Draft Thesis Write-Up: Ambient Air Quality and the risk of Acute Myocardial Infarction in Fiji

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# Chapter 1: Introduction

It is estimated that 91% of the words population live in places that exceeds the WHO air quality guidelines with the low- and middle-income countries experiencing the highest burden. Countries in the WHO Western Pacific and South-East Asia regions bear the greatest toll. Often, countries in these regions do not have the benefit of an integrated primary health care activities for early detection and treatment of people with risk factors similar to those in well developed nations.  (WHO, 2018).

In 2016, ambient air pollution was responsible for an estimated 4.2 million deaths worldwide every year of which 16%  of the deaths are from lung cancer, 25% from chronic obstructive pulmonary, 17% from ischaemic heart disease and stroke and 26% from respiratory infection(WHO, 2019). Air pollution in most developing countries originate from sources such as combustion processes from motor vehicles, open burning of wastes and emissions from industrial processes. Most of these emissions contain a heterogeneous mixture of pollutants often referred to as criteria pollutants includes particulate matter (PM), ground-level ozone, carbon monoxide, lead, sulphur dioxide and nitrogen dioxide(USEPA, 2018). These pollutants are of major health concern because of the various cumulative effect it has on the respiratory and cardiovascular health of humans. Principal among these pollutants are particulate matter with an aerodynamic of <10microns (PM10) and particulate matter with an aerodynamic diameter of <2.5microns (PM2.5) which has been reported to cause acute cardiovascular and respiratory morbidity and mortality (Pope & Dockery, 2006).

Impact of air pollution exposure became apparent early in the 20thcentury when mortality and morbidity increased dramatically after episodes  of exposure to high levels of air pollutants during the Meuse Valley Fog in 1930. But it wasn’t until the London Fog episode in 1952 whereby deaths increased sharply from the initial estimate of 3000-4000 deaths to about 12,000 deaths that the health effects of air pollution became too obvious to ignore(Vallero, 2014; BOUBEL, FOX, TURNER, & STERN, 1994). In this episodes it was evident that although the relative increase in morbidity and mortality for cardiovascular disease was lower than pulmonary disease, the absolute number of deaths or hospital admissions attributable to cardiovascular disease presented a larger public health impact.

Cardiovascular diseases (CVDs) are the leading cause of death globally with more people dying from CVDs than from any other cause. The World Health Organization estimates that in 2016, 17.9 million people died from cardiovascular diseases, representing 31% of all global deaths. Eighty five percent (85%) of these deaths are due to heart attack and stroke and three quarters of CVD deaths occur in low- and middle-income countries (WHO, n.d.). Moreover, of the 17 million premature deaths for people under the age of 70 due to non-communicable diseases, 82% were in low- and middle-income countries and 37% of these deaths were caused by cardiovascular diseases.

Air pollution has increasingly been recognized  as a risk factor for cardiovascular disease. Recent epidemiological studies and evidence have consistently shown the increased risk of cardiovascular disease with exposure to air pollutants. In 2004, the American Heart Association (AHA) Scientific Statement concluded that exposure to particulate matter air pollution contributes to cardiovascular morbidity and mortality, especially for myocardial infarction, stroke, cardiac arrhythmia and heart failure(Brook et al., 2004).

## Ambient air quality in Fiji

The Republic of Fiji is one of the developing nations nestled in the south pacific with an estimated population of 884, 167 people. Fifty seven per cent (57%) of these total population live in the urban areas and 43% live in the rural areas (FBoS, 2018).  There is no monitoring of ambient air quality in any city or town in Fiji that would provide an insight of the actual level of pollutants present in its immediate surroundings. However, industrial  and commercial occupational activities that has the potential to emit activities are required to carry out their own air quality monitoring and provide this monitoring data to the Department of Environment in Fiji for verification and recording purposes only.

In 2013, the Department of Environment in Fiji (DoE) collected information on potential air pollution sources through qualitative means. Qualitative data was obtained from public complaints, visual observations and local studies. From this data, the Department of Environment of Fiji established that the main sources of pollution in terms of the volume emitted are from vehicle emissions, burning of dumpsites, backyard burning or burning of household waste, industrial emissions, agricultural burning, emissions from incinerators, cooking in open stoves and dust from unpaved roads.

To date, the only study on air quality in Fiji that has been published was carried out  in 2015 by Isley and others. Their study focused on measuring ultrafine particle concentrations (PNCs) in order to demonstrate combustion emissions. The study by (Isley, Nelson, & Taylor, 2016) on the air quality in Suva found that PM2.5concentrations generally complied with the WHO annual average  of 10 g/m3. However, black carbon(BC) concentrations in the city of Suva were similar to cities in more industrialised countries such as Australia, New Zealand, England and Ireland. Isley and Taylor concluded that the increased vehicular traffic and increasing industrial activities immensely contribute to the particulate pollution in the city. Added to this is the widespread burning of wastes and the emissions from second-hand vehicles of low grade and older diesel vehicles.

## Cardiovascular disease in Fiji

Cardiovascular diseases accounts for 17.7 million deaths each year which is estimated to be 31% of all deaths worldwide. Developing nations like Fiji are experiencing a change in disease patterns with a significant decline in infectious diseases and a corresponding increase in non-communicable diseases such as diabetes, cardiovascular diseases and cancer(TAYLOR, LESWIS, & LEVY, 1989). In Fiji, non-communicable diseases (NCDs) accounts for an estimated 84% of all deaths of which 34 % are attributed to cardiovascular diseases and 22% from diabetes (WHO, 2018). While diabetes related deaths are not the leading cause of death in Fiji, NCDs like diabetes and cardiovascular diseases are a major concern. Cardiovascular diseases  that are of major concern in Fiji includes ischemic heart disease, stroke, hypertensive heart disease, cardiomyopathy and myocarditis and rheumatic heart disease. Of these cardiovascular, diseases the most common in Fiji is ischemic heart disease or coronary heart disease which includes heart conditions such as angina and myocardial infarction(IHME, 2018).

In 2018, the Ministry of Health and Medical Services in Fiji and the World Health Organization established that cardiovascular disease is the leading cause of deaths in Fiji. There are almost twice as many cardiovascular related deaths in Fiji as those from diabetes(PACNEWS, 2018). The major risk factors for cardiovascular diseases in Fiji are smoking, having an unhealthy diet, not physically active and misusing of alcohol. In addition, a person becomes more at risk of cardiovascular disease if they have raised blood pressure, elevated blood sugar and are overweight or obese.

The Ministry of Health and Medical Services, with support from development partners such as WHO is taking a proactive approach to combat NCDs. Early identification of those Fijians who are at risk of developing these diseases is paramount and is one of the main reason that all health facilities in Fiji are offering free screening services for NCDs and their risk factors. NCD screening is also provided in workplaces and communities so that risks are identified for individuals and advice on mitigating measures are given to make changes needed to prevent these life threatening diseases.

## Description of Lautoka City

The city of Lautoka is the second largest of the two cities in Fiji, and is nestled on the western part of Fiji’s main island, Viti Levu (refer to Fig 1). Lautoka is located 24 kilometres north of the town of Nadi where Fiji’s International airport is located. The city lies in the heart of Fiji’s sugar cane growing region and is affectionately known as the ‘Sugar City’ of Fiji. It covers an area of 16 square kilometres and is home to Fiji’s largest sugar mill. The city has an estimated population of 52,500 people from various social and ethnic backgrounds. The ethnic distribution of the population consists of indigenous Fijians which accounts for 43% of the population, Fijians of Indian descent accounts for 45% and people from other ethnic background makeup the remaining 7% of the population.



Shows the four divisions in Fiji and Lautoka City on the island of Viti Levu. Source:

As the second largest city, Lautoka harbours most industrial and commercial enterprises in the Western part of Fiji’s main land. This include timber mills, breweries, oil/ghee refining factory. aerated water and juice factory, concrete industries, soap factoy, engineering and steel workshops, flour mill, bakeries, etc.

The weather in Lautoka is similar to that of in Nadi as both are within close proximity. The rainy season is usually between the months of December to April and precipitation is quite low as compared to the capital of Fiji, Suva which is located in the central division. The average rainfall in a year is estimated at 2,050mm or 80.5 inches per year and during the dry season ( May to October), rainfall can be as low as 100mm or 4 inches per month. The temperature on western part of Viti Levu is a little higher then other locations in Fiji with a mean annual temperature of 280C.  The amount of sunshine too is higher in Lautoka which enjoys 7-8hrs of sunlight per day as compared to Suva which has an average of 5-6 hours of sunlight.

## Site Identification and description

Air quality data was collected within the Central Business District (CBD) of Lautoka City. Consent was granted by the Chief Executive Officer of the Lautoka City Council, Mr. Jone Nakauvadra and permission was also given to use the Council’s resources in setting up the air quality monitoring sensors (AQMS). A senior Environmental Health Officer by the name of Mr. Wally Atalifo was designated to guide me because of his knowledge of the city and to locate the best possible locations to mount the monitoring sensors. The sites that were chosen had to gather relevant data with with particular reference to my research objectives and research question. Three sites were initially selected within the CBD area to allow collection of ambient air from three separate but distinct locations. However, only two AQMS was used as the third one developed problems prior to being used.

The two sites are generally high volume used roads by all vehicles, workers and pedestrians. Site 1 as shown in Fig. 2 was chosen because of the volume of traffic and usage of this road by daily commuters into the city. This air quality monitoring site is at the juncture of Vitogo Parade (highlighted in yellow in Fig 2) and Narara parade . The traffic management plans for this city does not allow buses to use this section of the city in order to streamline the traffic flow within the city. Adjacent to this site are major clothing retail stores and on the other side is a public park for community recreational activities. A railway runs parallel to the Vitogo parade and is used to cart sugar cane from farms located in the outskirts of Lautoka City and from Nadi town. Site 2 is located  at the junction of Tukani Street and Vakabale Street. Traffic volume along this site is always high especially for buses and heavy trucks. Adjacent to Site 2 are restaurants and retail stores as well as the main bus station for the city. The public market is about 30m from this monitoring site and the density of people around this area on a daily basis is always high.



Shows the boundaries of the Lautoka City CBD. Source: Google Maps

## Rationale for research

Globally, a lot of research has been conducted and focused on urban population in developed countries and where issues relating to air pollution is readily apparent. Ambient air quality in developing countries has always been of concern because of the continuous increase in urban development, increasing population and the continuing reliance on fossil fuels as the main source of energy. In addition, most of the populace in developing counties live in the rural areas whereby households still rely on unprocessed biomass fuels in the form of wood and crop residues. The gaseous pollutants released from the combustion of these energy sources have varying and significant effects on human health and one that is of importance to this study is the effect it has on cardiovascular health. As a developing country, the most commonly known or traditional risk factors to cardiovascular diseases in Fiji are being overweight or obese, smoking, high blood pressure and high cholesterol levels, being physically inactive, having an unhealthy diet, diabetes and prediabetes and having a family history of early heart disease.

My area of specialisation in Fiji is Environmental Health, hence a systematic investigation of the association between an environmental variable (pollutants in air) and a health effect (heart disease) will provide the nous amongst health researchers in Fiji on the influence of environmental factors on human health. Air pollution is one of the largest risk factors that lead to a range of diseases.  Heart disease is a common cause of death and disability worldwide. In Fiji alone, 34% of annual deaths is from cardiovascular diseases(WHO, 2018).  Being from Fiji and an ardent advocate on impacts of environmental  exposures on human health, this research will provide the impetus in developing explanatory models of the linkage between air quality and risk of heart disease in the Fijian population.

This research is unique in the sense that it will be one of the first studies to quantitatively examine the exposure to air pollutants in an urban area in Fiji and risk of hospitalization due to acute myocardial infarction. The study will put into perspective the growing epidemiological evidence on pollutant levels and risk of hospitalizations from acute myocardial infarction for the city of Lautoka. Investigating the outcome of this study in order to develop policies and plans for ambient air quality is not within the scope of this thesis. This research is purely to examine the ambient air quality and risk of acute myocardial infarction in Lautoka.

## Research aims

The general aim of this research is to determine  if exposure to increased levels of criteria pollutants )particulate matter with an aerodynamic diameter of <2.5microns and <10 microns, carbon monoxide, sulphur dioxide, nitrogen dioxide and ground-level ozone) will lead to increased rate of emergency room hospitalizations due to heart disease ( also referred to as acute myocardial infarction in this thesis). To achieve this, three principal objectives were developed to be the focus of this research and are as follows:

1. Collecting data on the ambient air quality in the Lautoka Central Business District for a period of three months
2. Determine the ambient air quality in the Lautoka CBD and its potential health implications
3. Conducting a meta-analysis of published studies on the association between ambient air quality and risk of hospitalization due to myocardial infarction.
4. Lastly, on the basis of the meta-analysis and air quality data collected, I will develop predictive models on the risk of hospitalization due to acute myocardial infarction for the city of Lautoka.

## Thesis outline

The layout and content of this thesis is generally constructed to satisfy the aims of this research. Chapter One has introduced the research topic and the rationale for conducting the study. The general aims of the research and the study site has also been introduced thoroughly in this chapter. In addition,  a brief on the ambient air quality in Fiji and the outlook of cardiovascular health was also presented. Chapter two provides the literature review of what is known about the research topic. It examines the history of air pollution and the associated cardiovascular effects. In particular the health outcome of interest, acute myocardial infarction. Also provided here are the air pollution issues in Fiji and the drawbacks that the country has in terms of measuring and addressing air quality.  Chapter 3 introduces the study design adopted for this research and the methods selected to obtain the necessary quantitative measurements for this research. In particular, this part emphasizes the analysis methods used to provide precise pooled estimates in the meta-analysis. Chapter four provides the qualitative and quantitative output of all the approaches and procedures described  in chapter 3.  In this chapter, the exposure and outcome variables are combined and analysed to determine the relationship between them. Chapter five

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# Chapter 2: Literature Review

## Introduction

The objective of this chapter was to conduct an assessment on the body of research relating to air pollution and the cardiovascular effects.  The chapter commences with a brief history on the evolution of air pollution before the industrial revolution to the current air pollution problems and challenges. In addition to this is that it elaborates on how the advancement of technology has in a way mitigated the effects of air pollution and emission of pollutants in developed countries but not so much in developing countries. Also presented in this chapter are the cardiovascular effects of air pollution and how it has impacted the cardiovascular health of the population in certain countries described herein.

## History of Air Pollution

### Before the Industrial Revolution

Air pollution can be traced back to the tribes in early history whereby they lived nomadic lives to avoid the stench of decaying animals as well as foul odour from vegetable and human waste. These early tribesmen also learned to use fire and this new knowledge was quite important in their daily lives. The usage of fire would fill the inside of their living homes with the products of incomplete combustion and examples of this can still be seen in some primitive parts of the world (VALLERO, 2008). Later on, the invention of chimneys removed the combustion materials and cooking odours from the liveable spaces.

In the bronze and iron ages, industries associated with the production of  air pollution such as dust and fumes were from metallurgy, ceramics and preservation of animal products. These industries were responsible for the production of clay and bricks before 4000BC. The production and use of iron came into effect around 1000BC. During this period people relied on the use of charcoal as a source of fuel rather than coal or coke (BOUBEL, FOX, TURNER, & STERN, 1994). The burning of wood in fireplaces inside homes created emissions that were smoky and in AD 61 Roman philosopher Seneca said that,

*“ As soon as I had gotten out of the heavy air of Rome and from the stink of the smoky chimneys thereof, which, being stirred, poured forth whatever pestilential vapours and soot they had enclosed in them, I felt an altercation of my disposition”*

Later on in 1157, the wife of king Henry II of England, Eleanor of Aquitaine, decide to move away from Tutbury Castle because she cannot endure the burning of wood.  One hundred and sixty  years later the burning of coal was prohibited in London and in 1306 Edward I issued a Royal proclamation authorizing the use of sea coal in furnaces. By 1661, air pollution in London was a huge problem and it prompted John Evelyn to advise the parliament and King Charles II on the quality of air in London with possible remedies to control air pollution. These propose remedies are still relevant now in the 21st century(P.J.B., 1956).

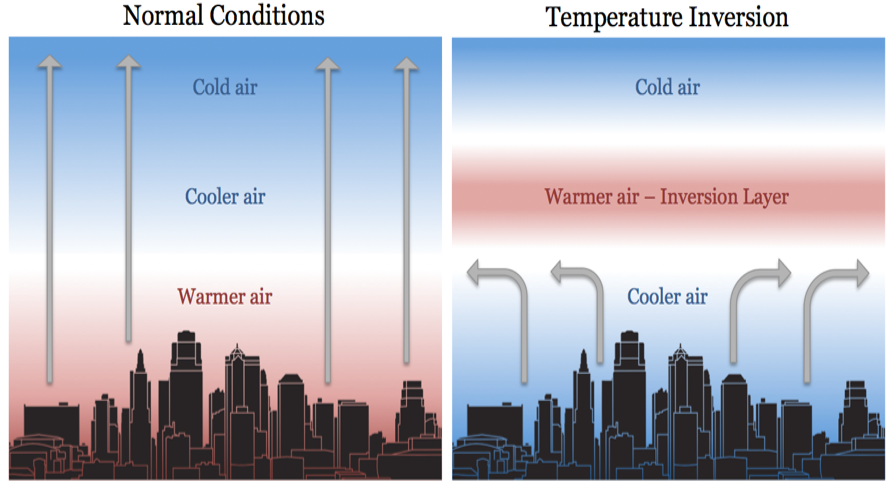
### The Industrial Revolution

The Western civilization embraced the industrial revolution because it brought prosperity, social changes and altered the direction of their history. Its’ downside was the over reliance of industries on coal as an energy source and the severe air pollution that it caused(Air pollution and health 1999). Harnessing of steam to move machinery and pump water began in the early years of the 18th century and culminated in 1784 with Watt’s reciprocating engine that was later replaced with the steam turbine in the twentieth century(*Fundamentals of Air Pollution*, 1973) .

A consequence of this era was a lot of pollution from the smoke and ash from the burning of coal in the boiler furnaces of stationary power plants, locomotives, home heating fireplaces and furnaces. In 1819, Great Britain took the initial steps to address the problem brought about by air pollution and in 1856 saw the creation of laws specifically for London. These laws were introduced to reduce the imminent threat of air pollution and deemed the emission of smoke as a public health nuisance (Beaver, 1955). In 1880, the United States of America developed municipal ordinances and regulations targeting the emission of black smoke and ash from industrial, marine and locomotive sources. Despite the introduction of new laws to curb the emission of smoke and ash in major industrialized countries, air pollution was at its worst towards the end of the 19th century. The principal engineering technological advancement in the control of air pollution were the stoker for mechanical firing of coal, scrubber for removing acid gases from effluent gas steams, cyclone and bag house dust collectors, and the introduction of physical and chemical principles into process design(BOUBEL, FOX, TURNER, & STERN, 1994).

### The 20th Century

In the early 1900s, there was still a reliance on the use of coal but technological advances such as the replacement of the steam engine with the electric motor was a major breakthrough. Towards the end of the 1stquarter of this century, the use of coal was being replaced by pulverized coal, oil and gas. But each of these new energy sources produce their own characteristic emissions to the atmosphere. In this period there was a decrease in ash emissions as oil had replaced the use of coal resulting in the significant increase in automobile production. The period 1925 to 1950 saw the emergence of the present day pollution problems such as the Meuse Valley Smog in 1930 whereby a combination of air pollution and climatic conditions killed sixty people (Firket, 1936). In 1948, an air inversion (an air inversion is an event in which air stops circulating and is trapped close to the ground. The combination of trapped toxic gases and early morning mists yields disastrous effects) episode similar to that shown in Fig 2.0 known as the Donora Smog in Donora, Pennsylvania killed almost 40 people and left nearly half of the town’s 14,000 residents with severe respiratory and cardiovascular problems (USEPA, 2017).  The city of Los Angeles also experienced the effects of smog in the 1940s as a result in the influx of cars and industries combined with a geography that traps fumes.



The picture on the left shows air flow in normal conditions and on the right show air flow during a temperature inversion. Source: Cotton NC State University

Towards the end of the first half of this century, major technological advancement and changes were made in major industrial countries like the United States and Great Britain. A good example was in Pittsburgh, Pennsylvania whereby the use of coal and oil as home heating fuel was substituted with the use of natural gas. In addition to this was the displacement of steam locomotives and replaced with diesel locomotives. Despite the growing problem of air pollution during the period 1925-1950, no country in the world adopted any pollution laws to try and mitigate harmful emissions. It was the state of California that enacted the first state air pollution law in the United States.

The period 1950-1980 was marked with a major pollution disaster in Great Britain which was later known as the ‘Great London Smog’.  During this period coal was the main source for generating power and heating homes in London. It was on the 5th of December 1952 that a high pressure weather system stalled over southern London and caused a temperature inversion. For five days,  a heavy fog combined with smoke from the fumes of vehicle exhaust and power plants created a public health disaster which resulted in an estimated of 4,000 premature deaths (Klein, 2012). The impact of the London Smog provided the impetus for the regulating of emissions with the enacting of the Clean Air Act of 1956. The new law required the markets to provide cleaner fuels for home owners , phasing out of coal in urban areas, power stations closed to residential areas were closed and height of chimneys were regulated. As a result of high air pollution levels, almost all European countries including Australia,New Zealand and Japan have created legislation to control air pollution. In the United States, federal laws were introduced in 1955 to support research in air pollution, training and technical assistance *(Fundamentals of Air pollution 1984).*Later in 1970, the United States Environment Protection Agency (USEPA) came into existence with the federal mandate of protecting the environment and human health. The second half of the twentieth century  also marked the increase in air pollution from automobiles, pollution from flue gases and emissions from combustion *(Vallero 2008).*In the 1970s, the buildup of greenhouse gases was a major concern as it was found to be contributing to the depletion of the stratosphere ozone layer. Also, the discovery of the Antarctic ‘ozone hole’ in 1985 and the emergence of uncontrolled changes in climate in 1990s saw the emergence of the major problem brought about by greenhouse gases such as chlorofluorocarbon (CFCs)(Elsom, 1992). Towards the end of this period, air quality monitoring systems and modelling of atmospheric processes came of age and highlighted the global concern of air pollution. Subsequently, in the early years of the 21stcentury,  global air pollution has taken on a greater urgency amongst the scientific community and the general public.

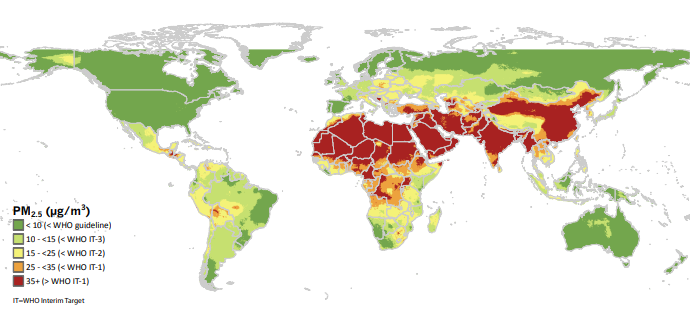
## Recent Ambient Air Pollution

Air pollution in the industrialized and developing world has changed drastically from the last century as a consequence of the rapid global population growth (Fenger, 2009). Economic development, energy consumption, urbanization, transportation and motorization are other driving forces of air pollution in urban cities(Chen & Kan, 2008). Nevertheless , the urban environment in most industrialized countries has improved from pollution caused by  power and heat generation(Fenger, 2009). In the 2005 Air Quality Guidelines Global update, the World Health Organization summarized that the annual average concentrations of PM10in European and North American cities were generally lower than 50g/m3as shown in Table 1. Asia, (“Air Pollution Regulatory Framework”, 2008)Africa and Latin America had the highest levels of PM10compared to other regions and SO2has substantially declined in the United States of America and Europe. Decline of SO2levels in Bangkok, Jakarta and New Delhi is attributed to the use of fuel with low sulfur content . Latin America and Africa also recorder moderate reduction in SO2levels (Chen & Kan, 2008).

Table 1: Ranges of annual average concentrations of PM10, NO2, SO2and 1 hr average maximum of ozone for different regions. *Source: WHO*

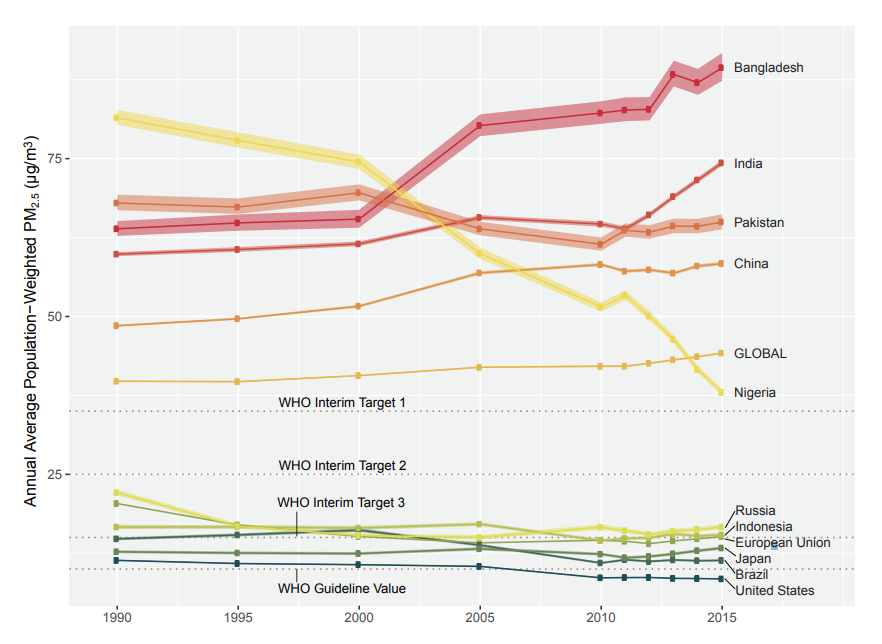
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|  |  |  |  |  | Region | Annual average concentration |  |  | Ozone (1hr max concentrations) |
|  |  |  |  |  |  | PM**10** | NO**2** | SO**2** |  |
|  |  |  |  |  | Africa | 40-150 | 35-65 | 10-100 | 120-300 |
|  |  |  |  |  | Asia | 35-220 | 20-75 | 6-65 | 100-250 |
|  |  |  |  |  | Australia/New Zealand | 28-127 | 11-28 | 3-17 | 120-310 |
|  |  |  |  |  | Canada/ United States | 20-60 | 35-70 | 9-35 | 150-380 |
|  |  |  |  |  | Europe | 20-70 | 18-57 | 8-36 | 150-350 |
|  |  |  |  |  | Latin America | 30-129 | 30-82 | 40-70 | 200-600 |

In contrast, (Chen & Kan, 2008) suggests that countries in transition have shown that traffic related air pollutants such as NO2and SO2tend to increase due to increasing number of motor vehicles. Mega cities such as Beijing, Tokyo, Osaka, New York, Los Angeles and Sao Paulo have recorded NO2levels that exceed the WHO criteria of 40g/m3. The recent report by WHO on the State of Global Air/2017 estimates that 90% of the world’s population reside in areas with unhealthy airand 50% live in areas where the PM2.5 concentrations were above the WHO interim target 1 (IT-1) of 35g/m3. Moreover, 64% of the world’s population reside in locations exceeding the interim target (IT-2) of 25g/m3. Countries such as China, India, Bangladesh and Pakistan are have recorded extreme PM2.5concentrations of above 75g/m3.(Health\_Effects\_Institute, 2017). The sources of these extreme PM2.5concentrations are from multiple sources of combustion emissions including household solid fuel use, coal-fired power plants agricultural and other open burning, transportation and industrial sources. In addition, this report indicates that the lowest annual average population-weighted PM2.5( 8g/m3)were recorded in Finland, Australia, New Zealand, Canada, Brunei, Sweden, Greenland and several Caribbean and Pacific Island countries.



Shows the comparison of the global annual average PM2.5concentrations in 2015 with WHO Air QUality guidelines. *Source: Health Effects Institute*

Furthermore, the report on the State of Global Air/2017 suggests that global weighted PM2.5concentrations has increased by 11.2% from 39.7g/m3in 1990 to 44.2g/m3in 2015. The rapid increase in the      PM2.5 concentrations was significant from the year 2010 onwards as shown in Figure 4. These increases reflects the changes in air pollution levels in some of the most populous countries in the world such as China, Bangladesh and those in the European region. The highest increase from 2010 to 2015 was experienced by Bangladesh and India whereas the United States of America, Brazil, Russia, Indonesia and countries in the European have seen a slight decrease in PM2.5 levels since 1990.



Shows the annual average population weighted PM2.5concentrations in the 10 most populous countries including the European Union. *Source: State of Global Air/2017*

he State of Global Air 2017 report further adds that the ozone levels have increased by 7% globally from 1990 to 2015. This is due to a combination of factors and principal among them is the increase in emissions of ozone precursors such as NO2combined with the warmer temperatures in developing countries such as Pakistan, Brazil and Bangladesh. The United States and the European Union have noted a decrease in ozone and PM2.5concentration levels by 5% and 2% respectively. The decrease in concentration levels in the USA and EU is due to the air quality management programs that has been in place in these countries since 1990.

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## Air Pollution in Developing Countries

Developing countries face particularly difficult choices  in balancing efforts to protect the environment with efforts to spur economic growth (Alberini et al., 1997). As a consequence, rapid urbanisation and increased migration to urban areas often lead to an influx in traffic and industrial activities. With the increase in traffic and industrial activities, emission of gaseous pollutants are often uncontrolled and contribute significantly to ambient air pollution in the urban areas(Mannucci & Franchini, 2017). Because of economic and other societal reasons, developing nations like Fiji often take a passive role in regulating ambient air quality in its urban areas.

The World Health Organization estimates that in 2016, 4.2 million premature deaths was caused by ambient air pollution in both cities and rural areas. From these deaths, 91 percent occurred in low-and middle-income countries and the greatest burden was in the WHO South-East Asia and Western Pacific regions (WHO, 2018). Modernization has seen a shift from the use of biomass fuel to petroleum products and electricity in developed countries. However, households continue to use biomass fuels and poverty is seen as one of the main barriers to the adoption and use of cleaner fuel sources(Gordon et al., 2014).

Indoor air pollution is a major public health threat and about 50% of the people in developing countries rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. The slow rate of development in most of these developing countries suggests that biomass fuels will continue to be used by the poor in years to come(Nigel Bruce, 2000). Majority of households in developing countries still use earth ovens and stoves whereby incomplete combustion results in substantial emission of pollutants. These cooking practices are inefficient, and use fuels and technologies that produce high levels of household air pollution with a range of health-damaging pollutants, including small soot particles that has the ability to penetrate deep into the lungs. In poor ventilated houses, indoor smoke can be 100 times higher than the acceptable levels for fine particles. Exposure is particularly high among women and young children, who spend most of their time near the domestic hearth(WHO, 2018). In these countries, indoor concentration of particles usually exceed the guideline levels by a large margin(Smith, Apte, Yuqing, Wongsekiarttirat, & Kulkarni, 1994). These high concentrations of particulate matter and carbon monoxide have shown to be associated with health effects that may occur among the children such as reduced lung function and exacerbation of chronic obstructive pulmonary diseases. This is in stark contrast to developed nations whereby the use of gas stoves has evolved into adopting stoves with cleaner energy sources like electricity

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## Characteristics of common Air Pollutants

The air we breathe can contain a variety of pollutants emitted into the atmosphere outdoors (also called ambient air) as well as into the air indoors. Air pollution can contain a mixture of solid particles, liquid droplets and gases from a variety of sources such as industry, motor vehicles, heating appliances, and tobacco smoke (NSW-Health, 2013). The World Health Organization has established that the pollutants with the strongest evidence of health effects are particulate matter are particulate matter (PM2.5& PM10), ground-level ozone (O3), nitrogen dioxide (NO2) and sulphur dioxide (SO2) and that adverse health consequences can occur as a consequence of short/long-term exposure to them(WHO, 2018). Due to their occurrence in the atmosphere, these pollutants are classified as indicator pollutants for fuel combustion and traffic related air-pollution(Chen & Kan, 2008). In the United States of America, the USEPA has categorized these four pollutants and two others, namely carbon monoxide (CO) and lead (Pb) as criteria pollutants. This is because these pollutants are commonly found all throughout the United States of America and has the tendency to cause harm to human health and the environment.

To satisfy the aims of this study, these pollutants were measured during the 3 months period in Fiji. Understanding their occurrence and sources (as outlined below) is imperative in addressing air pollution in Fiji.

### Particulate Matter (PM10 & PM2.5)

Often called particulates, constitutes a major class of pollutants. It comes in a variety of shapes and sizes and can be either liquid droplets or dry dusts, with a wide range of physical and chemical properties (C. David Cooper, 2011). Primary particles are emitted  directly into the atmosphere, such as diesel soot, whereas secondary particles are created through physiocochemical transformation of gases such as nitrate and sulphate formation form gaseous nitric acid and sulphur dioxide respectively. The numerous natural and anthropogenic sources of particulate matter include motor vehicle emissions, tire fragmentation and resuspension of road dust, power generation and other industrial combustion, smelting and other metal processing (Brook et al., 2004). Other sources are from agriculture, construction and demolition activities, residential wood burning, windblown soil, pollens and moulds, forest fires and combustion of agricultural debris, volcanic emissions and sea spray. Although there are thousands of chemicals that have been detected in particulate matter in different locations, some of the more common constituents include nitrates, sulphates, elemental and organic carbon, organic compounds (e.g. polycyclic aromatic hydrocarbons), biological compounds (e.g. endotoxin, cell fragments), and a variety of metals (e.g. iron copper, nickel, zinc and vanadium.

Because of the complex nature of particulate matter, its has been measured and regulated based primarily on mass within different size ranges. There was a shift in regulatory focus in 1987 from total suspended particles to particles that could readily penetrate and deposit in the tracheobronchial tree, or PM10(particulate matter with a median aerodynamic diameter of <10m). The USEPA regulated 24-hour and annual average standards for PM2.5(particulate matter with median aerodynamic diameter <2.5m) in 1997, comprising the size fraction that can reach the small airways and alveoli. The function of the alveoli is to exchange oxygen and carbon dioxide molecules to and from the blood stream. PM2.5 originates mostly from combustion sources and includes primary and secondary particles, whereas the coarse fraction derives mostly from natural sources such as crustal material (including windblown soil) and grinding processes (Brook et al., 2004). Important bioaerosols (e.g. endotoxin, pollen grains and fungal spores) are found mostly in the coarse fraction (and larger particles), although both endotoxin (an essential component of the cell wall of gram-negative bacteria) and the antigenic protein content of pollen grains can also adsorb onto the surface of fine particulate matter. Mostly, larger particles demonstrate a greater fractional deposition in the extrathoracic and upper tracheobronchial regions. whereas smaller particles (e.g. PM2.5) show greater deposition deep in the lungs. Although PM2.5 generally behaves as a regional pollutant, there can be considerable small-scale partial variability due to point source emissions (e.g. a smelter) or features such as street canyons in large cities . In addition, prevailing wind patterns can affect human exposures.

More recently, considerable research attention has been devoted to ultra fine particles (UFPs) < 0.1m in diameter, which results from combustion processes. UFPs tend to be short-lived, because they cluster and combine into larger particles. However, they demonstrate very high deposition in the alveoli, account for a major portion of the actual particles within particulate matter and have a high surface area to mass ratio. Such characteristics potentially  lead to enhanced biological toxicity. UFPs may even be able to pass directly into the circulatory system, which would allow them to be disseminated systemically (EPA, n.d.).

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### Nitrogen Oxides

Nitrogen oxides are reactive substances commonly understood to include nitric oxide (NO), nitrogen dioxide (NO2) nitrogen trioxide, nitrogen tetroxide (N2O4) nitrogen pentoxide (N2O5). These compounds are referred to collectively as “NOx”. Gaseous nitric acid (HNO3), a major source of particulate nitrate, is formed when NO2 reacts with hydroxyl radicals during the day and when N2O5 reacts with water vapour at night (USEPA, 1993). Other members of the larger family of nitrogen oxids include nitrous acid, nitrous oxide, peroxy=acetyl nitrate (responsible for some of the irritant effects of photo chemical smog), nitrites, nitroso compounds and other nitrogen containing acids. Most toxicological and epidemiological research has focused on NO2  because of the fact that NO2 is one of the regulated air pollutants for which standards are available worldwide. Also, NO from vehicular exhaust and power plant is largely converted to NO2,and NO2plays a primary role in the formation of tropospheric ozone (O3). The main anthropogenic source of NOxin ambient air is fossil fuel combustion in motor vehicles and industrial processes, particularly in power generation.  High temperature combustion results in the oxidation of atmospheric N2,first to NO and then to NO2(Brook et al., 2004). Motor vehicle emissions near busy streets can result in high local NOx concentrations. The typical daily NOx pattern consists of a low background concentration, with morning and late afternoon peaks resulting from rush-hour traffics. Nitrogen in fossil fuels such as coal can be oxidized to NO2 under oxygen-rich combustion conditions. NO2and NO ae both formed naturally as a result of bacterial metabolism of nitrogenous compounds and, to a lesser extent, from fires, volcanoes and fixation by lightning. The generation of tropospheric ozone and other photochemical oxidants is initiated with photolysis of NO2, whereas NO acts as an ozone scavenger(Lipsett, 2001).

Significant human exposure can also occur in nonoccupational indoor settings(Spengler et al., 1994). Unvented furnaces and stoves are the primary sources of indoor NOxalthough kerosene space heaters and cigarette smoke may also play a role(LEADERER, 1982). In urban areas, infiltration of ambient NO2 from vehicular emissions may also influence indoor exposures(Spengler et al., 1994).

### Carbon Monoxide

Carbon monoxide (CO) is a nearly ubiqutous product of incomplete combustion of carbon-containing fuels. Common outdoor sources include motor vehicles, engines on motorboats, lawnmowers, chain saws and other devices that require fossil fuel combustion; residential wood-burning, improperly adjusted gas-burning and oil appliances; coal combustion and tobacco smoking(Hampson, 1992).  In urban areas, the contributions of diesel and stationary source combustion are relatively small in comparison to gasoline powered engines(Harrison, 1999).

Carbon monoxide is an odourless, colourless and tasteless gas that binds to hemoglobin with an affinity 250 times that of oxygen, thereby interfering with the systemic delivery of oxygen to tissues. In addition, binding of carbon monoxide to hemoglobin causes causes an allosteric change in the conformattion of the oxyhemoglobin complex that increases the oxygen affinity of the remaining binding sites and interferes with the release of O2 at the tissue level(Brook et al., 2004). In the United States, the current ambient carbon monoxide concentrations suggest that in most circumstances, this pollutant serves more as an indicator of combustion-related pollution than as a direct toxicant. However, in some situations (e.g. insufficiently ventilated parking structures), carbon monoxide could attain concentrations sufficient to lead to physiologically meaningful increases in carboxyhemoglobin in persons with significant atherosclerotic disease or other cardiac conditions

### Sulphur Dioxide

Sulphur dioxide (SO2) is a highly irritating, colourless, soluble gas with a pungent odour and taste. When it comes into contact with water it forms suphurous acid, which accounts for its strong irritant effect on eyes, mucous membranes and skin. Sulphur dioxide is efficiently scrubbed from inhaled air in the upper airway. In the absence of anthropogenic activities, concentrations of ambient SO2 are very low, in the range of 1 ppb. In ambient air, the primary sources of SO2 include combustion of sulphur-containing fuels, especially in power plants and diesel engines (prior to the reformulation of diesel fuels) and roasting of metal sulphide ores. Sulphur dioxide is oxidized to sulphur trioxide, which, because of its strong affinity for water, can be rapidly hydrated to form sulphuric acid(WHO, 1987). Increased levels of sulphur dioxide have been associated with widespread illnesses in several 20thcentury air pollution catastrophes; however, much of the morbidity and mortality in these episodes may have been due to its role in the formation of particulate sulphates. In nonoccupational settings, SO2 is generally found at substantially lower concentrations indoors than outside; however, the use of kerosene space heaters can generate significant indoor concentrations(LEADERER, 1982).

### Ozone

Ozone (O3) is not emitted directly into the atmosphere and is formed by natural processes and by human activities. Is composed of three oxygen molecules joined together with the two basic oxygen molecule (O2)and and an additional third atom which makers ozone an unstable highly reactive gas. It is created  when emissions from vehicles and industries containing oxides of nitrogen and volatile organic compounds chemically react in the presence of sunlight (“Ground-level Ozone Basics | US EPA”, n.d.). It is a colourless-to-bluish gas with a characteristic odour associated with electrical discharges. Low level exposure is ubiquitous, because it is more readily formed during the summer months and is usually at the highest concentrations in the afternoon or early evening. Ozone is formed in the stratosphere by the action of solar radiation on molecular oxygen (O2). Because stratospheric O3 prevents high-energy UV radiation from penetrating the atmosphere, many terrestrial forms would be unable to survive without this O3layer.

Ozone has been recognized since the 1950s as the principal component of photochemical smog. In the troposphere, it is also known as ground-level ozone and is formed by the action of solar UV radiation on nitrogen oxides and reactive hydrocardons, both of which are emitted by motor, vehicles power plants, industrial boilers, refineries, chemical plants and pther industrial sources. The reaction sequence involves photolysis of nitrogen dioxide (NO2) to NO and oxygen atoms. Under desirable conditions, there is little accumulation of O3. However, volatile organic compounds (VOCs) can facilitate the oxidation of NO to NO2by alternative mechanisms. These reactions reduce NO scavenging, which allows O3concentrations to increase(Brook et al., 2004).

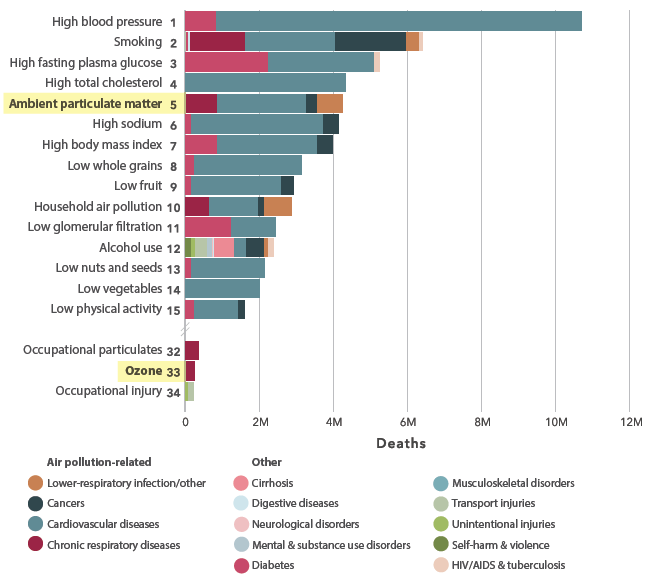
Ozone is most likely to reach unhealthy levels on hot sunny days in urban environments, but can still reach high levels during colder months. Ozone can also be transported long distances by wind, so even rural areas can experience high ozone levels. The typical formation of ground-level ozone in populated areas is characterized by a broad peak that lasts from the late morning until the late afternoon or early evening (USEPA, 2017). However, large-scale transport may result in elevated ozone concentrations that extend over into remote areas  far removed from the primary sources. Wind speed and direction, temperature inversion, addition of other O3 precursors and other factors affect the temporal O3 patterns downwind, so that peak concentrations may occur anytime from noon until  late in the evening (Lioy & Raymond V., 1989). Human activities are major sources of O3precursors, although there are also natural sources of ozone precursors which includes the intrusions on stratospheric O3. the action of lightning on molecular oxygen. Chemical reactions involving naturally occurring nitrogen oxides and other volatile organic compounds(USEPA, 1996).

## Global Burden of  Ambient air pollution

The Global Burden of Diseases, Injuries and Risk Factors study in 2015 (GBD 2015) estimated the burden of disease attributable to seventy-nine risk factors in 195 countries from 1990 to 2015. The study identified air pollution as a leading cause of global disease burden, especially in low-income and middle-income countries. Ambient air pollution in particular particulate matter with an aerodynamic  diameter of <2.5m was identified as a leading risk factor for global disease burden with an estimated 2.9 million attributable deaths in 2013. An additional 217,000 deaths were attributable to long-term ozone exposure(Collaborators, 2017).

According to (Cohen et al., 2017), ambient air pollution contributes substantially to the global burden of disease in 2015. This burden of disease has increased for the past 25 years (1990-2015) due to population ageing, increasing non-communicable disease rates and the increasing air pollution in low-income and middle-income countries. In addition, the global exposure to air pollution and its disease burden as shown in Fig 6 illustrates that the air pollutant PM2.5 was the fifth highest ranking risk factor for death in 2015. Particulate matter with an aerodynamic diameter of <2.5m was responsible for 4.2 million deaths from heart disease and stroke, lung cancers and respiratory illnesses. Ground level ozone was ranked as the 33rdrisk factor causing deaths and was responsible for an additional 254,000 deaths.

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Shows the global ranking of risk factors for total deaths from all causes for all ages and sexes in 2015. Source: State of Global Air-2017

Moreover, recent reviews by the USEPA and WHO have shown that the long-term exposure to ambient air pollution is responsible for increased mortality and morbidity from respiratory and cardiovascular diseases, lung cancer and shortens life expectancy.

## Health Impacts of Ambient Air Pollution

Ambient air pollution is a major cause of death and disease globally. The health effects range from increased hospital admissions and emergency room visits, increased mortality, to increased risk of premature death(Brunekreef & Holgate, 2002). Premature deaths linked to ambient air pollution  are mainly from heart disease, stroke, chronic pulmonary disease, lung cancer and acute respiratory illnesses among children. The World Health Organization estimates that world wide ambient air pollution accounts for

* 29% of all deaths and diseases from lung cancer
* 17% of all deaths and disease from acute lower respiratory infection
* 24% of all deaths from stroke
* 25% of all deaths and disease from ischaemic heart disease
* 43% of all deaths and disease from chronic obstructive pulmonary disease.

Pollutants of interest and with the strongest evidence  for public health concern include particulates  with an aerodynamic diameter <10m (PM10) and particulates with an aerodynamic diameter <2.5m (PM2.5), ozone (O3) sulphur dioxide (SO2) and nitrogen dioxide (NO2).

The health risks associated with particulate matter is well documented in terms of mortality and cardiovascular and respiratory effects. It is also used widely as an indicator to assess the health effects of exposure to ambient air pollution. Because of its small size PM10 can penetrate and be deposited in the upper respiratory tract. On the other hand, PM2.5 can penetrate further deep into the gas exchange region of the lungs (alveoli) and cause respiratory and cardiovascular impacts (Kampa & Castanas, 2008). Because of its toxicity and ability to penetrate deep into the respiratory system, particulate matter has been classified as a cause of lung cancer by the WHO’s International Research Agency for Research on Cancer (IARC).

The relationship between particulate matter and lung cancer was also observed in the study by (III, 2002) fine particulate and sulphur dioxide related pollution were associated with all-cause, lung cancer and cardiopulmonary mortality. They found that each 10-g/m3elevation in fine air particulate pollution was associated with about a 4%, 6%, 8% increased risk of all-cause, cardiopulmonary and lung cancer mortality respectively. Moreover, they concluded that long term exposure  to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Similar findings was made in the cohort of never-smokers by (Turner et al., 2011) where the researchers observed that each 10g/m3increase in PM2.5concentrations was associated with a 15-27% increase in lung cancer mortality. Turner and other concluded that their findings has contributed and strengthened the evidence that recent ambient concentrations of PM2.5are associated with small but measurable increases in lung cancer mortality.

Both short-term and long-term exposure to ambient air pollution can lead to reduced lung function, respiratory infections and aggravated asthma in both children and adults.  The cohort study by (JAMESGAUDERMAN et al., 2000) after appropriate adjustment found that ambient air pollution was correlated with statistically significant decreases in lung function. James Gauderman and others came to the conclusion that these finding suggests that exposure to air pollution may lead to a reduction in maximal lung function which occurs early in adult life, and ultimately to increased risk of chronic respiratory illnesses in adulthood. These findings were similar to the European birth cohort study by (Gehring et al., 2013) where a 20 g/m3increase in levels of NO2, Nitrogen oxides, and a 5g/m3of PM2.5was associated with small decreases in lung function. The authors of the European birth cohort study deduced that exposure to air pollution may result in reduced lung function in schoolchildren.

On the effects of air pollution on asthma, a prospective cohort study by (Jerrett et al., 2008) found significant associations between incident asthma and exposure to ambient NO2. In addition, the risks observed in this study suggest that air pollution contributes to the new onset of asthma. In the time series study by (Schwartz, Slater, Larson, Pierson, & Koenig, 1993), they observed a significant association on the daily counts of asthma emergency room visits for persons under 65 with PM10exposure on the previous day. But, Schwartz and others deduced from their study that the mechanisms triggering or exacerbating asthma remains unclear. However, a study by (Halonen et al., 2008), observed after accurate particle size segregation was used, nucleation mode was associated with asthma visits of children. Moreover, Halonen and others also observed a positive association for the pooled asthma-COPD visits with PM2.5. This study by Halonen et al., also found that coarse and accumulation mode particle levels was statistically significantly associated withe the pooled astham-COPD visits. Halonen and others concluded that smaller particle size (<250nm), gaseous air pollutants and traffic related PM2.5 (at longer lags) were significantly associated with hospital emergency room visits for asthma among children. In contrast, PM2.5 coarse particles and gaseous pollutants had a more immediate effect on the the pooled asthma-COPD visits of the elderly.

Furthermore, the World Health Organization suggests that maternal exposure to ambient air pollution is associated with adverse birth outcomes such as low birth weight (LBW), pre-term birth and small gestational age (SGA) births. In a meta-analysis study by (Dadvand et al., 2012) they found that term low birth weight was positively associated with a 10g/m3increase in PM10 ,OR = 1.03 (95% CI:1.01, 1.05) and PM2.5, OR = 1.10(95% CI:1.03, 1.18). Dadvand and others came to the conclusion that their combined effect estimates study supports an adverse impact of maternal exposure to particulate pollution on fetal growth . In addition, a population based study by  (Maroziene & Grazuleviciene, 2002) found that the risk of preterm birth increased by 25%, adjusted OR = 1.25 (95% CI: 1.07-1.46). Maroziena and Grazuleviciena concluded that a relationship exist between NO2exposure and the risk of preterm birth. Another study by (Malmqvist et al., 2011), they observed a  statistically significant association between  small for gestational age (SGA) births and both nitrogen oxides (NOx) and traffic density. In their subgroup analysis, Malmqvist and other observed an increased risk of SGA for girls, OR = 1.12(95% CI:1.01,1.24); they also observed increased risk among mothers who had not changed residency during pregnancy, OR = 1.09 (95% CI:1.01, 1.18). In this study the authors concluded that controlling for confounders is crucial, especially those that are linked to socioeconomic and spatial gradients.

According to the World Health Organization(WHO, 2019), there is emerging evidence that suggests ambient air pollution may affect diabetes and neurological development in children. This relationship between air pollution and diabetes was observed in a cohort study by (Raaschou-Nielsen et al., 2012)whereby the authors found a statistical significant association between long-term exposure (exposure to above 19.4g/m3 - upper quartile) to traffic-related air pollution at the residence and diabetes mortality, mortality rate ratio, MRR of 2.15 (95% CI 1.21, 3.83).  These findings were consistent with the meta-analysis study by (Janghorbani, Momeni, & Mansourian, 2014) whereby they  found that exposure to air pollution was associated with slight increase risk of diabetes and susceptibility of people with diabetes to air pollution. Moreover, the birth cohort study by (Guxens et al., 2012) observed the effects of exposure to air pollutants on infant mental development. Guxens and others concluded that their findings suggests that prenatal exposure to residential air pollutants may adversely affect infant mental development, but potential effects may be limited to infants whose mothers report low antioxidant intakes.

Even though all populations are affected by air pollution, the burden of disease is inequitable within and across countries. Most of the disease burden is evident in low-and middle-income countries including the poor and marginalized population. To some extent, this is due to the intense process of urbanization and industrial development  in a very short period of time and this phenomenon has had deleterious effects on the health of the people living in these countries(Mannucci & Franchini, 2017). These populations tend to inhabit locations near busy roads and industrial sites characterized by high levels of outdoor air pollution.

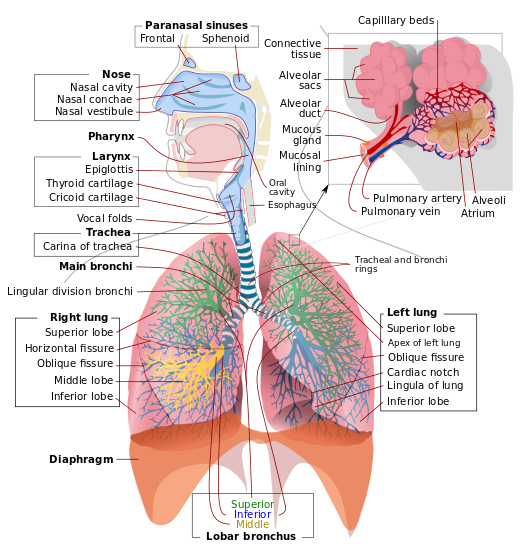
## Ambient Air pollution and Cardiovascular Disease

The cardiovascular system is also known as the circulatory system and includes the heart, arteries, veins capillaries and blood. These vital structures are critical in the process of pumping deoxygenated blood to the lungs for gas exchange as  well as pumping oxygenated blood to the body’s tissues to support their metabolic functions . The cardiovascular system has three major functions and includes:

1. Transportation of materials - the cardiovascular system transports blood to almost all of the body’s tissues. The blood delivers nutrients and oxygen and removes wastes and carbon dioxide to be processed or removed from the body.
2. Protection from pathogens - this is a another of its function and it protects the body through its white blood cells. White blood cells clean up cellular debris and fight pathogens that have entered the body. Platelets and red blood cells form scars to seal wounds and prevent pathogens from entering the body and liquids from leaking out. Blood also carries antibodies that provide specific immunity to pathogens that the body has previously been exposed to or has been vaccinated against.
3. Regulation of the body’s homeostasis - the cardiovascular system is instrumental in the body’s ability to maintain homeostatic *(maintaining internal stability owing to the coordinated response of its parts to any situation or stimulus that would tend to disturb its normal condition or function)* control of several internal conditions. Blood vessels help maintain a stable body temperature by controlling the blood flow to the surface of the skin. Blood vessels near the skin’s surface open during times of overheating to allow hot blood to dump its heat into the body’s surroundings. In the case of hypothermia, these blood vessels constrict to keep flood flowing only to vital organs in the body’s core. Blood also helps balance the body’s pH due to the presence of bicarbonate ions, which act as a buffer solution(Innerbody, n.d.).

Many serious conditions and diseases can cause our cardiovascular system to stop working properly. It is now well reasonably well established that both short-term and chronic air pollution exposures are related to cardiovascular diseases(Brook et al., 2004). Ever since the late 20th Century, epidemiological and clinical evidence has led to an increased concern about the potential harmful effects of ambient air pollution and its association with heart disease and stroke. Of special interest are common air pollutants that include particulate matter with aerodynamic diameter <10m (PM10) and aerodynamic diameter <2.5 (PM2.5),  carbon monoxide, oxides of nitrogen, sulphur dioxide, ozone and lead(Brook et al., 2004).

The most common route for these pollutants to enter the cardiovascular system is via inhalation. when inhaling air travels from the upper respiratory tract which includes the nasal cavity, pharynx, epiglottis and larynx to the lower respiratory tract consisting of the trachea, bronchi, bronchioles and lungs. Of all the pollutants that pass through the respiratory system (refer to Fig. 7), particulate matter with an aerodynamic diameter of <2m and <0.1m have the ability to penetrate the lung alveoli (where gas exchange of oxygen and carbon dioxide occur) and enter the blood stream whereby it exerts the adverse health effects(Franck, Odeh, Wiedensohler, Wehner, & Herbarth, 2011). Because of its very small size , most particulates with an aerodynamic diameter of 2m to 10m are deposited in the nasal cavities and upper airways. Particulat matter has been recognized as being associated with inducing adverse health effects because it may contain substances that can be transported to the respiratory tract(Lee, Kim, & Lee, 2014).



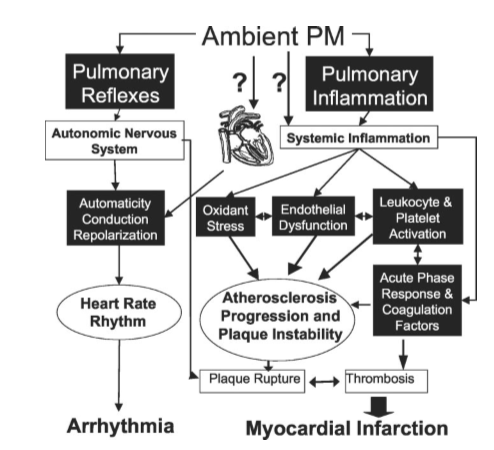
The respiratory system showing the upper and lower respiratory tract as well as the  alveoli

There is a growing body of epidemiological knowledge and studies that the greatest health threat due to air pollution is cardiovascular disease(Lee, Kim, & Lee, 2014). Because of its minute size, particulate matter can be inhaled deep into the lungs, with a portion depositing in the alveoli and entering the pulmonary circulation and apparently the systemic circulation (Sun, Hong, & Wold, 2010). Inhalation of particulate matter or ultrafine particles (UFPs) triggers inflammatory responses in the lung and increases the release of inflammatory mediators in the blood. This in turn can lead to various changes in the cardiovascular system, such as an increase in blood coagulability and the progression of atherosclerotic lesions(Nakane, 2011).

Studies from around the world have consistently shown that both short- and long-term exposures to pollutants, in particular Particulate Matter are associated with a host of cardiovascular illnesses including, myocardial ischaemia and infarctions, heart failure, arrythmias, strokes and increased cardiovascular mortality. Moreover, evidence from cellular/toxicological experiments, controlled animal and human exposures have demonstrated several mechanisms by which pollutant exposure may both trigger acute events as well as prompt the chronic development of cardiovascular diseases(Brook, 2008). In a series of epidemiological studies,(Dockery et al., 1993) and (Samet, Dominici, Curriero, Coursac, & Zeger, 2000) established that air pollution have adverse effects on the cardiovascular system and that brief exposures to air pollution have been associated with increased cardiovascular related morbidity and mortality. This pathology link have particular implications in low- and middle-income countries. These low- and middle-income countries are rapidly developing, hence, air pollution concentrations are continuing to rise. Moreover, the study by (Barnett et al., 2006) on the effect of air pollution on hospitalizations for cardiovascular disease in elderly people in Australia and New Zealand cities found that particulate matter (PM10& PM2.5), NO2, SO2 and CO were significantly associated with higher admissions amongst the elderly (65yrs) than the younger age group (15-64 yrs). The findings from this study showed that the elderly admissions increased for total cardiovascular disease (2.2%), all cardiac disease(2.8%), cardiac failure (6%), ischaemic disease(2.3%) and myocardial infarction (2.9%). The authors of this study in New Zealand and Australia concluded that their advanced age, frailty and with probably pre-existing heart conditions made the elderly population vulnerable. Interestingly, these associations were found at concentrations that were below normal air quality health guidelines and the authors suggest that these guidelines have to be revised and lowered if possible to improve cardiovascular health amongst the vulnerable population. Similar findings on the effects of pollutants on cardiovascular health  of the elderly population were also found in the study by (Liu et al., 2015)

### Potential Biological Mechanisms

The general biological mechanisms linking air pollution to cardiovascular disease involve direct effects of pollutants on the cardiovascular system, blood and lung receptors, and/or indirect effects mediated through pulmonary oxidative stress (an imbalance between the production of free radicals and the ability of the body to counteract or detoxify their harmful effects through neutralization by antioxidants)  and inflammatory responses(Brook et al., 2004). Direct effects may occur by way of agents that readily cross the pulmonary epithelium in to the circulation, such as gases and perhaps ultra fine particles(NEMMAR et al., 2001) along with soluble constituents of particulate matter with an aerodynamic diameter < 2.5 microns (PM2.5), e.g. transition metals. In addition, activation of pulmonary neural reflexes after particulate matter interactions with lung receptors may play a role. Subsequent changes in autonomic tone, under favourable circumstances might contribute to the instability of a vascular plaque or initiate cardiac arrhythmias. These direct effects of air pollution indicate a probable explanation for the occurrence of rapid (within a few hours) cardiovascular responses, such as increased myocardial infarctions(Peters, Dockery, Muller, & Mittleman, 2001). Less acute (several hours to days) and chronic indirect effects may occur via pulmonary oxidative stress/ inflammation induced by inhaled pollutants. Later on, this may contribute to a systemic inflammatory state, which may in turn be capable of activating haemostatic pathways, impairing vascular function, and accelerating atherosclerosis ( a disease in which the inside of an artery narrows due to build up of plaque)(Brook et al., 2004). A general  scheme showing potential mechanisms of the effects of ambient air pollutants (in particular, particulate matter) on the cardiovascular system is shown in Figure.



Illustrates the possible biological mechanisms linking particulate matter with cardiovascular disease. Source:(Brook et al., 2004)

### Ambient Air pollution and Acute Myocardial Infarction

Acute myocardial infarction is also referred to as heart attack, acute coronary syndrome or heart infarction and is a life-threatening condition. It occurs when blood flow to the heart muscle is abruptly cut-off, causing tissue damage. This is usually the result of a blockage in one or more of the coronary arteries. A blockage can develop due to build-up of plaque, a substance mostly made of fat, cholesterol and cellular waste products. Epidemiological studies for the past two decades have consistently shown that the level of air pollutants in the ambient air have been associated with hospitalizations due to acute myocardial infarction. For instance, the epidemiological study by  (Peters, Dockery, Muller, & Mittleman, 2001)  examined the effects of short-term exposure to fine particulate matter and risk of acute myocardial infarction in the greater Boston area, Massachusetts, USA. In their case-crossover study, Peters and colleagues observed that an increase in the levels of fine particulate matter (PM2.5) in the previous 2 hours was associated with an increased risk of myocardial infarction (MI) onset. They also observed a significant association between high 24-hour average concentrations of fine particulate matter and an increased risk of MI with a 24-hour delay. From these findings Peters and other suggested that increased concentrations of fine particulates in the air may elevate the risk of myocardial infarction after a few hours to a day of exposure.

In another case-crossover study by (Rasche et al., 2018), the authors examined the effect of the rapid changes in the levels of NO2,O3, PM10, oxides of nitrogen (NOx) and risk of myocardial infarction for the populace in the city of Jena, Germany. Rashe and colleagues observed that a 20g/m3and 8-20g/m3increase in NOxwas associated with up to 121% increased risk of MI with a lag time (Lag-2 days and Lag-3days). In addition, rapid changes in NO2(lag-1day) was associated in a close-to-linear risk of myocardial infarction. Moreover, increases in NO2by more than 8g/m3increased the risk of MI by 73%. In this study, PM10concentrations was not associated with any increased risk in MI. From these findings, Rasche and others suggested that the risk of Myocardial Infarction was not only dependent on short-term or long-term exposure to gaseous pollutants but also in the dynamic and magnitude of their increases.

In a similar study design to those discussed above, (Collart, Coppieters, Mercier, Kubuta, & Leveque, 2015) investigated the association between short-term exposure to air pollutants and the triggering of acute myocardial infarction (AMI) in Charleroi, a heavily polluted region in Wallonia, Belgium. Collart and colleagues observed that the risk of  AMI increased when, (1) PM10and NO2 concentrations increased by 10g/m3 and, (2) when there was a 1oC increase in temperature. From these findings Collart and others concluded that the effects of the short-term effects of  air pollutants was stronger in the warm period.

Furthermore, the findings from a meta-analysis study by (Mustafić et al., 2012) suggested that all the main air pollutants i.e. carbon monoxide, nitrogen dioxide, sulphur dioxide and particulate matter (PM2.5 and PM10) were significantly associated with a near-term increase in myocardial risk.

# Chapter 3: Population and Methods

## Introduction

The objective of this chapter is to provide clarity on the methods that was used in order to meet the aims and objectives of the thesis as outlined in chapter one. In addition, this chapter will demonstrate the adopted epidemiological design in order to impute the desired outcome.  Firstly, the study design for this thesis and why it was chosen will be discussed. Secondly, the procedure involved in the collection of the ambient air quality and weather data be presented and discussed. The processes involved in manipulating the raw dataset before it is being described and analysed will be presented.

Finally, a brief description on the crucial steps taken during the meta-analysis will be discussed.

## Study design

This study will employ a combination of meta-analysis, collection of field data on air quality by measuring criteria pollutants (excluding lead) and developing a dose-response model of the association between criteria pollutants and risk of myocardial infarction.

## Air quality and Weather data

The VAISALA Air quality transmitter (AQT) 400 series which consists of two products (AQT 420 & AQT 410)the AQT 420 was the equipment of choice to collect air quality and weather data as it measured most of the common air pollutants ( PM10, PM2.5, SO2, NO2, O3, CO) and weather variables (Temperature, relative humidity, atmospheric pressure). This is one of the latest air quality monitoring equipment that is accurate and uses high-precision instrumental methods in order to understand how contaminant levels fluctuate over short time periods ( hours or days). One of the distinct features of this instrument is that it compensates for the impact of ambient air conditions and is specifically designed for urban areas, road networks or industrial sites and around transportation hubs(Vaisala, 2018). Two air quality monitoring sensors (AQT 420) were mounted on two existing traffic camera poles (refer to Fig 9). The instruments mounted on each poles includes a flexible solar panel, one VAISALA Air Quality Transmitter AQT 420, a box containing a 12v rechargeable alkali battery, a solar panel regulator and a serial data logger. The AQT 400 series was located at a height of 3m from the ground as a precautionary measure from vandalism and theft Air quality data was collected between the months of August and October of 2018. Ambient air quality data on levels of particulate matter with an aerodynamic <10m(PM10) and aerodynamic <2.5m (PM2.5), sulphur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide (CO) and ozone (O3) were collected at the two air quality monitoring sites. The pollutants PM10and PM2.5 was measured as microgram per cubic meter (g/m3) and the pollutants CO, NO2, SO3, O3were measured as parts per billion (ppb). The VAISALA Air Quality Transmitter 400 series also measures meteorology data like temperature in degrees Celcius (0C), relative humidity in percentage (%) and pressure in hectopascal (hPa). Ambient air quality and meteorology data from the VAISALA Air Quality Transmitter 400 series was transferred into the serial data logger  and then stored in a universal serial bus (USB) flash drive in text document (txt).



Setting up and mounting of air quality monitoring sensors and its components

## Data Cleaning

Air quality data was then exported into a Microsoft excel spreadsheet, saved as a Microsoft excel document and named Site 1.  From the saved excel document, rows 1 - 6 were removed as they were readings when the equipment was being tested at the University of Canterbury Geography lab. Columns L, M and N were also removed as they had no relevance to the desired data set. A new row was then inserted at the top of the excel sheet and used as the headings for each pollutant and meteorology variable.Column A was then split into two columns and Column A was named as ‘Date’ and column B as ‘Time’. Column C was for the readings for the ‘Temperature, column d for ’Humidity’, column E for ‘Pressure’, column F for ’NO2’, column G for ’SO2‘, column H for ’CO’, column I for ’O3‘, column J for ’PM2\_5’ and column K for ‘PM10’. After all these steps were taken to clean the raw data, a new excel spreadsheet was created and the cleaned data pasted onto it and given the name Site 1\_data. These steps were replicated for the data obtained from Site 2 except that the first 6 rows was not removed. The cleaned data was then pasted onto a new excel spreadsheet and given the name Site2\_data. On the completion of cleaning the raw data from both sites, a third spreadsheet was also created and named ‘Master\_Sheet’. This Microsoft excel sheet contains cleaned data from both Site 1 and Site 2 merged into one data set for the Lautoka city.

## Data Processing

Now that the data is clean, it was imported into R Studio for further processing before beginning with the analysis. R Studio is a free and open-source integrated development environment for R, a programming language for statistical computing and graphics.  R-Studio uses the graphical environment of the computer to facilitate interactions with R for example, it has a Console window for typing code, a data window for reviewing data frame and other data structures, a work space for viewing all of the data loaded into R, Plot area for showing visualizations and a tabbed window control to show other displays(“About - RStudio”, n.d.).  Processing data meant that I had to manipulate the clean data in order to impute the mean or average hourly and daily values of the environmental exposure variables which includes temperature, pressure, humidity, nitrogen dioxide, sulphur dioxide, carbon monoxide, ozone, particulate matter with an aerodynamic diameter of <2.5 microns and particulate matter with an aerodynamic diameter of <10 microns.

In R Studio, a new project was created and the dataset ‘Master\_Sheet.xls’ with a total of 6,822 observations  of 11 variables was imported into R and renamed as *“aqdata”*. The R packages library(tidyverse), library (readxl), library(lubridate) was then loaded onto the R console as they will be used to process and later on analyse this dataset. These R packages makes it easier to work with dates and times and to read, write and format excel files. In addition, it is a coherent system of packages for data manipulation, exploration and visualization that share a common design philosophy.

Using the pipe function denoted by *“%>%”*and mutate function denoted by *“mutate()”*   the columns *“date”* and  *“time”* in the aqdata dataset was corrected so that the year was separated from the months, months were separated from the days, days were separated from the hours, hours were separated from minutes, minutes were separated from seconds. After running this code chunk in RStudio, 7 new variables were added to the existing  *aqdata* dataset and the new dataset with a total of 6822 observation of 18 variables was renamed as  *“aqdata\_time\_corrected”*. The new variables were year, month, day, hour, minute, second and date.

The final step in data processing was to group the data based on hour and then based on day. To do this in RStudio, the function *“group\_by”* was employed and the pipe function, *“%>%”*, was then used to impute and summarise the mean values of all the observations. The outcome of this final data processing step yielded a total of 121 observations of 13 variables and the final dataset was named, *“aqdata\_revised”*. Now that the final dataset has been imputed, the next step was out carry out the analysis.

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## Data Analysis

### Descriptive Statistics

The daily (24-hour) average or mean concentrations  for the pollutants, particulate matter with an aerodynamic diameter <2.5 and aerodynamic diameter <10 microns, sulphur dioxide, nitrogen dioxide, zone and carbon monoxide was imputed using the formula

and the values tabulated. Mean values for the weather variables, temperature, relative humidity and atmospheric pressure was also obtained. The range which included the minimum and maximum values for all these environmental variables was also obtained when imputing their mean values. Values for the 25th, 50th& 75thpercentiles were also acquired by arithmetic means and the value tabulated.

To measure the statistical relationship between the environmental variables (PM10, PM2.5, SO2, NO2, O3, CO, temperature, relative humidity and atmospheric pressure) Pearson’s correlation coefficient, *r*,  was employed. Pearson’s correlation coefficient is the best method of measuring the association between two variables of interest because it is based on the method of covariance. It provides information about the magnitude of the association, or correlation as well as the direction of the relationship.

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### Time Series

The time series approach was used to determine the construct the concentrations of the pollutants during the hours of a day. Concentrations for each hour was obtained and the mean imputed (i.e. concentrations for every 1 am of every day were obtained and the average imputed. This procedure was repeated for 2am, 3am, 4am and so on). This was done for the whole 24 hours period in order to be able to visualize the observations of the variables against time. Visualizing the air quality and meteorology data enables the identification of the nature and distinct features of the observations. All the time series analysis was done using the Microsoft Excel 2016 software program.

## Meta-Analysis

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The Participant-Intervention-Comparison-Outcomes (PICO)(Schardt, Adams, Owens, Keitz, & Fontelo, 2007) framework was used as a guide in developing the theory and framing of the question on air quality and risk of acute myocardial infarction hospitalizations. Information to assist in the formulation of the question was obtained from observational epidemiological studies such as case-cross over and time series studies. Hence, any Intervention was not appropriate and was replaced with the term ‘Exposure’. The question was then  developed according to the PICO framework and is as follows:

* P - General population ( both sexes, all ethnicity, all nationality)
* E - pollutants (PM2.5 and PM10)
* C - lower levels of pollutants
* O - higher levels of pollutants

On the basis of PICO, the meta-analysis research question was reframed to read, “ Compared with lower levels of exposure to air pollutants, what is the risk of acute myocardial infarction related hospitalizations for people who are exposed to higher levels of air pollutants”.

### Literature Search

Based on the PICO research question, a systematic review of literature was conducted. The literature search was done to identify published primary studies that evaluated the effect of  exposure to the common air pollutants and the risk of myocardial infarction. These pollutants are carbon monoxide (CO), nitrogen dioxide (NO2), sulphur dioxide (SO2), ground-level ozone (O3) and particulate matter with an aerodynamic diameter of < 2.5 microns (PM2.5) and particulate matter with an aerodynamic diameter of < 10 microns (PM10).

A comprehensive search on several electronic databases including Embase (between 1974 to December 10th, 2018) and The Ovid Medline and in-process and other non-indexed citations ( between 1946 to November 27, 2018) was conducted to search for all relevant published studies. To exhaust relevant literature on the desired studies other databases were accessed which included Google scholar, Toxnet and Web of Science.  In addition, the search for desired studies was extended to reference lists of eligible studies for additional published and unpublished data. In searching for relevant articles, the search terms were arranged using the Boolean logic(“DMP Provider | Data Management Platform & Solutions | Lotame”, n.d.). Moreover, specific search related vocabulary, symbols of truncation of expansion, and placement of the relevant terms in different sections of an article were also used to identify as much relevant studies as possible(Tuttle, von Isenburg, Schardt, & Powers, 2009).

The  Medical Subject Headings (MeSH) classification system was employed for the primary search in Ovid Medline, Embase  and Toxnet.  This search included a combination of key words such as ‘air pollution’, ‘air pollutants’, ‘particulate matter’, ‘vehicle emissions’, ‘ozone’, ‘carbon monoxide’, ‘sulphur dioxide’,  ‘nitrogen dioxide’ in the title or abstracts of a study. Similarly, the key words used to search for the desired outcome were myocardial Infarction, acute coronary syndrome, heart attack or heart infarction.

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### Study Selection (Inclusion and Exclusion criteria)

The selection of relevant studies was based on a scheme of inclusion and exclusion criteria so that only the desired studies were retrieved. Initially all studies that was selected had to be published online from the beginning of 2008 to the year 2018. Studies had to be primary studies and published only in the English language. After reading through the titles and abstracts, studies that evaluating the outcome of interest to the research i.e. outcomes had to be either ‘myocardial infarction’ or ‘acute myocardial infarction’ or -ST-Elevation Myocardial Infarction’ or ‘Acute coronary syndrome’ were retained and  others excluded.  In addition, studies had to include exposure to any of the common air pollutants which includes particulate matter with an aerodynamic diameter < 2.5 microns and < 10 microns, carbon monoxide, ground-level ozone, sulphur dioxide and  nitrogen dioxide. All studies that focused on special population groups was excluded and those studies that addressed the general population was included. Moreover, primary studies which employed the time series and case-crossover (Maclure, 1991) designs were selected and studies on any other design were excluded. Furthermore, all qualitative studies were excluded and all duplicates were removed.

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### Quality score assessment

The quality score used to assess the methodological quality of the selected studies were adapted from a scale based on previous systematic reviews and meta-analysis(Mustafić et al., 2012). The four components used to evaluate these articles are as follows: (1) Determining of MI (0 to 1 point). A score of 1 was given if the diagnosis of MI was coded in accordance with the International Classification of Diseases or based on angiographic criteria or in accordance with the clinical, laboratory and electrocardiographic criteria. A score of 0 was given if there was no description of diagnosis or the diagnosis was determined by patient history or other criteria. (2) The quality of air pollutant measurements (0 to 1 point). If measurements were performed at least daily and less than 25% missing data, a score of 1 was given. Measurements that were not performed hourly  and more than 25% missing data or there was no description of particulate measurements, a score of 0 was given (Mustafić et al., 2012). (3) Adjustment for confounders (0 to 1 point). Due to the differences in research methods used between case-crossover and time series studies, the methods of adjustment for confounders were also different. For time series studies, a score of 1 was given if adjustment for covariates was performed for several important covariates together, including long-term trends, seasonality, temperature, humidity, pressure or day of week. For case-crossover studies which control invariant and slowly changing confounders by the design itself, therefore, if an adjustment was made for temperature, humidity, pressure or day of week, a score of 1 was given. A score of 0 was given if studies did not do any adjustment of the above mentioned important covariates. Finally, if a study got maximum score in each component, it was considered to be of good quality. On the other hand, if one of the 3 components got a minimum score of 0, it was considered to be of low quality.

### Step 7: Testing for heterogeneity

In this test, the information obtained in steps 4 & 5 was first use to test the heterogeneity of the studies. Evaluating the statistical heterogeneity of the studies involved using a variation of chi-square test based on pooled estimate, the effect estimate of each individual study and the number of studies. This test is referred to as Q statistic test(Cochran, 1954) and the associated p-value noted, and further evaluated at 0.05 for the null hypothesis. The null hypothesis was that, “the results of the study are similar to each other or that there is no difference between the results of the studies included in the meta-analysis. The alternative hypothesis was that, ”the results of the study differ from each other". If the p-value rejects the null hypothesis, then the studies are heterogeneous; if the p-value fails to reject the null hypothesis, then the conclusion will be that the studies are homogeneous. If the studies are homogeneous, the results of the studies will be pooled together and two types of estimates reported: (1) fixed effects estimates based on the assumption that the studies that have been included in the research from an exhaustive set of studies; and (2) a random effect estimate where it will be assumed that the set of studies being included in the analysis from a ‘sample’ or random sample of studies of ‘all possible studies’.

### Step 8: Testing for Publication Bias

Here I will test for publication bias. Publication bias refers to a bias that occurs due to the fact that smaller studies and those with “equivocal estimates”(i.e. estimates that are inconclusive or those studies with negative estimates) are less likely to be published and therefore less likely to be captured in this meta-analysis than those studies that are large and have significant findings. Plotting the variance of the study estimates (variance of the effect estimate of a study is a function of its sample size) and the effects estimate itself will show that the cloud of point may define a funnel. The base of the funnel will be formed by studies that are small in size (hence large variance) and the effect estimates will vary all around the point estimate; the apex or peak of the funnel will be formed by those studies that are large sized (hence low variance) and all the estimates will be clouded around the point estimate obtained in the meta-analysis. If part of the funnel is missing, then that indicates that there was publication bias. This is referred to as the funnel plot. There are other tests, such as ‘Egger’s test’ that can statistically report the extent of publication bias. Eggers test(Egger, Smith, Schneider, & Minder, 1997) is a statistical test to assess the funnel plot asymmetry. In addition, Egger’s test plots the regression line between precision of the studies (independent variable) and the standardized effect (dependent variable). When there isn’t any publication, the regression line originates in the y-axis zero. If it is much further away from zero, this suggest further evidence of publication bias(Molina, n.d.). Nothing much can be done to remedy publication bias other than searching for ‘fugitive literature’ and contacting the research groups and other who can have studies that are small and remained unpublished or actually obtain the raw data from different sources.

### Step 9: Subgroup analyses and meta-regression

In the final step, I tested for meta-regression or subgroup analyses. Here, I sub grouped the data and analysed them separately using a regression model. I tested if the estimates were different for those in developing vs developing countries and also for those with different types of source apportionment. Source apportionment is when different sources contributes differently to air pollution. For example, do sources such as vehicle exhausts lead to higher admission rates than at coal burning plants? Is the association between PM10 and hospital admissions different for developing countries than for developed countries?

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### Data Extraction

Relevant information was extracted from all eligible studies obtained in accordance with the inclusion and exclusion criteria in the study selection process. Each eligible study was printed in PDF format and the full text read independently.  A standard ‘Summary of Findings’ form was created in Microsoft excel and a full description of each study characteristics was extracted as follows:  the author and year of publication, the locality, the pollution exposure, the nature of the outcome,  the study design, pollutant models, adjustments performed, effect measurement/effect size, the number of participants for the study and the critique quality score.

The case crossover studies included in the meta-analysis are (Hopke et al., 2015; Zhang et al., 2016; Hsieh, Yang, Wu, & Yang, 2010; Chang, Kuo, Liou, & Yang, 2013; Cheng, Tsai, & Yang, 2009; Bejot et al., 2011; Vencloviene, Grazuleviciene, Babarskiene, Dedele, & Grazulevicius, 2011; Nuvolone et al., 2011; Liu et al., 2018; Chiu, Weng, Chiu, & Yang, 2017; Pope et al., 2015; Argacha et al., 2016; Gardner et al., 2014; Rasche et al., 2018; Sahlén et al., 2019; Evans et al., 2016; Weichenthal et al., 2017; Bhaskaran et al., 2011; Xiaodong et al., 2015; Rich et al., 2013).

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# Chapter 4: Results

## Introduction

The objective of this chapter is to firstly, provide descriptive information on the ambient air pollutant levels in the city of Lautoka. Descriptive statistics on all the air quality and environmental variables will be presented to show the distribution of the observations with time. Air pollutants that showed significant levels will be visualized using graphs to highlight existing variations with time (hours of a day). The mean and range will be imputed to show the mean daily pollutant levels in comparison to the WHO air pollutant guideline values. Correlation coefficients will also be imputed and presented to demonstrate the statistical relationship between two variables. Secondly, the forest plot and the precise pooled estimate derived from the meta-analysis on the risk of myocardial infarction due to exposure to air pollutants will also be presented. Lastly, on the basis of the pooled estimate from the meta-analysis and air quality data the risk of myocardial infarction hospitalisation will be predicted.

## Descriptive statistics on air pollutants and weather variables

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Table 2 provides the 24-hour average descriptive statistics on air pollutant levels and meteorological variables. The overall mean daily PM10 and PM 2.5concentrations  and standard deviation was 73.42  33.23 g/m3and 10.87  5.8 g/m3 respectively. The range for daily PM10concentrations was a minimum concentration of 30.71 to a maximum concentration of 142.76 g/m3 whereas the range for daily PM2.5 concentrations was a minimum concentration of 3.95 to a maximum concentration of 31.75 g/m3. The overall daily mean concentrations and standard deviation for the pollutants SO2, NO2, O3 and CO were 0.09  0.17 ppb, 0.05 0.03 ppb, 0.004   0.03 ppb and 0.39  0.22 ppb respectively. The range for the 24-hour average of SO2concentrations was a minimum concentration of 0.012 to a maximum concentration of 0.76ppb, the range for NO2was a minimum concentration of 0.02 to a maximum concentration of 0.173 ppb, range for CO was a minimum concentration of -0.41 to a maximum concentration of 0.63ppb, range for ozone was a minimum concentration of -0.0009bto a maximum concentration of 0.109 ppb. The mean daily temperature was 27.53  2.07 0C with a range of a minimum temperature of 23.32 to a maximum temperature of 30.56 0C. The mean daily relative humidity was 67.72  6.77% with a range of a minimum relative humidity of 59.4 to a maximum relative humidity of 83.3 %. The mean daily atmospheric pressure was 1012  2.95 hPa with a range of minimum atmospheric pressure of 1004 hPa to a maximum atmospheric pressure of 1015 hPa.

Summary of the descriptive statistics for air pollutants and meteorology variables in the Lautoka CBD

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  |  |  | Percentiles |  |  |  |
| Exposure Variables | Mean**± SD** | Min | 0.25 | 0.5 | 0.75 | Max |
| PM10 | 73.42 ± 33.23 | 30.71 | 44.88 | 68.97 | 99.57 | 142.76 |
| PM2.5 | 10.87 ± 5.8 | 3.95 | 8.14 | 9.44 | 12.82 | 31.75 |
| SO2 | 0.09 ± 0.17 | 0.01 | 0.03 | 0.03 | 0.06 | 0.76 |
| NO2 | 0.05 ± 0.03 | 0.02 | 0.03 | 0.04 | 0.04 | 0.17 |
| CO | 0.39 ± 0.22 | -0.41 | 0.2 | 0.32 | 0.42 | 0.63 |
| O3 | 0.0 ± 0.03 | -0.0 | 0.0 | 0.0 | 0.01 | 0.11 |
| Temp(oC) | 27.53 ± 2.07 | 23.32 | 26.72 | 27.53 | 29.21 | 30.56 |
| Rel. Humidity (%) | 68.72 ± 6.77 | 59.4 | 63.7 | 67.36 | 71.94 | 83.3 |
| Pressure | 1012 ± 2.95 | 1004 | 1011 | 103 | 1014 | 1015 |

This is a caption

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Days | PM2.5 | PM10 | O3 | CO | SO2 | NO2 | Pressure | Humidity | Temperature |
| Monday | 7.22 | 40.77 | 0.0 | -1.88 | 0.01 | 0.01 | 1014.3 | 60.31 | 27.4 |
| Tuesday | 8.62 | 62.28 | 0.0 | 0.4 | 0.03 | 0.02 | 1013.3 | 74.58 | 27.63 |
| Wednesday | 8.89 | 59.83 | 0.01 | 0.27 | 0.2 | 0.03 | 1013.68 | 66.02 | 27.33 |
| Thursday | 12.65 | 77.92 | 0.02 | 0.3 | 0.04 | 0.06 | 1012.26 | 68.23 | 27.25 |
| Friday | 12.98 | 93.28 | 0.03 | 0.38 | 0.06 | 0.04 | 1012.19 | 65.49 | 28.21 |
| Saturday | 5.71 | 50.85 | 0.02 | -0.1 | -0.0 | 0.1 | 998.97 | 58.0 | 30.27 |

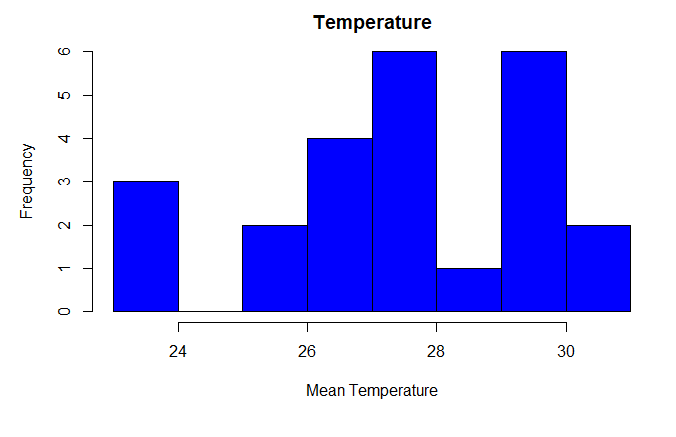
This is a caption

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| hours | mean\_pm10 | pm25 | no2 | so2 | ozone | CO | temperature | humidity | pressure |
| 1 | 77.23 | 8.16 | 0.03 | 0.03 | 0.0 | 0.31 | 27.0 | 69.41 | 1010.57 |
| 2 | 36.55 | 6.02 | 0.04 | 0.03 | 0.01 | 0.18 | 26.66 | 71.43 | 1003.72 |
| 3 | 30.71 | 4.59 | 0.17 | 0.04 | 0.0 | -0.41 | 25.29 | 75.9 | 1007.86 |
| 4 | 35.19 | 3.94 | 0.03 | 0.05 | -0.0 | 0.2 | 23.71 | 81.33 | 1013.06 |
| 5 | 39.82 | 4.18 | 0.03 | 0.06 | -0.0 | 0.23 | 23.32 | 83.3 | 1013.36 |
| 6 | 130.15 | 9.6 | 0.04 | 0.07 | -0.0 | 0.38 | 23.85 | 80.9 | 1013.87 |
| 7 | 94.4 | 12.66 | 0.05 | 0.06 | -0.0 | 0.63 | 25.84 | 73.71 | 1014.49 |
| 8 | 126.59 | 31.75 | 0.04 | 0.05 | 0.01 | 0.6 | 27.81 | 66.84 | 1015.07 |
| 9 | 106.42 | 15.25 | 0.03 | 0.01 | 0.01 | 0.32 | 29.17 | 63.67 | 1015.15 |
| 10 | 100.11 | 10.39 | 0.06 | 0.76 | 0.02 | 0.21 | 30.13 | 59.4 | 1015.01 |
| 11 | 41.43 | 8.29 | 0.07 | 0.46 | 0.11 | 0.01 | 30.56 | 59.82 | 1014.41 |
| 12 | 47.78 | 8.1 | 0.03 | 0.2 | 0.1 | 0.16 | 29.89 | 61.65 | 1013.47 |
| 13 | 45.55 | 8.3 | 0.02 | 0.03 | 0.01 | 0.43 | 30.0 | 61.17 | 1013.55 |
| 14 | 42.88 | 8.15 | 0.02 | 0.01 | 0.01 | 0.39 | 29.72 | 61.75 | 1013.01 |
| 15 | 47.47 | 9.29 | 0.03 | 0.02 | 0.0 | 0.39 | 29.33 | 63.95 | 1012.59 |
| 16 | 60.72 | 10.25 | 0.03 | 0.02 | 0.0 | 0.45 | 29.1 | 63.71 | 1012.54 |
| 17 | 78.0 | 11.65 | 0.13 | 0.03 | 0.05 | -0.08 | 28.75 | 65.54 | 1012.77 |
| 18 | 102.17 | 13.28 | 0.04 | 0.15 | 0.03 | 0.47 | 27.89 | 66.77 | 1013.03 |
| 19 | 99.39 | 15.88 | 0.03 | 0.03 | 0.0 | 0.41 | 27.43 | 66.64 | 1010.49 |
| 20 | 142.76 | 18.89 | 0.04 | 0.03 | 0.0 | 0.46 | 26.95 | 71.29 | 1011.01 |
| 21 | 85.07 | 14.62 | 0.04 | 0.03 | 0.0 | 0.34 | 26.74 | 71.53 | 1012.07 |
| 22 | 79.44 | 10.57 | 0.04 | 0.03 | 0.0 | 0.28 | 26.79 | 73.16 | 1015.07 |
| 23 | 57.73 | 9.09 | 0.04 | 0.03 | 0.01 | 0.26 | 27.64 | 68.43 | 1005.4 |
| 24 | 54.55 | 7.96 | 0.03 | 0.03 | 0.0 | 0.33 | 27.16 | 67.87 | 1010.59 |

Figure 9 illustrates the frequency distribution of the mean temperature (expressed as degrees Celsius, oC) for the Lautoka CBD. On the horizontal axis or x-axis is the mean temperature values and on the vertical axis or y-axis is the frequencies of these temperature values. Most of the days recorded a mean temperature between 27oC/28oC and 29oC/30oC whilst very few days recorded a temperature of less than 25oC.

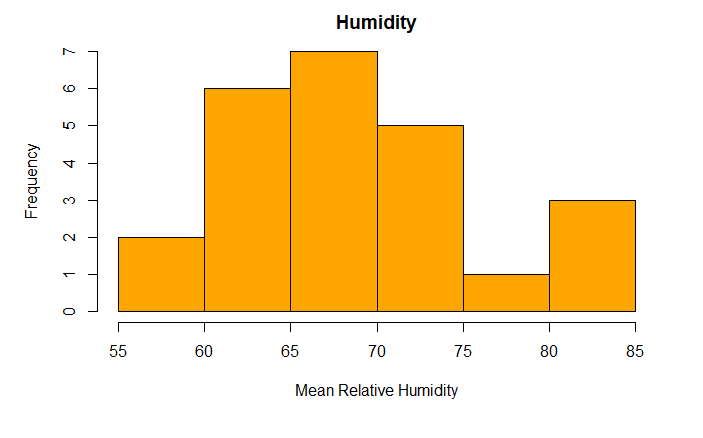
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Shows the histogram for the mean temperature (24-hr averages)

Figure 11 illustrates the frequency distribution of the mean relative humidity (expressed as percentage, %) for the Lautoka Central Business District. The horizontal axis or x-axis is the mean relative humidity values and on the vertical axis or y-axis is the frequencies of these rel. humidity values. Most of the days recorded a mean rel. humidity between 60 % to 70% whilst very few days had a rel. humidity between 75% and 80%.



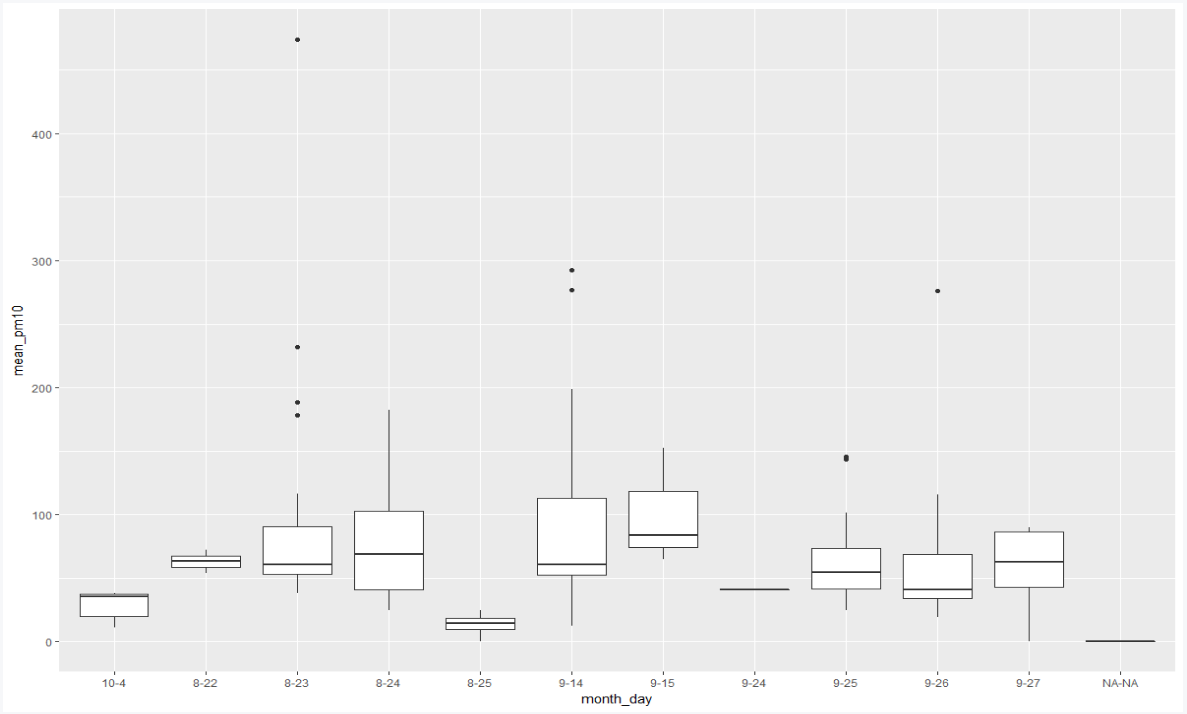
Shows the histogram for the mean ((24-hr averages) temperature

 Figure 12 illustrates the frequency distribution of the mean atmospheric pressure ( expressed as hectopascal, hPa) for Lautoka city. The horizontal axis or x-axis, is the mean atmospheric pressure values and on the vertical axis or y-axis is the frequencies of the atmospheric pressure values. Most of the days recorded a mean atmospheric pressure between 1010 to 1015 hPa.

## 

Figure 12 illustrates the

## 



Shows the boxplot for the pollutant PM10 and days of the month (month\_day)

## 

## Relationships among the air pollutants and weather variables

Table 3 displays the Pearson’s correlation coefficients, r,  values for the air pollutants and weather variables.  A strong (positive) linear relationship was observed between PM10and PM2.5 with a correlation coefficient, r = 0.76, p-value<0.001. A moderate (positive) linear relationship was observed between PM10and carbon monoxide, r = 0.51 and p=0.001. This moderate (positive) linear relationship was also exhibited between PM10and atmospheric pressure with a correlation coefficient, r = 0.33, p=0.2. No linear relationship was observed between PM10and SO2, NO2, O3, temperature or relative humidity. PM2.5 showed a moderate (positive) linear relationship with CO, r = 0.52, p=0.008. A weak (positive) relationship was observed between PM2.5 and atmospheric pressure, r = 0.3, p = 0.16. No linear relationship was observed between PM2.5 and SO2, NO2,O3, temperature or relative humidity. SO2showed a moderate (positive) linear relationship with the variables O3(r = 0.48, p=0.02), temperature (r = 0.38, p = 0.06) and pressure (r = 0.3,p =0.15). SO2also exhibited a moderate (negative) linear relationship with the weather variable, humidity (r = -0.4, p=0.06). No linear relationship was observed between SO2and NO2 or CO. A strong (negative) linear relationship was observed between NO2 and CO (r = -0.78, p<0.001). No linear relationship was observed between NO2and O3, temperature, relative humidity or atmospheric pressure. CO showed a weak (positive) linear relationship and a weak (negative) linear relationship atmospheric pressure (r = 0.31, p = 0.14) and O3 (r = -0.38, p = 0.06) respectively. No linear relationship was observed between CO and the weather variables temperature or relative humidity. A moderate (positive) relationship was observed between O3 and the weather variable temperature, r = 0.53, p = 0.007. A weak (negative) linear relationship was also observed between O3 and relative humidity, r = -0.49, p=0.01. No linear relationship was observed between O3 and atmospheric pressure. Temperature showed a strong (negative) linear relationship with relative humidity (r = -0.98, p <0.001). There was no linear relationship observed between temperature and atmospheric pressure. Moreover, no linear relationship was observed between relative humidity and atmospheric pressure.

Pearson’s correlation coefficients among air pollutants and weather variables

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Variables | PM**10** | PM**2.5** | SO**2** | NO**2** | CO | O**3** | Temperature | Humidity | Pressure |
| PM10 | 1 | 0.76 | 0.05 | -0.14 | 0.51 | -0.2 | -0.05 | 0.02 | 0.33 |
| PM2.5 |  | 1 | -0.07 | -0.14 | 0.52 | -0.09 | 0.19 | 0.22 | 0.3 |
| SO2 |  |  | 1 | 0.1 | -0.21 | 0.48 | 0.38 | -0.4 | 0.3 |
| NO2 |  |  |  | 1 | -0.78 | 0.18 | -0.1 | 0.11 | -0.18 |
| CO |  |  |  |  | 1 | -0.38 | 0.05 | -0.09 | 0.31 |
| O3 |  |  |  |  |  | 1 | 0.53 | -0.49 | 0.2 |
| Temperature |  |  |  |  |  |  | 1 | -0.98 | 0.2 |
| Humidity |  |  |  |  |  |  |  | 1 | -0.17 |
| Pressure |  |  |  |  |  |  |  |  | 1 |

## Descriptive information of studies in meta-analysis

This is a caption

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Author | Location | Exposures | Outcome | Study design | OR | lower\_CI | upper\_CI | N | quality\_score |
| Cheng, 2009 | Kaohsiung, Taiwan | SO2, NO2, PM10, CO, O3 | AMI hospital admissions, Daily counts of hospital admissions | Casecrossover | 1.41 | 1.27 | 1.57 | 9349 | 3 |
| Hsieh, 2010 | Taipei, Taiwan | SO2, NO2, PM10, CO, O3 | AMI hospital admissions, Daily counts | Casecrossover | 1.1 | 1.05 | 1.15 | 23420 | 3 |
| Bhaskaran, 2011 | England & Wales | O3,CO,NO2,SO2, PM10 | AMI admissions, daily counts | Casecrossover | 1.2 | 0.3 | 2.1 | 79288 | 3 |
| Nuvulone, 2011 | Tuscany, Italy | CO,NO2, PM10 | AMI hospitalisations | Casecrossover | 1.01 | 1 | 1.03 | 11450 | 3 |
| Wang, 2016 | Pudomng district, Shanghai, China | PM2.5, PM10,SO2,NO2, CO | AMI hospital admissions, Daily counts | Casecrossover | 1.16 | 1.03 | 1.29 | 972 | 3 |
| Liu, 2017 | 26 cities in China | PM2.5,CO,NO2,SO2 | STEMI hospital admissions | Casecrossover | 0.9 | 0 | 1.8 | 1531 | 3 |
| Weichenthal, 2017 | British Columbia, Canada | PM2.5 | AMI hospital admissions | Casecrossover | 1.06 | 1.03 | 1.08 | 1531 | 3 |
| Hopke, 2015 | Rochester, New York, USA | PM2.5 | STEMI admissions, daily counts | Casecrossover | 0.99 | 0.8 | 1.22 | 338 | 3 |
| Chang, 2013 | Taipei, Taiwan | PM2.5,PM10,CO,NO2,SO2,O3 | MI hospital admissions, daily counts | Casecrossover | 1.04 | 1 | 1.08 | 14353 | 3 |
| Evans, 2016 | Monroe County New York,USA | Wood smoke (Delta C), traffic pollution(BC),PM2.5, NO2, SO2, O3,CO | STEMI admissions, daily counts | Casecrossover | 1.17 | 0.99 | 1.39 | 362 | 3 |
| Weichenthal, 2017 | 16 cities in Ontario, Canada | PM2.5, NO2, O3 | MI emergency room visits | Casecrossover | 1.06 | 1.03 | 1.08 | 30101 | 3 |
| Pope, 2015 | Utah’s Wasatch Front, USA | PM10, PM2.5 | STEMI admissions, | Casecrossover | 1.02 | 0.97 | 1.08 | 1274 | 3 |
| Argacha, 2016 | Belgium | PM10,PM2.5,O3,NO2 | STEMI hospitalisations | Casecrossover | 1.03 | 1.0 | 1.05 | 11428 | 2 |
| Zhang, 2016 | Chaoyang, Beijing China | PM2.5, PM10,NO2, SO2,O3, CO | STEMI | Casecrossover | 1.05 | 1 | 1.11 | 2749 | 3 |
| Rasche,2018 | Jena, Germany | NO2, PM10, O3 | MI emergency visits and admissions | Casecrossover | 2.21 | 1.19 | 4.08 | 693 | 3 |
| Sahlen, 2018 | Stockholm, Sweden | NO2,SO2,O3,PM10, PM2.5 | MI admissions | Casecrossover | 1.03 | 1.0 | 1.05 | 14601 | 2 |
| Bejot, 2010 | Dijon, France | O3 | MI admissions | Casecrossover | 1.12 | 1.03 | 1.21 | 913 | 2 |
| Vencloviene, 2011 | Kaunas, Lithunia | NO2 | MI hospital admissions, daily counts | Casecrossover | 1.61 | 1.03 | 2.23 | 2895 | 3 |
| Rich, 2013 | New Jersey, USA | PM2.5 | MI admissions | Casecrossover | 1.13 | 1 | 1.27 | 1563 | 3 |
| Gardner, 2014 | Rochester, New York, USA | PM2.5 | STEMI adimissions | Casecrossover | 1.17 | 0.98 | 1.4 | 338 | 3 |
| Chiu, 2017 | Taipei, Taiwan | O3 | MI hospital admissions, daily counts | Casecrossover | 1.07 | 1.02 | 1.12 | 14353 | 3 |
| Rosenthal, 2013 | Helsinki, Finland | NO, NO2,O3,CO,SO2,PM2.5,PM10 | MI admissions | Casecrossover | 1.14 | 1.03 | 1.27 | 629 | 2 |

## Summary results for selected studies

The systematic search identified 31 studies (24 case-crossover studies and 8 time series studies) that fulfilled the inclusion and exclusion criteria. Eleven studies investigated the relationship between the common air pollutants (PM10,PM2.5, carbon monoxide, sulphur dioxide, nitrogen dioxide and ozone) and myocardial infarction, acute coronary syndrome, ST-elevation myocardial infarction admissions, 6 studies investigated the effects of PM2.5 and acute myocardial infarction, myocardial infarction, ST-elevation myocardial infarction admissions, 2 studies investigated the relationship between PM10, PM2.5 and acute coronary syndrome hospital admissions, 2 studies investigated the relationship between ozone and myocardial infarction hospital admissions, 1 study investigated the relationship between PM10, nitrogen dioxide, carbon monoxide and acute myocardial infarction hospitalisations, 1 study investigated the relationship between PM2.5, carbon monoxide, nitrogen dioxide, sulphur dioxide and ST-elevation myocardial infarction hospital admissions, 1 study investigated the relationship between PM2.5, nitrogen dioxide, ozone and myocardial infarction emergency room visits, 1 study investigated the relationship between PM10, PM2.5, O3, NO2 and ST-elevation myocardial infarction hospitalisations, 1 study investigated the relationship between PM10, nitrogen dioxide, ozone and myocardial infarction emergency visits/admissions, 1 study investigated the relationship between nitrogen dioxide and myocardial infarction hospital admissions, 1 study investigated the relationship between PM10, carbon monoxide and acute coronary syndrome hospital admissions, 1 study investigated the relationship between PM10, PM2.5, ozone, black smoke and ST-elevation myocardial infarction admissions, 1 study investigated the relationship between sulphur dioxide, nitrogen dioxide, suspended particle matter and acute coronary syndrome admissions, and 1 study investigated the relationship between PM10, carbon monoxide, nitrogen dioxide, sulphur dioxide and acute myocardial infarction admissions. These studies accounted for more than 500, 000 myocardial infarction patients. The lag time varied from 0 to 24 hours and 0 to 7 days. table 4 shows the summaries and results from each study included in the meta-analysis.

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# Chapter 5: Discussions

## Introduction

Despite the growing epidemiological research and evidence on the health impact of air pollution for the past two decades, there has been very little research undertaken in Fiji and surprisingly, none has been done in Lautoka. This thesis will be the first study to examine the ambient air quality in the Lautoka Central Business District and its potential health impacts, specifically, risk of acute myocardial infarction. To achieve this, the principle objectives that were developed are:

1. Determining the ambient air quality in the Lautoka CBD and its potential health implications
2. Conducting a meta-analysis on the association between ambient air quality and risk of hospitalization due to myocardial infarction
3. On the basis of the meta-analysis and air quality data, I will develop predictive models on the risk of hospitalization due to acute myocardial infarction for Lautoka.

Significant findings from the results are discussed in relation to the three principle objectives and in the context of international research and literature. Limitations of this research and future directions are also elaborated herein.

## Air Quality in the Lautoka CBD and heart disease

### Particulate Matter (PM10 and PM2.5)

This study showed that particulate matter (PM) levels, in particular PM10, was quite significant in the ambient air quality data gathered from the Lautoka Central Business District. The recommended 24hr mean concentrations for particulate matter with an aerodynamic diameter < 10microns (PM10) is 50g/m3(WHO, 2006). The daily mean PM10 concentrations recorded for the Lautoka CBD was 73.42g/m3 which was in excess of 23.42g/m3when compared with the WHO recommended guideline. Despite this high concentrations , the WHO’s Global Health Observatory (GHO) data reported that for urban areas mean concentrations of PM10 are expected to be within the range of less than 10g/m3to over 200g/m3. In addition, in a typical day (hours of the day) the analysed data observed an increase in PM10concentrations in the morning commencing after 5 am and was at its peak between 6am to 8am and in the afternoon PM10 concentrations started to increase exponentially after 3pm and peaked at 8pm before decreasing again(ref to Fig. 1). The daily mean PM2.5concentrations observed for Lautoka CBD during this study was 10.87 g/m3 but was well below the recommended WHO-24hr mean air quality guideline of 25 g/m3. In comparison, the WHO’s Global Health Observatory (WHO, 2018) data reported that the urban mean concentrations of fine particulate matter (PM2.5) in Fiji is 10.5g/m3and the expected range of PM2.5concentrations in urban areas is between less than 10g/m3to over 100g/m3. In a typical day (24 hours), it was observed that an increase in  PM2.5concentrations  occurred after 5am and was at its peak at 8am. Another increase in PM2.5 concentrations occurred in the afternoon starting at 4 pm and was at its peak at 8m before decreasing (refer to Fig 2). The two periods (morning and afternoon) whereby high concentrations of particulate matter (PM10and PM2.5) occurred are when the volume of vehicles (including buses and trucks) are at its highest in the city and this is a norm for most urban centres in Fiji. In these two periods, buses, trucks, other public transport and private vehicles converge into the city to pick and drop workers and students which could result in high particulate emission from these vehicles. Pearson’s correlation also indicated a strong (positive) linear relationship between the two pollutants suggesting the existence of multiple sources apart from vehicle emissions. In terms of the pollutant sources and volume emitted, the Department of Environment in Fiji in 2013 had generally agreed that emissions from vehicles were the major source of air pollution in Fiji.

Fine particles, namely PM2.5 are commonly associated with emissions from combustion or secondarily formed aerosol particles, as opposed to coarse particles ( < 10microns) PM10which is commonly associated with wind-blown dust, crushing and grinding actions or resuspension by vehicle movement(WHO, 2009). Findings from Studies by (Kleeman, Schauer, & Cass, 2000; <https://europepmc.org/abstract/med/8899908\textit{{Characterization} of fuel and aftertreatment device effects on diesel emissions. - Abstract - Europe PMC}>, 1996) concurred with this statement and their studies found that motor vehicles are among the major contributors of particulate matter emissions in the urban atmosphere. In comparison, major cities in the neighbouring countries of Australia and New Zealand have maintained low levels of PM10and PM2.5 (Barnett et al., 2005). This is despite the fact that these cities have larger populations, high number of vehicles and a thriving agricultural and industrial economy. Vehicle emissions along roads in urban areas are released in close proximity to human receptors, giving reduce opportunity for the atmosphere to dilute the emissions. Furthermore, concentrations of pollutants are significantly enhanced by the fact that many roads in cities have buildings alongside. As a result, these building reduces the effect of wind speed at the source of emissions by as much as an order of magnitude relative to that on an open road(Colvile, Hutchinson, & Warren, 2002). This observations by Colvile and others on road traffic and effects of urban air quality on human health is consistent with the layout of the Lautoka CBD, hence, the exposure methods of the public to air contaminants would be quite similar.

The decrease in particulate pollution in between the peak periods for the Lautoka city can be attributed to the low volume of traffic movement. This is also the time when most of the workers and students are at their workplaces or in schools respectively. Despite this low volume of traffic PM10concentrations were still high (30g/m3 - 70g/m3) late into the night and in the early hours of the morning. This could be attributed to other potential particulate sources of  notably from power generation, industrial combustion, wind blown pollens , agricultural activities and from open burning of wastes (Brook et al., 2004). Suffice to note that the duration of this study coincided with the sugar cane crushing season in Lautoka. In the crushing season, operation of the mill is on a 24 hour basis unless there is a mechanical breakdown. Apart from the emissions from the operation of the sugar mill, the burning of sugar cane farms to alleviate access for harvesting and to reduce volume of waste material remains an issue of concern in Fiji. In a study by (Mnatzaganian, Pellegrin, Miyamura, Valencia, & Pang, 2015) on the association between sugar cane burning and acute respiratory illnesses, the authors found a significant association between the incidence of respiratory distress  and the burning of sugar cane farms. From their findings, Mnatzaganian and others concluded that the more sugar cane farms that are burnt the higher the incidence of respiratory distress are likely to occur.

The health implications of particulate air pollution has been consistently shown in epidemiological studies.  Not only does these studies show an  association between particulate air pollution and the exacerbation of illnesses in people with respiratory disease but also increases in the number of deaths from cardiovascular and respiratory disease among the elderly(Seaton, Godden, MacNee, & Donaldson, 1995). High ambient particulate (PM10 and PM2.5) pollution have been associated with mortality and hospital admissions due to respiratory and cardiovascular diseases in both short-term and long-term studies(Brunekreef & Holgate, 2002). Epidemiological studies from around the world have consistently demonstrated that both short-term and long-term exposures to particulate matter are associated with a host of cardiovascular diseases, including myocardial infarction, heart failure, arrhythmias, strokes and increased cardiovascular mortality. Significantly, evidence from cellular/ toxicological experiments, animal and human exposures and human panel studies have indicated several mechanisms by which particle exposure may trigger acute events as well as expedite the chronic development of cardiovascular diseases(Brook, 2008).

### Ozone (O3)

The mean concentrations for the air pollutant ozone during the study period was very minimal and well below the WHO guideline of 100g/m3 (8-hour mean) (WHO, 2006) . Even though the concentrations of O3as negligible, the observations made on the mean concentrations within the hours of a day was characteristic of its nature. Ground-level ozone is a secondary pollutant which is created by chemical reactions between oxides of nitrogen and volatile organic compounds (VOC) in the presence of sunlight. It reaches unhealthy levels on hot sunny days in urban environments(USEPA, n.d.). In addition, it is usually at the highest concentration in the afternoon or early evening(NSW\_Health, n.d.). This phenomenon was observed in this study when ozone concentrations were at its peak in the midday between 11am and 12pm (refer to Figure 5). In the afternoon, ozone concentrations increased again between 4pm and 6pm. Temperature levels were also high during the ozone peak periods suggesting a relationship between the two variables. Moreover, Pearson’s correlation validated the existence of a linear (positive) relationship between the two variables, r = 0.53 and suggests that meteorological parameters like temperature can influence the concentrations of ozone. Apart from causing respiratory diseases, recent evidence have linked O3 with cardiovascular diseases as well. For example, the study by (Bejot et al., 2011) investigated the effects of shot-term exposure to O3 and its impact on ischemic heart and cerebrovascular disease. This case crossover study compared daily levels of urban O3pollution and the incidence of first-ever, recurrent, fatal and non-fatal ischemic cerebro-vascular events (ICVE) and myocardial infarction. The authors of this study observed that apart from ICVE, there was a marginally significant association between low levels of O3 and myocardial infarction with 1-day lag, OR=1.147(95%CI: 0.999-1.318). In addition, when adjusting for confounders like hypercholesterolemia an association between myocardial infarction and O3 was observed, OR=1.111(95%CI: 1.020-1.211). From these findings, the authors concluded and suggested that recurrent Myocardial Infarction could be triggered by short-term exposure to even low O3concentrations, specifically for individuals with severe vascular risk factors. Other studies such as that by (Cheng, Tsai, & Yang, 2009) where they investigated the association between air pollutant levels and in creased hospital admissions for myocardial infarction in a tropical city (Kaohsiung) in Taiwan yielded similar findings. Cheng and colleagues observed that on warm days (>25oC) concentrations of O3 were positively associated with increased daily hospitalizations due to myocardial infarction. The authors of this study concluded that increased concentrations of pollutants such as O3 increases the risk of higher frequency of hospital admissions due to myocardial infarction. The findings from these studies suggest that even in low concentrations, short-term exposure to O3 has the potential to cause adverse effects on the heart leading to high hospitalizations.

### Carbon monoxide (CO)

The average concentrations for the air pollutant carbon monoxide in this study was similar to ozone. WHO does not provide guideline values for CO but its is safe to say that the mean CO concentrations in the Lautoka CBD was very minimal when comparing it with the USEPA standards (USEPA, 2018). The primary sources of carbon monoxide in the ambient air around urban areas are commonly from cars, trucks and other vehicles or machinery that burn fossil fuels. Carbon monoxide binds with haemoglobin with an affinity more than oxygen to form carboxyhemoglobin in the red blood cells and interferes with the release of oxygen at tissue level(Brook et al., 2004). Even though the carbon monoxide concentrations observed during this study was very low, it is imperative to understand the impact it may have on the cardiovascular health of the Lautoka populace. For instance, in a study by (Allred et al., 1989) where they investigated the short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease, they observed a decrease of 5.1%(90CI:1.5 to 8.7%; p =0.02) and a decrease of 12.1%(90%CI:9.0-15.3%; p<0.000-1) in the length time to a threshold ischemic ST-segment change (ST end point) after carbon monoxide exposures that produced carboxyhemoglobin levels of 2% and 3.9% respectively. In the addition, the time for the onset of angina decreased by 4.2% at the 2% carboxyhemoglobin level and by 7.1% at the 3% carboxyhemoglobin level. Moreover, Allred and others concluded that low concentrations of carboxyhemoglobin aggravates myocardial ischemia during exercise in subjects with coronary heart disease. A similar study was conducted by (ANDERSON, 1973) whereby the author investigated the cardiovascular effects of exposure to low concentrations of CO on 10 men with stable angina pectoris. In this study the participants initially breathed air, then breathed air  with 50ppm CO or 100 ppm CO for  4hours on 5 consecutive days. Once they have been exposed to breathing CO a standard treadmill exercise ECG was recorded and the time of onset and duration of pain was recorded. The results of this study showed that the average carboxyhemoglobin (COHb) increased after breathing air with 50ppm CO (1.3% rise) and a 4.5% rise after 100ppm CO. In addition, there was no prolonged pain after breathing air with 50ppm CO but a duration of pain was significantly continuous after breathing air with 100ppm carbon monoxide. Findings from this study suggested that low levels of carbon monoxide can cause decrease exercise tolerance and worsening of myocardial ischemia in patients with angina pectoris. In another study by (Stieb, Szyszkowicz, Rowe, & Leech, 2009), the authors examined the associations  between common air pollutants and respiratory or cardiac conditions for cities in Canada. Stieb and colleagues observed that an increase in 0.7ppm CO was associated with an 2.6% increased hospital visits due to myocardial infarction/angina. From these findings, the authors concluded that daily mean levels of CO and NO2 was most consistent with emergency department visits for cardiac conditions.

### Nitrogen dioxide

Nitrogen dioxide concentrations in the Lautoka CBD was similar to that observed for carbon monoxide and ground level-ozone in that it was very low. Despite the low levels of NO2, its occurrence indicates emissions from motor vehicles and nearby industrial processes around Lautoka City. As a developing nation, understanding these emissions sources and how it may impact the cardiovascular health of its populace is warranted. Recent epidemiological studies have consistently shown the existence of relationship between NO2and increased hospital admissions due to heart disease. In a study by (Vencloviene, Grazuleviciene, Babarskiene, Dedele, & Grazulevicius, 2011), the authors investigated whether geomagnetic activity has an effect on the association between short-term exposure to NO2and emergency hospitalization for acute coronary syndrome (ACS). Vencloviene and colleagues observed that for patients under 65 years of age, an interquartile range increase (IQR) in ambient NO2 pollution on the day of admissions and previous day (lag 0-1 day) increases the risk of acute coronary syndrome by 24%(95%CI:0.96-1.6). From these findings, the authors concluded that variations in geomagnetic activity may increases the effect of traffic related air pollution on acute coronary syndrome.

### Sulphur dioxide

The sulphur dioxide levels  in the Lautoka CBD was also well below the guidelines established by WHO (refer to Table??). However, despite these low concentrations, epidemiological studies have shown that short-term exposure to SO2 has an impact on the cardiovascular health. This was observed in the study by (Liu et al., 2017) where the authors conducted a time-stratified case crossover study in 14 large Chinese cities to examine the association of short term exposure to air pollution and daily admissions due to acute myocardial infarction. In this study Liu and colleagues observed that an interquartile range (IQR) increase in sulphur dioxide on lag - 2 days was significantly associated with a 2% increase in acute myocardial infarction hospital admissions. The authors of this study concluded that people who live in these 14 cities are at an increased risk of acute myocardial infarction hospitalizations if they are being exposed to SO2 even for a short-term. Moreover, Liu and others even suggested that the findings from this study can be applicable to developing countries as well.

### Weather variables (Temperature, Relative humidity and Atmospheric Pressure)

The weather data gathered during this study is typical of the day to day weather for locations on the western side of Viti Levu in Fiji.  Since Lautoka CBD lies adjacent to the sea (refer to Figure 2), influences to the weather is mostly due to the surrounding ocean. In essence, Fiji weather is best described as tropical. This study coincided with the cooler season (May to November) persisting in Fiji with an average daytime temperature of just over 27oC. Environmental triggers of heart diseases have been under-recognized but have gained significant interest in the past decade(Pope et al., 1995). Of the three weather variables measured, ambient temperature has been consistently discussed in epidemiological studies to be associated with heart related diseases. For instance, in a study by (Claeys et al., 2015), the authors assessed the independent environmental triggers ( air pollutants and weather variables) of acute myocardial infarction and to identify which sub-population are more vulnerable to these triggers. Claeys and colleagues observed that for temperature was significantly correlated with STEMI in that for every 10oC decrease in temperature an 8% increase in the risk of  STEMI was apparent (adjusted incidence risk ratio (IRR) 0.92(95%CI: 0.89-0.96). From these findings, Claeys and others suggested that amongst the environmental triggers of STEMI, low temperatures was the most important. Other findings on the effect of weather variables on cardiovascular health was observed in 2 other epidemiological study by  (Royé, Zarrabeitia, Fdez-Arroyabe, Álvarez Gutiérrez, & Santurtún, 2018) and (Hori, Hashizume, Tsuda, Tsukahara, & Nomiyama, 2012). In the time series study by Roye and colleagues, they examined the relationship between ambient temperature and acute myocardial infarction hospital admissions in Cantabria, Spain. They observed an inverse but significant relationship between low ambient temperature and the number of hospital admissions due to acute myocardial infarction. From these findings, Roye and others suggested that environmental factors such as  ambient temperature makes an important contribution to mortality due to acute myocardial infarction. In addition to examining the effects of air pollutants, Hori and colleagues also employed a time series study to explore the effects of weather variables namely, air pressure and ambient temperature on the emergency admissions due to stroke and cardiovascular disease. They observed  that for every 1oC increase in ambient temperature there was a 7.83% increase in the daily admissions  for acute coronary syndrome  and heart failure, (95%CI: 2.06-13.25. They also observed that every 1hPa decrease in air pressure was associated with a 3.56%increase in emergency admissions due to heart failure, 95%CI:1.09-5.96). Findings from these studies suggests that both an increase and decrease in ambient temperature was associated to an adverse cardiovascular effect

(Che et al., 2014)

# Identification and characteristics of included study

A total of 21 case crossover studies were included in this meta-analysis  and there descriptions are as follows: The study by (Xiaodong et al., 2015) assessed the variations in acute myocardial infarction (AMI) hospital admissions  relative to air pollutant levels between 1st November 2013 and 27 April 2014. The pollutants included in the study are PM2.5,PM10, NO2,SO2, and CO whilst the definition of AMI admissions were consistent with the International Classification of Diseases, 9th revision (ICD-9). After imputing the 24 hour means for each pollutant, conditional logistic regression was used to calculate the exposure  effect and the effects estimate expressed as Odds Ratio (OR) per 50g/m3 3of each pollutant with pollutant with weights equal to the number of hospitalisations on that day. Xiaodong and colleagues observed that urban background levels of PM10, PM2.5 and CO were significantly associated with an increased risk in AMI. The study by (Weichenthal et al., 2017)  examined if hospital admissions for myocardial infarction (MI) was associated with short-term changes in outdoor PM2.5. The study was conducted in 3 regions in British Columbia, Canada between 2008 and 2015 with a total of 2,881 myocardial infarction cases. Age and sex of participants were stratified to determine the relationship between PM2.5and MI.  Conditional logistic regression was employed to describe the relationship between the PM2.5concentrations and the risk of MI. Adjustments were made for mean ambient temperature (lag0-3day). Weichenthal and colleagues observed that each 5g/m3of 3-day average PM2 was associated with an increased risk of MI; the authors concluded that that among  elderly patients a short-term  change in outdoor PM2.5 concentrations increases the risk of MI. The study by(Evans et al., 2016) examined the onset of STEMI after exposure to wood smoke and traffic pollution. This study was conducted in Monroe County, New York and included  a total of 362 STEMI patients between 1stJanuary 2007 to 30 September 2012. A time-stratified approach was employed that was similar to other case-crossover studies. Conditional logistic regression was employed to determine the relative odds of STEMI with interquartile increases in pollutant concentrations (lag0-72hr means). Adjustments were made for seasons (colder and warmer months), temperature and relative humidity. Evans and colleagues observed that for each 7.1g/m3increases I  increases in PM2.5increased the odds of STEMI. In addition, the authors also observed that increases in ozone and carbon monoxide concentrations in the previous hour also increased the odds of STEMIs. The case-crossover study by (Sahlén et al., 2019) examined the association of short-term risk of STEMI after exposure to  of air pollutants (NO2, SO2, O3, PM2.52.5, PM10). The study was conducted in Stockholm, Sweden from January 2000 - June 2014  with a total of  14, STEMI patients who are included in patients registered in SWEDEHEART (Swedish Web System for Enhancement and Development of Evidence Based Care in Heart Disease Evaluated According to Recommended Therapies). After imputing the averages of pollutant concentrations  2 weeks before and after exposure, conditional logistic regression was employed to analyse (lag0-24hrs) before hospital admissions. Multivariate single-pollutant models was also built with adjustments made for atmospheric pressure, precipitation relative humidity, wind velocity and temperature. Sahlen and colleagues observed that the risk of STEMI with NO2was strongest at 15-lag and PM2.5was strongest at 20-hr lag. From these findings Sahlen and others suggested that the risk of STEMI increases within hours of exposure to air pollutants. The study by (Rasche et al., 2018) examined the rapid changes in pollutant (NO2, NOx, O3, PM10) concentrations and the risk of MI. The study was conducted in the city of Jena in Germany from 1stJanuary 2003 to 31stDecember 2010 with a total of 693 MI cases.  A lag time of 0-4days was employed in the generalized linear mixed models (GLMM) to determine the risk of MI and environmental data. Multivariate analysis was also conducted with adjustments made for ambient air temperature, relative humidity, and atmospheric pressure. Rasche and colleagues observed that in the overall population, an increase of NOxby more than 20g/m3 and 8-20g/m3 increases the risk for MI by up to 121% (lag2-3days). In addition, the risk of MI in the overall population was also associated with the rapid changes of ambient NO2 concentrations whilst the risk  of MI increased by 73% with a lag of 1 day. Moreover, similar associations was observed in almost all subgroup analysis. From these findings Rasche and others suggested that the magnitude of increases in nitrogen oxide levels may be an independent risk factor for MI.  The study by(Rich et al., 2013) examined whether the fine particles (PM2.5) enriched with secondary species (sulphates, nitrates, elemental carbon, organic carbon and ammonium) enhanced the triggering of myocardial infarction (MI) in New Jersey, USA. Rich and colleagues used the time stratified case-crossover approach to make an approximation on the risk of transmural infarction increased tapered element oscillating microbalance (TEOM) levels in the 24 hours before emergency department arrival. Pearson’s correlation was employed to determine the relationship between the variables and conditional logistic regression employed to estimate the risk of MI with increased PM2.5 concentration with each 10g/m3increase in TEOM PM2.5 levels.. concentration with every . The authors observed  a larger relative odds of MI  when PM2.5 was laden with species (sulphates, nitrates, and organics) and  suggested that air pollution mixtures these days are enhanced by pollutants formed through atmospheric chemistry.(Gardner et al., 2014) investigated whether increased levels of ambient fine particulate pollution (PM2.5) in the previous 24 hours was associated with an increased risk in STEMI. The study was conducted in Rochester, New York for the period 1stJanuary 2007 to 31stJanuary 2010. The study population included all STEMI and non-STEMI (NSTEMI) patients who presented to the University of Rochester Medical Center (URMC).Conditional logistic regression was used to examine the association between increased PM2.5concentrations and risk of STEM or NSTEMI; analysis also accounted for shorter time lags (lag0-11hrs), longer time lags (lag0-95hrs)(lag0-11hrs) and co-morbidities (hypertension, dyslipidemia, diabetes and low-left ventricular ejection fraction). Adjustments was also made for mean temperature and mean relative humidity in the previous 3 hours. Gardener and colleagues observed that each 7.1g/m3increase in PM2.5concentrations was associated with an 18% increase in the risk of STEMI. Hypertensive patients were at a greater risk of STEMI after exposure to fine particulates in the previous hour than those non-hypertensive patients. Gardener and others concluded that pre-existing conditions such as hypertension puts and individual at a greater risk of STEMI when exposed to increased fine particle concentrations.(Argacha et al., 2016) examined the relationship between exposure to common air pollutants such as PM10, PM2.5, O3 and NO2 and hospital admissions due to STEMI. Significant comorbidities that may increase the risk of STEMI were also assessed. Conditional logistic regression was employed to analyse the association between air pollution and the risk of STEMI with a 0-day lag time between exposures with the outcome expressed as odds ratios. Risk estimates were calculated for each 10g/m3 increase in air pollutants and10oC increase in ambient temperature. Adjustments were made for ambient temperature and day of the week. Argacha and colleagues observed that the odds for an increase in STEMI was associated with every 10g/m3increase in PM10, PM2.5, NO2 concentrations. The elderly (>75yrs) was more at risk of STEMI. No effect was observed for O3.These findings prompted the authors to suggest that elderly people were more at risk of STEMI when exposed to PM.(Pope et al., 2015) investigated the effects of increased  short-term exposure to PM2.5and acute coronary syndrome (ACS)occurrences including STEMI, NSTEMI and unstable angina. The study was conducted in Utah’s Wasatch Front, USA for all patients who received coronary angiography between 10thSeptember 1993 to 15th May 2014. Conditional logistic regression was initially used to determine the relationship between exposure to PM2.5and ACS occurrences with the effect measures expressed as odds ratios per 10g/m3 increments of PM2.5. Adjustments were made for minimum temperature, dew point temperature and barometric pressure. Pope and colleagues observed that the Odds for ST-segment elevation myocardial infarction increased with each 10g/m3increase in concurrent-day PM2.5. From these observations, Pope and other suggested that the triggering of acute coronary events such as ST-segment elevation myocardial infarction may be attributed to increased exposure to fine particulate matter.(Chiu, Weng, Chiu, & Yang, 2017) investigated the short-term effects of ambient O3 on the daily hospitalizations of  MI. The study was conducted in Taipei, Taiwan for all MI (ICD9) patients registered under the Bureau of National Health Insurance over a 5 year period between 2006 to 2010. A time-stratified approach was used in this case-crossover study with the stratification of time into separate months to identify referent days falling on the same day of the week within the same month as the index day. A cumulative lag period of up to 2 previous days  (i.e. average air pollutant concentrations of the same and previous 2 days). Conditional logistic regression was used to explore the association between the ambient concentrations of O3 and the number of MI hospitalizations. Estimates of the associations was expressed as odds ratios and 95% confidence intervals with weights equal to the number of hospitalizations on that day. Adjustments was also made for ambient temperature, relative humidity and barometric pressure. Chiu and colleagues observed that in single-pollutant models, the increase in MI admissions was significantly associated with O3on both cold (<23oC) and warm (>23oC) days and an increase in MI admissions respectively. In 2 pollutant models MI admissions increased significantly on cool days. From these findings, Chiu and others suggested that short-term exposure to O3concentrations can increase the MI hospital admissions on both warm and cool days. (Liu et al., 2018) examined the effects of short-term exposure to PM2.5on MI hospital admissions. The study was conducted in 26 Chinese cities and STEMI hospital admissions for these cities were obtained from the Hospitalization Summary Reports for the period 1st January 2014 to 31stDecember 2015. Using the time -stratified case crossover design, the association between pollutant levels and STEMI hospitalizations was analysed using conditional logistic regression. A1-day lag was used in the analysis to control for the non-linear delayed effects temperature. To evaluate temporal associations, the models were fitted with different lag structures from the current day (lag0) through 5 lag days (lag5). To account for the fact that single-day models may underestimate the association, the associations were estimated with 3-day (lag0-2) and 6-day(lag0-5) moving averages of PM2.5levels. Adjustments were made for  other pollutants such as CO, NO2, SO2and public holidays. Liu and colleagues observed that an interquartile range (IQR) increase in PM2.5concentrations (47.5g/m3) at lags 2, 3, 4 and 0-5 days corresponded with 0.6% (95%CI:0.1 -1.1%), 0.8(95%CI:0.3-1.3%), 0.6%(95%CI:0.1-1.1%) and 0.9%(95%CI:0-1.8%) increases in STEMI admissions. Liu and others concluded that short-term increases in PM2.5may increase the risk of STEMI hospitalizations. (Chang, Kuo, Liou, & Yang, 2013) evaluated the short-term effect of exposure to PM2.5and MI (ICD9) hospitalizations. The study was conducted for individuals residing in Taipei city over a 5 year period, from 2006 to 2010. A time-stratified approach was used in this case-crossover study with the stratification of time into separate months to identify referent days falling on the same day of the week within the same month as the index day. A cumulative lag period of up to 2 previous days (i.e. average air pollutant concentrations of the same and previous 2 days). Conditional logistic regression was used to explore the association between the ambient concentrations  of PM2.5 and the number of MI hospital admissions. Estimates of the associations was expressed as odds ratios and 95% confidence interval with weights equal to the number of hospitalizations on that day. Adjustments was also made for daily average temperature, humidity and barometric pressure as well as for warm (>23oC) and cool days (<23o). Chang and colleagues observed that for single pollutant models increased MI admissions was significantly associated with an interquartile range increase (IQR) of PM2.5concentrations(17.46g/m3) on both cool (OR=1.05 (95%CI: 1.01-1.09) and warm (OR=1.10(95%CI:1.06-1.15)days. From these findings, the authors suggests that increased levels of PM2.5increases the risk of MI hospitalizations. (Hsieh, Yang, Wu, & Yang, 2010) examined the relationship between ambient pollutant levels and MI hospitalizations in Taipei city, Taiwan over an 11 year period from 1996 to 2006. A time-stratified approach was used in this case-crossover study.(Cheng, Tsai, & Yang, 2009) evaluated the relationship between concentrations of ambient air pollutants (SO2, PM10, NO2, CO, O3) and MI hospitalizations among individuals who live in Kaosiung over an 11 year period from 1996 to 2006. (Bejot et al., 2011) investigated the short-term effects of exposure to ozone on ischemic heart and cerebrovascular disease in Dijon, France from 2001 to 2007. Using a bi-directional case-crossover design analysis daily concentrations of urban O3 was compared with first-ever, recurrent, fatal and non-fatal ischemic cerebro-vascular events (ICVE) and myocardial infarction. Concentrations of SO2, NO2, CO and PM10were used to create bi-pollutant models. Using multivariate logistic modelling, the effects of O3exposure were calculated for every 10g/m3increase in pollutants. Adjustments was also done for all possible confounders such as weather variables and hypercholesterolemnia. Bejot and colleagues observed that in stratified analysis, O3was associated with MI incidence when hypercholesterolemnia was present, OR = 1.111(95%CI: 1.020-1.211) and the strength of the associations increased with increasing number of combined factors. Bejot and others deduced from these findings that even exposure to low concentrations of O3 can trigger MI especially among individuals with severe vascular risk factors. (Zhang et al., 2016) investigated the relationship between ambient air pollution and emergency department visits due to acute myocardial infarction in Chaoyang District, Beijing, China in 2014. A time-stratified approach was used in this case-crossover study with a lag 0-5days. PM concentrations were compared during the period of patients experiencing AMI (case period) with the times not experiencing AMI (control period). Relative risk of AMI was estimated by comparing PM exposure during case periods and control periods. Adjustments were made for temperature and relative humidity using a natural smooth spline with 3 degrees of freedom in each model. Conditional logistic regression modelling was used to estimate the association between exposure to air pollutants and acute myocardial infarction outcomes. Odds Ratios and 95% confidence intervals scaled to every 10 g/m3change of particulate matter concentrations was presented as risk of AMI, STEMI and NSTEMI. Single and multiple pollutant models was used to determine the independent/combined effects of air pollutants on AMI outcomes. Zhang and colleagues observed that each 10g/m3 3increases in models was used to determine the independent/combined effects of air pollutants on AMI outcomes. Zhang and colleagues observed that each 10g/m3increases. (Hopke et al., 2015) investigated whether fine particulate (PM2.5) emissions from different upwind origins were associated with cardiovascular effects among residents in Rochester, New York from 1st January 2007 to 31stDecember 2010. A time-stratified approach was used in this case-crossover study by contrasting pollutant levels prior to the acute coronary syndrome (case-period) to other periods when the subject did not have an acute coronary syndrome, matched to the case-period by month, weekday, and hour of the day (control periods). To calculate each hourly air mass location for the 24hrs prior to each case or control time period, NOAA hybrid single-particle lagrangian trajectory (HYSPLIT) was employed. HYSPLIT is a system for computing simple air parcel trajectories, dispersion and deposition simulations. Conditional logistic regression was used to examine whether PM2.5/STEMI associated was altered by whether the air mass passed through each of the 8 cardinal wind direction (ENE,ESE,SSE,SSE,SSW,WNW, WSW, NNW) sectors in the previous 24hours. Adjustments were made for mean temperature and mean relative humidity. Hopke and colleagues noted that when air passed through the West-Southwest (WSW) direction anytime in the last 24hours, a statistically significant association between STEMI with each 7.1g/m3increase in PM2.5 concentrations in the previous hour was observed, OR=1.27(95%CI: 1.08-1.22). In addition, the relative odds were largest when the time spent in the WSW was between 8-16hours. From these findings Hopke and others suggest that fine particles from the WSW direction are more potent in triggering STEMIs and that the direction of wind is related to emissions from coal-fired power plants and other industrial sources of the Ohio river valley.(Nuvolone et al., 2011) examined the relationship between air pollutants (PM10, NO2, CO) and AMI hospitalizations in Tuscany, Italy for the period January 2002 to December 2005. Nuvulone and colleagues used an area-specific case-crossover approach whereby the authors identifies cases and compares each subject’s exposure in a time period just before a case event (the case period) with the subject’s exposure at other times (control periods). This study design adjusts for individual characteristics that vary over time such as age, gender, and body mass index (BMI). Conditional logistic regression models were constructed to impute the odds ratios and their 95% confidence intervals. Adjustments were made for variables such as  apparent temperature influenza, epidemics and population decreases during vacation periods holidays. To impute immediate and delayed effects different lag patterns were considered using single day lags from lag0 (current day concentration) to lag5(5 days before the event day). Cumulative lags was also considered by imputing the  average between the same day and the previous 5 days (lag0-5) and the average between the previous 3 days and the previous 5 days (lag3-5). Finally, pooled estimates were obtained from the random effects meta-analysis. Nuvolone and others observed that all the pollutants measured were associated with AMI hospitalization with a meta-analytical odds ratio at lag2, 1.013(95%CI: 1.000-1.026) for PM10, 1.022 (95%CI:1.004-1.041) for NO2, and 1.007(95%CI: 1.002-1.013). The findings from this study suggests that AMI onset is associated with short-term exposure to air pollutants and that groups like the elderly (>75yrs), females, older patients with hypertension and chronic obstructive pulmonary disease are more vulnerable.(Sedgwick, 2013). The study by(Lin et al., 2013) evaluated the role of gaseous pollutants  on AMI hospitalization and mortality in Hong Kong over a 13 year period from 1998 to 2010. Using a time stratified case-crossover design the cases and controls were matched by the day of the week to account for any potential weekly patterns with a 1 day lag.  Adjustments was made for temperature, relative humidity and public holidays as confounding factors. Lin and colleagues observed that an in single pollutant models the risk of AMI mortality increased by 4.55% with every interquartile (IQR) increase in NO2 concentrations. AMI mortality also increased by 2.56% for every interquartile increase in SO2 concentrations. These findings suggest that in Hong Kong elevated concentrations of SO2 and NOincreases AMI related mortality. Finally the study by (Matsukawa et al., 2014) evaluated the impact of Asian dust (AD) and the incidence of acute myocardial infarction in patients with coronary heart diseases. The study participants were patients admitted into 4 hospitals in the Fukuoka prefecture, Japan from 2003 to 2010 . Matsukawa and colleagues used the time-stratified case crossover approach with the case period defined as the day of admission and control periods are the days of the week in the same month of the same year as the case period. Single lag effect was employed (0-5days) and cumulative lag ( days 0-1, days 0-5) to account for the persistence of AD, suspended particulate matter (SPM), NO2 and SO2 events over a few days. Conditional logistic regressions was employed to examine the association of exposure to AD, SPM, NO2 and SO2the occurrence of AMI. Stratified analysis according to age strata was also performed to examine the effect modification. Matsukawa and colleagues observed significant associations between AD and the incidence of AMI. In addition, significant associations was observed when AD was defined by the SPM (PM<7microns) and the risk of AMI hospitalisation. These findings suggest that exposure to AD prior to the onset of symptoms is associated with the incidence of AMI

## Overall analysis of systematic review and meta-analysis on the association between exposure to air pollutants and risk of AMI hospitalization

Recent epidemiological studies have reported the relationship between exposure to ambient air pollution and risk of hospitalizations due to acute myocardial infarction. Of the common air pollutants ambient particulate matter has gained wide concern, especially PM2.5 (Luo et al., 2015). In this systematic review and meta-analysis, 20 case-crossover studies was identified that strictly met the inclusion criteria from 2008 to 2018. The included studies provided epidemiological evidence from 137,846 participants overall. Included studies exhibited effect estimates that had a positive association between exposure to ambient air pollution and risk of  acute myocardial infarction hospital admissions or emergency department visits. The overall analysis of these studies showed a high degree of heterogeneity, I2 = 77%. Although this meta-analysis observed a high degree of heterogeneity, a meta-analysis study by (Cai, Li, Scott, Li, & Tang, 2016) suggest that such heterogeneity could be attributed to location of study and lag time used. The location of the selected studies and their different lag times are provided in **Table 6**whichshows varying lag times and locations. From the studies included in the meta-analysis, most number (5) of studies were from the United States of America, followed by Taiwan with 4 studies, 2 studies were from China and Sweden, and a study each from Japan, Italy, Canada, Belgium, France and Germany.  In addition, other possible reasons for the high degree of heterogeneity as discussed by Cai and colleagues could be attributed to the selection of only studies with significant associations as well as differences in average particulate matter.

When compared with the overall pooled estimates, the results obtained from the two subgroup analyses were basically similar in that there was still significant association between exposure to air pollutants and risk of AMI hospitalisation. However, when quantifying the level of heterogeneity, the degree of heterogeneity decreased from high in the overall analyses to moderate heterogeneity in both the subgroup analyses.

In the subgroup analysis on the risk of AMI hospitalization with short-term exposure to at least 5-10g/m3 increments of PM2.5was statistically significant; based on the random-effects meta-analysis model, a combined Odds Ratio (OR) of 1.06(95%CI: 1.03-1.09), p<0.0001 was observed. A moderate degree of heterogeneity was detected, I2 = 43% with evidence of publication bias.  Similarly, the subgroup analysis on short-term exposure to common air pollutants and risk of AMI hospitalizations in Western Pacific  based on the random-effects analysis model was also statistically significant with combined Odds Ratio (OR) = 1.05(95%CI: 1.03-1.08), p<0.0001. A moderate degree of heterogeneity was also observed, I2 = 39%.  Findings consistent with this study was observed by

In summary, based on the systematic review and meta-analysis of the 20 case-crossover studies, the pooled effect estimates suggests that short-term exposure to air pollutants in particular PM2.5was associated with AMI hospitalizations and emergency department visits. In addition the subgroup the analyses suggests that exposure to at least 5-10g/m3of PM2.5was significantly associated with an increased risk of AMI hospitalization. Findings in the systematic review and meta analysis studies by(Mustafić et al., 2012) and (Luo et al., 2015) was consistent  with the findings from this study. These two studies examined the pooled estimates on the relationship between short-term exposure to common air pollutants and the risk of myocardial infarction based on a random-effects meta-analysis model. A moderate degree of heterogeneity was observed and also evidence of publication bias in PM2.5 exposure.  Authors from both these studies suggested that the risk of AMI was significant after short-term exposure to common air pollutants.

Moreover findings from the studies by (Weichenthal et al., 2017; Argacha et al., 2016; Chang, Kuo, Liou, & Yang, 2013; Zhang et al., 2016; Sahlén et al., 2019; Rich et al., 2013; Evans et al., 2016; Gardner et al., 2014; Hopke et al., 2015; Pope et al., 2015) are consistent with the findings from both the overall and subgroup analysis in that short-term exposure to 5-10g/m3 increments of particulate matter with an aerodynamic diameter of <2.5 micron (PM2.5) is associated with an increased risk of hospital admissions due to acute myocardial infarction. The collective argument from these studies suggest that even at low concentrations, an increase of 5-10 of PM2.5 can trigger acute cardiovascular events like acute myocardial infarction and subsequently increase AMI hospitalizations

 (Borenstein, Hedges, Higgins, & Rothstein, 2009)

## Risk of AMI hospitalizations in Lautoka due to the ambient air quality in the Lautoka CBD

After carefully examining the results of the air quality data and the outcome of the meta-analysis, significant extrapolations can be made as to the risk of AMI hospitalisations in Lautoka. Of all the common air pollutants (PM10, PM2.5, O3, SO2, NO2, CO) measured in the Lautoka CBD, only PM10and PM2.5 exhibited concentrations that was significant and relatable to the objectives of this study.  Although the mean daily ambient PM2.5 in the Lautoka CBD was relatively low (9.34g/m3) as shown in Figure 19, the outcome of the meta-analysis as well as that observed in the study by (Mustafić et al., 2012)have shown that short-term exposure to low concentrations is associated with increased AMI hospitalisations. As was observed in the study by (Weichenthal et al., 2017),  short-term exposure to every 5g/m3 increments of a 3-day mean PM2.5 was associated with an increased risk of myocardial infarction among the elderly. Interestingly, the case-crossover study by (Hsieh, Yang, Wu, & Yang, 2010) observed that after a 2 day lag, PM10 was found to be statistically significant with increased hospital admissions de to myocardial infarction. Similar observations was made in the study by(Liu et al., 2017)

Daily PM10 concentrations exceeded the WHO recommended  guideline value and  as such poses a significant risk. Epidemiological studies have also observed associations  associations (Yu et al., 2018)

 (Maclure & and M. A. Mittleman, 2000) (Egger, Smith, & Phillips, 1997)

# Chapter: Conclusion

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