# DETERMINATION OF SPATIAL DISTRIBUTION OF CARDIOVASCULAR DISEASE RISK FACTORS AT THE DIKGALE, MAMABOLO, MOTHIBA HEALTH AND DEMOGRAPHIC SURVEILLANCE SITE, LIMPOPO PROVINCE, SOUTH AFRICA

**BY**

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DISSERTATION

Submitted in fulfilment of the requirements for the degree of

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**DECLARATION**

I declare that the dissertation hereby submitted to the University of Limpopo, for the degree of (MSc Medical Sciences in Chemical Pathology) has not been previously submitted by me for a degree at this or any other university; that it is my work in design and execution, and that all material contained herein has been duly acknowledged.

Makgobatlou, MD (Ms) September 2024

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**DEDICATION**

To the Lord Almighty for giving me strength and keeping me in good health.

In loving memory of my mother (Mrs. Moyahabo Makgobatlou) and my lovely siblings who recently passed on, for always wanting the best for me.

A special feeling of gratitude to my father, niece, fiancé, daughter and brother for their words of encouragement, support and everlasting love.

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**ABSTRACT**

**Determination of spatial distribution of cardiovascular disease risk factors at the Dikgale, Mamabolo, Mothiba health and demographic surveillance site, Limpopo province, South Africa.**

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**Background:** For proper management and control of diseases, epidemiological studies focused on the spatial distribution of communicable diseases. Little attention has been given to non-communicable diseases, especially amongst the black population.

**Aim:** To determine the spatial distribution of cardiovascular disease risk factors (hypertension, obesity, dyslipidaemia, diabetes mellitus, alcohol consumption, smoking) among adults residing in the Dikgale, Mamabolo, Mothapo health and demographic surveillance site (DIMAMO HDSS), Limpopo province, South Africa.

**Methodology:** This was a retrospective, descriptive, cross-sectional and quantitative research. The study used data collected from the Africa Wits-INDEPTH Partnership for Genomic Research (AWI-Gen) phase 1. A total sample size of 791 (women-549 and men-242) was used. Data extraction tool was used to obtain variables such as demographic status, socio-economic status, lifestyle, substance use, laboratory measurements, Geographic Information System (GIS) coordinates as well as the general health of the participants. The data were analysed using Statistical Package for Social Sciences (SPSS) software for statistical analysis and the local Moran I statistic for the spatial analysis.

**Results:** Most of the biomedical risk factors were more common among women compared to men, hypertension (51.7% vs 37.6%), obesity (49.5% vs 3.3%), dyslipidaemia (22.6% vs 15.0%) and diabetes mellitus (7.4% vs 4.6%). In contrast, most of the behavioural risk factors were more common among men compared to women, alcohol consumption (57.4% vs 13.8%) and smoking (82.6% vs 7.5%). Metabolic syndrome was more common in women (15.7%) compared to men (2.1%). In the total population, the proportions of metabolic syndrome and high Framingham score were 11.5% and 9.9% respectively. Binary and multivariate regression analysis

showed that diabetes and central obesity may have contributed to hypertension, high waist circumference may have contributed to the distribution of diabetes mellitus, obesity was found to be a determinant for the distribution of dyslipidaemia and there was no association between both alcohol consumption and smoking with social determinants.

**Conclusion:** Biomedical cardiovascular disease risk factors were more common among women and in clusters A and B whilst behavioural cardiovascular risk factors were more common among men and in cluster D. Most of the cardiovascular disease risk factors were more common in village clusters A and B. The reason for this may be attributed to age, gender and behavioural risk factors.

**Keywords:** cardiovascular risk factors (hypertension, diabetes mellitus, obesity, dyslipidaemia, alcohol consumption, smoking), spatial distribution**.**

## DEFINITION OF CONCEPTS

The defined terms below were used as they are unless stated otherwise.

**Cardiovascular diseases:**

Disorders that affect the blood vessels and heart (Fabiyi *et al.,* 2015).

**Cardiovascular disease (CVD) risk factors:**

These are behaviours, circumstances, or conditions that makes one susceptible to the development of cardiovascular disease (Psaltopoulou *et al.*, 2017).

**CVD non-modifiable risk factors:**

Non-modifiable CVD risk factors are risk factors that are less manageable with behaviour like age, gender (Darikwa *et al.,* 2020).

**CVD modifiable risk factors:**

Modifiable CVD risk factors are risk factors that can be manageable with behaviour like obesity (Darikwa *et al.,* 2020).

**Spatial distribution:**

Refers to the analysis of phenomena that can be linked to the way disease is scattered in a certain location as well as the population at high risk (Pfeiffer *et al.*, 2008).

## LIST OF ABBREVIATIONS

**AWI-gen** : Africa Wits-INDEPTH partnership for genomic research

**BMI** : Body mass index

**CV** : Co-efficient of variation

**CVD** : Cardiovascular disease

**DBP** : Diastolic blood pressure

**DHET** : Department of higher education and training

**DIMAMO HDSS** : Dikgale, Mamabolo, Mothapo health and demographic surveillance site

**GIS** : Geographic information system

**HDL-c** : High density lipoprotein cholesterol

**IDF** : International Diabetes Federation

**LDL-c** : Low density lipoprotein cholesterol

**LDL-Rs** : Low density lipoprotein receptors

**LVH** : Left ventricular hypertrophy

**MRI** : Magnetic resonance imaging

**NCEP ATP III** : National Cholesterol Education Program Adult Treatment panel III

**SAT** : Subcutaneous adipose tissues

**SBP** : Systolic blood pressure

**SPSS** : Statistical Package for Social Sciences

**TREC** : Turfloop Research Ethics Committee

**VAT** : Visceral adipose tissues

**VLDL** : Very low-density lipoprotein

**WHO** : World Health Organization

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# CHAPTER 1

## 1. INTRODUCTION AND PROBLEM STATEMENT

### 1.1 INTRODUCTION

According to (Roy *et al.*, 2017), cardiovascular diseases (CVDs) are a group of disorders that mainly affect the heart as well as blood vessels. Most of these diseases are related to a process called atherosclerosis (Rehman *et al.*, 2022). Globally, CVDs are the leading cause of morbidity (Kivimäki *et al.,* 2018) and mortality (Fabiyi *et al.*, 2015), affecting millions of people each year. South Africa is not left out as noted by (Sliwa *et al.*, 2022), this contributes to the growing threat to health among Africans as well.

The diagnosis of cardiovascular disease (CVD) includes plenty of methods depending on the specific disease that is being studied. The main methods of diagnosis include the determination of associated CVD risk factors through family and medical history, physical examination, laboratory and imaging tests (Laslett *et al.*, 2012). Cardiovascular disease risk factors, which are the focus of the present study, play a fundamental role in terms of CVD causation, having the risk factor/s elevate a person's chance of developing the disease (Psaltopoulou *et al.*, 2017).

These risk factors include those that are not modifiable: gender, age, ethnicity, family history (hypertension, dyslipidaemia, diabetes mellitus). The non-modifiable risk factors are difficult to manage as they cannot be altered with a change of behaviour (Umer *et al.*, 2017). On the other hand, the modifiable ones include obesity, smoking, alcohol consumption, physical inactivity (Darikwa *et al.,* 2020). Modifiable risk factors are termed modifiable because they can be controlled or reduced with a change of behaviour (Umer *et al.*, 2017)

Spatial distribution of disease is defined as the analysis of phenomena that can be linked to the way disease spread in a certain location as well as the population at risk (Nyirenda *et al.*, 2019). Spatial studies on chronic diseases like CVDs are unique, they are designed to examine the contextual characteristics of communities that may be afforded opportunities for chronic disease prevention and treatment (Casper *et al.*, 2019). To develop targeted, cost-effective and chronic health interventions, one needs

to understand the joint spatial clustering of CVD risk factors in areas of need (Darikwa *et al.,* 2020).

Globally, risk factors like hypertension, unhealthy diet, diabetes mellitus and tobacco smoking remain the leading cause of CVD (between the years 2000 and 2016) but in Eastern Europe, there has been a decrease in CVD morbidity and mortality over the same period (Thomas *et al.*, 2018). In South Africa, the (Darikwa *et al.,* 2020) study showed that the rates of CVDs and its associated risk factors were higher in urban areas than in rural areas. These findings are suggestive of a more diffusion process in urban districts. A national study of the 9 provinces of South Africa showed that there is a relatively high prevalence of non-communicable diseases even in the rural populations, with 57% and 43% prevalence in urban and rural areas respectively (Ajaero *et al.,* 2022). CVD risk factors are increasing in the rural population of South Africa. The Darikwa *et al.,* 2020 study included a few rural populations, therefore it will be beneficial to do a spatial analysis study to develop an appropriate intervention for the rural population.

## 1.2 PROBLEM STATEMENT

Spatial distribution of disease is the analysis of phenomena that can be linked to the way disease spread in a certain location. This helps in developing appropriate interventions and targeting affected populations. Most spatial studies on health conditions are mainly focusing on communicable diseases rather than non-communicable diseases. As a result, communicable diseases are better managed than non- communicable diseases as spatial analysis of these conditions helped in developing the appropriate intervention. There is thus a need to also use spatial analysis in developing an appropriate and necessary intervention for non-communicable diseases.

Cardiovascular diseases and associated risk factors are becoming common worldwide and thus serve as the ideal non-communicable disease that can be analysed using spatial analysis. According to our knowledge, there are limited studies that evaluated the spatial distribution of cardiovascular disease risk factors (hypertension, diabetes mellitus, obesity, dyslipidaemia, smoking and alcohol consumption) among rural black population in South Africa. This study aims to determine the spatial distribution of cardiovascular disease risk factors among black adults residing in the Dikgale, Mamabolo, Mothapo health and demographic surveillance site (DIMAMO HDSS).

# CHAPTER 2

## 2. LITERATURE REVIEW

This section gives background on cardiovascular diseases (CVDs), related risk factors and how spatial analysis is incorporated into the evaluation of the risk factors.

### 2.1 Introduction

According to (Roy *et al.*, 2017), the term cardiovascular is derived from 'cardio' meaning heart and 'vascular' meaning blood vessel, therefore cardiovascular diseases are a group of disorders that affect the heart and blood vessels (Fabiyi *et al,* 2015). Cardiovascular diseases are caused by or associated with plenty of health problems, many of which are related to a process called atherosclerosis (Rehman *et al.*, 2022). Atherosclerosis is a condition that develops when a plaque builds up in the walls of blood vessels, the buildup narrows the vessels, making it harder for blood to flow throughout the body (Saigusa *et al.*, 2022).

Cardiovascular diseases remain the leading cause of disease burden globally (Kivimäki *et al.,* 2018). About 17.3 million people died from cardiovascular diseases, this amount to about 30% of all global deaths out of which an estimated 7.3 million were because of stroke (Fabiyi *et al,* 2015). Africa is not left out of this burden as noted by (Thomas *et al.*, 2018), and this constitutes a growing threat to health among Africans. In South Africa cardiovascular diseases such as coronary artery disease and stroke account for more than a third of deaths in people older than 65 years (Sliwa *et al.*, 2022)

Other entities that contribute to CVDs are cardiovascular disease risk factors, which is the focus of this study.

"Understanding the joint spatial clustering of CVDs and associated risk factors to determine areas in need of enhanced integrated interventions would help develop targeted, cost-effective and productive mediations" (Darikwa *et al.,* 2020).

Having an understanding of how health outcomes are spatially distributed is a fundamental step in investigating the effect of environmental influences on health-related issues (Paquet *et al.*, 2016). Spatial studies on chronic diseases are unique because they are designed to examine the contextual characteristics of communities that may be afforded opportunities for chronic disease prevention and treatment

(Casper *et al.*, 2019). The main importance of the distribution of chronic diseases like cardiovascular diseases is that it helps to determine appropriate and necessary interventions which enable public health professionals the ability to precisely understand and address existing problems in chronic disease management (Casper *et al.*, 2019).

## 2.2 Cardiovascular disease risk factors

### 2.2.1 Non-modifiable risk factors

Risk factors for cardiovascular disease are certain behaviours, circumstances or conditions that make one susceptible to developing cardiovascular disease (Psaltopoulou *et al.*, 2017). Risk factors of CVDs have been identified over the years. The non-modifiable risk factors such as gender, age, ethnicity, family history are less manageable with behaviour (Darikwa *et al.,* 2020).

### 2.2.2 Modifiable risk factors

Modifiable risk factors are those that can be managed with a change of behaviour. Examples include obesity, high blood pressure, high blood glucose, high fat intake, low physical activity, harmful or excessive tobacco and alcohol use, high salt intake, educational level, employment status, (Darikwa *et al.,* 2020).

### 2.2.3 CVD risk factors

The present study focused on the following CVD risk factors: hypertension, diabetes mellitus, obesity, dyslipidaemia, alcohol consumption and cigarette smoking.

#### 2.2.3.1 Hypertension

According to (WHO, 2013), hypertension is systolic blood pressure ≥140mmHg and/or diastolic blood pressure ≥90 mmHg. Hypertension is the leading cause of cardiovascular disease and premature death worldwide (Mills *et al.,* 2020). According to (Ayogu *et al.*, 2021), hypertension was the major and most prevalent CVD risk factor in Africa. According to the Demographic and Health Survey in the rural area in Limpopo Province of South Africa, the prevalence of hypertension was found to be approximately 21%, and it is slightly more common in women than in men (Ntuli *et al.*, 2015)

Hypertension is a major risk factor for stroke, heart failure (Saiz *et al.*, 2022). Hypertension is the most prevalent risk factor for stroke and has been reported in

about 64% of patients with stroke (Cipolla *et al.,* 2018). One of the most important consequences of chronic hypertension is its effect on cerebral arteries and arterioles which leads to hypoperfusion of the brain (Ciacciarelli *et al.*, 2020). Decreased perfusion of the brain microcirculation is caused by hypertrophy and remodelling of the cerebral arteries and arterioles. Hypertension-induced hypertrophy and remodelling are considered detrimental in cerebral arterioles and this increases the chances of both haemorrhagic and ischaemic stroke (Cipolla *et al.,* 2018).

Hypertension is also associated with heart failure. Hypertensive heart disease represents illnesses from uncontrolled hypertension that may progress to heart failure (Slivnick *et al.,* 2019). In the most widely accepted model of hypertensive heart failure, left ventricular hypertrophy (LVH) is caused by chronic pressure overload (Guazzi *et al.,* 2017). Progressive hypertrophy changes in the heart lead to diastolic dysfunction which ultimately leads to increased left-sided filling pressures and diastolic heart failure (Kemp *et al.,* 2012). Eventually, a significant number of patients progress to systolic dysfunction in the presence of chronic volume and pressure overload (Guazzi *et al.,* 2017).

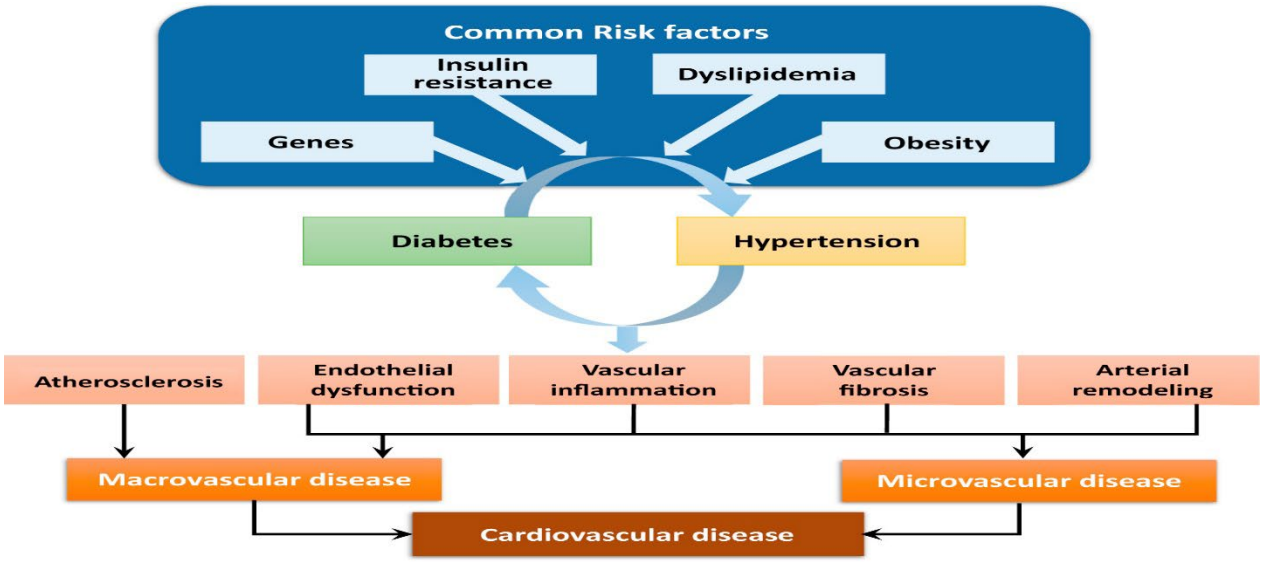
A spatial study conducted in Thailand showed that hypertension clusters occurred in Bangkok province as well as other metropolitan areas while significantly low rates clusters occurred in some northeast and southern provinces (Laohasiriwong *et al.*, 2018). This was mainly because of urbanization, better socioeconomic status and economic growth in the urban areas. These findings help in the facilitation of control and prevention of hypertension and assist researchers and health officials to come up with sound and effective ways to manage hypertension.

