

sizes based on their predicted penetration capacity into the lungs: inhalable coarse PM, with a size range of 10 µm or less in aerodynamic diameter (PM_{10}); fine PM, with an aerodynamic diameter of 2.5 µm or less; respirable PM with an aerodynamic diameter of 4 µm or less; and ultrafine particulate matter with an aerodynamic diameter equal to or less than 0.1 µm (USEPA, 2009). Particulate matter originates from a wide range of sources, such as agricultural and road dust, river beds, mining and construction sites. It is made up of different types of components, such as acid (nitrates and sulphates), organic chemicals, metals, dust particles, soil and water droplets (WHO, 2005). Some particles, such as smoke, dust, dirt and soot, are large enough to see with the naked eye but some particles are so small they can only be detected using an electron microscope. The sizes and characteristics of some PM are described below.

2.2.1.1 Coarse particulate matter

As mentioned above, the coarse particle (PM_{10}) is the particle with a size range of 10 µm or less in aerodynamic diameter (PM_{10}) and is primarily composed of crustal elements, organic debris, and metals from suspended road dust, which are produced by processes such as mechanical grinding, wind, resuspension of solid materials and agricultural activities (Kukkonen et al., 2005). Coarse particles can have biological substances such as fungi, pollen and endotoxins on their surfaces. These PMs can easily penetrate the lungs and usually get deposited in the upper and large airways of the respiratory tract via inhalation (Boogaard et al., 2010). Previous studies have only used $PM_{2.5}$ or PM_{10} as PM measurements, meaning the effects of particles of other sizes, especially $PM_{10\text{-}2.5}$, are not well understood (Pope III & Dockery, 2006). While $PM_{2.5}$ is more closely linked to adverse health effects than coarse particles (as $PM_{2.5}$ can reach inside the respiratory tract), PM_{10} has also been a subject of prime interest for epidemiological studies as it can cause serious human health issues (Behera et al., 2015). However, the health impacts of PM_{10} vary greatly depending on its composition (Gu et al., 2017), and its ability to travel and deposit in the human respiratory tract (Horemans et al., 2012) and their ability to increase its significant toxicity (Sarigiannis et al., 2017). Over the past decade, an increasing number of investigations have explored PM_{10} and its health-related effects (Clements et al., 2017; Solomon et al., 2012). Mortality (Giannini et al., 2017; Ortiz et al., 2017) and morbidity (Cohen et al., 2017; Requia et al., 2018) – particularly among elderly populations (Xia et al., 2017) and children (Chen,

Glonek, et al., 2016) – are the major health concerns linked to particulate air pollution. This mortality and morbidity are basically due to different diseases, such as cardiovascular disease (D'Souza et al., 2017), acute and chronic pulmonary emboli (Rojas-Bracho, Suh, & Koutrakis, 2000), asthma (Lopez-Villarrubia et al., 2016; Zhao et al., 2017), respiratory infectious diseases (Stafoggia et al., 2013), hypertension (Honda et al., 2017) and low birth weight (Ebisu, Berman, & Bell, 2016). The US-EPA has noted that coarse particles deposited in the upper airways are more relevant to asthmatic responses and irritation (U.S. EPA, 2015a). In fact, some studies around the world show that the relationship between PM_{10} and health impacts is far deeper and more complicated than originally thought. For example, in the USA, D'Souza et al. (2017) found a positive association between right ventricular dysfunction and coarse particle deposition among susceptible populations, including smokers and persons with emphysema. Time-series studies conducted in various places in Europe found evidence of an independent effect of PM_{10} as well as direct association with health impacts. However, the evidence was found to be stronger for fine particles in most urban areas (Brunekreef & Forsberg, 2005).

In the Australian context, the handful of topological studies suggest an association between coarse PMs and human health impacts. For example, Chen, Glonek, et al. (2016) showed that every increase of PM_{10} by 10 units increased by 8.3% the risk of an asthma hospital admission among children (0–17 years) during the cold season in Adelaide. A study conducted in Brisbane also found a 10 $\mu g/m^3$ increase of PM_{10} (especially in heavy traffic areas) was associated with a 4% increase in respiratory emergency hospital admissions (Chen, Mengersen, & Tong, 2007). Due to the accumulation of consistent findings linking PM_{10} with adverse health impacts, the United State Environmental Protection Agency (USEPA) set National Ambient Air Quality Standard (NAAQS) and Australia set the National Environmental Protection Measures (NEPM).

Overall, the majority of the research shows that PM_{10} exposure causes a small but significant increase in human morbidity and mortality (Anderson, Thundiyil, & Stolbach, 2012). Elemental composition analysis of PM_{10} is considered as a significant risk factor for an adverse health effect. This was beyond the scope of the study, however, we recommend the future study to consider this type of analysis.

2.2.1.2 Fine particulate matter

As mentioned above, fine particulate matter ($PM_{2.5}$) is defined as a particle with an aerodynamic diameter of 2.5 µm or less. It is inhalable particles or droplets and is considered a major air quality concern due to its adverse effect on human health. $PM_{2.5}$ is normally made up of sulphate and nitrate particles, organic and elemental carbon, and soil. However, the composition of the PM varies with season, place, weather and time. They can easily transport for hundreds (or thousands) of kilometres (Johansson, Norman, & Gidhagen, 2007) and persist for longer in the atmosphere (weeks or months) than PM_{10}. Due to their smaller size, they can easily penetrate deeper into the respiratory system and cause more hazards to human health (Dabass et al., 2018). For this reason, the WHO recommends using $PM_{2.5}$ rather than PM_{10} concentrations as air quality indicators (World Health Organization, 2006). The main sources of $PM_{2.5}$ are combustion from motor vehicles; the burning of coal, fuel oil and biomass; and road/soil dust. It consists mainly of crustal particles mechanically produced from agriculture, construction, mining, and road traffic, and particles of biological origin. Although significant evidence exists on the adverse effects of $PM_{2.5}$, it is challenging to quantify the contribution of each specific source or constituent to these adverse effects.

Due to rapid economic growth, along with industrial expansion and urbanisation, the occurrence of smog or haze episodes has increased in frequency over the past few decades. These episodes are characterised by high $PM_{2.5}$ levels and reduced visibility. For example, China has attracted considerable global attention to this issue. The drastic increase of its economic growth over the last 30 years has resulted in serious air pollution problems in many cities in China, mostly in metropolitan areas, including Beijing (Zhang, Zhang, & Xue, 2010), Shanghai (Zhou et al., 2016) and Guangzhou (Jihua et al., 2009). In the recent year, the high occurrence of $PM_{2.5}$ has not only increased the global concern due to its adverse health impacts but also has triggered their government to tackle the problems due to air quality.

Numerous epidemiological studies around the world have documented the association between exposure to ambient $PM_{2.5}$ and mortality (Kettunen et al., 2007; Maji et al., 2018). Globally, more than two million deaths are estimated to occur each year as a direct consequence of exposure

to air pollution due to damage to the respiratory system. Among these deaths, around 2.1 million are due to $PM_{2.5}$ and 0.47 million due to O_3 (Shah et al., 2013). For example, a study conducted in six cities of the United States found that a 10 μg/m^3 increase of $PM_{2.5}$ from mobile sources was associated with a 3.4% increase in daily mortality (Lin, Liu, et al., 2016). Similarly, the equivalent increase in $PM_{2.5}$ from coal combustion sources also accounted for a 1.1% increase in mortality; however, $PM_{2.5}$ generated from crustal particles was not associated with daily mortality. These results indicated that the particles generated from combustion from motors and the burning of coal are the major source of concern around the world (Lin, Liu, et al., 2016).

Therefore, including $PM_{2.5}$ as one of the six principal pollutants in National Ambient Air Quantity Standard (NAAQS) by USEPA in1997 (USEPA, 2009) and in National Environmental Protection (Ambient Air Quality) Measures by Australia in 1998 (Australian State of the Environment, 2016) was the best step taken for the improvement of public health protection, as many toxic elements are most concentrated in that particle mass size fraction. However, it is possible that varying levels of protection are still provided by that standard, depending on variations in local $PM_{2.5}$ composition and associated sources. Future standard-setting and emissions controls implementation may consider these differences, but the challenge remains to reduce the level of $PM_{2.5}$ concentrations to prevent human health impacts.

2.2.1.3 Ultrafine particulate matter

Ultrafine particulate (UFP) matter is in the nanoscale size particulate matter with an aerodynamic diameter equal to or less than 0.1 μm or 100 nm ($PM_{0.1}$) and consists of various suspended solids and liquid droplets. Generally, $PM_{0.1}$ is split into 'engineered nanoparticles' (ENPs) and other UFP matter.

Generally, ultrafine particulates have both natural and anthropogenic sources, including primary (directly emitted) and secondary formation from gas interaction (Bigazzi & Figliozzi, 2012). Hot volcanic lava, smoke and ocean spray are the major natural sources, whereas combustion reactions, emission from manufacturing and automobile sources are the main anthropogenic sources of $PM_{0.1}$. These particulates vary considerably in composition and particle number

concentration (PNC) over distance and time due to variation in the source of emissions, their formation process and meteorological influences. Higher UFP concentration typically occurs in urban areas near roadways, manufacturing industries, construction sites and so on. In most urban areas, peak UFP concentration is observed during morning rush hours, associated with motor vehicle emissions (Kim et al., 2002; Park et al., 2008).

Ultrafine particulates (including ENPs) are better able to move throughout the body as they can pass through biological membranes and cell walls. These particles are studied based on their number in the atmosphere, in contrast to regulated particles, such as $PM_{2.5}$ and PM_{10}, which are studied based on their mass. It is scientifically proven that the smaller the particle size, the larger the potential human health impacts. Ultrafine Particulates ($PM_{0.1}$) is even smaller than the cilia that help to filter out large particles from the nasal passages, meaning $PM_{0.1}$ can easily penetrate deep into the human body. In addition, due to its large surface-area-to-volume ratio, the particles can easily allow for the desorption of toxic components into human tissues (Chen, Hu, et al., 2016). The UFP contain trace metals such as cadmium, lead, zinc and bismuth, which can potentially reach the brain to cause brain damage (Health Effect Institue, 2013). The adverse health impacts of $PM_{0.1}$ are totally different from those of $PM_{2.5}$ and PM_{10}.

There is a range of potential human exposure activities, from occupational, manufacture and industrial processes to incidental by-product emissions where the exposed population at risk are children, elderly people, commuters and outdoor workers. The research on risk assessment of UFPs is still in the very early stages; currently, there is not enough research evidence on how to regulate UFPs and manage the associated health risks.

2.2.2 Gaseous air pollution

Hazardous chemicals escape to the environment in the form of gases via a number of natural and/or anthropogenic activities. These lead to gaseous air pollution that readily enters into the human respiratory system and may have adverse effects on human health (WHO, 2005). Gaseous air pollutants– such as carbon monoxide, oxide of nitrogen, sulphur dioxide and ozone– differ in

their chemical composition, emissions, reactions, disintegration and ability to diffuse over long or short distances. Exposure to these air pollutants has been associated with both acute and chronic effects on human health, affecting a number of different systems and organs (WHO, 2018). These effects range from upper respiratory tract irritations to chronic respiratory and heart diseases, including lung cancer, acute respiratory infections, asthmatic attacks and chronic bronchitis among children and adults. In addition, long- and short-term exposure to gaseous air pollutants have been linked to premature mortality and reduction in life expectancy. The sources and human health impact due to gaseous air pollutants are briefly discussed below.

2.2.2.1 Carbon monoxide

Carbon monoxide (CO) is an odourless, tasteless and colourless toxic contaminant in the atmosphere that is produced due to incomplete combustion of carbon-containing fuels, such as natural gas, coal, oil, gasoline and wood. Carbon monoxide is a particularly important contaminant to monitor because of its potent mammalian toxicity. In a global context, CO mixing ratios in the troposphere range from 40 to 200 ppb and have an average chemical lifetime of 30–90 days (Seinfeld & Pandis, 2016). The overall maximum concentration is observed during the local spring and the minimum occurs in later summer.

Human activities are mainly responsible for the introduction of increasing quantities of CO to the atmosphere. The major sources of CO are ethane oxidation (by OH), technological processes (combustion and industrial), biomass burning and the oxidation of non-methane hydrocarbons. Combustion is associated with the incomplete burning of fossil fuels containing carbon, such as gasoline, natural gas, oil, coal and wood. It is therefore estimated that about two-thirds of the CO comes from anthropogenic activities, including oxidation of anthropogenically derived CH_4 (Kim et al., 2017). In the US, vehicle exhaust contributes about 75% of all CO emissions, and up to 95% of all emissions are from cities (US Environmental Protection Agency, 2012). According to the US-EPA, the vehicular exhaust contains up to 100,000 ppm CO and levels can reach between 10 and 12 ppm within the passenger compartment of an automobile during heavy traffic. Even higher concentrations are encountered in semi-closed environments where continuous exposure to vehicular exhaust occurs, such as parking garages and narrow roads (US Environmental

Protection Agency, 2017). Thus, people who are in very vulnerable age groups, such as infants, children and the elderly, can be exposed to CO in a variety of commonly encountered environments.

Carbon monoxide intoxication continues to be one of the most common causes of morbidity and mortality due to poisoning (Burt et al., 2014). This gas is non-irritating and imperceptible in the air we breathe. Due to this, exposure to CO is often not recognised and acute CO toxicity is commonly misdiagnosed (Iqbal et al., 2012). However, the health risks associated with CO vary with its concentration and the duration of exposure. Some of these health effects include: headaches, oxidative stress, apoptosis, an increase in immune-mediated injury and risk of heart disease (Burt et al., 2014). This pollutant is dangerous to human health as even a small portion can prevent oxygen from being delivered through the body to major organs. In severe cases, it may even cause death (Chung, Lin, & Kao, 2015). Infants and foetuses are more susceptible to CO toxicity than adults due to their higher metabolic rates and the presence of foetal haemoglobin, which has a greater affinity for CO than adult haemoglobin (Kao & Nanagas, 2004).

To minimise CO-associated health risks, different countries have adopted their own acts and regulations. For example, as a result of the accumulation of visible dense smog that contained CO within many cities of the USA during the 1960s, the US government established the basis of the *Clean Air Act* in 1970 (US Environmental Protection Agency, 2012). Likewise, in 1999 Australia established the National Environmental Protection Measures (NEPM) with a focus on monitoring and reporting air quality in relation to the NEPM standards (National Environment Protection Council, 2011). Besides these acts and regulations, public education and awareness regarding the standards and their implementation are other options for further decreasing morbidity and mortality from CO poisoning. Some of this basic awareness includes professional maintenance of fuel heating systems, appropriate ventilation (including CO detectors) in workplaces, not idling motor vehicles in enclosed spaces, and so on. However, people should be aware of its emission during the day to day life and should be warned about the potential harm concentrated CO might cause to human health.

2.2.2.2 Oxides of nitrogen

Nitrogen dioxide (NO_2) and nitric oxide (NO) are referred to together as oxides of nitrogen (NO_x) (i.e. NOx=NO+ NO_2). NO_x also reacts with volatile organic compounds in the presence of sunlight to form smog, acid rain, fine particulate matters and ground-level O_3, all of which are associated with adverse health effects. NO_x gases are produced during the combustion of fossil fuels at high temperatures, such as by car engines, industrial furnaces and boilers. Once nitrogen gas released during fuel combustion, it then combines with oxygen atoms to create NO and further combines with oxygen to create NO_2. Under standard conditions, NO is a colourless gas and one of the principal oxides of nitrogen. NO_2 is also subject to extensive further atmospheric transformations that lead to the formation of photo-oxidants (including O_3) and other strong oxidants that participate in the conversion of NO_2 to nitric acid and the nitrate part of secondary inorganic aerosols. This is an unpleasant-smelling irritant gas that, at high concentrations, may cause inflammation in the respiratory airways (WHO, 2013).

Most of the NO_2 in cities comes from vehicle exhaust (80%). Only 1% of the total amount generates naturally in the atmosphere from lightning, plants, soil and water; the remaining NO_2 is generated from manufacturing industries and food processing (Department of the Environment and Energy, 2005c). Due to the photochemical reaction of NO_2, the newly generated pollutants are a major source of nitrate, sulphate and organic aerosols that can contribute significantly to total PM_{10} or $PM_{2.5}$ mass. Therefore, in 1971 NO_2 was considered in the first draft of the Environmental Protection Act as it was highly linked with a number of adverse health effects on the respiratory system.

In large cities with high levels of motor vehicle traffic, the amount of NO_x emitted into the atmosphere as air pollution can be significant. Figure 22 shows the contribution of NOx from motor vehicle and energy production in the EU during 2011 (European Union, 2013).

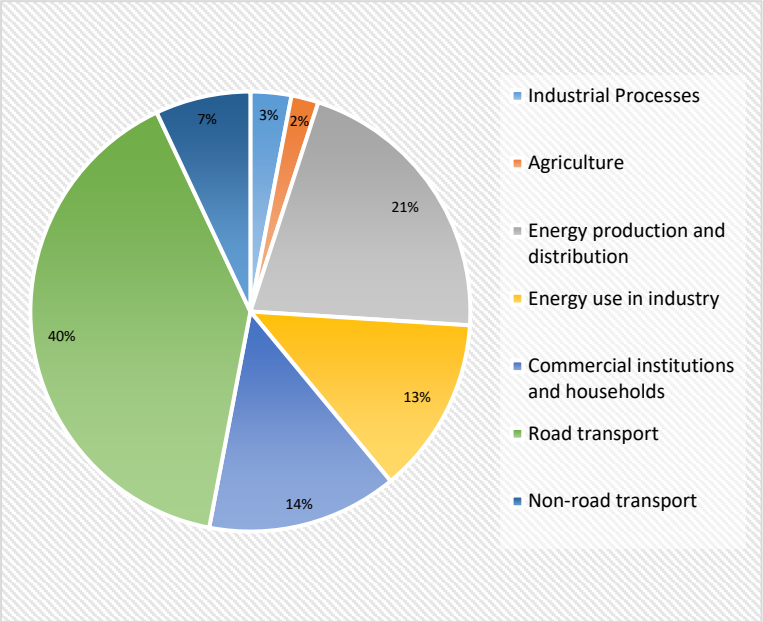


Figure 2.2: NOx emissions by sector group during 2011 (European Union, 2013)

In the Australian context, the major source of NO_x is the burning of fossil fuels such as coal, oil and gas. Most of the NO_2 in cities comes from vehicle exhaust (about 80%) and the remainder comes from petrol and metal refining, electricity generation from coal-fired power stations, manufacturing industries and food processing (Department of the Environment and Energy, 2005a). Since the 1990s, the concentration of NO_2 has sometimes increased for short periods, which does have an adverse health effect but is now thought to be acceptable for humans. However, since the implementation of the NEPM standard in 1998, air pollution authorities are monitoring levels to see if they are at an acceptable level. The current NEPM guideline value of NO_2 for a one-hour average period is 0.12 ppm (National Environment Protection Council, 2011).

NOx is very harmful to the ecosystem as well as human health as it can easily react with ammonia, moisture and other compounds to form nitric acid and related particles. These particles can easily penetrate deep into the lung tissue and can cause damage due to which in extreme cases, this can cause premature death. In some cases, the inhalation of such particles may cause or worsen respiratory diseases such as bronchitis and lung cancer (Esplugues et al., 2011; Grineski et al., 2011; Hamra et al., 2015).

To reduce such impacts among humans, guidelines have been set by the WHO and Australia. The main aim of these guidelines is to reduce the NOx concentration in the ambient air within the limit. The current WHO guideline values of NO_2 for a one-hour average period is 200 µg/m^3 and an annual average is 40 µg/m^3 (WHO, 2016).

2.2.2.3 Sulphur dioxide

Sulphur dioxide (SO_2) is a colourless gas that can be detected by taste and smell (it has a sharp, nasty smell) in the range of 1,000 to 3,000 µg/m^3. It is a predominant form of pollutant found in the lower atmosphere. This pollutant plays an important role in regional and global climate change. SO_2 has a short atmospheric lifetime and tends to concentrate on the source region, resulting in climatic effects with strong spatial and temporal variations (S.J. Smith et al., 2011).

The SO_2 in the atmosphere is emitted from both anthropogenic and natural sources. The world is developing rapidly, with a growing volume of economies resulting in an increase in commercial energy consumption over the past two decades (Arden P & Dockery, 2012). About 99% of the SO_2 produced from anthropogenic sources is due to the burning of fossil fuels in power plants and industrial facilities and from ore extraction and non-road equipment (Bentayeb et al., 2015; Department of the Environment and Energy, 2005b). The main natural source of SO_2 is volcanic activity; SO_2 is also found in a relatively high concentration in fossil fuels, which commonly contain 1–2% of sulphur by weight. The widespread of anthropogenic use of those concentrated fossils fuels has, therefore, greatly increased sulphur emissions into the atmosphere, which is substantially greater than natural emission on a global basis (Smith et al., 2011). SO_2 is also produced by motor vehicle emissions as the result of fuel combustion. These pollutants are linked with a number of adverse effects on human respiratory health (Kan et al., 2010).

The effects of air pollution on human health are complex as their individual effects vary from one person to another. The main health impacts of SO_2 are an irritation to the nose, throat, and airways, causing wheezing, coughing, a tight feeling around the chest and shortness of breath. The effects of SO_2 are felt very quickly and some people experience the worst symptoms within

few (10-15) minutes. SO_2 in the air can easily react with other substances to form sulphurous acid, sulphate particles or sulphuric acid, which can cause harm to the environment and health.

In Australia, the amount of SO_2 produced in the air is at acceptable levels in most cities and towns. The Australian government set a standard that resulted in SO_2 levels (the one-hour average period is 0.20 ppm, for a 24-hour period is 0.08 ppm and for a one-year period is 0.02 ppm) being decreased in the content of the fuel, reducing its emission level even further. Therefore, the SO_2 level in the air is not generally a problem in Australia. However, high concentration levels sometimes are found near chemical manufacturing industries, petrol refineries, mineral ore processing plant, power stations, and mining areas. It is, therefore, regular monitoring of the gaseous air pollution is required to prevent a human being from harmful diseases.

For example, Mt Isa and Kalgoorlie are the only areas where the concentrations of SO_2 in the air have been found to be higher than the NEPM guideline, but only occasionally (Department of the Environment and Energy, 2005b). Even though Australia experiences low levels of SO_2, the Australian government is alert to the issue and has taken steps to manage and reduce SO_2 concentration levels due to its significant adverse health effects. These steps include implementing national fuel quality standards, supporting the implementation of tighter vehicle emission standards and promoting alternative fuel. In 1999, the average sulphur content of diesel was 1300 ppm, which was reduced to 500 ppm in 2002 (Department of the Environment and Energy, 2005b). Australian state and territory governments have agreed on a NEPM for Ambient Air Quality through the National Environment Protection Council. The current NEPM guideline value of SO_2 for a one-hour average period is 0.20 ppm, for a 24-hour period is 0.08 ppm and for a one-year period is 0.02 ppm (Government of Western Australia, 2015).

2.3 Exposure to air pollution and its health impacts

Air pollution has become a global health problem and is considered as one of the most important environmental risk factors causing adverse health effects, especially for those living in urban areas. Growing evidence consistently demonstrates that living in an area with heavy vehicular

traffic plays a significant role in the risk of different kinds of diseases. Some of the human diseases associated with exposure to air pollution are explained below.

2.3.1 Respiratory health impacts

Over the past few decades, epidemiological research has confirmed that outdoor air pollution is a contributing cause of morbidity (Jacobs et al., 2017; Luong et al., 2016; Raaschou-Nielsen et al., 2016a) and mortality (Lelieveld et al., 2015c; Wheida et al., 2018). However, some effects may be related to short-term exposure (Mann et al., 2010; Su et al., 2016) and some due to long-term exposure (Beelen et al., 2014; Hoek et al., 2013). Recent evidence from several studies proved that air pollution contributes to the substantial worldwide burden of diseases, from acute lower respiratory tract infections to lung cancer (Jedrychowski et al., 1990; Mehta et al., 2013). There is some other evidence regarding the relationships between traffic pollution and adverse respiratory health outcomes, including asthma (Bowatte et al., 2018), wheezing (Brunst et al., 2015), decreased lung function (Rice et al., 2015), lung cancer (Beelen et al., 2008), increased lung inflammation markers (Kubesch et al., 2015), and increased medical visits and hospital admissions (Samoli et al., 2016). The diseases associated with air pollution and respiratory health are described below.

2.3.1.1 Asthma

Asthma has captured great attention for several years due to its increasing prevalence. Many urban areas in the developing world are undergoing rapid population growth accompanied by increasing outdoor air pollution through urbanisation and industrialisation. The presence of airborne pollutants in the ambient air has been identified as the primary cause of the allergic symptom of asthma (Rosenlund et al., 2009). There is increasingly convincing evidence of air pollution being associated with many signs of asthma worsening, such as pulmonary decrement (Hong et al., 2010; Liu et al., 2009), increased bronchial hyper-responsiveness symptoms (Andersen et al., 2008), increased anti-asthmatic medication use (Gent et al., 2003) and emergency visits (Andersen et al., 2008). The main symptoms of asthma include chest tightening,

coughing, wheezing and shortness of breath (Guarnieri & Balmes). Air pollutants – such as PMs, NO_2, CO_2 and O_3 – have been extensively studied and found to play an important role in causing asthma (Jang, 2012). The inhalation of NO_2 is injurious to the lungs as it can augment the degree of allergic airway inflammation and prolong allergen-induced airways in asthma (Pénard-Morand et al., 2010). Likewise, inhaling SO_2 is extremely sensitive for allergic asthma (Deger et al., 2012). As discussed earlier, PM can easily penetrate deep into the lungs, which increases the frequency and severity of asthma attacks (Nastos et al., 2010). This penetration can worsen bronchitis and other lung diseases by reducing the body's ability to fight infections (Trasande & Thurston, 2005).

In Australia, asthma is a common condition – affecting one in ten Australians – and it is considered as one of nine national health priority areas (Australian Bureau of Statistics, 2012). Australia has a high prevalence of asthma among children, which has increased since the 1980s (Orellano et al., 2017). The most common sources of air pollution – which is associated with asthma in Australia – are emissions from motor vehicles (Clare Walter, 2019), industrial production (Xue-yan Zheng, 2015), home heating sources and bushfires (Liu et al., 2015). In addition, drought-related dust storms are another source: these occur occasionally but produce extreme pollutants (Department of the Environment and Heritage, 2004). Moreover, the exposure to aeroallergens, such as pollens, combined with extreme weather events is also reported as one of the major triggers of asthma exacerbations in Australia (Murray et al., 2006). For example, epidemic thunderstorm asthma events are highly triggered by an uncommon combination of grass pollen levels and a particular type of thunderstorm that results in asthma symptoms developing in large numbers of people over a short period of time (Reddel et al., 2015). In several studies around the world, traffic-related air pollution has been shown to play a major role in developing asthma, allergies and respiratory infections, which is one of the major health issues facing children (Brandt et al., 2015; Hoek et al., 2012), adults (Cesaroni et al., 2008; Meng et al., 2007) and elderly people (Andersen et al., 2012; Halonen et al., 2009). However, in some studies, there were controversial results regarding the role of air pollution in the development of new-onset asthma, and the contribution of the risk factor remains unclear (Sarnat & Holguin, 2007). For example, although increasing evidence indicates that living near heavy traffic areas is associated with an increased rate of asthma, some well-designed studies

have found either no or very weak associations (Heinrich & Wichmann, 2004; Oftedal et al., 2009). This inconsistency may be due to an incomplete assessment of exposure to traffic-related air pollution. Therefore, further studies are needed to improve exposure estimates by measuring traffic-related air pollutants near participants' residences, schools and community places and identify their time/activities pattern in prediction models. Also, further research is required that can investigate the differential impact of traffic by genetic and other susceptibility factors, and to identify specific pollutants that underlie the adverse effect of traffic on asthma.

2.3.1.2 Lung cancer

Lung cancer is one of the most common human cancers. According to the WHO (2012), there were approximately 3.7 million deaths attributable to environmental air pollution. Among these deaths, 88% were attributed to different diseases in low- or middle-income countries, and the rest were due to lung cancer. In terms of estimated patterns of cancer incidence, in 2012, among 40 European countries, lung cancer was found to be the fourth most common type of cancer. As per this research, among 410,000 new cases, 353,000 deaths due to pollution-related lung cancer were recorded.

Although cigarette smoking is the biggest factor in the development of lung cancer, around 10–25% of lung cancer develops in those who have never smoked (Ghazipura, Garshick, & Cromar, 2019). Numerous recent studies have proved that the overall incidence of lung cancer within non-smoking populations is increasing (Gazdar & Zhou, 2018) due to various risk factors, such as occupational exposures to poor-air quality (Shankar et al., 2019), residential radon (Lorenzo-González et al., 2019) and socioeconomic position (Cao & Chen, 2019). Though there is some conflicting evidence about lung cancer rates in non-smokers, it is important to understand the risk factors other than smoking that contribute to lung cancer.

