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Human health risks of inhalable exposure to $PM_{2.5}$ in Pretoria, South Africa

by

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DECLARATION

I, Nandi Sisasenkosi Mwase, student number 17242496, hereby declare that this dissertation, "*Human health risks of Inhalable exposure to $PM_{2.5}$, Pretoria, South Africa,*" submitted in accordance with the requirements for the Magister Scientiae degree at University of Pretoria, is my own original work and has not previously been submitted to any other institution of higher learning. All sources cited or quoted in this research paper are indicated and acknowledged with a comprehensive list of references.

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Nandi Sisasenkosi Mwase

11 February 2020

DEDICATION

I dedicate this study to my family (my mother, grandparents, uncles, aunt, and siblings) who sacrificed so much for me to purse my studies, especially my grandmother who passed away this year and could not see me finish my Masters, but she would have been happy to know that I had been able to submit. My family not only gave financial support, but also emotional support throughout the journey.

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ABSTRACT

Aim: The aim of this project was to measure $PM_{2.5,}$ soot, black carbon, and UV particulate matter, and assess the health risks $PM_{2.5}$ poses to humans in Pretoria, as part of my MSc (Epidemiology) project.

Design: The study is a two-part study combining an exposure assessment and Human Health Risk Assessment study.

Setting: The study was conducted in an urban background area located in Pretoria, Gezina, South Africa. The area is mostly a residential area, away from the highway and without much heavy traffic**.**

Data and method: Gravimetric analysis was used to determine $PM_{2.5}$ concentrations every third day from 19 April 2019 to 23 April 2019. An estimate of possible health risks from exposure to airborne $PM_{2.5}$ was performed using the USA Environmental Protection Agency human health risk assessment framework. A scenario-assessment approach was utilised, where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for intermediate (24-hour) and chronic (annual) exposure periods for different exposure groups (infants, children, adults).

Outcome measures: Absence of major adverse health effects from exposure to airborne pollutants.

Results: The average annual ambient concentration of $PM_{2.5}$ was 21.5 ± 13.6µg/m^3, which was higher than the annual $PM_{2.5}$ World Health Organization air quality guideline. Infants and children, rather than adults, are more likely to be affected by 24-hour exposure. Additionally, for chronic annual exposure, $PM_{2.5}$ posed low health risks to sensitive individuals, with the severity of risk varying across exposed groups.

Conclusion: Levels of $PM_{2.5}$ posed a low health risk to people in Pretoria, however a follow-up study should investigate the risks posed by the $PM_{2.5}$ chemical composition. It is recommended that the City of Tshwane Air Quality Management Plan, which is currently under review, addresses local and long-range sources of $PM_{2.5}$ in the city.

Key Words:

Air pollution, $PM_{2.5}$, South Africa, exposure assessment, health risk assessment, meteorological conditions,

LIST OF ABBREVIATIONS

^{o}C	Degrees Celsius
APPA	Atmospheric Pollution Prevention Act
AQMP	Air Quality Management Plan
COPD	Chronic Obstructive Pulmonary Diseases
CVD	Cardiovascular Diseases
DEFF	Department of Environment, Forestry and Fisheries
HHRA	Human Health Risk Assessment
NAAQS	National Ambient Air Quality Standards
NEMA:AQA	National Environmental Management Act: Air Quality Act
NO_2	Nitrogen Dioxide
O_3	Ozone
$PM_{2.5}$	Particles matter smaller or equal to 2.5 µm in aerodynamic diameter
REVIHAAP	Review of Evidence on Health Aspects of Air Pollution
SAAQIS	South African Air Quality Information System
SD	Standard Deviation
SES	Socioeconomic status
SDGs	Sustainable Development Goals
SO_2	Sulphur Dioxide
WHO	World Health Organization
$\mu g/m^3$	Micrograms Per Cubic Meter

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CHAPTER 1: BACKGROUND

1.1. DEFINING THE RESEARCH PROBLEM

Air pollution has continued to be a major environmental concern and has been recognised as a vital public health risk.[1] This has been recognised by the World Health Organization (WHO) as well as the United Nations (UN), hence the establishment of the Air Quality guidelines in 1987,[2] which has been continually revised. As a member of the UN, South Africa has recognised the issue of air pollution and has promulgated environmental legislation as a control measure for air pollution and its adverse effects. Despite the legislative governance in the country, the monitoring of exposure levels and updating of Air Quality Management Plans (AQMP) have not been adequately followed through. The City of Tshwane (Pretoria) is one of the municipal areas that has not updated its AQMPs. There is a particular lack of efficient monitoring of particulate matter with the aerodynamic size of 2.5 µm ($PM_{2.5}$). Due to its size, $PM_{2.5}$ poses a higher threat to human health, therefore, the lack of monitoring of the levels of exposure to the population within Pretoria, hinders interventions, both legislative and non-legislative, from being adequately implemented or evaluated.

1.2. MOTIVATION AND RELEVANCE

The purpose of developing an AQMP is to empower the City of Tshwane (Pretoria) to meet its obligations as outlined in the Air Quality Act. This is intended to provide more efficient practices of air quality management and ensure a cost-effective and equitable reduction of emissions. The main goal is to assess the exposure levels of $PM_{2.5}$ within Gezina, an area based in the Tshwane Metropolitan. This assessment should then assist in improving air quality around Tshwane and reduce environmental health risks.

The exposure levels of $PM_{2.5}$ levels in Pretoria are not adequately monitored. The possible health impacts of air quality in South Africa, and specifically the City of Tshwane, has not been extensively researched and has created a major research gap. There is a need to assess the exposure levels of $PM_{2.5}$, and equally understand the interaction among the different pollutants as well as variables such as meteorological

variables, ultimately understanding the possible health risks that these could pose to the population.

1.3. AIM AND OBJECTIVES

1.3.1 AIM

The aim of this project is to measure $PM_{2.5}$ and assess the health risks that this pollutant poses to humans in Gezina, Pretoria.

1.3.2. OBJECTIVES

The proposed project objectives are:

(1) To determine the levels of $PM_{2.5}$, soot, black carbon and organic carbon at the HW Snyman Building, Prinshof Campus, University of Pretoria, for 13 months.

(2) To determine the correlation between $PM_{2.5}$, soot and other pollutants (PM_{10}, SO_2, NO_2, O_3; measured by the DEFF in Pretoria), to assess possible effects of other pollutants on $PM_{2.5}$ and soot.

(3) To determine the correlation between $PM_{2.5}$ and meteorological variables (rainfall, temperature, humidity and wind speed) and seasonal change (winter, summer, autumn, and spring), and to assess the effects meteorological variables may have on the concentration levels of $PM_{2.5}$.

(4) Conduct a Human Health Risk Assessment (HHRA) study to assess the health risks of inhalation exposure to $PM_{2.5}$ in Gezina, Pretoria.

1.4. OUTLINE OF THE DISSERTATION

In the current chapter, the general introduction to the research topic is given, and the problem statement, the significance of the study, the research aims and objectives are addressed.

In Chapter 2, the literature on the $PM_{2.5}$, the known adverse health effects, as well as an overview of the existing legislation and the current air pollution management in South Africa was reviewed.

In Chapter 3, the methods applied in the study are stipulated. This includes a detailed explanation of the two methodologies used to achieve the objectives of the study, namely, the exposure assessment and the human health risk assessment. The statistical analysis methods applied are also presented.

In Chapter 4, the results of the study are presented.

In Chapter 5, the results are discussed and limitations and strengths of the project are addressed. It also covers the conclusions and recommendations regarding the project results.

CHAPTER 2: LITERATURE REVIEW AND MOTIVATION

This chapter will give a brief review on air pollution and the relationship between PM and soot. It will summarise evidence of the human health effects and air pollution specifically and cardiovascular disease (CVD) and respiratory disease (RD), mention susceptible groups (i.e. prenatal exposure) and the relationship between air pollution and climate change indicators, such as temperature, relative humidity, wind speed and rainfall, give a brief history of air quality management in South Africa and internationally, compare the South African National Air Quality Standards (NAAQs) to those of the more protective World Health Organization (WHO) guidelines, present an overview of the methods used to conduct human health risk assessment studies and lastly, highlight the need for more research on $PM_{2.5}$ in South Africa.

2.1. AIR POLLUTION AND ITS SOURCES

Air pollution is the presence of one or more contaminants such as dust, fumes, gas, mist, odour, smoke or vapour in quantities that can be harmful to all living organisms.[3] The sources of particulate matter can be caused by indoor and outdoor sources. Indoor sources include cooking and forms of heating in the house.[4] Outdoor sources include traffic, manufacturing and other industrial procedures, which are commonly anthropogenic forms of pollution.[5] For the purpose of this study, outdoor pollution and sources are the focus of the study. Within the African context, due to rapid population growth this has led to increased use of vehicles, solid fuels for cooking and heating, and poor waste management practices, [6-7] all of which have resulted in a rising threat to the health of the population.[8] As a result, soot is an additional product of incomplete combustion of such fuels.

2.1.1. INDOOR POLLUTION

Indoor pollution, although not the focus of this study, has been associated with multiple adverse effects. Indoor pollution occurs within a household or a closed area where the ventilation is poor and there is incomplete combustion of biomass.[9] This incomplete combustion releases different concentrations of particulate matter, carbon monoxide, nitrogen oxides, sulphur dioxides and other toxic gases into the environment.[10]

Exposure to these pollutants has been associated with multiple adverse health effects that include chronic obstructive pulmonary disease (COPD), lung cancer, tuberculosis and particular acute lower respiratory infections. Not only is this common within South Africa, but associations have been observed in multiple low-to middle-income countries.[10-11] Other studies have indicated that in poor urban environments, which could refer to townships within South Africa, there is high paraffin usage indoors, while in high priority areas, such as the Vaal triangle, coal usage is a problem.[11] Reasons for the different sources of the heating, lighting and cooking in poor to semi-urban areas, is due to the low accessibility of electricity to power the different appliances; this suggests that poor areas within South Africa are highly dependent on fuels that produce significant polluting emissions.

2.1.2. OUTDOOR POLLUTION

Emissions from traffic, power generation, industrial emissions that are mainly from burning of fossil fuels and domestics use of coal, wood and paraffin, are some of the main sources of outdoor air pollution.[12-14] In addition, increased migration to urban areas increases the anthropometric activities, such as increased traffic and industrial activity, all of which increase outdoor emissions within an area.[15] These sources emit carbonaceous particles that are considered to be toxic to humans.[16] The most common air pollutants found in these emissions include, sulphur dioxide (SO_2), nitrogen oxides (NO_x), ozone (O_3) , volatile organic compounds (VOCs) and suspended particulate matter (SPM).[17] Literature would suggest that these pollutants are considered primary pollutants, all except O_3, which is referred to as a secondary pollutant because it occurs once nitrogen oxides and VOCs react in sunlight and stagnate air.[18] The NEMA: AQA states eight criteria pollutants, which include carbon monoxide (CO), NO_2, SO_2, O_3, particulate matter (PM_{10} and $PM_{2.5}$), benzene and lead (Pb).[19]

2.1.3. SOOT

Black carbon smoke (soot) is a product of the incomplete combustion of hydrocarbon-based fuels and is identified as a short-lived climate pollutant.[20] This is also the source of $PM_{2.5}$, hence the relation between the two in this study. The sources are very similar

to those of $PM_{2.5}$, and includes car and industrial emissions, indoor cooking, and outdoor cooking involving coal burning (i.e. barbeque).[20-21]

Soot, also referred to as black carbon and elemental carbon, intensively absorbs light and heat.[22] It positively affects the radiation balance in the atmosphere, and it has been suggested to make a significant contribution to global warming.

2.2. GLOBAL KNOWLEDGE ON AIR POLLUTION AND HEALTH

Air pollution is a major risk factor leading to an increase in morbidity and mortality in several countries. In 2014, the World Health Organization (WHO) reported that one in every eight deaths globally is due to air pollution exposure.[23] Due to this evidence, the United Nations (UN) and WHO have stipulated air pollutant guidelines.[24] These guidelines help UN signatories adopt the levels of emission within the countries to promote a better quality of air. Due to the complex mixture of air pollution, the guidelines have identified air pollutant components as criteria air pollutants, which include: particulate matter (PM), sulphur dioxide (SO_2), ground-level ozone (O_3), carbon monoxide (CO), benzene, lead and nitrogen dioxide (NO_2).[25]

PM levels are expressed by the mass of particles matter smaller or equal to 2.5 µm in aerodynamic diameter ($PM_{2.5}$) and particles matter smaller or equal to 10 µm in aerodynamic diameter (PM_{10}) in aerodynamic diameter. According to the WHO, of the 91 countries monitoring air pollutants, over half are experiencing air pollution levels of almost 2.5 times higher than the WHO standard.[26] With the various pollutants, literature indicates their association with many adverse health effects affecting multiple populations.[27] There has been evidence that indicates that gaseous pollutants, such as SO_2, NO, NO_2 and O_3 to have harmful effects.[27-28] The most evident trends in Cardiovascular Diseases (CVDs) and Respiratory disease. Evidence also indicates that as much as pollutants affect notifiable vulnerable groups within a community, their condition increases their risk of being affected as compared to rest of the community; these groups include the elderly, young and pregnant women.[27,29]

Research has indicated that outdoor air pollution and its major components, including particulate matter, are carcinogenic to humans.[30] Literature also supports a causal link

between $PM_{2.5}$ and cardiovascular and respiratory ill health. Long-term exposure to $PM_{2.5}$ can activate a range of problems that include atherosclerosis, adverse health outcomes, childhood respiratory problems, cognitive and diabetic problems. Air pollutants are also a contributing factor to premature mortality rates globally.[31] The reason for this, due to $PM_{2.5}$, is sometimes referred to as fine particulate matter, having a size that enables it to remain suspended in the air for a longer period of time, high penetration ability and thus increasing the likelihood of being inhaled.[32] Black carbon has been associated with conditions as a result of short to medium-term exposure; such conditions include systemic inflammation and oxidative stress, impaired heart rate deceleration capacity, and blood pressure [33-35]

There was an estimated 3.2 million premature deaths associated with the exposure to fine particulate matter across the world, the majority of those deaths being cardiovascular diseases.[36] Further reports in 2012 indicated that 12% of global mortality was due to diseases associated with air pollution.[28,37-38] The WHO has attempted to set regulated guidelines of exposure for all signatories to standardise within their counties.[28,38] Exposure to these air pollutants can come from both outdoor sources and with some air pollutants, indoor exposure is possible. The United Nations has worked in conjunction with the WHO and placed improvement of air quality in the Sustainable Development Goals.[39] By providing guidelines that are adaptable for government legislation, it contributes to a broader means of controlling one of two categories of air pollution, indoor and outdoor pollution. Exposure to outdoor air pollutants is essentially beyond the control of individuals and requires action by public authorities at the national, regional and even international levels. It is through the implementation of legislation that countries can achieve the Sustainable Development Goals (SDGs), which seek to address and improve air quality by 2030. The first one being SGD goal 3, target 9 that seeks to " reduce the number of deaths and illnesses caused by hazardous chemicals and air, water and soil pollution and contamination."[39]

2.2.1. AIR POLLUTION AND CARDIOVASCULAR DISEASE

The WHO published in 2013 the Review of Evidence on Health Aspects of Air Pollution (REVIHAAP) report.[40] The REVIHAAP report established that PM_{10}, NO_2, SO_2, ground-level O_3, as well as individual metals (arsenic, cadmium, nickel, lead) and polycyclic

aromatic hydrocarbons were risk factors for numerous human health effects.[40] Cardiovascular disease morbidity and mortality are some of the health effects included in the REVIHAAP report.[40] The numerous epidemiological studies that reported the adverse health effects of PM_{10}, NO_2 and SO_2 prompted the need to update the 2005 WHO air quality guidelines.[40]

Studies conducted in both developed and developing countries strengthened the argument that ambient air pollution increases CVD mortality and hospital admissions.[40] However, CVD mortality was used as the health outcome in most of the studies.[40] More studies are needed that also focus on CVD symptoms and CVD hospital admissions.