#### 2.2.3.2 Diabetes Mellitus

Hyperglycaemia which is chronic, is the primary symptom of a variety of metabolic illnesses together, which is referred to as diabetes mellitus. Diabetes mellitus is caused by either impaired insulin secretion, impaired insulin action, or both (Petersmann *et al.*, 2019). According to ( Atlas, International Diabetes Federation (IDF), 2015), approximately 75–80% of people with diabetes die due to cardiovascular complications (Animaw *et al.,* 2017). The Atlas, IDF estimates that worldwide, 415 million people have diabetes, 91% of those with diabetes mellitus were found to have type 2 diabetes mellitus (Atlas, International Diabetes Federation, 2021). Type 2

diabetes mellitus accounts for over 90% of diabetes and this proportion is higher if it is adjusted for the older and urban population (Pheiffer *et al.*, 2021). The burden of diabetes and diabetes-related mortality and disability are reported to be rising in Africa due to sedentary lifestyles as well as urbanisation (Animaw *et al.,* 2017). Type 2 diabetes mellitus is a major source of morbidity and mortality in South Africa (Pheiffer *et al.*, 2021).

Diabetes is associated with both macrovascular (involving large arteries such as the aorta, carotid) and microvascular (involving small arteries and capillaries) disease (Petrie *et al.,* 2018). Several pathways, including oxidative stress and inflammation, are involved in the development of vascular problems associated with diabetes, including chronic hyperglycemia and insulin resistance (Ke *et al.*, 2022). Because hypertension is connected with vascular dysfunction and injury, it is a significant risk factor for diabetes-related vascular problems (Figure 1).




Petrie *et al.,* 2018

**Figure 1: illustrates the vascular mechanisms by which cardiovascular disease is predisposed by diabetes and hypertension.**

Obesity, dyslipidemia, insulin resistance, and genetics are common risk factors for CVD. These conditions exacerbate hypertension and diabetes, promoting endothelial dysfunction, atherosclerosis, vascular inflammation, and structural remodelling that result in both macrovascular and microvascular illness. When diabetes and

hypertension coexist, there may be significant vascular damage and endothelial dysfunction (Petrie *et al.,* 2018).

Reducing the microvascular and macrovascular consequences of diabetes mellitus may require managing hypertension as a comorbidity and focusing on strategies to improve vascular health because diabetes is linked to an increased risk of CVD, which is exacerbated when coexisting with hypertension (Petrie *et al.,* 2018).

A study conducted by (Huang *et al.*, 2020) in China found that there was a persistent hot spot for diabetes mellitus in the city centres as compared to rural areas. People who reside in the city are more likely to suffer from diabetes mainly because of lifestyle differences between urban and rural areas.

2.2.3.3 Obesity

Clinically, obesity is diagnosed when the body mass index (BMI) is >30 kg/m$^2$. Obesity is defined by the WHO as an abnormal or excessive buildup of fat that poses a risk to one's health. Globally, the prevalence of obesity is increasing and this is thought to be due to the lifestyles related to high calorie intake and low energy expenditure (Petrie *et al.,* 2018).

Obesity, which is a worldwide epidemic has major effects on cardiovascular health (Landecho *et al.*, 2019). Obesity predisposes individuals to acute coronary syndrome, atrial fibrillation, heart failure, ischemic stroke, which are the main causes of cardiovascular hospitalization and mortality (Kotsis *et al.*, 2018).

Atrial fibrillation is one of the important cardiac arrhythmia associated with significant mortality and morbidity (Javed *et al.,* 2021). Research indicates that the primary pathogenic pathway that can result in the development of atrial fibrillation in the obese population is left atrial dilatation along with elevated inflammatory factors (Javed *et al.,* 2021), this is mainly because of the effect of fat deposits on the epicardium (Kotsis *et al.*, 2018).

Heart failure is also prevalent in the obese population (Id *et al.*, 2019). The mechanism in which obesity leads to cardiac problems is thought to be due to the increase in blood volume and cardiac output, which results in an increase in blood returning to the heart. This leads to dilatation of the left and right ventricular walls and finally reduction in the pump strength of the left ventricle, which causes left ventricular hypertrophy (heart

failure), (Id *et al.*, 2019). An independent risk factor for stroke is obesity (Kernan *et al.*, 2013). These risk factors cause structural alterations in both the small and large arteries. These alterations are linked to silent lesions in the small arteries of the brain, such as lacunar infarcts, white matter lesions, and microbleeds that can be seen on brain magnetic resonance imaging (MRI) (Kotsis *et al.*, 2018). It is acknowledged that a significant risk factor for stroke is the existence of silent small vessel disease in the brain (Kernan *et al.*, 2013).

Adipose tissue has been reported to be able to synthesise and release a wide range of hormones, cytokines, extracellular matrix proteins, growth factors, and vasoactive agents. These substances are collectively referred to as adipokines, and they have been shown to affect numerous physiological and pathological processes (Landecho *et al.*, 2019).

Subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) can both be measured to determine fat distribution among obese individuals (Lytle *et al.*, 2021). Ultrasound provides the most accurate results in terms of measuring the layers of subcutaneous adipose tissue, subcutaneous tissue ranges from 1.65 mm to 14.65 mm in males and from 3.30 mm to 18.20 mm in females (Störchle *et al.*, 2018). Visceral adipose tissue is linked with metabolic consequences of obesity and is usually characterized by measuring the VAT area at the level of L4 to L5 intervertebral space (Cho *et al.*, 2019). These measurements are commonly done by computed tomography (CT), but are not limited to CT, VAT can also be measured using ultrasound as well (Cho *et al.*, 2019). The visceral fat area associated with an increased risk of obesity is 103.8 cm squared (Lytle *et al.*, 2021).

Due to increased fat accumulation, adipose tissue undergoes hypertrophy and hyperplasia which leads to adipose tissue enlargement and are associated with the development of metabolic derangements and elevated cardiovascular risk in obesity (Landecho *et al.*, 2019). When different deposits of fat are released into the bloodstream and travel to distant sites (such as the heart and arteries), they are able to put forth their biological effects in an endocrine manner, increased adipocytes, primarily at the perivascular site, alter the local generation of angiotensinogen, which plays an essential role for the circadian regulation of blood pressure (Garcia-barrio et al., 2018), ultimately affecting the myocardium's vascular walls are directly impacted by this (Landecho *et al.*, 2019).

A study in Nigeria, showed that the people with high-paying and stable jobs are more likely to be overweight while those from poverty backgrounds had obesity (Osayomi, 2020). This suggests that spatial patterns of obesity and overweight are related to economic status.

#### 2.2.3.4 Smoking

Tobacco remains one of the leading causes of preventable death in Western countries. It is responsible for 700,000 deaths a year in Europe (Bouabdallaoui *et al.*, 2021). The prevalence of adult smokers in South Africa was 17.6% with 15.9% smoking daily and 1.7% non-daily smokers (Darikwa *et al.,* 2020). The prevalence of smokers in the Dikgale rural area of Limpopo was 13.7%, of which 81.3% were daily smokers (Maimela *et al.*, 2016).

The mechanisms by which tobacco leads to CVD are not yet clear (Dhar *et al.*, 2020). However, it is thought that the air we inhale from the atmosphere is oxygen-rich. Through blood vessels, oxygenated blood is returned to the heart and other areas of the body by the lungs after exchanging oxygen for carbon dioxide. When blood is circulated throughout the body carrying cigarette smoke, it contaminates various bodily parts with the toxins from cigarettes (Dhar *et al.*, 2020). Carbon monoxide gas, nicotine poison, and the whole aerosol remnant known as tar are the main ingredients of tobacco smoke. The compounds found in cigarettes other than nicotine provide the highest risk of cardiovascular disease. When inspired, nicotine causes an increase in the rate of heart and blood pressure (Dhar *et al.*, 2020).

In Zambia, a study showed that cigarette smoking is higher in separated or divorced individuals and it was lower in those with some formal education (Nyirenda *et al.*, 2019). Therefore, cigarette smoking interventions should consider targeting specific demographics, socioeconomic factors.

#### 2.2.3.5 Dyslipidaemia

Dyslipidemia is defined as increased levels of serum total cholesterol, low-density lipoprotein cholesterol (LDL-c), triglycerides, or decreased serum high-density lipoprotein cholesterol (HDL-c) levels (Hedayatnia *et al.*, 2020). Dyslipidaemia is associated with poor cardiovascular outcomes, even though this is the case, markers

of this condition are often ignored in clinical practice (Halcox *et al.*, 2017). The high prevalence of dyslipidaemias may give signs of cardiovascular disease in a population known to be undergoing lifestyle changes with urbanisation (Khine *et al.,* 2016). Previously, abnormal lipid levels were not common among the South African black population, however, a recent study at George Mukhari referral hospital reported that currently there is an increased request for lipid profile testing indicating that abnormal lipid levels are now becoming common (Khine *et al.,* 2016). This is supported by a study that has reported that the prevalence of high LDL-c cholesterol and low HDL-c cholesterol are increasing (Maimela *et al.*, 2016).

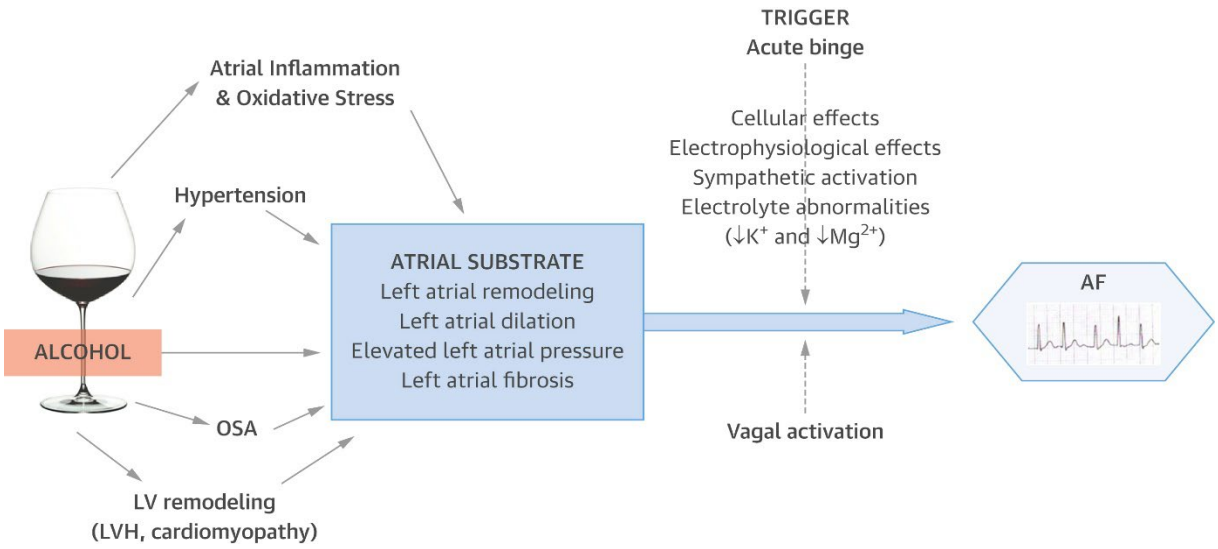
The progression of dyslipidaemia is associated with atherosclerotic cardiovascular disease which is caused by the atherogenic contribution of LDL-c that has been oxidised (ox-LDL-c) (Liu *et al.*, 2017). Oxidation of LDL-c involves a series of complex processes, it occurs when there are high levels of native LDL-c in the body, these LDL-c particles react with free radicals, molecules that are not stable, exposure to toxins, also diseases are transformed to ox-LDL-c. According to (Trpkovic *et al.*, 2015), LDL-c receptors (LDL-Rs) are responsible for the identification and internalization of the native LDL-c, this is a very important contribution to the metabolism of cholesterol. An experiment of incubating high levels of native LDL-c with macrophages revealed that the macrophages ultimately transform into lipid-laden foam cells, which are important in the formation of atherosclerotic plaques. This suggests that LDL-Rs on macrophages may be up-regulated in conditions that have elevated LDL-c levels (Liu *et al.*, 2017).

According to the findings of a study conducted in South Africa, urban informal and rural informal populations had a lower risk of dyslipidaemia as compared to those in urban formal settlements (Dwane *et al.,* 2020). Also, people with higher education qualifications had significantly higher rates of dyslipidaemia (Dwane *et al.,* 2020). Therefore, demographics as well as educational level play roles in the development of dyslipidaemias.

#### 2.2.3.6 Alcohol

The association between alcohol intake and CVD is in two ways, it is safeguarding at low amounts and harmful at high intake (Voskoboinik *et al.*, 2016). Despite what has been said above, some writers argue that alcohol intake is also harmful even in low

amounts (Arnett *et al.*, 2019). However, a recent study supports that alcohol in low amounts is of benefit to the cardiovascular system (Chiva-Blanch *et al.,* 2019). However, more levels of alcohol intake on a regular basis may also increase the risk of CVD (Voskoboinik *et al.*, 2016). Alcohol consumption also appears to have a continuous positive relationship with the risk of atrial fibrillation (Toma *et al.,* 2017).




Voskoboinik *et al.*, 2016

**Figure 2: Shows habitual Alcohol Consumption and atrial fibrillation pathophysiology.**

AF = atrial fibrillation; $K^+$ = potassium; LV = left ventricle; LVH = left ventricular hypertrophy; $Mg^{2+}$ = magnesium; OSA = obstructive sleep apnea

Habitual alcohol consumption predisposes to atrial fibrillation by direct effects on the left atrial substrate and interaction with other atrial fibrillation risk factors, including hypertension, obstructive sleep apnea and left ventricular dysfunction (Voskoboinik *et al.*, 2016).

According to a study done in Canada, taking away alcohol was associated with increased alcohol consumption as compared to on-site consumption (Sherk *et al.*, 2018). This means that for proper intervention to reduce alcohol abuse, customers should consume alcohol at the bar and not take any home.

### 2.3 Spatial analysis and its use in assessing CVD risk factors.

Spatial distribution of disease refers to the analysis of phenomena that can be linked to the way disease spread in a certain location as well as the population at risk

(Nyirenda *et al.*, 2019). This is carried out by extracting or creating new information about a set of geographical features to perform routine examinations, also for research purposes (Mena *et al.*, 2018). The use of mapping as an epidemiological tool by the geographic information system (GIS) is a fundamental step in the production of disease maps and enables users to spatial referencing and manipulation of data (Fabiyi *et al.,* 2015).

Geospatial studies mostly use maps to establish correlations of chronic diseases such as CVDs as well as related risk factors with geographical position (Lwin *et al.,* 2012). These maps play a vital role in visualizing epidemiological health situations and they can be easily understood (Lwin *et al.,* 2012). Spatial pattern detection can be a useful tool for understanding the geographical distribution of CVD risk factors, previous studies have demonstrated that socioeconomic status, education level, air pollution, infrastructure, urbanization have effects on the occurrence of CVD (Rahnama *et al.,* 2019). Once these are known, appropriate interventions can be implemented.

Globally, risk factors like hypertension, unhealthy diet, diabetes mellitus and tobacco smoking remain the leading cause of CVD (between the years 2000 and 2016) but in Eastern Europe, there has been a decrease in CVD morbidity and mortality over the same period (Thomas *et al.*, 2018). Nationally, the (Darikwa *et al.,* 2020) study showed that the rates of CVDs and risk factors were higher in urban areas than in rural areas. These findings are suggestive of a more spatial diffusion process among urban districts. CVD risk factors are increasing in the rural population of South Africa. (Darikwa *et al.,* 2020) the study included a few rural populations; therefore, it will be beneficial to do a spatial analysis study to develop an appropriate intervention for the rural populations.

## 2.3 PURPOSE OF STUDY

### 2.3.1 Study aim

To determine the spatial distribution of cardiovascular disease (CVD) risk factors at Dikgale, Mothapo, Mamabolo health and demographic surveillance site (DIMAMO HDSS), Limpopo Province, South Africa.

### 2.3.2 Objectives

To determine the prevalence of CVD risk factors among adults participating in the DIMAMO HDSS.

To correlate the CVD risk factors with spatial analysis variables among adults participating in the DIMAMO HDSS.

To determine the determinants of the spatial distribution of CVD risk factors in the DIMAMO HDSS.

## 2.4 RESEARCH QUESTION

What is the spatial distribution of cardiovascular disease risk factors in the DIMAMO villages of Limpopo province?

# CHAPTER 3

# 3. RESEARCH METHODOLOGY

## 3.1 Research design

This research was quantitative, retrospective, descriptive and cross-sectional study.

Quantitative research is the type of research that gives the researcher a way of making statistics from the data collected, the data obtained can be counted (Heale *et al.,* 2015). This study was quantitative because the data was analysed statistically. Descriptive research aims to describe the patterns at which diseases occur concerning variables such as person, place, and time (Snyder, 2019). This study was descriptive because it described the spatial distribution of cardiovascular risk factors.

A cross-sectional study is a type of research whereby data is collected from different individuals at a single point in time (Monakali *et al.*, 2018). This study was cross-sectional because it used data that was collected only once (from the AWI-gen phase) and there was no follow-up. A retrospective study is a study that uses existing data that have been collected (Snyder, 2019). This study was retrospective because secondary data collected from AWI-gen (Africa Wits-INDEPTH Partnership for Genomic Studies) Phase 1 was used.