A study conducted by the International Agency for Research on Cancer (IARC) found strong evidence of an association between diesel engine exhaust (especially particulate matter) and the risk of lung cancer (WHO, 2012). Therefore, the IARC classified diesel engine exhaust as carcinogenic to human health. Similarly, several toxicological studies found long-term exposure

to particulate matter from traffic-related air pollution to be associated with the incidence of high risks for lung cancer (Puett et al., 2014; Zhang, Li, & Gao, 2017), mainly in non-smokers (Yorifuji et al., 2010) and people who have never smoked (Turner et al., 2011). For example, the WHO has estimated that long-term exposure to $PM_{2.5}$ is responsible for approximately 5% of all cancers, including bronchus, trachea, and lung (Ezzati et al., 2004). Likewise, the follow-up of the Harvard Six Cities cohort study (Laden et al., 2006b) and American Cancer Society Cohort study (Pope III et al., 2002) also found that $PM_{2.5}$-related air pollution is a predictor of lung cancer and mortality. Several other studies have investigated the association between lung cancer and not only particulate matters but also other selected gases, such as sulphur dioxide (Beeson, Abbey, & Knutsen, 1998) and nitrogen dioxide (Vineis et al., 2006). These studies also found an association between elevated long-term exposures to an ambient concentration of selected gases with the incidence of lung cancer.

In the case of Australia, lung cancer is a leading cause of death and is ranked fifth among the most common cancers diagnosed (MacKenzie et al., 2008). This cancer is responsible for almost one in five cancer deaths in Australia. In 2010, around 81% of lung cancers in Australia were due to smoking tobacco; the remainder were due to different causes (Cancer Australia, 2011). The exact contribution of air pollution to lung cancer is not well studied in Australia. However, in 2003, long-term exposure to urban air pollution was estimated to be the cause of 351 lung cancer deaths Nationwide (Begg et al., 2007).

Assessment of exposure to traffic-related air pollution is therefore important due to the widespread presence of its emissions in the environment and its toxicological relevance, both of which need to be addressed immediately.

2.3.1.3 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD), is a chronic inflammatory disease of the airways, pulmonary vessels and lung parenchyma. It is anticipated to be ranked fifth among the conditions with a high burden on society and third among the most important causes of death for both genders worldwide by 2020 (Ling & van Eeden, 2009; Salvi & Barnes, 2009). Cigarette smoking is

one of the leading cause of COPD, but this does not explain its increasing prevalence worldwide (Peeler, 2019). It is therefore important to understand the actual environmental factors that are contributing to this burden (Salvi & Barnes, 2009).

With the rapid urbanisation of the world's population, understanding the harmful effects of exposure to urban air pollution, especially from traffic, is an important issue for the urban planner (Anderson et al., 1997; Ghanbari Ghozikali et al., 2016; Ko & Hui, 2012). Numerous epidemiological studies have suggested that there is an association between air pollutants and different diseases, such as airway hyper-responsiveness, asthma and respiratory infections, which are important determinants of COPD (de Marco et al., 2011). Damage to the lung tissue by air pollutants is due to a local and secondary inflammatory response (Hogg & Van Eeden, 2009). The repeated inhalation of pollutants is believed to be central to the effects of long-term exposure and to the chronic and progressive nature of COPD (Ling & van Eeden, 2009). There is clear evidence that long-term exposure to a higher density of traffic-related air pollution is significantly associated with COPD, resulting in increased morbidly and mortality (Andersen et al., 2011). For example, a study conducted in 10 cities in the USA found a strong association between long-term exposure to PM_{10} and an increasing number of COPD patients in hospital (Zanobetti, Schwartz, & Dockery, 2000). It was observed that for every 10 $\mu g/m^3$ increment of PM_{10}, there was an increase in hospital admissions of 2.5% due to COPD among the elderly population (older than 65 years). Similarly, a study conducted in six European cities (London, Paris, Amsterdam, Barcelona, Milan and Rotterdam) also found an association between air pollution and increased COPD hospital admissions (Anderson et al., 1997). Comparison between women living close (<100 m) and further away from busy roads found that the chances of developing COPD were 1.79 times higher (95% CI: 1.06–3.02) among those living close than those who live further away from busy roads(Schikowski et al., 2010). The subsequent follow-up of this study with lung function assessments among the subgroup of elderly women, conducted in 2008–2009, found a decrease in COPD with improvement in air quality (Schikowski et al., 2010). However, there are a few studies that have conflicting results; therefore, a causal relationship between outdoor air pollution and COPD cannot be verified. For example, a study from Nottingham in the UK found no significant cross-sectional association between people living

in close proximity to heavy traffic routes and COPD (Pujades-Rodriguez et al., 2009). In some studies, the relationship between outdoor air pollution and its health effects were grouped on COPD instead of asthma (Halonen et al., 2008). However, in spite of some conflicting results, it appears that ambient air pollutants are significant triggers for exacerbating COPD, from increasing its symptoms to hospital admission and even sometimes mortality (Ko & Hui, 2012; Schikowski et al., 2010).

In the case of Australia, there were 4,761 deaths attributed to COPD in 2006 (Gall, Krysiak, & Prescott, 2010) and 4,207 in 2003 (Begg et al., 2007). Although COPD is attributed predominantly to smoking tobacco, exposure to air pollution is also a suspected risk factor for COPD (Trupin et al., 2003) in Australia. Data on the prevalence of COPD due to air pollution is limited; however, some epidemiological studies have found an association between them (Moore et al., 2016). For example, according to Trupin et al. (2003), one in five cases of COPD was estimated to be attributable to air pollution such as PM, CO, and NO_2 exposure. Similarly, Simpson et al. (2005) also found a significant association between air pollution and hospital admission due to COPD. However, more research is needed in Australia to identify the actual cause and manage COPD diseases worldwide. In addition, continuing efforts are needed to prevent the disease by avoiding smoking and improving ambient and occupational air quality.

2.3.2 Cardiovascular disease

Cardiovascular disease (CVD) is the number one cause of death in developed countries and is the leading cause of loss of life due to morbidity and mortality (Naghavi et al., 2017). The main causes of CVD are genetics (Nikpay et al., 2015), smoking (Burns, 2003) and exposure to air pollution (McGuinn et al., 2019). Over the past few decades, the epidemiological and clinical evidence has confirmed the great concern about ambient air pollution (Brook, Rajagopalan, Pope, Brook, Bhatnagar, Roux, et al., 2010) and its attendant mortality and morbidity related to cardiovascular diseases (Goldberg, Burnett, & Stieb, 2003). Globally, around 92% of the population lives in places where the guidelines for air pollution established by WHO are not met (World Health Organization, 2016). As a result, exposure to air pollution is responsible for an estimated 3.3

million deaths: >2.1 million from ischemic heart disease, >1.1 million from stroke and 17 million deaths from cardiovascular disease worldwide (Forouzanfar et al., 2016; Institue for Health Metrics and Evaluation, 2016).

Accumulating epidemiological studies have shown that acute exposure to particulate matters from traffic-related air pollutants is significantly associated with increased CVD (Cohen et al., 2017; Hart et al., 2014). Particulate matter, especially fine and ultrafine particles, plays the most important role in heightening the risk of CVD through different mechanisms, including inflammation and systemic oxidative stress. These mechanisms could drive atherosclerosis progression and other long-term effects as well as serve as triggers of the event through changes in vascular function, autonomic balance and plague stability (Brook, Rajagopalan, Pope, Brook, Bhatnagar, Roux, et al., 2010; McGuinn et al., 2019). For example, Hart et al. (2014) conducted a large prospective study among women and found 39% who lived within 50 m of a major roadway to have an increased risk of sudden cardiac death compared with women living 500 m away or further. It is also suggested that the long-term effects of fine and ultrafine particulate matter have a greater impact on cardiovascular mortality compared with the short-term effects (Newby et al., 2014). The long-term exposure studies have investigated the cardiovascular effects of air pollution using annual variations in pollutant concentration, whereas short-term exposure studies have investigated the effects using hourly or daily variations. However, there are still limited data regarding the long-term effects of air pollution in relation to cardiovascular diseases (Lipsett et al., 2011).

In Australia, CVD is a major cause of death, to which 43,477 deaths were attributed in 2017 – it kills one Australian every 12 minutes (Australian Bureau of Statistics, 2018a). Despite improvements over the past few decades, it is still one of Australia's biggest health problems, not only impacting the population but also creating a significant burden on the economy. Many factors contribute to CVD in Australia, including smoking tobacco, environmental factors, insufficient physical activity, poor diet, excessive alcohol consumption and genetic factors (Heart Foundation, 2014). Besides these risk factors, numerous studies have investigated the growing evidence of the association between air pollution and CVD (Wilmot et al., 2012). For example, according to a study conducted by Hansen et al. (2012), despite the relatively low concentration

of air pollution in Australian cities, PM concentrations are found to be highly associated with the increase in morbidity due to CVD in Adelaide. This increasing morbidity in Adelaide is attributed to the high concentrations of PM_{10} and $PM_{2.5}$ in the ambient air during the cold season. However, the effects of $PM_{2.5}$ were found to be higher compared to PM_{10}. Similarly, a study conducted by Barnett et al. (2006) also found an association between the emission of air pollution such as CO, NO_2 and PM with adult cardiovascular hospital admissions, especially among the elderly, at pollution concentrations below the normal health guidelines. However, the available data regarding the link between air pollution and CVD all over Australia is limited. Therefore, further research is needed to determine the strong evidence of the association between ambient air pollutant and CVD and reduce the number of health impacts related to cardiovascular disease.

2.4 International air quality guidelines

The main purpose of managing air quality is to protect public health and the environment from the adverse effects of air pollutants. The WHO has established air quality guidelines that aim to provide a uniform scientific basis for understanding the effects of air pollution on human health (WHO, 2005). These guidelines address all regions of the world to provide uniform targets for air quality that would protect the large majority of individuals from adverse health effects.

Air quality management consists of different strategies, including risk assessment, setting air quality and emission standards, monitoring and enforcement, implementation of control measures and risk communication. Normally, these strategies are set by each country to protect the health of their citizens, which is an important component of national risk management and environmental policies. The use of air quality standards, however, has become the cornerstone of air quality management. The role of this management, adopted and enforced by regulatory authorities, is to define the level of acceptable air pollution for a country or region (WHO, 2005). WHO guidelines have recommendations and suggests that each country should simply adopt them as standards. However, previous epidemiological evidence shows that even if a country meets the standards, there is still the possibility of adverse effects. Because of this, the national standards vary from country to country according to the approach adopted for minimising health risks, technical feasibility, economic status, cultural conditions encountered in a given location

and various other political and social factors. The WHO acknowledges that when formulating policy and targets, a government should always consider their own local circumstances very carefully before adopting the International guidelines directly as a legally based standard.

The updated WHO standards for air pollutants in 2005 are shown in Table 2.1 below:

Table 2. 1: WHO standard for ambient air pollutants, 2005

Pollutant	Concentration	Average
$PM_{2.5}$	10 µg/m^3	Annual Mean
	25 µg/m^3	24-hour Mean
PM_{10}	20 µg/m^3	Annual Mean
	50 µg/m^3	24-hour Mean
O_3	100 µg/m^3	8-hour Mean
NO_2	40 µg/m^3	Annual Mean
	200 µg/m^3	1-hour Mean
SO_2	20 µg/m^3	24-hour Mean
	500 µg/m^3	10-minute Mean

(WHO, 2005)

Further, WHO periodically reviews the accumulated scientific evidence to update their guidelines on air quality. The most recent update was completed by WHO Europe in 2005 (World Health Organisation Europe, 2013).

Similarly, the United States Environmental Protection Agency has set National Ambient Air Quality Standards for six principal pollutants which are called as “criteria air pollutants”. Units of measure for the standards are parts per millions (ppm) by volume, parts per billion (ppb) by volume, and micrograms per cubic meter of air (µg/m^3). These standards are also periodically reviewed and are listed below:

Table 2. 2: National Ambient Air Quality Standards for six principle pollutants

Pollutants		Primary/Secondary	Averaging Time	Level	Form
Carbon Monoxide		Primary	8 hours	9 ppm	Not to be exceeded more than once per year
			1 hour	35 ppm	
Lead		Primary and Secondary	Rolling 3 month average	0.15 µg/m^3	Not to be exceeded
Nitrogen Dioxide		Primary	1 hour	100 ppb	98th percentile of 1-hour daily maximum concentrations, averaged over 3 years
		Primary and Secondary	1 year	53 ppb	Annual Mean
Ozone		Primary and Secondary	8 hours	0.070 ppm	Annual fourth-highest daily maximum 8-hours concentration, averaged over 3 years
Particle Pollution	$PM_{2.5}$	Primary	1 year	12.0 µg/m^3	Annual mean, averaged over 3 years
		Secondary	1 year	15.0 µg/m^3	Annual mean, averaged over 3 years
	PM_{10}	Primary and Secondary	24 hours	35 µg/m^3	Not to be exceeded more than once per year on average over 3 years
Sulfur Dioxide		Primary	1 hour	75 ppb	98th percentile of 1-hour daily maximum concentrations, averaged over 3 years
		Secondary	3 hours	0.5 ppm	Not to be exceeded more than once per year

(Source: United States Environmental Protection Agency)

These guidelines are an important tool for worldwide use and are intended to support actions that aim for the optimal achievable level of air quality in order to protect public health in different contexts. In addition, air quality standards are an important instrument of risk management and environmental policy and should be set by each country to protect the health of its citizens.

2.5 Ambient air quality in Australia

2.5.1 Introduction

Although air quality in Australian cities is considered better than other cities around the world, air pollution is still a major concern as it been estimated that urban air pollution contributes approximately 1% to the total burden of diseases in Australia (Xia et al., 2015). Poor air quality has a major impact on the environment, health and the economy. It impacts not only the visual environment but also the quality of life. As explained above (section 2.3), more than two million Australians are suffering from asthma and hundreds of thousands are affected by other respiratory disorders that can be worsened by air pollution (Reddel et al., 2015; Woolcock et al., 2001). Ambient air quality in Australia is mostly affected by increasing human activity and climate change. The population of Australia continues to grow, with increasing urban density and expanding community boundaries. As a consequence, ambient air quality in Australia is significantly affected by human activities such as transportation, energy consumption and resource use. The size of an urban centre and the presence of major industrial facilities significantly affect air quality. While emissions put ongoing pressure on air quality, some of this pressure is reduced by improving infrastructure and equipment; for example, cleaner vehicles, cleaner energy – including renewable energy – and cleaner industrial and commercial operations.

Since 1991, particulates and O_3 have been the air pollutants of concern in Australia, with peak concentrations at or above national air quality standards. This pollution shows no consistent downward trend in major cities, such as Sydney and Melbourne (Department of the Environment and Energy, 2004). The Australian Government plays a significant role in supporting the states and territories to reduce the concentrations of particulate matter and O_3 levels. Air toxics (also called 'hazardous air pollutants') originate from different sources, such as industrial emissions, motor vehicle emissions, solid fuel combustion and materials such as paints and adhesives in new buildings. This toxic air has the potential to cause serious harm to humans and/or the

environment. To assess the risks posed by these pollutants, all Australian governments have been working together. In June 2003, the Australian Government established an air toxins program to focus attention on this area. After recognising the potential health impacts, Australia has set up national benchmarks for five priority air toxins in ambient air within the National Environment Protection Measure (NEPM). More about the NEPM is described below.

2.5.2 National Environmental Protection Measures

A national environment protection measure (NEPM) is legislation designed to protect particular aspects of the environment in a consistent way across state, territory and Commonwealth Jurisdictions in Australia. The NEPM sets national standards for the six key air pollutants (CO, SO_2, O_3, NO_2, Pb and PMs) to which Australians are exposed and provides a nationally consistent framework for the monitoring and reporting of those pollutants. As a statutory entity within the Environment Protection and Heritage Council (EPHC), the overall goal of the NEPM is to attain ambient air quality that allows for the adequate protection of human health and wellbeing (NEPC, 2010). The standards are legally binding on each level of Government in all states. Here, the NEPM itself does not comply or direct pollution control measures, it requires participating jurisdictions, such as commonwealth, states and territories, to undertake reporting and monitoring activities and provide data to assist jurisdictions in formulating air quality policies.

At the national level, the Australian Government's Council related to the environment meet regularly to discuss and deal with common concerns, including air quality. These ministerial meetings provide a forum for agreement on priorities and resources for the improvement of the NEPM standard, its related programs and policies, and for developing related studies. For more than a decade, Australia has been following the national standards and their goal for the betterment of ambient air quality. These national standards, policies and programs were developed based on strong empirical evidence about the health impacts of major pollutants. The standards are protected by law and are regularly monitored in all major cities, with results reported publicly. The achievement of 10 years' air quality goals of the NEPM depends upon the action taken by the state and territory governments to control point and non-point sources of

air pollution. Although the achievement of the goals of the NEPM is in the hands of the states and territories, the system of public reporting allows members and interest groups to put pressure on governments and regulators if the activities or progress regarding air quality improvement slow down. The updated NEPM standards for air pollutants in 2010 are shown in Table 2.3:

Table 2. 3: Updated NEPM standards for air pollutants (EPHC, 2010)

Pollutant	**Averaging period**	**Maximum (ambient) concentration**	**Goal within 10 years (maximum allowable exceedances)**
CO	8 hours	9.0 ppm	1 day a year
NO_2	1 hour	0.12 ppm	1 day a year
	1 year	0.03 ppm	None
Photochemical Oxidant	1 hour	0.10 ppm	1 day a year
	4 hours	0.08 ppm	1 day a year
SO_2	1 hour	0.20 ppm	1 day a year
	1 day	0.08 ppm	1 day a year
	1 year	0.02 ppm	None
Pb	1 year	0.50 µg/m^3	None
PM_{10}	1 day	50 µg/m^3	5 days a year
PM2.5	1 day	25 µg/m^3	Goal is to gather sufficient data nationally to facilitate a review of the standard as part of the review of this measure scheduled to commence in 2005.
	1 year	8 µg/m^3	

2.5.3 National Clean Air Agreement

On 15 December 2015, the 'National Clean Air Agreement' was established by the Australian state and federal environmental ministers to ensure clean and healthy air in the future. This helps governments to prioritise national action and address air quality issues. The Agreement seeks to ensure that the community continues to enjoy clean air, and addresses the impacts on human health and the environment if they arise (Department of Environment and Energy, 2015). Also, a key focus of this agreement is to encourage the development of partnerships with the business

and non-government sectors to achieve and sustain improved air quality outcomes. The Agreement establishes a basis of priority for action now and into the future and provides a means to develop practical, cost-effective and outcome-focused measures to address all the issues prioritised.

To achieve this, the Agreement requires reliable data and information to support the decision-making process and to satisfy the public need. The Agreement's work plan clearly outlines the priority actions, roles and responsibilities of different levels of government and the timeframes for implementing agreed actions. This work plan is reviewed every two years to maintain accountability and ensure its continued relevance.

All states and territories have their own primary responsibility for monitoring and managing air quality in their jurisdictions. Also, they have their own legislation and strategies in place to manage air quality, including point source emissions from a particular industry or facility where local circumstances play a key role. They monitor air quality under the same NEPM standard that protects public health and they report to the Australian government. Not only the government but also businesses and the community need to be active to ensure a clean air future. If any issues arise, such as vehicle and fuel quality, the Australian Government plays a lead role in managing it. In addition, the Australian Government is able to take a national approach that has been recognised by all other governments. The National Clean Air Agreement further seeks to foster continued coordinated effort between governments to ensure practical, efficient and effective responses to air quality issues are prioritised under the agreement (Department of Environment and Energy, 2015).

2.5.4 Air quality in Perth

2.5.4.1 Perth geographic and demographic information

Perth is the capital city of Western Australia and Australia's fourth-most populous city. It is considered to be one of the most isolated cities in the world. According to the 2016 census, the city had an estimated population of 2.14 million people, which accounts for 8.58% of the national

population (Population Australia, 2019). Also, looking back over the last eight years of Perth's population, Perth has the fastest population growth rate ranging from 9.58% (2012) to 0.99% (2015). The first settlement area in Perth was on the Swan River with the city's Central Business District and port (Fremantle). The population of the city increased substantially as a result of rising Western Australia gold mining in the late 19th century. The central business district of Perth is surrounded by the Swan River to the south and east, Kings Park on the western end, and the northern border is covered with railway reserve. Perth is part of the South West Land Division of Western Australia with the majority of the metropolitan area of Perth located on the Swan Coastal Plain. The density of the city increased by 7.5 people per square km and reached 315 people per square km as of the year 2014.

Data on Perth's population rise from 2012–2017 are shown in Table 2.3 below:

Table 2. 4: Population of Perth, Australia from 2012-2017

Year	Population in Million	**Growth Rate (%)**
2012	1.83	9.5
2013	1.97	7.6
2014	2.02	2.5
2015	2.04	0.9
2016	2.14	4.9
2017	2.20	2.8

Source: (Population Australia, 2018)

The majority of Perth's annual rainfall occurs during the colder seasons, generally between May and September. Perth is an example of a 'hot-summer Mediterranean climate' (Geerts & Linacre, 2002). The winters are relatively cold and wet, and the lowest temperature recorded in Perth is -0.7^{0}C on 17 June 2006. However, the temperature reaching below zero is a very rare occurrence. Summers are generally hot and dry with temperatures ranging from 30–40^{0}C from December to March. February is usually the hottest month of the year. The highest temperature recorded in Perth is 46.2^{0}C on 23 February 1991 (Bureau of Meteorology, 2012). According to the CSIRO, average daily temperatures greater than 16^{0}C are considered as a warm season and less than 12^{0}C as a cold season (Penman, Lemckert, & Mahony, 2006). Summers are generally dry but

sometimes sporadic rainfall occurs in the form of short-lived thunderstorms, which also cause heavy rainfall in the north-west of Western Australia. The sea breeze, locally known as the 'Fremantle Doctor' blows from the southwest most summer afternoons and provides much relief from the hot north-easterly winds. Due to this wind, the temperature often falls below 30^0C a few hours later. According to the Bureau of Meteorology, Perth has an average of 8.8 hours of sunshine per day – 3200 hours of annual sunshine – making it the sunniest capital city in Australia (Bureau of Meteorology, 2012). Since the 1970s, the rainfall pattern has changed in Perth as well as in southwest Western Australia. There has been a reduction in winter rainfall with a greater number of extreme rainfall events happening in the summer months (Indian Ocean Climate Initiative, 2009).

The population and the meteorological conditions play a significant role in increasing or decreasing the concentration of ambient air pollution in the Perth metropolitan area.

2.5.4.2 Air pollution trends in Perth

In Western Australia, the Department of Water and Environmental Regulation (DWER) has the role of protecting and maintaining the quality of air following NEPM. DWER is responsible for providing technical, strategic and policy advice on air-quality-related problems, such as poor

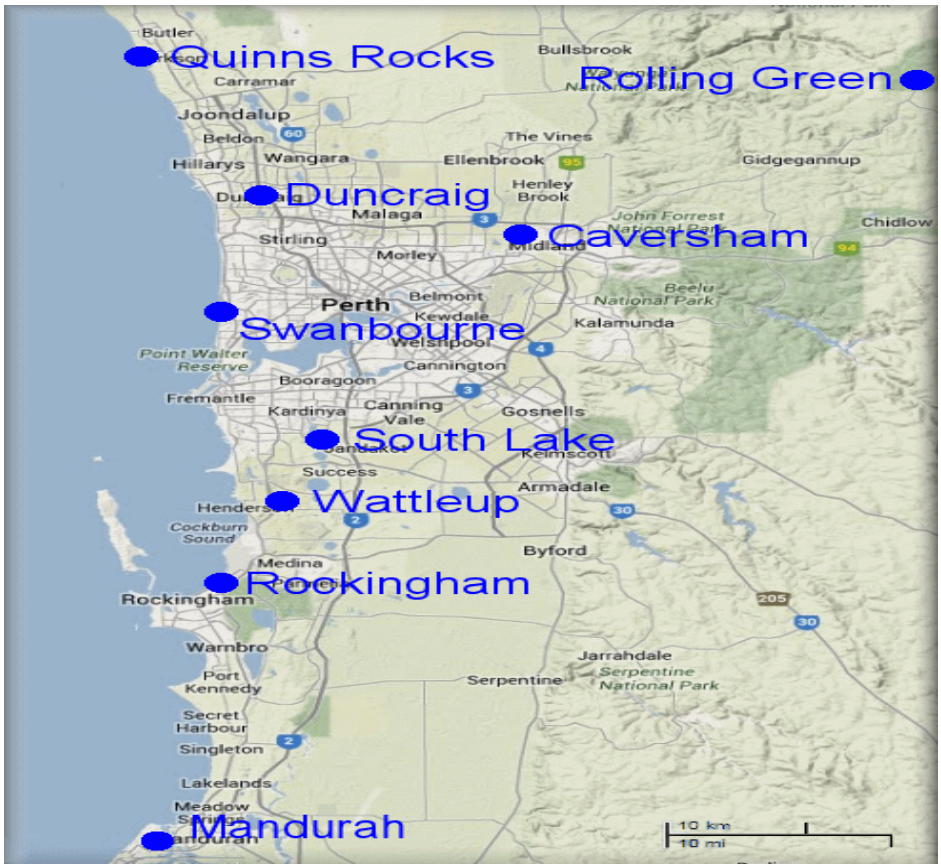


Figure 2.3: Department of Water and Environment Regulation air quality monitoring sites in Perth, WA 2016

ambient air quality, emissions from industries, odour modelling, air toxicity and health standards (Department of Water and Environmental Regulation, 2018). Around 13 different air quality monitoring sites are set up and operated by DWER in Western Australia, among which eight sites are within the Perth metropolitan region: Caversham (CA), South Lake (SL), Duncraig (DU), Swanbourne (SW), Quinns Rocks (QR), Rolling Green (RG), Wattleup (WT) and Rockingham (RO) Figure 2.3). The more detail information about these monitoring sites are described in the table below:

Table 2. 5: The detail information on air quality monitoring sites in Perth

Site Name	Location	Population Pattern	Type of pollution monitored	Source of pollution	Pollution Level
Caversham (CA)	Semi-rural area northeast of the Perth CBD	Low population density	CO, NO, O_3, PM_{10}, $PM_{2.5}$, SO_2,	Brick manufacturing industries, vineyards	High O_3 in 2015, PM_{10} and $PM_{2.5}$ occasionally exceeded NEPM standard
South Lake (SL)	Close to Kwinana industrial area	Growing population	CO, NO, O_3, SO_2, PM_{10}, $PM_{2.5}$	Wood fires, industries	High CO in 2016, low SO_2 in 2017, PM_{10} and $PM_{2.5}$ occasionally exceeded NEPM standard
Duncraig (DU)	Approx. 200 m west of Mitchel Freeway	Dense population	CO, NO, PM_{10}, $PM_{2.5}$	Vehicles, domestic wood fires, domestic wood heater, heavy machine working in the vicinity of monitoring site	PM_{10} and $PM_{2.5}$ occasionally exceeded NEPM standard, high PM_{10} level in 2015 and 2017
Swanbourne (SW)	Western coastal suburb of Perth	Dense population	NO, O_3, PM_{10},	Wood fires, vehicles	PM_{10} occasionally exceeded NEPM standard
Quinns Rock (QR)	Outer coastal suburb of Perth	Moderate population density	NO, O_3, $PM_{2.5}$	Vehicles, wood fires	$PM_{2.5}$ occasionally exceeded NEPM standard
Rolling Green (RG)	Outer east rural suburb of Perth	Low population density	NO, O_3	Vehicles, domestic wood fires	High O_3 in 2015
Wattleup (WT)	South metropolitan site, 25 km south of Perth	Growing population	SO_2	Kwinana Industrial Area.	Low SO_2 in 2017
Rockingham (RO)	South coastal site, 35 km south of Perth	Moderate to dense population	NO, O_3, SO_2	Woodfire, vehicles	High NO in 2016, low in SO_2 in 20178

Source: Department of Water and Environment Regulation air quality monitoring sites in Perth, WA 2016

2.5.5 Air quality in other states of Australia

In New South Wales (NSW), air quality is monitored by the Department of Planning, Industry and Environment. Generally, air quality in NSW remained good during 2018 for much of the time based on the information collected from the 43 stations of the air quality network (NSW Government, 2018). However, some areas, such as the southwest and northwest of Sydney, experience poor air quality due to ozone and particulate matters in 2017 (New South Wales Government, 2017). Regional dust storms and bushfires, along with human activities (such as motor vehicles, mining, industry, and power generation and residential wood heaters as well as non-road vehicles and equipment), contribute particle concentration and ozone. These sometimes cause poor air quality across NSW that exceeds the national air quality standards. In Sydney, suburbs such as Rozelle, Liverpool, Chullora, Campbelltown West, Earlwood and Prospect also experienced a spike in air pollution during 2015, with pollution levels climbing above the national standards (NSW Environmental Protection Authority, 2015; NSW Government, 2018). Similarly, the Hunter Valley in Newcastle was also found to have a higher level of $PM_{2.5}$ than the recommended Australia levels in 2015 (NSW Environmental Protection Authority, 2015). The large concentration of these particles in the Hunter Valley is due to coal mining, the stockpiling of fuel and its transport on uncovered rail wagons to Stockton and Newcastle. Similarly, Wagga Wagga, which is a major regional city in the Riverine region of NSW, was also found to have the highest concentration of PM_{10} over a day-long reading in 2015 that was almost triple the national standard of 50 μg/m3 (New South Wales Government, 2017). These outcomes were due to agricultural activities. However, since the 1980s the air quality in NSW has improved with the reduction of ambient NO_2, CO, SO_2, and lead levels.