PM_{10} and $PM_{2.5}$ are known to aggravate CVD conditions, primarily due to their size that allows them to enter the circulatory system readily.[27,29,41] Long term exposure of the pollutants leads to an increase in mortality, while short-term exposure to $PM_{2.5}$ aggravates multiple conditions such as acute heart failure and myocardial infractions.[16,42] Long-term exposure has also been associated with an increase in coronary heart disease and acute myocardial infarction.[16,43] Other studies argue that cardiovascular mortalities are strongly associated with $PM_{2.5}$ particles produced from biomass burning sources as opposed to a moderate association with roadway and industry sources;[32] this suggests that along with the presence of $PM_{2.5,}$ the sources of emission contribute to the risk of cardiovascular disease. Corrigan *et al.*[42] further indicate that a change in National Ambient Air Quality Standards (NAAQS) of $PM_{2.5}$ levels can result in the improvement in public health and a reduction in cardiovascular mortality rates.[42]

Although particulate matter has a strong association to both cardiac mortality and hospitalisation, gaseous pollutants such as SO_2 and nitrogen oxides have equal associations.[29,41,44-45] Long term exposure to NO_2 has also been associated with cardiovascular mortality.[46] Short-term exposure to particulate matter and gaseous pollutants can be associated with various cardiovascular outcomes. Short-term exposure to $PM_{2.5}$ and NO_2 can result in the increased admissions for arrhythmia.[47] While short-term exposure to $PM_{2.5}$, SO_2, NO_2 and CO has led to an increased risk of

hospitalisation and death caused by congestive heart failure.[48] An increased risk in stroke has also been strongly associated with short-term exposure to SO_2, NO_2 and CO; in addition, some evidence shows an association between stroke and ozone exposure.[49-51]

A case-crossover epidemiology study conducted in Cape Town 2012, reported that outdoor air pollution exposure posed a higher risk of dying from CVD than in developed countries,[52] despite the air quality levels of Cape Town averaging similar to some European cities. A significant increase of 2.6% and 3.4 in CVD mortality was observed in SO_2 and NO_2, respectively. However, there was no significant association observed between PM_{10} and CVD mortality. A follow-up study in 2017 was conducted in Cape Town, Durban and Johannesburg,[53] The meta-analysis revealed a mortality risk of 1.0% (0.3%; 1.7%), 1.0% (-0.3%; 2.3%) and 0.9% (-0.9%; 2.7%) for CVD mortality following a 10 $\mu g.m^{-3}$ increase in the 2-day cumulative average of PM_{10}, NO_2 and SO_2 during 2006-2010, respectively.[53]

2.2.2. AIR POLLUTION AND RESPIRATORY DISEASE

More recent studies conducted in both developed and developing countries strengthened the conclusion of the REVIHAAP report, i.e. that ambient air pollution increases RD mortality and hospital admissions.[40] However, RD mortality was used as the health outcome in most of the studies. More studies are needed that focus on RD symptoms and RD hospital admissions.

Similar to the cause of cardiovascular diseases, the sizes of pollutants PM_{10} and $_{2.5}$ easily enter the respiratory system and are absorbed quicker than they can be expelled from the system. Particulate matter along with gaseous pollutants increase the prevalence of both respiratory diseases and their symptoms.[54] COPD can be developed through short and long term exposure.[55] Long-term exposure to air pollution can lead to a decline in lung function, most notably in older patients.[41,56] Within in children, studies have shown that asthma attacks increase when in close proximity to air pollution sources.[57] Other studies have identified the young age group to have modification effect when exposed to air pollution, due to the larger surface area of their lungs and fragile frames.[58] This increases their risk of paediatric asthma attacks.

Literature reports there is a strong association between the short-term effects of exposure to ambient air pollution and hospital admissions due to pneumonia, bronchitis and asthma symptoms in children under the age of 18 years old.[59] Studies indicated that hospital admissions for asthma attacks were associated with $PM_{2.5}$ NO_2 and O_3,[60] while others show a positive association between PM_{10}, $PM_{2.5}$ and NO_2 and respiratory hospital admissions.[55] Particulate matter is also associated with the aggravating of acute conditions such as emphysema and bronchitis. Without adequate interventions to reduce emissions of gaseous pollutants, such as NO_2, the degradation of respiratory health could be worse in the future.[61-62]

In addition, soot has also been found to have serious adverse health outcomes. Similar to $PM_{2.5}$, and its readily inhalable size, it can cause respiratory problems including increasing hospital admissions.[21,63]

Wichmann and Voyi (2012) reported an increase of 1.3% (-1.4%; 4.0%) and 2.0 % (-1.6%; 5.7%) in RD mortality per inter-quartile range increase in PM_{10} (12 $\mu g.m^{-3}$) and NO_2 (12 $\mu g.m^{-3}$), respectively in Cape Town, South Africa, during 2001-2006. In contrast, a decrease of -0.5% (-3.6%, 2.6%) was observed per inter-quartile range (8 $\mu g.m^{-3}$) increase in SO_2.[52] A follow up study by Thabethe (2017) reported an overall excess mortality risk of 0.4% (-0.4%; 1.1%), 1.2% (-0.2%; 2.6%) and -1.9% (-3.7%; 0.0%) observed for RD mortality following a 10 $\mu g.m^{-3}$ increase in the 2-day cumulative average of PM_{10}, NO_2 and SO_2 during 2006-2010, respectively.[53]

2.2.3. AIR POLLUTION AND PRENATAL EXPOSURE

PM_{10}, $PM_{2.5}$ and gaseous pollutants, such as SO_2 and O_3, have been associated with adverse health outcomes during pregnancy affecting both the mother and her unborn child. Infants having utero exposure to particulate matter and ozone are at high risk of stunted growth and low birthing weights. [64-66] In addition, expectant mothers are at a higher risk of developing the onset of Gestational Diabetes Mellitus (GDM) upon exposure to particulate matter and ozone. [67-69]

2.2.4. AIR POLLUTION AND CANCER

Lung cancer is the most prominent type of cancer associated with air pollution.[70] Long-term exposure to ambient particulate matter has been identified to increase the risk of developing cancer, predominantly lung cancer.[71] Other studies have shown that long-term exposure to NO_2 and $PM_{2.5}$ increase the odds of developing lung cancer, with further investigation needed to explore the association of O_3 and cancer.[72] Aside from the actual size of the particles, literature has argued that the main chemical composition of the particulate matter contributes to the cancerous element of the air pollutants.[73] Studies have shown that even low exposure to ambient air pollutants can increase the risk of cancer, due to vapours, metallic compounds and metals.[70] Dependent on the source of the particulate matter, the elements found have different properties, either cancerous or non-cancerous.[73]

2.2.5. AIR POLLUTION AND DIABETES

The focus on air pollution and diabetes has recently gone under investigation. Recent studies have shown a positive association between PM_{10} and type 2 diabetes;[74] with an increased exposure of 10 $\mu g/m^3$ $PM_{10,}$ the odds of developing type 2 diabetes increases by 1.23.[75] There have been strong suggestions that indicate particulate matter is strongly associated with the development of diabetes.[76-77]

2.3. CONTRIBUTING FACTORS

Multiple contributing factors could increase exposure of air pollutants to a community. Within this section, only two have been identified and although not an exhaustive list, these two factors, mostly uncontrollable to the individual, are socioeconomic status (SES) and meteorological factors.

2.3.1. SOCIO-ECONOMIC STATUS

Areas located in middle to low SES areas are more prone to use heating methods that disperse high emission. Within these areas is a high dependence on fuels such as charcoal, wood, paraffin and these fuels are known to increase emissions. In addition, there has been a higher mortality rate due to air pollution within those areas compared to areas of higher SES. [78-81] The former, are neighbourhoods located closer to higher traffic congestion and industrial areas, which are implied to have higher air pollution

concentrations.[81] Developing counties are also affected by the difference in air pollution compared to developed countries. Developing counties have less stringent air quality laws and this results in the allowance of higher emissions, placing their communities at higher risks to exposure. [82]

2.3.2. METEOROLOGICAL FACTORS

2.3.2.1. SEASONAL CHANGE

The reviewed literature indicated that meteorological conditions can significantly influential the concentrations of particulate matter as well as gaseous pollutants. Most notably, the concentrations are highest during colder months of the year.[83-86] This is due to a number of factors, such as relative humidity, stronger inversion, low wind speeds, and little to no precipitation.[84,87-89] Warmer seasons allow a release of emission out into the troposphere, away from direct contact. [85] These factors are most prominent in areas that experience dry winters and wet summers. Other literature would suggest that particulate matter is higher in warmer months where there are no rains and lower in the colder months where there are higher wind speeds and high rains.[90] Similarly, soot absorption coefficients are recorded to be higher in winter months, when more heating activities are taking place.[91]

2.3.2.2. RAINFALL

The presence of rainfall clears the atmosphere by removing air pollutants that are present, as a result it decreases the presence of such.[92] It has also been reported that wind speed, relative humidity and temperature, have a significant influence on the concentration of particulate matter within the atmosphere.[88,93]

2.3.2.3. STABILITY PATTERNS AND CLIMATE CHANGE

However, the continuous influence of climate change could possible shift the patterns commonly observed, and meteorological influences may change.[94] Multiple human activities contribute to air pollution emissions that contribute to climate change. As a result, climate change may influence human health effects of air pollution by changing levels, chemical composition and transboundary movement.[95] In addition, emissions,

transport, dilution, chemical transformation and eventual deposition of air pollutants, can be influenced by weather conditions such as temperature, humidity, wind speed and direction and mixing height.[96] Temperature inversions can limit both vertical and horizontal dispersion of air pollution.[97] Higher temperatures increase chemical reactions that lead to ground level O_3 and secondary particle formation. NO_2 absorbs visible solar radiation and contributes to impaired atmospheric visibility; as an absorber of visible radiation it could have a potential direct role in global climate change.[96-97]

The Intergovernmental Panel on Climate Change (IPCC) indicates rising temperature as one of the key climatic changes, as it has direct and indirect effects on health.[98] The rising temperature around the world is of concern.[99] In South Africa, an increase of 3–4°C in ambient temperature is projected along the South African coast and 6–7°C inland within the next 100 years as a result of climate change.[100] Recent reviews summarised the evidence that temperature, in conjunction with air pollutants, have the ability to cause damaging effects on human health.[101-104] Furthermore, the greatest burden of climate change will be in low- and middle-income countries (such as South Africa) due to a high burden of existing vulnerabilities such as poverty, informal housing with poor protection against heat, inadequate public health services, pre-existing diseases such as TB, HIV/AIDS, dementia, diabetes, chronic respiratory and cardiovascular diseases.[105-107]

Wichmann (2017) conducted the first epidemiological study that investigated the association between apparent temperature (Tapp) and all-cause mortality in Cape Town, Durban and Johannesburg, South Africa, during 2006-2010.[108] A 3.3%, 2.6% and 2.8% increase in mortality per IQR increase in the 2-day cumulative lag of Tapp was observed in Cape Town, Durban and Johannesburg, respectively, above the city-specific thresholds. The city-specific Tapp thresholds were 18.6 °C, 24.8 °C and 18.7 °C, respectively, for Cape Town, Durban and Johannesburg.[108] The elderly were more at risk in Cape Town and Johannesburg. No difference in risk was observed for males and females in the three cities. In the meta-analysis, an overall significant increase of 0.9% in mortality per 1 °C increase in the 2-day cumulative lag of Tapp was observed for all age groups combined in the three cities. For the ≥65 year group a significant

increase of 2.1% in mortality was observed. The risks for all age groups combined and the elderly are similar to those reported in studies from developed and developing countries. [108] A follow-up study by Makunyane (2018) investigated the association between RD and CVD mortality and Tapp in six major cities in South Africa, namely Cape Town, Durban, East London, Johannesburg, Pretoria and Port Elizabeth during 2006-2010.[109] The study concluded that the heat effects in six cities for RD and CVD mortality per 1°C increase in the 2-day cumulative average of Tapp was 0.50% (-0.03%;1.03%) and 0.13% (-0.47%;0.74%), respectively. Stronger associations were observed for the elderly (≥65 years).

2.4. SOUTH AFRICA KNOWLEDGE ON AIR POLLUTION AND EXISTING LEGISLATION

Air pollution is one of the nine health and environmental risks that are highlighted as potential key risks according to the South African National Department of Health. [110] This serves as an indicator that the South African government recognises the potential harm air pollution can have on human health. South Africa also has adapted supporting environmental legislation that adheres to the WHO's guidelines.[19] It is presumed that South Africa has records of experiencing high levels of pollutions, mainly experienced in industry focused areas.[111] However, studies have shown that anthropogenic emissions are worse in urban areas, specifically low-income residential areas.[112-113] The main sources of pollution in South Africa are emitted from human activity, biomass fuel use, transportation and household emissions.[114] The legislation surrounding air pollution in South Africa has progressed over a number of years.

2.4.1. NATIONAL AMBIENT AIR QUALITY STANDARDS AND WHO GUIDELINES

Tables 2.1 and 2.2 summarise the NAAQS in South Africa and the more protective WHO guidelines.[115-116]

Table 2.1: South African National Ambient Air Quality Standards

Pollutant	Averaging Period	Concentration	Frequency of Exceedances	Compliance Date
PM_{10}	24 Hours	75 µg/m^3	4	1 January 2015
	1 Year	40 µg/m^3	0	1 January 2015
$PM_{2.5}$ (added in 2012)	24 Hours	40 µg/m^3	4	1 January 2016 - 31 December 2029
	1 Year	20 µg/m^3	0	1 January 2016 - 31 December 2029
NO_2	1 Hour	200 µg/m^3	88	Immediate
	1 Year	40 µg/m^3	0	Immediate
SO_2	10 Minutes	500 µg/m^3	526	Immediate
	1 Hour	350 µg/m^3	88	Immediate
	24 Hours	125 µg/m^3	4	Immediate
	1 Year	50 µg/m^3	0	Immediate
Ground-level O_3	8 Hours	120 µg/m^3	11	Immediate
CO	1 Hour	30 mg/m^3	88	Immediate
	8 Hour	10 mg/m^3	11	Immediate
Lead	1 year	0.5 µg/m^3	0	Immediate
Benzene	1 year	5 µg/m^3	0	1 January 2015

The 2005 WHO guidelines are based on epidemiological evidence conducted prior to 2005[115] and are current.[117] Since 2005, the evidence base for adverse health effects related to short- and long-term exposure to the criteria air pollutants have become much larger and broader.[40]

Table 2.2: World Health Organization air quality guidelines of 2005

Pollutant	Averaging Period	Concentration
PM_{10}	24 Hours	50 µg/m^3
	1 Year	20 µg/m^3
$PM_{2.5}$	24 Hours	25 µg/m^3
	1 Year	10 µg/m^3
NO_2	1 Hour	200 µg/m^3
	1 Year	40 µg/m^3
SO_2	10 Minutes	500 µg/m^3
	1 Hour	Not applicable
	24 Hours	20 µg/m^3
	1 Year	Not applicable
Ground-level O_3	8 Hours	100 µg/m^3
CO	1 Hour	35 mg/m^3
	8 Hour	10 mg/m^3
Benzene	1 year	No safe level of exposure can be recommended

2.4.2. ATMOSPHERIC POLLUTION PREVENTION ACT

The Atmospheric Pollution Prevention Act (AAPA) established in 1965, was the first initiative to address air pollution that was predominantly from industrial emissions.[111] The APPA (act 45 of 1965) sought to control pollution at the source and attempted to set guidelines for common pollutants that included SO_2, NO_2 and ozone,[118] however, due to the prominent downfalls of the APPA was repealed. The Department of

Environmental Affairs and Tourism rolled out the National Environmental Management: Air Quality Act in 2004 to address the short falls of the APPA.[119]

The NEMA: AQA was later established to provide a more comprehensive legislative structure for environmental management in South Africa, although these standards are considerably lenient in comparison. Nonetheless, extensions to the NEMA promulgated after 2005 affect environmental management in South Africa in a significant manner. The South African Government further instituted the NEMA: AQA. The act clearly stipulated guidelines to pollution control of ambient air to multiple parties including polluter, supervisory bodies, as well as the general public.[19] The metropolitan councils were charged with the responsibility of implementing the Act at the local governmental level, which included the completion of Air Quality Management Plans (AQMP) and the mid- and long-term review of such AQMP.[120]

2.4.3. AIR QUALITY MANAGEMENT OF THE CITY OF TSHWANE

The Air Quality Management Plan for the City of Tshwane (Pretoria) was developed and approved on 15 September 2006, by the Mayoral Committee, and was meant as a management and performance-monitoring tool for air quality control and to provide a baseline assessment of air quality issues in Tshwane. The AQMPs for municipalities were implemented by the Government to decentralise responsibility of air quality monitoring at local level rather than national level. These were to work by meeting the main obligations stated in the Air Quality Act.[121]

Some of the objectives include achieving and sustaining acceptable air quality levels in Pretoria and also minimising health risks and harm to the environment. The AQMP provided the importance of air quality measurement “tools,” such as emission inventory, air quality and meteorological monitoring and atmospheric dispersion modelling.[121] The intention of these tools was to provide comprehensive emission inventory, to facilitate the effective characterisation of spatial and temporal variations in air pollutant concentrations. Despite the initial implementation of these plans in 2006-2008, there have been no consistent updates.