AWI-gen is Africa Wits-INDEPTH (University of the Witwatersrand, Johannesburg, and the International Network for the Demographic Evaluation of Populations and Their Health) partnership for Genomic Studies (AWI-gen), is a National Institute of Health–funded Collaborative Centre of the Human Heredity and Health in Africa (H3Africa) (Gómez-Olivé *et al.*, 2017). The main aim of the AWI-gen study is to study environmental and genetic factors that are associated with cardio-metabolic disease in populations in Africa (South Africa, Kenya, Ghana and Burkina Faso) (Ali *et al.*, 2018). The phase 1 project recruited participants from August 2013 to August 2016 (Ali *et al.*, 2018). The project has 2 phases, phase 2 was initiated to investigate any cardiometabolic changes that may occur in phase 1 participants over time, It (phase 2) was initiated after about five years of phase 1 cessation (Ramsay *et al.*, 2018). This study used data from phase 1 because phase 2 was not yet complete and quality checks were also not finalised.

## 3.2 Sampling

### 3.2.1 Study area

The Dikgale, Mamabolo, Mothapo health and demographic surveillance site (DIMAMO HDSS) is a unit conducting population surveillance in the Dikgale, Mamabolo and Mothiba villages, the villages are situated next to the University of Limpopo which is about 40km from Polokwane, the capital city of Limpopo province. DIMAMO HDSS participants are predominantly Africans who speak Northern Sotho (Mashinya *et al.*, 2020).

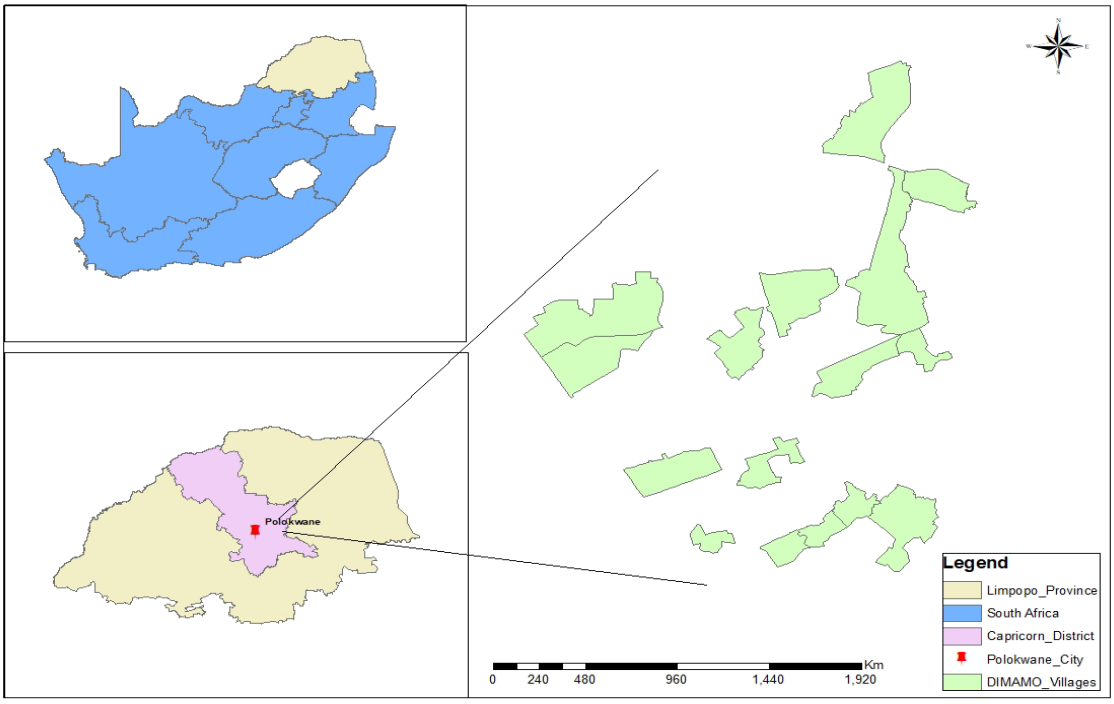



**Figure 3: DIMAMO villages**

Shows where the DIMAMO villages are situated in Limpopo province.

### 3.2.2 Study population

The target area has an estimated population size of approximately 100 000 (Ramsay, 2021). The study population comprised adults (40-60 years) who participated in the AWI-Gen1.

Participants who participated in the AWI-gen1 phase was about 1400. There was no sampling for the present study, the results obtained from all participants in AWI-gen1 was considered when coming to data analysis, provided they contain all relevant and required information for the study.

### 3.2.3 Sample design

Participants in AWI-gen phase 1 were selected using convenient sampling. Convenient sampling means that the participants were selected according to their availability (Stratton, 2021).

### 3.2.4 Sample size

Slovin's formula was used since the estimated total population was known.

$$N/(1+N*e^2)$$

N= known population number of the villages of interest (100 000)

e=margin of error (5%)

$=100000/1+100000*0.05^2$

=100000/251

=398

The calculated minimum sample size is 398.

### 3.2.5 Inclusion criteria

The AWI-gen project came fourth because there was an alarming increase of cardiovascular and metabolic diseases in Africa and less research and interventions were in place (Ali *et al.*, 2018). Therefore, the West Africa (Burkina Faso and Ghana), East Africa (Kenya) and South Africa were recruited to take part in the project. The participants were then selected among the centres of the participating countries. The inclusion criteria for AWI-gen project were men and women of 40-60 years comprising of approximately equal numbers of men and women participants. However, later in the project, it was noted that the DIMAMO HDSS had approximately 70% of women participants.

For this study, participants who participated in the AWI-gen1 phase conducted at DIMAMO HDSS were included. The participants were all considered to increase the statistical strength of the study.

### 3.2.6 Exclusion criteria

Any participant with incomplete data (from the AWI-gen1 phase at DIMAMO HDSS) required for the study was excluded.

### 3.3 Data collection

The current study was retrospective, secondary data was used. The data of variables of interest were extracted from the database using an extraction tool. The researcher (I) in the present study worked with the AWi-Gen2 data collection team to gain experience and knowledge on how the data was collected and captured.

#### 3.3.1 Questionnaire

In the AWI-gen phase 1 project, information on alcohol consumption, smoking, level of education, marital status, employment status, demographic information, lifestyle was obtained through a questionnaire that was translated into Northern Sotho and further explained to participants for full understanding.

#### 3.3.2 Anthropometric measurements

In the AWI-gen phase 1 project, the standing height was measured using a stadiometer. Participants' height measurement was done without shoes, heavy clothing (jackets) and hair accessories.

Body weight was done using a weighing scale. Participants were asked to take off heavy outfits and their shoes. The weight was then recorded.

The participants' body mass index (BMI) was then calculated. BMI is an individual's weight in kilograms divided by the square of the height in meters.

Waist circumference was measured by wrapping a tape measure around their waist (level of the navel) while participants were standing with their hands out of the way.

#### 3.3.3 Measurements of visceral adipose tissue and subcutaneous adipose tissue

visceral and subcutaneous adipose tissue in the AWI-gen phase 1 project were measured using the LOGIQ-e ultrasound system.

#### 3.3.4 Blood pressure

In the AWI-gen phase 1 project, blood pressure was measured using automated Omron blood pressure monitors. The monitor handled the inflation and deflection of the cuff and display the blood pressure (systolic and diastolic blood pressures were measured) and pulse reading on the screen. Blood pleasure was measured from the participants while seated, relaxed and silent. Three readings were done; the mean was recorded. After deflating the cuff, participants waited for five minutes before the next blood pressure measurement was done until three readings were obtained.

### 3.3.5 Blood collection

In the AWI-gen phase 1 project, the participants were requested to fast overnight before the morning of blood collection. The volume of blood taken was about 6 mL and 3 mL for red-top and grey-top (containing sodium fluoride) tubes respectively. After blood withdrawal, participants were given incentive food. There was pre- and post-counseling of HIV testing for participants. Blood samples were collected by qualified nurses from the participants using red, grey-top blood collection tubes. Grey-top tubes containing sodium fluoride were used to collect plasma for glucose determination. Red-top tubes were used to collect serum for the determination of serum lipids. The blood was centrifuged, then serum was transferred into plain cryotubes and stored at -80 degrees Celsius.

### 3.3.6 Laboratory analysis

In the AWI-gen phase 1 project, the stored serum and plasma samples were frozen and transported to the Sydney Brenner Institute for Molecular Bioscience Biobank at University of the Witwatersrand in Johannesburg for biochemical analysis (glucose and serum lipid levels) (Ramsay *et al.*, 2016).

#### 3.3.6.1 Determination of triglycerides (TG) levels

Serum TG was measured, using an automated spectrophotometry method on an AU 480 auto-analyser supplied by Beckman Coulter, which was calibrated, using a standard reference material (system calibrator).

Method procedure: 66 µL of the reagent 1 and 17 µL of reagent 2 were mixed with 57 µL and 10 µL of diluent, respectively. The reaction mixture was amalgamated with 1.6 µL of the sample and incubated for 800 seconds at the wavelength of 660 nm.

The formula was not applicable at TG concentrations greater than 4.5 mmol/L.

#### 3.3.6.2 Determination of total cholesterol (TC) levels

Serum TC was measured using an automated spectrophotometry method on an AU 480 auto-analyser supplied by Beckman Coulter which was calibrated, using a standard reference material (system calibrator).

Method procedure: 24 µL of reagent 1 and 0 µL of reagent 2 were mixed with 96 µL and 0 µL of diluent, respectively. The reaction mixture was amalgamated with 1.6 µL of the sample and incubated for 600 seconds at the wavelength of 540 nm.

3.3.6.3 Determination of high-density lipoprotein cholesterol HDL-c

Serum HDL-c was measured, using an automated spectrophotometry method on an AU 480 auto-analyser supplied by Beckman Coulter, which was calibrated, using a standard reference material (system calibrator).

Method procedure: 144 µL of the reagent 1 and 48 µL of reagent 2 were mixed with 0 µL and 0 µL of diluent, respectively. The reaction mixture was amalgamated with 1.6 µL of the sample and incubated for 700 seconds at the wavelength of 600 nm.

3.3.6.4 Calculation of low-density lipoprotein cholesterol (LDL-c) levels

The measurements of TC, HDL-c and TG were used for the calculation of LDL-c by the Friedewald formula (LDL-c = TC - HDL-c - 0.2 × TG) (Hong *et al.*, 2023).

3.3.6.5 Determination of blood glucose

Glucose was determined, using an AU480 auto-analyser supplied by Beckman Coulter. Method performance: 40 µL of the reagent 1 and 20 µL of reagent 2 were mixed with 120 µL and 20 µL of diluent, respectively. The reaction mixture was amalgamated with 1.6 µL of the sample and incubated for 660 seconds at the wavelength of 340 nm.

**3.4 Data analysis**

3.4.1 Diagnosis

3.4.1.1 Hypertension is defined as systolic blood pressure of ≥140 mmHg and/or diastolic blood pressure of ≥ 90 mmHg (WHO, 2013). Participants with a systolic blood >140 mmHg or diastolic blood pressure of >90 mmHg or both were considered hypertensive.

3.4.1.2 According to (WHO, 2021), obesity is defined as a body mass index of >30 kg/m$^2$. Participants with a BMI of greater than 30 kg/m² were considered obese. Waist circumference of ≥80 cm for women and of ≥90 cm for men was considered increased (WHO, 2021).

3.4.1.3 Normal blood tests result for fasting glucose test: < 7 mmol/l (Standards of Medical Care in Diabetes, 2021). Participants with fasting glucose levels of ≥7 mmol/l were considered hyperglycaemic (diabetic).

3.4.1.4 Reference values for visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT): The optimal cut-off values for VAT are 134.6 cm (men) and 91.1 cm

(women) (Meredith-Jones *et al.*, 2018). The subcutaneous tissue thickness ranges from 1.65 mm to 14.65 mm in men, whereas it is from 3.30 mm to 18.20 mm in women (Störchle *et al.*, 2018). Female participants with VAT of greater than 134.6 cm, SAT of greater than 18.20 mm were considered to have excessive adipose deposits (Störchle *et al.*, 2018). Males with VAT of greater than 91.1 cm, SAT of greater than 14.65 mm were considered to have excessive adipose deposits (Störchle *et al.*, 2018).

3.4.1.5 Dyslipidaemia

High-density cholesterol (HDL-c), the participant had low HDL-c when it was lower than 1.0 mmol/l (women), 0.9 mmol/l(men) (Noubiap *et al.*, 2018).
Low-density cholesterol (LDL-c), the participant had increased LDL-c when it was greater than 2.7 mmol/L (Zhang *et al.*, 2021).

Triglyceride was high if greater than 1.81 mmol/l (men) and 1.52 mmol/l (women) (Masana *et al.*, 2021).

Total cholesterol was high if greater than 5.2 mmol/l (Masana *et al.*, 2021)

3.4.1.6 Metabolic syndrome

According to the National Cholesterol Education Program Adult Treatment panel III (NCEP ATP III) criteria, metabolic syndrome is diagnosed by having any three of the following risk factors: high waist circumference, diabetes mellitus, dyslipidaemia, hypertension (Ismael, 2021).

International Diabetes Federation (IDF) criteria says that high waist circumference is required plus any 2 of the following risk factors: diabetes mellitus, dyslipidaemia, hypertension (Sun *et al.*, 2022).

Being diagnosed with metabolic syndrome, means that one's chance of developing cardiovascular disease is increased compared to those without the diagnosis of metabolic syndrome.

3.4.1.7 Framingham score

Framingham risk score is calculated from considerations of the following variables: age, gender, smoking status, total cholesterol, high density lipoprotein-c (HDL-c), systolic blood pressure, hypertension medication. If the Framingham score is >20%, there is a low risk of cardiovascular disease in the next 10 years. If the Framingham

score is ≤20%, there is a high risk of developing cardiovascular disease in the next 10 years (Santos *et al.*, 2020).

3.4.1.8 Assessment of number of risk factors

The aim of grouping the cardiovascular risk factors (no risk factor, 1, 2, 3 or more risk factor/s) was to categorise the participants according to the guidelines of metabolic syndrome. Being diagnosed with metabolic syndrome increases the chances of cardiovascular disease. Having no risk factor was associated with having a low chance of developing cardiovascular disease compared to those with 3 or more risk factors.

3.4.2 Statistical analysis

Data was analysed statistically using Statistical Package for Social Sciences (SPSS) version 27.0 software. Distribution of continuous variables was assessed using Shapiro-Wilk test. The variables which were normally distributed were expressed as the mean ± standard deviation. The variables that were not normally distributed were expressed as the median and interquartile range (IQR) and were transformed using log form to convert it to a normal distribution.

The qualitative data collected was analysed descriptively and the variables were expressed in percentages. All categorical characteristics of the participants were expressed as a percentage. For all analysis, statistical significance was set at a probability ($p$) of 0.05.

A t-test was used to compare means between 2 independent groups. A chi-square test was used to compare categorical variables. For comparison of 3 or more groups, analysis of variance (ANOVA) was used. A chi-square test was used to compare categorical variables between groups.

To determine the determinants of spatial distribution of CVD risk factors, the spatial data (coordinates) were correlated with sociodemographic variables (age, gender, level of education and marital status)

3.4.3 Spatial analysis

Spatial data analysis included mapping the distribution of standardized incidence ratios (SIRs) for each of the cardiovascular risk factors (obesity, diabetes mellitus, dyslipidaemia, smoking, alcohol consumption, hypertension) in males and females by the community. An SIR is a value that represents the ratio of the observed incidence

of disease to the expected incidence of disease, where a value of zero indicates a community with no observed cardiovascular disease risk factor cases. There are three spatial statistics to determine spatial clustering ( local Moran's I, global Moran's I, and Getis-Ord G*) (Kedron *et al.*, 2021).

The local Moran I statistic outlines whether or where local clustering occurs, which is not provided by the global Moran's I statistic. The Gi* statistic is effective in the measurements of high and low rates (Chance Scott *et al.,* 2016). A cluster & outlier analysis tool in a geographic information system (GIS) is used to calculate the local Moran's I statistic and associated Z-score. Calculated spatial exploratory data help determine spatial autocorrelation of prevalence and inequality values among the entire sample. Spatial analyses will be performed using GeoDa version 1.6.6 (GeoDa Center for Geospatial Analysis and Computation, Tempe, AZ) and maps were produced using QGIS version 2.4.0.

3.4.4 Relationship between spatial data and sociodemographic variables

To determine the determinants of spatial distribution of CVD risk factors, the spatial data (coordinates) were correlated with sociodemographic variables (age, gender, education, marital status) using multivariate logistic regression analysis (appendix v).

### 3.5 Ethical consideration

The research obtained an ethical clearance certificate from the Turfloop Research Ethics Committee (TREC) at the University of Limpopo (TREC/97/2023: PG-Renewed).

The AWI-gen phase 1 project's aims, objectives and procedures of the project were explained to participants in their home language (Northern Sotho) after which they got an opportunity to ask questions and they were made aware that participation in the project was purely voluntary and they could choose to not continue with their participation at any point. The procedures, risks and benefits of the project were explained. The participants who agreed to volunteer were then given an informed consent form translated into their home language (Northern Sotho) to sign. In the present study, participants' consent does not apply as it used retrospective data from the AWI-gen phase 1 project.

Confidentiality means not discussing information provided by an individual with others, whilst anonymity means presenting research findings in ways that ensure individuals cannot be identified (Hoft, 2021). To ensure confidentiality, the clinical and laboratory information and health status of the participants were not discussed with people who were not part of the research team.

Anonymity was ensured by using research identifiers and the names of the participants were omitted.

## CHAPTER 4

## 4. RESULTS

The purpose of this section is to determine how the aim and objectives were addressed. The section shows the characteristics (demographics, anthropometric, biochemical) of participants by gender. Furthermore, it determines the distribution of characteristics amongst the village clusters. It also correlates the CVD risk factors with spatial analysis variables.

### 4.1 Characteristics of the participants by gender.

The purpose of the study was to determine the spatial distribution of CVD risk factors. However, CVDs are determined by looking at different factors that include age, gender, blood pressure, glucose levels, serum lipids, smoking, alcohol consumption. Therefore, the determination of the prevalence and levels of these determinants play a fundamental role when coming to conclusions.

**Table 1: characteristics of participants.**

In the present study, there were 791 participants (549 women and 242 men) out of the 1415 participants who took part in the Awi-gen1 phase. Six hundred and twenty-four (624) participants were excluded from this study due to incomplete data required for the study.

| Variable | Total Population | Women | Men | P-value |
|---|---|---|---|---|
| **N** | 791 | 549 | 242 | - |
| **Age (years)** | 52.47±8.24 | 52.48±8.06 | 52.45±8.64 | 0.951 |
| **Height (m)** | 1.62±83.06 | 1.60±65.31 | 1.70±66.80 | <0.001 |
| **Weight (kg)** | 70.86±21.43 | 76.0±23.57 | 60.78±11.19 | <0.001 |
| **BMI (kg/m$^2$)** | 28.01±5.24 | 30.81±8.05 | 21.67±4.08 | <0.001 |
| **Waist circumference (cm)** | 89.58±17.58 | 93.51±15.90 | 80.64±11.60 | <0.001 |

| | | | | |
|---|---|---|---|---|
| **High waist circumference ((%) n)** | 60.8 (481) | 79.4 (436) | 18.6 (45) | <0.001 |
| **Obesity (% (n))** | 35.4 (280) | 49.5 (272) | 3.3 (8) | <0.001 |
| **SBP (mmHg)** | 125.32±19.55 | 126.20±19.03 | 123.52±20.54 | 0.023 |
| **DBP (mmHg)** | 72.11±97.26 | 78.10±68.494 | 78.10 ±68.50 | 0.764 |
| **Hypertension (% (n))** | 47.4 (375) | 51.7 (284) | 37.6 (91) | <0.001 |
| **Glucose (mmol/l)** | 5.35 ±2.57 | 5.43 ±2.65 | 5.17±2.37 | 0.190 |
| **Diabetes mellitus (% (n))** | 6.6 (55) | 7.4 (44) | 4.6 (11) | 0.147 |
| **Total cholesterol (mmol/l)** | 4.13±1.17 | 4.21±1.20 | 3.93±1.06 | <0.001 |
| **High cholesterol (% (n))** | 20.3 (160) | 22.6 (124) | 15.0 (36) | 0.021 |
| **Triglycerides (mmol/l)** | 0.95 (0.70-1.34) | 0.95 (0.68-0.95) | 0.97 (0.71-1.40) | 0.143 |
| **High Triglycerides (% (n))** | 18.2 (144) | 18.6 (102) | 17.2 (42) | 0.065 |
| **HDL-c (mmol/l)** | 1.18±0.41 | 1.16±0.36 | 1.23±0.49 | 0.022 |
| **Low HDL-c (% (n))** | 30.2 (238) | 30.8 (169) | 28.7 (69) | 0.559 |
| **LDL-c (mmol/l)** | 2.25±1.03 | 2.64±1.0 | 2.26±1.03 | <0.001 |
| **High LDL-c (% (n))** | 41.2 (326) | 46.9 (258) | 28.3 (68) | <0.001 |
| **Smoking (% n))** | 30.5 (241) | 7.5 (42) | 82.6 (199) | <0.001 |
| **Alcohol consumption (% (n))** | 27.2 (215) | 13.8 (76) | 57.4 (139) | <0.001 |
| **No risk factor (% (n))** | 12.8 (101) | 15.7 (86) | 6.2 (15) | <0.001 |
| **1 risk factor (% (n))** | 27.7 (219) | 30.4 (167) | 21.5 (52) | 0.010 |
| **2 risk factors (% (n))** | 37.7 (298) | 34.2 (188) | 45.5 (110) | 0.003 |

| | | | | |
|---|---|---|---|---|
| **3 or more risk factors (% (n))** | 21.9 (173) | 19.7 (108) | 26.9 (65) | 0.024 |
| **No formal education ((%) n)** | 9.2 (73) | 10.2 (576 | 7.0 (17) | 0.153 |
| **Primary education ((%) n)** | 35.5 (279) | 35.0 (192) | 36.0 (87) | 0.804 |
| **Secondary education ((%) n)** | 52.9 (419) | 52.9 (291) | 52.9 (128) | 0.994 |
| **Tertiary education ((%) n)** | 2.5 (20) | 1.8 (10) | 4.1 (10) | 0.057 |
| **Not married ((%) n)** | 20.9 (165) | 20.6 (113) | 21.5 (52) | 0.773 |
| **Married ((%) n)** | 46.8 (370) | 47.5 (261) | 45.0 (109) | 0.516 |
| **Divorced ((%) n)** | 2.9 (23) | 2.2 (12) | 4.5 (11) | 0.069 |
| **Widowed ((%) n)** | 16.9 (134) | 20.4 (112) | 9.1 (22) | <0.001 |
| **Employment ((%) n)** | 31.3 (247) | 29.2 (160) | 36.1 (87) | 0.054 |
| **Metabolic syndrome NCEP ((%) n)** | 14.2 (112) | 18.2 (100) | 5.0 (12) | <0.001 |
| **Metabolic syndrome IDF ((%) n)** | 11.5 (91) | 15.7 (86) | 2.1 (5) | <0.001 |
| **High Framingham score ((%) n)** | 9.9 (78) | 1.6 (9) | 28.5 (69) | <0.001 |

*p-value ≤0.05=there is a significant difference in observed results, p-value>0.05=no significant difference, normally distributed data is presented as mean±SD, mean (IQR) for not normally distributed data and categorical data as ((%) n).*

4.1.1 The mean age of participants was 52.47. There was no significant difference in mean age between men and women (p=0.951).

4.1.2 The mean BMI of the participants was 28.01 kg/m². There was a significantly high proportion of women with obesity compared to men as p <0.001. The proportion of obesity was 35.4% in the total population and was significantly higher in women

compared to men (p≤0.001). The mean value for waist circumference was 89.58 cm. The proportion of participants with increased waist circumference was 60.8% of the total population. The proportion of participants who had increased waist circumference was significantly higher in women compared to men (p≤0.001).

4.1.2 The mean SBP and DBP of the participants were 125.32 and 72.11 mmHg respectively. There was no significant difference in DBP (p=0.764). SBP was significantly higher in women compared to men, the p-value was 0.023. The proportion of participants with hypertension was 47.4% in the total population and was significantly higher in women compared to men at p<0.001.

4.1.3 The mean glucose of the participants was 5.35 mmol/l. There was no significant difference in glucose levels (p=0.190) between men and women. The proportion of participants with diabetes mellitus was 6.6% of the total population. The difference in the proportion of participants with diabetes mellitus was also not significant (p=0.147) between men and women.

4.1.4 The mean value of total cholesterol was 4.13 mmol/l. Total cholesterol level was significantly higher in women compared to men (p≤0.001). The proportion of participants who had high cholesterol was 20.3% in the total population and was significantly higher in women compared to men (p=0.021). The mean value of LDL-c in the total study population was 2.25mmol/l and was significantly higher in women compared to men (p≤0.001). The proportion of participants who had high LDL-c levels was 41.2% and was significantly higher in women compared to men (p≤0.001). Likewise, HDL-c level was significantly lower in women compared to men (p≤0.001). The proportion of participants who had low HDL-c levels was 30.2% and there was no significant difference between women and men (p=0.559). The mean value of triglycerides was 0.95 mmol/l. There was no significant difference in triglyceride levels between women compared to men (p=0.143). The proportion of participants who had high triglyceride was 18.2%. The difference in the proportion of participants who had high triglycerides was also not significant between women and men (p=0.065).

4.1.5 The proportion of participants who were smokers was 30.5%. There were significantly more smokers among men compared to men as p≤0.001. The proportion of participants who were consuming alcohol was 27.2%. Significantly, more men were consuming alcohol compared to women as p≤0.001.

4.1.6 The proportion of participants with no risk factors was 12.8% in the total study population and was significantly lower in men compared to women (p <0.001). The proportion of participants with one risk factor was 27.7% in the total study population and was significantly higher in women compared to men (p=0.010).

The proportion of participants with two risk factors was 37.7% in the total study population and was significantly higher in men compared to women (p=0.003). The proportion of participants with three or more risk factors was 21.9% in the total study population, men had a significantly higher proportion of three or more risk factors as compared to women with p=0.024.

4.1.7 The proportion of participants with no formal education was 9.2% of the total population and there was no significant difference in the proportion of participants with no formal education between men and women (p=0.153). The proportion of participants with primary education was 35.5%of the total study population and there was no significant difference in the proportion of participants with primary education between men and women (p=0.804).

The proportion of participants with secondary education was 52.9% of the total study population and there was no significant difference in the proportion of participants with secondary education between men and women (p=0.994). The proportion of participants with tertiary education was 2.5% of the total study population and there was no significant difference in the proportion of participants with tertiary education between men and women (p=0.057).

4.1.8 The proportion of participants who were single was 20.9% of the total study population and there was no significant difference in the proportion of single participants between men and women (p=0.773). The proportion of participants who were married was 46.8% of the total study population and there was no significant difference in the proportion of married participants between men and women (p=0.516).

The proportion of participants who were divorced was 2.9% of the total study population and there was no significant difference in the proportion of divorced participants between men and women (p=0.069). The proportion of widowed participants was 16.9% of the total study population and there was a significantly higher proportion of widows in women than in men (p=<0.001).

4.1.9 The proportion of participants who were employed was 31.3% of the total study population and there was a significantly higher proportion of employment in men than in women (p=0.054).

## 4.2 **The distribution of cardiovascular disease risk factors amongst the village clusters in the Dikgale, Mamabolo, Mothapo health and demographic surveillance site (DIMAMO HDSS).**

### 4.2.1 Presentation of the DIMAMO HDSS villages

The purpose of this section is mainly to compare the distribution of cardiovascular disease risk factors amongst the village clusters. The participants were from 12 villages, but some villages had a very low number of participants, so the villages were then grouped into 4 clusters. The villages were grouped according to proximity to each other and approximation of equal participant numbers per cluster. The total number of participants was 791 and thus the number of participants in clusters A, B, C and D was 196, 208, 180 and 207 respectively. Figure 4 shows how the DIMAMO villages are situated and how they were grouped.

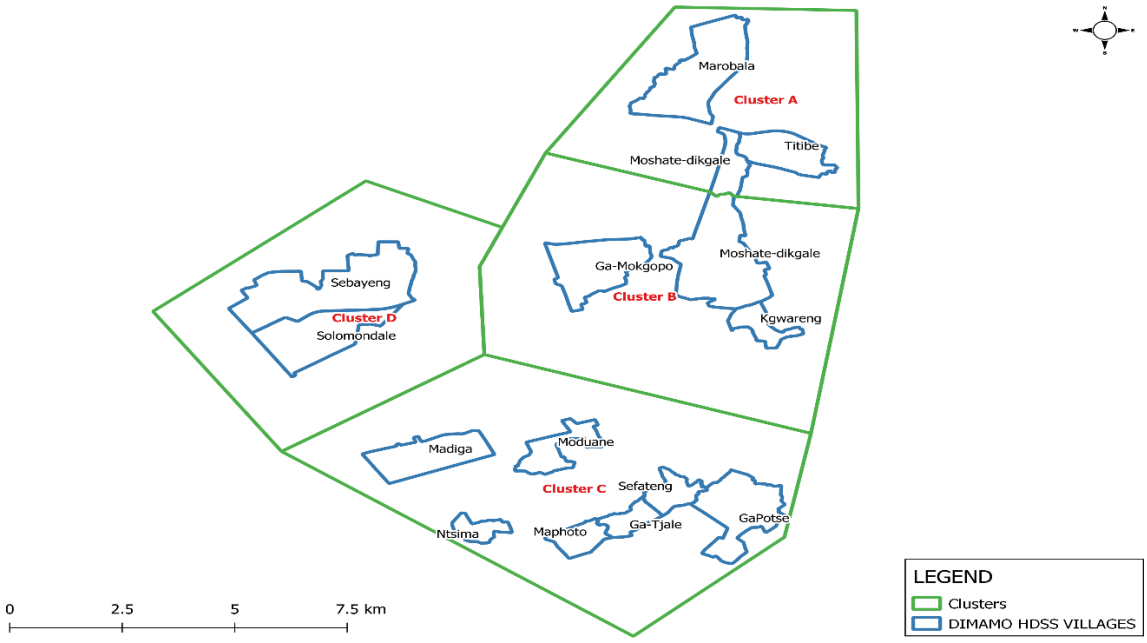



**Figure 4: DIMAMO villages**

**Table 2: distribution of characteristics by village clusters.**

Table 2 shows how the characteristics of the participants are distributed within the village clusters.

| Variable | Total population | Cluster A | Cluster B | Cluster C | Cluster D | P-value |
|---|---|---|---|---|---|---|
| **N** | 791 | 196 | 208 | 180 | 207 | - |
| **Female ratio ((%) n)** | 68.4 (541) | 75.5 (148) | 71.2 (148) | 65.0 (117) | 65.7 (136) | 0.083 |
| **Age (years)** | 52.47±8.24 | 52.11±8.15 | 52.77±7.78 | 52.99±8.34 | 52.0±8.70 | 0.604 |
| **Waist circumference (cm)** | 90.15±16.07 | 91.07±16.59 | 92.43±15.48 | 88.59±16.47 | 88.35±15.56 | 0.027 |
| **High Waist circumference ((%) n)** | 60.8 (481) | 63.8 (125) | 69.2 (144) | 56.7 (102) | 53.1 (110) | <0.001 |
| **Obesity ((%) n)** | 35.4 (280) | 40.3 (79) | 41.3 (86) | 29.4 (53) | 30.0 (62) | 0.013 |
| **Central obesity ((%) n)** | 59.3 (471) | 63.3 (124) | 67.3 (140) | 55.6 (100) | 51.7 (107) | 0.005 |
| **Hypertension ((%) n)** | 47.4 (375) | 51.0 (100) | 49.3 (103) | 42.2 (76) | 46.6 (96) | 0.354 |
| **Diabetes mellitus ((%) n)** | 6.3 (50) | 7.1 (13) | 6.1 (13) | 6.8 (12) | 6.3 (12) | 0.887 |
| **Total cholesterol (mmol/l)** | 4.10±1.2 | 4.20±1.01 | 4.36±1.63 | 4.08±1.23 | 3.93±1.36 | 0.345 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **High total cholesterol ((%) n)** | 20.3 (161) | 20.6 (40) | 21.7 (45) | 20.7 (37) | 18.0 (37) | 0.821 |
| **Triglycerides (mmol/l)** | 1.12±0.64 | 1.04±0.64 | 0.88±0.25 | 1.23±0.84 | 0.94±0.49 | 0.770 |
| **High triglycerides ((%) n)** | 18.6 (147) | 19.6 (38) | 18.2 (38) | 18.0 (32) | 18.4 (38) | 1.023 |
| **HDL-c (mmol/l)** | 1.18±0.41 | 1.18±0.35 | 1.15±0.20 | 1.15±0.36 | 1.07±0.42 | 0.134 |
| **Low HDL-c ((%) n)** | 41.2 (326) | 53.3 (104) | 37.1 (77) | 41.7 (75) | 33.8 (70) | <0.001 |
| **LDL-c(mmol/l)** | 2.52±1.03 | 2.36±0.95 | 2.88±1.44 | 2.52±1.07 | 2.69±1.29 | <0.001 |
| **High LDC-c ((%) n)** | 30.2 (239) | 24.7 (48) | 31.9 (66) | 33.2 (61) | 30.2 (63) | 0.291 |
| **Smoking ((%) n)** | 18.8 (149) | 14.8 (29) | 17.3 (36) | 18.3 (33) | 24.6 (51 | 0.817 |
| **Alcohol consumption ((%) n)** | 33.7 (267) | 20.0 (39) | 26.5 (55) | 21.9 (39) | 31. 6 (65) | 0.063 |
| **No formal education ((%) n)** | 9.2 (73) | 7.1 (14) | 6.8 (14) | 11.1 (20) | 12.1 (25) | 0.015 |
| **Primary education ((%) n)** | 35.3 (279) | 34.7 (68) | 36.2 (75) | 37.8 (68) | 32.9 (68) | 0.768 |
| **Secondary education ((%) n)** | 52.9 (415) | 54.1 (106) | 54.1 (112) | 50.0 (90) | 53.1 (110) | 0.839 |
| **Tertiary education ((%) n)** | 2.5 (20) | 4.1 (8) | 2.9 (6) | 1.1 (2) | 1.9 (4) | 0.285 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **Not married ((%) n)** | 20.9 (165) | 17.9 (35) | 19.7 (41) | 21.7 (39) | 24.2 (50) | 0.446 |
| **Married ((%) n)** | 46.8 (370) | 51.5 (101) | 47.1 (98) | 48.9 (88) | 40.1 (83) | 0.121 |
| **Divorced ((%) n)** | 2.9 (23) | 3.1 (6) | 3.8 (8) | 2.8 (5) | 1.9 (4) | 0.712 |
| **Widowed ((%) n)** | 16.9 (134) | 16.3 (32) | 19.2 (40) | 17.2 (31) | 15.0 (31) | 0.704 |
| **Employment ((%) n)** | 31.3 (248) | 33.7 (66) | 30.8 (64) | 27.2 (49) | 33.2 (69) | 0.527 |

*p-value≤0.05=there is a significant difference in observed results, p-value>0.05=no significant difference.*

4.2.2 Distribution of age

The mean age of participants among the clusters was 52.47. There was no significant difference in mean age between the village clusters (p=0.604).

4.2.3 Distribution of hypertension

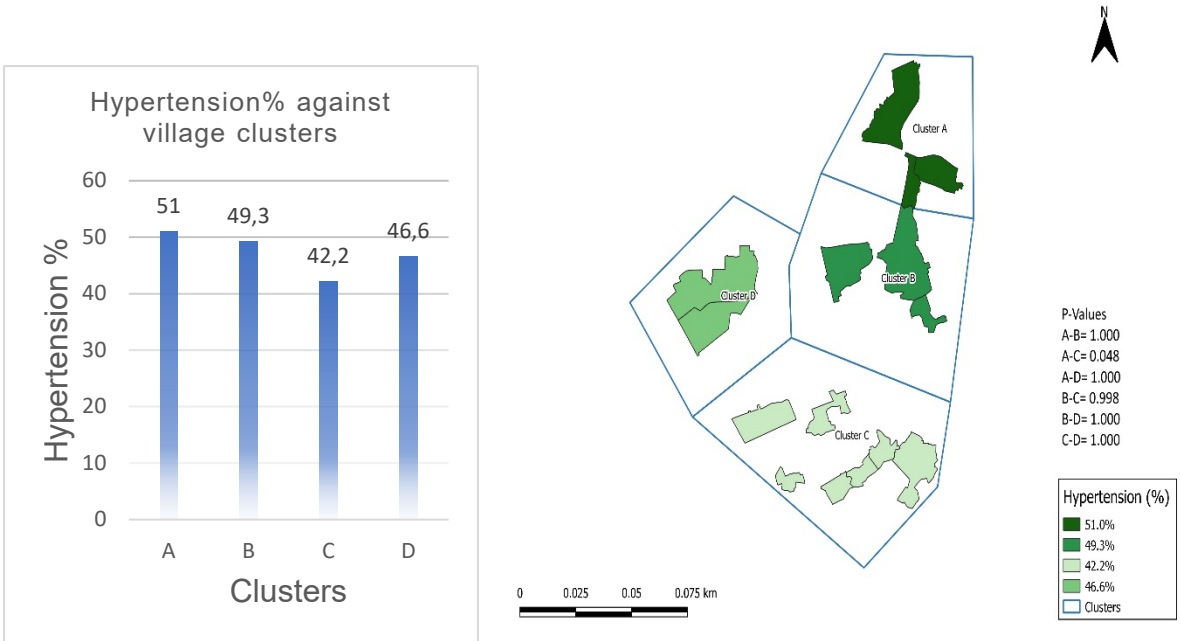



**Figure 5**: Distribution of hypertension among village clusters

**Figure 5** shows that hypertension was prevalent in the population as it was noted in the figure (47.5% had hypertension). There was no significant difference in the proportion of hypertension among the clusters (p-value>0.05). However, the difference in the proportion of hypertension was only significantly higher in cluster A compared to cluster C (p=0.048).

Binary regression analysis showed that participants with high glucose were significantly 3.970 times more likely to have hypertension and those with high waist circumference were significantly 2.476 times more likely to have hypertension, furthermore, binary regression analysis showed that participants who were smokers were 0.676 less likely to have hypertension (Appendix III: Table 3.1). This was supported by multivariate regression analysis which also showed that participants with high glucose levels were 3.157 times more likely to have hypertension, furthermore, participants with high waist circumference were 2.770 times more likely to have hypertension, furthermore, multivariate regression analysis showed that participants who were smokers were 0.557 times less likely to have hypertension (Appendix III: Table 3.2).

### 4.2.4 Distribution of diabetes mellitus

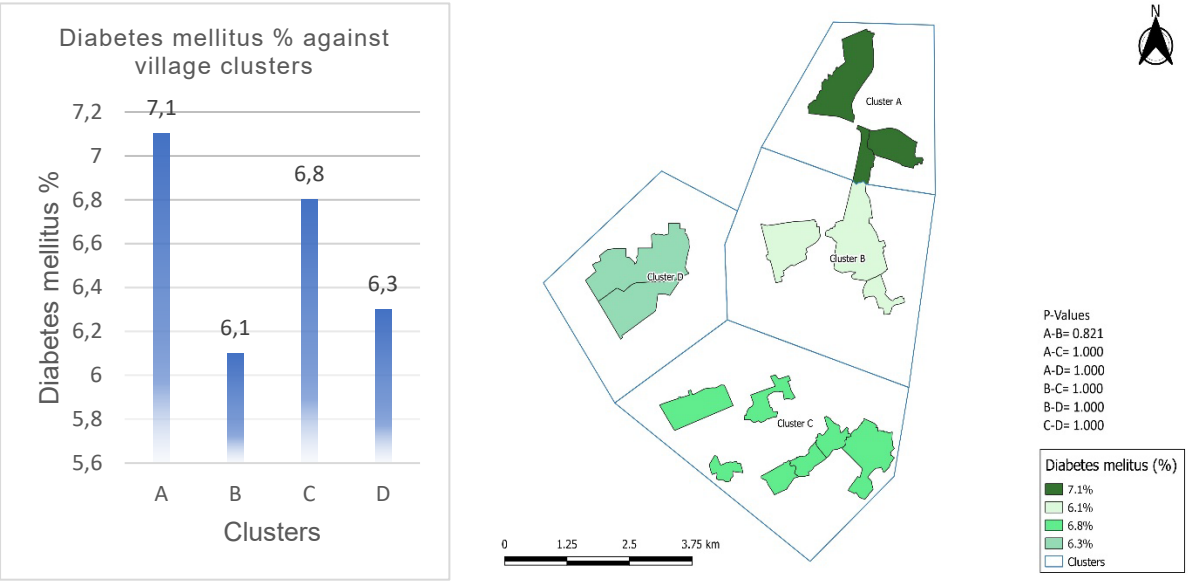



**Figure 6**: Distribution of diabetes mellitus among village clusters

Figure 6 shows that diabetes mellitus was not as prevalent in the population as it was noted in the figure (about 6% of the population had diabetes mellitus). There was no

significant difference in the proportion of diabetes mellitus among all clusters as the p-values were all greater than 0.05.

Binary logistic regression showed that participants with high waist circumference were significantly 3.136 times more likely to have diabetes mellitus (Appendix III: Table 3.3). This was supported by multivariate regression analysis which also showed that participants with high waist circumference were 2.996 times more likely to have diabetes mellitus (Appendix III: Table 3.4).

4.2.5 Distribution of dyslipidaemia.

The proportion of high cholesterol was 20.3%, there was no significant difference in high cholesterol proportion among the clusters (p=0.821). The proportion of triglycerides was 18.6%, there was no significant difference in triglyceride proportion among the clusters (p=1.023). The proportion of low HDL-c was 41.2%, there was a significantly higher proportion of low HDL-c in cluster A compared to other clusters (p<0.001). The total proportion of high LDL-c was 30.2%, there was no significant difference in the proportion of high LDL-c among clusters (p=0.291).

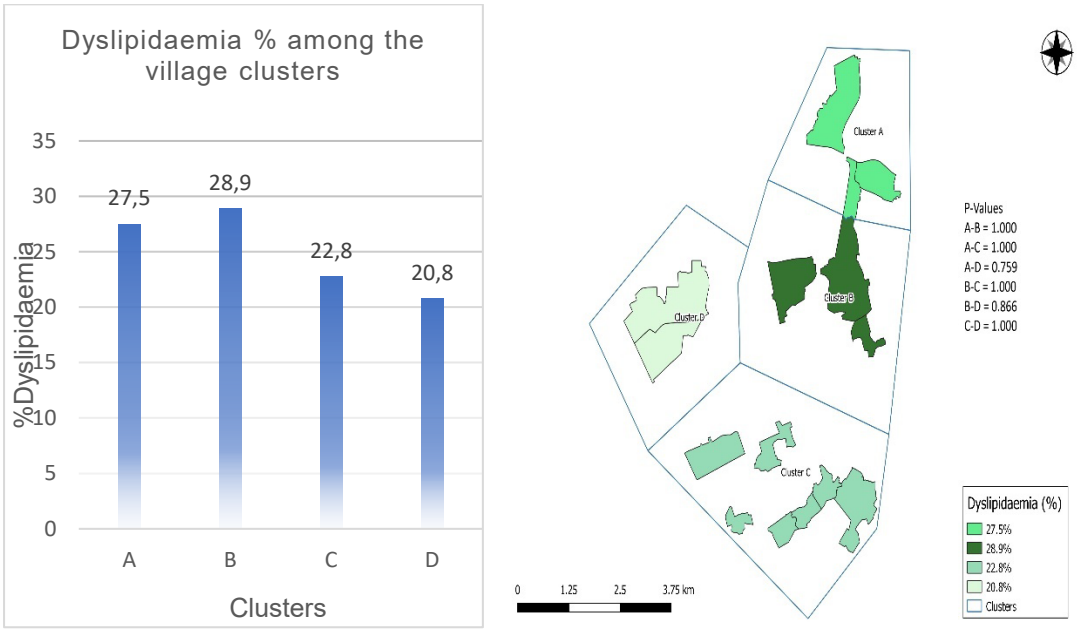



**Figure 8**: Distribution of dyslipidaemia among village clusters

Figure 8 shows that the overall proportion of dyslipidaemia among the clusters was about 25%. There was no significant difference in the proportion of dyslipidaemia among the individual clusters as p>0.05 among all groups.

Binary logistic regression showed that participants with high BMI were significantly 2.058 times more likely to have dyslipidaemias (Appendix III: Table 3.5). This was supported by multivariate regression analysis which also showed that participants with high BMI were 2.973 times more likely to have dyslipidaemias (Appendix III: Table 3.6).

### 4.2.6 Distribution of obesity

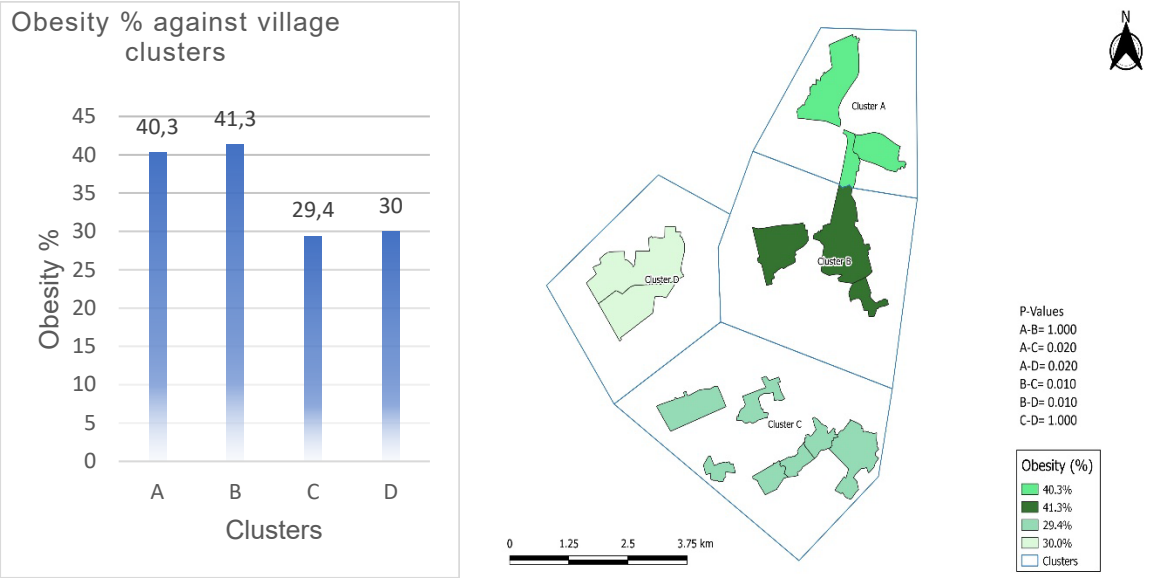



**Figure 7**: Distribution of obesity among village clusters

Figure 7 shows that the population had 35.4% of people with obesity. There was no significant difference in the proportion of obesity in clusters A & B. However, there was a significantly higher percentage of obesity in cluster A than C, cluster A than D, cluster B than C and cluster B than D with p-values of 0.020, 0.020, 0.010, and 0.010 respectively.

Binary logistic regression showed that participants with high LDL-c were significantly 2.147 times more likely to have obesity and those with high blood pressure were significantly 2.006 times more likely to have obesity (Appendix III: Table 3.7). This was supported by multivariate regression analysis which also showed that participants with high LDL-c were 1.746 times more likely to have obesity and those with high blood pressure were 1.435 times more likely to have obesity (Appendix III: Table 3.8).

Binary regression analysis showed that alcohol consumers were significantly 0.595 times less likely to have obesity (Appendix III: Table 3.7). However, multivariate

regression analysis showed that alcohol consumers were 1.532 times more likely to have obesity (Appendix III: Table 3.8).

4.2.7 Distribution of smoking.

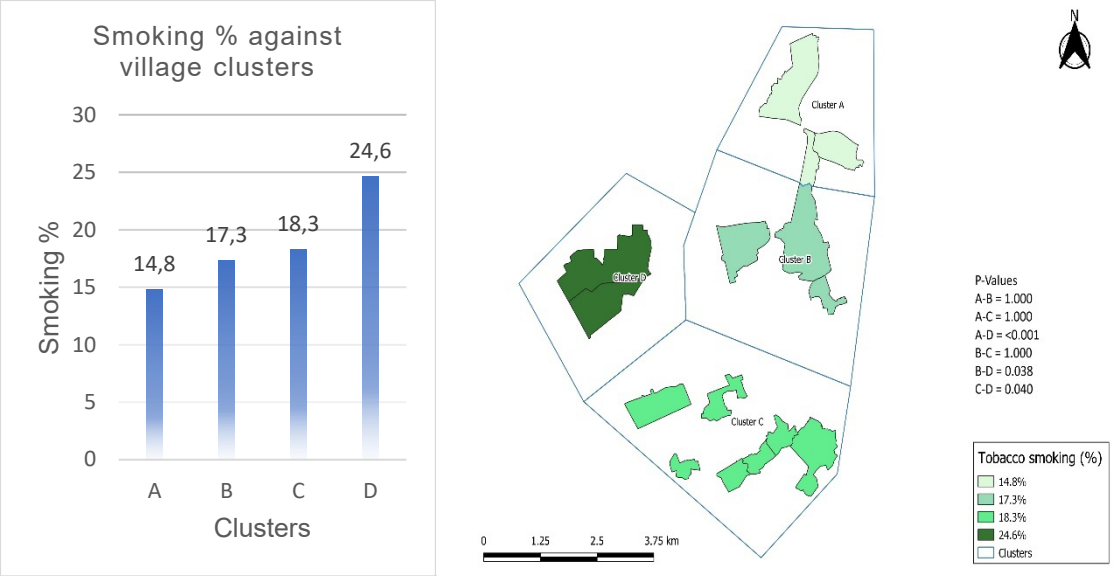



**Figure 9**: Distribution of smoking among village clusters

Figure 9 shows that the percentage of smokers was about 18% in the population as noted in the figure. There was no significant difference in the percentage of smokers in clusters A & B, A & C and B & C as the p-value > 0.05. there was a significantly higher percentage of smokers in cluster D than in A, D than in B and D than in C with p-values of <0.001, 0.038 and 0.040 respectively.

Binary logistic regression showed no association between smoking and level of education and marital status (Appendix III: Table 3.9). However, multivariate regression analysis showed that married participants were 0.541 times less likely to be smokers (Appendix III: Table 3.10).

4.2.8 Distribution of alcohol consumption.

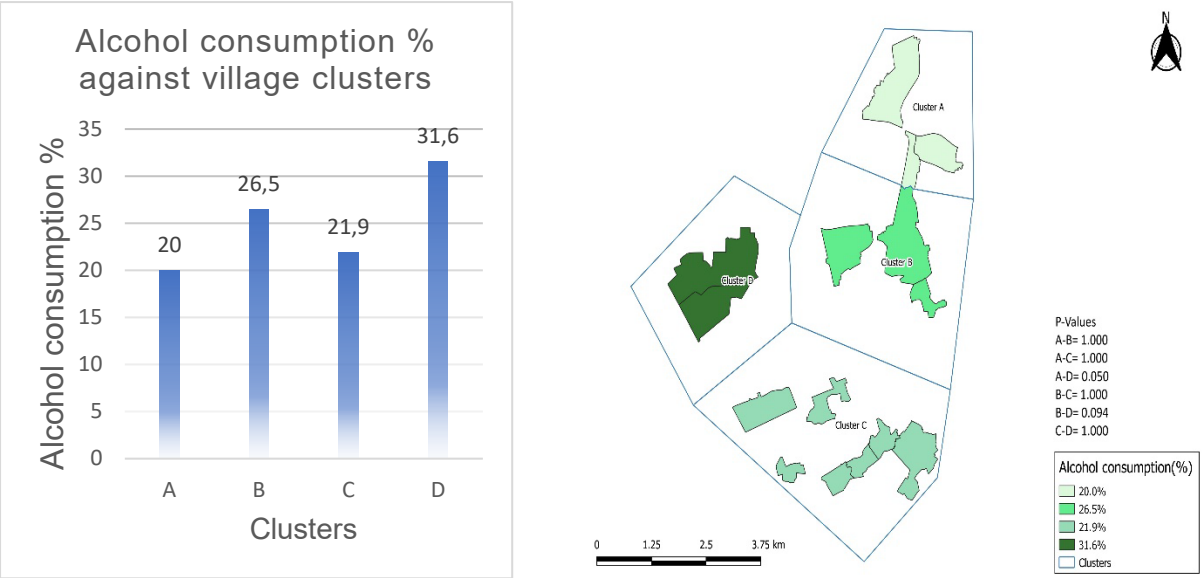



**Figure 10**: Distribution of alcohol consumption among village clusters

Figure 10 shows that the proportion of people consuming alcohol was about 27% as noted in the figure. There was no significant difference in alcohol consumption among all other clusters as the p-value > 0.050, except there was a significantly higher proportion of alcohol consumption in cluster D compared to A.

Binary logistic regression showed that single participants were significantly 0.453 times less likely to be alcohol consumers, furthermore, participants who had no formal education were significantly 5.830 times more likely to be alcohol consumers (Appendix III: Table 3.11). This was supported by multivariate regression analysis which also showed that single participants were 0.551 times less likely to be alcohol consumers and those with no formal education were 2.393 times more likely to be alcohol consumers (Appendix III: Table 3.12).

4.2.9 The proportion of participants who had no formal education was 9.2%. There was a significant difference in the proportion of participants who had no formal education in cluster A compared to clusters C and D (p=0.015). The proportion of participants who had primary education was 35.3%. There was no significant difference in the proportion of participants who had primary education (p=0.768). The proportion of participants who had secondary education was 52.9%. There was no significant difference in the proportion of participants who had secondary education (p=0.839). The proportion of participants who had tertiary education was 2.5%. There

was no significant difference in the proportion of participants who had tertiary education (p=0.285).

4.2.10 The proportion of participants who were single (marital status) was 20.9%. There was no significant difference in the proportion of people who were single among the clusters (p=0.446). The proportion of participants who were married was 46.8%. There was no significant difference in the proportion of people who were married among the clusters (p=0.121). The proportion of participants who were divorced was 2.9%. There was no significant difference in the proportion of people who were married among the clusters (p=0.712). The proportion of widowed participants was 16.9%. There was no significant difference in the proportion of people who were widowed among the clusters (p=0.704).

4.2.11 The proportion of those who were employed was 31.3%. There was no significant difference in the proportion of participants who were married among the clusters (0.527).

### 4.3 Spatial distribution of the modelled CVD risk factors among the village clusters.

The figure below shows how the CVD risk factors are distributed among the village clusters.

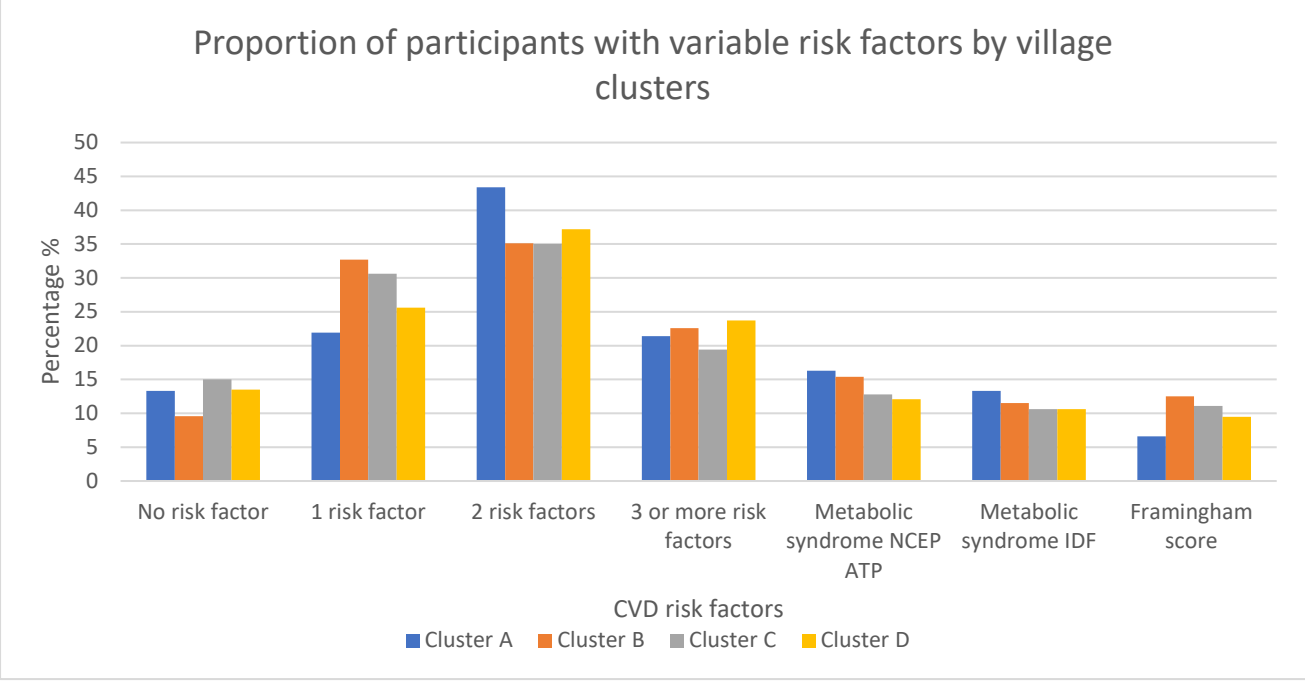


**Figure 11: Spatial distribution of the modelled CVD risk factors among the village clusters.**

4.3.1 Among the clusters, 12.8% had no risk factor for CVD and there was no significant difference in the proportion of participants with no risk factor for CVD (p=0.422). Among the clusters, 27.7% had 1 risk factor for CVD and there was no significant difference in the proportion of participants with 1 risk factor for CVD (p=0.071).

4.3.2 Among the clusters, 37.7% had 2 risk factors for CVD. A borderline significantly higher proportion of participants with 2 risk factors for CVD was observed in cluster A compared to cluster B (p=0.052). Also, a borderline significantly higher proportion of participants with 2 risk factors for CVD was cluster A compared to cluster C (p=0.054). Among the clusters, 21.9% of participants had 3 or more risk factors for CVD and there was no significant difference in the proportion of participants with 3 or more CVD risk factors between the village clusters (p=0.777).

4.3.3 Among the clusters, 14.2% of participants had metabolic syndrome according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criterion. There was no significant difference among the proportion of participants with metabolic syndrome defined by NCEP ATP III (p=0.565) between the village clusters. Cluster A had the highest proportion of participants with metabolic syndrome defined by NCEP ATP III.

Binary logistic regression showed that participants who were alcohol consumers were significantly 0.465 times less likely to have metabolic syndrome (NCEP ATP III) (Appendix III: Table 3.13). This was supported by multivariate regression analysis which also showed that participants who were alcohol consumers were 0.469 times less likely to have metabolic syndrome (NCEP ATP III) (Appendix IV Table 3.14).

Among the clusters, 11.5% of participants had metabolic syndrome according to the IDF definition. There was no significant difference among the proportion of participants who had metabolic syndrome defined by the International Diabetes Federation (IDF) (p=0.822). Cluster A had the highest proportion of participants with metabolic syndrome defined by IDF.

Binary logistic regression showed that participants who were alcohol consumers were significantly 0.357 times less likely to have metabolic syndrome (IDF) (Appendix III: Table 3.15). This was supported by multivariate regression analysis which also showed that participants who were alcohol consumers were 0.364 times less likely to have metabolic syndrome (IDF) (Appendix IV Table 3.16).

4.3.4 Among the clusters, 9.9% of participants were found to be at risk of developing cardiovascular disease in the next 10 years (high Framingham score). Cluster B had the highest proportion of participants with the risk of developing cardiovascular disease in 10 years.

Binary logistic regression showed that participants who were alcohol consumers were significantly 4.420 times more likely to develop CVD in the next 10 years (Appendix III: Table 3.17). This was supported by multivariate regression analysis which also showed that alcohol consumers were 4.331 times more likely to develop cardiovascular disease in the next 10 years (Appendix III: Table 3.18).

# CHAPTER 5

## 5. DISCUSSION

This section discusses the findings obtained from the present study about the characteristics of the study population and the spatial distribution of CVD risk factors, as well as the association between the CVD risk factors with the determinants of their spatial distribution.

### 5.1 Characteristics of the study population

The study aimed to determine the spatial distribution of cardiovascular disease risk factors in rural African areas, however, some community-dependent variables may affect the spatial distribution of these CVD risk factors. It is thus critical that the characteristics of participants are assessed before spatial distribution analysis.

5.1.1 Age and Gender.

The age of the participants who took part in this study was 40-65 years. Cardiovascular diseases and their risk factors usually present later in life (Thomas *et al.*, 2018). (Seki *et al.*, 2022) showed that as one gets older their chances of developing medical conditions increase, suggesting that their chances of developing CVDs increase too with ageing. Therefore, the age group used in this study is appropriate for the assessment of CVD risk.

In this study, there was a higher proportion of women participants compared to men. This agrees with studies conducted in the Dikgale area (Limpopo province) (Ntuli *et al.*, 2015) and sub-Saharan Africa (Nonterah *et al.*, 2021). A study conducted in Florida showed that although men agree to participate in health research studies, the number of them (men) who avail themselves to participate in the actual study is far too less compared to women (Otufowora *et al.*, 2021), hence women's participation in studies is often higher than men.

A study by the National Institutes of Health, 2021 (Kanaya *et al.*, 2022), showed that women wanted to participate in studies because they were interested in knowing more about their health status, to be observed/checked by health professionals/researchers and they were also grateful that through these studies, researchers can gain new

knowledge in health-related issues and then come up with possible and helpful interventions.

## 5.2 Spatial distribution of CVD risk factors in the DIMAMO population.

### 5.2.1 Spatial analysis of obesity and central obesity

In this study, the proportion of participants who were obese was over a third of the total population. The prevalence of obesity was found to be higher in women in this study than in men. This agrees with what was found in a study conducted by (Agyemang *et al.*, 2015) in Sub-Saharan Africa. A South African study showed that women have higher BMI compared to men, hence the high prevalence of obesity seen in women compared to men (Abarca-Gómez *et al.*, 2017).

A study conducted in America showed results that were similar to those found in this study, that the proportion of obesity was seen mostly in women compared to men (Chooi *et al.*, 2019). The reason why women tend to have increased chances of obesity was reported to be biological (Jastreboff *et al.*, 2019). Naturally, women have more lipid content compared to men, especially during childbearing periods (Jastreboff *et al.*, 2019).

In women, oestrogen plays a fundamental role in causing obesity. In reproductive women, oestrogen works with adipose tissue genes to increase gluteo-femoral subcutaneous adipose tissue mass and decrease central adipose tissue mass, this is a protective effect. However, after menopause, the reduction in levels of oestrogen together with aging, increases the total adipose tissue mass (Leeners *et al.*, 2017).

The proportion of obesity in this study was higher in cluster B and lower in cluster C.

In this study, both binary and multivariate regression analysis showed that there was a positive association between gender and alcohol consumption with obesity. In this study, binary and multivariate regression analysis showed that there was no association between obesity and level of education, marital status and employment status.

### 5.2.2 Spatial distribution of hypertension

In this study, the proportion of participants who had hypertension was about half of the total population. The proportion of hypertension in women was higher in this study than the proportion of hypertension in men, which is in contrast with what was reported

in the study conducted in Kenya (Mohamed *et al.*, 2018). The (Mohamed *et al.*, 2018) study showed that participants were mostly young adults (18-29 years), compared to the present study (40-65-year-olds). South African women have increased BMI (overweight, obese) compared to women in Kenya, and an increase in BMI is a risk factor for hypertension, this is another reason why South African women have a high prevalence of hypertension compared to women in Kenya (Mohamed *et al.*, 2018) as it was reported above that more women were obese as compared to men in this present study.

Hypertension showed a significantly higher proportion in cluster A compared to cluster C (which had the lowest proportion of participants with hypertension).

In this study, although not significant, participants from cluster A had the highest proportion of both hypertension and high glucose levels. Binary regression analysis showed a positive association between high glucose levels and hypertension, this was then supported by multivariate regression analysis which also showed a positive association between high glucose levels and hypertension. A study conducted by (Bosu, 2016), showed that there was a strong association between hypertension and diabetes mellitus (high glucose levels), in most cases, people with diabetes mellitus often present with increased levels of systolic blood pressure (SBP) and/or diastolic blood pressure (DBP). The (Bosu, 2016) study was in agreement with what was found in this study, meaning that diabetes mellitus may have been a determinant of hypertension in this study.

In this study, obesity may have not contributed to the proportion of hypertension because the proportion of hypertension was higher in cluster A while the proportion of obesity was higher in cluster B. This was supported by both binary and multivariate regression analysis that there was no association between hypertension and obesity. Hypertension was shown to have a positive association with obesity (Kotsis *et al.*, 2018), however, in this study, obesity may have not been a determinant for the distribution of hypertension.

Binary regression analysis in this study showed that there was a positive association between a high waist circumference and hypertension and this was also supported by the multivariate regression analysis. Hypertension was found to have a positive

association with high waist circumference (Id *et al.*, 2019) This suggests that central obesity was a better predictor of hypertension as compared to general obesity.

Both binary and multivariate regression analyses showed that there was no association between alcohol consumption and hypertension. This means that participants who were consuming alcohol were less likely to have hypertension. The commonly consumed alcohol by the participants in the study area used in the present study was found to be the African traditional beer (Choma et al., 2007). According to (Olas *et al*., 2020), the effect of alcohol consumption on health is dependent on the constituents of the alcohol and the chemical components. The micronutrients and elements found in traditional beer may be protective of hypertension. This may thus reduce the chances of hypertension.

In this study, women participants were higher in cluster A compared to women participation in the other clusters, this may have resulted in a higher proportion of hypertension in cluster A. This was supported by binary regression analysis and a study conducted by (Mills *et al.,* 2020), which reported that hypertension was seen mostly in the women population compared to men (Mills *et al.,* 2020). Therefore, this suggests that female gender may have been a determinant for the distribution of hypertension in this study.

5.2.3 Spatial distribution of diabetes mellitus.

In the present study, the proportion of participants with diabetes mellitus was found to be less than ten percent of the total population. The proportion of participants with diabetes mellitus was found to be significantly higher in women compared to men in this study, this agreed with what was reported in a study conducted in South Africa (Erzse *et al.*, 2019).

The highest proportion of diabetes mellitus in this study was seen in cluster A, and the lowest proportion of diabetes mellitus was seen in cluster B (However, the difference between the proportion of diabetes mellitus in cluster A and cluster B was not significant).

Although not significant, the proportion of high waist circumference in this study was also seen to be higher in cluster A. This was supported by both binary and multivariate regression analysis that showed a positive association between high waist circumference and diabetes mellitus. A study by (Aras *et al.,* 2021), showed that there

was a positive relationship between diabetes mellitus and an increased waist circumference. This suggests that high waist circumference may have been a determinant of diabetes mellitus and hence the proportion of diabetes mellitus was high in cluster A villages.

5.2.4 Spatial distribution of dyslipidaemia.

In the present study, dyslipidaemia was defined as having one or more of the following (high LDL-c, low HDL-c, high triglycerides, and/or high total cholesterol).

In the present study, the prevalence of dyslipidaemia was found to be about a fifth of the total population. The proportion of dyslipidaemia was increased in women than in men, this agreed with what was found in a study conducted in Malaysia (CM *et al.*, 2020). Another study conducted in South Africa also found that dyslipidaemia was more common in women (Mansfield *et al.,* 2022). In the present study, it was found that obesity was common among the women population. According to a study conducted in Slovenia by (Močnik *et al.,* 2023), the majority of the patients who were obese were also found to have dyslipidaemia, this suggests that obesity in women may have contributed to the high distribution of dyslipidaemia as compared to men in this study.

The proportion of dyslipidaemia in this study was higher in cluster B and lower in cluster D. The proportion of obesity (high BMI) was also increased in cluster B. This suggests that participants with high BMI were positively associated with dyslipidaemia. This was supported by both binary and multivariate analysis which showed that a high BMI was positively associated with dyslipidaemia. A study by (Marques-vidal *et al.*, 2023) showed that there was a strong association between dyslipidaemia and obesity (Marques-vidal *et al.*, 2023). This suggests that obesity may be a determinant in the distribution of dyslipidaemia.

Elderly adults were seen to have a greater chance of having dyslipidaemia due to their age (Noubiap *et al.*, 2018) but in this study, age may have not been a determinant for dyslipidaemia since there was no significant difference in the proportion of age in all clusters and again no significant difference in the mean age of participants. Both binary and multivariate regression also showed that there was no association between age and dyslipidaemia in this study population.

5.2.5 Spatial distribution of alcohol consumption

In the present study, the proportion of alcohol consumption was found to be a third of the total population. The proportion of alcohol consumption was higher in men compared to women. This agreed with other studies conducted in South Africa and in the same study area as the present study (Mashinya *et al.*, 2018), (Pheiffer *et al.*, 2021). This was also in agreement with a global study conducted by Amari, 2020, which showed that the prevalence of alcohol consumption was higher in men compared to women.

In this study, the proportion of alcohol consumption was found to be higher in cluster D and lower in cluster A.

In the present study, the proportion of people who had no formal education was higher in cluster D (However, the difference was not significant compared to other clusters). This suggests that the level of education may affect the distribution of alcohol consumption.

Binary logistic regression showed that there was a positive association between participants with no formal education and alcohol consumption. The positive association between no formal education and alcohol consumption was supported by multivariate regression analysis. A study conducted by (Nyirenda *et al.*, 2019), showed that there was a positive association between low levels of education (no formal education) and alcohol consumption.

In this study, the proportion of single participants was seen to be higher in cluster D compared to all the other clusters. This suggests that single participants tend to consume alcohol more than those with other different marital statuses. However, both binary and multivariate regression analysis showed that there was no association between being single and alcohol consumption. A study by (Olas *et al.,* 2020) showed that there was no association between being single and alcohol consumption.

5.2.6 Spatial distribution of smoking

In the present study, the prevalence of smoking was found to be about a fifth of the total population. The proportion was found to be higher in men compared to women in this study. This is in agreement with what was reported globally and among the various African populations (Boua *et al.*, 2021). The reason why men smoke more than women may be cultural or social. Men smoke more than women because they feel that if they

don’t smoke, they are not considered real men by society, also men do not have to keep their bodies healthy due to childbearing reasons (Davey *et al.,* 2020).

In this study, the proportion of smoking was found to be higher in cluster D and lower in cluster A.

In the present study, the proportion of participants who had no formal education was higher in cluster D (However, the difference was not significant compared to other clusters). This suggests that level of education may be associated with the distribution of smoking. Binary logistic regression showed no association between the level of education and marital status with smoking. However, after adjusting for known confounders (age and gender) in multivariate regression analysis it was shown that there was a negative association between married participants and smoking. (Maimela *et al.*, 2016) reported that there was a positive association between single and divorced marital status with smoking.

### 5.2.7 Distribution of CVD models

CVD risk factors do not act individually causing CVD but act in symphony (Damen *et al.*, 2016), therefore the CVD models are considered to be a better assessment of cardiovascular risk compared to assessment of individual factors.

#### 5.2.7.1 Spatial distribution of metabolic syndrome

In this study, the proportion of participants who had metabolic syndrome was less than a fifth of the total population and it was higher in cluster A.

Again, although not significant, there were more women participants in cluster A compared to other clusters. This suggests that women were more likely to have metabolic syndrome than men. A study showed that metabolic syndrome was higher among women compared to men (Huang, 2009). Binary and multivariate regression analysis showed that there was no association between gender and metabolic syndrome, which disagreed with what was reported by (Huang, 2009).

In this study, binary and multivariate regression analysis showed that there was a negative association between alcohol consumption and metabolic syndrome diagnosed by either NCEP ATP III or IDF criteria.

Binary and multivariate regression analysis showed that there was no association between the level of education, marital status and employment status with metabolic syndrome.

Metabolic syndrome is diagnosed by using certain CVD risk factors, therefore village clusters that had a higher proportion of general CVD risk factors are thus expected to have a higher proportion of metabolic syndrome.

The proportion of participants with hypertension and diabetes mellitus was higher in cluster A and it was noted that hypertension and diabetes mellitus form part of metabolic syndrome (Fahed *et al.*, 2022), this may have contributed to the higher distribution of metabolic syndrome in cluster A. Factors that affect the distribution of individual risk factors may thus indirectly determine the distribution of metabolic syndrome.

5.2.7.2 Spatial distribution of high Framingham score

In this study, the proportion of participants who had higher Framingham score was seen in cluster B.

Binary logistic regression showed a positive association between smoking and the chances of developing CVD in the next 10 years. This was supported by multivariate regression analysis which also showed a positive association between smoking and the chances of developing CVD in the next 10 years.

Binary and multivariate regression analysis showed that there was no association between the level of education, marital status and employment status with a high Framingham score.

# CHAPTER 6

## 6. CONCLUSION, LIMITATIONS AND RECOMMENDATIONS.

This section gives an overview of the conclusion of the study, limitations and recommendations.

### 6.1 CONCLUSION

The study aimed to determine the spatial distribution of cardiovascular disease (CVD) risk factors while the objectives were to determine the prevalence of CVD risk factors, to correlate the CVD risk factors with spatial analysis variables and to determine the determinants of the spatial distribution of CVD risk factors.

In the present study, the proportion of hypertension was higher in cluster A. The determinants that may have contributed to a higher proportion distribution of hypertension in cluster A are age, gender (women), central obesity (high waist circumference) alcohol consumption, diabetes mellitus. Binary and multivariate regression analysis showed that diabetes mellitus and central obesity may have contributed to the distribution of hypertension.

Although not significant, the proportion of diabetes mellitus was higher in cluster A. It was found that high waist circumference may have been a determinant for the distribution of diabetes mellitus in this population. Binary and multivariate regression analysis showed that high waist circumference was positively associated with diabetes mellitus.

The proportion of dyslipidaemia was seen to be higher in cluster B. The determinant that may have contributed to the high proportion of dyslipidaemia was found to be obesity. Binary and multivariate analysis showed that obesity was a determinant for the distribution of dyslipidaemia in this study.

The proportions of alcohol consumption and smoking were seen to be higher in cluster D. The determinants that may have contributed to alcohol consumption and smoking include low levels of education, unemployment and marital status. Binary and multivariate regression analysis showed a positive association between alcohol consumption and no formal education, however, both regression analyses showed no association between smoking and the above-mentioned social determinants.

The proportion of metabolic syndrome was higher in cluster A. Binary and multivariate regression analysis showed no association between metabolic syndrome and level of education, marital status, employment status.

In this study, the proportion of a higher Framingham score was seen in cluster B. Binary and multivariate regression analysis showed no association between a higher Framingham score and level of education, marital status, employment status.

Biomedical CVD risk factors (hypertension, obesity, dyslipidaemia, diabetes mellitus) were more common among women and in clusters A and B whilst behavioural CVD risk factors (smoking and alcohol consumption) were more common among men and in cluster D. Most of the cardiovascular disease risk factors were more common in village clusters A and B. The reason for this may be older age, gender and behavioural risk factors.

The overall conclusion from the present study is that different cardiovascular risk factors are distributed differently in different village cluster. The distribution of these risk factors is determined by demographic factors such as age, gender, lifestyle factors such as smoking and alcohol consumption and biological factors such as obesity and or central obesity

## 6.2 LIMITATIONS

The cross-sectional study design did not allow the determination of incidence rates. This study used data from a retrospective study which used convenience sampling, therefore selection bias could not be avoided. Again, convenience sampling means that the results obtained may not be representative of the whole population as not all the villages have relatively the same denominator. To adjust for this, the villages were clustered in groups of almost relatively same denominator. However, the study may provide a picture of what could be found when random sampling was to be used.

There was an imbalance in gender distribution, women participants were higher than men participants. Although the researcher was aware of this during analysis of data, gender bias could not be avoided in this study.

## 6.3 RECOMMENDATIONS

To validate the findings obtained in the present study, a longitudinal study design is recommended where incidence rates could be assessed. Furthermore, a stratified

simple random sampling should be employed to ensure that all village clusters have relatively the same denominator.

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# APPENDICES

## APPENDIX I: Scholarship approval letter

higher education & training
Department:
Higher Education and Training
REPUBLIC OF SOUTH AFRICA

Private Bag X174, PRETORIA, 0001. 123 Francis Baard Street PRETORIA, 0002, South Africa
Tel: (012) 312 5911, Fax: (012) 321 6770
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*Enquiries: Ms S Andrease; Tel: 012 312 6039; e-mail: Andrease.S@dhet.gov.za*

Prof M Mokgalong
Vice-Chancellor
University of Limpopo
Private Bag X1106
**SOVENGA**
0727

By e-mail: frances.pratt@ul.ac.za

Dear Professor Mokgalong

**SELECTION OUTCOMES OF PARTICIPANTS FOR PHASE 1 OF THE NURTURING EMERGING SCHOLARS PROGRAMME SCHOLARSHIP AND INTERNSHIP**

The Department of Higher Education and Training (the Department) is pleased to inform you that the recommendation to award the Nurturing Emerging Scholars Programme (NESP) Scholarship and Internship (Phase 1) to the following candidates has been approved:

- Ms Murangi Mabel Ramarumo: Medical Microbiology
- Ms Maite Dineo Makgobatlou: Chemical Pathology
- Mr Tebogo Abie Mokwana: Mathematics Education

The University is requested to inform the successful candidates about the outcome of their applications and ensure that signed contracts (attached) are submitted to the Department within 30 days from receipt of this letter. The attached table provides details of the outcome for each position applied for.

This information should be sent to Ms Salvation Andrease (Andrease.S@dhet.gov.za).

Yours sincerely

**Dr T Lewin**
**Acting Deputy Director-General: University Education**



**Date: 16 March 2021**

## APPENDIX II: Ethics clearance certificate

**University of Limpopo**
**Department of Research Administration and Development**
**Private Bag X1106, Sovenga, 0727, South Africa**
**Tel: (015) 268 3935, Fax: (015) 268 2306, Email: tukiso.sewapa@ul.ac.za**

**TURFLOOP RESEARCH ETHICS COMMITTEE**

**ETHICS CLEARANCE CERTIFICATE**

**DATE:** **09 April 2024**

**PROJECT NUMBER:** **TREC/97/2023: PG-Renewed**

**PROJECT:**

| | |
|---|---|
| **Title:** | Determination of spatial distribution of cardiovascular disease risk factors at the Dikgale, Mamabolo, Mothiba Health and Demographic Surveillance Site, Limpopo Province, South Africa |
| **Researcher:** | MD Makgobatlou |
| **Supervisor:** | Mr SSR Choma |
| **Co-supervisor:** | Dr T Satekge |
| **School:** | School of Medicine |
| **Degree:** | Master of Science (Medical Sciences) |

**PROF D MAPOSA**
**CHAIRPERSON: TURFLOOP RESEARCH ETHICS COMMITTEE**

The Turfloop Research Ethics Committee (TREC) is registered with the National Health Research Ethics Council, Registration Number: **REC-0310111-031**

**Note:**

i) **This Ethics Clearance Certificate will be valid for one (1) year, as from the abovementioned date. Application for annual renewal (or annual review) need to be received by TREC one month before lapse of this period.**

ii) **Should any departure be contemplated from the research procedure as approved, the researcher(s) must re-submit the protocol to the committee, together with the Application for Amendment form.**

iii) **PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES.**

*Finding solutions for Africa*

**APPENDIX III: Table 3: Binary and multivariate regression analysis of determinants of cardiovascular disease risk factors.**

**Table 3.1: Binary regression analysis of determinants of hypertension.**

| | | Hypertension | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Age** | | 1.068 (1.048-1.088) | <0.001 |
| **Gender** | | 0.562 (0.413-0.766) | <0.001 |
| **Smoking** | | 1<br>0.676 (0.419-1.091) | -<br>0.010 |
| **Alcohol consumption** | | 1<br>1.154 (0.826-1.613) | -<br>0.402 |
| **Marital status** | Single | 1<br>1.092 (0.635-1.876) | -<br>0.751 |
| | Married | 1<br>1.460 (0.903-2.360) | -<br>0.123 |
| | Divorced | 1<br>1.768 (0.676-4.624) | -<br>0.245 |
| | Widowed | 1<br>1.129 (0.638-1.996) | -<br>0.677 |
| **Level of education** | No formal education | 1<br>0.939 (0.321-2.748) | -<br>0.909 |
| | Primary | 1<br>0.943 (0.354-2.515) | -<br>0.906 |
| | Secondary | 1<br>0.988 (0.378-2.579) | -<br>0.980 |
| | Tertiary | 1<br>0.736 (0.298-1.821) | -<br>0.507 |
| **High LDL-c** | | 1<br>1.119 (0.790-1.584) | -<br>0.527 |
| **High Cholesterol** | | 1<br>1.336 (0.847-2.107) | -<br>0.213 |
| **High glucose** | | 1 | - |

| | | |
|---|---|---|
| | 3.970 (1.909-8.254) | <0.001 |
| **High BMI** | 1<br>1.123 (0.752-1.678) | -<br>0.570 |
| **High Waist circumference** | 1<br>2.476 (1.646-3.722) | -<br><0.001 |
| **Employment** | 0.855 (0.625-1.170) | 0.328 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.2: Multivariate regression analysis of determinants of hypertension.**

| | | First model | | Last model | |
|---|---|---|---|---|---|
| **Covariates** | | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | | 1.067 (1.045-1.089) | <0.001 | 1.065 (1.044-1.087) | <0.001 |
| **Gender** | | 1.074 (0.633-1.822) | 0.792 | - | - |
| **Smoking** | | 0.568 (0.371-0.872) | 0.010 | 0.557 (0.381-0.814) | 0.003 |
| **Alcohol consumption** | | 1.287 (0.891-1.859) | 0.179 | - | - |
| **Marital status** | Single | 1.097 (0.512-1.966) | 0.757 | - | - |
| | Married | 1.020 (0.709-2.035) | 0.495 | - | - |
| | Divorced | 2.197 (0.785-6.146) | 0.134 | - | - |
| | Widowed | 0.982 (0.530-1.820) | 0.953 | - | - |
| **Level of education** | No formal education | 0.886 (0.291-2.693) | 0.831 | - | - |
| | Primary | 0.996 (0.366-2.712) | 0.994 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| | Secondary | 0.982 (0.370-2.602) | 0.970 | - | - |
| | Tertiary | 0.845 (0.289-2.114) | 0.825 | - | - |
| **High LDL-c** | | 1.043 (0.734-1.482) | 0.815 | - | - |
| **High Cholesterol** | | 1.269 (0.806-1.999) | 0.303 | - | - |
| **High glucose** | | 3.269 (1.552-6.884) | 0.002 | 3.157 (1.510-6.601) | 0.002 |
| **High BMI** | | 1.382 (0.916-2.085) | 0.123 | - | - |
| **High Waist circumference** | | 2.431 (1.566-3774) | <0.001 | 2.770 (2.003-3.830) | <0.001 |
| **Employment** | | | | | |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.3: Binary regression analysis of determinants of diabetes mellitus.**

| | | Diabetes mellitus | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Age** | | 1.043 (1.000-1.089) | 0.053 |
| **Gender** | | 0.605 (0.305-1.200) | 0.150 |
| **Smoking** | | 1<br>0.311 (0.064-1.522) | -<br>0.149 |
| **Alcohol consumption** | | 1<br>0.949 (0.473-1.907) | -<br>0.884 |
| **Marital status** | Single | 1<br>1.044 (0.451-3.744) | -<br>0.947 |
| | Married | 1<br>1.113 (0.358-3.460) | -<br>0.854 |

| | | | |
|---|---|---|---|
| | Divorced | 1<br>0.539 (0.051-5.683) | -<br>0.607 |
| | Widowed | 1<br>0.731 (0.190-2.807) | -<br>0.648 |
| **Level of education** | No formal education | 1<br>1.099 (0.422-2.862) | -<br>0.847 |
| | Primary | 1<br>1.005 (0.555-1.821) | -<br>0.987 |
| | Secondary | 1<br>1.078 (0.609-1.906) | -<br>0.798 |
| | Tertiary | 1<br>1.854 (0.278-1.721) | -<br>0.517 |
| **High LDL-c** | | 1<br>0.869 (0.443-1.705) | -<br>0.684 |
| **High Cholesterol** | | 1<br>1.039 (0.451-2.394) | -<br>0.928 |
| **High BMI** | | 1<br>0.707 (0.349-1.432) | -<br>0.336 |
| **High Waist circumference** | | 1<br>3.136 (1.161-8.468) | -<br>0.024 |
| **Employment** | | 1<br>0.691(0.351-1.360) | -<br>0.285 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.4: Multivariate regression analysis of determinants of diabetes mellitus.**

| | first step | | Last step | |
|---|---|---|---|---|
| **Covariates** | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | 1.046 (1.003-1.090) | 0.036 | 1.034 (0.996-1.073) | 0.083 |
| **Gender** | 1.335 (0.509-3.504) | 0.557 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| **Smoking** | | 0.349 (0.074-1.632) | 0.181 | - | - |
| **Alcohol consumption** | | 0.999 (0.509-1.959) | 0.997 | - | - |
| **Marital status** | Single | 1.023 (0.286-3.662) | 0.972 | - | - |
| | Married | 1.134 (0.365-3.522) | 0.827 | - | - |
| | Divorced | 0.532 (0.050-5.630) | 0.600 | - | - |
| | Widowed | 0.702 (0.184-2.677) | 0.604 | - | - |
| **Level of education** | No formal education | 1.099 (0.422-2.862) | 0.854 | - | - |
| | Primary | 1.005 (0.555-1.821) | 0.945 | - | - |
| | Secondary | 1.078 (0.609-1.906) | 0.787 | - | - |
| | Tertiary | 1.854 (0.278-1.721) | 0.517 | - | - |
| **High LDL-c** | | 0.862 (0.440-1.690) | 0.666 | - | - |
| **High Cholesterol** | | 1.018 (0.443-2.340) | 0.966 | - | - |
| **High BMI** | | 0.685 (0.342-1.373) | 0.286 | - | - |
| **High waist circumference** | | 2.893 (1.117-7.495) | 0.029 | 2.996 (1.298-6.919) | 0.010 |
| **Employment** | | 0.691(0.351-1.360) | 0.285 | - | - |

*$p<0.05$, $\beta$= beta-coefficient correlation*

**Table 3.5: Binary regression analysis of determinants of dyslipidaemia.**

| | | Dyslipidiaemia | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Age** | | 1.013 (0.985-1.042) | 0.366 |
| **Gender** | | 0.617 (0.407-0.937) | 0.023 |
| **Smoking** | | 1<br>0.821 (0.389-1.736) | -<br>0.606 |
| **Alcohol consumption** | | 1<br>0.853 (0.531-1.370) | -<br>0.510 |
| **Marital status** | Single | 1<br>0.604 (0.287-1.273) | -<br>0.185 |
| | Married | 1<br>0.932 (0.494-1.757) | -<br>0.827 |
| | Divorced | 1<br>0.939 (0.269-3.284) | -<br>0.922 |
| | Widowed | 1<br>0.730 (0.341-1.561) | -<br>0.417 |
| **Level of education** | No formal education | 1<br>0.728 (0.203-2.612) | -<br>0.626 |
| | Primary | 1<br>0.504 (0.160-1.594) | -<br>0.244 |
| | Secondary | 1<br>1.078 (0.609-1.906) | -<br>0.798 |
| | Tertiary | 1<br>0.854 (0.278-1.721) | -<br>0.517 |
| **High glucose** | | 1<br>0.630 (0.281-1.414) | -<br>0.263 |
| **High blood pressure** | | 1<br>1.142 (0.754-1.732) | -<br>0.531 |
| **High BMI** | | 1<br>2.058 (1.246-3.401) | -<br>0.005 |
| **High Waist circumference** | | 1 | - |

| | | |
|---|---|---|
| | 1.291 (0.695-2.398) | 0.419 |
| **Employment** | 1<br>1.175 (0.794-1.737) | -<br>0.420 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.6: Multivariate regression analysis of determinants of dyslipidaemia.**

| | | First step | | Last step | |
|---|---|---|---|---|---|
| **Covariates** | | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | | 1.015 (0.989-1.041) | 0.256 | - | - |
| **Gender** | | 0.677 (0.321-1.429) | 0.306 | - | - |
| **Smoking** | | 0.833 (0.432-1.607) | 0.586 | - | - |
| **Alcohol consumption** | | 810 (0.526-1.249) | 0.341 | - | - |
| **Marital status** | Single | 0.684 (0.339-1.380) | 0.289 | - | - |
| | Married | 0.978 (0.532-1.798) | 0.943 | - | - |
| | Divorced | 0.752 (0.365-1.550) | 0.440 | - | - |
| | Widowed | 0.959 (0.278-3.310) | 0.947 | - | - |
| **Level of education** | No formal education | 0.816 (0.239-2.787) | 0.745 | - | - |
| | Primary | 0.607 (0.199-1.857) | 0.380 | - | - |
| | Secondary | 0.732 (0.248-2.161) | 0.572 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| | Tertiary | 0.854 (0.278-1.721) | 0.517 | - | - |
| **High glucose** | | 0.631 (0.283-1.404) | 0.259 | - | - |
| **High blood pressure** | | 1.078 (0.726-1.600) | 0.710 | - | - |
| **High BMI** | | 1.917 (1.196-3.072) | 0.007 | 2.973 (1.608-3.343) | <0.001 |
| **High Waist circumference** | | 1.159 (0.666-2.017) | 0.602 | - | - |
| **Employment** | | 0.161 (0.199-0.640) | 0.420 | - | - |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.7: Binary regression analysis of determinants of obesity.**

| | | Obesity | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Age** | | 0.985 (0.958-1.013) | 0.286 |
| **Gender** | | 0.035 (0.017-0.072) | <0.001 |
| **Smoking** | | 1<br>0.602 (0.228-1.592) | -<br>0.306 |
| **Alcohol consumption** | | 1<br>0.595 (0.391-0.906) | -<br>0.016 |
| **Marital status** | Single | 1<br>0.903 (0.439-1.859) | -<br>0.782 |
| | Married | 1<br>1.399 (0.730-2.680) | -<br>0.321 |
| | Divorced | 1<br>0.308 (0.071-1.341) | -<br>0.117 |
| | Widowed | 1<br>1.514 (0.725-3.165) | -<br>0.270 |

| | | | |
|---|---|---|---|
| **Level of education** | No formal education | 1<br>0.504 (0.112-2.266) | -<br>0.371 |
| | Primary | 1<br>0.596 (0.148-2.401) | -<br>0.467 |
| | Secondary | 1<br>0.639 (0.163-2.511) | -<br>0.521 |
| | Tertiary | 1<br>0.564 (0.378-0.821) | -<br>0.534 |
| **Employment** | | 1<br>1.236 (0.872-1.751) | -<br>0.233 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.8: Multivariate regression analysis of determinants of obesity.**

| | | First step | | Last step | |
|---|---|---|---|---|---|
| **Covariates** | | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | | 1.066 (1.042-1.091) | <0.001 | 0.976 (0.950-1.002) | 0.073 |
| **Gender** | | 0.141 (0.055-0.361) | <0.001 | 0.153 (0.065-0.362) | <0.001 |
| **Smoking** | | 1.237 (0.378-4.048) | 0.725 | - | - |
| **Alcohol consumption** | | 0.516 (0.330-0.805) | 0.004 | 1.523 (0.340-1.805) | 0.003 |
| **Marital status** | Single | 1.097 (0.612-1.966) | 0.757 | - | - |
| | Married | 1.202 (0.709-2.035) | 0.495 | - | - |
| | Divorced | 2.197 (0.785-6.146) | 0.134 | - | - |
| | Widowed | 0.982 (0.530-1.820) | 0.953 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| **Level of education** | No formal education | 0.886 (0.291-2.698) | 0.731 | - | - |
| | Primary | 0.996 (0.366-2.712) | 0.994 | - | - |
| | Secondary | 0.982 (0.370-2.602) | 0.970 | - | - |
| | Tertiary | 0.564 (0.378-0.821) | 0.534 | - | - |
| **Employment** | | 0.212 (0.178-1.419) | 0.233 | - | - |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.9: Binary regression analysis of determinants of smoking.**

| | | Smoking | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Age** | | 0.953 (0.922-0.985) | 0.0004 |
| **Gender** | | 48.740 (26.769-88.743) | <0.001 |
| **Marital status** | Single | 1<br>1.134 (0.534-2.410) | -<br>0.743 |
| | Married | 1<br>0.636 (0.326-1.240) | -<br>0.184 |
| | Divorced | 1<br>2.313 (0.592-9.033) | -<br>0.228 |
| | Widowed | 1<br>1.323 (0.537-3.256) | -<br>0.543 |
| **Level of education** | No formal education | 1<br>0.729 (0.151-3.256) | -<br>0.694 |
| | Primary | 1<br>1.014 (0.261-3.941) | -<br>0.984 |
| | Secondary | 1<br>1.639 (0.163-2.511) | -<br>0.561 |
| | Tertiary | 1 | - |

| | | | |
|---|---|---|---|
| | | 1.564 (0.378-0.821) | 0.534 |
| **Employment** | | 1<br>1.229 (0.787-1.919) | -<br>0.366 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.10: Multivariate regression analysis of determinants of smoking.**

| | | First step | | Last step | |
|---|---|---|---|---|---|
| **Covariates** | | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | | 0.953 (0.922-0.985) | 0.004 | 0.962 (0.935-0.990) | 0.008 |
| **Gender** | | 8.740 (6.763-8.743) | <0.001 | 47.367 (26.469-84.764) | <0.001 |
| **Marital status** | Single | 1.134 (0.534-2.410) | 0.743 | - | - |
| | Married | 0.636 (0.326-1.240) | 0.184 | 0.541 (0.335-0.874) | 0.012 |
| | Divorced | 2.313 (0.592-9.033) | 0.228 | - | - |
| | Widowed | 1.323 (0.537-3.256) | 0.543 | - | - |
| **Level of education** | No formal education | 0.729 (0.151-3.525) | 0.694 | - | - |
| | Primary | 1.014 (0.261-3.525) | 0.984 | - | - |
| | Secondary | 0.678 (0.183-2.514) | 0.561 | - | - |
| | Tertiary | 0.845 (0.258-0.730) | 0.534 | - | - |
| **Employment** | | 0.206 (0.227-0.819) | -0.336 | - | - |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.11: Binary regression analysis of determinants of alcohol consumption.**

| Alcohol consumption | | | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Age** | | 0.974 (0.952-0.996) | 0.024 |
| **Gender** | | 13.807 (9.165-20.800) | <0.001 |
| **Marital status** | Single | 1 | - |
| | | 0.453 (0.246-836) | 0.011 |
| | Married | 1 | - |
| | | 0.603 (0.351-1.035) | 0.066 |
| | Divorced | 1 | - |
| | | 0.551 (0.182-1.668) | 0.292 |
| | Widowed | 1 | - |
| | | 0.773 (0.413-1.450) | 0.423 |
| **Level of education** | No formal education | 1 | - |
| | | 5.830 (1.655-20.410 | 0.006 |
| | Primary | 1 | - |
| | | 3.654 (1.143-11.679) | 0.068 |
| | Secondary | 1 | - |
| | | 2.526 (0.812-7.653) | 0.109 |
| | Tertiary | 1 | - |
| | | 0.494 (0.397-0.756) | 0.458 |
| **Employment** | | 1 | - |
| | | 1.178 (0.828-1.678) | 0.362 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.12: Multivariate regression analysis of determinants of alcohol consumption.**

| | First step | | Last step | |
|---|---|---|---|---|
| **Covariates** | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | 0.974 (0.952-0.996) | 0.024 | 0.973 (0.951-0.995) | 0.015 |

| | | | | | |
|---|---|---|---|---|---|
| **Gender** | | 13.807 (9.165-20.800) | <0.001 | 13.456 (9.035-20.040) | <0.001 |
| **Marital status** | Single | 0.453 (0.246-0.836) | 0.011 | 0.551 (0.341-0.889) | 0.015 |
| | Married | 0.603 (0.351-1.035) | 0.066 | - | - |
| | Divorced | 0.551 (0.182-1.668) | 0.292 | - | - |
| | Widowed | 0.773 (0.413-1.450) | 0.423 | - | - |
| **Level of education** | No formal education | 5.830 (1.665-20.410) | 0.006 | 2.393 (1.332-4.299) | 0.004 |
| | Primary | 3.654 (1.143-11.679) | 0.029 | - | - |
| | Secondary | 2.526 (0.812-7.852) | 0.109 | - | - |
| | Tertiary | 0.602 (0.738-0.791) | 0.588 | - | - |
| **Employment** | | 0.164 (0.180-0.829) | 0.362 | - | - |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.13: Binary regression analysis of determinants of metabolic syndrome by NCEP definition.**

| | Metabolic syndrome NCEP | |
|---|---|---|
| **Covariates** | 95% CI | p-value |
| **Age** | 0.981 (0.941-1.024) | 0.383 |
| **Gender** | 2.346 (0.742-7.417) | 0.147 |
| **Smoking** | 1<br>0.377 (0.064-2.228) | -<br>0.282 |
| **Alcohol consumption** | 1<br>0.465 (0.301-0.719) | -<br><0.001 |

| | | | |
|---|---|---|---|
| **Marital status** | Single | 1<br>0.394 (0.120-1.291) | -<br>0.124 |
| | Married | 1<br>0.333 (0.117-0.948) | -<br>0.039 |
| | Divorced | 1<br>0.163 (0.012-2.242) | -<br>0.175 |
| | Widowed | 1<br>0.436 (0.136-1.396) | -<br>0.162 |
| **Level of education** | No formal education | 1<br>0.286 (0.040-2.022) | -<br>0.210 |
| | Primary | 1<br>0.785 (0.144-4.294) | -<br>0.781 |
| | Secondary | 1<br>0.568 (0.108-2.996) | -<br>0.505 |
| | Tertiary | 1<br>0.738 (0.348-1.827) | -<br>0.654 |
| **Employment** | | 1<br>1.129 (0.727-1.753) | -<br>0.588 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.14: Multivariate regression analysis of determinants of metabolic syndrome by NCEP definition.**

| | First model | | Last model | |
|---|---|---|---|---|
| **Covariates** | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | 0.981 (0.941-1.024) | 0.383 | - | - |
| **Gender** | 2.346 (0.742-7.417) | 0.147 | - | - |
| **Smoking** | 0.377 (0.064-2.228) | 0.282 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| **Alcohol consumption** | | 0.465 (0.301-1.753) | <0.001 | 0.469 (0.305-0.720) | <0.001 |
| **Marital status** | Single | 0.394 (0.120-1.291) | 0.124 | - | - |
| | Married | 0.333 (0.117-0.948) | 0.039 | - | - |
| | Divorced | 0.163 (0.012-2.242) | 0.175 | - | - |
| | Widowed | 0.436 (0.136-1.396) | 0.162 | - | - |
| **Level of education** | No formal education | 0.286 (0.040-2.022) | 0.210 | - | - |
| | Primary | 0.786 (0.144-4.294) | 0.781 | - | - |
| | Secondary | 0.568 (0.108-2.996) | 0.505 | - | - |
| | Tertiary | 0.845 (0.289-2.114) | 0.825 | - | - |
| **Employment** | | 1.129 (0.727-1.753) | 0.588 | - | - |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.15: Binary regression analysis of determinants of metabolic syndrome by IDF definition.**

| | Metabolic syndrome IDF | |
|---|---|---|
| **Covariates** | 95% CI | p-value |
| **Age** | 0.993 (0.945-1.043) | 0.007 |
| **Gender** | 1.833 (0.248-13.526) | 0.552 |
| **Smoking** | 1<br>0.444 (0.035-5.630) | -<br>0.531 |
| **Alcohol consumption** | 1<br>0.357 (0.217-0.589) | -<br><0.001 |

| | | | |
|---|---|---|---|
| **Marital status** | Single | 1<br>0.902 (0.220-3.701) | -<br>0.887 |
| | Married | 1<br>0.424 (0.121-1.492) | -<br>0.998 |
| | Divorced | 1<br>1.745 (0.685-4.724) | -<br>0.258 |
| | Widowed | 1<br>0.689 (0.177-2.678) | -<br>0.591 |
| **Level of education** | No formal education | 1<br>0.301 (0.035-2.579) | -<br>0.273 |
| | Primary | 1<br>0.676 (0.108-4.220) | -<br>0.676 |
| | Secondary | 1<br>0.399 (0.067-2.399) | -<br>0.316 |
| | Tertiary | 1<br>0.637 (0.371-1.741) | -<br>0.558 |
| **Employment** | | 1<br>1.463 (0.912-2.347) | -<br>0.114 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.16: Multivariate regression analysis of determinants of metabolic syndrome by IDF definition.**

| | First model | | Last model | |
|---|---|---|---|---|
| **Covariates** | β (95% CI) | p-value | β (95% CI) | p-value |
| **Age** | 0.993 (0.945-1.043) | 0.779 | - | - |
| **Gender** | 1.833 (0.248-13.526) | 0.552 | - | - |
| **Smoking** | 0.444 (0.035-5.630) | 0.531 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| **Alcohol consumption** | | 0.357 (0.217-0.589) | <0.001 | 0.364 (0.222-0.595) | <0.001 |
| **Marital status** | Single | 0.902 (0.220-3.701) | 0.887 | - | - |
| | Married | 0.424 (0.121-1.492) | 0.181 | - | - |
| | Divorced | 2.187 (0.775-6.147) | 0.144 | - | - |
| | Widowed | 0.689 (0.177-2.278) | 0.591 | - | - |
| **Level of education** | No formal education | 0.301 (0.035-2.579) | 0.273 | - | - |
| | Primary | 0.676 (0.108-4.220) | 0.676 | - | - |
| | Secondary | 0.399 (0.067-2.3.33 | 0.316 | - | - |
| | Tertiary | 0.847 (0.287-2.112) | 0.822 | - | - |
| **Employment** | | 1.463 (0.912-2.347) | 0.114 | - | - |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.17: Binary regression analysis of determinants of high Framingham score.**

| | | High Framingham score | |
|---|---|---|---|
| **Covariates** | | 95% CI | p-value |
| **Alcohol consumption** | | 1<br>4.420 (2.679-7.736) | -<br><0.001 |
| **Marital status** | Single | 1<br>0.626 (0.151-2.593) | -<br>0.518 |
| | Married | 1<br>0.922 (0.296-2.870) | -<br>0.889 |

| | | | |
|---|---|---|---|
| | Divorced | 1<br>2.181 (0.376-12.638) | -<br>0.384 |
| | Widowed | 1<br>0.862 (0.213-3.484) | -<br>0.835 |
| **Level of education** | No formal education | 1<br>1.197 (0.021-1.874) | -<br>0.158 |
| | Primary | 1<br>0.238 (0.038-1.491) | -<br>0.125 |
| | Secondary | 1<br>0.278 (0.048-1.622) | -<br>0.155 |
| | Tertiary | 1<br>0.936 (0.398-1.711) | -<br>0.587 |
| **High glucose** | | 1<br>1.064 (0.245-4.612) | -<br>0.934 |
| **Employment** | | 1<br>0.594 (0.339-1.040) | -<br>0.069 |

*p<0.05, β= beta-coefficient correlation.*

**Table 3.18: Multivariate regression analysis of determinants of high Framingham score.**

| | | First model | | Last model | |
|---|---|---|---|---|---|
| **Covariates** | | β (95% CI) | p-value | β (95% CI) | p-value |
| **Alcohol consumption** | | 4.420 (2.526-7.736) | <0.001 | 4.331 (2.488-7.540) | <0.001 |
| **Marital status** | Single | 0.626 (0.151-2.593) | 0.518 | - | - |
| | Married | 0.922 (0.296-2.870) | 0.889 | - | - |
| | Divorced | 2.181 (0.376-12.638) | 0.384 | - | - |

| | | | | | |
|---|---|---|---|---|---|
| | Widowed | 0.947 (0.562-1.780) | 0.803 | - | - |
| **Level of education** | No formal education | 0.197 (0.021-1.874) | 0.158 | - | - |
| | Primary | 0.238 (0.038-1.491) | 0.125 | - | - |
| | Secondary | 0.278 (0.048-1.622) | 0.155 | - | - |
| | Tertiary | 0.751 (0.245-2.478) | 0.725 | - | - |
| **High glucose** | | 1.064 (0.245-4.612) | 0.934 | - | - |
| **Employment** | | 0.577 (0.321-1.038) | 0.067 | - | - |

*p<0.05, β= beta-coefficient correlation.*