In Victoria, air quality in Melbourne, Geelong and the Latrobe Valley region is generally good. However, Brooklyn (a suburb of Melbourne), sometimes experiences poor air quality due to local sources of air pollution, such as industries, bushfires and planned burns (Environment Protection Authority Victoria, 2018). Bushfires, the Hazelwood mine fire, prolonged drought conditions and dust events had a major impact on Victoria's air quality during 2003 and 2006–2009. The air quality of the Port Phillip Region during this year was associated with particles and O_3 whereas

the Latrobe Valley was associated with particles only. The main aim of the NEPM goal for PM_{10} particles is that the daily air quality objective should not be exceeded more than five days in a year. However, the goal was not achieved at four air monitoring sites – Box Hill, Brooklyn, Footscray and Geelong – mostly in February on hot and dry days due to bushfires, urban emissions and raised dust. Overall, Victoria experienced an increasing number of days where the $PM_{2.5}$ level's daily standard of 25 μg/m^3 was exceeded in 2017 due to urban sources. Similarly, there have been a few instances of ozone levels being exceeded over the last few years and most have been associated with large bushfires. As a result, levels of ozone that exceed recommended levels have been main concerns for Victoria over the past few years. However, according to the Environmental Protection Authority monitoring report, the concentrations of CO, NO_2 and SO_2 occur at low levels, though NO_2 concentrations have increased marginally over the last five years (Environment Protection Authority Victoria, 2018)

In South Australia, the Environmental Protection Authority conducts long-term ambient air quality monitoring around the state. The goals of the NEPM were met for the pollutants SO_2, CO, NO_2 and O_3. The main sources of air pollution in South Australia are bushfires, industry, light and heavy vehicles and dust storms. Throughout Adelaide, the quality of the air was usually good to very good in 2018 (Environmental Protection Agency South Australia, 2019). However, in 2015, the quality of the air in South Australia did not meet the NEPM standard in a few places. For example, SO_2 and PM_{10} in Port Pirie, and PM_{10} in Whyalla and Hummock Hill were higher than the NEPM standard.

In Queensland, the air quality is generally good. However, in some places, monitored air pollutants, such as $PM_{2.5}$ and PM_{10}, occurred at levels that exceeded the NEPM standard, primarily as a result of smoke from vegetation burning (Government of Queensland, 2017). According to a report by the government of Queensland, concentrations of PM_{10} during 2002, 2003, 2005 and 2009 were above the standard of 50 μg/m^3 due to major dust storms and/or bushfires. Similarly, ozone measured in the South East of Queensland, Gladstone and Townsville was found to exceed the NEPM air quality standards in two different years (2011 and 2015). Both exceedances were associated with added emissions of precursor pollutants from bushfires coupled with favourable weather conditions for photochemical smog formation. According to

the Queensland government, higher temperatures are predicted in the near future, as climate change is likely to increase the potential for ozone formation. However, even though major industries consistently emit pollutants throughout the year, the concentrations of NO_2, SO_2 and CO remain within the national standard.

Similarly, in the Northern Territory, the Environmental Protection Authority monitors the quality of air at Winnellie and Palmerston stations (Northern Territory Environment Protection Authority, 2015). Generally, the quality of air in the Northern Territory is good; however, there is some pollution at the top end of the region due to smoke from distant and local vegetation burning during the dry season, mainly constituting $PM_{2.5}$. Gases such as CO, NO_2, and SO_2 all occur at very low levels compared to large cities in other parts of Australia, whereas O_3 occurs at moderate levels due to natural processes. Most of the time, NO_2 levels are found at higher concentrations than other gases near busy roads.

2.5.6 Sources of air pollution and its health impacts in Australia

As mentioned in section 2.2, pollution sources are often characterised as either anthropogenic (human-made) or natural sources. In terms of air quality, most focus is placed on anthropogenic emissions because emissions from natural sources are generally very difficult to control. The most important anthropogenic sources of air pollution in Australia are described below.

2.5.6.1 Biomass burning

The burning of biomass is the combustion of organic matters such as crops, forest residue and vegetation (Granier et al., 2011). Burning can be from natural or manmade fires. For example, human-initiated burning includes the burning of vegetation for land clearing and land-use change, or it could be natural, such as lightening-induced fires. According to scientific estimates, 90% of biomass burning is due to human activities and only a small percentage is due to natural fires (The National Aeronautics and Space Administration, 2001). In Australia, fire is a common practice every year for land management, where it is used to burn the by-product of crops such as wheat or rice stubble, sugarcane waste, rice husks and forest residue (van der Kaars et al.,

2000). The annual contribution of total carbon emissions from the burning of biomass is 87%, from wildfire is 10% and 3% is from prescribed burns (Department of Climate Change, 2008). During extreme fire years, in the southern part of Australia biomass burning emits as much carbon to the atmosphere as savannah fires. In these cases, even though they often occur for a short duration, the pollution events can be severe and leave a strong perception of poor air quality for the residents they impact. Therefore, the smoke fires generate every year due to biomass burning are one of the main public health concerns in Australia (Martin et al., 2013). The health impacts of biomass burning include eye and skin irritation, heart disease, cancer and respirable diseases, such as breathing problems, respiratory tract irritation, bronchitis, and increased severity of asthma.

Biomass burning releases a large number of airborne particles and traces of other gases including carbon dioxide, CO, water vapour, PMs, hydrocarbon, NOs and thousands of other compounds, (Hurst, Griffith, & Cook, 1994). The actual composition of smoke depends upon the type of wood-burning, the surrounding temperature, and the speed and direction of wind blowing. The smoke from biomass burning tends to have UFPs, which are a more significant threat to public health, as they can go deeply into the lungs (Sigsgaard et al., 2015). In fact, the pollution can result in significant health impacts due to several factors, such as the concentration of air pollutants, the length of exposure, individual susceptibility, age and the presence of any pre-existing lung or heart disease (Laumbach & Kipen, 2012). Pregnant women and unborn children are also potentially susceptible to smoke from biomass burning, which contains many of the same compounds as found in cigarette smoke (Wylie et al., 2014).

To manage and reduce the level of pollution from vegetation burning, different sustainable environmental activities are encouraged throughout Australia. Some good examples of these activities are the use of wheat stubble to produce strawboard and soil fertilizer instead of burning it (Russell-Smith et al., 2013). In some areas, the wheat stubble can also be returned to the soil to reduce soil erosion and moisture loss. Rice, wheat and sugarcane waste can also be used to produce ethanol. This way, Australia can manage and predict air quality outcomes from biomass burning in their areas.

2.5.6.2 Fossil fuel combustion

Australia is the world's eighth-largest energy producer and contributes about two-and-a-half percent of the world's energy production (Bureau of Resources and Energy Economics, 2014). To produce such energy, Australia is one of the most coal-dependent countries in the world (Diesendorf & Saddler, 2003). Currently, the primary sources of energy in Australia are coal, natural gas and oil-based products, with the coal industry producing approximately 38% of Australian's total greenhouse gas emissions.

In 2008, Australia's net CO_2 emissions from all energy sectors were 566.2 million tonnes, among which the transport sector produced 13.9% and industrial processes produced 5.4% (Parliament of Australia, 2010). The main emission of CO_2 in Australia comes from the combustion of fossil fuels. These fossil fuels include coal, oil and oil derivatives such as petrol and methane gas (also called natural gas). Due to this, Australia's greenhouse gas emissions increased by 1.5% in 2017, largely driven up by the increasing use of fossil fuel in various sectors, such as electricity, transport, stationary energy, agriculture, fugitive emissions, industrial processes, waste and land use, land-use change and forestry (Climate Council, 2017). The use of fossil fuel has large-scale detrimental effects via the production of greenhouse gases (Galdos et al., 2013; Smeets, Bouwman, & Stehfest, 2007), which are increasing the greenhouse effect on our planet and causing global warming (Meinshausen et al., 2009). In Australia, global warming is regarded as the most salient environmental issue and is believed to have contributed to Australia's worst drought (Braganza et al., 2003), flood (Franks & Kuczera, 2002) and heavy rainfall (King, Karoly, & Henley, 2017). Past studies have indicated that a certain group of people are associated with a significantly higher risk of health issues due to global warming, such as respiration illness due to heatwaves; mortality due to cold, drought or storms; changes in air quality; and changes in the ecology of infectious diseases (Kovats et al., 2001; Stott, Stone, & Allen, 2004).

In conclusion, there is a clear link between fossil fuel burning and adverse health impacts. Due to this, a rapid transition to more renewable energy sources and efficient energy use is urgently needed. In order to identify the potentially harmful effects of fossil fuel on human health, research should be undertaken to quantify and characterise the risks.

2.5.6.3 Bushfire

Bushfire is one of the most common sources (both natural and anthropogenic) of emission and is an ever-increasing problem in Australia. Bushfires produce a large amount of smoke that contains PMs, polycyclic aromatic hydrocarbons, aldehydes, CO, organic acids, volatile organic compounds and O_3 due to reactions with sunlight (Rappold et al., 2017). Approximately 90% of PMs produced from bushfires consist of PM_{10}, which causes respiratory problems (Tham et al., 2009). These PMs from bushfires are an increasing and unregulated source of air pollution across wide geographic areas and frequently affect major populations. Bushfires are likely to increase due to global warming and the practice of burning vegetation, thus increasing the risk of exposure to air pollution among the wider population (Haikerwal et al., 2015).

In Australia, more than 800 endemic species of vegetation are dominated by fire-adapted eucalypts (Sharples et al., 2016). Due to this vegetation and the climatic conditions, the bushfire season is becoming longer and episodes of severe fire weather more frequent. The occurrence of more frequent bushfires causes increasing disease and deaths due to particulate air pollution (Morgan et al., 2010). Bushfires are common over the tropical savannas of the north of Australia where some parts of the land burn on an annual basis. However, in the south-east, where the majority of the population resides, a Mediterranean climate featuring hot and dry summers and wet winters is susceptible to large wildfires that threaten life and property. The primary reason for the high chance of bushfire in the south-east is the climate of the region. The second reason is the encroachment on the bushland of expanding urban populations around the fringes of cities. The dry, hot summer boosts the occurrence of fire hazards, which are worsened by the periodic droughts that occur as part of natural inter-annual climate variability. Studies conducted by Suppiah et al. (2007) and Clarke, Smith, and Pitman (2011) predicted that south-eastern Australia will become hotter and drier in the future as an effect of climate change. This study found that the number of very high and extreme fire danger days could increase by 4–25% by 2020 and 15–70% by 2050 across most of south-eastern Australia due to increasing greenhouse gases. During bushfires, large areas of land can be covered with a thick layer of smoke that can travel hundreds of kilometres from the actual fire source, potentially involving major cities and exposing millions of people.

The concentration of PMs in Australia is generally low but do occasionally exceed air quality guidelines due to bushfires in surrounding bushland (Bradstock et al., 2010; National Environment Protection Council, 2011). Studies have shown that PMs (both $PM_{2.5}$ and PM_{10}) are responsible for respirable health impacts and the associations between them are even stronger during bushfire periods (Martin et al., 2013). According to Morgan et al. (2010), every 10 $\mu g/m^3$ increase of PM_{10} due to bushfire was associated with a 1.24% increase in all respiratory disease admission, including chronic obstructive pulmonary disease and asthma among adults. However, researchers did not find consistent associations with bushfire and cardiovascular admission or mortality. In Melbourne, Tham et al. (2009) found that elevated levels of PM_{10} increase the risk for exposed people of attending emergency departments for respiratory conditions. In Brisbane, Chen, Verrall, and Tong (2006) study found that daily respiratory hospital admission rates consistently increased with increasing levels of PM_{10} during bushfire periods. In Darwin, Johnston et al. (2002) found a significant increase in asthma patients with every 10 $\mu g/m^3$ of PM_{10} increase during bushfire periods. The strongest effect was seen on days when PM_{10} was above 40 $\mu g/m^3$.

In the past few decades, researchers began to pay more attention to the smaller fractions of PM ($PM_{2.5}$) rather than PM_{10} for two reasons. First, small-sized particles remain in the atmosphere for longer periods of time, and second, they can penetrate deeper into the respiratory system where they promote local and systemic inflammation. The most commonly investigated and established adverse health impacts of $PM_{2.5}$ exposure from bushfire smoke relates to pulmonary diseases, such as asthma, chronic obstructive pulmonary disease, and infections (Henderson & Johnston, 2012), and an increase in hospital admissions and emergency department visits (Elliott, Henderson, & Wan, 2013). Several studies conducted in Sydney also found that particulate air pollution – especially $PM_{2.5}$ – was associated with daily mortality and hospital admissions due to respirable diseases (Barnett et al., 2006; Morgan et al., 2010; Simpson et al., 2005). However, the relationship between them appeared stronger during bushfires compared to non-bushfire periods.

Given that bushfire is likely to increase in frequency and intensity in Australia, the increase in PM concentrations observed during burning events and the likely adverse health impacts associated with this increase indicate the need for further research in this area.

2.5.7 Health impacts in relation to traffic air pollution in Australia

Traffic-related air pollution is one of the main reasons for the high concentration of air pollution in Australia (Pereira et al., 2010; Xia et al., 2015). In particular, traffic congestion increases emissions and degrades ambient air quality. It is well established that with an increasing population, traffic on roads has significantly increased all around the world over the past 20 years (Duong & Lee, 2011). Australia has one of the highest rates of motor vehicle ownership in the world, with over 90% of Australian households having one or more registered motor vehicles (Australia Bureau of Statistics, 2018). Public and active modes of transportation only account for a small percentage of travel, despite the fact that in major cities, approximately 20% of trips to work are less than 5 km (Australia Bureau of Statistics, 2018). Most commuters prefer to use their own car for a distance that could easily be replaced by an active mode of transport such as bicycle riding or walking. Due to the growing numbers of motor vehicles, the continual addition of air pollutants into the ambient air of Australia is rapidly increasing (Bowatte et al., 2018).

The large majority of these vehicles use an internal combustion engine that burns fossil fuels or gasoline as an energy source. The process of incomplete combustion of oil or fuel contributes to air pollution by releasing a variety of emissions into the atmosphere. The most common air pollutants released from vehicles include PM, NO_2, CO, NO, SO_2, Pb, and Volatile Organic Compounds (VOCs). The in-combustion rate and release of pollutants depend on the type of vehicle. The numerous technologies used in vehicles for fuel combustion can play a significant role in the production of air pollutants. Exhaust emissions contribute fine PM with an aerodynamic diameter of less than 2.5 ($PM_{2.5}$, $PM_{0.1}$), whereas non-exhaust emission is associated with the coarse mode of PMs, such as PM_{10} (Kam et al., 2012). Populations that live in proximity to high traffic routes are the most susceptible to PM-related health effects (Choudhary & Tarlo, 2014), and the most sensitive demographics are children (Vieira et al., 2012), the elderly (Adar et al., 2007), women (Hystad et al., 2015) and healthy adults (Zuurbier et al., 2011).

Although the detailed physiological phenomenon of PMs attacking the human body remains unclear, several studies have proved that PM components – including elemental carbon, organic carbon, NO_2, NO and SO_2 – play a significant role in toxicity (Bowatte et al., 2018; Palleschi et al., 2018). In 1996, the USA estimated that over 600 million people in urban areas around the world are exposed to hazardous levels of traffic-generated air pollutants (Cacciola, Sarva, & Polosa, 2002). As a consequence, traffic air pollution and its association with public health impacts are grabbing attention from local societies, environmental and health research communities, regulating agencies, governmental organisations and industries. In Australia, recent studies have reported similar concerns showing an association between traffic air pollution and human health (Pereira et al., 2010; Xia et al., 2015). To find out the actual association between pollution and health issues, various studies have been conducted in different Australian cities using different methodologies, such as time-stratified case-crossover (Pereira et al., 2010), comparative risk assessment health models (Xia et al., 2015), AusRod dispersion model (Pereira et al., 2011) and so on. In Perth, a limited number of studies have been conducted in relation to traffic air pollution and its associated human health impacts. For example, Pereira et al. (2014) conducted a study regarding traffic air pollution and its impact during the perinatal period. As per this study, the pollution (up to 0.49% per 10 $\mu g/m^3$ increase in locally derived traffic emissions) caused the pre-labour rupture of membranes and reduced foetal growth. Similarly, another study conducted by the same researcher in 2013 also found an increase in pre-eclampsia by 12% during exposure to traffic-related air pollution (Pereira et al., 2013). In Sydney, a study conducted by Jalaludin et al. (2006) showed that traffic-related particulates are strongly associated with the number of emergency attendances due to cardiovascular diseases (CVD) and stoke during cold periods compared to warm among people aged 65+ years. However, Hansen et al. (2012) proved that the concentrations of PM_{10} are significantly higher in the warm season which explains the increasing number of hospital admissions due to CVD. In Tasmania, a study conducted by Bui et al. (2013) found that frequent exposure to heavy vehicles was associated with increased asthma severity. Similarly, in Adelaide, Xia et al. (2015) conducted a study to find out the environmental and health benefits due to shifting 40% of vehicle transport to an alternative transport mode, such as bicycle riding or walking. The results showed that the annual average urban $PM_{2.5}$ concentration was reduced by approximately 0.4 $\mu g/m^3$, resulting in net health benefits of an

estimated 13 deaths/year prevented due to improved air quality. Also, Barnett et al. (2006) conducted a study in five Australian cities (Brisbane, Canberra, Melbourne, Perth and Sydney) to estimate the association between air pollution and human health impacts. The results suggested that traffic-related air pollution (CO, NO_2 and PMs) is significantly associated with cardiovascular hospital admissions, especially among the elderly, at pollution levels lower than the established guidelines.

Despite some evidence, further research is still needed to fully understand how PMs and other pollutants affect human health in Australia. In order to understand the actual contribution of traffic emissions to the environment and the impact they may cause to human health, it is important to understand the chemical composition of the gasoline or fuel used, its quantitative contribution, and sources. Moreover, it is also important to understand the properties of traffic emissions, such as physical shape, structure, particle size distribution, and the temporal and spatial variations, which have a significant relationship with the cause of disease in a human body (Watson et al., 2007). A few decades ago, researchers and policymakers focused on exhaust emissions; subsequently, the regulations and technologies have been revised and have resulted in zero vehicle tailpipe emissions to ambient PM concentration (Mathissen et al., 2011). Even with zero tailpipe emissions, some research shows that traffic will continue to contribute to fine and ultrafine particulates through non-exhaust emission (Kumar et al., 2013), which contributes nearly 90% of the total emission (Pant & Harrison, 2013).

Australia has also implemented other strategies to maintain good ambient air quality (Department of the Environment, 2016); however, some pollutants still exceed the current national standards, such as ground-level O_3 and PMs (Department of Environment and Energy, 2015). Hence, it is important to monitor the ambient air quality and interpret the adverse health impacts associated with increased levels of air pollution in the near future.

2.6 Multiple-path model of particle deposition in lungs

2.6.1 Introduction

The deposition of PM in the lungs varies based on the amount of air inhaled and exhaled. It also varies according to physical and physiological factors, such as the depth, frequency and route of breathing and the duration of breathing in the exposed area. As required by the Environmental Protection Act, researchers from the Chemical Industry Institute of Toxicology (CIIT) started research regarding the deposition of PMs and have made significant contributions to the current understanding of particle dosimetry in the respiratory tract, and its deposition and clearing mechanism (ARA, 2014). They developed a scientifically sophisticated and user-friendly computational model for estimating particle dosimetry for laboratory animals. The researchers from CIIT developed 10 statistically-based 'virtual' lung structure models to examine the process with randomness and asymmetry of the airway branching system. These models were used to predict particle deposition in the lung assuming simple and idealistic lung geometries. This trial enabled them to gain insight into the effects of the lungs' branching pattern and the size of the deposition of PMs.

Finally, the Multiple-Path Particle Dosimetry (MPPD) model was developed to integrate more realistic asymmetries in lung structure and calculate the particle deposition at individual airway levels. Initially, this model was used to estimate the deposition of particles for laboratory rats using measurements available in the literature. Later, CIIT extended the MPPD model to calculate deposition for a range of particle sizes and breathing rates in adult human lungs. Currently, the new version of this software (MPPD v3.04) is available from CITT free of charge at www.ara.com/products/multiple-path-particle-dosimetry-model-mppd-v-304.

2.6.2 Application of the Multiple-Path Particle Dosimetry model

According to this model, inhaled particles may deposit in various regions of the respiratory system depending on five deposition mechanisms: impaction, settling, diffusion, interception and electrostatic deposition. The degree of particle deposition depends on particle size, shape and density, airway geometry and the breathing pattern of the individual (either nose or mouth breathing). Despite this, the deposition could differ for different groups of people, such as adults, children or people with respiratory diseases (Ham, Ruehl, & Kleeman, 2011; Hansen et al., 2012; Hofmann, 2011). This model calculates the deposition and clearance of monodisperse and poly-disperse aerosols in the respiratory tract for particles ranging in size from ultrafine (0.01 µm) to coarse (up to 100 µm) (Figure 2.4). The density and size of the particles in the surroundings are the most important factors that must be taken into account when using dosimetry models to estimate lung burdens (Miller et al., 2013; Oravisjärvi et al., 2011). In fact, the region of the airway in which particles deposit is most highly dependent on particle size (Møller et al., 2008; Scheuch et al., 2006). Therefore, regional deposition is an important factor in assessing the possible effects on health posed by inhaled particles. However, deposition of particles in the lung also varies according to other factors, such as exposure concentration, distance and time of exposure, exertion rate, and route of breathing. The models used are based on both single-path and multiple-path methods for tracking airflow and calculating aerosol deposition in the lung. The single-path method calculates particle deposition in a typical path per airways generation whereas the multiple-paths calculate particle deposition in all airways of the lungs and also provide lobar and airway-specific information. A multiple-path model is usually chosen in various

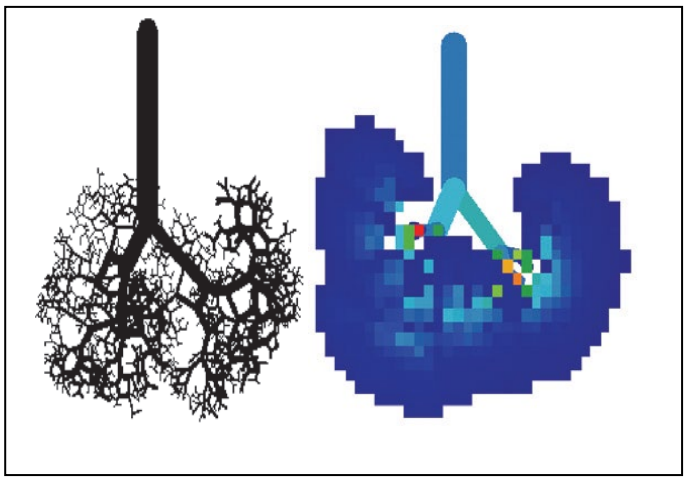


Figure 2.4: Visualisation from MPPD of deposition fraction of PM in human lungs

studies because it represents a relatively realistic human airways structure (Anjilvel & Asgharian, 1995). The information of the MPPD model in more detail is described in table 2.6 below:

Table 2. 6: Information about the MPPD model

Respiration Parameters	**Airway Morphometry**
Species	Human
Type of Model	Yeh/Schum Symmetric, Yeh/Schum 5-Lobe, Stochastic Lung, Age-Specific Symmetric, Age-Specific 5-Lobe, Weibel, PNNL Symmetric and PNNL Asymmetric
Human Airways Parameter	Functional Residual Capacity, Upper Respiratory Tract Volume, Breathing Frequency, Tidal Volume, Inspiration Fraction and Breathing Scenario (nasal, oral oronasal-mouth breather, oronasal-normal augmenter and endotracheal)
Particle Properties	Density, Aspect Ratio, Diameter of the particles, Aerosol Concentration and Acceleration of Gravity

Numerous studies have been conducted regarding the deposition of particles in the human lungs using the MPPD model in different scenarios. For example, Sánchez-Soberón et al. (2015) researched on exposure to three size fractions of outdoor PM (PM_{10}, $PM_{2.5}$ and PM_1) and its influence on the health of three population groups (children, adults and elderly) using the MPPD v 2.11 model. The results of this study showed that the highest pulmonary deposition doses for the three PM sizes were recorded for elderly people. Due to high-intensity activities, the children were also observed with a higher deposition of $PM_{2.5}$ in the pulmonary regions of the lungs than the adults. These observations have implications for the expected adverse health effects for vulnerable groups of the population. Likewise, some research has focused on the estimation of alveolar lung burdens of copper using MPPD models among workers engaged in tasks requiring various levels of exertion as reflected by their minute ventilation (Miller et al., 2013).

To assess the impact of inhaled particle matter on the basis of exertion rate, understanding regional deposition and subsequent physiological impacts are critical steps. In future, the study should focus on modelling realistic exposures (gender, physical activities) and perform studies to elucidate mechanisms of injury in both bronchial and pulmonary airways that might impact performance.

2.6.3 Improvement of the Multiple-Path Particle Dosimetry Model

The MPPD model software has undergone continual improvement since its initial release (Miller et al., 2016). For example, the model version v 2.11 has additional functionality to calculate deposition and clearance of more complex aerosols simultaneously to handle up to four overlapping log-normal distributions. For use in the MPPD model, each of the individual mode size distributions of the particles had to be combined into single size distribution. As per the input parameters provided by the user for each mode, single multimodal size distribution is constructed computationally to reflect the overall distribution of particle size. This size range of multimodal particle distribution is then divided into the logarithmically spaced intervals. The total number of particles in each size interval is then calculated in order to assist the MPPD model in calculating deposition and clearance. This type of feature is useful whenever the atmospheric exposure is multimodal, as is often the case with workplace exposure and also can occur in inhalation toxicology studies.

An additional feature is able to adjust the total aerosol deposition for the amount of a particular particle “X” when “X” is mixed with some other aerosol. This feature is particularly important for workplace exposure where the particles of interest are part of the total dust aerosol that is measured. Recently, a new version of MPPD software (vs 3.04) was introduced, which has an advanced and modified function and is also able to handle up to four overlapping log-normal distributions, enabling more complex aerosol distributions to be modelled for deposition and clearance calculations (Puisney, Baeza-Squiban, & Boland, 2018). For both animals and humans, this feature is able to specify clearance rates and mucous velocities so any type of particle can be modelled. This additional feature enables more information to be established, such as lung geometry/morphology in humans as well as rats. The deposition and clearance of particles in mice, rats, monkeys, sheep and pigs can, therefore, be easily examined.

The updated version achieved a greater resolution in its imaging capabilities, which provides an opportunity to expand our knowledge of the lung structure of various species and add refined models to MPPD (Pauluhn, 2017). Only basic physiological data are needed for a number of

species that would supplant the algometric relationships that have had to be used for some of the variables.

2.7 Summary

Exposure to outdoor air pollution represents the biggest environmental risk to human health in various ways, leading to increased mortality and morbidity (WHO, 2018). The WHO estimates that air pollution contributed to around 4.2 million premature deaths annually around the world in 2016. Numerous epidemiological studies have shown that fine particulate air pollution is especially associated with cardiovascular and respirable diseases. Further, the adverse health effects of other air pollutants, such as coarse and ultrafine particulate matter, oxide of nitrogen, sulphur dioxide and carbon monoxide are also widely reported in the literature.

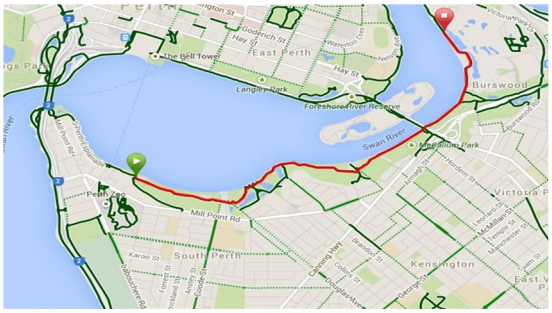
Many Australian studies have also reported that exposure to traffic air pollution is associated with an increase in mortality and hospital admissions due to respiratory and cardiovascular disease, and other short-term health effects (Xia et al., 2015). In an urban environment, air pollution exposure while commuting via different modes of transport in traffic micro-environments may lead to a high proportion of total daily exposure to ambient air pollution. Cyclists, in particular, experience higher uptakes of air pollution due to their relatively high physical exertion levels and subsequent breathing frequency compared to other commuters (passengers on buses or trains; car drivers) (Kingham et al., 2013). However, the deposition rate varies from one cyclist to another due to breathing rate, which varies between individuals and depends on gender, fitness and lung capacity. In Australia, very limited research has been conducted regarding traffic-related air pollution and the personal exposure of cyclists. In addition, there is a lack of consensus among researchers regarding particle size deposition into the respiratory tract during physical activity. Furthermore, the evidence about the magnitude and specific regional deposition of the potential dose difference between genders is also very limited. Due to these limitations, the potential effects of accurate exposure – dose relationships that could be used to develop health risk assessment models for cyclists – are lacking (Buonanno et al., 2012). To address these gaps, this study aims to examine the exposure level to PM among bicycle commuters, and simulate the deposited dose of PM as a function of respiration rate,

physical exertion, particle characteristics and gender to gain a better understanding of the health effects and compare the standard exposure approaches to methods that account for individual variance and exertion.