As of now, the City of Tshwane has nine monitoring stations distributed across the municipality, Bodibeng, Booyseng, Ekandustria, Hammanskraal, Mamelodi, Olivienhoutbosch, Pretoria West, Rosslyn and Tshwane Market. On the DEFF website, South African Air Quality Information Systems (SAAQIS), it is indicated that of the nine stations, only three (Bodibeng, Hammanskraal and Rosslyn) record $PM_{2.5}$ concentrations, and of these three only one (Hammanskraal) records relatively consistent and comprehensive results. The same site also indicates that the most recent report conducted in the City of Tshwane was in 2011,illustrating there has not been a comprehensive air quality report done to show the levels of $PM_{2.5}$ within the CoT in 8 years. The report does not give results of $PM_{2.5}$ levels, and shows that even then the information on the air pollutant was not known nor was it monitored. Without information on the concentrations levels of $PM_{2.5}$, how are control measures to be implemented to preserve the health of the community? Levels not consistently monitored also means the DEFF lacks the information on how much areas exceed or are below the stipulated standards.

2.5. HUMAN HEALTH RISK ASSESSMENT

A human health risk assessment (HHRA) is essentially a public health tool designed to improve the health of the public via assessing potential risks and the extent of those risks.[122] This tool has a four-part process that considers a myriad of elements to generally assess the pollutant concentrations the public is exposed to, the population groups at risk and the toxic effects of the pollutant of interest.[122]

The HHRA has four main features, namely hazard identification, dose-response assessment, exposure assessment and risk characterisation. Conducting a HHRA is a cost-effective, reliable method of assessing the potential health effects of an identified pollutant. It also assists in identifying the possible short term and long term effects of the pollutant that adults and children are exposed to.[123] However, the risk assessment has its limitations. This may include a lack of epidemiological and toxicological data on the pollutant of interest. The inability to determine synergistic effect of pollutants, and difficulty to determine possible influencing factors such as behavioural factors within the population.[123]

2.5.1. HAZARD IDENTIFICATION

Hazard identification is a process of researching existing literature of the pollutant of interest and identifying the potential and associated health effects. The objective of hazard identification is to determine whether there is scientific evidence presenting the pollutant of interest as harmful to humans.[123-124] According to the WHO, hazard identification looks at whether the pollutant of interest constitutes a health risk to humans and what conditions can the hazard occur.[122] This establishes the potential hazards the pollutant of interest poses and the extent of this hazard.[122] The assessment considers existing epidemiological and toxicological data, as well as the biological and chemical information of the pollutant of interest.[122]

2.5.2. DOSE-RESPONSE ASSESSMENT

The dose-response assessment is the process that considers the relationship between the exposed dose of the hazard and possible severity of its effects on the body.[73,124]

2.5.3. EXPOSURE ASSESSMENT

The exposure assessment determines if people are in contact with the potentially hazardous pollutant. It considers the concentration, the route of exposure and media, as well as the duration of exposure. The objective of this stage is to determine the concentrations to which the target populations are exposed;[125] the degree, frequency and length of exposure in the target population.[125]

2.5.4. RISK CHARACTERISATION

Risk characterisation is a summary, integration and evaluation of scientific evidence, reasoning and conclusions of a risk assessment.[126] This stage considers the possible carcinogenic and/or non-carcinogenic risks that may come from exposure to the pollutant.[126] These risks include the likelihood of developing cancer over a lifetime of exposure and the non-carcinogenic effects from exposure, all established using the hazard quotient.[122,126]

2.6. MOTIVATION AND RELEVANCE

The purpose of developing an AQMP is to empower the City of Tshwane (Pretoria) to meet its obligations as outlined in the Air Quality Act. This is intended to provide

efficient practices of air quality management and ensure a cost-effective and equitable reduction of emissions. The main goal is to assess the exposure levels of $PM_{2.5}$ within Tshwane Metropolitan. This assessment should then assist in improving air quality in Tshwane and reduce environmental health risks.

Few studies have been done across the country in quantifying PM2.5, and even fewer of these studies have conducted health risk assessments to assess the risk this pollutant poses to the community. Although some studies have shown possible health threats to high-risk communities, these were mostly located in industrial areas. Less concentration has been made to communities living in of urban locations, where the main sources of pollution are traffic, local fires, indoor cooking and so on. The values of exposure are unknown and desperately need to be quantified, mainly because of these highly populated areas.

CHAPTER 3: METHODOLOGY

This chapter is presented in three sections: the laboratory analytical methodologies, the fieldwork and lastly, the method of the Human Health Risk Assessment (HHRA) applied in this project.

3.1. STUDY SETTING

The study site was located at the School of Health Systems and Public Health (SHSPH), University of Pretoria, Gezina, in the City of Tshwane. Samples were collected on the roof of the HW Snyman South Building, Prinshof campus (S 25°43'53" E 28°12'01"), as seen in Figure 3.1. Gezina is an urban suburb area in the CoT (Pretoria). The study site was a favourable choice for sampling due to the assurance of safety and a continuous supply of electricity. The equipment was placed on the rooftop away from individuals to prevent any interferences with the equipment, as opposed to passive sampling.

Figure 3.1: Image of sampling site, derived from Google Earth

3.2. STUDY AREA

The sample site was located in an urban area, which was referred to as a “cleaner” area compared to an industrial area. In this area, there were no industrial manufacturing companies, nor was it located near a freeway or highway. Although located near the Steve Biko Gezina Road, which is a major road in the area, it is 5 km to 10 km away from the central business district (Pretoria CBD). The sampling site, however, is located 270 m away from the Tshwane District hospital incinerator. The areas’ most notable sources of air pollution are nearby traffic and burning of various materials done by the homeless to keep warm, although this would be more prevalent during the winter season. There are four notable seasons experienced in South Africa: summer (December, January and February), autumn (March, April and May), winter (June, July and August) and spring (September, October and November). The area experiences wet summers and dry winters, similar to the rest of South Africa.

The $PM_{2.5}$ measurements were collected over a 13 month period (19 April 2018 to 23 April 2019) every third day for 24 hours (see Appendix 1). In addition, duplicate samples were taken every fifth measurement. Overall, sampling occurred over 124 days, including the 25 duplicates (overall, 149 samples were collected).

3.2.1. TRAINING AND PROCEDURES

Initial training was conducted in May 2017 when the researcher, who was doing BSc Environmental Health Honours degree studies, was assisting a PhD student with his $PM_{2.5}$ measurements. Further training was obtained by Dr Nico Claassen, School of Health Systems and Public Health, during a module the researcher attended in 2018, namely Methods of Exposure Assessment; the module is part of the coursework required in this MSc (Epidemiology) degree programme

3.3. EXPOSURE ASSESSMENT

The exposure assessment determined $PM_{2.5}$ concentrations as well as the soot, black carbon and organic carbon composition within the area of interest (Gezina area). The $PM_{2.5}$ measurements were collected every third day for 24 hours from 9 am to 9 am over 13 months (19 April 2018 to 23 April 2019) (see Appendix 1). In addition, duplicate samples were taken every fifth measurement. Overall, sampling occurred over 124 days, including the 25 duplicates (overall, 149 samples were collected). The period 9 am to 9 am was selected for practical reasons as starting and stopping a measurement at midnight was not practical.

3.3.1. GRAVIMETRIC ANALYSIS

Gravimetric analysis is the analytical technique used to determine an analytic mass, in this study it was the mass of $PM_{2.5}$.

Gravimetric analysis of the $PM_{2.5}$ collected on filters was done at the Air Quality Laboratory, SHSPH, as it has a Mettler Toledo microbalance. The filters were weighed before and after sampling on the microbalance to determine the mass of the collected $PM_{2.5}$. The weighing followed a standard operating procedure (SOP), where three field blanks were used before and after each batch of 20 filters. The SOP used for the weighing procedure was a modified version of the SOP used in the ULTRA study.[127]

The filters were conditioned for at least 24 hours before weighing in the weighing room of the Air Quality Laboratory, SHSPH. The temperature and relative humidity are maintained at 21.0±1.0^{o}C and 50±5%, respectively in the weighing room.[127]

In order to neutralise the static charge on the filters before weighing, they were put through an alpha radiation source (Po-210). This increased the precision of the measurements of the samples. The filters were pre-weighed for a maximum of two months before use in fieldwork and post-weighed for the same period after use. Triplicate measurements were done on each filter, if the result differed by more than 5 µg, extra weighing of the filter was repeated;[127] this was continued until the requirement had been met.

The field blank mass was first calculated to determine if there was an increase or decrease. The determined value was then subtracted from the calculated mass (µg):

$$M = W_2 – W_1 – B \qquad \text{(Equation 1)}$$

Where:

W_1 = adjusted filter weight before sampling (µg)

W_2 = adjusted filter weight after sampling (µg)

B = mean adjusted filter weight change of field blank filters (µg)

The weights determined in W_2 and W_1, were averages of triplicate measurements done on each filter at every weight session. The average weight was controlled for deviation of the control filter weights on weighing from the nominal value. The average deviation for the blank filters was done by subtracting the nominal value of the three blank filters. For the filters exposed on the roof, the average deviation of two exposed filters from the nominal value of the two exposed values were subtracted. The nominal value was the average value of all eight weighing sessions.

The limit of detection (LoD) is evaluated by weighing batches of blank filters according to ISO/CD 15767.[128] Pre- and post-weighed filters were stored in a refrigerator at 4°C, at the Air Quality Laboratory, SHSPH.

3.3.1.1. EQUIPMENT AND MATERIALS

$PM_{2.5}$ samples were collected on 37 mm filters (2µm pore size) Teflon filters (Zefon International: Sampling Equipment specialists 5350 SW 1st Lan. Ocala, FL 34474 USA) using small a GilAir pump and a BGI cyclone. The medium in 37 mm diameter filter provides a cross-sectional surface area; the pore size prevents particulates

greater than the pore size from being collected. Pre- and post-flow rates were measured using the Gilian Gilibrator: primary flow air calibrator, Range: 20cc – 6 LMP

For duplicate measurements, two pumps and two cyclones were be used.

3.3.1.2. SAMPLING

On the day of sampling, the filter with the appropriate sample number was removed from the refrigerator where it was stored. The pump was removed from the power plug, which kept the internal battery of the pump charged and ready for use for the 24 hours sampling period. The pump was then switched on in order to prepare the unit before calibration could commence. While this was being done, the filter was removed from the Petri dished in which it was stored and transferred, with its support pad, into a cassette using a flat-nosed tweezer (Figure 3.2 below (a)).

(a) (b)

Figure 3.2: Images of preparation of sample unit (a) support pad and filter in cassette (b) prepared sample unit

Next, the cyclone was attached to the cassette containing the filter (Figure 3.2 above (b)). The unit was securely sealed with insulation-tape to prevent any leaks both into and out of the unit. The bubble solution was put into the calibration unit thereafter the

unit containing the filter was attached to the calibration unit as well as the pump, as seen in Figure 3.3 below.

Figure 3.3: GilAir pump connected to the calibration unit

An average flow rate is taken after three readings. The pumps were calibrated to be ± 4 L/min. After the flow rate was taken, the unit was taken up to the sampling site, as seen in Figure 3.4.

Figure 3.4: Sample unit placed on the sample site (HW Snyman South rooftop)

After the 24-hour period had elapsed, the bottom end of the cyclone was removed to ensure that the large particles that were captured were removed and the unit could safely be turned without contaminating the sample. Post-calibration took place in the laboratory, where an average was taken once again. The filter was removed and placed in a petri dish and put back into storage in the refrigerator.

3.3.2. SOOT MEASUREMENTS

A modified SOP was used for the soot analyses of $PM_{2.5}$ collected on filters (i.e. reflectance analyses). This procedure was similar to that of the ULTRA study.[129-130] All black soot index analyses were done using the M43D smoke stain reflectometer (Diffusion Systems Ltd., London, UK) at the Air Quality Laboratory of the SHSPH

Reflectance was measured on each filter using a five-point method. This started from the centre, followed by each quadrant; the average reflectance was then calculated. The soot measurements were conducted in a batch of 25 filters. After each batch, three filters were selected at random and measured for a second time to ensure the measurements did not differ by a maximum of 3%. If a difference was seen, the batch was re-done. The correction due filter was adjusted by measuring the filed field blank. The black soot index was calculated using the following calculation:

$a = (A/2V) * In (Rf/Rs)$ (Equation 2)[131-132]

Where:

Rs is the average reflectance of the sampled filter

Rf is the average reflectance of field blank filters

V is the sampled air volume (m^3)

A is the area of the stain on the filter ($780 * 10^{-6} m^2$).

The absorption coefficient (*a*) is expressed in $10^{-5}m^{-1}$

3.3.3. BLACK CARBON AND UPVM

Black carbon (BC) and UVPM (a proxy for organic carbonaceous particulate matter absorbing UV light at 370nm) were performed using a Model OT21 Optical Transmissometer (Magee Scientific Corp., Berkeley, CA USA) at the office of the co-supervisor. The additional absorption in the UV light, at 370 nm, due to the organics indicated the presence of biomass burning.[133-135] UVPM is a proxy for organic carbon species, expressed in mass concentration.

3.3.4. WEATHER AND OTHER AIR POLLUTION DATA

The City of Tshwane (Pretoria) has a network of nine monitoring sites that continuously monitor air pollutants similar to the United States Environmental Protection Agency and in accordance with ISO 17025 guidelines (National Environmental Management: Air Quality Act, 2004) (as seen Table 3.1). The air pollution data are stored in the South African Air Quality Information System (SAAQIS), which is managed by the South African Weather Service (SAWS). Air pollution data between 19 April 2018 and 23 April 2019 were obtained from the SAAQIS, hourly concentrations of three air pollutants were obtained and used in this study: SO_2, NO_2, PM_{10}, CO and O_3. 24-hour averages (9 am to 9 am) were then calculated from the data obtained. $PM_{2.5}$ is measured at four of the nine air monitoring sites (Figure 3.5), but there are many data gaps.

However, there was a lot of missing data and some stations had no recorded information. Therefore, the data used as a comparison in this study was from the Pretoria West station that had the most complete data and was one of the closest in proximity to the sampling site.