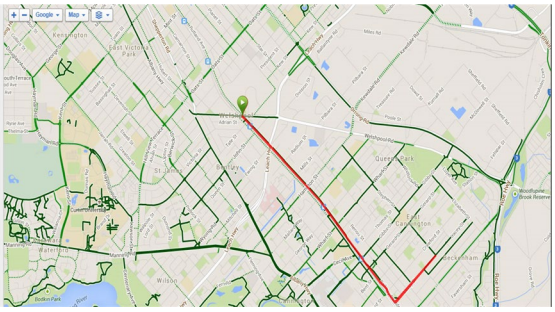
CHAPTER 3 : METHODS

3.1 Introduction

This study was conducted in the metropolitan area of Perth, the capital city of Western Australia (WA) and the fourth-most populated city in Australia. To achieve the aim and objectives of the study, four popular predetermined bicycle commuter routes across the Perth metropolitan area were selected (Figure 3.1) based on the number of vehicles and information provided by the Department of Main Roads (Main Roads, 2002) and Bicycling Western Australia (https://www.bwa.org.au) (Figure 3.1). Two routes were selected from suburban areas and away from major traffic roads, and two other routes were located in areas close to major vehicular traffic roads (such as freeways) where the movement of vehicles is significantly higher compared to the suburban roads (Table 3.1). The selected cycling routes are also very popular among cyclists and the cycling volume is high during the daytime. Before commencing the study, the cycling routes were monitored to ensure they were in appropriate conditions for the study. Each cyclist was expected to ride 5 km (from the start to the turnaround point and back to the initial location). The cycling routes located within suburban areas and away from vehicular traffic roads (community routes) were named route 1 and route 2, and those close to freeways (major routes) were named route 3 and route 4. More information about the routes is provided below.



Route 1



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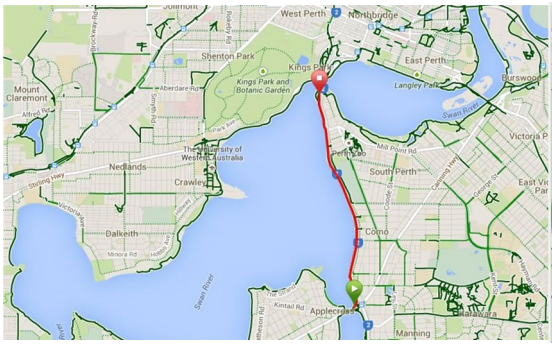
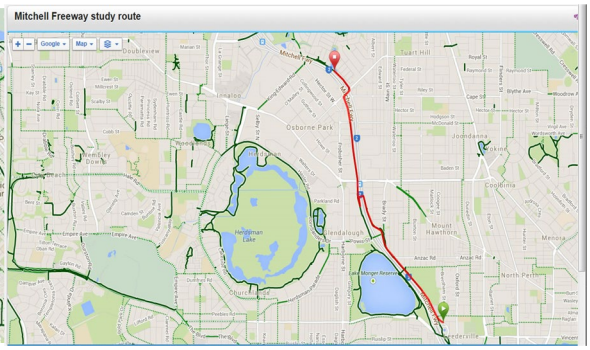


Route 3



Route 4

Figure 3.1: Maps of four cycling routes

3.2 Sampling Sites

3.2.1 Route 1 (Mill Point Road)

Route 1 is situated near Coode St in South Perth Esplanade WA 6151, which is near the south bank of the Swan River (Sir James Mitchell Park). The city of Perth is located approximately 1 km north of this route. According to Main Roads WA, the average weekly (Monday to Friday) number of vehicles that passed near Mill Point Road in 2015–2016 (two years) was 21,120, including both cars and trucks (https://trafficmap.mainroads.wa.gov.au/map). The distance between Mill Point Road and route 1 is approximately 200–245 meters. The average number of cyclists that

commuted through this route per week (Monday to Friday) in 2015–2016 was 1,148. Although Mill Point Road is not classified as a busy road, it is in close proximity to a diesel-powered ferry station (Mends St Jetty), car parks, and restaurants. Also, part of the cycling route is close to a major intersection between Mill Point Road and Labouchere Road. A sampling site for measuring ambient air pollution was selected near the Observation Deck, South Perth Esplanade, South Perth, WA. The distance between monitoring equipment and route 1 was approximately 308 meters. More details with regard to air pollution monitoring are provided in section 3.4 below.

3.2.2 Route 2 (Railway Parade)

Route 2 is located on Railway Parade in Welshpool WA 6106, which is situated southeast of the Perth CBD (approximately 8.8 km). This route is close to Railway Parade (approximately 1–2 metres away). The surrounding neighbourhood consists of residential houses, schools, a railway line (20 meters away) and light industries such as repair and sprays painting (within 100–800 meters). This route is also close to another road (Sevenoaks Street) that runs parallel to Railway Parade (approximately 10 meters away). There are two intersection points close to the sampling site: one is between Welshpool Road and Railway Parade (approximately 60 meters away) and the second one is between Leach Highway and Railway Parade (approximately 500 meters away). The average weekly number of vehicles counted for this route in 2017–2018 (two years) was 2,630 including both cars and trucks. The ambient air monitoring equipment was installed at the front yard of St Joseph's School, 140 Railway Parade, Queens Park WA. The distance between the monitoring equipment and route 2 was approximately 13 meters.

3.2.3 Route 3 (Kwinana Freeway)

Route 3 is located in South Perth near the Mill Point Reserve, 5.0 km from the Perth CBD and on average 7 meters away from the Kwinana Freeway. According to Main Roads WA, the average number of cyclists commuting through this route per week in 2017–2018 was 2,132. The Kwinana Freeway is considered one of the busiest freeways within the Perth Metropolitan area,

with private cars, public buses, and heavy trucks moving from both directions – south and north of Perth's CBD. The average weekly number of vehicles counted in 2017–2018 (two years) was 193,252 (Main Roads, 2018). The ambient air quality monitoring equipment was installed within the Mill Point Reserve area, South Perth, approximately 35 meters away from this cycling route.

3.2.4 Route 4 (Mitchell Freeway)

Route 4 is located close to Richmond Reserve, North Perth which is on average 7 meters away from the Mitchel Freeway. This route is about 3 km away from the Perth CBD and is also close to three other major roads: the exit ramp to Vincent Street (approximately 15 meters away), Oxford Street (approximately 220 meters away) and Vincent Street (approximately 230 meters away). According to Main Roads, WA, the average vehicular flow on the Mitchell Freeway per week during 2015–2016 (two years) was 161,184. The number of cyclists commuting through this route per week in 2015–2016 was 1,207. The ambient air quality monitoring equipment was installed beside the route, near the end of Melrose Street, Leederville. The distance between the monitoring equipment and route 4 was approximately 118 meters. The total number of vehicles close to the selected cycling routes are provided in Table 3.1.

Table 3. 1: The major roads and number of vehicles near the four cycling routes

Route	Year	Major Roads	Average volume of vehicles (Mon-Fri)	Direction of vehicles	Type of vehicles (%): both directions		Distance to the road (approx.)	Total no. of vehicles
					Cars	Trucks		
Route 1	2015–2016	Mill Point Rd, East of Onslow street	21,120	Both	96.3	3.7	160 m	21,120
Route 2	2017–2018	Railway Parade (Welshpool Beckenham) North of Wharf St	2,630	Both	91.6	8.4	1.0 m	
	2014–2015	Sevenoaks St	13,746	Both	94.6	5.4	45 m	35,930
	2013–2014	Welshpool Road	19,554	Both			60 m	

Route 3	2017–2018	Kwinana Freeway (at narrow bridge)	191,064	Both	94.0	6.0	20 m	193,252
	2013–2014	Mill Point Road	2,188	Going North	94.4	5.6	150 m	
Route 4	2013–2014	Mitchell Freeway (near Melrose Street)	71,933	Going South	NA*	NA*	70 m	
	2015–2016	Mitchell Freeway (near end of Melrose Street)	89,251	Going North	NA	NA	130 m	214,273
	2015–2016	Ramp to Vincent St from M. Freeway (south)	16,267	Both	94.7%	5.3%	15 m	
	2013–2014	Oxford Street (close to end of Melrose Street)	12,991	Both	95.6%	4.4%	220 m	
	2013–2014	Vincent Street (Close to end of Melrose street)	23,831	Both	NA	NA	230 m	

Source: https://trafficmap.mainroads.wa.gov.au/map

3.3 Study population and sample size

3.3.1 Sample size

The sample size (N) of 122 subjects was estimated to detect a large effect of 0.40 according to Cohen's effect conventions for the main effect of a group (four routes: two in community areas and two in major roads) with a power of 80% at 5% significant level. Accounting for less than a 20% drop out rate, 156 subjects were sufficient for the study. In this study, we applied the definition of the Commonwealth Scientific and Industrial Research Organisation (CSIRO) for warm and cold seasons. The warm season is defined as the yearly period when the average daily temperature is greater than 16^0C and the cold season as having an average daily temperature of less than 12^0C (Penman, Lemckert, & Mahony, 2006).

3.3.2 Selection criteria

The study population consisted of cyclists residing in Perth who (already) cycled at least one of the selected four routes while commuting to work. The participants were selected randomly from Perth cycling clubs, Curtin University and the general community, with the selection criteria including non-smokers (including never smoked and current non-smokers who quit smoking), males and females, aged between 20 and 55 years who cycle at least 150 km/week – ideally along one of the four study routes. Having cardiovascular and other chronic health conditions (but not asthma) were exclusion criteria for the study. Potential participants who had asthma were accepted in the study because according to the Australian Bureau of Statistics in 2018, approximately 2.7 million Australians had asthma (11.2%) diagnosed by a doctor (Australian Bureau of Statistics, 2018b). Perth has one of the highest rates of asthma prevalence in Australia, between 19 and 23.4% among adults (James et al., 2010a). In total, 122 individual cyclists were recruited for the study. Cyclists were divided into four groups according to the route they normally commute. Due to an insufficient number of cyclists, the participants were asked to ride an extra route in addition to their original one to increase the statistical power of the study. No participant repeated all four routes. Each cyclist was requested to cycle the same route in both cold and warm seasons. Unfortunately, some cyclists were not available to do both cold and warm seasons, so the total number of participants' rides were 282.

3.4 Data collection procedures

In this study, the monitoring of ambient concentrations of PMs (such as PM_1, $PM_{2.5}$, PM_4, PM_{10} and PM_{total}) and selected gases – including CO, SO_2, NO, NO_2 (i.e. NO + NO_2 = NOx) – was conducted at each cycling route. Each cyclist was also assessed for personal exposure to respirable particulate matter (PM_4). The Multiple-Path Particle Dosimetry (MPPD 3.04) model was used to establish the lung-deposited dose of PMs with different size for each cyclist according to their exertion rate.

3.4.1 Ambient air pollution monitoring

Ambient air quality monitoring was conducted at each cycling route whilst participants were cycling in the morning peak hours between 6:30 am and 9:30 am for five days/week during the cold and warm seasons. The morning peak hour was selected as the most appropriate time to conduct the monitoring because according to previous studies the highest exposure was consistently observed during these hours (Hunter et al., 2012). The equipment used in the study included a DustTrakTM DRX Aerosol Monitor 8533b (TSI Inc., MN, USA), Testo 350 gas analyser, P-Trak Model 8525, Diffusion Dryer DDU 570**,** Thermodenuder TDD 590, Universal Scanning Mobility Particle Sizer 2700(USMPS), TinyTag 2TM data logger (GeminiData Loggers (UK) Ltd.) and Anemometer. That equipment was used throughout the study and was set up 200 m away from each cycling route at about 0.5 meters above the ground level and was programmed concurrently to record data for a period of four hours. A detailed description of the instruments is provided below.

3.4.1.1 DustTrak TM DRX Aerosol Monitor 8533

The DustTrak TM DRX Aerosol Monitor 8533b (TSI Inc., MN, USA) is a real-time dust monitor that is used to simultaneously measure both mass- and size-fractionated concentrations of particles (TSI, 2016). It is a battery-operated data-logging and light-scattering laser photometer that gives real-time aerosol mass readings (Figure 3.2). This desktop monitor uses laser photometry at 90^{o} light scattering that provides real-time particle concentration in mg/m^3 in five fractions: PM_1, $PM_{2.5}$, PM_4, PM_{10} and total inhalable PM size fractions (PM_{total}). This instrument measures particle concentrations ranging from 1 µg/m^3 (0.001 mg/m^3) to high concentration up to 15 x 10^4 µg/m^3 (150 mg/m).). DustTrak TM DRX Aerosol Monitor 8533 was selected because it can be used in high mass concentration environments, ability to generates custom calibration factors with integrated gravimetric references sampling capability based on aerosol of interest. Also, it

Figure 3.2: Picture of DustTrakTM DRX Aerosol Monitor 8533

can significantly reduce mass conversion errors using particle size and count data due to particle density, refraction index and shape. In addition, this equipment can also be used in the temperature zone of 0 to 50 ^{o}C and between 0-98% non-condensing relative humidity. This equipment was programmed to record data every 10 seconds during the four hours of sampling duration.

Regarding calibration, there are two factory defaults, one is the “Ambient Call” and the other is the “Factory Call”. The ambient call is appropriate for outdoor ambient dust or fugitive dust monitoring whereas the factory call is appropriate for most workplace aerosol monitoring.

3.4.1.2 Testo 350 analyser

For emission monitoring, the Testo 350 analyser was used, which is a portable electrochemical gas analyser that consists of a ’Control Unit’ and ‘Analyser Box’ within the set of the instrument (Figure 3.3). This instrument simultaneously measures O_2, CO, NO, NO_2, NOx, SO_2, H_2S, CO_2 and CH (total hydrocarbons). Testo 350 analyser was selected for this study because this device has exclusive sensor design, patented gas paths, active sampling conditioning, intelligent automatic data logging and testing program which work together seamlessly providing a lightweight and simple to use emission monitoring solution. The control unit is small in size compared to the Analyser box whose measurement interface provides a multitude of field configurations which makes testing faster to set up and easier to perform. Besides this, control units have other beneficial function such as long Bluetooth range of 300 feet and real-time colour graphics, intuitive operation of the control unit which allow to view collected data in a graph or numeric values and can work as a data storage device and download it as the convenience. This equipment was programmed to record data every 1 second during the four hours of sampling duration.

Figure 3.3: Picture of Testo 250

Regarding calibration, O_2, CO, NO, NO_2, NOx, SO_2, H_2S, CO_2 and CH sensors can be tested and adjusted using test gas. If unrealistic readings are displayed, the sensor should be checked (calibrated) and, if required, adjusted. To ensure that specific accuracies are retained, Testo recommends testing every six months and recalibration when required.

3.4.1.3 Universal scanning mobility particle sizer

A universal scanning mobility particle sizer (USMPS) was used to measure the size and number concentration of ultrafine particles (Figure 3.4). The USMPS system is well suited to extremely

Figure 3.4: Universal Scanning Mobility Particle Sizer

precise measurements of particle size distributions within the range of 4 to 600 nm. This system includes a classifier termed as an electrical mobility classifier (DEMC), which is also known as a differential mobility analyser (DMA), in which the aerosol particles are selected according to their electrical mobility and passed into its outlet. The electrical charges carried by these particles are then measured in a downstream 'Charme aerosol electrometer'. The particle size distributions of an aerosol generated by the USMPS on the touch screen is shown in Figure 3.5.

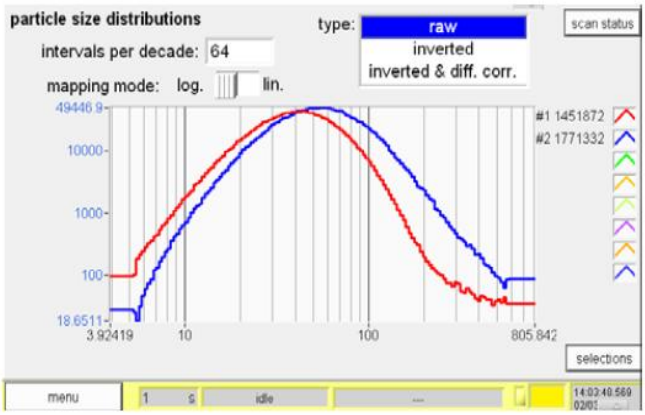


Figure 3.5: Typical particle size distribution measured by USMPS

The scanning procedure for measuring the size and number of ultrafine particles from ambient air with USMPS and other instruments is explained below, with a schematic diagram in Figure 3.6.

1. First Scan: An initial scan was performed for five minutes using only the USMPS system with no inlet pre-conditioning (apart from the essential neutraliser).
2. Second Scan: A second scanning was conducted placing a diffusion dryer at the inlet. The environmental aerosol is conditioned at first by entering it into the diffusion dryer containing silica gel, which removes moisture from the particles, and then passed through the neutraliser and USMPS to measure the 'dry' particle size.
3. Third Scan: A third scanning was performed by replacing the diffusion dryer with a Thermodenuder. The Thermodenuder removes precursors and volatile particles from an aerosol sample for subsequent measurement of solid dry particles.
4. The initial scan was then repeated. All scans were run for five minutes.

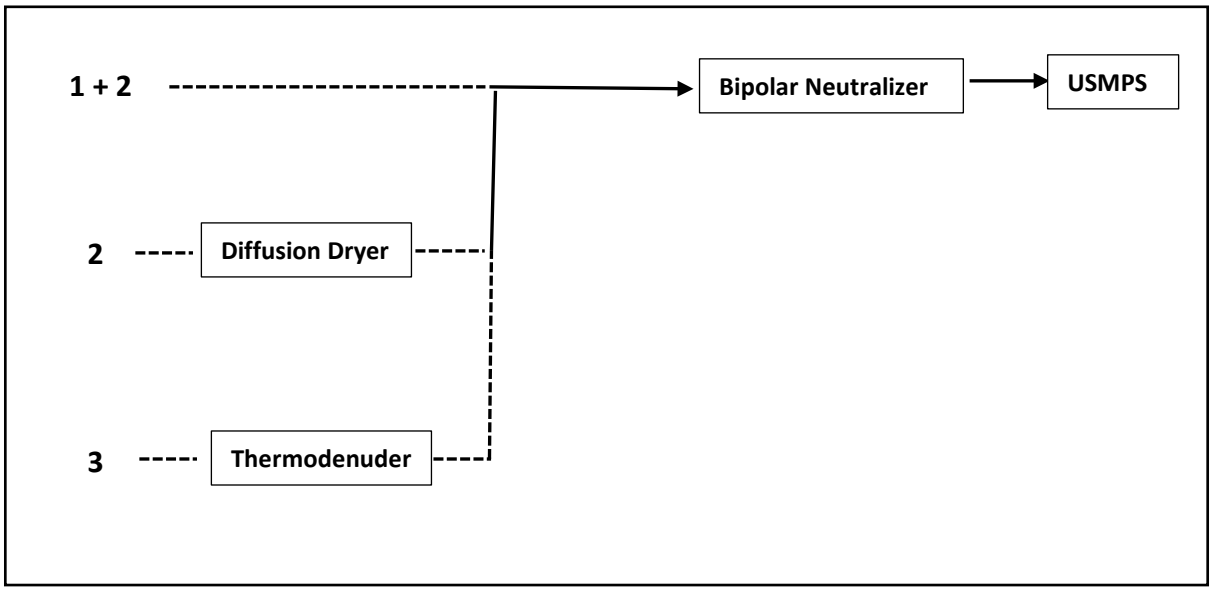


Figure 3.6: Schematic diagram of instrument configuration

As mentioned above, the diffusion dryer and Thermodenuder, along with the neutraliser and USMPS, were used to measure the size and number concentration of ultrafine particles in the ambient air. However, the data collected from the first, second and third configurations were not significantly different between each other. It was therefore concluded that the particles were predominantly dry and solid so the configuration 1/4 scans were used in the results. The scan time of five minutes was used in all studies as a balance between short measurement times while minimising measurement artefacts such as diffusion broadening.

All three scan configurations were performed at each site during the time the cycling test was conducted. However, some components of the USMPS failed during the study, required the P-Trak to be used as a replacement device.

3.4.1.4 P-Trak

The TSI-P-Trak ultrafine condensation particle counter model 8525 (Figure 3.7) was used as an alternative device to measure the ultrafine particulate levels in the ambient air. This portable instrument was used for all

Figure 3.7: P-Trak Model 8525

routes in both seasons. The P-Trak uses a liquid isopropyl alcohol-based, condensation particle-counting technique and laser photometry to count particle number concentration. P-Trak can easily operate in temperatures between 32 and 100 ^{o}F (0 to 38 ^{o}C) and measurement ultrafine particle counts ranges from 0 to 500,000 pt/cc. Only one P-Trak was used throughout the study. This equipment was programmed to record data every 10 minutes during the four hours of sampling duration. For calibration, P-Trak needs to be return to the factory for annual cleaning and adjustment.

3.4.1.5 Anemometer

Figure 3.8: Anemometer/VelociCal

An anemometer (VelociCalc, TSI) was used to measure the temperature, relative humidity and wind speed (Figure 3.8). This instrument is used in our study because it is very portable, handheld as well as multi-function ventilation test instrument featuring a menu-driven user interface for easy operation. It can store up to 38.9 days of data collected at one-minute log interval. However, the stored data can be recalled, reviewed on-screen and downloaded for easy reporting. This equipment was programmed to record data every second during the four hours of sampling duration. Other meteorological data, such as rainfall, were obtained from the Bureau of Meteorology (www.bom.gov.au).

3.4.2 Personal exposure monitoring

Similar to the ambient monitoring equipment, the personal exposure monitoring of respirable particulate air pollution was also conducted in morning peak hours between 6:30 am and 9:30 am for five days/week during the cold and warm seasons. The morning peak hour was selected as the most appropriate time to conduct the monitoring because according to previous studies the highest exposure was consistently observed during these hours (Hunter et al., 2012).

Participants were scheduled for the test; upon their arrival, they were asked to sit and relax for 5–10 minutes before commencing the test. They were then briefed about the monitoring equipment, and the study procedure they were required to follow was explained. For personal exposure assessment, a Bio Harness 3.0 heart rate monitor (Figure 3.9) was used to measure the

Figure 3.9: The Bio Harness 3.0 heart/breath rate monitor

heart and respiration rate of the participants during cycling (Shields et al., 2013). The device is worn on the chest with the help of a strap that incorporates an electrocardiography (ECG) heart-rate and respiration-rate monitor. This device is also linked to a portable modem that transmits sensor-reading data and Global Positioning System (GPS) coordinates to a computer via its software. Also, it provides a facility to detect and transmit single-lead ECG signals to be received by Bluetooth-/USB-enabled EGC instruments.

At the same time, the SKC respirable sampler was used to measure the personal exposure of respirable particles among cyclists (Figure 3.10). The “waist bag” was used to hold the SKC pump and the cyclone was clipped onto the collar of the cyclist’s shirt around 30 cm diameter of the breathing zone. During the sampling, the airflow rate was maintained at two litres/minute. Before each test, the SKC pump was calibrated and charged, and the cyclone was made ready with the filter paper inside. Before sampling, filter papers were desiccated for 24 hours to remove any moisture. The usual method of flow measurement (calibration) for the higher flows was achieved using rotameter. The cyclone was then clipped onto the collar of the cyclist’s shirt near the breathing zone.

Each cyclist was expected to ride 10 km (from the start to the turnaround point i.e. 5 km and back to the initial location i.e another 5 km). At the beginning of each test, cyclists were asked to ride the bike slowly and maintain 65% of their maximum heart rate (E1), representing the 'low exertion rate'. After completing the 'low exertion' round trip (10 km), the filter paper on the

Cyclon

SKC Pump

Figure 3.10: SKC Cyclone Sampler

cyclone was removed and a new filter paper was replaced for the 'high exertion' round trip. During the second part of the test, participants were then asked to ride the bike as fast as they could to maintain 85–90% of their heart rate (E2), considered the 'high exertion rate'. On average, each participant spent 4 hours for both low and high exertion ride. The maximum heart rate was estimated as per the formula provided by Robergs and Landwehr (2002):

Heart Rate = Hr=220-participants' age (3.1)

The used filter paper was then stored in a covered petri dish to desiccate it for 24 hours in the laboratory. To determine the dust concentration, the weight of the filter paper was measured before and after use and followed the equation as per the Australian Standard (AS 2985-2009):

a. Calculate the weight of dust collected, from the following equation:

w = (w2 – w1) – (b2 – b1) (3.2)

Where w = blank corrected weight of dust collected on the filter, in milligrams

w1 = weight of filter before sampling, in milligrams

w2 = weight of filter after sampling, in milligrams

b1 = average weight of blank filter before sampling, in milligrams

b2 = average weight of blank filter after sampling, in milligrams.

b. Calculate the average flow rate (Q), and the volume of air (V) passed through each filter for the duration of sampling from the following equations:

$$\mathbf{Q} = \frac{\mathbf{Q1+Q2}}{\mathbf{2}} \qquad \textbf{(3.3)}$$

$$\mathbf{V} = \frac{\mathbf{Q\ x\ t}}{\mathbf{1000}} \qquad \textbf{(3.4)}$$

Where Q = average flow rate in litres per minute, Q1 = initial flow rate in litres per minute, Q2 = final flow rate in litres per minute, t = sampling duration in minutes, V = air volume in cubic metres.

c. Calculate the average concentration (C) of respirable dust from the following equation:

$$\mathbf{C} = \frac{\mathbf{w}}{\mathbf{v}} \qquad \textbf{(3.5)}$$

Where C = particulate matter concentration in milligrams per cubic metre, w = net weight of dust, blank corrected in milligrams, V = air volume in cubic metres.

3.4.3 Respiratory particle deposition model

3.4.3.1 Validation of the model

The MPPD model was calibrated and validated for each participant based on spirometry testing (lung function test) and aerosol deposition measurement using a low concentration of Di-Ethylhexyl Sebacate (DEHS) in the School of Public Health laboratory, Curtin University. These tests were conducted after completion of field data collection in the warm season which lasts for one and a half months.

For the validation process, participants were requested to inhale and exhale medical-grade air containing a low concentration of nebulized Di-Ethylhexyl Sebacate (DEHS) in order to measure lung particle deposition. This process was conducted three times for each participant. Inhaled and exhaled particles were measured using an optical aerosol spectrometer (PALAS, WELAS 2100).

The ratio between inhaled and exhaled particle concentrations was then calculated for each particle size group to determine deposition rates in each participant (Kim & Jaques, 2004). Breathing frequency and heart rate were obtained from the Bio Harness 3.0 heart rate monitor (section 3.3.2.1). A default model value for the upper respiratory tract (URT) as 50 ml was used and Functional Residual Capacity (FRC) was calculated from the formula presented by Stocks and Quanjer (1995) as follows:

FRC (in litre) for adult = 2.34 * Height (meter) + 0.01 * Age (years)-1.09 (3.6)

For lung function testing, the Micro I Diagnostic Spirometry was used to determining Forced Expiratory Volume in one second (FEV1) of lung function for each participant. The test was conducted at the laboratory of the School of Public Health, Curtin University. This device is a compact device with a rechargeable battery and is portable to use (Figure 3.11).

Figure 3.11: Micro I spirometer (Source: CareFusion)

Participants were asked to sit upright in a chair with legs uncrossed and feet flat on the ground. A nose clip was used to make sure that air did not come out of the nose during the test. The participants were also asked to follow certain steps, including: a) breathe in completely and pause for less than 1 second; b) Place mouthpiece in their mouth and close their lips to form a good seal then blow the air out as fast, hard and far as possible until unable to blow any longer; and c) Repeat the same procedures three times to grab the best FEV1 of all (according to the operating manual from CareFusion).

3.4.3.2 Multiple-Path Particle Dosimetry model

As mentioned in section 1.1 (introduction chapter), the Multiple-Path Particle Dosimetry (MPPD V3.04) model calculates the deposition and clearance of monodisperse and polydisperse aerosols (deposition probability) in different regions of the lungs – i.e., Head, TB and Pulmonary (section 1.1) – for particles ranging in size from ultrafine (0.01 µm) to coarse (up to 100 µm). This model calculates the predicted deposition of particles during one breathing cycle, which includes inspiration, pause and expiration.

To calculate the particle deposition in the lungs of the cyclists, the following steps were followed:

a. **Step 1:** Run the MPPD model: The parameters used by MPPD model to calculate deposition comprises four areas includes particle properties (density, diameter and aspect ratio), exposure scenario and air morphometry of each cyclist and the possibility to evaluate the

deposition or deposition and clearance of the particles. The model initially calculates particle deposition fraction (deposition probability) during one breathing cycle that includes inspiration, pause and expiration. Deposition fraction is the fraction of inhaled particles that deposit in the lungs. Given breathing parameter (breathing frequency per minute from breath rate monitor) and lung's parameter (Functional Residual Capacity from spirometer and FRC from equation 3.6 and a default model value for URT as 50 ml), the amount of particles deposited in the lung can be calculated from the Mass Mean Diameter (MMD) from the DustTrak (explained below). More detail regarding the parameters input used in the MPPD model is shown in Table 3.2.

Table 3. 2: Parameters input used in the MPPD Model

Cycling Pattern	Male						Female					
	Functional Residual Capacity (litre)			Breathing frequency per minute			Functional Residual Capacity (litre)			Breathing frequency per minute		
	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.
High-speed ride	3525.67	2977.60	3879.60	36	17	55	3284.36	2900.38	3663.70	36	21	51
Low-speed ride	3526.17	2977.60	3879.60	28	18	50	3298.06	2900.38	3663.70	29	17	44
Respiration Parameters	**Airway Morphometry**											
Species	Human											
Model	Yeh/Schum 5-lobe											
Upper Respiratory Tract Volume (ml)	50											
	Particle Properties											
Density (g/cm^3)	1											
Aspect Ratio	1.0											
Mass Median Diameter (MMD) µm range	0.1-10.0											
Geometric Standard Deviation (g/cm^3) (Diameter)	1											
Geometric Standard Deviation (g/cm^3) (Length)	1											
Correlation (µm)	0.0											
	Exposure Scenario											
Acceleration of Gravity (cm/s^2)	981.0											
Body Orientation	Upright											
Tidal Volume (ml)	2000											
Inspiration Fraction	0.5											
Breathing Scenario	Oronasal-Normal Augmenter											

The output from this model gives the 'particle deposition probability' curve where the x-axis represents the size of the particles and y-axis represents the number of particles of that particular size. This information was used to determine the 'deposition of particle size distribution' curve, which is also the graphical representation of the 'frequency function'. This curve showed an accurate picture of the particle deposition in the lungs according to their size.

The calculation of Mass Median Diameter from DustTrak and calculation of the fraction of the total mass of the deposited particles are explained below

a. Mass Median Diameter calculation from DustTrak

The 'number' or 'count' of particles discussed in step 1 are terms used to discuss the distribution of particles. Another term used is 'mass distribution' as a function of particle size. As mentioned earlier, the particle distribution number gives the fraction of the total number of particles in any size range, whereas the distribution of mass gives the fraction of the total mass of the particles contributed by particles in any size range.

In order to determine the median of the size classes (PM_1, $PM_{2.5}$, PM_4 and PM_{10}), the midpoint (median) was calculated after subtracting smaller size fractions (Hinds, 1999). The median of the mass-based distribution is called a mass median diameter (MMD). MMD is defined as the diameter for which half of the mass is contributed by particles larger than the MMD and half by particles smaller than the MMD. It is the diameter that divides the graphical representation of the distribution of mass into two segments of equal area.

The MMD counter's size classes – including 0.1 µm, 0.562 µm, 1.778 µm, 3.652 µm and 6.494 µm – were determined according to Hinds (1999) formula below and the DustTrak size class data (TSI, 2018):

$$\mathbf{d_{mm} = \sum m_i d_i \text{ divided by } M} \qquad \mathbf{(3.7)}$$

Where, d_{mm} is mass median diameter, m_i is mass collected from DustTrak, d_i is the midpoint of particles, M is total of the mass collected.

The midpoint of the particles is calculated as follows:

If the midpoint of PM_1 is M_1, then the midpoint of $PM_{2.5}$ is $M_{2.5}$-M_1; for PM_4 is M_4-$M_{2.5}$ and for PM_{10} is PM_{10}-M_4.

b. As mentioned earlier, the particle distribution number gives the fraction of the total number of particles in any size range whereas the distribution of mass gives the fraction of the total mass of the particles contributed by particles in any size range. Calculation of the fraction of the total mass of the deposited particles

As mentioned above (in section a), the distribution of particle mass gives the fraction of the total mass contributed by particles measured by DustTrak, USMPS and P-Trak ($PM_{0.1}$).

The fraction of the deposited total number of particles (df) with a different diameter between dp + dd_p is calculated by the formula below (Hinds, 1999):

$$\mathbf{df = f(d_p)\, dd_p} \qquad \mathbf{(3.8)}$$

Where, $f(d_p)$ is the frequency function and dd_p is the differential interval of particle size. This function is the mathematical representation of the curve and the area under the curve is calculated by the following formula:

$$\int_0^\infty \boldsymbol{f(dp)ddp = 1.0} \qquad \mathbf{(3.9)}$$

In frequency functions, the interval of particle size may be from zero to infinity, as in equation 3.9, or it may be between the given size of the particle between 'a' and 'b', or it may be even a tiny interval dd_p.

The area under the frequency function curve between two sizes ('a' and 'b') equals the fraction of particles whose diameters fall within this interval. Mathematically, this is expressed as follows:

$$\boldsymbol{fab = \int_a^b f(dp)ddp} \quad \textbf{(3.10)}$$

In this equation, the fraction of particles with diameters exactly equal to diameter b is zero, because the interval width is zero.

$$\boldsymbol{fbb = \int_b^b f(dp)ddp = 0} \quad \textbf{(3.11)}$$

The size distribution information can also be presented as a cumulative distribution function, $F(d_p)$, which is defined by the formula below:

$$\boldsymbol{F(a) = \int_0^a f(dp)ddp} \quad \textbf{(3.12)}$$

Where F(a) gives the fraction of the particles having a diameter less than 'a'.

b. Step 2: Calculation of the deposition dose of particles in the lungs

Deposition doses of particles are the function of ambient PM concentrations, the deposition fraction (DF) in each region, the distance travelled (D) and the weight of participants (wt). The following equations were used to estimate the deposition dose (Sánchez-Soberón et al., 2015):

Absolute total dose (µg) (ATD) = PMresp x DFpm **(3.13)**

Where, PM_{resp} =Average concentration of respirable particles and DFpm=deposition fraction of PM from model

Absolute Dose per cyclist (µg/km) = ATD/Wp/D **(3.14)**

Where ATD is calculated from equation 3.12, where 'Wp' is the weight of each participant, 'D' is the distance travelled by each participant, i.e. 5 km at each low and high exertion rates.

3.5 Data management and statistical analysis

After completing the environmental and personal PM monitoring, the logged data was recovered from the Dust Trak, Testo, USMPS, P-Trak, Anemometer and TinyTag for further analysis. Negative data from the DustTrak were considered missing values.

The demographic statistics of the study participants were presented in frequency and percentages for categorical variables (age and smoking status). The chi-square test was applied to examine the association between the cyclists' characteristics (age and smoking status) and genders. To assess the association between age group and the four routes, Fisher's exact test was applied, whereas the chi-square test was used to examine the association between smoking status ('never-smoked' and 'currently non-smoker') and the four cycling routes. The continuous variables (height, weight, BMI, and heart and breath rate) were summarised by using the median and interquartile range (IQR). The Mann-Whitney U test was applied to examine the differences in these physical characteristics between the two genders, whereas the Kruskal Wallis test was applied to examine the difference between the four routes since the data was not normally distributed.

Median, IQR, minimum and maximum values of the collected data for the PMs (PM_1, $PM_{2.5}$, PM_4, PM_{10}, and PM_{total}) and selected gases (CO, NO_2, NO and SO_2) were presented for each cycling route. Since the data did not follow a normal distribution, even after the log transformation, the Kruskal Wallis Test was applied to compare the PM concentrations among the four routes. The Mann-Whitney U test was further used as a post hoc test to examine the PM difference between each pair of routes if the Kruskal Wallis test revealed an overall significant difference. Furthermore, to determine the correlation between the meteorological parameters (rainfall, humidity, wind velocity and temperature) and the air pollutant variables (PMs and selected gases), Spearman's rank correlation coefficient was calculated. The above procedure was carried out first on the overall data and then replicated separately for data disaggregated by 'cold' and 'warm' seasons to examine the role of the season on data variations between routes. To compare the repeated measurements of ambient PMs and gas concentration the Wilcoxon signed rank test was applied.

To compare the personal concentration of cyclists between four routes, the Kruskal Wallis test was applied as the data was not normally distributed. Further, the Mann-Whitney U test was used to examine the personal exposure difference between each pair of routes. To compare the repeated measures of the personal concentration of the cyclists in the cold and warm seasons, the Wilcoxon signed-rank test was applied.

The deposition dose of PM of different sizes (0.1 µm, 0.562 µm, 1.778 µm, 3.652 µm and 6.494 µm) in different regions of the respiratory tract (Head, Pulmonary and Tracheobronchial) was obtained from the MPPD Model (refer section 3.4.3). The deposition dose of PM concentrations was presented in mean, standard deviation, median and IQR. One Way-ANOVA was applied to compare the PM concentration between the four routes. The Bonferroni test was further used as a post hoc test to examine the PM difference between each pair of routes if the One Way-ANOVA test revealed an overall significant difference.

The regional cumulative deposition curves as a function of particle size in both the pulmonary and TB regions of the respiratory system were then compared between four different groups: a) four routes; b) males and females; c) high and low cycling exertions; and d) cold and warm seasons.

All data were entered into and analysed using IBM SPSS version 22.0 (IBM SPSS Statistics for Window, Version 22.0. Armonk, NY: IBM Corp). All tests were two-sided and a p-value of less than 0.05 was considered statistically significant.

3.6 Ethical considerations

This project required Curtin Human Research Ethics Committee (HREC Ethics A) approval, which was achieved on 18th September 2014. According to the ethics requirements, all potential participants were given an information sheet explaining the purpose of the study, and those who were willing to participate were asked to sign a 'consent to participate' form. All the study data were kept strictly confidential and stored in the 'R Drive' of Curtin University. Throughout the study, the privacy of the participants was protected at all times by

creating a password in the laptop to access the data. Also, the filled-in forms are stored in a locker provided by Curtin University. The participants were also informed that they have the right to withdraw at any point in the study. Throughout the study, only five participants dropped out of the research due to various reasons. The study received ethics approval from the Human Research Ethics Committee at Curtin University before commencing, with approval number HR 183/2014.

CHAPTER 4 : RESULTS

4.1 Introduction

This chapter describes the results related to the demographic features of the study subjects, including their heart and breathing rates. The chapter also provides the results from the air quality and personal exposure monitoring to respirable particles. The outcome of the MPPD modelling in relation to the lung deposition dose of particulate matter is also presented in this chapter.

4.2 Demographic characteristics of the study subjects

A total of 122 individual cyclists participated in the study and their demographic characteristics and physical parameters are presented by gender in Table 4.1 and Table 4.2, respectively.

Table 4. 1: Demographic characteristics of the participants (N=122) as per gender

Participants' Characteristics	**Male** **n (%)**	**Female** **n (%)**	**Total** **n (%)**	**p-value[@]**
Age (years)				
20–29	10 (10.1)	4 (17.4)	14 (11.5)	
30–39	20 (20.2)	6 (26.1)	26 (21.3)	
40–49	40 (40.4)	8 (34.8)	48 (39.3)	
>49	29 (29.3)	5 (21.7)	34 (27.9)	0.210
Smoking Status				
Currently non-smoker	29 (29.3)	4 (17.4)	33 (27.0)	
Never smoked	70 (70.7)	19 (82.6)	89 (73.0)	0.249

[@]Chi-square test was applied to examine the proportional difference of age and smoking status between two gender groups

The participating cyclists were males and females aged between 20 and 55 years (Table 4.1). Among 122 participants, 99 (81.15%) were males and 23 (18.85%) were females. Most of the males (40.4%) and females (34.8%) were in the age group of 40–49 years. With regard to smoking status, of 122 participants, 33 (27%) were 'currently non-smokers' and 89 (73%)

'never smoked'. The proportion of 'currently non-smoker' was higher among males (29.3%) than females (17.4%) whereas more females (82.6%) reported that they had 'never smoked' compared to males (70.7%). No significant association was found between smoking status and gender.

Table 4. 2: Physical measurements of the participants (N=122) as per gender

Physical Measurements	**Male (n=97)** **Median (IQR)**	**Female (n=21)** **Median (IQR)**	**Total** **Median (IQR)**	**p-value[#]**
Height (m)	1.780 (0.094)	1.690 (0.123)	1.763 (0.100)	<0.001
Weight (kg)	80.600 (13.250)	64.000 (12.550)	78.100 (16.025)	<0.001
BMI (kg/m^2)	25.217 (4.040)	23.125 (4.773)	24.871 (4.315)	0.003
Heart Rate (bpm)				
Low-Exertion Heart Rate	119.107 (21.632)	124.309 (31.341)	119.343 (22.584)	0.420
High-Exertion Heart Rate	146.806 (21.469)	151.585 (26.392)	147.471 (20.711)	0.817
Breathing Rate (bpm)				
Low-Exertion Breathing Rate	26.720 (6.551)	29.132 (8.479)	26.774 (6.374)	0.165
High-Exertion Breathing Rate	35.491 (9.649)	41.394 (4.475)	36.755 (9.502)	0.133

[#] Mann-Whitney U test was applied to examine the difference in physical measurements between two gender groups, n: number of participants

The height, weight and Body Mass Index (BMI) of each participant were measured and the summary statistics are presented in Table 4.2. The test showed that, on average, the BMIs of male participants were significantly higher than the female participants ($p<0.05$).

The study also recorded the heart and breathing rates of each male and female participant during the high- and low-exertion ride and the results are reported in Table 4.2. It is evident that a higher heartbeat rate was recorded for females when compared with male participants during both high- and low-exertion cycling; however, no significant gender differences were observed during high- ($p=0.817$) and low- (0.420) exertion cycling.

Similarly, female participants had a higher breathing rate during both high and low exertion but the differences were also not significant.

The demographic characteristics and physical measurements of the study subjects were compared based on the route(s) cycled and results are presented in Table 4.3 and Table 4.4, respectively. As mentioned in the methodology chapter (section 3.2.2), the participants were

asked to cycle more than one route in addition to their original route due to the insufficient number of cyclists recruited for the study. The two tables include only those participants who were assigned to their original cycling route before repeating any other route.

Table 4. 3: Demographic characteristics of participants (N=122) per cycling route

Participants Characteristics	Route 1	Route 2	Route 3	Route 4	Total	P-value*
	n (%)	n (%)	n (%)	n (%)	n (%)	
Age (years)						
20–29	4 (10.3)	1 (10.0)	2 (5.3)	7 (20.0)	14 (11.5)	
30–39	9 (23.1)	3 (30.0)	6 (15.8)	8 (22.9)	26 (21.3)	
40–49	15 (38.5)	2 (20.0)	19 (50.0)	12 (34.3)	48 (39.3)	0.809
>49	11 (28.2)	4 (40.0)	11 (28.9)	8 (22.9)	34 (27.9)	
Gender						
Male	32 (82.1)	9 (90.0)	29 (76.3)	29 (82.9)	99 (81.1)	0.850
Female	7 (17.9)	1 (10.0)	9 (23.7)	6 (17.1)	23 (18.9)	
Smoking Status						
Currently non-smoker	9 (23.1)	3 (30.0)	11 (28.9)	10 (28.6)	33 (27.0)	0.575
Never smoked	30 (76.9)	7 (70.0)	27 (71.1)	25 (71.4)	89 (73.0)	

** Chi-square test (Fisher's exact test for age) was applied to examine the proportional difference between the four routes*

According to Table 4.3, the highest proportion of the participants belonged to the age group 40–49 years of age, followed by the 50 years and above. This was true in all groups except for route 2; however, no significant association was found between age groups and cycling routes (p=0.809).

The percentage of male participants was observed to be higher compared to females in each cycling route but the difference was not significant (p=0.850).

A higher proportion of currently non-smoking cyclists were found in route 2 (30%) and the lowest proportion was found for route 1 (23.1%). However, the opposite was found in terms of 'never smokers'. The Chi-Square test showed that there were no significant differences between the two groups of smoking status (p=0.575).

Table 4. 4: Physical measurements of participants (N=122) per cycling route

Physical Measurements	Route 1 Median (IQR)	Route 2 Median (IQR)	Route 3 Median (IQR)	Route 4 Median (IQR)	Total Total Median (IQR)	p-value∞
Height (m)	1.773 (0.085)	1.747 (0.111)	1.752 (0.147)	1.770 (0.114)	1.763 (0.103)	0.774
Weight (kg)	82.100 (16.475)	76.250 (25.400)	75.900 (18.100)	80.600 (15.450)	78.100 (16.025)	0.098
BMI (kg/m^2)	25.471 (5.342)	23.669 (5.559)	24.098 (3.645)	25.823 (4.142)	24.871 (4.315)	0.208
Heart Rate (bpm)						
Low-Exertion Heart Rate	118.264 (19.520)	108.377 (0.000)	120.495 (16.872)	123.583 (27.647)	119.343 (22.584)	0.233
High-Exertion Heart Rate	144.316 (21.399)	154.902 (44.593)	146.441 (13.520)	150.419 (26.170)	147.471 (20.711)	0.698
Breathing Rate						
Low-Exertion Breathing Rate	26.538 (5.069)	26.280 (0.000)	28.998 (7.096)	24.675 (8.815)	26.774 (6.374)	0.150
High-Exertion Breathing Rate	37.575 (10.985)	35.552 (16.120)	38.900 (7.317)	35.980 (7.9969)	36.755 (9.501)	0.738

∞Kruskal Wallis test was applied to examine the difference in physical measurements between the four routes.

Table 4.4 illustrates the summary statistics of height, weight, BMI, and heart and breathing rate of each participant according to the cycling routes. The table shows that the average height and weight of the participants who followed route 1 were higher compared to cyclists from other routes, whereas the BMI was higher among the participants from route 4. However, no significant difference in the height (p=0.774), weight (p=0.098) or BMI (P=0.208) were observed between the four cycling groups.

This table also reports the average heart and breathing rate of cyclists in each route during high- and low-exertion cycling. The highest average heart rate was recorded among participants riding in route 4 and the lowest among those in route 2, but the difference was not statistically significant (p=0.233).

With regard to the breathing rate, the cyclists who cycled in route 3 had higher breathing frequency during both high- ($p=0.738$) and low- ($p=0.150$) exertion riding compared to the others routes, but no significant difference was established.

4.3 Background concentration of particulate matter

The distribution levels of ambient PM concentrations (PM_1, $PM_{2.5}$, PM_4, PM_{10} and PM_{total}) according to the cycling route are presented in Table 4.5.

The concentration of smaller particles (PM_1) tended to be higher in route 1, while route 3 was found to have a higher concentration of larger particles ($PM_{2.5}$–PM_{10}). The results demonstrated that the PM concentrations were statistically significant between cycling routes ($p<0.05$) except for PM_{total}.

Table 4.5: Comparison of ambient PM concentrations between cycling routes

Particulates	N	PM Concentrations (µg/m^3) Median (IQR)	Min	Max	p-value$^{\infty}$
PM_1 µm					
Route 1	130	12.972 (8.823)$^{\#\wedge}$	3.333	73.572	
Route 2	36	9.924 (5.604) $^{\#\&}$	4.000	23.042	
Route 3	144	12.540 (7.151) $^{\&\$}$	5.800	34.713	<0.001
Route 4	144	8.0661 (6.740) $^{\wedge\$}$	2.631	135.019	
$PM_{2.5}$ µm					
Route 1	130	12.974 (8.691) $^{\#\wedge}$	3.433	74.287	
Route 2	36	10.569 (6.952) $^{\#\&}$	4.108	24.325	
Route 3	144	13.283 (7.194) $^{\&\$}$	6.400	34.968	<0.001
Route 4	144	8.137 (7.767) $^{\wedge\$}$	2.770	137.057	
PM_4 µm					
Route 1	130	12.979 (7.957) $^{\# \wedge}$	3.535	75.168	
Route 2	36	10.992 (8.321) $^{\#\&}$	4.415	25.042	
Route 3	144	14.077 (7.179) $^{\$\&}$	6.602	35.272	<0.001
Route 4	144	8.484 (8.412) $^{\wedge\$}$	2.810	138.629	
PM_{10} µm					
Route 1	130	13.510 (11.821) $^{\#\wedge}$	3.595	76.914	
Route 2	36	12.819 (10.101) $^{\#}$	5.123	27.475	
Route 3	144	14.863 (8.739) $^{\$}$	6.657	35.899	0.001
Route 4	144	10.660 (14.851) $^{\wedge\$}$	2.960	140.238	
PM_{Total} µm					
Route 1	130	13.868 (18.176)	3.595	77.567	
Route 2	36	15.879 (10.601)	5.985	31.258	
Route 3	144	15.952 (12.556)	6.695	36.053	0.082
Route 4	144	12.444 (15.119)	2.980	140.610	

∞ Statistically significant difference between the four routes was assessed by using the Kruskal-Wallis Test,
#, ^, &, $ same characters indicate a statistically significant difference between two routes using the Mann-Whitney U test;
n indicates the number of observations.

In order to incorporate seasonal differences in ambient PM concentrations, air quality was monitored twice per year, in warm and cold seasons.

Table 4.6 shows the comparison of ambient PM concentrations between the four cycling routes for each season. The results from cold season monitoring showed that the concentration of particles (PM_1–PM_{total}) was recorded to be higher in route 3 compared to the other three routes. However, during the warm season, the highest PM concentrations were measured in route 1 and the differences were significant between the routes in each season ($p<0.05$).

Overall, the results showed that routes 1 and 3 were the most polluted while route 2 was the safest cycling route in terms of PM exposures.

Table 4.6: Comparison of ambient concentrations of PM between routes per season

Particulates	Cold Season					Warm Season				
	n	PM (µg/m³) Median (IQR)	Min	Max	p-value℃	n	PM (µg/m³) Median (IQR)	Min	Max	p-value℃
PM_1 µm										
Route 1	52	10.609 (7.410)#$	6.726	32.633		78	13.156 (9.224)$#	3.333	73.572	
Route 2	18	5.390 (6.093) #&	4.000	15.240	0.002	18	10.250 (7.146) #	5.177	23.042	<0.001
Route 3	64	13.044 (7.910)&@	6.538	34.713		80	9.285 (6.851) @	5.800	20.942	
Route 4	52	8.565 (8.243)$@	2.631	29.065		92	7.695 (6.740) $@	3.074	135.019	
$PM_{2.5}$ µm										
Route 1	52	11.586 (7.724)#	6.779	33.300		78	13.331 (8.628)$	3.433	74.287	
Route 2	18	5.569 (7.304) #&	4.108	15.520	0.003	18	10.569 (7.712)	5.478	24.325	0.002
Route 3	64	14.066 (7.798)&@	6.569	34.968		80	9.700 (7.600) @	6.400	21.835	
Route 4	52	9.234 (8.731)@	2.770	30.145		92	8.081 (7.273) $@	3.022	135.057	
PM_4 µm										
Route 1	52	12.559 (8.255)#$	6.974	34.727		78	13.452 (7.957) $	3.535	75.163	
Route 2	18	5.846 (8.686)#&	4.415	15.840	0.001	18	10.992 (7.968)	6.013	25.042	0.001
Route 3	64	14.550 (7.443)&@	6.602	35.272		80	10.368 (8.044) @	6.905	22.800	
Route 4	52	10.012 (9.481)$@	2.810	31.350		92	8.359 (8.123) $@	3.432	138.629	
PM_{10} µm										
Route 1	52	13.428 (9.588 #$	7.132	37.627		78	13.510 (11.504)^ $	3.595	76.914	
Route 2	18	6.615 (10.616)#&	5.123	17.106	0.002	18	12.819 (6.662) %	9.743	27.475	0.006
Route 3	64	14.863 (6.060)&@	6.657	35.899		80	11.798 (11.057)^	7.536	24.708	
Route 4	52	12.000 (11.785)$@	2.960	33.925		92	9.362 (14.667) $%	4.395	140.238	
PM_{Total} µm										
Route 1	52	13.571 (10.973)#^	7.153	41.656		78	17.023 (25.647)^ $	3.595	77.567	
Route 2	18	7.015 (11.065)#&	5.985	18.494	0.002	18	15.916 (6.442) %	14.398	31.258	0.018
Route 3	64	15.952 (6.653)^&@	6.695	36.053		80	12.638 (14.398)^	7.959	35.964	
Route 4	52	13.931 (15.416)@	2.980	35.846		92	11.237 (38.545) $%	5.021	140.610	

℃Statistically significant difference between the four routes was assessed by using the Kruskal-Wallis Test in each season,

#, ^, &, $, @, % same character indicates a statistically significant difference between two routes using the Mann-Whitney U test; n indicates the number of observations.

The ambient PM concentrations were also compared between the cold and warm seasons for each route and the results are presented in Table 4.7.

Table 4.7: Seasonal difference in ambient PM concentrations per cycling route

Particulates	Cold Season		Warm Season		p-value¥
	n	PM Concentrations (µg/m³) Median (IQR)	n	PM Concentrations (µg/m³) Median (IQR)	
PM_1 µm					
Route 1	52	10.609 (7.410)	52	13.395 (16.394)	0.029
Route 2	18	5.390 (6.093)	18	5.250 (7.146)	0.327
Route 3	64	13.044 (7.910)	64	9.285 (7.012)	0.385
Route 4	52	8.565 (8.243)	52	7.695 (6.740)	0.985
Overall	186	8.903 (7.965)	268	10.250 (7.685)	0.256
$PM_{2.5}$ µm					
Route 1	52	11.586 (7.724)	52	13.800 (16.437)	0.032
Route 2	18	5.569 (7.304)	18	5.600 (7.712)	0.327
Route 3	64	14.067 (7.798)	64	9.700 (7.600)	0.434
Route 4	52	9.234 (8.731)	52	8.081 (7.273)	0.978
Overall	186	9.367 (7.724)	268	10.569 (8.906)	0.277
PM_4 µm					
Route 1	52	12.559 (8.255)	52	14.481 (16.391)	0.035
Route 2	18	5.846 (8.686)	18	5.992 (7.968)	0.445
Route 3	64	14.550 (7.443)	64	10.368 (8.044)	0.442
Route 4	58	9.906 (11.201)	58	8.454 (8.123)	0.327
Overall	186	10.042 (8.208)	268	10.992 (8.570)	0.121
PM_{10} µm					
Route 1	52	13.428 (9.588)	78	15.267 (17.020)	0.024
Route 2	18	6.615 (10.616)	18	6.820 (6.662)	0.144
Route 3	64	14.863 (6.060)	80	14.550 (9.386)	0.313
Route 4	52	11.076 (12.187)	92	9.362 (14.667)	0.197
Overall	186	12.000 (9.486)	268	12.900 (13.224)	0.056
PM_{total} µm					
Route 1	52	13.571 (10.973)	52	17.867 (34.370)	0.002
Route 2	18	7.015 (11.065)	18	15.906 (6.442)	0.031
Route 3	64	15.952 (6.653)	64	16.510 (14.218)	0.846
Route 4	58	14.040 (13.841)	58	13.237 (38.545)	0.167
Overall	186	15.471 (10.728)	268	14.482 (18.153)	0.005

¥ *Statistically significant difference between the two seasons was assessed by using Wilcoxon signed-rank test for each route, n indicates the number of observations.*

The overall analysis indicated that the concentration of each particle size was recorded as higher during the warm season (route 1 and 2) compared to the cold season. However, no significant differences were observed between them except route 1.

4.4 Background concentration of selected gases

During the study, the ambient level of selected gases – CO, NO, NO_2 and SO_2 – was monitored in the four cycling routes and the results are presented in Table 4.8. The study results indicated that the median levels of CO and SO_2 were higher in route 4 compared to other routes but the differences were not significant ($p>0.05$). However, higher concentrations of NO and NO_2 were recorded for route 3 and their differences were significant ($p<0.05$). Overall, route 3 appeared to be the most polluted route in terms of gas concentrations.

Table 4.8: Comparison of ambient concentration of gases between routes

Gases	n	Gas Concentration Median (IQR)	Min.	Max.	p-value$^{\infty}$
CO (ppm)					
Route 1	24	3.741 (7.591)	1.873	9.823	0.088
Route 2	6	0.808 (0.966)	0.809	1.774	
Route 3	26	2.054 (2.771)	1.943	5.815	
Route 4	64	4.054 (13.586)	0.632	57.488	
NO (ppm)					
Route 1	24	0.205 (0.30)$^{\#}$	0.000	0.784	<0.001
Route 2	6	0.099 (0.477)^	0.100	0.577	
Route 3	26	0.377 (0.236) $^{\#\wedge\%}$	0.059	1.129	
Route 4	64	0.193 (0.379) $^{\%}$	0.022	1.746	
NO_2 (ppm)					
Route 1	24	0.117 (0.170) $^{\#}$	0.004	0.443	<0.001
Route 2	6	0.096 (1.421)$^{\&}$	0.0.96	1.517	
Route 3	26	0.359 (0.498) $^{\#\%}$	0.089	1.485	
Route 4	64	0.091 (0.075)$^{\& \%}$	0.015	1.310	
SO_2 (ppm)					
Route 1	24	0.191 (0.288)	0.001	0.390	0.221
Route 2	6	0.074 (0.036)	0.074	0.110	
Route 3	26	0.201 (0.150)	0.010	0.674	
Route 4	64	0.241 (0.385)	0.016	0.581	

$^{\infty}$Statistically significant difference between the four routes was assessed by using the Kruskal-Wallis Test, #,^,&,% Same character indicates a statistical significant difference between two routes using the Mann-Whitney U test; n indicate the number of observations.