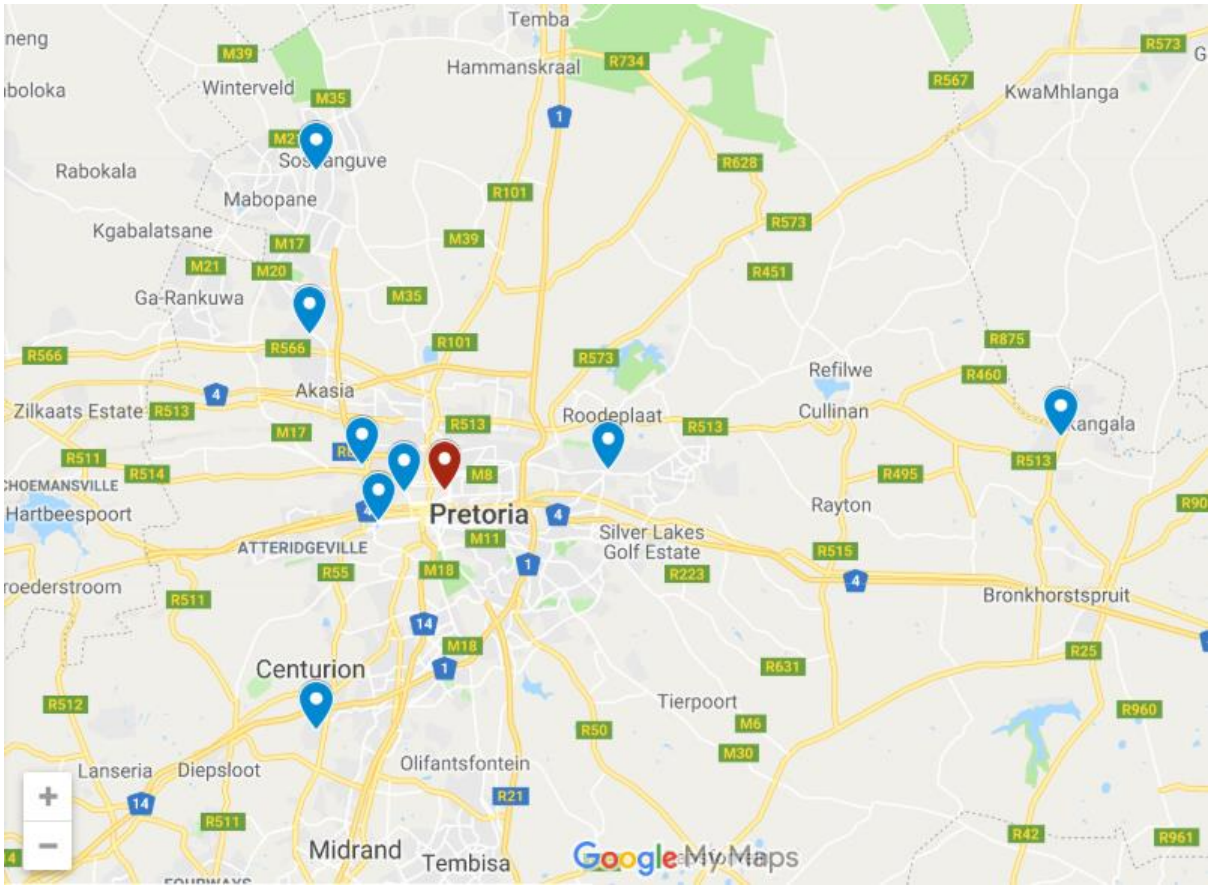

Figure 3.5: City of Tshwane nine air monitoring stations and sampling site at the School of Health Systems and Public Health, University of Pretoria

Table 3.1: Classification and year of establishment of air pollution monitoring stations in the City of Tshwane

Station	Suburb	Classification	Coordinates	Year Established	Measured Pollutants
Bodibeng	Soshanguve	Residential & traffic	25° 29'34,155"S 28°5'37,495"E	2011	SO_2, NO_2, PM_{10}, CO O_3, NO, NOx, $PM_{2.5}$
Booysens	Claremont	Residential & traffic	25°42'49,205"S 28°07'55,539" E	2009	SO_2, NO_2, PM_{10}, CO, NO, NOx
Ekandustria	Bronkhorspruit	Industrial	25°41'23,617"S 28°42'47.800"E	2012	SO_2, NO,NO_2, NOx PM_{10} CO, O_3, $PM_{2.5}$
Hammanskraal	Hammanskraal	Not stated	25°23'7.512"S 28°15'16.56"E	Not stated	SO_2, NO,NO_2, NOx PM_{10} CO, O_3, $PM_{2.5}$, PM coarse
Mamelodi	Mamelodi	Residential, Industrial & traffic	25°43'00,408"S 28°20'11"700 E	2009	SO_2, NO,NO_2, NOx PM_{10} CO, O_3,
Olievenhoutbosch	Centurion West	Residential	25°54'42,035"S 28°5'34,638"E	2009	SO_2, NO,NO_2, NOx PM_{10} CO, O_3, $PM_{2.5}$, PM coarse
Pretoria West	Pretoria West	Industrial & traffic	25°45'19,611"S 28°8'45,922"E	2005	SO_2, NO,NO_2, NOx PM_{10} CO, O_3, $PM_{2.5}$,
Tshwane Market	Pretoria West	Industrial & traffic	25°44'23,612"S 28°9'57,773"E	2014	SO_2, NO,NO_2, NOx PM_{10} CO, O_3, $PM_{2.5}$,H_2S
Rosslyn	Pretoria North	Industrial	25°37'30,528"S 28°5'41,089"E	2005	SO_2, NO,NO_2, NOx PM_{10} CO, O_3, $PM_{2.5}$, PM coarse

The meteorological data, which included temperature (°C) and relative humidity (%), wind speed (m/s) rainfall (mm) and wind direction (º), were obtained from the SAWS.

$PM_{2.5}$ levels were compared with an Aeroqual instrument (Figure 3.6). This instrument records continuous monitoring data $PM_{2.5}$. Thus as a measure of reliability, the levels recorded via gravimetric analysis were compared.

Hourly data was obtained, then averaged into daily averages (9 am to 9 am). The Aeroqual had missing data between 19 April 2018 and 15 May 2018, thus the comparison was from 16 May 2018 to 20 April 2019.

Figure 3.6: Image of Aeroqual instrument on the roof of the HW Snyman South building, University of Pretoria

3.4. HUMAN HEALTH RISK ASSESSMENT (HHRA)

The HHRA is used to estimate the probable adverse health effects to humans when exposed to a given environmental pollutant.[124,136-137]

For $PM_{2.5}$ this had only been conducted once before in the City of Tshwane.[73] The HHRA framework comprises four components:

- Hazard identification
- Exposure assessment
- Dose-response
- Risk characterisation

3.4.1. HAZARD IDENTIFICATION

The identification of $PM_{2.5}$ as harmful and its associated health risks was performed in a literature review (stated in chapter 2). Currently, there is clear evidence for long-term and short-term human health effects. This justifies the reasoning of $PM_{2.5}$ as the pollutant of interest.

3.4.2. DOSE-RESPONSE

Dose-response assessment is the manner in which an individual reacts to a particular exposure, was not performed in this study. The degree of the work requires a comprehensive screening as well as additional health data that is presently not available in South Africa.[136] Instead, a comparison was made between the observed $PM_{2.5}$ concentration levels and the daily South African National Ambient Air Quality Standard (NAAQS) of $PM_{2.5}$ (40 µg/m^3) (i.e. intermediate exposure) and the annual NAAQS of $PM_{2.5}$ (25 µg/m^3) (i.e. continuous exposure). The latter served as Reference Exposure Limit (REL). The daily $PM_{2.5}$ WHO guideline of (25 µg/m^3) and annual $PM_{2.5}$ WHO guideline of (10 µg/m^3) was also used as a REL, seeing as they are more protective than the daily and annual $PM_{2.5}$ NAAQS. This approach has been done in other studies.[124,136]

3.4.3. EXPOSURE ASSESSMENT

Inhalation was the most important route of exposure (not ingestion or dermal contact) and that people were exposed to 24 hours per day.[136]

This step focused on the $PM_{2.5}$ concentrations observed within the environment and the time spent in the presence of the air pollutant. In order to investigate the exposure, equations were used to characterise the risks posed by exposure to $PM_{2.5}$, namely:

- The United States Environmental Protection Agency (USEPA) Exposure Factors Handbook
- EPA Integrated Risk Information System (IRIS) equations.[136]
- Studies in South Durban[137] and Pretoria West,[124] South Africa, were adapted the equations from the USEPA and IRIS.

The long-term inhalation rates for adults and children (including infants) were presented as daily rates (m^3/day). It was assumed that chronic mean inhalation rates for infants, children and adults (males and females combined, unadjusted for body-weight) range from 6.8 m^3/day for infants from birth to 1 year, 13.5 m^3/day for children aged 6 to 12 years to 13.3 m^3/day for adults aged 19 to 75 years.[124] The intermediate mean inhalation rates for infants, children and adults (males and females combined, unadjusted for body-weight) range from 0.3 m^3/day for infants from birth to 1 year, 1.2 m^3/day for children aged 6 to 12 years to 1.2 m^3/day for adults aged 19 to 75 years

The Average Daily Dose (ADD) was calculated as follows:

$$ADD = (C \times IR \times ED)/ (BW \times AT)$$ (Equation 3) [124,136-137]

Where:

ADD is the dose the population of Pretoria may be exposed to without suffering negative health risks, which was expressed in µg/kg/day.

C is the average value of the $PM_{2.5}$ concentration measured during from 2018 to April 2019 expressed in µg/m^3.

IR (Inhalation Rate) is the amount of contaminated medium (air) inhaled per unit time or event, expressed in m^3/day.

EF (Exposure Frequency) was calculated on the basis that a person will be absent from study area for 14 days annually, which is rounded off as 350 days as done in other studies.[124,137]

BW is the average body weight (kg).

AT is the period over which exposure is averaged (1 year = 365 days). For non-carcinogens the AT equals ED (years) multiplied by 365 days .[137]

ED (Exposure Duration) expressed is in days. This is calculated as follows

$$ED = ET \times EF \times DE \qquad \text{(Equation 4)}[124]$$

Where:

ET is the exposure time (hour/day).

EF is the exposure frequency (days/year).

DE is the duration of exposure (year).

As done in other local studies, conducted in Durban and Pretoria West,[73,137] a scenario-assessment approach was utilised, where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for intermediate (24-hour) and chronic (annual) exposure periods for different exposure groups (infants, children, adults).

3.4.4. RISK CHARACTERISATION

The risks posed by inhalation exposure to $PM_{2.5}$ in the population of Pretoria were characterised in terms of the potential risk to symptoms or disease in the exposed population. The information compiled in the previous three steps (hazard identification, exposure assessment and dose-response assessment) was integrated into the risk characterisation step to quantify the non-carcinogenic potential health risks in the exposed population, expressed as a Hazard Quotient (HQ).

The HQ was calculated using the following equation:[138]

HQ = ADD/REL (Equation 5)[124]

Where:

HQ is the Hazard Quotient (which is always unit less)

ADD is the Field Average Daily Dose calculated (in µg/kg/day)

REL is the maximum exposure limit.[139] For the current study, the 24-hour $PM_{2.5}$ South African standard (40 µg/m^3), annual $PM_{2.5}$ South African standard (25 µg/m^3) (see appendix 3) and the 24-hour $PM_{2.5}$ WHO guideline (25 µg/m^3) and the annual $PM_{2.5}$ WHO guideline (10 µg/m^3), were used. The guidelines for interpreting the HQ calculations were as follows:[136 140-141]

HQ <0.1: no hazard exists

HQ 0.1-1.0: the hazard is low

Q 1.1-10: the hazard is moderate

HQ >10: hazard is high

3.5. ETHICS APPROVAL

Ethics approval (Reference No: 507/2018) was obtained from the Research Ethics Committee of Faculty of Health Sciences at the University of Pretoria (see Appendix 2). The project did not involve human or animal participants.

3.6. DATA ANALYSIS

Analysis of the $PM_{2.5}$ concentration, along with the soot, BC and organic carbon composition (UVPM), was done using a Microsoft Excel 2013 spreadsheet. All statistical analyses were done using STATA statistical software version 15. All figures were done using Microsoft Excel 2013.

Descriptive statistics for $PM_{2.5,}$ soot, BC, UVPM and the other pollutants measured by the City of Tshwane along with the weather variables were reported, such as minimum, mean, and standard deviation and maximum values along with time-series.

Tests for skewness and kurtosis, as well as Shapiro-Wilk tests for normality were conducted on the exposure variables to determine whether the variables had Gaussian distribution or not.

Non-parametric tests were applied due to skewed distributions. The Kruskal–Wallis test, Wilcoxon's rank-sum test (also known as the Mann-Whitney two-sample statistic) and Spearman rank correlation analysis were applied. Kruskal–Wallis tests were applied to test whether an exposure variable differed significantly across day of the week (Monday to Sunday), and seasons (spring, summer, autumn, winter). Wilcoxon's rank-sum tests were applied to test whether an exposure variable differed significantly between weekdays and weekends, or between weather conditions (dry/wet; windy/calm). Dry days were defined as days that had less than 0.2mm of rainfall. Calm days were classified to be between 0 and 1.5m/s, windy days between 1.6- 5.4m/s, this is according to the Beaufort value system. Linear regression was done between BC and UVPM to determine the regression coefficients.

Spearman rank correlation analyses were performed to determine the correlation coefficients between the exposure variables.

A Wilcoxon sign rank test was used to compare the medians of $PM_{2.5}$ concentrations collected via gravimetric analysis and from the Aeroqual instrument.

CHAPTER 4: RESULTS

In this chapter, the results of the Exposure assessment will be presented. The Human Health Risk Assessment (HHRA).

4.1. DESCRIPTIVE STATISTICS

Table 4.1 summarises the descriptive statistics on the data collected over the 13-month period from 19 April 2018 to 23 April 2019.

In total, 124 $PM_{2.5}$ samples were collected. The $PM_{2.5}$ levels ranged from 2.9 $\mu g/m^3$ to 89.9 $\mu g/m^3$, with a mean of 21.5 $\mu g/m^3$ and a standard deviation of 13.6. Within the same table were the p-values of both the tests for skewness and peakness of the data ($p<0.01$ and $p<0.01$, respectively). These values suggested that the data had skew distribution therefore, non-parametric tests were applied. The average soot levels of 2.04×10^{-7} 10^{-5} m^{-1} indicated that reflectance of the samples were high and soot levels were very low.

Figure 4.1 demonstrates the temporal variation of daily average $PM_{2.5}$ and soot concentrations in Pretoria (Gezina) from 19 April 2018 to 23 April in 2019. A seasonal trend can be observed with its peak in winter and drop in summer.

Table 4.2 shows the averages of $PM_{2.5}$ as the seasons changed from autumn (March to May), winter (June to August), spring (September to November) and summer (December to February).

Table 4.1: Descriptive statistics of $PM_{2.5}$, soot, BC, UVPM and meteorological conditions measured at the School of Health Systems and Public Health, University of Pretoria.

	n	Mean	SD	Min	Max	Skewness statistic	Kurtosis Statistic
$PM_{2.5}$ ($\mu g/m^3$)	124	21.5	13.6	2.9	89.9	***<0.01**	***<0.01**
Soot ($10^{-5}m^{-1}$)	124	0.02	0.14	2.04e-07	1.5	<0.01	<0.01
BC ($\mu g/m^3$)	124	2.6	2.2	-0.2	9.6	<0.01	<0.01
UVPM ($\mu g/m^3$)	124	2.0	1.5	-0.1	6.6	<0.01	<0.01
Wind speed (m/s)	115	1.4	0.6	0	2.8	0.02	0.88
Wind direction	115	119.5	53	0	219	0.19	0.2
Temperature (^0C)	115	18.3	6.3	0	26.3	0.29	<0.01
Relative humidity (%)	115	49.9	20.5	0	86	0.9	0.4
Rainfall (mm)	115	.4	0.9	0	4.3	<0.01	<0.01

SD-Standard Deviation, Min-minimum, Max-Maximum

This sample had a very high reflectance measure, i.e. soot level was very low

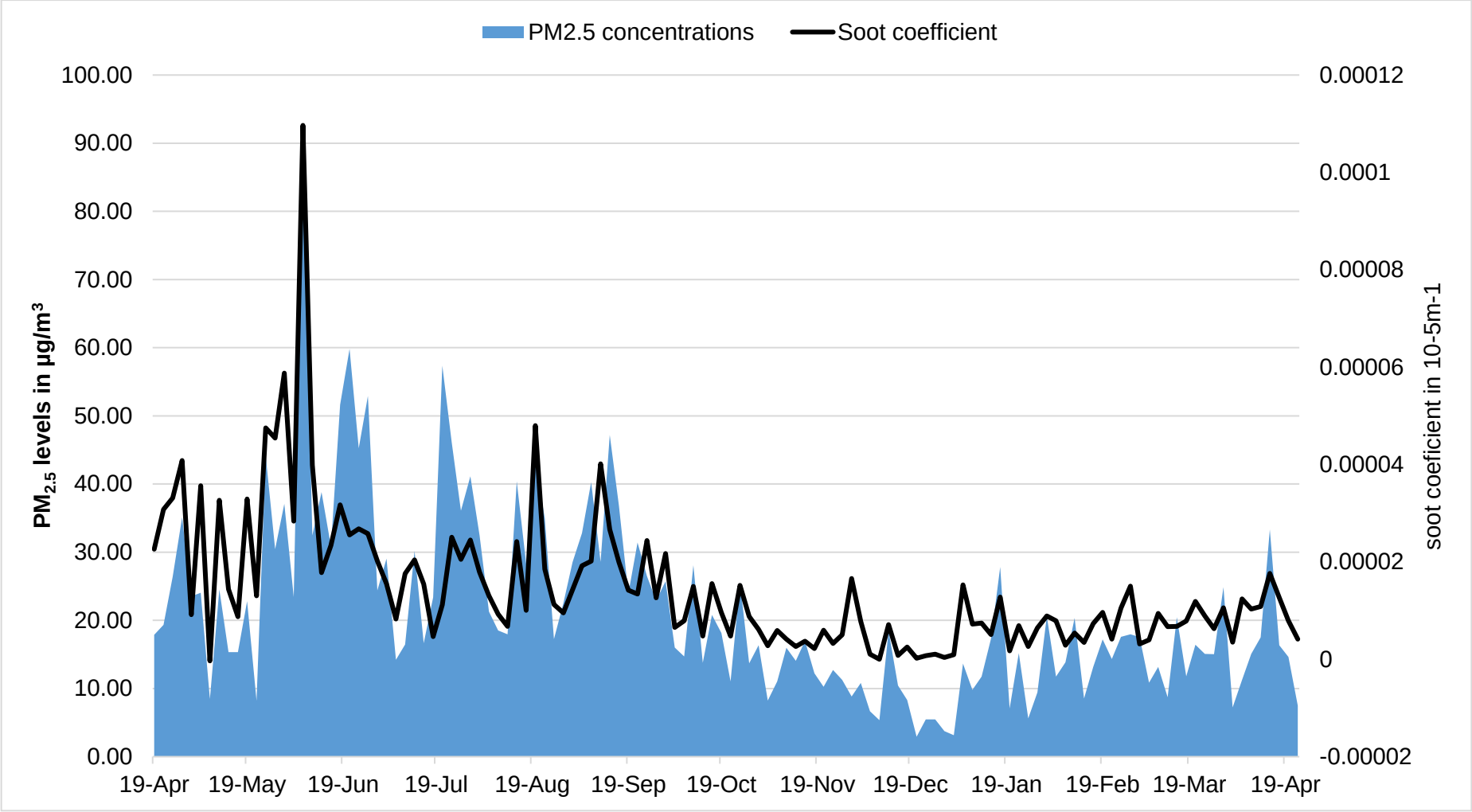

Figure 4.1: Comparison between $PM_{2.5}$ and soot levels measured at the School of Health Systems and Public Health, University of Pretoria.