The concentrations of the selected gases between the four cycling routes were measured during the cold as well as warm seasons and the findings are presented in Table 4.9. During

the cold season, route 4 in regards to CO, NO, SO_2 and route 3 in NO_2 appeared to be the most polluted cycling routes with respects to gas exposure. We established a statistically significant difference ($p<0.05$) in the concentration of all gases between the four cycling routes.

Due to missing data during the warm season, gas concentrations are only presented for route 1 and route 4. It appears that route 1 is more polluted than route 4 but the differences were not significant ($p>0.05$).

Overall, during the cold months, routes 3 and 4 appeared to be most polluted in terms of gases while during the warm season higher concentrations were measured in route 1.

Table 4.9: Comparison of ambient concentrations of gases between routes per season

Selected Gases	n	Cold Season				Warm Season				
		Gases Median (IQR)	Min.	Max.	p-value[∞]	n	Gases Median (IQR)	Min.	Max.	p-value[◊]
CO (ppm)										
Route 1	6	3.307 (0.000)[#]	3.307	3.307		18	4.175 (7.950)	1.873	9.823	
Route 2	6	0.808 (0.966) [#&$]	0.809	1.774	0.001	N/A	N/A			0.822
Route 3	26	2.054 (2.771) [&%]	1.943	5.815		N/A	N/A			
Route 4	40	4.297 (2.980) [$%]	0.632	18.543		24	3.177 (56.460)	1.028	57.488	
NO (ppm)										
Route 1	6	0.296 (0.000) [#]	0.296	0.300		18	0.205 (0.350)	0.000	0.784	
Route 2	6	0.099 (0.477) [#&$]	0.100	0.577	<0.001	N/A	N/A			0.347
Route 3	26	0.377 (0.236) [&]	0.059	1.129		N/A	N/A			
Route 4	40	0.432 (0.499) [$]	0.066	1.746		24	0.097 (0.147)	0.022	0.194	
NO_2 (ppm)										
Route 1	6	0.174 (0.000) [#^]	0.164	0.175		18	0.117 (0.195)	0.004	0.443	
Route 2	6	0.096 (1.421) [#]	0.096	1.517	0.048	N/A	N/A			0.507
Route 3	26	0.359 (0.498) [%]	0.089	1.485		N/A	N/A			
Route 4	40	0.134 (0.284) [^%]	0.073	1.310		24	0.075 (0.087)	0.015	0.111	
SO_2 (ppm)										
Route 1	6	0.191 (0.000) [#]	0.191	0.192		18	0.288 (0.313)	0.001	0.390	
Route 2	6	0.074 (0.036) [#&]	0.074	0.110	0.020	N/A	N/A			0.086
Route 3	26	0.201 (0.150) [&]	0.010	0.674		N/A	N/A			
Route 4	40	0.398 (0.323)	0.010	0.581		24	0.067 (0.192)	0.006	0.241	

[∞]Statistically significant difference among the four routes was assessed by using the Kruskal-Wallis Test,

◊ Statistically significant difference between the two routes was assessed by using the Mann-Whitney U test,

#, ^, &, %, $ same characters indicate a statistically significant difference between two routes using the Mann-Whitney U test; n indicates the number of observations.

4.5 Correlation between meteorological parameters and air pollutants

The Spearman Correlation Coefficient was used to investigate the correlation between particulate matter, selected gases and meteorological parameters such as rainfall, humidity, wind velocity and temperature. The results are presented in Table 4.10.

The results showed that the PMs were statistically correlated with CO, NO_2 and rainfall, but negatively correlated with temperature, as expected. Wind velocity and humidity were also negatively correlated with PMs but the relationship between them was not significant ($p>0.05$).

Regarding selected gases, the results indicated that CO and NO_2 were positively correlated with PMs. In regard to the meteorological parameters and gases, NO and NO_2 were negative but significantly correlated with temperature and humidity, whereas they were positively correlated with the level of rainfall. That is when the level of temperature and humidity increases, the concentration levels of NO and NO_2 decreases but when rainfall increases, these gases concentrations increases and they are statistically significant. In the case of CO, this gas was negatively correlated with humidity but positively with rainfall. Only wind velocity was negatively correlated with SO_2.

Table 4.10: Correlation coefficient between particulate matter, gases and meteorological parameters

Air Pollutants	PM_1	$PM_{2.5}$	PM_4	PM_{10}	PM_{total}	CO	NO	NO_2	SO_2	Wind Vel. (m/s) (r_s)	Temp. (^{0}C) (r_s)	Humidity (%) (r_s)	Rainfall (mm)(r_s)
PM (µg/m³)													
PM_1		0.996**	0.990**	0.943**	0.852**	0.463**	0.109	0.276**	-0.013	-0.040	-0.210**	-0.037	0.200**
$PM_{2.5}$	0.996**		0.997**	0.960**	0.878**	0.478**	0.083	0.238**	-0.021	-0.087	-0.198**	-0.064	0.230**
PM_4	0.990**	0.997**		0.973**	0.876**	0.450**	0.114	0.263**	0.013	-0.083	-0.040**	-0.035	0.255**
PM_{10}	0.943**	0.960**	0.973**		-0.040**	0.533**	0.111	0.222**	-0.057	-0.087	-0.102*	-0.068	0.243**
PM_{total}	0.852**	0.878**	0.876**	0.949**		0.639**	0.031	0.116	-0.189	-0.078	-0.078	-0.100	0.198**
Gases (ppm)													
CO	0.463**	0.487**	0.450**	0.533**	0.639**		-0.033	0.038	-0.295**	0.049	-0.017	-0.134**	0.164**
NO	0.109	0.083	0.114	0.111	0.031	-0.033		0.878*	0.906*	0.139	-0.235*	-0.243**	0.131*
NO_2	0.276**	0.238**	0.263**	0.222**	0.116	0.038	0.878**		0.660**	0.057	-0.383**	-0.332**	0.304**
SO_2	-0.013	-0.021	0.013	-0.057	-0.189*	-0.295**	0.906**	0.660**		-0.260*	0.007	-0.094	0.148
Meteorological Data													
Wind Vel.	-0.040	-0.087	-0.083	-0.087	-0.078	0.049	0.139	0.057	-0.260*		-0.052	0.056	-0.172*
Temp	-0.210**	-0.198**	-0.147**	-0.102*	-0.078	-0.017	-0.235**	-0.383**	0.007	-0.052		0.110*	0.043
Humidity	-0.037	-0.064	-0.035	-0.068	-0.100	-0.134**	-0.243**	-0.332**	-0.094	0.056	0.110*		-0.576**
Rainfall	0.200**	0.230**	0.255**	0.243**	0.198**	0.164**	0.131*	0.304**	0.148	-0.172*	0.043	-0.576**	

*** Spearman correlation coefficient (r_s) is significant at 5% significance level (two-tailed)*
**Spearman correlation coefficient (r_s) is significant at 1% significance level (two-tailed)*

4.6 Personal exposure levels to particles among cyclists

A comparison of the personal exposure to respirable particles (PM_4) was conducted between the four cycling routes according to gender, season and cycling exertions and the results are presented in Table 4.11.

The statistical analysis showed that the PM_4 concentration was significantly ($p=0.038$) higher among participants who cycled in route 1 and the lowest concentration was recorded in route 2.

To examine the seasonal variation in exposure to PM_4 among cyclists, the experiment was performed twice over a year during the cold and warm seasons. During the cold months, exposure to PM_4 was higher among participants who followed route 4, whereas during the warm season the concentration was higher in route 1. No significant differences were established between routes for both cold and warm seasons.

Personal exposure to PM_4 during the low- and high-speed rides were also measured and compared between the four cycling routes. During both rides (high and low), the exposure of PM_4 was higher in route 1 compared to other routes but the differences were not significant ($p>0.05$).

Overall, route 1 appears to be the most polluted route in terms of personal exposure to PM_4 compared to the others.

Table 4.11: Comparison of personal exposure to PM_4 among cyclists between routes for different seasons, gender and cycling exertion

Groups	Route	N	Personal Exposure Concentration of PM_4 (µg/m^3) Median (IQR)	p-value$^{\infty}$
ROUTES	Route 1	132	84.175 (160.625)$^{\#\$}$	
	Route 2	12	15.893 (298.763)	
	Route 3	136	36.364 (208.053)$^{\#}$	0.038
	Route 4	109	52.247 (148.381) $^{\$}$	
SEASONS				
Cold	Route 1	64	93.400 (170.034)	
	Route 2	n/a	n/a	
	Route 3	56	85.848 (256.008)	0.741
	Route 4	46	117.108 (183.507)	
Warm	Route 1	68	62.267 (163.945)	
	Route 2	12	35.893 (298.763)	
	Route 3	80	40.710 (136.682)	0.084
	Route 4	63	36.005 (93.914)	
EXERTIONS				
Low-Speed	Route 1	66	90.247 (204.714)	
	Route 2	6	15.893 (245.962)	
	Route 3	68	56.059 (280.162)	0.173
	Route 4	54	51.690 (142.528)	
High-Speed	Route 1	66	77.567 (150.952)	
	Route 2	6	31.786 (369.660)	
	Route 3	68	24.272 (138.939	
	Route 4	55	52.247 (160.051)	0.070
GENDERS				
Male	Route 1	112	87.413 (157.892)	
	Route 2	12	15.893 (298.763)	
	Route 3	110	36.659 (220.960)	0.188
	Route 4	87	56.117 (142.045)	
Female	Route 1	20	80.485 (312.328)	
	Route 2	n/a	n/a	
	Route 3	26	30.431 (130.324)	0.074
	Route 4	22	21.762 (189.220)	

$^{\infty}$Statistically a significant difference between the four routes was assessed by using the Kruskal-Wallis Test,
 # and $ same character indicates statistically significant difference between two routes using the Mann-Whitney U test;
N=number of participants.

Personal exposure to PM_4 was also compared according to seasons, exertion rates and gender between the cycling routes (Table 4.12).

Table 4.12: Comparison of personal exposure to PM_4 levels among cyclists in each route between seasons, cycling exertion and gender.

Routes	**No of participants**	**Personal Exposure Concentration of PM_4 ($\mu g/m^3$)** **Median (IQR)**	**Personal Exposure Concentration of PM_4 ($\mu g/m^3$)** **Median (IQR)**	**p-value¥**
		Cold Season	**Warm Season**	
Route 1	62	98.353 (161.938)	69.661 (160.050)	0.068
Route 2	n/a	n/a	n/a	
Route 3	56	85.848 (256.008)	20.710 (127.051)	0.026
Route 4	45	127.681 (183.854)	20.568 (103.306)	0.004
Overall	**163**	**93.400 (219.064)**	**31.133 (139.860)**	**<0.001**
Routes	**No of participants**	**Low-Speed Exertion**	**High-Speed Exertion**	
Route 1	65	87.413 (207.337)	77.567 (150.952)	0.212
Route 2	6	20.893 (245.962)	31.786 (369.660)	1.000
Route 3	68	56.059 (280.162)	24.272 (138.939)	0.013
Route 4	54	51.670 (142.528)	54.182 (162.235)	0.789
Overall	**193**	**64.020 (207.799)**	**50.693 (157.421)**	**0.030**
Routes	**No of participants**	**Male**	**Female**	
Route 1	20	85.794 (121.220)	80.485 (312.328)	0.494
Route 2	n/a	15.893 (298.763)	n/a	
Route 3	26	36.659 (220.960)	30.432 (130.324)	0.078
Route 4	n/a	21.762 (189.220)	56.117 (142.045)	0.081
Overall	**46**	**62.267 (181.017)**	**51.229 (181.033)**	**0.770◊**

¥*Statistically significant difference between the two seasons was assessed by using the Wilcoxson signed-rank test for each route,*

◊ *Statistically significant difference between the two genders was assessed by using the Mann-Whitney U test.*

Overall, exposure to PM_4 was significantly higher among cyclists during the cold season compared to the warm season ($p<0.05$).

The results also showed that the overall contribution to personal exposure was significantly ($p<0.05$) higher among cyclists cycling at low speed compared to the high-speed riding.

Furthermore, a substantial difference in PM_4 exposure between males and females was also established. In this study, the male population was exposed to higher PM_4 concentrations compared to the female population although the difference was not significant ($p=0.770$).

4.7 Deposition of particles among cyclists

The deposition of PM with different mean sizes (0.1 µm, 0.562 µm, 1.778 µm, 3.652 µm and 6.494 µm) in cyclists' lungs was computed using the MPPD model (section 3.4.3.2) and the results are described below. These sizes were selected as the midpoint of all the size fractions measured by DustTrak and U-SMPS, as mentioned in section 3.4.3.2.

4.7.1 Regional deposition fraction concentration of the particles in the respiratory system

The mean particle deposition fraction conc. in each region of the human respiratory system– head, TB and pulmonary – were compared between the cyclists and the results are presented in Table 4.13, Table 4.14 and Table 4.15 respectively.

Table 4.13: Deposition fraction conc. **of the PM in the head region of the human airway according to the cycling route**

Size (µm)	Route	n	Regional Deposition Fraction Conc. in Head Region (µg/m^3)		p-value©
			Mean ± SD	Median (IQR)	
0.1					
	Route 1	170	0.011 ± 0.004	0.012 (0.000)	0.032
	Route 2	46	0.013 ± 0.000	0.013 (0.000)	
	Route 3	178	0.015 ± 0.005$^{\#}$	0.015 (0.000)	
	Route 4	161	0.014 ± 0.000$^{\#}$	0.014 (0.000)	
0.562					
	Route 1	170	0.021 ± 0.003$^{\#}$	0.021 (0.000)	<0.001
	Route 2	46	0.022 ± 0.002$^{\$}$	0.022 (0.000)	
	Route 3	178	0.035 ± 0.036$^{\#\$\&}$	0.023 (0.000)	
	Route 4	161	0.023 ± 0.002$^{\&}$	0.022 (0.000)	
1.778					
	Route 1	170	0.089 ± 0.008$^{\#}$	0.089 (0.01)	<0.001
	Route 2	46	0.091 ± 0.005$^{\$}$	0.091 (0.01)	
	Route 3	178	0.099 ± 0.022$^{\#\$\wedge}$	0.093 (0.01)	
	Route 4	161	0.090 ± 0.008^	0.091 (0.01)	
3.652					
	Route 1	170	0.079 ± 0.006$^{\#}$	0.080 (0.000)	<0.001
	Route 2	46	0.078 ± 0.005$^{\$}$	0.080 (0.000)	
	Route 3	178	0.072 ± 0.015$^{\#\$\wedge}$	0.078 (0.010)	
	Route 4	161	0.077 ± 0.005^	0.079 (0.010)	
6.494					
	Route 1	170	0.015 ± 0.008	0.015 (0.010)	

	Route 2	46	0.013 ± 0.005	0.013 (0.010)	0.968
	Route 3	178	0.011 ± 0.057	0.011 (0.010)	
	Route 4	161	0.014 ± 0.006	0.013 (0.010	

© Statistically significant difference among the four traffic routes was assessed by using One Way ANOVA,#,@,%,&,$ same characters indicate the statistically significant difference between two routes using the Bonferroni test, n indicates the number of observations.

Table 4.13 presents the average mass concentration of PMs reaching the head region of the human airway during cycling. The results showed that the majority of small size particles (0.1−1.778) were deposited among the cyclists who cycled in route 3 compared to the other routes and the difference was significant (p=0.032). However, this is not consistent with the observations for larger particles ($PM_{3.652}$ and $PM_{6.494}$ µm) where the highest deposition was recorded for route 1 but a significant difference was only observed for $PM_{6.494}$.

Table 4.14: Deposition fraction conc. **of PM in the TB region of the lungs according to the cycling route**

Size (µm)	Route	n	Regional Deposition Fraction Conc. in TB Region (µg/m³)	p-value©	
			Mean ± SD	Median (IQR)	
0.1					
	Route 1	174	0.068 ± 0.013	0.067 (0.000)	0.521
	Route 2	46	0.069 ± 0.000	0.068 (0.000)	
	Route 3	178	0.070 ± 0.007	0.069 (0.000)	
	Route 4	162	0.069 ± 0.005	0.068 (0.000)	
0.562					
	Route 1	174	0.047 ± 0.008	0.049 (0.000)	0.033
	Route 2	46	0.049 ± 0.002	0.050 (0.000)	
	Route 3	178	0.050 ± 0.003	0.059 (0.000)	
	Route 4	162	0.048 ± 0.005	0.050 (0.000)	
1.778					
	Route 1	174	0.059 ± 0.012	0.058 (0.001)	0.360
	Route 2	46	0.060 ± 0.0051	0.060 (0.001)	
	Route 3	178	0.058 ± 0.014	0.061 (0.010)	
	Route 4	162	0.061 ± 0.007	0.060 (0.010)	
3.652					
	Route 1	174	0.130 ± 0.043	0.121 (0.050)	0.247
	Route 2	46	0.138 ± 0.036	0.130 (0.030)	
	Route 3	178	0.137 ± 0.060	0.138 (0.070)	
	Route 4	162	0.139 ± 0.040	0.131 (0.060)	
6.494					
	Route 1	174	0.254 ± 0.520	0.271 (0.030)	0.001
	Route 2	46	0.269 ± 0.019$^{\#}$	0.275 (0.020)	
	Route 3	178	0.243 ± 0.083$^{\#\&}$	0.274 (0.030)	
	Route 4	162	0.265 ± 0.032$^{\&}$	0.275 (0.030)	

© Statistically significant difference among the four traffic routes was assessed using One Way ANOVA, #,& same characters indicate the statistically significant difference between two routes using the Bonferroni test, n indicates the number of observations.

The MPPD model for the TB region (Table 4.14) showed that the highest depositions for $PM_{0.1}$ and $PM_{0.562}$ were recorded among cyclists following route 3; however, a significant difference between the four routes was only observed for $PM_{0.562}$ (p=0.033).

In regards to $PM_{1.778}$ and $PM_{3.652}$, the results showed that a higher deposition was recorded for route 4 but no significant differences between the four routes were observed for either PM.

Likewise, the deposition of large particles ($PM_{6.494}$) was found to be significantly higher among cyclists in route 2 compared to the other routes (p=0.001).

Table 4.15: Deposition fraction Conc. **of the PM in the pulmonary region of the lungs according to the cycling route**

Size (µm)	Route	n	Regional Deposition Fraction Conc. in Pulmonary Region (µg/m^3)		p-value©
			Mean ± SD	Median (IQR)	
0.1					
	Route 1	170	0.149 ± 0.020	0.151 (0.030)	
	Route 2	46	0.148 ± 0.015	0.148 (0.020)	
	Route 3	176	0.145 ± 0.019	0.144 (0.020)	0.177
	Route 4	162	0.149 ± 0.030	0.148 (0.020)	
0.562					
	Route 1	170	0.055 ± 0.014$^{\#}$	0.055 (0.010)	
	Route 2	46	0.054 ± 0.006	0.053 (0.010)	
	Route 3	178	0.051 ± 0.008$^{\#\$}$	0.051 (0.010)	0.004
	Route 4	162	0.053 ± 0.009$^{\$}$	0.054 (0.010)	
1.778					
	Route 1	170	0.112 ± 0.029$^{\#}$	0.113 (0.030)	
	Route 2	46	0.109 ± 0.019	0.107 (0.020)	
	Route 3	178	0.010 ± 0.030$^{\#\$}$	0.101 (0.030)	<0.001
	Route 4	162	0.110 ± 0.023$^{\$}$	0.109 (0.030)	
3.652					
	Route 1	170	0.183 ± 0.052$^{\#}$	0.189 (0.060)	
	Route 2	46	0.177 ± 0.039$^{\$}$	0.180 (0.050)	
	Route 3	178	0.151 ± 0.062$^{\#\$\wedge}$	0.162 (0.08)	<0.001
	Route 4	162	0.179 ± 0.046^	0.179 (0.070)	
6.494					
	Route 1	170	0.054 ± 0.039$^{\#}$	0.048 (0.050)	
	Route 2	46	0.045 ± 0.030	0.042 (0.040)	
	Route 3	178	0.037 ± 0.038$^{\#\$}$	0.027 (0.050)	<0.001
	Route 4	162	0.049 ± 0.037$^{\$}$	0.040 (0.050)	

© Statistically significant difference among the four traffic routes was assessed by using One Way ANOVA, #,$,^ same characters indicate the statistically significant difference between two routes using the Bonferroni test, n indicates the number of observations.

The deposition of PMs in the pulmonary region of the respiratory tract was observed to be significantly higher for route 1 ($p<0.05$) compared to other routes and significant for most PMs ($p=0.177$) except for $PM_{0.1}$ (Table 4.15).

It is evident from the MPPD model that the overall deposited fraction of smaller particles with a size between 0.1 and 3.652 μm was higher in the pulmonary region whereas the deposition of coarse particles ($PM_{6.494}$) was mainly concentrated in the TB region, and this is consistent for all cycling routes.

Route 1 and route 2 appeared to be the most polluted cycling routes in terms of deposition of PMs in the TB and pulmonary regions of the lungs.

4.7.2 Regional cumulative deposition of particulates in the respiratory system

Figures 4.1–4.4 show the Regional Cumulative Deposition (RCD) curves as a function of particle size in both the pulmonary and tracheobronchial (TB) regions of the respiratory system. The RCD data were calculated and averaged for all participants split variously into four different groups: the four cycling routes (Figure 4.1), gender (Figure 4.2), high and low exertion levels (Figure 4.3); and cold and warm seasons (Figure 4.4).

General trends in Figures 4.1–4.4 indicate that, in the pulmonary region, the deposition rate decreased for particles with size 4–5 μm (approximately) and no deposition occurred for particles with a size of 7 μm or larger. However, in the TB region, larger particles were the main size of deposited particles, with deposition rate decreasing above approximately 7–8 μm.

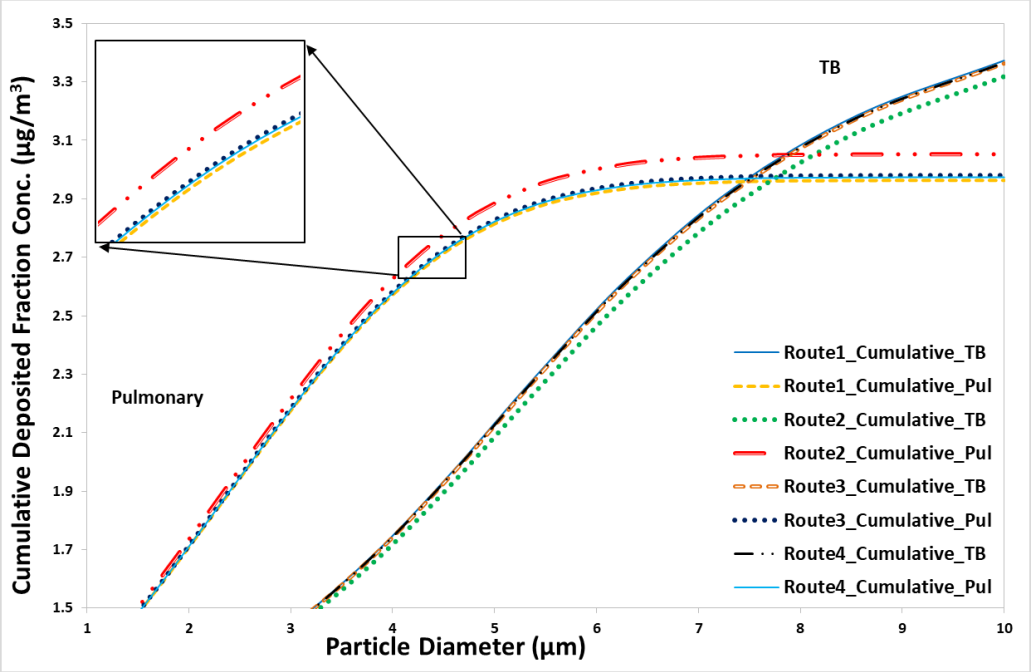


Figure 4.1: Comparison of RCD of particles in the respiratory system between cyclists cycling in the four routes

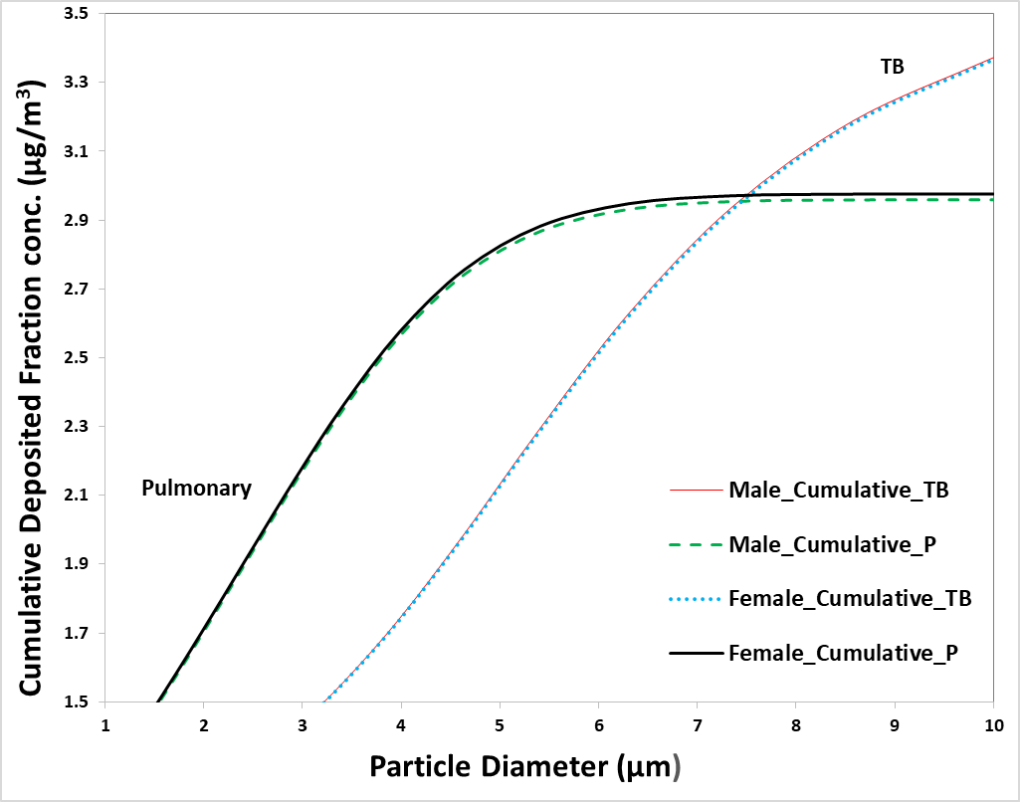


Figure 4.2: Comparison of RCD of particles according to gender

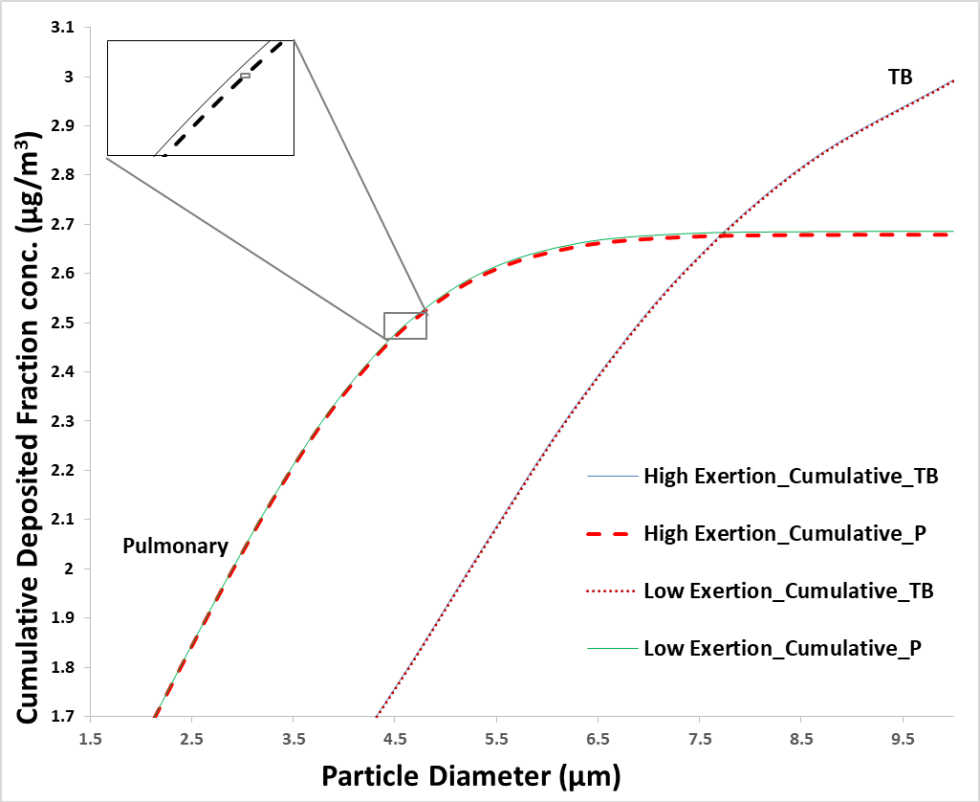


Figure 4.3: Comparison of RCD of particles according to exertion

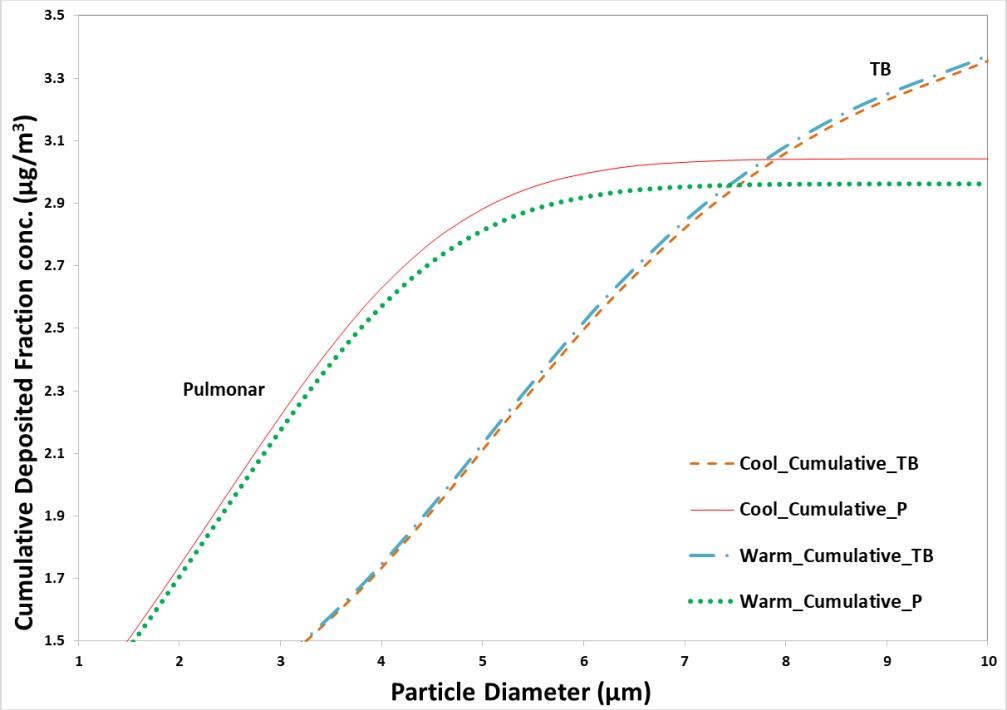


Figure 4.4: Comparison of RCD of particles according to season

In Figure 4.1, participants cycling in route 2 had the highest deposition of particles in the pulmonary region whereas those cycling in route 1 had the lowest value. This is in contrast with the TB region where the highest particle deposition was observed for participants cycling route 1 and the lowest among those cycling in route 2. The total difference in the deposited dose in the pulmonary region between route 2 (3.052 μg/m^3) and route 1 (2.963 μg/m^3) was 3%.

According to the model presented in Figure 4.2, females have higher particle deposition in the pulmonary region compared to males, with the opposite trend observed for the TB region. The total deposited dose in the pulmonary region for males was 2.960 μg/m^3 and for females was 2.976 μg/m^3, representing a 0.54% increase for females.

This study demonstrated that different exertion rates during cycling (Figure 4.3) demonstrated very little effect. The overall difference was only 0.13% between the high- and low-exertion groups in the pulmonary region. A trend of a similar magnitude was reversed in the case of the TB region.

A seasonal difference of 2.7% was observed in the deposition rate of particles in the pulmonary region with a higher deposition rate noted during the cold season (3.042 μg/m^3) compared to the warm season (2.963 μg/m^3). The trend was reversed in the TB region (Figure 4.4).

4.7.3 Relationship between ambient exposed concentration and the deposited dose of respirable PM in the respiratory system of cyclists

Figures 4.5–4.8 show the deposited dose for cyclists under different scenarios. If the Y-axis scale in these figures were changed, the figures could also represent deposition probability for each particle size and total deposition (in each region) for each scenario. It is therefore evident that some difference in particle deposition probability exists between some of the

studied groups.

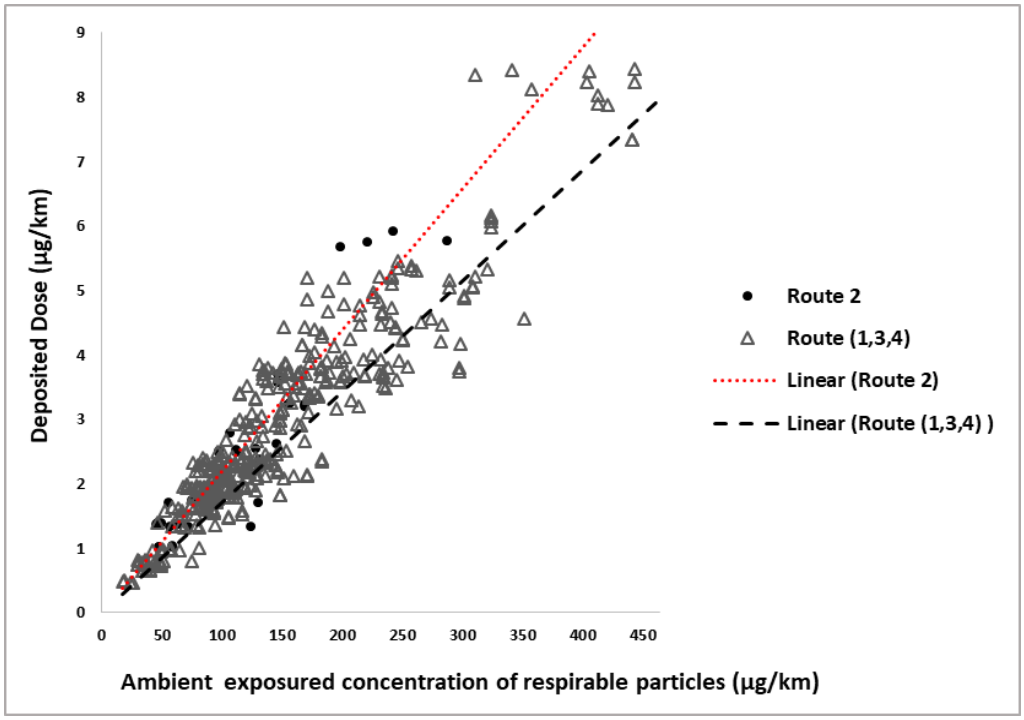


Figure 4.5: Relationship between ambient respirable exposure concentration and deposited dose in the respiratory tract of cyclists during cycling in the four routes

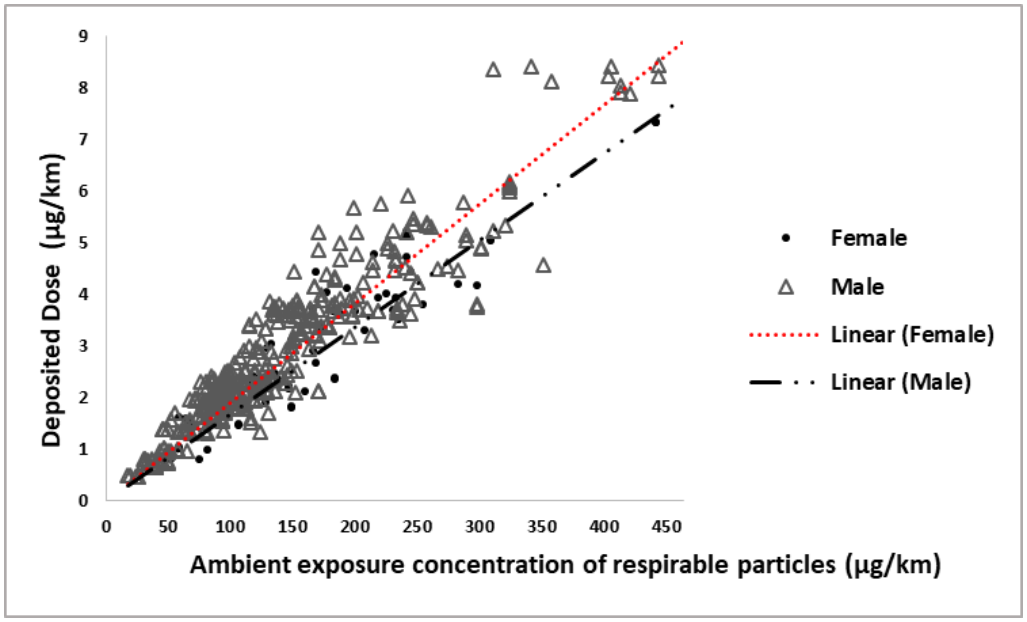


Figure 4.6: Relationship between ambient respirable exposure concentration and deposited dose in the respiratory tract of male and female cyclists

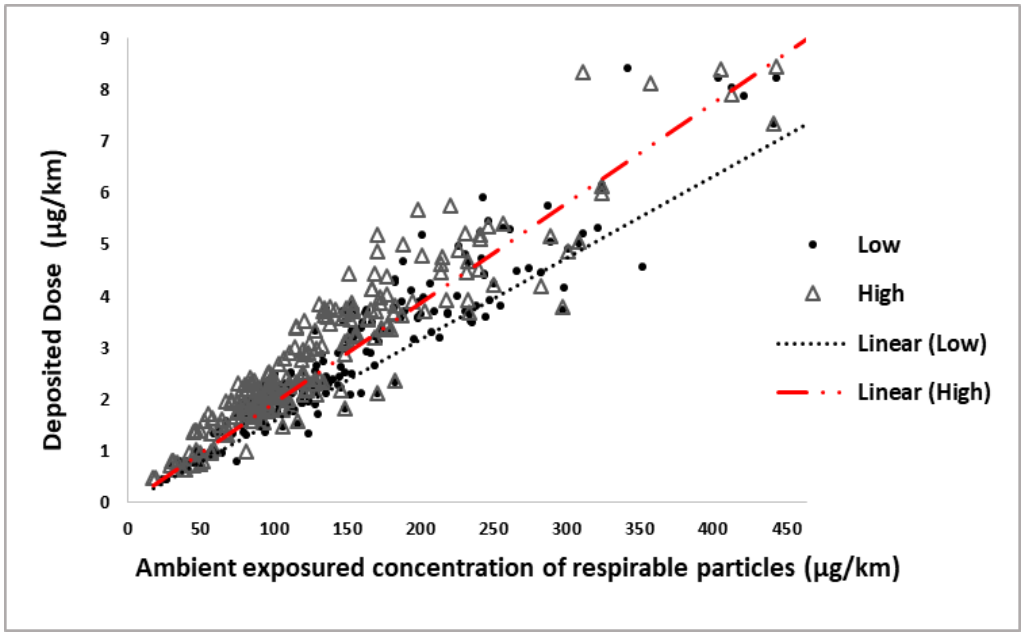


Figure 4.7: Relationship between ambient respirable exposure concentration and deposited dose in the respiratory tract of cyclists while cycling during high and low exertions

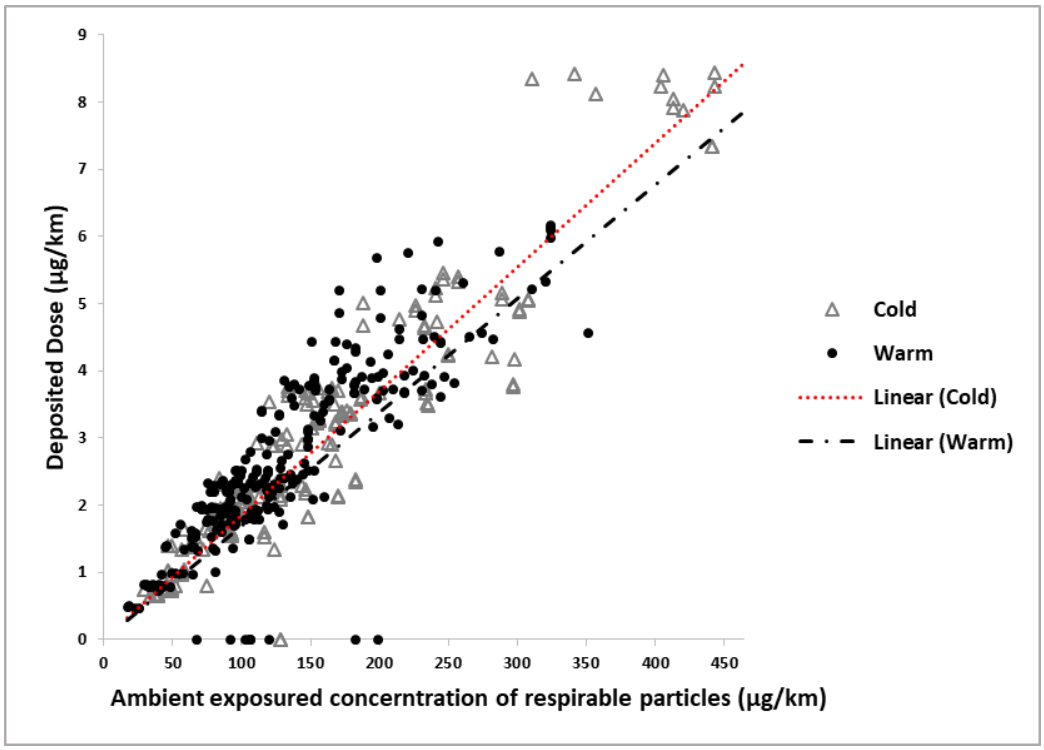


Figure 4.8: Relationship between ambient respirable exposure concentration and deposited dose in the respiratory tract of cyclists during cold and warm seasons

As a general rule, when assessing environmental air pollution exposure among individuals, the only exposed concentration of particles is considered, without correction for deposition

probability (National Environment Protection Council, 2011). It is, therefore, useful to compare the ambient concentrations of PM at first and then analyse the deposited particles in our cases (i.e. between four routes, males and females, high and low exertions, cold and warm seasons), and that may permit deposition adjustments to be made to the exposure data.

For groups (routes, gender, seasons) with the largest differences in Regional Cumulative Deposition (RCD) (see section 4.7.2), it is useful to plot the deposition dose of respirable particulate matter (PM_4) versus ambient exposure concentrations (Figures 4.5–4.8).

Figure 4.5 (route 2 vs other routes grouped) showed the most noticeable difference; however, the differences between males and females (Figure 4.6), between high and low exertion (Figure 4.7) and between cold and warm season (Figure 4.8) showed a smaller difference. As these relationships are perhaps more applicable beyond the current study, it is useful to include the equations of the straight line:

$$\boldsymbol{y = mx} \tag{4.1}$$

for route,

$$d_{r(2)} = 0.022 \cdot C_a, \tag{4.2}$$

$$d_{r(1,3,4)} = 0.017 \cdot C_a, \tag{4.3}$$

for gender,

$$d_f = 0.019 \cdot C_a, \tag{4.4}$$

$$d_m = 0.017 \cdot C_a, \tag{4.5}$$

for exertion,

$$d_h = 0.019 \cdot C_a, \tag{4.6}$$

$$d_l = 0.016 \cdot C_a, \quad (4.7)$$

and for season,

$$d_c = 0.019 \cdot C_a, \quad (4.8)$$

$$d_w = 0.017 \cdot C_a, \quad (4.9)$$

where 'C_a' is the ambient concentration, 'd' is deposited dose and the subscripts and *r, f, m, h, l, c* and *w* represents route, female, male, high, low, cold and warm, respectively. The numbers after the '*r*' subscript represent the route number.

CHAPTER 5 : Discussion

5.1 Introduction

This chapter discusses the findings of the study in four sub-sections: 1) ambient air quality; 2) effect of meteorological parameters on air pollution; 3) measured physiological parameters and exposure, and; 4) simulated dosimetry. The final section of this chapter describes the limitations of the study, which need to be considered in future research to ensure consistent results.

5.2 Ambient air quality

This study found that the concentrations of PMs in route 1 (community route) and route 3 (major route) were significantly higher than those recorded in the other two routes. In fact, an increased concentration of PM_1 was noted in route 1, whereas $PM_{2.5}$, PM_4 and PM_{10} were higher in route 3 compared to the other routes, and they are all significantly different. The overall concentrations of PM in route 1 and route 3 (8.0661–15.952 µg/m^3) were well below the NEPM standard ($PM_{2.5}$ is 25 µg/m^3 and PM_{10} is 50 µg/m^3).

Despite the lower level of vehicular movement near route 1 (Table 3.1), the study measured higher PM_1 concentrations for this route when compared to the other three routes (Table 4.5). Its close proximity to potential sources of particulate matters may have impacted the PM_1 concentrations in route 1, as discussed below.

a) There was a diesel ferry station close to the monitoring site, which could be a significant source of PM1 and other gases such as NOx, SOx, CO, CO_2 (Tichavska & Tovar, 2015). This is in agreement with other studies conducted by Cooper, Peterson, and Simpson (1996) and Peng et al. (2016), who found that cruise ships and ferries release tons of gases and $PM_{2.5}$ within port areas. Similarly, Tichavska and Tovar (2015) found that 70% of selected pollutants

(such as NOx, SOx, $PM_{2.5}$ and CO) in the local environment of ports was due to ferry vessels, and Corbett (2002) estimates 443 tons/year of PM emissions from ships.

b) The second potential explanation for the high PM_1 concentration recorded in route 1 is the close proximity of parking areas to where the monitoring equipment was set up. During measurements, continuous in- and outflow of heavy and light vehicles were observed, which could be a major source of PMs in this area.

Though studies on air pollution in parking lots are very limited, there are a few published reports that describe the scenario of PM concentration levels in parking areas. For example, in the study conducted by Zhao and Zhao (2014), higher PM_1 concentrations were measured in the morning and evening rush hours near parking areas.

c) The presence of several traffic lights (intersections) and subsequent vehicular congestion could be another reason for the recorded high PM_1 concentrations near route 1. Benson (1984) found that increased duration of traffic congestion has the potential to greatly increase pollutant emissions and degrade air quality, particularly near large roadways. The congestion usually changes the driving pattern, which results in an increased number of stops and starts, speedups and slowdowns, which increase emissions compared to normal driving (Lusk, Wen, & Zhou, 2014). Congestion also lowers the average speed of each vehicle, which increases travel time and exposure. However, congestion may also reduce the dispersion of vehicle-related pollutants as vehicle-induced turbulence depends on vehicle speed (Benson, 1984). According to Sjodin (1998), vehicles travelling at low speed also increases pollutant concentration from roadway sources, such as road dust (Sjodin et al., 1998). Taking all these into account suggests that a high volume of traffic should not be considered as a single indicator of pollution levels.

Another finding of this study was that route 3 (major route) was monitored as having higher concentrations of $PM_{2.5}$–PM_{10} compared to the other routes in the study. The possible reason for this observation might be the high volume of traffic flow (Table 3.1), which is the most common source of PMs (Barrowcliffe et al., 2002; Lin, Li, et al., 2016). The coarse fraction

component ($PM_{2.5}$–PM_{10}) mainly comes from particles whirled up from the road due to the wheel of the vehicles, whereas fine particles ($PM_{2.5}$) originate from the combustion of fuel from the engine. Several previous investigations have also reported a correlation between high traffic volume and increment of PMs and our findings are consistent with these results (Boogaard et al., 2009; Kaur & Nieuwenhuijsen, 2009; Kaur, Nieuwenhuijsen, & Colvile, 2005; Morawska et al., 2008). However, in most studies, UFP and fine PM number concentrations were given utmost importance as these particle sizes are more closely related to traffic emissions, and fewer studies have explored respirable particulate matter (PM_4).

Significant seasonal variations in PM concentration between the four routes were also observed in this study (Table 4.6). During the warm season, the highest PM concentrations were recorded in route 1 (community route), whereas during the cold season route 3 was found to be a highly polluted route with regards to particulate air pollution. We recorded higher PM concentrations in route 1 during the warm season, which could be due to the combustion emissions from the local or nearby (city) transportation (vehicles and ferries), meteorological conditions (high temperature) or infrastructure – such as tall buildings that trap the pollutants within the area. In contrast, the high PM concentrations in route 3 during the cold season could be due to a thermal inversion layer (Jayamurugan et al., 2013), which facilitates the accumulation of air emitted from the high volume of traffic on the freeway. This is consistent with the results reported in a study by Mishra et al. (2012) showing that PM concentrations are usually modified not only by the sources of pollution but also by different environmental scenarios, such as seasonal variation (summer or winter), the effects of meteorological parameters with respect to the volume of traffic, and the spatial representativeness of the monitoring station's location in the study area.

Comparing the overall concentration of PMs among the four routes between two seasons (warm and cold), the results showed a higher concentration of PMs during the warm season compared to the cold months (Table 4.7). This finding is consistent with another study, conducted by Majewski, Kleniewska, and Brandyk (2011), who found that a lower intensity of heat directly reduces PM emissions during cold seasons, whereas during summer the

concentration of PM increases due to the increase in air temperature. However, in contrast to this finding, studies conducted by Panther, Hooper, and Tapper (1999) and Schauer, Niessner, and Pöschl (2003) showed higher concentrations of ambient particulate matters during the winter season and lower during summer due to wind effects. Similarly, Chan and Kwok (2001) also showed higher concentrations of large-sized particles during winter and reduced levels of exposure in summer due to washout effects, wind direction and relative humidity.

For selected gases, the inter-microenvironment variation was quite different from the pattern shown by PM concentrations. The results showed that route 3 had higher concentrations of NO and NO_2 and route 4 had higher concentrations of CO and SO_2 than the other routes. The overall concentration of CO in the four routes was between 0.808–4.054 ppm, which is below the NEPM standard (9.0 ppm per 1 day a year), whereas NO_2 (0.091–0.359 ppm) and SO_2 (0.074-0.241) were above the standard (NO_2 is 0.12 ppm per 1 day a year and SO_2 is 0.20 ppm per 1 day a year). The higher concentration of gases in routes 3 and 4 supports the hypothesis that high traffic volume is directly related to the increased concentration of ambient air pollution and suggests that route choice can affect commuter exposure to pollutants associated with vehicle exhaust. Moreover, these findings are consistent with previous studies that measured the higher concentration of gases (CO, NO_2 and PM) on routes of high traffic volume (Han & Naeher, 2006; Zhang & Batterman, 2013). For example, the study conducted by Jarjour et al. (2013) showed that the concentration of CO was higher in high-traffic routes compared to those with low traffic. Similarly, a study conducted by Hertel et al. (2008) also showed higher NO_2 and CO concentrations near high-traffic routes.

Not only traffic volume but also specific characteristics of urban environments, such as close proximity to major roads, street canyons, tree-lined streets and infrastructure also play a significant role in determining the level of air pollution in the cycling microenvironment. In this study, routes 3 and 4 run parallel with and very close to freeways (3–4 meters), which could be a reason for their high levels of gases compared to other routes. Also, there is no trees (only bushes) present between the routes and the freeways, which can act like a barrier and do not allow further dispersion of the gases along the cycling areas. This is in agreement

with the findings established by the Hatzopoulou, Weichenthal, Dugum, Pickett, Miranda-Moreno, Kulka, et al. (2013) who proved that with every 5-metre increase in distance between cycling routes and traffic roads, the gaseous pollutants decrease by 2.5%.

The findings of this study suggest that cyclists riding in close proximity to high traffic volumes are exposed to higher concentrations of air pollution. However, as mentioned above, the high concentrations of particles are not always due to high traffic volume but sometimes also due to other specific characteristics of the urban environment – such as traffic congestion, street canyons, vegetation and infrastructure – as well as other sources of air pollution. This finding implies that urban planners and policymakers should not only focus on reducing traffic-related air pollution but should also give importance to planting vegetation barriers between cycling routes and vehicular traffic routes and relocating cycling routes further away from traffic and other sources of air pollution for the betterment of cyclists' health.

5.3 Effect of meteorological parameters on air pollution

Pollutants become airborne from the ground surface and the formation of secondary pollutants is controlled not only by the rate of emission but also by meteorological parameters, such as wind velocity, temperature, rainfall and humidity (Giri, Murthy, & Adhikary, 2008; Kaur & Nieuwenhuijsen, 2009; Thai, McKendry, & Brauer, 2008).

This study established several significant relationships between ambient PM concentrations and meteorological factors. It is assumed that the meteorological factors were the dominant source of the day-to-day variability in air quality. In fact, notable variations of PMs were observed in relation to ambient temperature. As the temperature increased, the condensation or coagulating rates increased, resulting in a more rapid loss of particulate matters and thus lowering the PM concentration levels. This finding is consistent with other studies conducted by Kittelson, Watts, and Johnson (2004) and Vinzents et al. (2005). Although there is increasing evidence for sequential correlations between PM and temperature, the relationships between them are inconsistent. For example, studies

conducted by Pearce et al. (2011), and Giri, Murthy, and Adhikary (2008), found a positive correlation between PM concentrations and temperature.

Furthermore, the study showed that increased precipitation significantly increased PM concentrations. This finding is consistent with the study by Tanner and Wong (1997), who found a positive correlation between the concentration of PMs and rainfall due to the advection properties of rain. Wet conditions are generally associated with disturbed atmospheric conditions, increased humidity and trapped PMs, which lead to increased exposure levels to PM. In contrast to this result, the study conducted by Giri, Murthy, and Adhikary (2008) showed a negative correlation between PM_{10} and rainfall, explaining that rain exhibited good washing effects on PM_{10} concentration in Kathmandu, Nepal. Not only this, but the study conducted by Keary et al. (1998) also showed a negative correlation between the concentration of PM_{10} and rainfall.

As expected, wind velocity is negatively correlated with PMs; however, no significant correlation between them was observed. Increased wind speed may affect PM concentrations by diluting primary sources, resulting in decreased pollution concentration (Vinzents et al., 2005). Similarly, if measurements were conducted upwind from a specific source then the concentrations of particles would be lower (Berghmans et al., 2009).

In this study, we also observed some influence of meteorological parameters on gaseous air pollution. In most other studies, wind velocity was found to influence the gaseous air pollutant levels (Elminir, 2007; Levy et al., 2003). The prevailing wind and its direction of flow help in transporting moisture or aerosol particulates from distant sources, which can play an important role in the seasonal variation of turbidity. In the current study, the wind speed was found to be significantly but also negatively correlated with SO_2 concentrations. This result is acceptable where the wind velocity helps to clean the ambient air pollution (Elminir, 2007). In agreement with this finding, the study conducted by Zhang et al. (2015) interpreted that horizontal dispersion is the major cause in decreasing gaseous concentration while increasing wind velocity. However, all the other gases (CO, NO and NO_2) were positively correlated with wind velocity but the correlation was not significant.

A negative correlation was found between temperature and NO, NO_2 and CO, and the correlation was significant with NO and NO_2. This negative correlation is due to the convection process – i.e., hot air above 2 m from the ground surface moving upward and cold air above 25 m moving downward – which helps in reducing the concentration of NO_x and CO. In agreement with this result, the analysis conducted by Jayamurugan et al. (2013) revealed that NO_x and CO have a very weak but negative correlation with temperature due to the convection process. In contrast, the study conducted by Aldrin and Haff (2005) found decreased temperature may be attributed to a reduction of a photochemical reaction of nitrogen that could ultimately reduce NO_x.

Analysis of the surface wind velocity and temperature alone do not adequately explain the variability in the concentrations of air pollutants. Therefore, analyses of the meteorological parameters affecting ambient gaseous concentrations of air pollution should also include an indicator of humidity content. The statistical analysis demonstrated that gaseous pollutants such as CO, NO, NO_2 and SO_2 were found to have a negative correlation with humidity; however, the correlation was not significant with SO_2. This is in agreement with the findings of a study conducted by Zhang et al. (1993). This study revealed that as relative humidity increases air pollutants absorb more water, increase their size and volume, and become deposited in the ground. High humidity may also indicate precipitation events accompanied by in-cloud scavenging, which highly affect the gaseous (CO, NO and NO_2) concentration (Kasper & Puxbaum, 1998). Similarly, findings from a study conducted by Elminir (2005) also showed higher concentrations of NO_2 at a lower relative humidity because the reaction of NO_2 with OH decreases. However, in contrast, the same study conducted by Elminir (2005) showed a positive correlation between SO_2 and CO concentrations with relative humidity, which can be attributed to the influence of clean, free tropospheric air masses. Likewise, the study conducted by Aldrin and Haff (2005) also showed no significant correlation between humidity and NO.