The temporal variation of BC and UVPM can be seen in Figure 4.2. It illustrates the high concentrations between the months of June and August, winter months. Then the lower concentrations around October to December, summer.

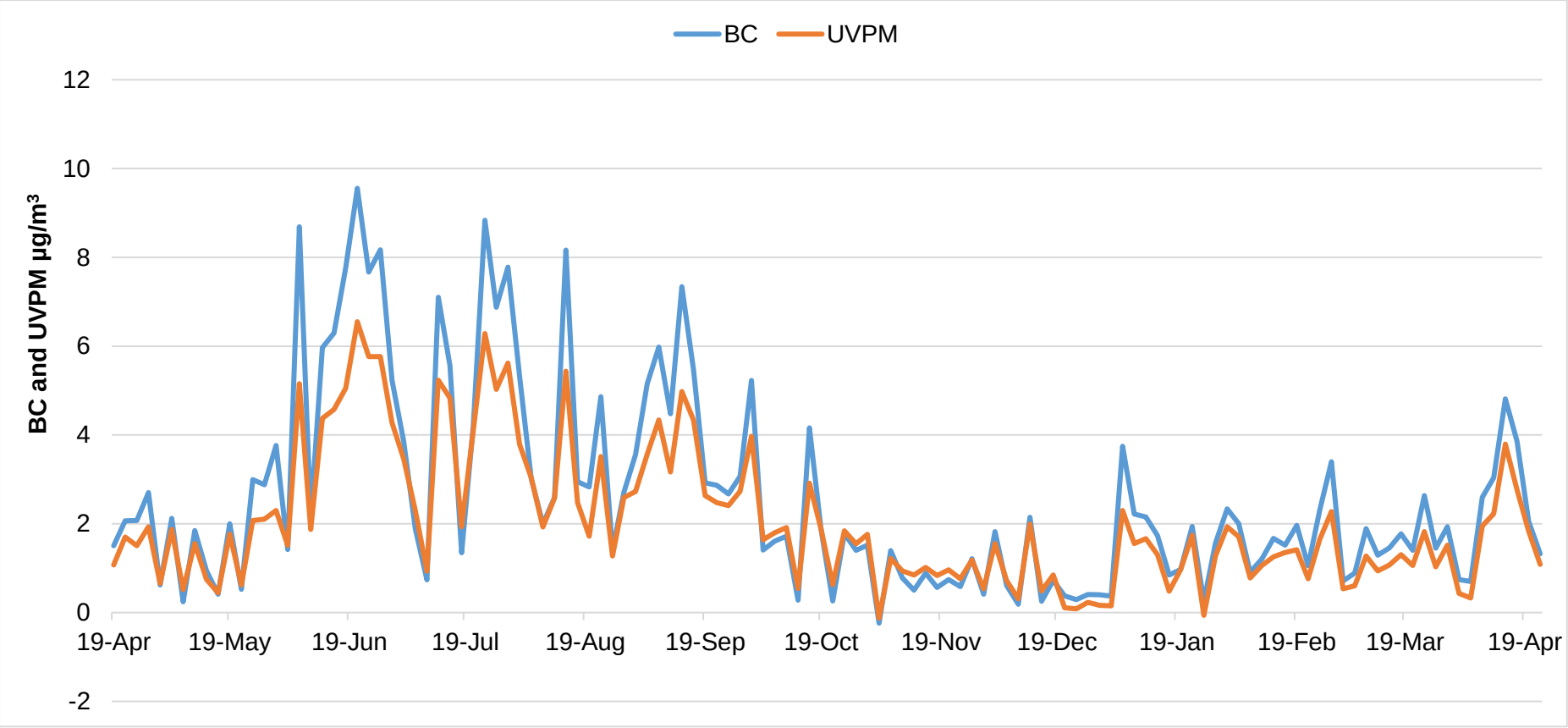

Figure 4.2. Temporal variation of BC and UVPM measured at the School of Health Systems and Public Health, University of Pretoria from April 2018 to April 2019

Table 4.2: Descriptive statistics of $PM_{2.5}$ concentrations ($\mu g/m^3$) across seasons, measured at the School of Health Systems and Public Health, University of Pretoria from 19 April 2018 to 23 April 2019

Season	Average	SD	Min	Max
Autumn	19.1	9.1	7.2	44.1
Winter	34.6	16.6	14.2	89.9
Spring	20.8	9.9	8.2	47.2
Summer	11.8	6.1	2.9	27.8

4.1.1. BLACK CARBON (BC) AND ULTRA-VIOLET ABSORBING PARTICULATE MATTER (UV-PM)

Figure 4.3 shows the linear correlations between $PM_{2.5}$ levels, BC, UVPM and soot. Fairly good correlations are observed at lower $PM_{2.5}$ levels and quite a few outliers are observed.

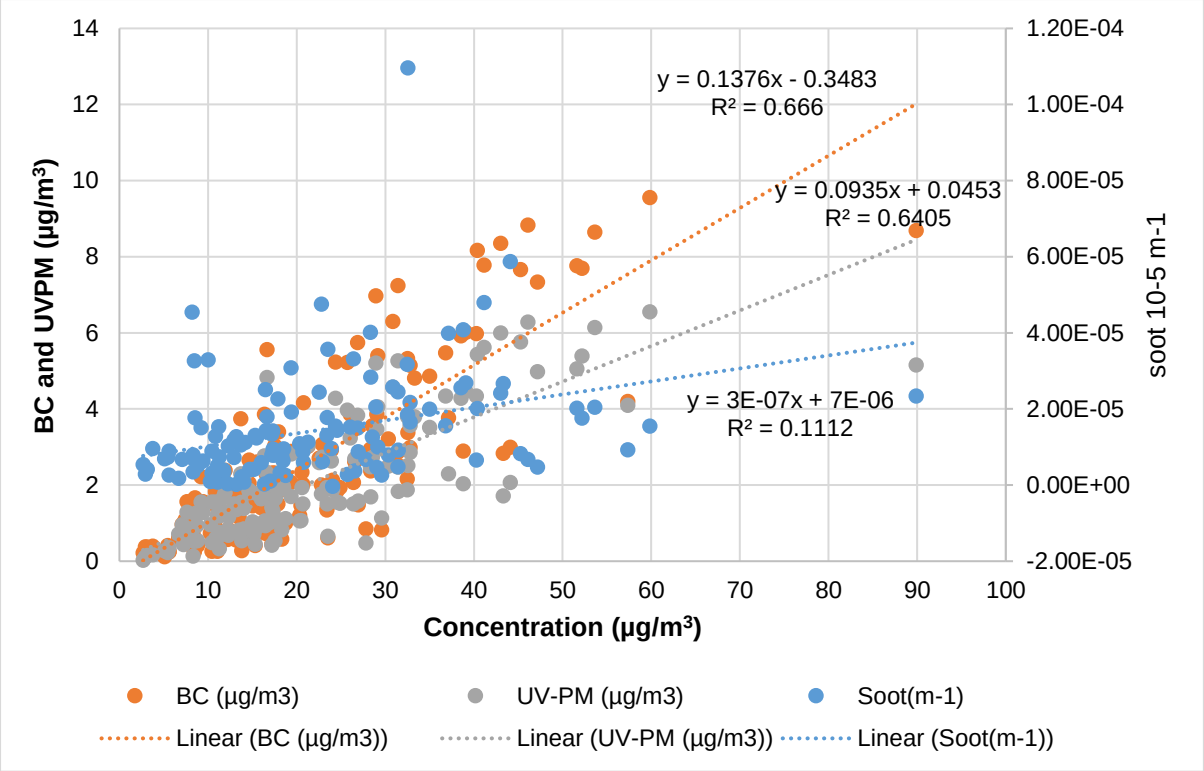

Figure 4.3 Distribution of BC and UVPM measurements content in soot exposure at levels of $PM_{2.5}$, measured at School of Health Systems and Public Health, University of Pretoria from 19 April 2018 to 23 April 2019

Figure 4.4 illustrates the linear regression of UV-PM verses BC produced the coefficients a= 0.281 and b=0.6799 (R^2=0.9627). UVPM, or organic carbonaceous particulate matter absorbing UV light at 370 nm. It indicates that the particle loading effect was corrected for at 0.6799 µg.There is a good linear relationship between BC and UVPM.

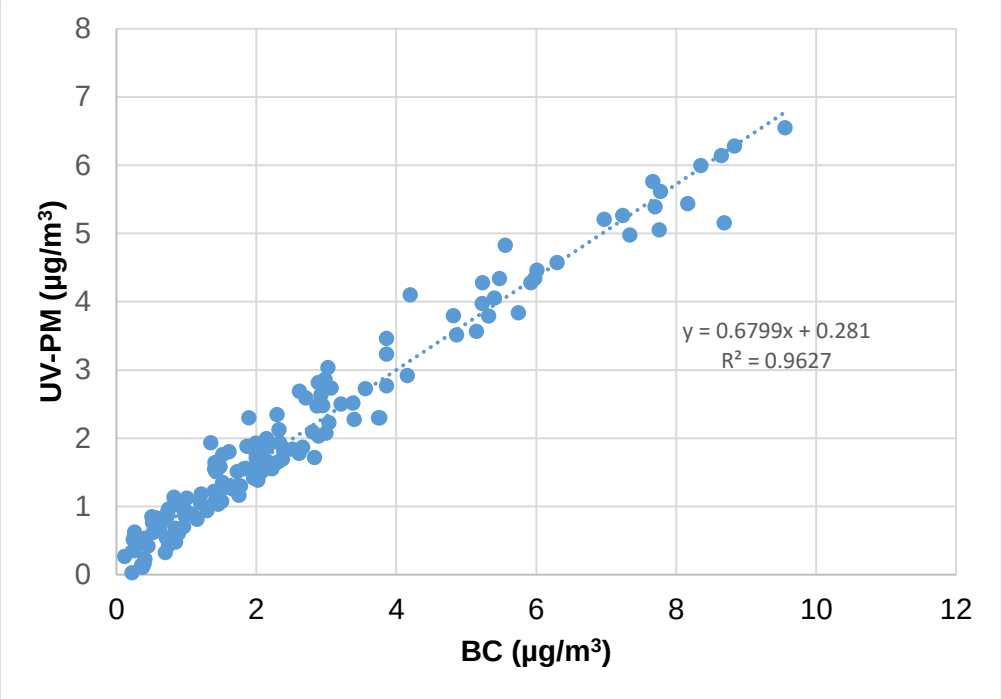

Figure 4.4: Relationship between UVPM and BC, measured at the School of Health Systems and Public Health, University of Pretoria from 19 April 2018 to 23 April 2019

4.2. INFERENTIAL STATISTICS

4.2.1. $PM_{2.5}$ AND OTHER CRITERIA POLLUTANTS

Table 4.3 shows the descriptive statistics of the criteria pollutants, namely NO_2, SO_2, O_3, CO and PM_{10}, obtained from the City of Tshwane. The data shows missing data for all pollutants and no data recorded for PM_{10}.

Table 4.3: Descriptive statistics for criteria pollutants (NO_2,SO_2, O_3, CO,PM_{10})* along with the data collected at the School of Health Systems and Public Health, University of Pretoria from 19 April 2018 to 23 April in 2019

	n	Mean	SD	Min	Max
$PM_{2.5}$ ($\mu g/m^3$)	124	21.5	13.6	2.9	89.9
Soot ($10^{-5}m^{-1}$)	124	0.016	0.1	2.04e-07	1.52
BC $_5$ ($\mu g/m^3$)	124	2.6	2.2	-0.2	9.6
UVPM $_5$ ($\mu g/m^3$)	124	2.0	1.5	-0.1	6.6
NO_2($\mu g/m^3$)	8	9.5	12.9	0.2	34.5
SO_2 ($\mu g/m^3$)	63	18.7	7.9	6.6	41.5
O_3 ($\mu g/m^3$)	94	40.4	20.97	-64.2	93.8
PM_{10}	0	-	-	-	-
CO ($\mu g/m^3$)	30	1.8	0.95	0.3	3.4

*Measured at the Pretoria West monitoring site by the City of Tshwane

Table 4.4 illustrates the correlation between $PM_{2.5}$, soot, BC, UVPM measured at the School of Health Systems and Public Health, University of Pretoria, and other the criteria air pollutants measured by the City of Tshwane. The results show that there a no statistically significant correlations between $PM_{2.5}$ and soot with the other criteria air pollutants. No correlation analysis was performed using the NO_2 and PM_{10} data, due to the lack of sufficient data. BC and UVPM have a significantly strong positive correlation with the $PM_{2.5}$ levels, 0.87 and 0.76 respectively.

Table 4.4: Correlation of $PM_{2.5}$, soot and other criteria pollutants (exposure pollutants)

	$PM_{2.5}$	Soot	BC	UVPM	CO	O_3
Soot	0.63	-				
	0.04					
BC	0.87	0.39	-			
	<0.01*	1				
UVPM	0.76	0.24	0.95	-		
	0.01*	1	p<0.01*			
CO	0.5	0.1	0.55	0.54	-	
	0.37	1	0.17	0.19		
O_3	0.26	-0.11	0.36	0.36	0.01	-
	1	1	1	1	1	
SO_2	-0.14	0.23	-0.36	-0.39	0.09	-0.35
	1	1	1	1	1	1

*Significant levels p<0.05

4.2.2. $PM_{2.5}$ AND METEOROLOGICAL CONDITIONS

Table 4.5 reports the correlations between $PM_{2.5}$, soot and the meteorological conditions (wind speed, temperature, relative humidity and rainfall). This was determined using Spearman's correlation. The results show that soot had a strong positive correlation with $PM_{2.5}$ (0.79). The test also revealed that wind speed, temperature, humidity and rainfall had statistically significant negative relationships with $PM_{2.5.}$ Wind speed, temperature and rainfall had moderate correlations -0.47 (p<0.01),-0.45 (p<0.01) -0.34 (p<0.01), respectively. While humidity had a weak correlation with $PM_{2.5}$,-0.29 (p=0.02).

Table 4.5: Correlation relationship $PM_{2.5}$, soot and meteorological conditions

	PM2.5	Wind speed	Temp	Humidity
Soot	0.79			
	<0.01*			
Wind speed	-0.47	-		
	<0.01*			
Temp	-0.45	0.50	-	
	<0.01*	<0.01		
Humidity	-0.29	0.23	0.19	-
	0.02	0.15	0.58	
Rainfall	-0.34	0.38	0.31	0.53
	<0.01*	<0.01	<0.01	<0.01

*Significant levels p<0.05

Comparisons were made between weekdays and weekends, dry and wet weather conditions, windy and calm weather conditions and between months. The averages are reported in Table 4.6. There is no significant difference for $PM_{2.5}$ levels observed on weekdays and weekends (p=0.73). However, there is a significant difference for $PM_{2.5}$ levels sampled during dry and wet conditions (p<0.01) as well as during windy and calm conditions (p=0.01).

Table 4.6: Average $PM_{2.5}$ levels ($\mu g/m^3$) across months, measured at the School of Health Systems and Public Health, University of Pretoria from 19 April 2018 to 23 April in 2019

	Jan	Feb	Mar	Apr	May	Jun
Average($\mu g/m^3$)	12.1	15.5	15.4	15.4	23.1	44.9
SD	7	3.9	4.8	8.2	11.2	20.1
Min	3.1	8.5	8.7	7.2	8.2	23.4
Max	27.8	20.6	24.9	33.3	44.1	89.9
	Jul	**Aug**	**Sept**	**Oct**	**Nov**	**Dec**
Average($\mu g/m^3$)	31.1	27.7	31.9	18.5	12.2	7.8
SD	14.2	9.6	7.6	5.7	2.9	4.6
Min	14.2	17.2	22.9	11.1	8.2	2.9
Max	57.4	43.3	47.2	28.1	17	18.5

Table 4.7 lists the average $PM_{2.5}$ levels on weekdays (Monday to Friday) and weekends (Saturday to Sunday).

Table 4.7: Average $PM_{2.5}$ levels ($\mu g/m^3$) on weekdays and weekends, measured at the School of Health Systems and Public Health, University of Pretoria during 19 April 2018 to 23 April in 2019

	Weekdays	Weekends
Average	22.2	19.9
SD	14.4	11.6
Min	2.9	3.8
Max	89.9	57.4

Table 4.8 indicates the average $PM_{2.5}$ levels during days with different weather conditions, i.e. dry or wet, calm or windy conditions. The results indicated higher $PM_{2.5}$ levels in dry conditions compared to wet conditions and during calm conditions compared to windy conditions.

Table 4.8: Average $PM_{2.5}$ ($\mu g/m^3$) on dry/wet and windy/calm days, measured at the School of Health Systems and Public Health, the University of Pretoria from 19 April 2018 to 23 April in 2019

Dry	Wet	Windy	Calm
24.3	14	16.1	24.8

Dry = 0mm rainfall, Wet >0mm rainfall

Calm=0-1.5 m/s, Windy< 1.6-5.4 m/s (wind speeds did not exceed 5.4 m/s)

$PM_{2.5}$ levels differed significantly across months ($p<0.01$) and seasons ($p<0.01$), but not across days of the week ($p=0.85$).

Tables 4.9 and 4.10 indicate the post hoc test results after conducting the Kruskal-Wallis tests across months and seasons, respectively.

Table 4.9: Post hoc test for the Kruskal-Wallis test for months

Dunn's Pairwise Comparison of $PM_{2.5}$ by months						
	January	**February**	**March**	**April**	**May**	**June**
February	0.14					
March	0.17	0.45				
April	**0.05**	0.30	0.26			
May	**<0.01**	0.09	0.07	0.19		
June	**<0.01**	**<0.01**	**<0.01**	**<0.01**	**<0.01**	
July	**<0.01**	**<0.01**	**<0.01**	**0.02**	0.12	0.11
August	**<0.01**	**<0.01**	**<0.01**	**0.02**	0.14	0.10
September	**<0.01**	**<0.01**	**<0.01**	**<0.01**	**0.04**	0.28
October	**0.04**	0.25	0.21	0.43	0.24	**<0.01**
November	0.49	0.14	0.17	**0.05**	**<0.01**	**<0.01**
December	0.15	**0.02**	**0.02**	**0.002**	**<0.01**	**<0.01**

The significant differences indicated in bold

Table 4.10: Post hoc test for the Kruskal-Wallis test for seasons

Dunn's Pairwise Comparison of $PM_{2.5}$ by seasons			
	Autumn	**Winter**	**Spring**
Winter	0.1		
Spring	**<0.01**	**<0.01**	
Summer	**<0.01**	**<0.01**	0.21

The significant differences indicated in bold

4.3. COMPARISON ON GRAVIMETRIC MEASUREMENTS WITH AEROQUAL

Figure 4.5 shows the $PM_{2.5}$ concentrations collected in the two techniques, namely gravimetric analysis and the real-time continuous Aeroqual instrument. There was no significant difference ($p=0.07$) between the median $PM_{2.5}$ concentrations recorded using the two techniques.

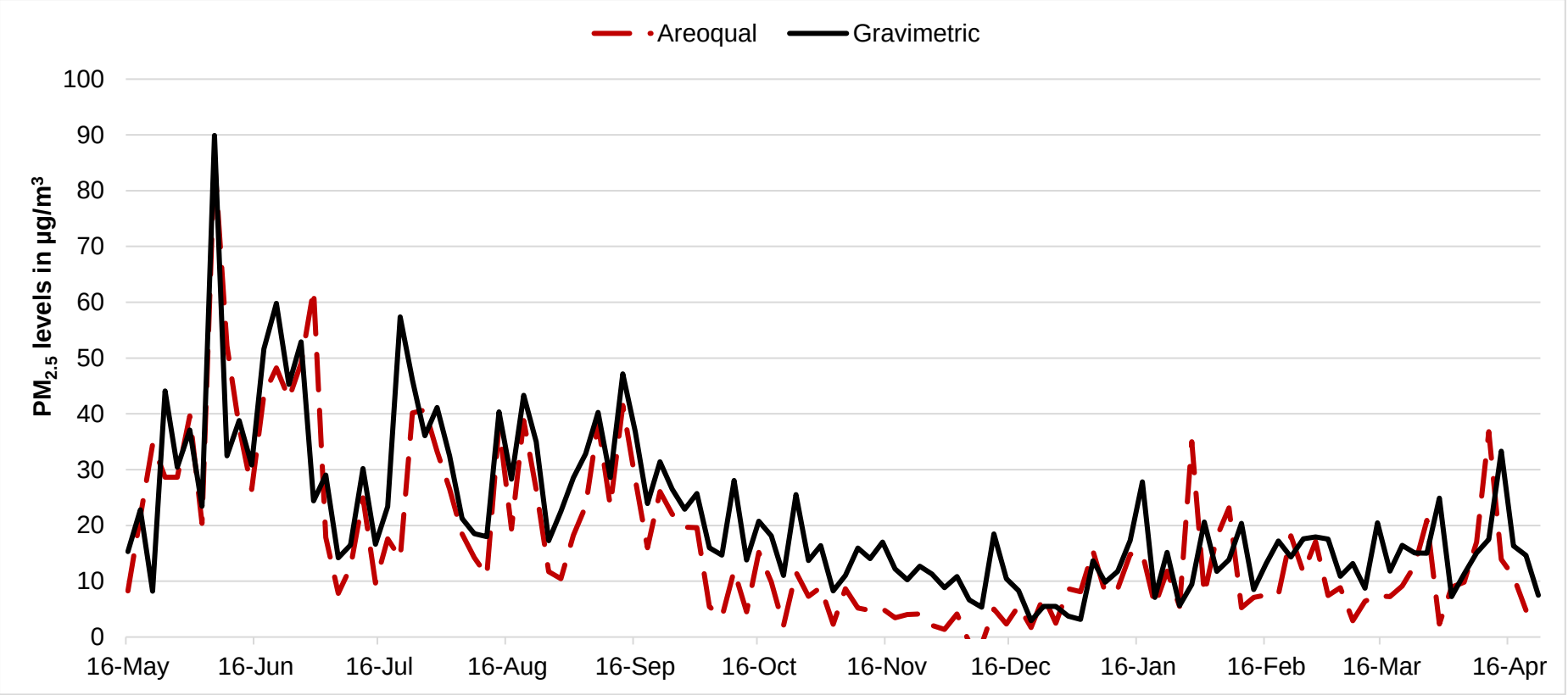

Figure 4.5 Comparison between $PM_{2.5}$ levels obtained with gravimetric analysis against the real-time continuous Aeroqual instrument, measured at the School of Health Systems and Public Health, University of Pretoria from 19 April 2018 to 23 April in 2019

4.4. HUMAN HEALTH RISK ASSESSMENT

4.4.1. HAZARD IDENTIFICATION

$PM_{2.5}$ has been identified as a hazard in the Literature Review: refer to Chapter 2.

4.4.2. DOSE-RESPONSE ASSESSMENT

Figure 4.5 illustrates the $PM_{2.5}$ concentrations against the 24-hour average benchmark concentrations of the daily South African standard and the WHO guideline, 40 µg/m^3 and 25 µg/m^3, respectively. However, the South African standard has a number of exceedance of 4 (Table2.1), extending the limit value to 44 µg/m^3.

The recorded $PM_{2.5}$ levels indicated 7.3 % (9/124), which were above the South African standard, while 29.8% (37/124) were above the WHO's guideline.

4.4.3. EXPOSURE ASSESSMENT

The information applied in the following tables (e.g. exposure frequency, exposure duration, averaging time, inhalation rate) were obtained from tables used in similar studies conducted in South Africa.[124,137]

Table 4.11: Exposure frequency, exposure duration and averaging time

Exposure group	Exposure frequency (days/year)	Exposure duration (year)	Averaging time (days)
Infant (birth to 1 year)	350	1	365 (=1*365)
Child (6 to 12 years)	350	12	4380 (=12*365)
Adult (19 to 75 years)	350	30	10950 (=30*365)

Source: From Matooane and Diab[137] , Morakinyo *et al.*[124] and US Environmental Protection Agency[138]

Table 4.11 shows the averaging time of the three exposure groups over 350 days of the year. The exposure durations are the average number of years to which each group is exposed. As the exposure group-age increases, the averaging time increases respectively.

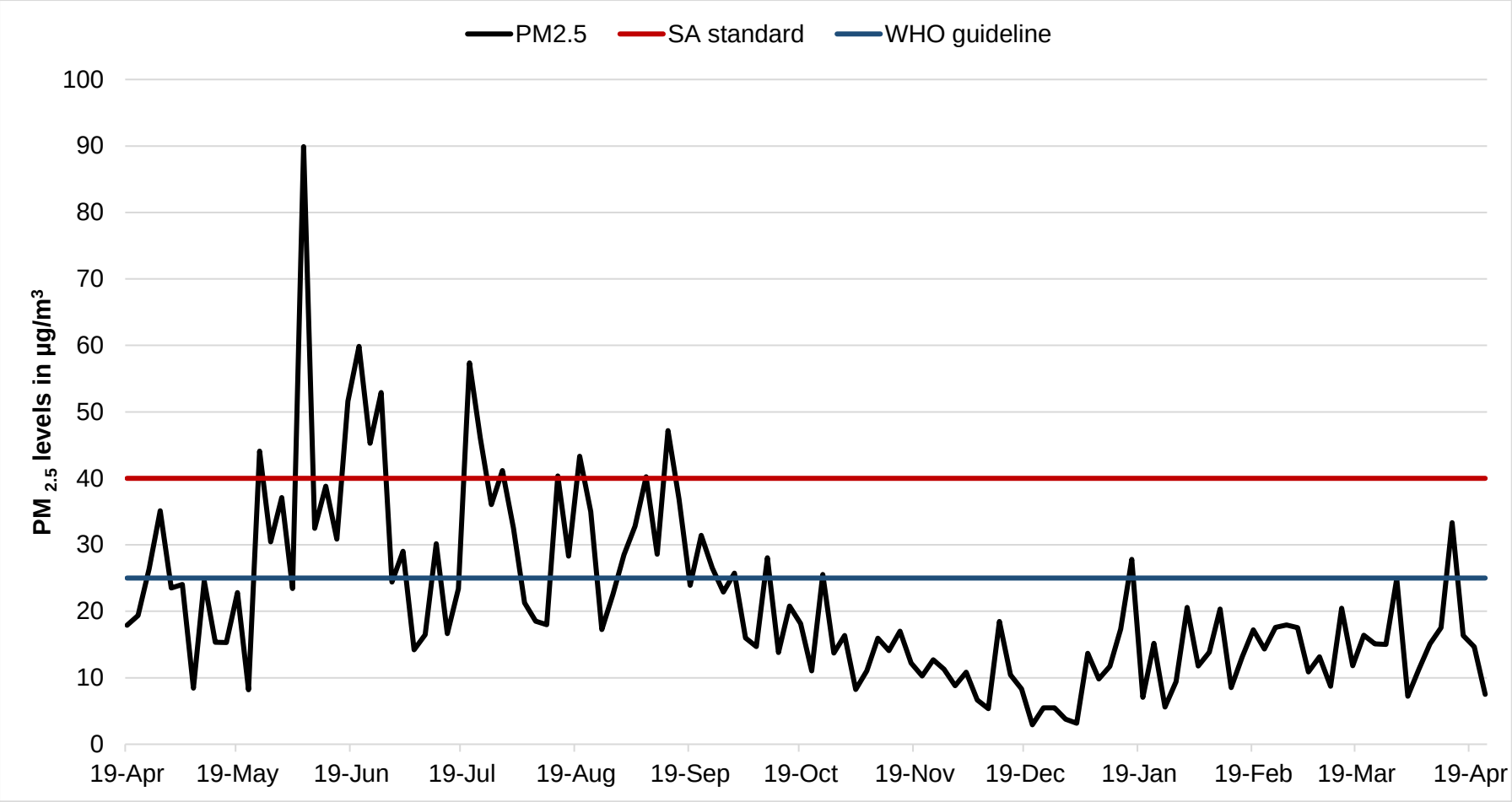

Figure 4.6: $PM_{2.5}$ concentrations against the 24-hour averages of the South African Standard and World Health Organization guidelines, 40 µg/m³ and 25 µg/m³, respectively

Table 4.12 shows the estimated exposure time values for each exposure group (infants, children, adults), which was based on the average and continuous scenarios for, intermediate and chronic exposure periods.[124,137-138] Default values were used for inhalation rates and body weights and presented in Table 4.12 for each exposure group.

Table 4.12: Exposure time (hours) for normal and worst-case scenarios for intermediate and chronic exposures

	Intermediate		Chronic	
Exposed group	**Normal**	**Worst case**	**Normal**	**Worst case**
Infant (birth to 1 year)	1	24	14.6	350
Child (6 to 12 years)	6	24	1050	4200
Adult (19 to 75 years)	3	24	1312.5	10500

Source: Adapted from Matooane and Diab[137] , Morakinyo *et al* [124]and US Environmental Protection Agency[138]

14.6 ((=350/24)*1); 1050 ((=4200/24)*6); 1312.5 ((=10500/24)*3)

350 (=1*350); 4200 (=12*350); 10500 (=30*350)

Table 4.13 shows the inhalation rates of the different exposure groups and their corresponding body weights assumed for this study.

Table 4.13: Averaging inhalation rates and body weights of the exposed population

Mean Inhalation rate (m^3/day)			
Exposed group	**Intermediate exposure**	**Chronic exposure**	**Mean body weight (kg)**
Infant (birth to 1 year)	0.3	6.8	11.3
Child (6 to 12 years)	1.2	13.5	45.3
Adult (19 to 75 years)	1.2	13.3	71.8

Source: Adapted from Matooane and Diab[137] , Morakinyo *et al* [124]and US Environmental Protection Agency[138]

Table 4.14 reports on the intermediate Average Daily Dose (ADD) for inhalation (m^3/day) for the exposure groups; there is no difference in doses between infants and children, but a lower dose can be seen adults (0.57, 0.57 and 0.36, respectively). However, the chronic exposure in Average Daily Dose (ADD) for inhalation shows an evident increase among the exposure groups. The infant dose is almost double the child dose and almost three times the adults' dose (12.93 in infants, 6.40 in children and 3.98 in adults).

Table 4.15 shows the ADD of the exposure groups, exhibiting the same trend as that in Table 4.14, i.e. the doses are highest in winter and lowest in summer (Table 4.16).

Autumn (Table 4.17) and spring (Table 4.18) have similar doses of ADD. The intermediate ADD doses are slightly different between infants and children. The difference is more evident in chronic exposure doses.

Table 4.14: Calculated average daily dose for intermediate exposure and chronic exposure based on the $PM_{2.5}$ concentration during the entire year (21.5 µg/m^3)

Age groups (years)	Body Weight (kg)	Inhalation Rate (m^3/day)		Average Daily Dose-intermediate (µg/kg/day)	Average Daily Dose – chronic (µg/kg/day)
		Intermediate	Chronic	Intermediate	Chronic
Infant (birth to 1 year)	11.3	0.3	6.8	0.57	12.93
Child (6 to 12 years)	45.3	1.2	13.5	0.57	6.40
Adult (19 to 75 years)	71.8	1.2	13.3	0.36	3.98

The values in the table were derived using Equation 3

Table 4.15: Calculated average daily dose for intermediate exposure and chronic exposure based on the $PM_{2.5}$ concentration during winter (34.6 µg/m^3)

Age groups (years)	Body Weight (kg)	Inhalation Rate (m^3/day)		Average Daily Dose-intermediate (µg/kg/day)	Average Daily Dose – chronic (µg/kg/day)
		Intermediate	Chronic	Intermediate	Chronic
Infant (birth to 1 year)	11.3	0.3	6.8	0.92	20.8
Child (6 to 12 years)	45.3	1.2	13.5	0.92	10.3
Adult (19 to 75 years)	71.8	1.2	13.3	0.578	6.4

The values in the table were derived using Equation 3

Table 4.16: Calculated average daily dose for intermediate exposure and chronic exposure based on the $PM_{2.5}$ concentration during summer (11.8 µg/m^3)

Age groups (years)	Body Weight (kg)	Inhalation Rate (m^3/day)		Average Daily Dose-intermediate (µg/kg/day)	Average Daily Dose – chronic (µg/kg/day)
		Intermediate	Chronic	Intermediate	Chronic
Infant (birth to 1 year)	11.3	0.3	6.8	0.31	7.09
Child (6 to 12 years)	45.3	1.2	13.5	0.31	3.51
Adult (19 to 75 years)	71.8	1.2	13.3	0.2	2.18

The values in the table were derived using Equation 3

Table 4.17: Calculated average daily dose for intermediate exposure and chronic exposure based on the $PM_{2.5}$ concentration during autumn (19.1 µg/m^3)

Age groups (years)	Body Weight (kg)	Inhalation Rate (m^3/day)		Average Daily Dose-intermediate (µg/kg/day)	Average Daily Dose – chronic (µg/kg/day)
		Intermediate	Chronic	Intermediate	Chronic
Infant (birth to 1 year)	11.3	0.3	6.8	0.51	11.48
Child (6 to 12 years)	45.3	1.2	13.5	0.51	5.69
Adult (19 to 75 years)	71.8	1.2	13.3	0.32	3.53

The values in the table were derived using Equation 3

Table 4.18: Calculated average daily dose for intermediate exposure and chronic exposure based on the $PM_{2.5}$ concentration during spring (20.8 µg/m^3)

Age groups (years)	Body Weight (kg)	Inhalation Rate (m^3/day)		Average Daily Dose-intermediate (µg/kg/day)	Average Daily Dose – chronic (µg/kg/day)
		Intermediate	Chronic	Intermediate	Chronic
Infant (birth to 1 year)	11.3	0.3	6.8	0.55	12.54
Child (6 to 12 years)	45.3	1.2	13.5	0.55	6.21
Adult (19 to 75 years)	71.8	1.2	13.3	0.35	3.86

The values in the table were derived using Equation 3

4.4.4. RISK CHARACTERISATION

As mentioned before, a scenario-assessment approach was utilised, where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for intermediate (24-hour) and chronic (annual) exposure periods for different exposure groups (infants, children, adults).

For the results in Table 4.19, the daily $PM_{2.5}$ South African standard (40 µg/m^3) and annual $PM_{2.5}$ South African standard (25 µg/m^3) were applied as the Reference Exposure Limits. The estimated hazard quotients are less than 1 for all the age groups during both scenario-assessment approaches (normal or worst-case) regardless of intermediate (24-hour) and chronic (annual) exposure periods.

Table 4.19: Hazard quotients for normal and worst-case exposure scenarios to $PM_{2.5}$ during intermediate and chronic exposure

	Intermediate exposure		**Chronic exposure**	
	Worst-case	**Normal**	**Worst-case**	**Normal**
Average for infants	0.32	0.01	0.52	0.02
Average for children	0.01	0.01	0.26	0.02
Average for adults	0.01	0.01	0.16	0.01

Daily $PM_{2.5}$ South African standard (40 µg/m^3) and annual $PM_{2.5}$ South African standard (25 µg/m^3) applied as the Reference Exposure Limit

For the results in Table 4.20, the daily $PM_{2.5}$ WHO (25 µg/m^3) and annual $PM_{2.5}$ WHO guideline (10 µg/m^3) were applied as the Reference Exposure Limits. The estimated hazard quotients are less than 1 for most of the age groups during both scenario-assessment approaches (normal or worst-case) regardless of intermediate (24-hour) and chronic (annual) exposure periods. However, the HQs in Table 4.20 are larger than those in Table 4.19, i.e. the risk increases when the more protective WHO guideline is applied as the exposure limit.

Table 4.20: Hazard quotients for normal and worst-case exposure scenarios to $PM_{2.5}$ during intermediate and chronic exposure

	Intermediate exposure		Chronic exposure	
	Worst-case	**Normal**	**Worst-case**	**Normal**
Average for infants	0.52	0.02	1.29	0.06
Average for children	0.26	0.02	0.64	0.06
Average for adults	0.16	0.01	0.40	0.04

Daily $PM_{2.5}$ WHO guideline (25 µg/m^3) and annual $PM_{2.5}$ WHO guideline (10 µg/m^3) applied as the Reference Exposure Limit

Chapter 5: DISCUSSION

5.1. EXPOSURE ASSESSMENT

The purpose of this study was to provide baseline information on the concentration levels of $PM_{2.5}$ in the urban area of Gezina, Pretoria, thereafter identify potential health risks of inhalable $PM_{2.5}$ exposure.

In total, 124 observations were recorded, derived from the 149 samples acquired. The duplicates were averaged to get one recording for the day. The results indicated an average daily mean of 21.5 µg/m^3, with the standard deviation of ±13.6. This average falls below the South African daily average standard, but exceeds the WHO daily average guideline. The minimum daily average concentration was observed in January 2019, at 2.9 µg/m^3 and highest daily average concentration of 89.9 µg/m^3 was observed in the month June 2018. The lowest concentrations were obtained when the temperatures and rainfall were the highest. The highest concentration was obtained during the winter period, when it is often expected that heating appliances and combustion for heating purposes are at their highest, respectively.[92]

The median of 17.5 µg/m^3 lies below the standard and the guideline. Due to the data being skew (Shapiro-Wilk test, $p<0.01$), non-parametric tests were conducted to test for the differences among days, months and across seasons. Differences between weekdays and weekends were tested as well as differences between different conditions, such a dry and wet as well as windy and calm conditions.

The concentration levels during winter varied from 14.2 µg/m^3 and 89.9 µg/m^3 with an average of 34.5 µg/m^3 ±16.6, while summer varied 2.9 µg/m^3 and 27.8 µg/m^3 with an average of 11.8± 6.1 µg/m^3; this was in an urban area. Comparing this to the results of a study conducted in an industrial area[73], where the measurements varied from 22.4 and 67.2 µg/m^3 with a mean of 38.3±8.4 µg/m^3 in winter, and 16.6 to 43.3 µg/m^3 and an average of 22.3±4.1 µg/m^3 in summer.[73] It is evident that the summer average from the urban area is lower than that of the industrial area, however the winter

concentrations of the two different areas are close to each other. The study[73] did not report information on autumn and spring.

Soot and UVPM are chemical components of $PM_{2.5}$ and the results show that there were strong positive correlations between the components. This relationship is supported by other studies.

5.1.1. CRITERIA AIR POLLUTANTS INTERACTION WITH $PM_{2.5}$

The $PM_{2.5}$ concentration levels were put against the criteria pollutants PM_{10}, CO, NO_2, SO_2 and O_3. The data used was received from the nine DEff air monitoring stations. The majority of the monitoring stations had insufficient data to make comprehensive comparisons, therefore the Pretoria West station data was used to make the comparisons as it had the largest 'complete' data set. The data from the Pretoria West site did however have a lot of missing data, therefore the conclusions of the results may have been heavily influenced. It is for this reason there was no information on the correlation between $PM_{2.5}$ and PM_{10} and NO_2. The study failed to show any significant association between $PM_{2.5}$, CO, O_3 and SO_2, however, studies would suggest that there are moderate to strong relationships between $PM_{2.5}$, SO_2, CO and NO_2; correlations between $PM_{2.5}$ and O_3 are either weak or uncorrelated.[142-143]

In a study conducted in Pretoria West in 2017,[124] concentration levels of PM_{10}, SO_2, O_3, NO_2 and CO were recorded by the DEFF at the Pretoria West air monitoring station. The annual averages for PM_{10}, SO_2 and NO_2 were, 48.3 µg/m^3 ± 43.4, 11.5 µg/m^3 ±11.6 and 18.7 µg/m^3 ± 25.4, respectively.[124] During the study, no 24-hour averages were provided for CO and O_3. Within this study the annual averages recorded were CO 1.8 µg/m^3 ± 0.95, SO_2 18.7 µg/m^3 ± 7.9, NO_2 9.5 µg/m^3 ± 12.9 and O_3 40.4 µg/m^3 ± 20.97; there was no data provided for PM_{10}. When comparing the two areas, an industrial area compared to the urban area, this was the following conclusion. The concentration levels of NO_2 have decreased in comparison to the concentration levels in 2018-2019. However, the SO_2 levels collected in 2016 are lower than those collected from 2018 to 2019, suggesting an increase over the span of two years. The comparison may not be

conclusive due to the amount of missing data, consequently, a fair comparison cannot be made for PM_{10}, CO and O_3.

5.1.2. METEOROLOGICAL EFFECTS ON $PM_{2.5}$ CONCENTRATIONS

In this study there were moderate relationships seen between $PM_{2.5}$ levels, wind speed, temperature, and rainfall. The relationships were statistically significant negative relationships, wind speed, temperature and rainfall had moderate correlations, -0.47 ($p<0.001$), -0.45 ($p<0.001$) -0.34 ($p=0.003$), respectively. Relative humidity did not have a strong relationship with $PM_{2.5}$, -0.29 ($p=0.02$). This would suggest that as the wind speed increased, the $PM_{2.5}$ concentrations would, to a certain extent, decrease. Research has shown that the movement of air disperses $PM_{2.5}$ and reduces the particles in the atmosphere.[92] This would equally occur in the presence of rainfall. Often in literature, it does indicate that as the meteorological conditions, namely, temperature, wind speed, relative humidity and rainfall increase, the $PM_{2.5}$ decreases,[88,93] to which this study's results concur with previous findings. This is despite a portion of the weather data received being missing, which may have affected the findings.

Studies have stated that $PM_{2.5}$ concentrations may be sensitive to the changes in climate, but not as sensitive as the emissions in the area.[94] Varying weather patterns, increased winds, and warmer winters could be affecting the concentration levels. However, as stated in Chapter 2, the study area was an urban area, suggesting that in an area of a different nature, such as an industrial area, there could have been different results.

There was no significant difference in the $PM_{2.5}$ levels measured during weekdays and weekends. However, there was a significant difference in dry and wet conditions ($p=0.002$). According to literature, when there is the presence of rainfall the particulate matter and other air pollutants are moved with the rain and "washes out" from the environment.[92] Thus the likelihood is that the concentration will be higher in drier conditions than that of wet conditions. There was a significant difference between

windy and calm conditions, (p=0.001), insinuating that there are higher $PM_{2.5}$ concentrations during calmer conditions and it would be expected that there are lower concentrations when wind speeds are higher.[144]

Significant differences were seen among months and across the seasons, p<0.001 and p<0.001, respectively. However, it was not evident that there was a difference within days of the week, p=0.85. The results further show that the colder month, as well as seasons, do differ from the warmer months and seasons. The findings are supported by literature, which indicates that the concentration levels of $PM_{2.5}$ are bound to differ across seasons. Multiple studies have recorded higher concentrations of particulate matter and gaseous pollutants,[124,145-146] reduced precipitation, minimal air movement as well as low wind speed, strong inversion and low relative humidity often occurring during winter.[87-89,147] $PM_{2.5}$ concentrations are higher in colder months to which the results of this study agree; in addition, higher concentrations of $PM_{2.5}$ are mostly observed in winter time.[148] However this observation can only be seen in areas that have dry winters and wet summers, as in areas that experience wet winters and dry summers the literature will be explained differently.[90]

5.1.3. BLACK CARBON (BC) AND ULTRAVIOLET PARTICULATE MATTER (UVPM)

The linear regression relationship between the $PM_{2.5}$ levels were measured from the sampling site and the BC. The slope, intercept, and R^2 values were on 0.35, 0.14, 0.67, respectively. The relationship between the $PM_{2.5}$ levels and the UVPM, the slope, intercept and R^2 values were on 0.05, 0.09 and 0.64, respectively. In addition, a strong relationship was shown between BC and UVPM and chemical components of $PM_{2.5}$. [149], indicating importance of investigating the different chemical components on $PM_{2.5.}$

5.1.4. COMPARISON WITH AREOQUAL INSTRUMENT

A comparison between the $PM_{2.5}$ levels obtained by the gravimetric method and the $PM_{2.5}$ levels obtained by the Areoqual instrument were made. The results indicated a strong positive correlation between the two processes. A Wilcoxon sum rank test indicated there was no difference between the two samples (p=0.07). Hence, using the small GilAir pumps was a valid option to measure $PM_{2.5}$ as there was no significant difference between the $PM_{2.5}$ levels measured by the two methods. However, the results provided by the Aeroqual instrument were not complete. The comparison was only made from the 16 May 2018 to 20 April 2019, due to missing data from the Aeroqual instrument.

5.2. $PM_{2.5}$ CONCENTRATIONS IN ACCORDANCE WITH HUMAN HEALTH RISK ASSESSMENT

The health risk assessment was conducted with slight modification. Nonetheless, all four stages were conducted. This method enabled the estimations of the nature and the probability of any adverse effects in humans upon being exposed to chemicals such as $PM_{2.5.}$ [150] There are two types of classification that the risk can be classified under, either carcinogenic or non-carcinogenic. [150]

5.2.1. HAZARD IDENTIFICATION

The hazard identification was identified in Chapter 1 (literature review), where the associated health risks were identified. The literature review also revealed that $PM_{2.5}$ was a non-carcinogenic risk, despite the knowledge presented that there are elements within $PM_{2.5}$ that are classified as carcinogenic. This conclusion was made because there was no chemical analysis done on the composition of the collected $PM_{2.5}$ samples.

5.2.2. DOSE-RESPONSE

The dose-response assessment stage explores the relationship between the concentration (dose) and the health effects, as well as the severity of the effect the pollutant is likely to cause in the body. [151] In a conventional HHRA, the dose-response assessment is the amount of the pollutant taken into the body and estimated as a function of concentration and the length of exposure.[124,137] Due to the nature of this study as a health impact study, it was established that only outward exposure to the community was to be considered. Figure 5.1 below illustrates the manner in which the pollutant would further progress upon entering the body. The circle shows the area of concentration for this study only concentrating on the exposure of the chemical. The route of exposure identified was inhalation and the exposure media the air.

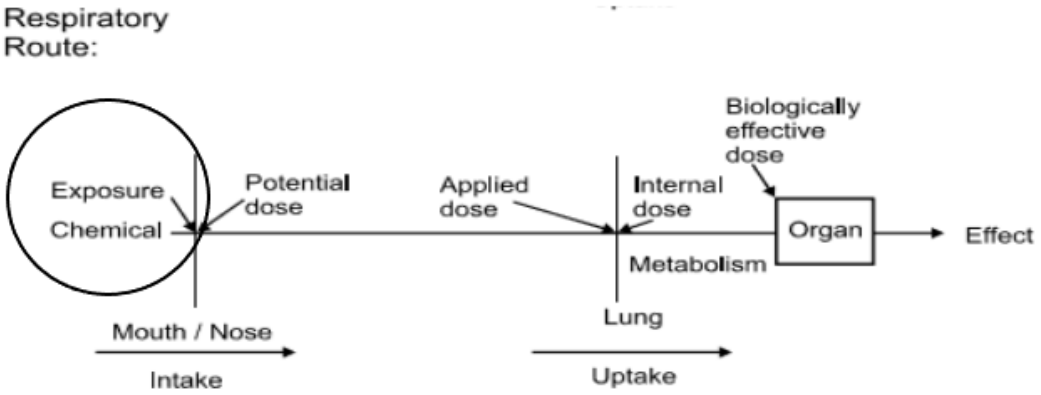

Figure 5.2: Image from US EPA[139], indicating the area of exposure the study focused on

Due to the complexity of this aspect of the HHRA, a dose-response was not performed in this study instead a comparison between the measured ambient concentration of $PM_{2.5}$ and REL was done. The RELs were the South African National Air Quality standard and the WHO guideline, 40 µg/m^3 and 25 µg/m^3, respectively.10.5 % of the recorded $PM_{2.5}$ levels were above the SA standards, while 29.8% were above the WHO guidelines. This could be attributed to a number of different factors. Firstly, it could be a result of the location of the sampling site. Being an urban area with very little activity occurring in the area, it could be anticipated that the pollution levels were not high. The sources of hydrocarbon could only be attributed to the traffic and the

hospital incinerator that was located close to the sampling area. There were also some outside fires observed, but these could not be considered a major source of pollution, as they were scarce and random. Literature often explains that higher pollution areas are often close to the highway, industrial areas and areas where burning is common.[10,152-153]

5.2.3. EXPOSURE ASSESSMENT

The exposure assessment identifies the population exposed to the hazard, the magnitude and the duration of exposure to the hazard. In the case of this study, the major focus was on the contact exposure of $PM_{2.5}$ rather than the contact exposure and the actual entry (internal dose) of $PM_{2.5}$.[125,154] Within Pretoria, where the sampling took place, there is an estimated population of 741 651 people, as per a census done in 2011.[155] The gravimetric analysis provided the magnitude of the $PM_{2.5}$ concentrations. The major route of exposure was inhalation. A scenario-based assessment method was used as recorded in other studies of a similar nature. In such a scenario, there are two main situations accounted for, the first being the average exposure, which is considered normal, the other is the worst-case scenario, which takes into consideration the possibility of continuous exposure.[124,137] The simulation is considered for annual exposure, and chronic exposure periods.[154]

The different scenarios are determined in different age groups, namely infants (under a year old), children (between 6-12 years old) and adults (19 -75 years old). It is also wise to consider the different “special groups,” such as pregnant women, the already ill and invalids. Due to the nature of $PM_{2.5}$, the reason for separating the age groups is due to the difference in size, physiology, behaviour and activity levels. Often the inhalation rates of children differ from those of adults.[154] There are lower inhalation rates in infants under the age of 1 year old and an approximated average weight of 11.3 kg, while adults between the ages of 19 to 75 years old have the highest inhalation rates in both the intermediate and chronic exposure phases. The estimated dose was much higher in infants than adults, 12.93 µg/kg/day compared to 3.98µg/kg/day. In children the dose rate was 6.4 µg/kg/day, although not as high as infants, but did amount to almost double the adults’ dose. This indicates that adults

have lower doses than children and infants. There is a further difference when seen that the estimated doses change over season, where in infants, children and adults the higher estimated doses are experienced during the winter period, 20.8 µg/kg/day, 10.3 µg/kg/day, 6.4 µg/kg/day, respectively. The lowest estimated doses are anticipated during the summer period 7.09 µg/kg/day, 3.51 µg/kg/day and.2.81 µg/kg/day. Nonetheless, infants are observed to have higher doses and the doses reduce as the age group increases.

Infants and children have a higher resting metabolic rate and oxygen consumption per unit of body weight than adults because of their rapid growth and relatively larger lung surface area per unit of body weight.[125] Despite adults having larger lungs than infants and children, they have a smaller lung capacity, thus a lower inhalation rate. In addition, the volume of air inhaled by adults is lower than that of infants and children over a similar period. The air passing through the lungs of a resting infant is almost double that of a resting adult.[125,154] Lastly, infants may not be as exposed to the outside so this could reduce their exposure to $PM_{2.5,}$ but in the case of children aged 7 to 12 years old they are the most active outside in the open air, thus this age group has an increased likelihood of exposure.

According to the EPA handbook (US EPA, 2011), exposure is the chemical concentration at the boundary of the body (US EPA, 1992).[125] This is to say that the external dose is not the same as that of the internal dose; often the external dose is higher than the internal dose. This is due to the complex exchange of oxygen and carbon dioxide occurring in the body.[154] Infants and children, rather than adults, are more likely to be affected by 24-hour exposure due to the contact they have with the environment.

5.2.4. RISK CHARACTERISATION

The hazard quotient was less than 1 in both the DEFF exposure limit and the WHO exposure limit, indicating that the probability of an adverse health outcome occurring among healthy and/or sensitive individuals is not high.[126] Despite the potential harm

that infants and children may face, sensitive individuals, such as the ill, pregnant women and invalids, are not at a high risk of developing severe adverse health effects in their conditions. Despite having hazard quotients less than 1, the DEFF hazard quotient was higher than that of the WHO. Although low risks were found there is evidence that a higher exposure limit reduces the hazard quotient of a substance.

The hazard quotient of less than 1 can also be interpreted to state there were no non-carcinogenic risks concluded from this study. However, this study did not investigate the heavy metals of which $PM_{2.5}$ consists. Without that analysis, it cannot be concluded indefinitely that there are no carcinogenic risks.

Although $PM_{2.5}$ levels do not seem to pose and health risks in Pretoria, it is important to consider the levels of chemical components of $PM_{2.5}$.[156] This indicates the components of $PM_{2.5}$ may have carcinogenic and non-carcinogenic effects, and should therefore be investigated.

5.3 STRENGTHS AND LIMITATIONS

The measurements for the exposure assessment were taken using state of the art equipment that were properly calibrated improving the reliability of this study. The $PM_{2.5}$ levels were measured over an entire year, not just for a few weeks or a season. Hence, temporal changes could be observed. The study also measured some chemical components of $PM_{2.5}$ such as BC, UVPM and soot. Few studies in South Africa focus on HHRA on $PM_{2.5}$. Lastly, this study used the WHO's guideline as an REL, not just the more lenient NAAQS.

Limitations to this study were that the comparative data was not complete, namely the meteorological and criteria pollutant data collected by the City of Tshwane. This had a serious effect on the results concerning the true influence that these conditions have on $PM_{2.5}$ levels. The general lack of air pollution data measured by the City of Tshwane led to inconclusive results that cannot be validated, these data are not reliable.

5.4 CONCLUSIONS

In conclusion, thus far the mean $PM_{2.5}$ levels around the study are reasonable, as they did not exceed the South African standard of 40 µg/m^3, however, levels did exceed the WHO guideline of 25 µg/$m^{3.}$ From the study, a significant correlation between the meteorological and $PM_{2.5}$ levels were observed. It was observed that highest average of $PM_{2.5}$ levels were in winter, throughout the study period. No significant correlation was observed between the criteria pollutants measured by the City of Tshwane and the $PM_{2.5}$ levels.

Despite the reasonable $PM_{2.5}$ concentration levels, the HHRA did reveal that infants (0-1 years old) and children (6-12 years old) were the most at risk upon exposure to $PM_{2.5}$; the levels increase the daily doses they are exposed to, compared to adults..

5.5. RECOMMENDATIONS

It is recommended the City of Tshwane Air Quality Managements Plan, which is currently under review, address local and long range sources of $PM_{2.5}$ in the city. Other suggestions include similar studies to be done to monitor the $PM_{2.5}$ concentrations, as well as the influence of the meteorological conditions. Health impact studies can be adapted to explore indoor exposure as well as outdoor exposure to $PM_{2.5.}$ In addition studies that include passive sampling among Tshwane residents should be considered to measure the exposure of $PM_{2.5}$ at that level. Studies must look into the trajectory of the $PM_{2.5}$ within Pretoria, which can be done using the Hybrid Single-Particle Lagrangian Integrated Trajectory Model (HYSPLIT model). This could assist in determining the outside sources of the pollutant. Studies can be done on the filters collected from this study to determine the chemical composition of the $PM_{2.5}$ samples to determine the carcinogenic/non-carcinogenic risk of the metals; this can be done using X-ray Fluorescence Chemical analysis.

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7. APPENDICES

Appendix 1: Measurement calendar 19 April 2018 to 23 April 2019

Date	Collection day	Filter #	Date	Collection day	Filter #	Date	Collection day	Filter #	Date	Collection day	Filter #
2018			21 Jul	154	F185	25 Oct	186 **Dupe**	F223, 224	26 Jan	218	F262
19 Apr	123	F148	24 Jul	155	F186	28 Oct	187	F225	29 Jan	219	F263
22 Apr	124	(F149)	27 Jul	156 **Dupe**	F187,188	31 Oct	188	F226	1 Feb	220	F264
25 Apr	125	(F150)	30 Jul	157	F189	3 Nov	189	F227	4 Feb	221 **Dupe**	F265,266
28 Apr	126 **Dupe**	(F151, 152	2 Aug	158	F190	6 Nov	190	F228	7 Feb	222	F267
1 May	127	(F153)	5 Aug	159	F191	9 Nov	191 **Dupe**	F229, 230	10 Feb	223	F268
4 May	128	(F154)	8 Aug	160	F192	12 Nov	192	F231	13 Feb	224	F269
7 May	129	f155)	11 Aug	161 **Dupe**	F193, 194	15 Nov	193	F232	16 Feb	225	F270
10 May	130	(F156)	14 Aug	162	F195	18 Nov	194	F233	19 Feb	226 **Dupe**	F271,272
13 May	131 **Dupe**	(F157,158)	17 Aug	163	F196	21 Nov	195	F234	22 Feb	227	F273
16 May	132	(F159)	20 Aug	164	F197	24 Nov	196 **Dupe**	F235,236	25 Feb	228	F274
19 May	133	(F160)	23 Aug	165	F198	27 Nov	197	F237	28 Feb	229	F275
22 May	134	(F 161)	26 Aug	166 **Dupe**	F199, 200	30 Nov	198	F238	3 Mar	230	F276
25 May	135	F 162	29 Aug	167	F201	3 Dec	199	F239	6 Mar	231 **Dupe**	F277,278
28 May	136 **Dupe**	F 163, 164	1 Sept	168	F202	6 Dec	200	F240	9 Mar	232	F279
31 May	137	F165	4 Sept	169	F203	9 Dec	201 **Dupe**	F241,242	12 Mar	233	F280
3 Jun	138	F166	7 Sept	170	F204	12 Dec	202	F243	15 Mar	234	F281

6 Jun	139	F167	10 Sept	171 **Dupe**	F205, 206	15 Dec	203	F244	18 Mar	235	F282
9 Jun	140	F168	13 Sept	172	F207	18 Dec	204	F245	21 Mar	236 **Dupe**	F283,284
12 Jun	141 **Dupe**	F169, 170	16 Sept	173	F208	21 Dec	205	F246	24 Mar	237	F285
15 Jun	142	F171	19 Sept	174	F209	24 Dec	206 **Dupe**	F247,248	27 Mar	238	F286
18 Jun	143	F172	22 Sept	175	F210	27 Dec	207	F249	30 Mar	239	F287
21 Jun	144	F173	25 Sept	176 **Dupe**	F211, 212	30 Dec	208	F250	2 Apr	240	F288
24 Jun	145	F174	28 Sept	177	F213	**2019**	209	F251	5 Apr	241 **Dupe**	F289,290
27 Jun	146 **Dupe**	F175, 176	1 Oct	178	F214	2 Jan	210	F252	8 Apr	242	F291
30 Jun	147	F177	4 Oct	179	F215	5 Jan	211**Dupe**	F253,254	11 Apr	243	F292
3 Jul	148	F178	7 Oct	180	F216	8 Jan	212	F255	14 Apr	244	F293
6 Jul	149	F179	10 Oct	181 **Dupe**	F217, 218	11 Jan	213	F256	17 Apr	245	F294
9 July	150	F180	13 Oct	182	F219	14 Jan	214	F257	20 Apr	246 **Dupe**	F295,296
12 Jul	151 **Dupe**	F181,182	16 Oct	183	F220	17 Jan	215	F258	23 April	247	F297
15 Jul	152	F183	19 Oct	184	F221	20 Jan	216 **Dupe**	F259,260			
18 Jul	153	F184	22 Oct	185	F222	23 Jan	217	F261			

*Dupe is the day of duplication

Appendix 2: Ethical Clearance

UNIVERSITEIT VAN PRETORIA
UNIVERSITY OF PRETORIA
YUNIBESITHI YA PRETORIA

Faculty of Health Sciences

The Research Ethics Committee, Faculty Health Sciences, University of Pretoria complies with ICH-GCP guidelines and has US Federal wide Assurance.
- FWA 00002567, Approved dd 22 May 2002 and Expires 03/20/2022.
- IRB 0000 2235 IORG0001762 Approved dd 22/04/2014 and Expires 03/14/2020.

13/09/2018

Approval Certificate
New Application

Ethics Reference No: 507/2018

Title: Human health risks of inhalation exposure to PM2.5 in Pretoria, South Africa

Dear Miss Nandi Sisasenkosi Mwase

The **Amendment** as described in your documents specified in your cover letter dated 27/08/2018 received on 27/08/2018 was approved by the Faculty of Health Sciences Research Ethics Committee on its quorate meeting of 12/09/2018.

Please note the following about your ethics approval:
- Ethics Approval is valid for 1 year
- Please remember to use your protocol number (**507/2018**) on any documents or correspondence with the Research Ethics Committee regarding your research.
- Please note that the Research Ethics Committee may ask further questions, seek additional information, require further modification, or monitor the conduct of your research.

Ethics approval is subject to the following:
- The ethics approval is conditional on the research being conducted as stipulated by the details of all documents submitted to the Committee. In the event that a further need arises to change who the investigators are, the methods or any other aspect, such changes must be submitted as an Amendment for approval by the Committee.

We wish you the best with your research.

Yours sincerely

Dr R Sommers, MBChB; MMed (Int); MPharMed,PhD
Deputy Chairperson of the Faculty of Health Sciences Research Ethics Committee, University of Pretoria

The Faculty of Health Sciences Research Ethics Committee complies with the SA National Act 61 of 2003 as it pertains to health research and the United States Code of Federal Regulations Title 45 and 46. This committee abides by the ethical norms and principles for research, established by the Declaration of Helsinki, the South African Medical Research Council Guidelines as well as the Guidelines for Ethical Research: Principles Structures and Processes, Second Edition 2015 (Department of Health).

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Lefapha la Disaense tša Maphelo

Appendix 3: Daily Reference Exposure Limit for $PM_{2.5}$, South African standard.

Table 12: South African AQI bands for NO_2, SO_2, O_3, PM_{10}, $PM_{2.5}$ and CO.

Air Quality Status	Summary Message	Bands	NO_2 Bands (ppb)	NO_2	SO_2 Bands (ppb)	SO_2	Ozone Bands (ppb)	Ozone	PM10 Bands (ug/m3)	PM10	PM2.5 Bands (ug/m3)	PM2.5	CO	CO Bands (ppb)
Low	Good	1	0-66	0	0-115	0	0-26	0	0-25	0	0-12	0	0	0-10000
		2	67-133	67	116-231	116	27-53	27	26-50	25	13-26	13	10000	10001-20000
		3	133-200	133	232-350	232	54-80	54	51-75	51	27-40	27	20000	20001-30000
Moderate	Moderate	4	201-267	201	351-400	351	81-107	81	76-85	76	41-50	41	30000	30001-35000
		5	268-334	268	401-450	401	108-134	108	86-95	86	51-60	51	35000	35001-40000
High	Unhealthy	6	335-400	335	451-500	451	135-160	135	96-105	96	61-70	61	40000	40001-45000
		7	401-467	401	501-550	501	161-187	161	106-115	106	71-80	71	45000	45001-50000
Very High	Very Unhealthy	8	468-534	468	551-600	551	188-213	188	116-125	116	81-90	81	50000	50001-55000
		9	535-601	535	601-650	601	214-240	214	126-136	126	91-100	91	55000	55001-60000
Hazardous	Hazardous	10	>602	602	>651	651	>241	241	>136	136	>101	101	60000	>60000

Table 13: Health messages to be communicated to the public for the different AQI bands.

AQI	Levels of Health Concern	Accompanying health messages for at-risk individuals*	Accompanying health messages for the general population
Low	Good	**Enjoy** your usual outdoor activities.	**Enjoy** your usual outdoor activities.
Moderate	Moderate	Adults and children with lung problems, and adults with heart problems, **who experience symptoms**, should **consider reducing** strenuous physical activity, particularly outdoors.	**Enjoy** your usual outdoor activities.
High	Unhealthy	Adults and children with lung problems, and adults with heart problems, should **reduce** strenuous physical exertion, particularly outdoors, and particularly if they experience symptoms. People with asthma may find they need to use their reliever inhaler more often. Older people should also **reduce** physical exertion.	Anyone experiencing discomfort such as sore eyes, cough or sore throat should **consider reducing** activity, particularly outdoors.
Very High	Very Unhealthy	Adults and children with lung problems, adults with heart problems, and older people, should **avoid** strenuous physical activity. People with asthma may find they need to use their reliever inhaler more often.	**Reduce** physical exertion, particularly outdoors, especially if you experience symptoms such as cough or sore throat.
Hazardous	Hazardous	Adults and children with lung problems, adults with heart problems, and older people, should **avoid** strenuous physical activity. People with asthma may find they need to use their reliever inhaler more often.	**Reduce** physical exertion, particularly outdoors, especially if you experience symptoms such as cough or sore throat.

Appendix 4: Proof of Proof reader

Gill Smithies

Proofreading & Language Editing Services

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Work Certificate

To	Ms. N.S. Mwase
Address	Faculty of Health Sciences, School of Health Systems and Public Health, University of Pretoria
Date	10/12/2019
Subject	Dissertation: Human health risks of Inhalable exposure to $PM_{2.5}$, Pretoria, South Africa
Ref	NSM/GS/01

I, Gill Smithies, certify that I have edited the following for language, grammar and style,

Dissertation: Human health risks of Inhalable exposure to $PM_{2.5}$, Pretoria, South Africa, by N. S. Mwase, to the standard as required by the University of Pretoria.

Gill Smithies