In the current study, rainfall was significantly and positively correlated with most gases, including CO, NO and NO_2. This finding is very difficult to interpret for gas pollution, whereas for PMs, a lack of precipitation delays the dispersion of the particles and reduces the

concentrations of some pollutants, such as PM_{10} and $PM_{2.5}$ (Vardoulakis & Kassomenos, 2008). However, Gatz and Nelson Dingle (1971) tried to explain that positive correlations between rainfall and gaseous air pollution are due to the advection process. In contrast, the studies conducted by Li et al. (2014) and Elminir (2007) established a negative association between rainfall and gaseous air pollution due to the dilution of gaseous particles by the water droplet and evaporation, followed by the scavenging of accumulated aerosol particles. Similarly, the study conducted by Lim, Jickells, and Davies (1991) and Aldrin and Haff (2005) also proved that the correlation between gaseous air pollution and rainfall was negative. The inconsistency of the above findings might be due to different study design, geographical locations, time span or time scale. However, our findings suggest that meteorological factors should also be considered in quantitative evaluations of ambient air pollution along different cycling routes to maximise the validity of such comparisons.

5.4 Measured physiological parameters and exposure

According to previous researchers, route choice as a proxy for traffic volume is likely to be an important determinant of exposure to commuters. In this study, personal exposure of PM_4 concentration among cyclists was observed to be significantly different between the four selected cycling routes. The highest median PM_4 exposure level was observed in route 1 and this is in agreement with the measured ambient background concentrations, which were found to be highest in the same route as explained in section 5.1. However, having high PM_4 exposure concentration near a low traffic route (route 1) was an unexpected result. These findings are very difficult to interpret because, in many studies, the researcher explains the mechanism of high personal exposure of PM_{10} but very few researchers discuss the only respirable particulate matter (PM_4). The increment of personal exposure of PM_4 in route 1 is probably due to different reasons (as explained in section 5.1). The first reason could be due to route 1's close proximity to a diesel ferry station, found to be an important source of cyclists' exposure to respirable particles. In agreement with this finding, the review study conducted by Knibbs, Cole-Hunter, and Morawska (2011) found that personal exposures to ultrafine particulate matter were higher among ferry commuters compared to cyclists, bus or

automobile passengers, and walkers. The second reason behind high personal concentrations of PM_4 among cyclists who cycled in route 1 was the numerous heavy and light vehicles that parked in and around route 1, especially during the morning rush hours when the number of vehicular movement with the engine running at the same time is very high (Liu, He, & Chan, 2011). Furthermore, engine idling, which is common when a vehicle is about to start or stop in a parking area, has been shown to cause higher exposure to PMs compared with moving vehicles. During idling conditions, the turbulent dispersion induced by the wake of a moving vehicle is absent, which is the reason behind the high PM exposure. Not very similar but very close to this study, the findings of research conducted by Ramachandran et al. (2005) concluded that higher levels of particulate matters were observed in parking ramp attendants compared to bus drivers.

The city of Perth is located approximately 1 km north of route 1, and Mill Point Road is approximately 245 meters away. This close proximity could be another reason for the high personal exposure concentrations of PM_4 among cyclists, as the pollutants produced from the high volume of congested vehicular movement in the city and Mill Point Road get dispersed due to wind (McNabola, Broderick, & Gill, 2008). Some previous studies have already proved the relationship between high traffic volume and wind velocity associated with higher exposure concentrations among commuters. For example, a study conducted in the UK observed significantly higher personal exposure concentration (20–50%) of particulate matters among commuters near a highly congested vehicular route compared to less congested route (Adams et al., 2001; Kingham et al., 1998). Similarly, in a London-based study, Briggs et al. (2008) observed positive and statistically significant correlations between car and truck density and the personal exposure concentration among walkers. These results are in agreement with similar investigations conducted in various locations where factors such as traffic congestion, emission rates, wind velocity and seasonal variation constantly play important roles in increasing the exposure concentration of particulates (Morawska et al., 2008; Strak et al., 2010). However, in contrast, the study conducted by Zuurbier et al. (2010), found no significant difference in particulate matter concentrations ($PM_{2.5}$ and PM_{10}) between high-traffic and low-traffic routes.

The findings of this study also show that personal exposure concentration to respirable particles among cyclists was higher than ambient background particle concentrations. This is obvious because of the high respiration rate of cyclists as well as the close proximity of cycling routes to traffic, whereas in the case of ambient particle concentration, the monitoring equipment was set up in a fixed site. This result is consistent with a previous study conducted by Kaur, Nieuwenhuijsen, and Colvile (2007) where the personal exposure concentration of $PM_{2.5}$ was higher compared to the urban background concentration measured from a fixed monitoring station. According to Kaur, the proximity of a fixed monitoring station to the pollutant source and the dispersion effects it encounters via the meteorological parameters determine the magnitude of pollutants, which is different from the concentration experienced by humans. This is the reason European regulations encourage that fixed monitoring stations associated with measuring traffic air pollution need to be situated in such a way that they represent the air quality in a surrounding area of at least 200 m^2 and not just the area in the immediate vicinity of the traffic's route (European Commission, 2000).

In addition to the above, another explanation behind the high personal exposure concentration of PMs compared to the ambient background concentration could be the different methodology used in measuring particulate matter. In this study, the gravimetric method was used to measure personal exposure to respirable particles, whereas DustTrak was used to monitor ambient PM concentrations. The DustTrak is a real-time optical monitor, which is associated with some limitations while monitoring ambient PM concentration (Sorensen et al., 2011; Wallace et al., 2011). For example, the different wavelengths of light employed to illuminate the particles from the DustTrak will have different sensitivities at different sizes depending on the wavelength. Due to this, a monitor with smaller wavelengths will be more sensitive to particles with smaller diameters compared to larger particles. Therefore, different aerosol mixtures with the same mass concentration as determined by a gravimetric monitor may have different mass concentration estimates compared to an optical monitor (Wallace et al., 2011). Though the exact relationships of real-time and gravimetric measurements used to set air quality standards are often unknown, the real-time monitor is known to be more significant, substantial and systematic compared to the gravimetric

method. According to the study conducted by Wallace et al. (2011), the results from the gravimetric analysis were 1.0–1.92 times higher than the real-time monitor. However, the exact relationship between the different methods used to calculate PM concentrations depends on the size of the particles and the environment being sampled.

The findings of this study also established a seasonal difference in exposure concentration of particulates among commuters (Table 4.11), which is in agreement with the results from previous studies (Adams et al., 2001; Sørensen et al., 2005). According to the results of this study, the overall PM_4 exposure concentration among the four routes was found to be significantly higher during the cold season compared to the warm season. These higher concentrations of particles recorded during the cold season may be due to the lower temperature and higher relative humidity, which can increase the condensation and coagulation, leading to increased PM concentrations. However, this result is not in agreement with the findings of a study conducted in Dublin, Ireland, where PM concentration exposure was found to be higher in summer than in winter (Adams et al., 2001).

A significantly higher concentration of personal exposure to PM_4 found during the low-speed cycling compared to high-speed riding could be explained by the time taken for travelling. This is consistent with a study by Tsai, Wu, and Chan (2008), according to which the longer the time spent commuting, the larger the total uptake of pollutants. In this study, the time duration of cycling 10 km was longer during the low-speed ride compared to the high-speed ride.

The main finding of this study is that personal exposure to respirable particles was highly influenced by the speed of cycling, in which respirable rate, as well as travelling time, plays a significant role in the uptake of pollutants. Also, other sources of air pollution (besides traffic volume) are key reasons for higher exposure to PMs among cyclists. Future studies should consider these parameters while researching the potential health benefits of cyclists.

5.5 Simulated dosimetry

This is the first study to investigate the deposition dose of PMs in the lungs of male and female cyclists cycling in four different cycling routes in Perth, WA. We used the MPPD model to estimate the potential difference in the lung deposition of PM with different sizes according to the exertion rate. The deposition fraction conc**.** was then compared between four routes (1, 2, 3 and 4). According to our findings, the deposition fraction conc**.** of smaller particles with a size between 0.1 and 3.652 µm was higher in the pulmonary region, whereas the deposition of coarse particles size of 6.494 µm was mainly in the Tracheobronchial (TB) region of the lungs. This finding was consistent for all four cycling routes and it is in agreement with other studies where the small-sized particles were found to be highly deposited in the peripheral airways (pulmonary region) and the coarse-sized particles in the upper airways (TB region) (Darquenne, 2012; Sracic, 2016).

In the present study, the regional cumulative deposition (RCD) curve was estimated as a function of particle size in the pulmonary and TB regions of the respiratory system. These curves represent four different groups: the four cycling routes, gender, high and low exertion levels, and cold and warm seasons. A notable difference was observed between those four groups, which were in accordance with the different ambient PM concentrations measured in each route. While comparing the four cycling routes (Figure 4.1), the deposition rate of particles in the pulmonary region decreased above approximately 4–5 µm and no deposition occurred for particles 7 µm and larger. Whereas, in the TB region, the deposition rate of larger particles decreased approximately above 7–8 µm. This is due to the fact that particles of different sizes have different mechanisms of deposition; i.e., fine and coarse particles tend to deposit by sedimentation whereas ultrafine particle deposition is by diffusion (Scheuch et al., 2006). Due to the different mechanisms of deposition, it is likely that deposition in the different regions of the lungs varies between fine and large particles (Donaldson, Tran, & MacNee, 2002).

Even though the deposition fraction conc**.** of particles in the pulmonary region was found to be higher in route 1 compared to the other three routes (Table 4.15), the cumulative

deposition in the same region was found to be higher among cyclists who cycled in route 2 (Figure 4.1). These findings are very difficult to compare with the results from previous studies because of the different methodologies adopted in the research. For example, some research did not consider the deposition of particles in particular regions of the lungs, such as the pulmonary region (Saber & Heydari, 2012), whereas other studies did not discuss the activities undertaken by the participants (Hussein et al., 2015). Similarly, in some studies, the deposition fractions conc**.** of particles were calculated by using the characteristics of the respiratory system of an adult with single values for tidal volume and breathing frequency (Ham, Ruehl, & Kleeman, 2011). On the other hand, other studies calculated the deposition factor with specific values for different age groups, such as tidal volume, functional residual volume, breathing frequency and volume of the upper respiratory tract (De Winter-Sorkina & Cassee, 2002). As a consequence, the present study indicates that regional deposition varies with the size of the particulate matter (Donaldson, Tran, & MacNee, 2002), so health effects do not depend only on the particle deposition fraction conc.; rather, they are determined by the cumulative dose of the particles deposited in particular regions of the lungs.

Generally, the results obtained from other studies indicate that the distribution of deposited particles depends on gender and age (Bennett, Zeman, & Kim, 1996). However, some studies indicate that the distribution of deposited particles in the respiratory system is very similar between genders engaged at a similar activity level (Salma et al., 2002). In the present study, the deposition of PMs in the pulmonary region of the lungs was found to be higher in females compared to males, while the opposite was observed for the TB region (Figure 4.2). The study conducted by Kim and Hu (1998) also follows the same trend – i.e., particle deposition in the lungs was greater in women compared to men for coarse particles regardless of flow rates. Likewise, Li et al. (2016) also estimated that the deposition of small particles was found to be higher among females compared to males. Neither study mentions the region of the lung.

The differential deposition of particles for different genders may be related to the differences in lung size between males and females. In the present study, the average lung size of female subjects (average FRC=3465.85 ml) was smaller than the male subjects (average FRC=3526.68 ml). Both male and female subjects inhaled the same aerosols but not with the same

breathing patterns (both genders were asked to do the high- and low-speed ride but males took less time compared to females in both rides), which could result in different outcomes. Another possible explanation for the different deposition dose among genders could be the potential for particles to penetrate deeper into the lungs. Although all subjects inhaled aerosols with the same tidal volumes, aerosols may have penetrated deeper into the lungs and this may result in a greater deposition for subjects with smaller lung sizes. Some researchers have already proved that the dimensions of the upper airways – particularly the larynx – of females are much smaller compared to males even if the lung or body size is the same. Due to this, it may be expected that the airflow environment of the upper airways is more turbulent for women than men, which can result in greater deposition of ultrafine particles in the lungs (Kim, Hu, & Ding, 1998; Kim & Hu, 1998). In contrast to this study, some researchers concluded there is no correlation between lung size and the deposition dose of particles (Blanchard & Willeke, 1984; Jaques & Kim, 2000). However, it should be noted that lung deposition is governed primarily by three factors: particle size, breathing pattern and geometry of the lungs. Further, the deposition of particles in the lungs also depends on the proximity of traffic sources and the uncontrolled effects of relative humidity on measurements, which cannot be excluded. These findings may have significant implications in health risk assessment concerning inhaled particles among men or women; further, they will be useful for targeted aerosol delivery information in pharmaceutical and clinical studies.

Exertion also plays a major role in how particles enter the respiratory system. As mentioned above, the deposition of the inhaled particles depends on many factors, such as exposure concentration, exposure duration, breathing pattern of the person, whether they breathe through the mouth or nose, the structure of respiration anatomy and morphometry of the person, and activity the person is involved in (Scheuch et al., 2006); (Hinds, 1999). Increased intensity of physical exercise results in larger numbers of small-sized particles penetrating deeper into the respiratory system (Sracic, 2016). A higher deposition level is due not only to exertion but is also partly caused by switching from nose-only breathing to the mixed nose and mouth breathing (Salma et al., 2015). Though the oral deposition efficiencies are consistently smaller than those for nasal deposition, more particles can reach the respiratory

system compared to only nose breathing. The activity level of an individual, however, plays an important role in exposure to particles and thus should be taken into account whenever assessing the potential health consequences of air pollution.

In the present study, the cumulative deposition of PMs in the pulmonary region of the lungs was found to be higher during low-exertion cycling; however, in the TB region, the observations were the opposite (Figure 4.3). This is consistent with the result where the data showed that personal exposure during low-speed riding was higher compared to the high-speed ride (Table 4.12). In the pulmonary region (Figure 4.3), the overall cumulative deposition gradually increased as the diameter of particles increased but narrowed off (4–5 μm) and reached an asymptote in the case of large particles (>7 μm). As explained in section 5.4, the deposition dose of particles also depends on the time spent riding. The higher deposition at low exertion levels could be due to more time spent during the low-speed ride compared to the high-speed ride. This is consistent with a study conducted by Salma et al. (2002), who demonstrated that the deposition of particles in the entire respiratory system during high activity (high exertion) decreases because the proportions of exhaled particles also grow when activity levels increase. The MPPD model used does not incorporate incapability adjustment to particle deposition. Sometimes, inhalation may differ as per the size and wind speed with which particles in the breathing zone actually enter the respiratory tract via the nose or mouth (or both) (Hofmann, 2011). Along with particle sizes and entry point, meteorological variables such as temperature and humidity may also significantly impact the inhaled particle deposition (Kittelson, Watts, & Johnson, 2004); (Vinzents et al., 2005). However, in contrast to this result, the study conducted by Saber and Heydari (2012) showed that the stronger the breathing intensity (high exertion), the higher the deposition dose in every respiratory region regardless of particle size. Despite this kind of inconsistency in the results, it is proved that the activity level of an individual plays an important role in the deposition of particles in the respiratory system and should be taken into account when assessing the potential health risks posed by traffic-related air pollutants.

Under different ambient conditions, a particle's composition and size distributions are obviously different. As mentioned above, particle deposition in the human respiratory system

is closely linked to the size of the particles (Hussain, Madl, & Khan, 2011), so it is expected to be different under different pollution levels and seasons. In the present study, we demonstrated how particles of different sizes were distributed in the respiratory system of cyclists during the cold and warm seasons (Figure 4.4). The deposition dose was found to be higher in the pulmonary region of the lungs of cyclists who cycled during the cold season compared to the warm season. This is consistent with the result for personal exposure concentration, where the data shows that personal exposure during the cold season was higher compared to the warm season (Table 4.12). The deposition fraction gradually increases in the pulmonary region until the particle size ranges from 5 μm and remains constant as particle sizes increase. Inconsistent with this result, the study conducted by Lu et al. (2015) also found that deposition was higher in winter but the comparison was made between winter (90%) and spring (86%). However, the study conducted by Li et al. (2016) found no differences between summer and winter, instead of finding a difference between winter (higher) and spring (low). Regardless of season and pollution concentration, the deposition in the pulmonary region is always highest among all other regions of the lungs. Although other studies have indicated that the health effects of particles are more closely related with the size and concentration of deposited particles, (Cohen et al., 2017; Raaschou-Nielsen et al., 2016b), the effects of deposition episodes such as cold seasons should also be considered.

The findings of this study indicate that cycling at both high and low exertion rates influences the deposition of particles for both warm and cold seasons. The study findings imply that gender should be discussed separately when conducting exposure assessment and that the level of physical activity should also be taken into account when assessing potential health consequences.

5.6 Limitations of the study

This study has several limitations and they are as follows:

a. Due to an insufficient number of cyclists recruited for the study, the participants were asked to repeat more than one route in addition to the original route they already participated in. Therefore, the number of cyclists in each route is not the same.

b. Due to the breakdown of USMPS, the P-Trak was substituted during the middle of the data collection, which reduced the number of measurements of USMPS data. The P-Trak also has a lower size range for counting.
c. Due to the breakdown of the Testo 350 analyser during the warm season, we have limited recordings for gases and were not able to compare for seasonal differences.
d. Also, the traffic information for each route on every cycling day was not collected. Instead, the data was requested from Main Roads, Western Australia and it was not related to the actual year of the study data collection. In order to overcome this limitation, the traffic volume of each year was averaged and assumed as a constant traffic flow at each point of time.

Regardless of its limitations, this study is the first in Australia to assess the ambient quality of air and the deposition of particles in the lungs of cyclists who cycle in different routes of the Perth metropolitan area. This study will be useful for policymakers and urban planners in developing strategies for constructing new cycling routes considering the potential adverse impacts of close proximity to busy roads.

CHAPTER 6 : CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

This study contributes to a small but growing body of literature that investigates the network of relationships between active cycling, exposure to ambient PM levels and its association with health impacts (through the literature review). Based on the results, this study concluded that the concentration of air pollution in ambient air, the physiological parameters of cyclists and their activity pattern during cycling have a high impact on the particle deposition in their respiratory tract. This approach, however, implies the availability of reliable data for specific case studies in the future.

A shift from automobiles to bicycles may be endorsed as a healthy means of transportation in terms of reducing the risk of developing adverse health effects among cyclists. However, the individual cyclist may receive increased exposure to traffic-related respirable particles because of their increased ventilation rate and close proximity to congested-traffic air pollution. Therefore, cycling near polluted routes can be unsafe in terms of particle deposition in the lungs. Nevertheless, choosing a cycling route close to a low traffic road is not the only consideration in reducing exposure to air pollutants; it is essential to also consider nearby additional sources of pollution, such as ferry stations, industries and car parking areas. In particular, this study indicates that the best way to mitigate exposure to pollution among cyclists is to cycle on a designated network of streets that are 'bicycle boulevards,' which are away from pollution sources (Jarjour et al., 2013).

According to WHO, there is no safe concentration for PM (WHO, 2018); nevertheless, in this study, the levels of PM did not exceed the NEPM/WHO standards at any point during the sampling period.

Furthermore, this study concluded that the gender, season and exertion rate should be considered while calculating the deposition dose among cyclists. This can offer more accurate results that will help in model validation, reduce uncertainty and clarify the contribution of personal exposure to actual health effects among cyclists.

Overall, the results obtained in this study can be used to develop approaches to consistently determine the deposited dose based on the exertion of cycling (and other populations exposed to environmental or occupational pollutants combined with physical exertion) and ambient PM levels, and evaluate the impact of the inhalation and uptake of air pollutants on human health.

6.2 Recommendations

To assess the health effects of deposited particles on cyclists' performance, long-term monitoring of PM concentrations and exposure for comprehensive chemical analysis of pollutants is recommended along Perth Metropolitan area where the emission of pollutants is high.

It is also recommended that policymakers focus on constructing or modifying the particular network of cycling routes to 'bicycle boulevards' that run away from busy streets or congested areas. Bicycle boulevards would potentially mitigate exposure to pollution among cyclists, which would eventually encourage people to cycle as an active mode of transportation for the betterment of their health. An increase in the number of bike commuters would, in turn, decrease the number of traffic on roads, leading to a lower concentration of pollution in ambient air.

Moreover, it is also recommended that, near cycling routes, other sources of air pollution associated with the combustion of fossil fuel be significantly reduced; for example, motorised traffic transportation, industrial emissions and so on. Policymakers should, therefore, focus on preparing strategies for renovating technologies and reducing emissions in ambient air – for example, by increasing the use of electric buses and cars. Furthermore, urban planners

can also consider designing cycling infrastructure that minimises the occurrence of stops along the traffic route and allows fast access through polluted urban areas in order to reduce the uptake of air pollution for cyclists. In addition, policymakers should consider adding vegetation near traffic roads and cycling routes.

Finally, incorporating physiological parameters and breathing rates in determining deposited doses, rather than exposed concentrations, would seem to be a logical next step to apply to all areas of environmental and occupational exposure assessment.

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APPENDICES

Appendix A

FORM OF CONSENT

I,.....

Given names Surname

have read the information sheets explaining the details of the study entitled **'Evaluation of exposure to particulate air pollution and its lung deposition among cyclists in the Perth Metropolitan Area'.**

I have been given an opportunity to ask questions to the investigators. I agree to participate in this study and understand that I may withdraw from the study at any stage.

I consent to the collation of my data for research purposes only. I have been assured that my personal details will not be released to anyone outside this investigating group, and that stored data will be de-identified.

This study has been approved by the Curtin University Human Research Ethics Committee (Approval Number HR 183/2013). The Committee is comprised of members of the public, academics, lawyers, doctors and pastoral carers. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hrec@curtin.edu.au .

Date.....

PARTICIPANT'S SIGNATURE.....

WITNESS SIGNATURE.....

Appendix B

INFORMATION SHEET FOR PARTICIPANTS

Dear Participant,

We would like to invite you to participate in the study titled 'Evaluation of exposure to particulate air pollution and its lung deposition among cyclists in the Perth Metropolitan Area' *conducted by Anu Shrestha, PhD student in the School of Public Health, Curtin University*. The aim of the study is to assess exposures to particulate air pollution (fine dust) and its deposition in the lungs among cyclists commuting in Metropolitan Area of Perth.

There are three (3) stages in the study:

- In the first stage cyclists will be asked to complete one page online questionnaire to determine their eligibility to participate in the research.
- In the second stage, all participants will be asked to come to Curtin University for no more than 30 min for physical assessment. The physical assessment consists of height and weight measurements and also spirometer test (Lung Function Test) in resting position. Spirometer measures ventilation, the movement of air into and out of the lungs and provides indication of how well the lungs perform.
- In third stage, all participants will be asked to cycle one among the four selected routes (10 km in high and 10 km in low levels of speed) by carrying small and lightweight monitoring equipment including heart rate and respiration rate monitor, and personal dust monitor. The cyclists are requested to cycle not more than 1 hour on one morning in cold and one morning in warm season.

  Four selected routes are 1. Douglas Ave, South Perth (Mends Street Jetty to near Marathon Club. 2. Kwinana Freeway (Near Red Box cafe, South Perth to Canning Bridge, Como). 3. Mitchell Freeway (End of Melrose St, Leederville to near Potenza Ave, Stirling

)and Railway Parade, Welshpool (Opposite to Jaram Australia, Welshpool to Junction of Crawford St and Lacey St)

We would be most grateful if you would be prepared to take part in all three stages. You may withdraw your participation at any point in time during the study.

In all three stages the data gathered will be kept confidential and the data and information generated will only be used for research purposes. The results will be presented in an aggregated form; and no individual participation will be identified.

This study has been approved by the Curtin University Human Research Ethics Committee (Approval Number HR 183/2014). The committee is comprised of members of the public, academics, lawyers, doctors and pastoral careers. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hrec@curtin.edu.au.

Note: All study participants will be eligible to go in a draw to win gift vouchers from a local cycle shop to a total value of $1000.

Thank you and your assistance will be highly appreciated. If you have any further queries, please do not hesitate to contact us:

Anu Shrestha (PhD Student)

Email: anu.shrestha@postgrad.curtin.edu.au

Mobile: 0406195434

Dr Krassi Rumchev

Tel: 08 9266 4342

Email: K.Rumchev@exchange.curtin.edu.au

Appendix C

INFORMATION SHEET FOR PHYSICAL ASSESSMENT

Dear Participant,

We would like to invite you to take part in the second stage of study titled 'Evaluation of exposure to particulate air pollution and its lung deposition among cyclists in the Perth Metropolitan Area' *conducted by Anu Shrestha, PhD student in the School of Public Health, Curtin University*.

This assessment includes the following measurements:

1. **Height and Weight Measurements** with a scale available in the School of Public Health Laboratory.
2. **Spirometry Testing:** The main aim of this test is to measure the lung function of the participants. Each participant will be asked to sit on a chair and take a deep breath and blow hard into the spirometer. This test will be repeated three times and the best value will be chosen for the analysis.
3. **Nebulizing Test:** As part of the spirometry testing we wish to measure the lung deposition of particulate matter (air pollutants) in order to calibrate the computational Multiple Path Particle Dosimetry (MPPD) model. This is conducted through inhalation and exhalation of medical grade air containing a very low concentration of nebulized diethylhexyl Sebacate (DEHS) in order to measure lung particle deposition.
   Note: The test is not recommended for anyone who suffers from respirable symptoms o for this test.

The physical assessment is voluntary and you can choose to take part in all or some of the measurements and we would be most grateful for your participation. All data gathered during the physical assessment will be kept confidential and will be used only for research purposes. The results will be presented in an aggregated form; and no individual participation will be identified.

Thank you and your assistance will be highly appreciated. If you have any further queries, please do not hesitate to contact us:

Anu Shrestha (PhD Student)
Email:
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Dr Krassi Rumchev
Supervisor
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Appendix D

Online questionnaire to determine the eligibility to participate

Q1. What is your full name?

Q2. What is your gender?

Q3. What is your age group?

Q4. Do you usually cycle to work in the morning?

Q5. How much distance in km do you ride during the week (Monday to Sunday)?

Q6. Which routes do you cycle most often?

Q7. Do you suffer from cardiovascular disease or any other chronic health condition?

Q8. Contact details:

Appendix E

Checklist for Cyclists

1. Read the information sheet sent by the investigator
2. Choose the time and date to participate in the research
3. Come to the place suggested by the investigator
4. Sign the consent form
5. Carefully listen the brief instruction given by the investigator
6. Put on the personal sampler and bio-harness monitor
7. Make it “ON” both the instruments and start riding in low exertion level (low speed).
8. After riding 5 km (looking at the map for end point), turnover and come back to the place where you have started (without turning off the instrument)
9. The investigator will make it “OFF” both instrument and will change the filter of the pump and again start the riding
10. Investigator will make it “OFF” both instrument and will change the cassette of the personal sampler and make it “ON” both the instrument and start riding again in high exertion level (high speed), turnover and come back to the place where you have started (without turning off the instrument)
11. Altogether, cyclists need to cycle 10 km in high and 10 km in low speed.
12. Cyclist need to come to Curtin University (investigator will call and fix the appointment) for physical measurement and test lung function.

Appendix F

Safety measures for cyclists

Please do not forget about your safety measures while cycling for the study such as:

a. Always check behind before you turn around or stop.
b. Slow down gradually and indicate to other what you are going to do.
c. Do not stop or turn around until it is safe to do so.
d. Please remember, safety is the most important thing and maintaining heart rate up high is the secondary. So, do not speed up high if there is any dangerous situation or if you do not feel comfortable to so.
e. Not to overtake unless it is safe to do so, even if this means your heart rate goes down a bit.
f. When turning, crossing bridge your heart rate may drop down below the recommendation level. Do not worry about it. Its ok to speed up once it is safe to do so
g. Please give me a call in my number 0406195434 if you got an accident or run into other difficulties such as flat tyre.

Appendix G

Format for data collection during cycling test

Date	Time	Name	Code	Cassette No	Low		Cassette No	High		Heart Rate Number	Consent Form	Remarks
					Start Time	End Time		Start Time	End Time			

Appendix H

Format for filter paper information during cycling test

Route Name…….. **Season……**

S.No.	Before	After	COD/speed	Date	S.No.	Before	After	COD/speed	Date

Appendix I

Format for physical assessment data collection

Date	**Name**	**Code No.**	**Time**	**Spirometer Test**			**Height**	**Weight**	**Nebulizing inhalation time**				
				FEV1	FEV1	FEV1			**Promo Reading**				
									Pure Air 1	**1**	**2**	**3**	**Pure Air 2**
				1.FEV1: 2. FVC: 3. FV1/FVC: 4. PEF	1.FEV1: 2. FVC: 3. FV1/FVC: 4. PEF	1.FEV1: 2. FVC: 3. FV1/FVC: 4. PEF			Start.Time: End. Time:	Start.Time: Flow Rate:	Start.Time: Flow Rate:	Start.Time: Flow Rate:	Start.Time: End Time: