# THE RISK OF METABOLIC SYNDROME AS A RESULT OF LIFESTYLE AMONG ELLISRAS RURAL POPULATION: ELLISRAS LONGITUDINAL STUDY

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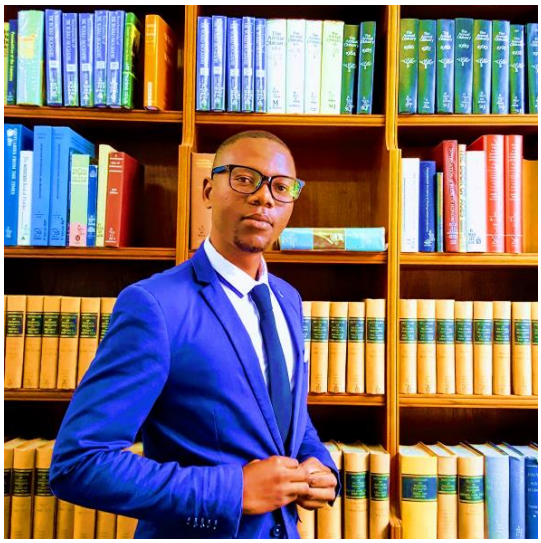

*"He who receives ideas from me, receives instruction himself without lessening mine; as he who lights his taper at mine, receives light without darkening me." ~ Thomas Jefferson*

## DEDICATION

I dedicate this dissertation to my family, relatives and friends. I would like to express special gratitude to my mother (Geminah Sekgala) and Grand Mother (Mmakgabo Sekgala), who continuously encouraged and supported me through my studies. My brothers (Joseph, Phillimon, Dipuo and Thimothy and my sister Ashley Sekgala) for being supportive and believing in me. Futhermore, I also dedicate this dissertation to my uncle and his wife (Sello Morudi) and (Conny Morudi) for their great support and motivation through my studies. I would also like to thank my dearest friend Sebolelo “Sebo” Khumalo for the words of encouragement and being supportive throughout my studies.

## DECLARATION

I declare that THE RISK OF METABOLIC SYNDROME AS A RESULT OF LIFESTYLE AMONG ELLISRAS RURAL POPULATION: ELLISRAS LONGTUDINAL STUDY is my own work and that all sources that I have used or quoted have been indicated by means of complete reference and that this work has not been submitted before for any other degree at any other institution.

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Full names Date

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## ABSTRACT

***Introduction*:** There is an increased trend in the prevalence of hypertension in children and adolescents in African countries. There are complications in diagnosing hypertension in children and adolescents due to the variation of blood pressure (BP) values with age, gender and height. The progression of the health transition with non-communicable diseases (NCDs) adds significantly to the disease burden, despite infectious diseases and undernutrition remaining persistent in both low and middle-income countries. Metabolic syndrome (MetS) is a global problem associated with the clustering of several cardiovascular risk factors. South African evidence suggests an upsurge of NCDs amidst the existence of communicable diseases (CDs) such as HIV/AIDS and tuberculosis. Moreover, NCDs and CDs in the country are influenced by socio-demographic factors; and thus tend to be more prominent in certain segments of the population.

***Aim and Objectives:*** The aim of this study was to perform blood pressure to height ratio and to determine lifestyle risk factors associated with metabolic syndrome among the Ellisras rural population aged 6-30 years, who are part of the ELS.

***Methods and materials:*** The current study is based on secondary data analysis of the Ellisras Longitudinal Study (ELS) and was conducted in two phases. Phase 1 included data analysis of all the participants in the ELS. This sample included a total number of 9002 children and adolescents (4678 boys and 4324 girls), aged 6-17 years. Parents or guardians provided written informed consent. Phase 2 consisted of biochemical analysis from a subsample of participants in the ELS. The subsample included 624 participants (306 males and 318 females) aged 18-30 years at the time the study was conducted. All participants underwent a series of anthropometric measurements (waist circumference and height) according to the standard of the International Society for the Advancement of Kinanthropometry (ISAK). The waist circumference (WC) measurements were taken to the nearest 0.1 cm, using a soft measuring tape. Metabolic syndrome was defined according to the International Diabetes Federation (IDF) criteria. Metabolic syndrome risk factors included total cholesterol (TCHOL), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), elevated fasting blood glucose (FBG), elevated blood pressure (BP) and high waist circumference (WC). A dietary intake questionnaire was also administered to each participant and self-administered questionnaire was used to collect data on

lifestyle factors, including smoking and alcohol intake. Dietary intake variables used in the linear regression method were log transformed prior to analysis because of their skewed distribution. Receiver-operating characteristic (ROC) curve was used to assess the accuracy of BPHR to screen children with prehypertension and hypertension. The optimal systolic BPHR (SBPHR) and diastolic BPHR (DBPHR) cut-off points for hypertension were determined. Sensitivity/specificity, positive predictive values and negative predictive values were calculated.

***Results:*** The optimal thresholds for defining prehypertension was 0.77 in children aged 6-10 years and 0.73 in adolescents aged between 11 and 17 years for systolic BPHR and 0.55 in children and 0.53 in adolescents for diastolic BPHR, respectively. The corresponding values for hypertension stage 1 were 0.76 and 0.73 for SBPHR and 0.50 and 0.58 for DBPHR, respectively. The BPHR is an accurate tool for screening elevated BP in Ellisras children aged 6-17 years. This can help to prevent the misclassification of children and adolescent hypertension. Furthermore, this tool can be used to screen children before the development of prehypertension and hypertension. Moreover, it can be used to manage hypertension in Ellisras children, ultimately reducing the risks of developing hypertension and associated cardiovascular disease in adulthood. Overall, the prevalence of metS was 23.1% (8.6% males and 36.8% females). Females appeared to have higher mean values for WC, FBG, TCHOL and LDL-C than males (82.14, 5.62, 4.62 and 2.97, respectively). The only significant gender difference observed was on WC ($p<0.001$). Males on the other hand had higher mean values for HDL-C, TG, SBP and DBP than females (1.20, 1.06, 125.91 and 71.44, respectively). The only significant difference observed in this case was on SBP ($p<0.001$). No significant age group differences were observed in all the metabolic risk factors with the exception of DBP where the older (25-30 years) participants presented with high SBP than the younger age group (18-24 years) (70.96 mmHg vs 68.78 mmHg, $p<0.05$). While, majority of females had significantly high WC, elevated total cholesterol and LDL-C, and reduced HDL-C; majority of males had elevated BP, SBP and DBP. No significant age and gender differences were observed on dietary intake. However, according to the linear regression analysis, no association between log total energy, log added sugar, log SFA and log MUFA with metabolic risk factors. There was a low and negative significant association between log fibre with SBP and DBP (β:-0.004, $p=0.003$ and β:-0.004, $p=0.046$), respectively, crude. After adjusting for the potential

confounding factors, log fibre was also associated with FBG (β:-0.028, p=0.046). Log PUFAs was inversely associated with FBG, HDL-C and SBP crude. Log trans fatty acids was inversely associated with WC, HDL-C and SBP crude. Both log PUFAs and log trans fatty acids were not associated with any metabolic risk factors after adjusting for potential cofounding factors. Log protein was inversely associated with SBP both crude and adjusted for potential cofounding factors. On predicting the actual risk using the logistic regression analysis, participants who had high dietary energy intake were significantly less likely to present with larger WC, low HDL-C and high LDL-C (OR: 0.250 95%CI [0.161;0.389], OR: 0.306 95%CI [0.220;0.425] and OR: 0.583 95%CI [0.418;0.812], respectively), but more likely to presents with elevated FBG, high TCHOL, high TG and hypertension (OR: 1.01 95%CI [0.735;1.386], OR: 1.039 95%CI [0.575;1.337], OR: 1.186 95%CI [0.695;2.023], OR: 5.205 95%CI [3.156;8.585], respectively) crude. After adjusting for age, gender, smoking and alcohol status, high energy intake was more likely to increase two times high the large WC and elevated FBG among study participants (OR: 2.766 95%CI [0.863;3.477] and OR: 2.227 95%CI [1.051;3.328], respectively). Furthermore, low dietary fibre intake was nearly four times more likely to increase the low HDL-C, crude (OR: 3.864 95%CI [1.067;13.988]) crude. Those participants who consumed high trans fats were more likely to present with high FBG (OR:1.424 95%CI [0.985;2.060]), but less likely to present with LDL-C (OR: 0.540 95%CI [0.321;0.906]) crude. However, after adding potential cofounding factors, participants with high fatty acid were less likely to present with high FBG (OR: 0.672 95%CI [0.441;1.023]).

***Conclusions:*** MetS is prevalent in young adults in Ellisras and is differentiated by age and gender with more females at an increased rate by virtue of their body size status, reduced HDL-C, elevated FBG and high LDL-C and the diet they consume that is in most cases high energy, more carbohydrates, high added sugar and SFA. Therefore, identifying groups that are at an increased risk and those that are in their early stages of MetS will help improve and prevent the increase of the metS in the future. These results have high policy implications.

**KEY CONCEPTS**

Metabolic syndrome; risk factors; blood pressure; blood pressure to height ratio; cardiovascular disease; dietary intake; rural South African.

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## LIST OF ABBREVIATIONS

| | |
|---|---|
| AACE | American Association of Clinical Endocrinology |
| AHA/NHLBI | American Heart Association/National Heart, Lung, and Blood Institute |
| AUC | Area under curve |
| BMI | Body Mass Index |
| BP | Blood pressure |
| BPHR | Blood Pressure to-Height Ratio |
| CDL | Chronic Diseases of lifestyle |
| CHD | Coronary Heart Disease |
| CVDs | Cardiovascular diseases |
| DBP | Diastolic blood pressure |
| DBPHR | Diastolic blood pressure to height ratio |
| DCCT | Diabetes Control and Complications Trial |
| EGIR | European Group for the Study of Insulin Resistance |
| ELS | Ellisras Longitudinal Study |
| FAO | Food and Agriculture Organization |
| FBG | Fasting Blood Glucose |
| FFAs | Free Fatty Acids |
| GI | Glycemic Index |
| HDL-C | High Density Lipoprotein-Cholesterol |

| | |
|---|---|
| HIV/AIDS | Human Immunodeficiency Virus infection and Acquired Immune Deficiency Syndrome |
| HPA | Hypothalamic-Pituitary-Adrenal axis |
| IDF | International Diabetes Federation |
| IGT | Impaired Glucose Tolerance |
| IOTF | International Obesity Task Force |
| IQR | Inter Quartile Range |
| IR | Insulin Resistance |
| ISAK | International Society for the Advancement of Kinanthropometry |
| LDL-C | Low Density Lipoprotein-Cholesterol |
| metS | Metabolic syndrome |
| MiRNA | Micro RNAs. |
| MRC | Medical Research Council |
| MUFAs | Monounsaturated Fatty Acids |
| NCD | Non-communicable Diseases |
| NCEP:ATP | National Cholesterol Education Program Adult Treatment Panel |
| NEFA | Non-Esterified Fatty Acid |
| NHANES | National Health and Nutrition Examination Survey |
| OGTT | Oral glucose tolerance test |
| OR | Odds Ratio |
| PUFAs | Polyunsaturated Fatty Acids |
| RAAS | Renin-Angiotensin-Aldosterone System |
| RDA | Recommended Dietary Allowance |
| ROC | Receiver operating characteristics |
| SA | South Africa |

| | |
|---|---|
| SBP | Systolic blood pressure |
| SBPHR | Systolic blood pressure to height ratio |
| SD | Standard Deviation |
| SNS | Sympathetic Nervous System |
| SSA | Sub-Saharan Africa |
| TCHOL | Total cholesterol |
| TG | Triglycerides |
| TB | Tuberculosis |
| TEM | Technical Error of Measurements |
| US | United States |
| WHO | World Health Organisation |

# CHAPTER 1

## 1. PROBLEM STATEMENT AND AIM OF THE STUDY

## 1.1. PROBLEM STATEMENT

As far as twenty years ago the World Health Organization (WHO) projected that non-communicable diseases (NCDs) would account for approximately three quarters of all deaths in developing countries by 2020 (WHO, 1997). Metabolic syndrome (metS) has been considered to be one of the fastest developing NCDs entities in the world (Ford *et al*., 2004). Based on current population estimates, nearly 100 million people have metS (Roberts *et al*., 2013). The metS is comprised of interconnected risk factors as a result of lifestyle; such as elevated blood pressure (BP), waist circumference (WC), fasting blood glucose (FBG) and low high density lipoprotein cholesterol (HDL-C) (Alberti *et al*., 2006). According to Moreno Franco *et al*. (2014), there is an association between dietary intake and metS risk factors. However, studies that examine the combination of dietary intake and risk factors of metS in the black rural South African population are limited.

## 1.2. RATIONALE

Metabolic syndrome is a collective global public health problem that is categorized by a group of metabolic risk factors in persons. Metabolic syndrome is associated with an increased risk of diabetes mellitus and cardiovascular diseases (Galassi *et al*., 2006). Moore *et al*. (2015) maintained that major risk factors for the metS include: elevated WC, BP, FBG and low HDL-C. According to Okada *et al*. (2016), an increased WC is generally associated with abdominal obesity and it is used in the definition of metS. Among other risk factors, WC was said to be a contributing risk factor in elderly people's attainment of developing metS (Gozashti *et al*., 2014). Moreover, Li *et al*. (2011) suggested that measurement of low HDL-C might also serve as a simple and convenient way to identify individuals at high risk of having metS. Current evidence indicated that low HDL-C was able to predict metS in young adult populations (Liu and Reaven, 2013).

Elevated BP among children and adults has become a serious public health problem worldwide (Ingelfinger, 2014). Elevated FBG is an epidemic in industrialised countries as it is associated with high morbidity and mortality rates (Ogden *et al*., 2006). In other studies, multiple incidences of metabolic abnormalities were reported

in participants with elevated FBG (Eckel *et al*., 2005; Tamang *et al*., 2013). Therefore, additional research is required in this field.

Preliminary results of the Ellisras Longitudinal Study (ELS) showed that there was a slight increase in the prevalence of overweight and hypertension over time (Monyeki *et al*., 2008). Furthermore, significant relationships between BP and WC and body mass index (BMI) at a younger age were reported cross-sectionally (Monyeki *et al*., 2008). However, little is known about the relationship between dietary intake and MetS status in the Ellisras rural population.

### *1.2.1. Aim of the study*

The aim of this study was to determine lifestyle risk factors associated with metabolic syndrome among the Ellisras rural population aged 6–30 years, who are part of the ELS.

The objectives of this study were:

i. To determine the performance of blood pressure to height ratio as a screening tool for elevated blood pressure in rural children.
ii. To determine the prevalence of risk factors for metabolic syndrome (total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, hypertension, triglycerides, waist circumference and fasting blood glucose levels).
iii. To determine the association between dietary intake and metabolic syndrome risk factors.
iv. To determine the risk of developing metabolic syndrome

## 1.3. SCIENTIFIC CONTRIBUTION

The results of this study addressed the current prevalence of metabolic syndrome among rural young adults in South Africa in order to improve interventions to reduce metS. The aim and objectives highlight the risk factors that contribute to the development of the metS. Furthermore, the results of the study were presented to the Ellisras community for the purposes of education.

## 1.4. STRUCTURE OF THE DISSERTATION

1. Chapter 1 Problems statement, rationale and aim of the study
2. Chapter 2 Literature review
3. Chapter 3 Materials and methods
4. Chapter 4 Results and Discussion
5. Chapter 5 Summary, conclusion and recommendations
6. Articles published to International peer reviewed Journals will be compiled as an addendum

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# CHAPTER 2

## 2. LITERATURE REVIEW

## INTRODUCTION

In 1997, the WHO estimated that non-communicable diseases (NCDs) would account for approximately three quarters of all deaths in developing countries by 2020 (WHO, 1997). By 2008, NCDs caused about 36 million deaths globally, with 80% of these occurring in low-income and middle-income countries (Wild and Green, 2000; WHO, 2012). This increased disease burden occurs as a result of the increased prevalence of various risk factors for NCDs such obesity, blood pressure, cholesterol and tobacco use (Boutayeb and Boutayeb, 2005). In order to address the NCD epidemic, Ezzati, *et al* (2005), recommended that prevention of obesity should be prioritised from the early stages of economic growth, followed by the personal intervention for blood pressure and cholesterol at a population level.

A number of studies have reported the increasing epidemic of NCDs in Sub-Saharan Africa (SSA), these include cancers, cardiovascular diseases and metabolic diseases such as overweight/obesity, type 2 diabetes mellitus, chronic lung disease and depression (Ezzati *et al*., 2005; Mensah, 2008; Connor *et al*., 2007; Daar *et al*., 2007). Stroke and several other vascular diseases are also known to contribute to the NCD burden (Connor *et al*., 2007). The emerging epidemic of NCDs in SSA have been attributed to the changes in economic growth, global urbanization, lifestyle habits, the nutritional transition and aging (Longo-Mbenza *et al.*, 2010; WHO, 2000; Moore *et al*., 2015; Yusuf et al., 2001). The increasing burden of NCDs will escalate further over the coming decades if the necessary precautions to prevent this trend are not taken (Abegunde *et al*., 2007). Therefore, early intervention and diagnoses of these NCD risk factors will be beneficial to the countries in SSA.

South Africa as a developing country is also undergoing a health transition in form of a rising epidemic of infectious diseases and NCD, as well as perinatal and maternal problems and challenges related to injuries and violence (Bourne *et al*., 2002; Bradshaw *et al*., 2003). As such SA is experiencing a quadruple burden of disease.

Metabolic syndrome (metS) has been considered to be one of the fastest developing NCDs entities in the world (Ford *et al*., 2004; Mottillo *et al*., 2010). Metabolic syndrome is characterized by a group of risk factors that co-exist in an individual. Therefore, metS shares similar risk factors to that of NCDs, such as elevated blood

pressure, glucose intolerance and insulin resistance. These risk factors have been associated with obesity, thereby suggesting the interrelation between NCDs and metS (McKeigue *et al*., 1998). Furthermore, the relationship between obesity and the metS was reported in a study in which surgical removal of visceral fat was associated with improved insulin sensitivity and delayed development of type 2 diabetes mellitus (Gabriely *et al*., 2002).

Worldwide, the metS pandemic is considered as a high economic cost complex disorder and it has become one of the major public health challenges (Kassi *et al*., 2011). The risk factors associated with this syndrome are linked to diabetes and CVDs in both adolescents and adults (Bao *et al*., 1994; Bao *et al*., 1996; Eckel *et al*., 2005). Stern *et al*. (2004) emphasized that individuals who have metS are fivefold more likely to develop type 2 diabetes. Based on current population estimates, nearly 100 million people have metS (Roberts *et al.*, 2013). In which around 20% is reported in adults without type 2 diabetes and approximately 80% in adults living with type 2 diabetes (Agomuoh *et al*., 2006).

The prevalence of metS increased from 29.2 to 34.2% in the U.S according to the National Health and Nutrition Examination Survey (NHANES) between 1999 and 2006 (Mozumdar and Liguori, 2011). For the past decades, the prevalence of type 2 diabetes mellitus and obesity has been on the rise (Cook *et al*., 2003). There was approximately 65% prevalence of the US adult population with overweight. This might explain the increasing epidemic of metS in high income countries. Similar increase was observed in Asian countries (Nestel *et al*., 2007).

The subject of metS has received much attention in Africa recently, due to increasing awareness of its association with CVD related deaths (Vorster, 2002; Bruno *et al*., 2004; Isezuo and Ezunu, 2005). Previously, the burden of diseases among Africans was mainly attributable to infectious diseases. Nowadays, however, Africa is experiencing an epidemiological transition with increased cardiovascular disorders, which has resulted in a double burden of disease on the continent (Okafor, 2012). The evidence of the link between metS and diabetes is a milestone, given that the prevalence of metS is significantly higher in diabetic patients than healthy individuals of Nigeria (Ogbera, 2010).

Metabolic syndrome is now very common even in the South African population. The prevalence of individual risk factors of the metS are reported in a few South African provinces. The prevalence of metS ranged from 42.6% to 62.0% in the Western Cape (Kruger and Nell, 2017; Erasmus *et al.*, 2012) to 5.9% in Eastern Cape (Sekokotla *et al*., 2017), 22.1% in Kwazulu Natal (Motala *et al*., 2011) and 52.2% and 39.7% in the rural and urban Free State (Van Zyl *et al*., 2012). To date, there is no study done in Limpopo province to determine the prevalence of metS. Further studies in rural South African areas are needed to determine if the prevalence of metS is heterogeneous and come-up with ways to prevent the metS from becoming a social and economic problem in the near future.

## 1. Criteria for the diagnosis of metabolic syndrome

There are several criteria in the literature for diagnosing metS including the WHO, 1998 (Alberti *et al.*, 1998); the European Group for the Study of Insulin Resistance (EGIR); 1999 (Balkau and Charles, 1999); the National Cholesterol Education Program Adult Treatment Panel (NCEP: ATPIII), the American Association of Clinical Endocrinology (AACE), 2003 (Einhorn *et al*., 2003); the American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI), 2004 (Grundy *et al*., 2004) and the International Diabetes Federation (IDF) (Europe) (Alberti *et al*., 2005). These different diagnostic criteria for metS are summarised in Table 1.

Table 1 Criteria for the diagnosis of metabolic syndrome

| **WHO, 1998 (Alberti *et al*., 1998) Insulin resistance is defined as type 2 diabetes mellitus or (IFG) (> 100 mg/dl) or (IGT), plus two of the following are present** | **EGIR (1999) (Balkau and Charles, 1999) Insulin resistance is defined as insulin levels > 75th percentile of non-diabetic patients, plus two of the following are present** | **NCEP: ATPIII, 2001 (NCEP, 2001) MetS is diagnosed if any three of the following are present** | **AACE, 2003 (Einhorn *et al*., 2003) MetS is diagnosed if IGT plus two or more of the following are present** | **IDF (2005) (Alberti *et al*., 2005) MetS is diagnosed if Central obesity (defined as waist circumference but can be assumed if BMI > 30 kg/m2) with ethnicity-specific values, waist circumference must be of Europeans, > 94 cm in men and > 80 cm in women plus two of the following are present.** | **AHA/NHLBI, 2004 (Grundy *et al*., 2004) MetS is diagnosed if any three of the following are present** |
|---|---|---|---|---|---|
| Abdominal obesity (BMI) > 30 kg/m2 | Waist circumference ≥94 cm in men, ≥80 cm in women. | Waist circumference > 102 cm in men, > 88 cm in women. | BMI ≥25 kg/$m^2$ | Triglycerides ≥150 mg/dl | Waist circumference ≥102 cm in men,≥ 88 cm in women. |
| Triglycerides ≥150 mg/dl HDL-cholesterol < 40 mg/dl in men and < 50 mg/dl in women. | Triglycerides ≥150 mg/dl, HDL-cholesterol < 39 mg/dl in men or women. | Triglycerides ≥150 mg/dl | Triglycerides ≥150 mg/dl, HDL-cholesterol < 40 mg/dl in men and < 50 mg/dl in women. | HDL-cholesterol < 40 mg/dl (1.03 mmol/L) in men and < 50 mg/dl (1.29 mmol/L) in women. | Triglycerides ≥150 mg/dl. |
| BP ≥140/90 mmHg | BP ≥140/90 mmHg or taking antihypertensive drugs. | HDL-cholesterol < 40 mg/dl in men and < 50 mg/dl in women. | BP ≥130/85 mmHg | BP ≥130/85 mmHg | HDL-cholesterol < 40 mg/dl in men and < 50 mg/dl in women. |
| Microalbuminuria (urinary albumin secretion rate >20 µg/min or albumin-to-creatinine ratio >30 mg/g or | Fasting glucose >110 mg/dl | BP ≥130/85 mmHg | . | Fasting blood glucose ≥100 mg/dl | BP ≥130/85 mmHg |
| | | Fasting blood glucose ≥110 mg/dl | | | |

*MetS=metabolic syndrome; IFG=impaired fasting glucose; IGT=impaired glucose tolerance; HDL-C=high-density lipoprotein cholesterol; BP=blood pressure; BMI=body mass index; EGIR =european group for the study of insulin resistance; NCEP:ATPIII=national cholesterol education program adult treatment panel III;AACE=american association of clinical endocrinology criteria; IDF=international diabetes federation; AHA/NHLBI=american heart association/national heart, lung, and blood institute*

Table 1 shows that the WHO considered screening metS focusing on the insulin resistance (IR) or type 2 diabetes mellitus as they are important components of metS. Microalbuminria was considered among other risk factors included in the diagnosis of metS. EGIR considered an IR as the major criteria for metS with WC but not BMI. In 2001, NCEP: ATPIII and AHA/NHLBI proposed the new definition in which all the risk factors were considered significant as long as more than three are present. Additionally, IDF published a new definition that differed from the other definitions as IR was not considered a major component of metS. There is no specific criteria for diagnosing the metS in South Africa. However, the IDF developed the new definition for Sub-Saharan countries. For the purpose of this study, IDF criteria is used for diagnosing the metS. IDF focused on the WC as a prerequisite for diagnosing metS. Waist circumference is a surrogate measure of central obesity, the cornerstone of metS (Kassi, 2011). Several independents researches done is South Africa and Africa in general used the IDF criterion (Kelliny *et al*., 2008; Longo-Mbenza *et al*., 2010; Motala *et al*., 2011; Erasmus *et al*., 2012; Kruger and Nell, 2017). Elevated BP, triglycerides and HDL-C were considered the same risk level in all definitions. All these discrepancies in the definition of metS have influenced the prevalence of metS at an individual and population level. The need to have one practical definition to accurately diagnose an individual with metS is crucial.

## 2. Determinants of metabolic syndrome

Metabolic syndrome (metS) is a collective global public health issue that is associated with a cluster of risk factors that co-exist in an individual (Oron-Herman *et al*., 2008; Eckel *et al*., 2010). These risk factors can be divided into controllable and uncontrollable risk factors.

### 2.1. Controllable risk factors

Clustering risk factors of the metS have become the leading cause of mortality and mortality in both developing and developed countries (Li *et al*., 2013). These risk factors are untreatable but can only be controlled and managed, therefore it is important to note the controllable risk factors of the metS. These include excess body fat around the waist, high blood pressure, high blood sugar or IR, elevated

cholesterol levels, sedentary lifestyle; poor nutritional dietary intake, physical inactivity and tobacco smoking and alcohol abuse.

### 2.1.1. Increased waist circumference (WC)

According to Okada *et al*. (2016), an increased WC is generally associated with abdominal obesity and it is used in the definition of metS. The cut off points for elevated WC are (≥94cm in males, ≥80cm in females) (Alberti *et al*., 2009). Among other risk factors, WC was said to be the biggest contributing risk factor to developing metS in elderly participants (Gozashti *et al*., 2014). Reportedly, 24-65% and 43-78% of European females and males, respectively, were living with obesity and had been diagnosed with metS (van Vliet-Ostaptchouk *et al*., 2014). Subsequently, these epidemiological transitions regarding the components of metS have recently been observed in SSA and have been considered to have extended to an epidemic stage, in early adulthood as well (Okafor, 2012). In Nigeria, the prevalence of metS was 28% (Oguoma *et al*., 2015). These evidence the great associations between weight gain and metS. Meanwhile, the performance of waist-to-height ratio is found to the better predictor in Nigerian population (Oguoma *et al*., 2015).

#### 2.1.1.1. Complications associated with elevated waist circumference

Abdominal obesity (measured by waist circumference (WC)) refers to the excessive body fat which gas become the most important public health issue. Amongst other complications, WC is shown to be associated with an increased risk of Myocardial Infarction, stroke and early mortality (WHO, 2008). However, these diseases are not associated with general obesity measured by BMI (Larsson *et al*., 1984). In short, large amounts of free fatty acids (FFAs) are released the portal system in the liver by the intra-abdominal fat mass and disturbs hepatic insulin absorption (Björntorp, 1990). Intra-abdominal fats cause the turnover of FFAs distribution in the organs (Weiss, 2007). Consequently, cardiovascular diseases (CVDs) increases as a result of increased free fatty acids (FFAs) as shown in figure 3.

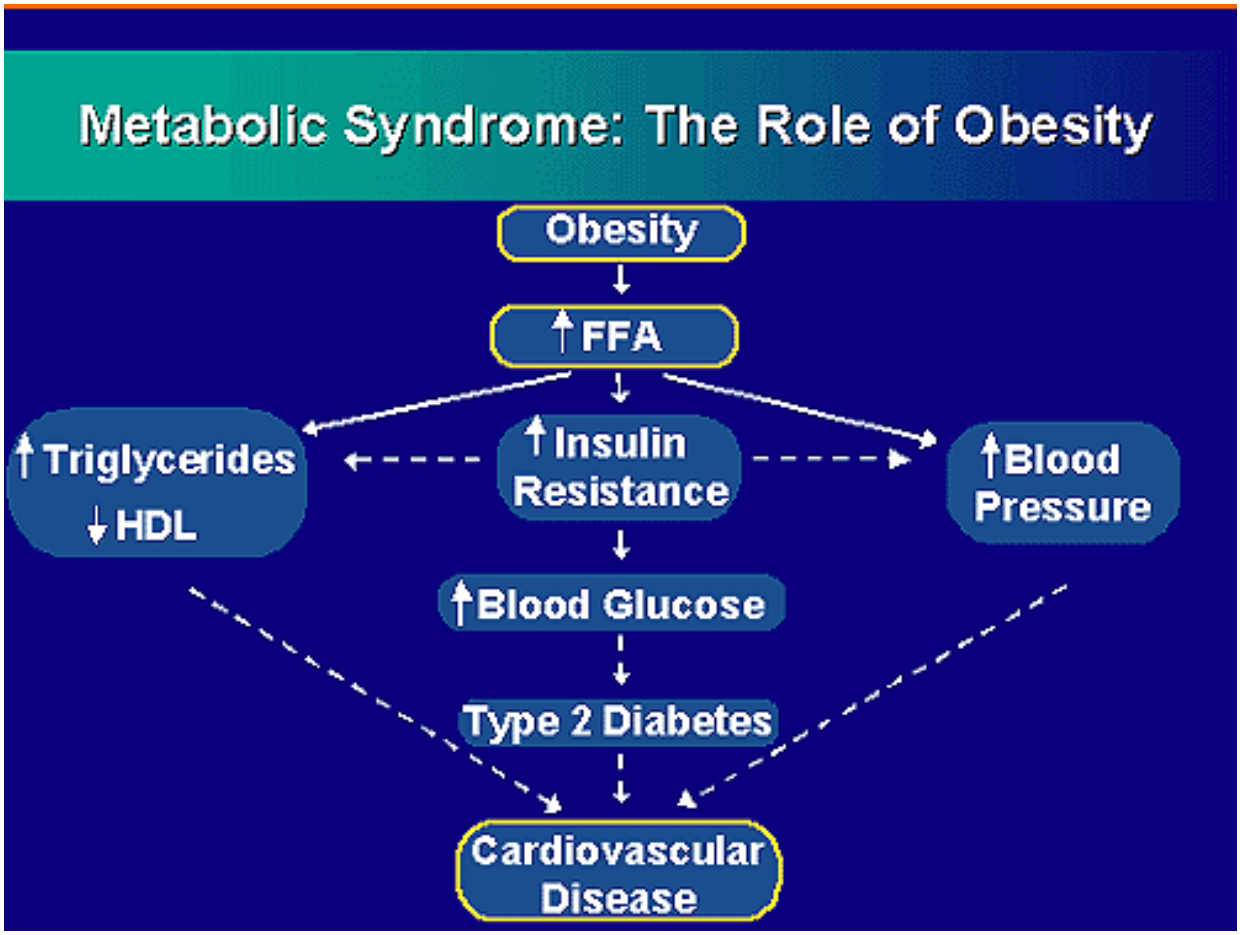


**Figure 3: The pathways of obesity influencing the cardiovascular diseases.** *FFA= free fatty acids; HDL=high-density lipoprotein.*

### 2.1.2. High blood pressure

Blood pressure (BP) can be defined as the pressure exerted by blood against the vessels in the body (Nobrega *et al*., 2014). The cut-off points for high blood pressure are (≥130 mmHg systole and/or ≥85 mmHg diastole) (Alberti *et al*., 2009). BP is commonly associated with metabolic abnormalities such as obesity, dyslipidemia, glucose intolerance (Saad *et al*., 2004). Both hyperinsulinemia and hyperglycemia activate the Renin angiotensin system (RAS) which result in the elevated expression of angiotensinogen, Angiotensin II and Angiotensin I receptors. This leads to the development of hypertension, especially in individuals with insulin resistance (Malhotra *et al*., 2001). Studies showed that, insulin resistance and hyperinsulinemia activate the sympathetic nervous system (SNS) and cause the kidneys to increase sodium reabsorption and consequently cardiac output (Morse *et al*., 2005).

Aldosterone is also produced by the adipocytes in response to the Angiotensin II as shown in figure 1 (Briones *et al*., 2012).

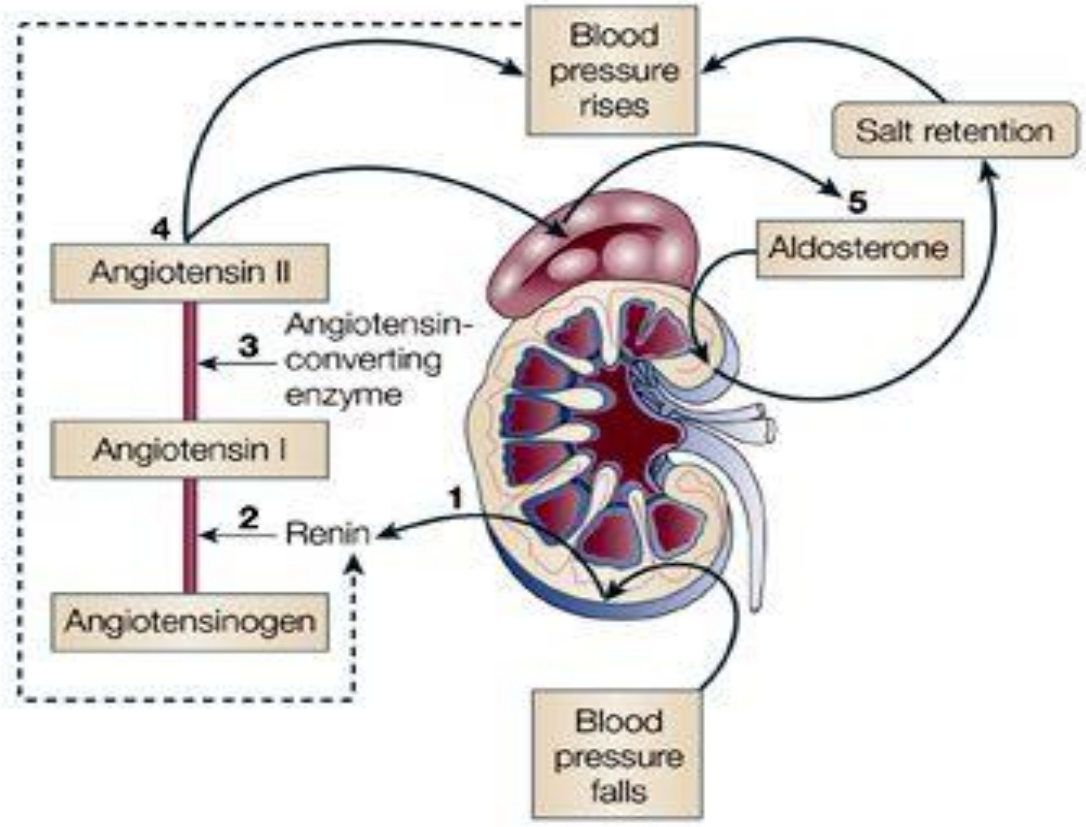


**Figure 1: A summary of Renin–angiotensin–aldosterone system (RAAS)** (Brewster and Perazella, 2004).

An elevated BP among children and adults has become a serious public health problem worldwide (Ingelfinger, 2014). Elevated BP is one of the most noticeable risk factors of the metS. Studies have examined the multiple incidences of metabolic abnormalities in participants with elevated blood sugar (Haffner *et al*., 1992; Kemper, 2004). Two types of hypertension can occur as a result of elevated blood pressure:

i. Primary hypertension

Primary hypertension, also known as essential hypertension, occurs with unknown secondary causes and it accounts for about 95% of all forms of hypertension. However, it is complexed to track the pathogenesis of primary hypertension as it appears multifactorial (Carretero and Oparil, 2000). There are reported risk factors

associated with primary hypertension including age, genetic factors, excessive weight gain and obesity (Aronow *et al.*, 2011).

ii. Secondary hypertension

The onset age of the secondary hypertension is not clear, therefore is complex to screening it. Although, health professionals identify its symptoms through clinical and laboratory measures. This type of hypertension is proposed to be caused by chronic kidney diseases, Cushing syndrome, use of certain drugs, coarctation of aorta, pheochromocytoma, primary aldosteronism, sleep apnea, obstructive europhaphy and thyroid or parathyroid disease (Viera and Neutze, 2010).

#### 2.1.2.1. Complications associated with high blood pressure

Complications that are associated with untreated hypertension progressions are damage to several organs such as the heart (left ventricular hypertrophy, coronary atherosclerosis), brain (stroke, vascular dementia), kidneys (nephrosclerosis, albuminuria, proteinuria), arteries (peripheral artery disease, atherosclerosis), and eyes (retinopathy) (Nadar *et al*., 2006; Korhonen *et al*., 2015). However, kidney and artery damage may accumulate in the treatment resistant stage. Therefore, in order to prevent cardiovascular disease in the future, high blood pressure should be diagnosed and managed at an early age.

#### 2.1.2.2. Measurements of blood pressure

i. Intra-arterial monitoring

The most direct measurement of BP, considered as the gold standard is intra-arterial monitoring (Verdecchia, 2000). Using this method, a catheter for intra-arterial monitoring is inserted into an artery and pressure waves will display on a monitor. Intra-arterial monitoring provides a beat-to-beat record of BP and is used in the intensive care unit and during surgery (Gupta and Lipsitz, 2007). Due to it's invasive nature, this technique in not appropriate for use in screening or in noncritical care settings (Verdecchia, 2000; Jones *et al*, 2003).

ii. Clinical measurements

In a clinic setting, the measuring of BP is recommended to be done by trained personnel. The proposed standard commonly used to measure BP is the upper arm at the brachial artery, because devices and techniques that measure BP at alternative sites such as wrist and finger are highly prone to error, and are therefore not recommended in guidelines (Pickering, 2005). As such, these devices will not be included in this review.

- Auscultatory method

The auscultatory method is a manual device that requires trained personnel to detect Korotkoff sounds using a stethoscope. Korotkoff sounds are made by the turbulent flow of blood past the restricted area created by the inflated cuff. The readings from the brachial artery are displayed on a mercury or aneroid sphygmomanometer. However, as with other methods, error and bias are included in this method. These include differences in auditory acuity and terminal digit rounding. Errors and bias can be minimized by the positioning of the patient and arm, cuff size and placement, cuff inflation and deflation, number and timing of measurements, and unique Korotkoff sounds. Although, these recommendations are not grounded on the systematic review of the literature. Bearing in mind these potential sources of error, the auscultatory method using a mercury sphygmomanometer correlates well with simultaneous intra-arterial BP (r=0.94 to 0.98) when performed correctly and was considered the gold standard for clinic-based measurements for many years. However, environmental issues related to the possibility of mercury spillage and the banned use of mercury sphygmomanometers, the role of this method has been reduced. Aneroid sphygmomanometers use a lever and bellows system to measure pressure and have been used as a mercury-free alternative. “Hybrid” sphygmomanometers are newer devices with an electronic pressure gauge in place of the mercury column, but BP is still determined using the auscultatory method.

- Oscillometric Method

Oscillometric sphygmomanometers use a pressure transducer to assess the oscillations of pressure in a cuff during gradual deflation. The point of maximum oscillation corresponds to the mean intra-arterial pressure. Systolic and diastolic BP measurements are then calculated based on an empirically derived algorithm. Investigators have cited several advantages to these devices, especially when they

are fully automated and can be programmed to complete several measurements after a period of rest at appropriate intervals without requiring the presence of medical personnel. The ability to obtain multiple readings while a patient rest alone in a quiet room may mitigate the increased BP seen in some persons only when in medical settings (isolated clinic hypertension) (Pickering *et al*., 2005; Myers, 2010).

#### 2.1.2.3. *Blood pressure in children and the need to develop a tool-blood pressure to height ratio*

There is an increased trend in the prevalence of hypertension in children and adolescents in African countries (Feber and Ahmed, 2010). Despite the presence of undernutrition in children in both the developing and developed countries, hypertension is emerging in rural children (Monyeki and Kemper *et al*., 2008). It has been well established that hypertension in children and adolescents tracked into adults, thereby increasing the future risk of cardiovascular diseases (CVDs) and mortality (Labadarios *et al*., 2001; Kiessling *et al*., 2008). Hypertension in children and adolescents was usually asymptomatic and caused left ventricular hypertrophy and carotid intimal medial thickness (Stergiou *et al*., 2011). Therefore, early diagnosis of prehypertension and hypertension in children is important to reduce the risk of CVDs in adulthood. Identification of hypertension in children and adolescents is difficult compared to adults because of differences in systolic blood pressure (SBP) and diastolic blood pressure (DBP) values due to age, gender and height (Hansen *et al*., 2007).

#### 2.1.2.4. *Blood pressure to height ratio as a tool to screen elevated blood pressure in children*

This method of screening for hypertension in children and adolescents was previously preceded in Han children (Lu *et al*., 2011). Blood pressure to height ratio (BPHR) was found to be an accurate and non-age dependent tool to identify hypertension in those children (Lu *et al*., 2011; Xi *et al*., 2014). In addition, optimal thresholds were stated. The same study done in Iran reported that the performance of BPHR used to identify hypertension was high in ethnic, age group, children and adolescents (Kelishadi *et al*., 2016) although there were no consistent cut-off point

values. There was no study that reported on the performance of BPHR among rural South African children.

### 2.1.3. Elevated fasting blood glucose

Elevated fasting blood glucose (FBG) can be described as a state of higher than normal fasting glucose concentration mostly due to insulin resistance (IR) as it inhibits the ability for the cell to transport glucose from the blood streams to the muscles tissue, adipose tissue and liver cells (WHO, 2016). The cut-off points for fasting blood glucose is (≥5.6 mmol/L) (Alberti *et al*., 2009). The most accepted scientific hypothesis proves that, the pathophysiology of metS is through the involvement of IR (Kaur, 2014). This is the reason that metS was referred to as "insulin resistant syndrome". Nonetheless, hyperinsulinaemia, that occurs as a result of defective insulin action that is explained by insulin resistance. The excessive circulation of fatty acids influences the increase in insulin resistance (Shoelson *et al*., 2006). The abundance of fatty acids reduces the insulin sensitivity by inhibiting the insulin-mediated glucose uptake in the muscles (Roden *et al*., 1996).

Among the predicted 366 million people that will live with diabetes mellitus in 2030 in the world, 298 million will be from developing countries (Wild *et al*., 2000). This shows that diabetes contributes towards more than one million deaths in the world. The prevalence of type 2 diabetes remarkably increased in African countries. In 2010, there was 46% prevalence of type 2 diabetes mellitus in rural Angola populations (Evaristo-Neto, 2010). South Africa is holding the second largest prevalence of type 2 diabetes among SSA countries (Popkin *et al*., 2012; IDF, 2014). Diabetes mellitus is characterized as type 1 (insulin dependent) and type 2 (non-insulin dependent) diabetes mellitus.

#### 2.1.3.1. *Complications associated with elevated fasting blood glucose*

Type 2 diabetes has detrimental health effects. When not treated it often forms advanced glycation end products (AGEs) which consequently results in dyslipidemia, inflammatory, vascular and thrombotic complications (Balagopal *et al*., 2011). Diabetes is known as the world highest cause of vasculature complications such as atherosclerosis which in turn lead to other disorders including stroke, small and large vesicle disease, and coronary heart disease (Labarthe, 1998). Complications

associated with diabetes kills 3.2 million people every year in the world (Hasnain and Sheikh, 2009). Subsequently, 90% of all diabetes have developed main cause of illness and premature death (Atlas, 2003). Therefore, there is a need to uproot the pandemic of metS in rural areas of the South African population, to maximize life expectancy.

#### 2.1.3.2. *Measurement of fasting blood glucose*

i. Oral glucose tolerant test (OGTT)

An enzymatic technique (employing hexokinase and glucose-6-phosphate dehydrogenase) is used for glucose measurement. Glucose is tested immediately after blood collection, however, in the case of blood samples, the blood should be collected into a container with glycolytic inhibitors and stored in ice-water until separated before analysis (Unwin *et al*., 2002).

There is on-going debate about the use of Oral glucose tolerant test (OGTT) in clinical and epidemiological studies. However, (WHO, 1999) recommended the use of this test, even though the ADA discouraged its use due to its greater cost, lower reproducibility and inconvenience (Puavilai *et al*., 1999). Nonetheless, OGTT can be used for the diagnosis of both 2-h plasma glucose and impaired glucose tolerance (IGT). This test can confirm and exclude an abnormality of IGT in people without symptoms (Unwin *et al*., 2002).

ii. Glycated haemoglobin (HbA1c)

The study conducted by Rohlfing *et al*, (2002), defined the relationship between $HbA_{1c}$ and diabetes as assessed in the Diabetes Control and Complications Trial (DCCT). There was a predictable relationship between diabetes and HbA1c. The HbA1c tests are not available in most countries across the world. The laboratory test measurements are not accurate as it is affected by several factors such as anaemia, pregnancy, abnormalities of haemoglobin and uraemia (Goldstein *et al*., 2004). Therefore, this test is not recommended for diagnostic test of diabetes (Unwin *et al*., 2002).

### 2.1.4. Abnormal Lipids profile

Total cholesterol (TCHOL) can be described as a fat-like substance found in the blood stream as well in the body organs and nerve fibres (Krauss *et al*., 2004). The cut-off point is >5.1 mmol/L (Mancia *et al*., 2013). There are two types of cholesterol namely; low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C).

Low-density lipoprotein cholesterol (LDL-C) is referred to as “bad” cholesterol because it deposits its cholesterol on the walls of arteries (Krauss *et al*., 2004). It is also the type of cholesterol that becomes oxidized and damages the lining of the arteries for accommodating mineral and fat deposits. The cut-off point is >3 mmol/L (Mancia *et al*., 2013).

High-density lipoprotein cholesterol (HDL-C) is considered “good” cholesterol because it sticks on firmly to the cholesterol it carries without letting them attach to arterial walls. It reduces the size of a plaque to keep cholesterol in solution and moves it safely throughout the body (Krauss *et al*., 2004). The HDL-C cut-off point is (<1.0 mmol/L Male; <1.3 mmol/L Female) (Alberti *et al*., 2009).

Triglycerides (TG) are type of fats found in the body; the body converts any calories it does not use into triglycerides (Jaworski *et al*., 2007). The cut-off point is (≥1.7 mmol/L) (Alberti *et al*., 2009).

#### 2.1.4.1. *Complications associated with abnormal lids profiles*

Both elevated LDL-C and triglycerides increases the chances of developing coronary heart diseases (CHDs). Coronary heart disease is defined as a condition in which plaque builds up inside the coronary arteries (Figure 4). Plaque is made up of cholesterol, fat, calcium, and other substances found in the blood that leads to the atherosclerosis.

Individuals with metS have demonstrated the presence of dyslipidemia (Isezuo and Ezunu, 2005; Garrido *et al*., 2009). In this regard, reduced HDL-C and hypertriglyceridemia are two major forms of dyslipidemia. Increased LDL-C on other hand has an effect in reducing HDL-C (Ogbera, 2010). The issue of dyslipidaemia need to be addressed across the world population (Akpa *et al*., 2006).

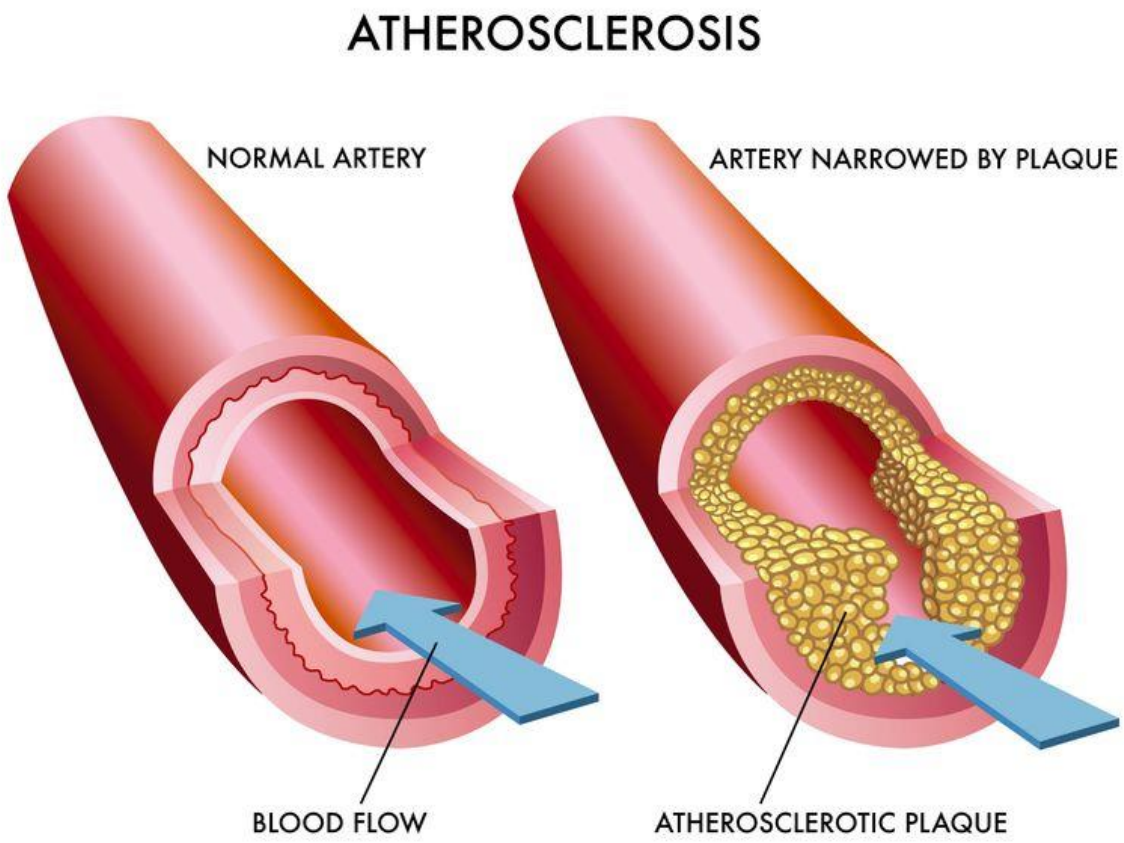


**Figure 4: Shows the normal artery and narrowed artery by plaques.**

http://anatomy-medicine.com/diseases-of-the-blood-vessels/156-atherosclerosis.html

2.1.4.2. *Measurement of cholesterol and triglycerides*

Cholesterol and triglycerides are measured enzymatically in serum or plasma in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol and glycerol, respectively. (Artiss and Zak, 2000). While LDL-C is calculated from measured values of cholesterol, triglycerides (TG) and HDL-C using Friedewald equation: (Friedewald *et al*., 1972).

[LDL-C] = [cholesterol] - [HDL-C] - [TG]/5

2.1.5. Environment and diet

South African current and available national and regional studies, suggested that dietary intake amongst rural South Africans is lower than the recommended daily allowance due to the insufficient amount of food consumed (Chikhungu *et al*., 2014; Mchiza *et al*., 2015). Lack of dietary diversity resulting in inadequate intake of both macronutrients and micronutrients including vitamins and trace elements or minerals contributes to health challenges (Fichera and Savage, 2015). Nonetheless, SA is undergoing a nutritional and epidemiological transition in which people are adopting a sedentary lifestyle and westernised food habits (Temple *et al.,* 2006). The western diet refers to the over-consumption of foods high in sugar, salt and fat, which consequently lead to weight gain among people and attributes to CVDs and other associated risk factors (Naidoo and Wills, 2006).

#### 2.1.5.1. *Sedentary lifestyle and urbanization*

Sedentary lifestyle and urbanization were the two factors contributing the most to the increased prevalence of metS (Okafor, 2012; Beltrán-Sánchez *et al*., 2013). The high prevalence of metS in developed countries can be due to increased consumption of high energy dense foods. People's lifestyle changes with economic growth and urbanization (Shi *et al*., 2008). The rapid transition in lifestyle due to urbanization has led to the change in health of the population from a lower mortality rate to higher prevalence of metS in developing countries (Cook and Dummer, 2004). There is 27.3% and 66.5% physical inactivity level in the South African rural and urban population, respectively (Van Zyl *et al*., 2012). In addition to physical inactivity, tobacco smoking and alcohol consumption are major public health issues in South Africa (SA) and other parts of the world (Reddy *et al*., 1998; Patel *et al*., 2007; WHO, 2008; WHO, 2015). Smoking and alcohol use contributes significantly to the diseases and premature deaths in the world (WHO, 2015; Rehm *et al*., 2003; Rehm *et al*., 2009).

#### 2.1.5.2. *Dietary intake habits*

Nutritional transition refers to a change in dietary intake from plant-based food sources which are high in fibre and low in fats to a high energy dense "western diet" which is high in fat and low in fibre (FAO, 2006; Popkin, 2003). Such dietary practices expose South Africans to obesity. As a result, the increasing levels of overweight and obesity/central obesity indicate an elevated risk for metabolic

disease. Dietary intake is a lifestyle factor contributing to metS since it is significant for good health and normal growth (Zarei *et al*., 2013). Good health and normal growth are explained by strong body immune system and less illness (Amine *et al*., 2002). Amine *et al* (2002) stipulated that the transition in diet and lifestyle occurred with industrialisation, urbanization, economic growth and market globalization for the past few decades. Consequently, all these occurrences influenced the health and nutritional status of the population, especially in developing countries.

#### 2.1.5.3. *Complication associated with poor nutritional intake and physical inactivity*

The consumption of a western diet that is high in fats and carbohydrates with low physical activity increased the prevalence of metS (Das, 2015). Excessive energy is stored as fats; therefore, visceral fats have the distinct gene expression pattern that is associated with insulin resistance, lower the HDL-C and increase LDL-C particle numbers (Neeland, *et al*., 2013). More importantly, the visceral fats are converted into fatty acids which are released in to the blood streams and transported into the liver and stored as triglycerides (Bergman *et al*., 2007; Klop *et al*., 2013). Hypertriglyceridemia results, after the fatty acid flux stimulates the hepatic output of LDL-C (Nikolic *et al*., 2013). Excessive triglyceride in the blood is then transferred to LDL-C and because more attached to the hepatic lipase which results in the breakdown of triglycerides and reduces the LDL-C particle numbers (Klop *et al*, 2013). Clinically, small dense LDL-C is more atherogenic as is more prone to oxidation and uptake into the arterial wall (Nikolic *et al*., 2013). In addition, monocyte/macrophage and adipocyte-derived factors may have direct atherothrombotic effects that promote the development of atherosclerotic cardiovascular events. Common genetic variants and environmental factors may influence the development of atherosclerosis at multiple levels through influences on central adiposity, innate immunity, glucose and lipoprotein metabolism, and vascular function (Reilly and Rader, 2003).

## 2.2. Non controllable risk factors

### 2.2.1. Age

Traditionally, aging has been regarded as a natural process and consequently for attaining the disease (Hayflick, 2007). CVDs, type 2 diabetes, hypertension and cancers are example of aging-associated diseases.

The epidemic of metS used to be an adults issue in the past (Kylin, 1923; Vague *et al*., 1947; Avogadro *et al*., 1996; Alberti *et al*., 2006). Today it is common in younger age groups due to the spread of obesity from childhood (Weiss *et al*., 2004; Cook *et al*., 2003; Cruz and Goran, 2004). The prevalence of metS increased from 11% in age group 20–29 years to 89% in age group 70–79 in 2009 (Ogbera, 2010). This shows that metS increases with age (Ogbera, 2010; Kelliny *et al*., 2008). The World Health Organisation (WHO) together with International Obesity Task Force (IOTF) have shown the proportion of increase in obesity and overweight from children age 5 to adults (WHO, 2004; Lobstein *et al*., 2004). For the past few decades, Bulatao (1993) found that aging is the key driver of diseases such as CVDs. The age of an individual demonstrates a greater proportion of disease susceptibility (Ordovas, 2007).

### 2.2.2. Gender

Although males and females are alike in several ways, there are other biological and behavioural differences between the two genders. Such differences have an impact on a wide range of diseases (Regitz-Zagrosek, 2012).

Gender-specific differences in the prevalence of metS have been reported satisfactorily. In which female have significantly higher prevalence of metS when compared to males (Ilanne-Parikka *et al*., 2004; Kelliny *et al*., 2008; Ogbera, 2010; Okafor, 2012, Motala *et al*., 2009). However, this is contrary to other findings (Puepet *et al*., 2009). Therefore, the investigation of different gender with regard to metS requires a closer attention.

### 2.2.3. Ethnicity

The burden of multiple disease varies by ethnicity. Some diseases are more prevalent in certain ethnic groups (Mathur *et al*., 2011).

Differences in the ethnicity and socioeconomic status influence the characteristics of the metS. Asians have a higher prevalence of metS than the white population (Araneta *et al*., 2002). Moreover, the International Diabetes Foundation (IDF) published new criteria for metS which accommodates the different populations, ethnicities and nationalities. The risk and values of the association between diabetes and CVDs varies amongst different populations. For example, South Asian, where the prevalence of diabetes and CVDs were high even at smaller WC which could not meet the criterion standard of white population (Reaven, 2006). If there could be many other studies on metS focusing on different population, it could shed a light not only on the pathophysiology of the syndrome but also on the genetic level, and such might help on treatment strategies.

### 2.2.4. Genetics

A genetic disease is any disease that is caused by an abnormality in an individual's genome. Some genetic disorders are inherited from the parents, while other genetic diseases are caused by acquired changes or mutations in a pre-existing genes (Oti and Brunner, 2007).

Genetic factors–both genetic and environmental factors influence metS (Neel, 1962).

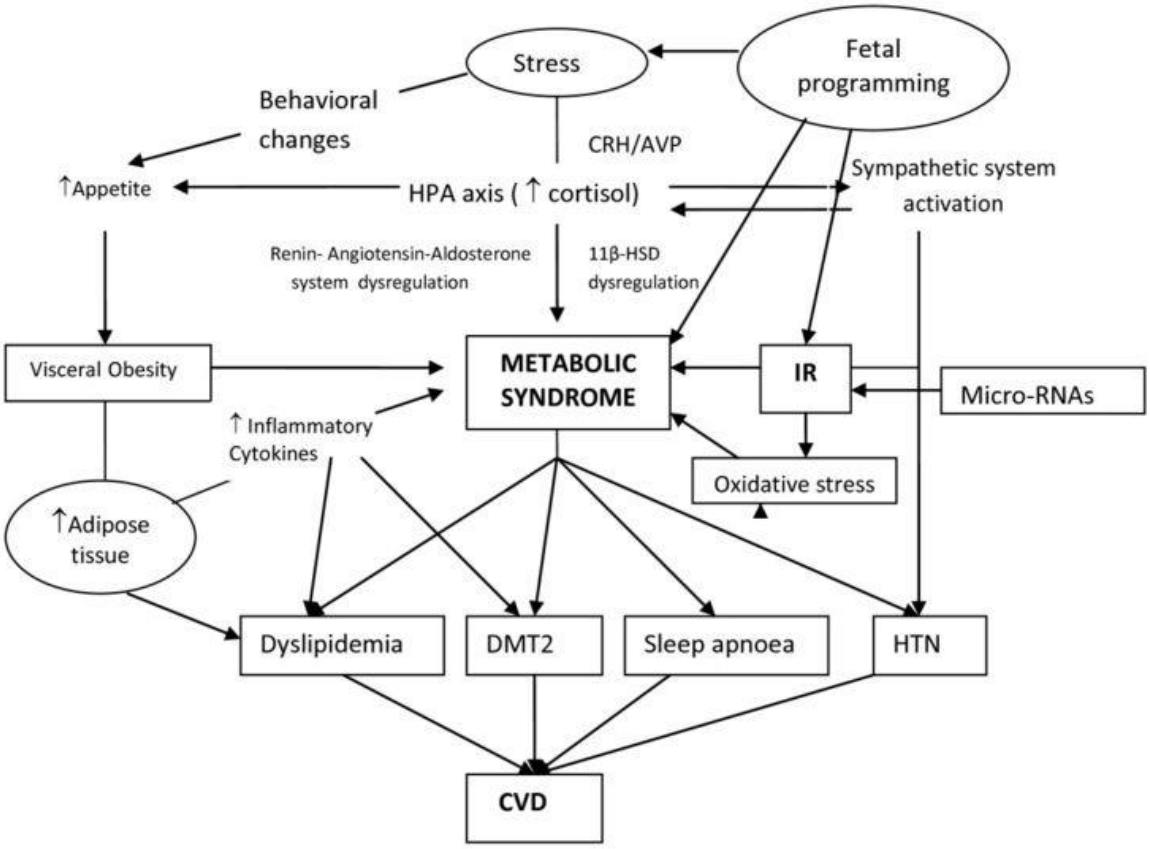


**Figure 2: The pathogenesis cardiovascular diseases in the metabolic syndrome** (Kassi *et al*., 2011). *IR=insulin resistance; HTN=hypertension; HPA axis= hypothalamic-pituitary-adrenal axis; DMT2=type 2 diabetes mellitus; CVD= cardiovascular disease; CRH=corticotropin releasing hormone; AVP=arginine vasopressin.*

Despite the many physiological mechanisms of risk components associated with metS, many of pathways remain unclear, more especially with regard to genetics and environmental factors (Ordovas, 2007). Briefly, in figure 2, several underlying factors play a role in the pathogenesis of metS apart from insulin resistance (IR) and obesity. Deregulation of the hypothalamic-pituitary-adrenal axis (HPA), autonomic nervous system (ANS) as well as stress increases the cellular oxidative stress, renin-angiotensin-aldosterone system (RAAS) activity, intrinsic tissue glucocorticoid action and micro RNAs (miRNAs).

## 2.3. SUMMARY

The metS is a constellation of interrelated risk factors that metabolically origin that are associated with the development of atherosclerotic CVDs. The pandemic of metS considered as elderly syndrome and being more prevalence in developed countries in the past. Majority of studies that focused on the subject of metS, came-up with the clear connection between this syndrome with type 2 diabetes mellitus and cardiovascular diseases. Type 2 diabetes mellitus and cardiovascular disease are most leading cause of morbidity and mortality worldwide.

There is a need to diagnose metS in the early stages to prevent and manage the emerging of metS. Various definitions for diagnosing metS have been developed in different population settings. Amongst all existing definition groups for diagnosing the metS, there is no unified definition adopted. The role of different ethnicity and cultural differences remain the cornerstone not to use uniform definition. Nonetheless, International Diabetes Federation (IDF) developed the new definition for Sub-Saharan countries. However, the hindrance still exists due to the unavailability of metabolic cut points in the black rural countries.

Healthy nutritional dietary intake should modify the attributable risk of the unwanted exposure amongst population. Food and Agriculture Organization (FAO) and World Health Organisation (WHO) have emphasized the concept of “balanced diet” as the first line of eating behaviour. Intake of unhealthy diet such as high-energy dense, saturated fats and salt and refine carbohydrates results in an increase chances of gaining weight which subsequently increases the metS. Most existing studies focused only on metS as one entity while fewer included risk factors such as smoking, physical activity, alcohol consumption and socio-economic status.

The intake of food determines the large extent of people’s health, growth and developments. Therefore, there is need to profile the association of dietary intake as lifestyle risk for the determinant of metS.

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# CHAPTER 3

## 1. MATERIALS AND METHODS

### 1.1. GEOGRAPHICAL AREA

Ellisras (known as Lephalale) is considered as one of the deep rural areas in the western part of the Limpopo province in South Africa (SA). The villages are approximately 70km away from the Ellisras town (23°40S 27°44W), adjacent to the Botswana border. The population is about 50,000 dispersed across 42 settlements (Sidiropoulos *et al*., 1997). The main sources of employment for the Ellisras residents are the Iscor coal mine and the Matimba electricity power station. The remaining workforce is mostly involved in subsistence farming and cattle rearing, while a few are involved in education and civil services. Poverty, unemployment and low life expectancy are common in rural South African settings and the Ellisras rural population is not exempted from this (Stats SA, 2002).

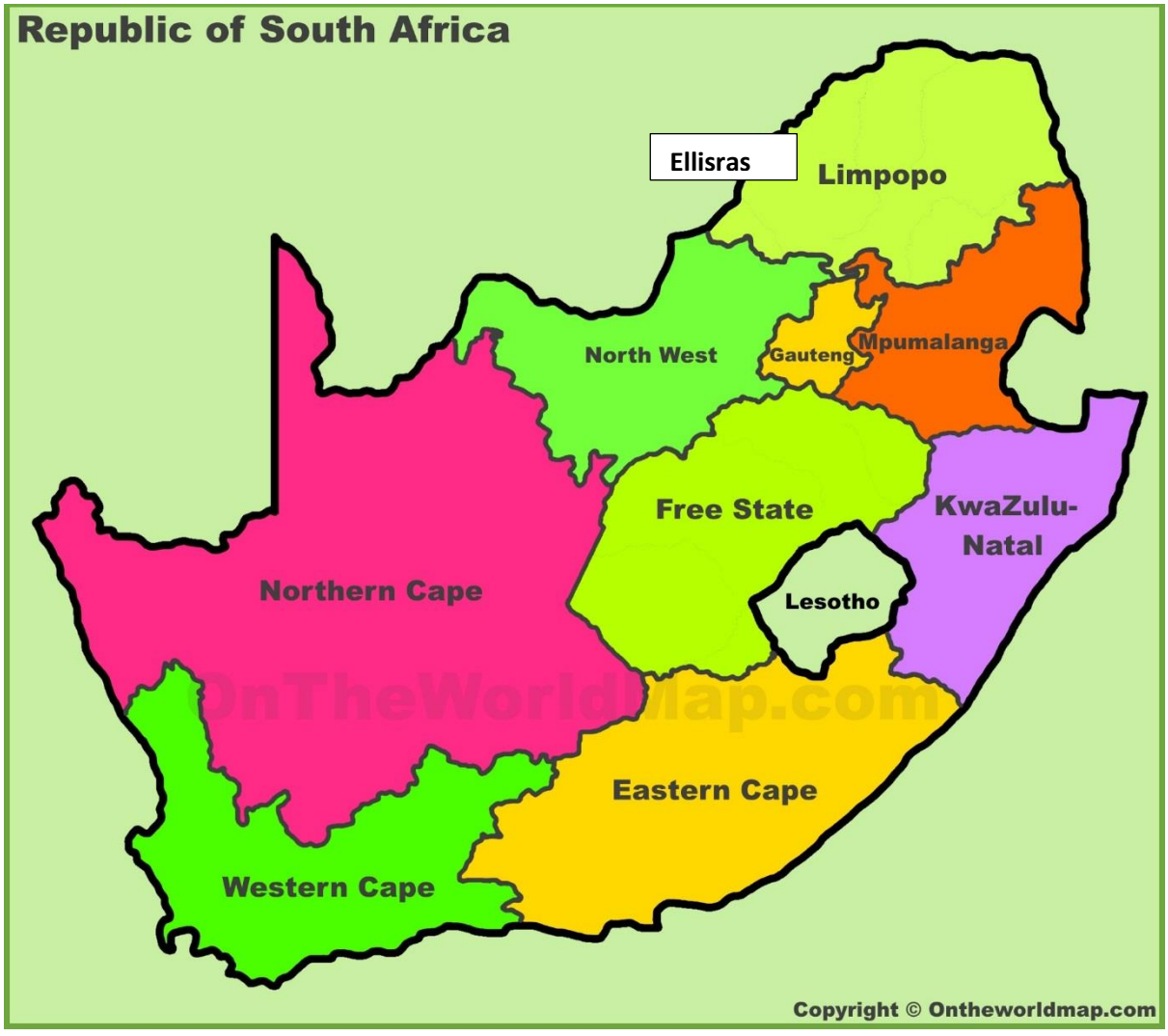


**Figure 5: The South African map showing the Ellisras area**

## 1.2. SAMPLE AND RESEARCH DESIGN

The Ellisras Longitudinal Study (ELS) was initiated in 1996 and applied a cluster sampling method (Monyeki *et al*., 1999; Monyeki *et al*., 2000). The study was undertaken at 22 schools (10 pre-schools and 12 primary schools). These school were randomly selected from a pool of 68 schools within the Ellisras area. Birth records were obtained from the principals of each school. Only those records that were verified against health clinic records were used to determine the age of potential participants. Each of the 22 selected schools were assigned a grade with the expectation that most of the children in a particular age category (i.e. 3, 4,...9,10) would be found in that grade.

The current study is based on secondary data analysis of the ELS and was conducted in two phases. Phase 1 included data analysis of all the participants in the ELS. This sample included a total number of 9002 children and adolescents (4678 boys and 4324 girls), aged 6-17 years. Parents or guardians provided written informed consent. Phase 2 consisted of biochemical analysis from a subsample of participants in the ELS. The subsample included 624 participants (306 males and 318 females) aged 18-30 years at the time the study was conducted. The Ethics Committee of the University of Limpopo granted ethical approval prior to the survey and the participants signed the informed consent forms (MREC /P/204/2013:IR).

## 1.3. MEASUREMENTS

### 3.3.1. Anthropometry

All participants underwent anthropometric measurements (waist circumference and height) according to the standard of the International Society for the Advancement of Kinanthropometry (ISAK) (Norton and Olds, 1996). The waist circumference (WC) measurements were taken to the nearest 0.1 cm, using a soft measuring tape. Height measurements were taken to the nearest 0.1 cm in barefooted study participants.

### 3.3.2. Blood pressure

At least three blood pressure (BP) readings of systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken using an Omron electronic Micronta

monitoring kit (Omron Healthcare Europe B.V, Hoofddorp, the Netherlands) after the child had been seated for 5 min or longer at 5 minutes intervals (Collaboration, 2017). The bladder of the device contained an electronic infrasonic transducer that monitors the BP and pulse rate, displaying these concurrently on the screen. This versatile instrument has been designed for research and clinical purposes (Ramoshaba *et al*., 2017). In a pilot study, conducted before the survey, a high correlation (r = 0.93) was found between the readings taken with the automated device and those taken with a conventional mercury sphygmomanometer.

To perform the blood pressure to height ratio (BPHR), hypertension was defined according to the 2004 National High Blood Pressure Education Program Working Group definition (Lu *et al*., 2011). The following equation for BPHR was used: systolic blood pressure to height ratio (SBPHR) = SBP (mm Hg)/height (cm) and diastolic blood pressure to height ratio (DBPHR) = DBP (mmHg)/height (cm) (Lu *et al*., 2011).

*Definition for hypertension*

The United States (US) National High Blood Pressure Education Program working group was used to define prehypertension and hypertension and followed the age, gender and height specificity for BP (National High Blood Pressure Education Program, 1996). Prehypertension was defined as SBP/DBP≥90th but ≤95th percentile or SBP/DBP≥120/80 mmHg and <130/90 mmHg. Hypertension stage 1 was defined as SBP/DBP≥$95^{th}$ percentile. Hypertension stage 2 was defined as SBP/DBP≥$99^{th}$ percentile+5 mm Hg. These BP references were used as gold standard (Falkner *et al*., 2004)

**Figure 6: Picture shows blood pressure measurements**

### 3.3.3. Dietary intake

Dietary intake was measured using a validated 24-hour recall method (Langenhoven, 1991). Senior Northern Sotho speaking dietetic students of the University of Limpopo, specifically trained to use the 24-hour recall method, completed interviews with participants regarding their dietary intake over the previous 24-hours. For each participant, interviews took place on one weekday and on one weekend day. An average of the two days 24-hour dietary intake was then compiled for each participant. Estimated portion sizes of foods consumed were recorded in as much detail as possible, using a pre-tested questionnaire and food models, simulating average portions of local foods (Frisancho, 1990). The different

forms of dietary fibre (total, soluble and insoluble fibre) consumed were calculated using the Food Finder 111 analysis package and are recorded and presented as grams. Briefly, the amount of food items consumed by each individual (breakfast, lunch, supper) were captured into Food Finder 111 software version 3.0 (May 2014) (FoodFinder Database). All food items were analysed and the output was saved on to an excel spreadsheet. The raw data was imported into the statistical package of the social sciences (SPSS) version 24.0 for statistical analysis.

Furthermore, a self-administered questionnaire was used to collect data on lifestyle factors, including smoking and alcohol intake.

### 3.3.4. Biochemical parameters

*Fasting blood glucose*

Fasting blood samples were collected into 4 ml grey top vacutainer tubes [vacutainer BD$^{TM}$] containing sodium fluoride and oxalate. Samples were then placed in a cooler box with ice (0–8$^{0}$C) on site prior to analysis. The fasting blood glucose (FBG) was measured using the glucose oxidase method, on a Beckman LX20$^{®}$ auto analyser (Beckman Coulter, Fullerton, CA).

*Lipid profile*

At the laboratory, fasting blood samples were centrifuged at 2500 rpm for 15 minutes to obtain plasma. Total cholesterol (TCHOL), triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) were measured using the enzymatic assay kits on a Beckman LX20$^{®}$ auto-analyser (Beckman Coulter, Fullerton, CA). Plasma was stored at -80$^{0}$C prior to analysis. Low-density lipoprotein cholesterol (LDL-C) was calculated using Friedewald equation:
([LDL-C] = [TCHOL] – [HDL-C] – [TG] / 5) (Friedewald *et al*., 1972).

*Criteria for metabolic syndrome diagnosis*

MetS was diagnosed using the new harmonised guidelines of the international diabetes federation (IDF) which requires large waist circumference (WC) of ≥94cm (males) and ≥80cm (Females) in addition to two of the following criteria: low HDL-C of <1.0 mmol/L (males) and <1.3 mmol/L (females), high TG of ≥1.7 mmol/L),

elevated BP (≥130 mmHg systole and/or ≥85 mmHg diastole), or FBG (≥5.6 mmol/L) (Alberti *et al*., 2009).

All measurements were done with an AU480 Chemistry System from Beckman Coulter (Brea, Calif). The instrument was calibrated according to standard procedures. All measurements were done in triplicate and the percentage of the coefficient of variation (CV) was calculated. Measurements were repeated when the CV > 5%. All biochemical analyses was done in the Medical Science Unit of the Department of Pathology and Medical Science at University of Limpopo.

### 1.4. QUALITY CONTROL

All training of anthropometric measurements was done in accordance with the standard procedures of the International Society for the Advancement of Kinanthropometry (ISAK) (Norton and Olds, 1996). Reliability and validity of anthropometric measurements have been reported elsewhere (Monyeki *et al*., 2002). In brief, the absolute and relative values for intra- and inter-tester technical error of measurements (TEM) for stature, ranged from 0.04-4.16 cm (0.2-5.01%) and circumference measurements ranged from 0.0-3.4 cm (0-4%) (Monyeki *et al*., 2002).

**Figure 7: Picture of fieldworkers capturing a data**

### 1.5. STATISTICAL ANALYSIS

Descriptive statistics for continuous variable such as age, height, SBP, DBP, SBPHR, DBPHR, WC, FBG, TCHOL, HDL-C, TG, LDL-C, energy, free fatty acids (FFAs), protein, carbohydrate, added sugar, fibre, saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), polyunsaturated fatty acids (PUFAs), trans fatty acids are expressed as the mean ± SD or median and inter-quartile range (IQR) for Ellisras participants aged 6-30 years. The independent t-test was applied to test the significance level ($P < 0.05$) between genders and age groups.

The $X^2$ test was used for categorical variables. For the prevalence estimates, the international diabetes federation (IDF) criteria were applied to participants who had

elevated WC in addition to two or more of other risk factors of other risk factors of metS.

*Receiver operating curve analysis*

Receiver operating characteristics curve (ROC) analysis was performed to assess the performance of SBPHR and DBPHR as an accurate tool for screening elevated SBP and DBP, respectively. The area under the curve (AUC) and 95% confidence interval (CI) for the BPHR index was performed to assess the discrimination power of the test. The AUC typically ranged from 0.5 to 1, representing the power that had poor discrimination from the one that had the perfect discrimination. A good test has an ROC skewed to the upper left corner with AUC of 1, whereas an AUC of 0.5 means that the test performs no better than chance (Schisterman *et al*., 2001; Zhou *et al*., 2009). Prehypertension and hypertension were defined by the determined optimal thresholds of the BPHR index and were used as predictive variables to compare with the gold standard. The sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and AUC (95% CI) were calculated to assess the performance of determined optimal thresholds.

The United States (US) National High Blood Pressure Education Program working group (National High Blood Pressure Education Program, 1996), was used to define prehypertension and hypertension and followed the age, gender and height specificity for BP. Prehypertension was defined as SBP/DBP≥90th but ≤95th percentile or SBP/DBP≥120/80 mmHg and <130/90 mmHg. Hypertension stage 1 was defined as SBP/DBP≥$95^{th}$ percentile. Hypertension stage 2 was defined as SBP/DBP≥$99^{th}$ percentile+5 mmHg (Falkner *et al*., 2004).

*The association between dietary intake and metabolic syndrome risk factors*

Linear regression analysis was used to investigate the association of dietary intake with various metS risk factors. The data was further unadjusted and adjusted for age, gender and energy. Dietary intake variables used in the linear regression method were log transformed prior to analysis because of their skewed distribution.

Logistic regression analysis was used to investigate the risk of dietary intake on metS risk factors. Chi-square tests were used for proportions between genders and age groups

Unadjusted and adjusted for age, gender, smoking and alcohol intake odds ratios was used to show the influence of dietary intake on metS risk factors.

*Statistical package and significance level*

All the statistical analyses were done using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA, 23.0). A p-value of $< 0.05$ was considered statistically significant.

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# CHAPTER 4

## 4. RESULTS AND DISCUSSION

## 4.1. Development of height and blood pressure to height ratio in the ELS children aged 6-17 years between 1999-2003 (Phase 1)

Table 2 shows the descriptive statistics for the height and BP by age and gender of ELS sample over time (1999-2003). As expected, the mean height for both genders increases with age overtime (134.27 boys, 134.54 girls (1999); 139.32 boys, 140.25 girls (2000); 142.96 boys, 145.18 girls (2001); 146.54 boys, 148.32 girls (2002); 150.87 boys, 152.34 girls (2003)). However, girls had significantly higher levels for changes in height compared to boys ($P<0.05$) in each years. Similar results were observed in He and Karlberg (2001). The increase in height over time is well documented and is linked to puberty stage during growth (Salerno *et al*., 1997; He and Karlberg, 2001; Zeitlin *et al*., 2003). The levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP) were high in girls but increased with age in both genders overtime ($P<0.001$). Similarly, the levels of systolic blood pressure to height ratio (SBPHR) and diastolic blood pressure-to-height ratio (DBPHR) increased in both genders overtime, although higher in girls than in boys. Peters *et al*. (2012), also reported the same trend of increasing blood pressure over time in children. These common findings are not surprising since blood pressure is proven to be closely correlated with height in children (Voors *et al*., 1977).

Table 2 Descriptive statistics for the development of height and blood pressure by age and gender of ELS children overtime (1999-2003).

| | 1999 | | 2000 | | 2001 | | 2002 | | 2003 | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **Boys(n=984) M±SD** | **Girls(n=907) M±SD** | **Boys(n=936) M±SD** | **Girls(n=877) M±SD** | **Boys(n=926) M±SD** | **Girls(n=839) M±SD** | **Boys(n=890) M±SD** | **Girls(n=823) M±SD** | **Boys(n=942) M±SD** | **Girls(n=878) M±SD** |
| **Age (y)** | 10.17±1.88 | 10.21±1.79 | 11.01±1.90 | 11.03±1.82 | 11.56±1.90 | 11.65±1.80 | 12.39±1.91 | 12.47±1.88 | 13.14±1.91 | 13.48±1.86 |
| **Height (cm)** | 134.27±9.40 | 134.54±7.80 | 139.32*±9.18 | 140.25*±9.05 | 142.96**±8.18 | 145.18**±7.15 | 146.54*±11.24 | 148.32*±10.9 | 150.87*±11.4 | 152.34*±10.7 |
| **SBP (mmHg)** | 97.77**±7.84 | 99.58**±8.61 | 99.42*±8.50 | 100.93**±8.3 | 94.55**±7.25 | 96.76**±8.05 | 102.77**±14.8 | 105.5**±14.6 | 105.15**±10.5 | 107.9**±10.8 |
| **DBP (mmHg)** | 60.59**±6.14 | 61.92**±6.26 | 65.47*±6.70 | 66.48*±7.19 | 61.85**±5.75 | 63.26**±6.12 | 61.36**±11.31 | 63.34**±10.8 | 66.98**±7.98 | 68.59**±7.57 |
| **SBPHR** | 0.73**±0.06 | 0.74**±0.07 | 0.72±0.07 | 0.72±0.06 | 0.66**±0.06 | 0.68**±0.06 | 0.70*±0.09 | 0.71*±0.08 | 0.70*±0.07 | 0.71*±0.08 |
| **DBPHR** | 0.45**±0.05 | 0.46**±0.05 | 0.59**±0.07 | 0.60**±0.06 | 0.44*±0.05 | 0.44*±0.05 | 0.70*±0.09 | 0.71*±0.08 | 0.45*±0.06 | 0.45*±0.06 |

*SBP=systolic blood pressure; DPB=diastolic blood pressure; SBPHR=systolic blood pressure to height ratio; DBPHR=diastolic blood pressure to ratio; M=mean; SD=standard deviation; *P<0.05, **P<0.001.*

*Selection of optimal thresholds of SBPHR/DBPHR for Identifying elevated blood pressure in Ellisras children*

Table 3 shows the selection of optimal thresholds of SBPHR and DBPHR for identifying elevated SBP and DBP in Ellisras children and adolescents aged 6-17 years. Because the optimal thresholds of BPHR between age groups 11-13 and 14-17 years were very close or similar to one another, the average was considered and defined as adolescents, while age groups 6-10 were defined as children (Monyeki *et al*., 1999). For SBPHR, 0.77 was selected as an optimal threshold for prehypertension among children, whereas 0.73 was selected for adolescents. For DBPHR, 0.55 was selected in children and 0.53 in adolescents. The corresponding hypertension stage 1 (SBP≥95th percentile) for SBPHR was 0.75 and 0.73 for children and adolescents, respectively; while the DBPHR for children and adolescents were 0.50 and 0.58, respectively. The discriminatory ability of optimal cut-offs of SBPHR and DBPHR for identifying prehypertension, hypertension stages 1 and 2 was satisfactory. The cut-offs seem to be better at predicting stage 2 hypertension of which the majority of children have been screened in the current study and this was consistent with the findings of other research (Lu *et al*., 2011). Therefore, this test becomes more sensitive and specific to the severe condition (Kanchanaraksa, 2008). The results of thesis could be used by Clinicians in their own practices in Ellisras rural area and similar rural settings in South African populations.

Table 3 Selection of optimal thresholds of SBPHR/DBPHR for Identifying elevated BP in ELS children aged 6-17 years, (Boys n=4678), (Girls n=4324)

| | **90 percentile ≥ SBP<90th percentile** | | | | **SBP≥95th percentile** | | | | **SBP>99th percentile + 5 mm Hg** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **Children(6-10years)** | | **Adolescents(11-17years)** | | **Children(6-10years)** | | **Adolescents(11-17years)** | | **Children(6-10years)** | | **Adolescents(11-17years)** | |
| | **Boys** | **Girls** | **Boys** | **Girls** | **Boys** | **Girls** | **Boys** | **Girls** | **Boys** | **Girls** | **Boys** | **Girls** |
| **Threshold** | 0.75 | 0.78 | 0.72 | 0.73 | 0.75 | 0.77 | 0.71 | 0.74 | 0.76 | 0.81 | 0.74 | 0.75 |
| **Total** | 0.77 | | 0.73 | | 0.76 | | 0.73 | | 0.79 | | 0.75 | |
| **Sensitivity** | 0.732 | 0.819 | 0.821 | 0.798 | 0.809 | 0.848 | 0.800 | 0.789 | 0.867 | 0.955 | 0.871 | 0.923 |
| **Total** | 0.776 | | 0.810 | | 0.829 | | 0.795 | | 0.911 | | 0.897 | |
| **Specificity** | 0.675 | 0.771 | 0.717 | 0.701 | 0.684 | 0.741 | 0.667 | 0.734 | 0.731 | 0.878 | 0.808 | 0.792 |
| **Total** | 0.723 | | 0.709 | | 0.713 | | 0.701 | | 0.805 | | 0.80 | |
| **AUC(95% CI)** | 0.764(0.678-0.850) | 0.836(0.785-0.888) | 0.836(0.798-0.874) | 0.815(0.787-0.843) | 0.856(0.811-0.900) | 0.862(0.788-0.936) | 0.797(0.726-0.868) | 0.832(0.790-0.873) | 0.884(0.800-0.968) | 0.935(0.849-1.000) | 0.908(0.863-0.954) | 0.934(0.892-0.975) |
| **Total** | 0.80(0.732-0.869) | | 0.826(0.793-0.859) | | 0.859(0.800-0.918) | | 0.815(0.758-0.871) | | 0.910(0.825-0.984) | | 0.921(0.878-0.965) | |
| | **90 percentile ≥ DBP<90th percentile** | | | | **DBP≥95th percentile** | | | | **DBP>99th percentile + 5 mmHg** | | | |
| **Threshold** | 0.54 | 0.55 | 0.53 | 0.53 | 0.54 | 0.58 | 0.58 | 0.58 | 0.59 | 0.57 | 0.57 | 0.61 |
| **Total** | 0.55 | | 0.53 | | 0.5 | | 0.58 | | 0.58 | | 0.59 | |
| **Sensitivity** | 0.707 | 0.681 | 0.887 | 0.947 | 0.809 | 0.727 | 0.745 | 0.725 | 0.800 | 0.500 | 0.710 | 0.708 |
| **Total** | 0.694 | | 0.920 | | 0.768 | | 0.735 | | 0.65 | | 0.709 | |
| **Specificity** | 0.61 | 0.625 | 0.939 | 0.949 | 0.605 | 0.684 | 0.728 | 0.701 | 0.721 | 0.639 | 0.692 | 0.747 |
| **Total** | 0.618 | | 0.944 | | 0.645 | | 0.715 | | 0.68 | | 0.720 | |
| **AUC(95% CI)** | 0.713(0.660-0.765) | 0.722(0.668-0.775) | 0.820(0.713-0.926) | 0.812(0.728-0.895) | 0.806(0.753-0.860) | 0.752(0.660-0.844) | 0.812(0.753-0.872) | 0.795(0.755-0.836) | 0.868(0.798-0.938) | 0.647(0.555-0.739) | 0.814(0.753-0.876) | 0.847(0.795-0.899) |
| **Total** | 0.718(0.664-0.77) | | 0.816(0.721-0.912) | | 0.779(0.707-0.852) | | 0.804(0.754-0.854) | | 0.758(0.677-0.839) | | 0.831(0.774-0.888) | |

*AUC=area under the curve; BP=blood pressure; CI=confidence interval; DBP=diastolic blood pressure; DBPHR=diastolic blood pressure to height ratio; ELS= ellisras longitudinal study; SBP= systolic blood pressure; SBPHR=systolic blood pressure to height ratio.*

*Performances of optimal thresholds of SBPHR/DBPHR for identifying prehypertension and hypertension in Ellisras children aged 6-17 years*

Table 4 shows the prehypertension, hypertension stages 1 and 2 redefined using the optimal thresholds of BPHR index and compared with the gold standard**.** Performance of the optimal thresholds of the SBPHR/DBPHR for detecting prehypertension, hypertension stages 1 and 2 was satisfactory based on sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV), regardless of age groups. In all prehypertension, hypertension stages 1 and 2, the NPVs were very high (≥98%), although PPVs were lower (ranging from 7–14%). The sensitivity and specificity of these methods were <70%. The NPV was above 98% for both children and adolescents indicating that all individuals with normal BP was identified. However, the PPV results indicated that individuals with true prehypertension, hypertension stages 1 and 2 only accounted to 6.9–13.5% of all screened hypertension. In other words, about 86.5–93.1% of individuals with normal BP will be misclassified into the hypertension group. The results suggested that the optimal cut-offs of SBPHR and DBPHR were accurate and acceptable for screening children with increased risk of hypertension but should not be deliberated as the diagnostic criteria. The screened hypertension individuals using SBPHR and DBPHR should further be examined and confirmed by medical professionals. The optimal thresholds of SBPHR and DBPHR for screening hypertension were established by Xi *et al*. (2014). The optimal thresholds for prehypertension were 0.81 in children and 0.70 in adolescents for SBPHR, while DBPHR was 0.52 in children and 0.46 in adolescents, and were similar to the results of Ellisras children and adolescents aged 6-17 years.

Table 4 Performances of optimal thresholds of SBPHR/DBPHR for identifying prehypertension and hypertension in ELS children aged 6-17 year.

| | **Prehypertension** | | **Hypertension(stage 1)** | | **Hypertension(stage 2)** | |
|---|---|---|---|---|---|---|
| | **6-10 years** | **11-17 years** | **6-10 years** | **11-17 years** | **6-10 years** | **11-17 years** |
| **Threshold (SBPHR/DBPHR)** | 0.77/0.55 | 0.73/0.53 | 0.76/0.56 | 0.73/0.58 | 0.79/0.58 | 0.75/0.59 |
| **Sensitivity** | 0.780 | 0.809 | 0.831 | 0.798 | 0.911 | 0.900 |
| **Specificity** | 0.983 | 0.701 | 0.710 | 0.700 | 0.998 | 0.800 |
| **PPV** | 0.135 | 0.135 | 0.074 | 0.069 | 0.089 | 0.084 |
| **NPV** | 0.983 | 0.985 | 0.993 | 0.992 | 0.998 | 0.997 |

*DBPHR=diastolic blood pressure to height ratio; ELS=ellisras longitudinal study; NPV=negative predictive value; PPV=positive predictive value; SBPHR=systolic blood pressure to height ratio.*

## 4.2. Metabolic syndrome risk factors in the ELS subsample, aged 18-30 years (Phase 2)

Table 5 shows the mean values for the metabolic syndrome (MetS) risk factors and dietary intake of the participants, by age group and gender. Overall, females appear to have higher mean values than males for waist circumference (WC), fasting blood glucose (FBG), total cholesterol (TCHOL) and low-density lipoprotein cholesterol (LDL-C), while they have lower mean values for high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), systolic blood pressure (SBP) and diastolic blood pressure (DBP) than males. Consistent differences have been reported in many studies (Van Zyl *et al*., 2012; Benmohammed *et al*., 2016; Kruger and Nell, 2017). This trend is consistent, when disaggregating the sample by age groups. However, the only significant differences between males and females were recorded for WC (75.09±9.53 and 82.14±14.37, respectively) and SBP (125.91±12.78 and 114.23±10.84, respectively). This significant gender difference was also observed in the 25-30 year group. The same trend was found in Yoon *et al*. (2014). With regard to differences between age groups, most risk factors were higher in the older age group (25-30 years), except for FBG and HDL-C (5.56±0.91 and 1.16±0.31) which were higher in 18-24 year age group compared to 1.16±0.31 and 1.14±0.35 in 25-30 year age group, respectively). There were no significant differences between age groups for all the measured risk factors except for the DBP where the mean value was 68.78±9.37 in age group 18-24 years compared to 70.96±10.05 in age group 25-30 years ($p<0.05$). However, the majority of studies did not dichotomize their study participants into age groups. Nonetheless, the increase in metS risk factors with age is well known. The WC mean values increased with age and differ amongst gender (higher in females than males). Similarly reported in Van Zyl *et al*. (2012). Therefore, more studies focused on gender with regard to the pathophysiological mechanism of central fat distribution are required in addition to those studies on the proportion of genetic predisposition, environmental factors and dietary diversity that might influence the development of this syndrome. Total energy intake in this study falls below the dietary reference intakes (DRIs) for both males and females. However, females tend to have a higher energy intake than males. This was also

observed in studies conducted in other rural South Africa (SA) (Kruger *et al*., 2006; Steyn and Temple, 2012). This supports that, the large proportion of black South African consume less energy than recommended (Labadarios *et al*., 2011(a); Labadarios *et al*., 2011(b)). This was also observed in a Kwazulu-Natal black population (Kolahdooz *et al*., 2013).

Table 5 further provides information regarding the macronutrients consumed. Females consumed more carbohydrates, added sugar, fibre and saturated fat; while males consumed more total fats, proteins and mono-and poly-unsaturated fats. Females had a higher intake of added sugar than males. This was expected since high energy median values were found in females compared to males. This is explained by the rapid progression of nutritional and health transition in SA. The median fibre intake observed for both genders were lower than the Recommended Dietary Allowance (RDA) (Schneider *et al*., 2007). Our results were in line with that of a study conducted in the North West province, where fibre intakes were within the recommended amounts (Wentzel-Viljoen and Kruger, 2005). High dietary fibre intake is stipulated to lower the development of obesity since its effect contributes to a decrease in appetite (Slavin, 2005). Male participants had a higher intake of protein than females. There is evidence that intake of a high protein protect against the metS, hence males had low prevalence of metS as compare to females, although the high dietary protein in the management for metS is still controversial (Wojcik *et al*., 2016).

Table 5 Descriptive statistics for metS risk factors of Ellisras adults by age group and gender

| | 18-24 years | | | 25-30 years | | | 18-30 years | | |
|---|---|---|---|---|---|---|---|---|---|
| **Risk factors** | **Males(n=103)** | **Females (n=101)** | **Total (N=204)** | **Males(n=203)** | **Females(217)** | **Total (N=420)** | **Males(n=306)** | **Females(n=318)** | **Total (N=624)** |
| | **M±SD** | **M±SD** | **M±SD** | **M±SD** | **M±SD** | **M±SD** | **M±SD** | **M±SD** | **M±SD** |
| **WC (cm)** | 72.07±7.27 | 78.05±13.36 | 75.03±11.11 | 76.62*±10.17 | 84.04*±14.46 | 80.46±13.09 | 75.09*±9.53 | 82.14*±14.37 | 78.68±12.73 |
| **FBG (mmol/L)** | 5.54±0.91 | 5.78±0.91 | 5.56±0.91 | 5.40±0.84 | 5.63±1.77 | 1.16±0.31 | 5.45±0.87 | 5.62±1.55 | 5.53±1.26 |
| **TCHOL (mmol/L)** | 4.02±0.87 | 4.07±1.03 | 4.04±0.95 | 4.04±0.95 | 4.35±1.13 | 4.20±1.06 | 4.03±0.92 | 4.62±1.11 | 4.15±1.03 |
| **HDL-C (mmol/L)** | 1.23±0.34 | 1.09±0.28 | 1.16±0.31 | 1.19±0.39 | 1.10±0.31 | 1.14±0.35 | 1.20±0.37 | 1.10±0.30 | 1.15±0.34 |
| **TG (mmol/L)** | 0.96±0.60 | 0.87±0.48 | 0.92±0.54 | 1.11±0.67 | 1.00±0.52 | 1.05±0.60 | 1.06±0.65 | 0.96±0.51 | 1.01±0.59 |
| **LDL-C (mmol/L)** | 2.61±0.71 | 2.80±0.89 | 2.71±0.81 | 2.63±0.81 | 3.05±0.96 | 2.85±0.92 | 2.62±0.78 | 2.97±0.95 | 2.80±0.89 |
| **SBP (mmHg)** | 123.20±12.30 | 112.61±9.16 | 117.95±12.06 | 127.29*±12.37 | 114.98*±11.49 | 120.93±13.41 | 125.91**±12.78 | 114.23**±10.84 | 119.96±13.05 |
| **DBP (mmHg)** | 68.89±9.58 | 68.66±9.20 | 68.78*±9.37 | 72.73±10.35 | 69.31±9.50 | 70.96*±10.05 | 71.44±10.34 | 69.10±9.39 | 70.25±9.88 |
| **Dietary intake** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** | **Median(IQR)** |
| **Energy (kj)** | 3520.0(3646.50) | 3314.0(2919.0) | 3486.0(3299.50) | 2886.0(3967.50) | 3674.0(3992.50) | 3213.50(3953.50) | 3029.0(3874.0) | 3474.0(3482.0) | 3310.0(3591.0) |
| **Fatty acids (%)** | 23.18(17.73) | 20.71(22.37) | 21.43(20.01) | 22.22(26.10) | 22.75(25.80) | 22.50(26.80) | 22.6(21.51) | 22.1(24.26) | 22.3(23.50) |
| **Protein (%)** | 14.48(11.67) | 11.55(9.24) | 13.17(10.45) | 12.07(13.75) | 11.68(12.78) | 12.03(13.36) | 12.9(12.34) | 11.7(11.19) | 12.3(11.76) |
| **Carbohydrate (%)** | 61.81(24.49) | 65.98(28.80) | 63.31(23.73) | 63.76(35.57) | 62.29(33.42) | 62.79(34.60) | 62.8(30.82) | 63.7(30.91) | 63.0(31.41) |
| **Added sugar (g)** | 24.40(39.83) | 34.70(49.30) | 27.70(40.65) | 24.00(49.70) | 25.80(38.45) | 25.80(39.90) | 24.0(45.50) | 26.0(36.00) | 25.8(39.50) |
| **Fibre (g)** | 5.60(8.18) | 5.90(7.80) | 5.70(7.85) | 3.80(7.15) | 4.80(9.85) | 4.05(8.43) | 4.3(7.00) | 5.1(9.00) | 4.6(8.40) |
| **SFAs (%)** | 5.83(6.42) | 4.56(8.42) | 5.28(7.75) | 4.14(10.06) | 5.28(10.62) | 5.02(10.44) | 4.8(8.54) | 5.0(9.91) | 4.9(9.36) |
| **MUFAs (%)** | 8.20(9.55) | 5.39(10.50) | 6.67(10.03) | 5.19(13.77) | 6.97(14.95) | 6.48(14.79) | 6.6(11.75) | 6.4(14.01) | 6.5(12.90) |
| **PUFAs (%)** | 5.07(7.37) | 2.97(7.67) | 4.02(7.63) | 2.97(7.74) | 4.16(8.61) | 3.44(8.39) | 3.7(7.57) | 3.4(8.18) | 3.7(7.95) |
| **Trans fatty acids (%)** | 0.18(1.10) | 0.12(1.05) | 0.14(1.05) | 0.12(0.40) | 0.12(1.27) | 0.12(0.81) | 0.1(0.49) | 0.1(1.17) | 0.1(0.95) |

*Data on lipid profile and anthropometry are presented as M±SD, while dietary intake data is presented as median (IQR). n=number of participants; WC=waist circumference; FPG=fasting blood glucose; TCHOL=total cholesterol; HDL=high density lipoprotein; LDL=low density lipoprotein; TG=triglycerides; CHO= carbohydrates; SBP=systolic blood pressure; DBP=diastolic blood pressure; SFAs= saturated fatty acids; MUFAs = monounsaturated fatty acids; PUFAs= polyunsaturated fatty acids; TFAs=trans fatty acids; IQR=interquartile range; M=mean; SD=standard deviation; **p <0.001; * P<0.05*

Globally, the prevalence of the metS is on the rise, and this increase is the case in developing countries, in Africa and in South Africa (SA). The interesting thing is that in SA the metS prevalence seems to be differentiated by gender and ethnicity in that it is higher in black (>60%) compared to white (±55%) South Africans when the Joint Statement definition criterion is used (Hoebel *et al*., 2011). However, when other definition criteria are used (especially the IDF definition criteria) the prevalence becomes lower in black Africans (46.5%) when compared to white Africans (74.1%) (Erasmus *et al*., 2012). Motala *et al*. (2011) on the other hand showed that the metS prevalence is different amongst genders in that more females (25%, 21.2% and 16.8%) present with metS when compared to their male counterparts (10%, 11.2% and 7.9%) using JIS, IDF and ATP 111 definition criteria, respectively. Furthermore, when the WHO definition criteria is used it appears as though the prevalence of metS is estimated to be high (59.1%) in African countries such as Nigeria when compared to other developing countries like Turkey (19%) (Isezuo and Ezunu, 2005; Can and Bersot, 2007). However, when the NCEP-ATP III and IDF definition criteria are used the metS prevalence is shown to be higher in Turkey (38% and 42%, respectively), than in Africa (Cameron *et al*., 2007).

In the current study, the prevalence of metS was estimated using the IDF criteria. Overall, the prevalence of metS was 23.1% (8.6% males and 36.8% females). This prevalence is lower than the prevalence shown in South African coloured participants in Erasmus *et al*. (2012) study, but they are higher than the ones shown in black North West residents (9.5% for females and 6.8% for males), participating in Hoebel *et al*. (2011) study. Additionally, the current population had high metS prevalence than that reported in low-income black South Africans (Owolabi *et al*., 2018). However, their males had higher prevalence than those in the current study. We have to bear in mind that the definition criteria used for metS in these studies were the IDF criteria.

On examining factors that seemed to influence metS prevalence in the current study, it appears as though age and gender were the main determinants of this condition. In fact, being older influenced the health status of the participants especially the mean SBP and adiposity as shown by the values that were higher in the older age group compared to the younger age group. The majority of females also presented with larger WC and higher levels of total plasma cholesterol and lower levels of HDL-C

mean values. The South African National Health and Nutrition Examination Survey (SANHANES) reported the same results (Shisana *et al*., 2014). Furthermore, mean SBP values also differed significantly by gender with males presenting with higher mean values than females in the same survey (Shisana *et al*., 2014). Systolic and diastolic blood pressure also seemed to increase with age. However, no significant gender differences were observed in terms of total cholesterol (TCHOL).

## 4.3. The prevalence of metabolic syndrome risk factors and dietary intake among subsample aged 18-30 years

*The overall prevalence of the metS risk factors in the total sample, male and female age group 18-30 years*

Figure 8 shows the observed prevalence of each metS risk factor of the total sample of participants. Overall, significantly more females presented with increased WC than males (51.9% vs 4.6%). These results are similar with the South African evidence which suggested that in the North West province, which is located close to Ellisras (Limpopo Province), the majority of females (43.5%) had a WC that was above 88cm compared to 8% of males that presented with a WC above 102 cm (Shisana *et al*., 2014). On the contrary, Bacopoulou *et al*. (2015), presented evidence in which WC was higher in males than females. This is explained by the fact that central fat distribution is more dominant in males than females. Waist circumference was similar amongst gender in other studies (Nasila *et al*., 2013; Longo-Mbenza *et al*., 2011). There is evidence that increased visceral adiposity leads to the insulin resistance that is central in the pathogenesis of metS and it is further, associated with the production of adipocytokines, which leads to the low grade inflammatory response observed in metS (Omuse *et al.*, 2017). Several prospective studies have consistently reported a protective effect of increased HDL-C against cardiovascular diseases (CVDs), in which low levels are associated with metS (Wong *et al*., 2012).

The trends of gender difference was also observed with elevated TCHOL (22.0% for females vs 11.1% for males), and LDL-C (42.8% for females vs 26.8% for males), as well as reduced HDL-C (77.7% for females vs 29.4% for males). Similarly, other studies also reported that reduced HDL-C was prominently higher in females than in males (Kuk and Ardern, 2010; Peer *et al*., 2015). Significantly more males presented with increased SBP, DBP and hypertension (33.0%, 9.2% and 34.0%, respectively)

compared to their female counterparts (7.9%, 5.0% and 9.4%, respectively) (SBP and hypertension were $p<0.001$, while DBP was $p<0.05$). These findings were in contrast to those of Kruger and Nell (2017) as they reported higher BP in females than in males. The differences in these results can be explained by factors other than the criteria used, as both of studies used the same definition. Overall, the highest prevalence risk factors are reduced HDL-C, elevated FBG, and increased LDL-C (ranging from 54.0%, 46.3% and 34.9%, respectively). The lowest prevalence risk factors are increased DBP, increased TG and high TCHOL (ranging from 7.1%, 9.9% and 16.7%, respectively). The current study population did not show any significant differences between FBG and TG among genders. Whereas, Kruger and Nell (2017) found that, only TG showed differences between males and females. This is explained by the testosterone concentration which is associated with metS (Grosman *et al*., 2014).

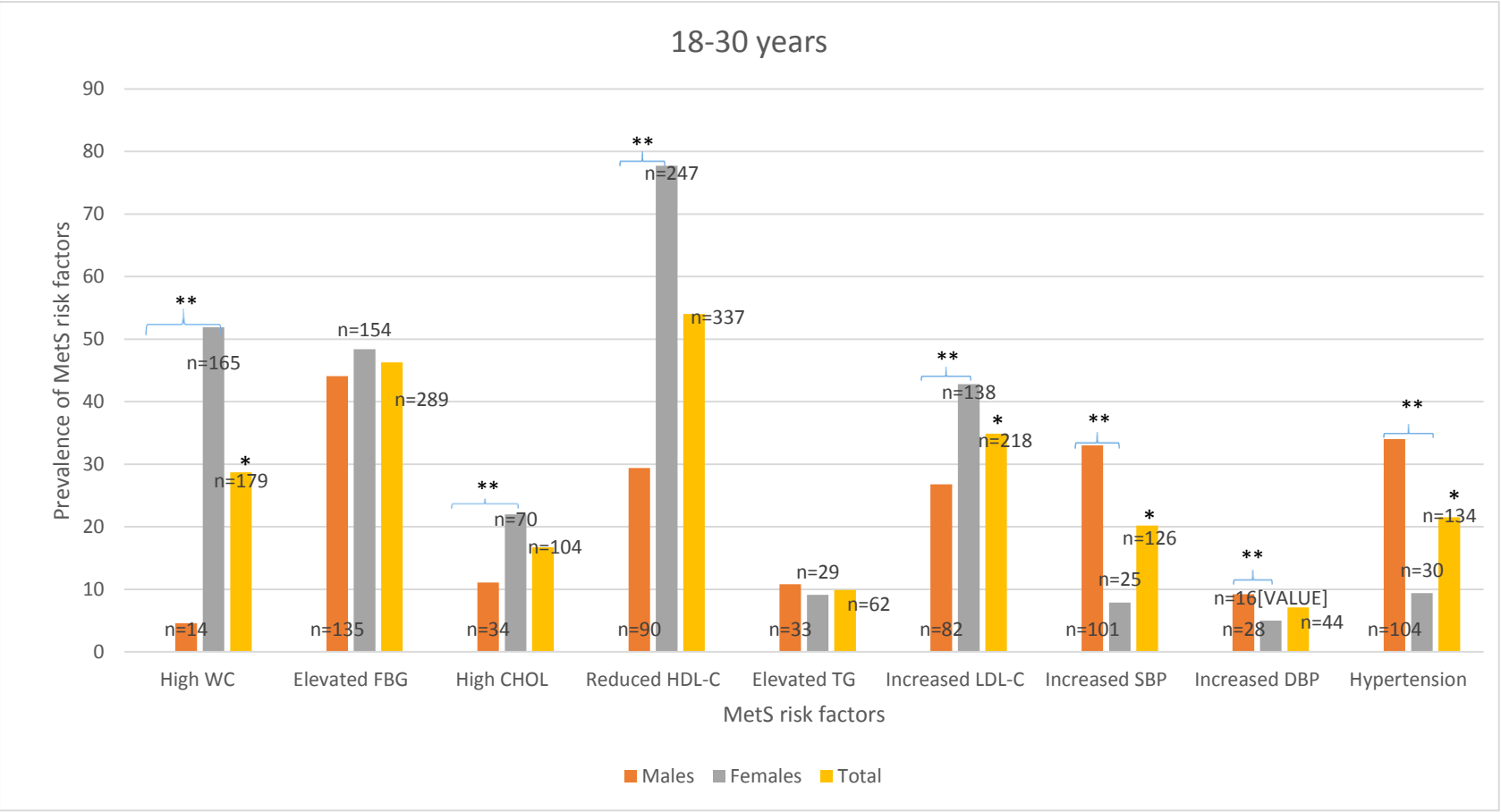


**Figure 8 Prevalence of the metabolic syndrome risk factors in the total sample, males and females in 18-30 years of Ellisras young adults.**

*n=number of participants; WC=waist circumference; FBG=fasting blood glucose; CHOL=cholesterol; TG=triglycerides; HDL-C=high-density lipoprotein cholesterol; LDL-C=low-density lipoprotein cholesterol;**p<0.001; *p<0.05.*

*The prevalence of the metS risk factors in the total sample, male and female aged-group 18-24 years*

The gender difference trend was observed even in figure 9, when the sample data was disaggregated by age groups in that, significantly more females aged 18-24 years presented with larger WC and a high reduced HDL (37.6% and 77.2%) than males (1.9% and 24.3%). Similar results have been reported in many studies (Van Zyl et al., 2012; Benmohammed *et al*., 2016; Kruger and Nell, 2017). Several prospective studies have consistently reported a protective effect of HDL-C against cardiovascular diseases (CVDs), in which low levels are associated with metS (Wong et al., 2012). Males within the younger age group (18-24 years) had significantly higher DBP and hypertension levels than females (23.3% vs 5.0% and 23.3% vs 8.9%, respectively). Similarly, Berry *et al*. (2017) reported the age difference with respect to hypertension. The same trend was observed in the Demographic and Health Survey (DHS) report; however, females had a higher blood pressure than their male counterparts (Puoane *et al*., 2002). The highest prevalence risk factors in this particular age group are reduced HDL-C, elevated FBG and increased LDL-C (ranging from 50.5%, 47.5% and 28.4%, respectively). Similar findings were reported by Motala *et al*. (2011). The continuation of the current trend will put this population at an increased risk of CVDs. Especially, cerebrovascular disease since artery disease is still uncommon in Africans (Mayosi *et al*., 2009). The lowest prevalence risk factors are increased DBP, elevated TG and high TCHOL (ranging from 6.9%, 7.8% and 13.7%, respectively).

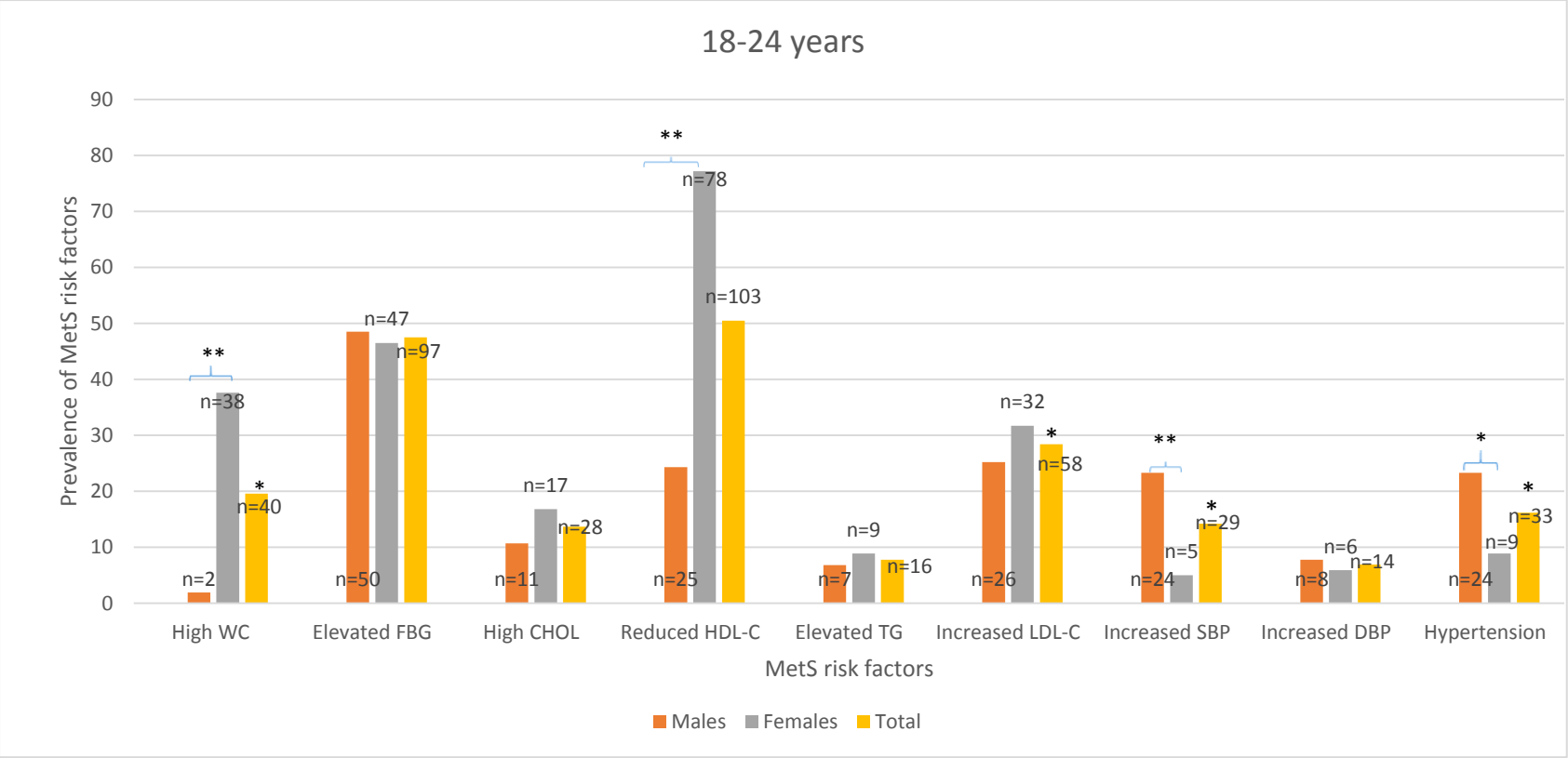


**Figure 9 Prevalence of metabolic syndrome risk factors in the total sample, males and females in age group 18-24 years of Ellisras young adults**

*n=number of participants; WC=waist circumference; FBG=fasting blood glucose; CHOL=cholesterol; TG=triglycerides; HDL-C=high-density lipoprotein cholesterol, LDL-C=low-density lipoprotein cholesterol, **p<0.001; *p<0.05.*

*The prevalence of the metS risk factors in the total sample, male and female aged-group 25-30 years*

As shown in Figure 10, consistent trends were observed in the older age group (25-30 years), as larger WC and high reduced HDL-C were significantly higher in females compared to males (58.5% vs 5.9% and 77.9% vs 32.0%, respectively). Similar results were observed in other studies ((Van Zyl *et al*., 2012; Benmohammed *et al*., 2016; Kruger and Nell, 2017). In addition, females had significantly higher TCHOL than males (24.4% vs 11.3%). Similar to the younger age group (18-24 years), males presented with higher SBP, DBP and hypertension than females (37.9% vs 9.2%, 9.9% vs 4.6% and 39.4% vs 9.7%, respectively). This was the case in previous results of the same population when the participants were still in their childhood stage (Sekgala *et al*., 2017). Changes in blood pressure between gender and age group over time in rural population requires close attention. Overall, the highest prevalence risk factors in this older age group are reduced HDL-C, elevated FBG and increased LDL-C (ranging from 55.7%, 45.7% and 38.1%, respectively). Similar, results were reported by Motala *et al*. (2011). The lowest prevalence risk factors are increased DBP, elevated TG and high TCHOL (ranging from 7.1%, 11.0% and 18.1%, respectively). The significant role plays by these risk factors in influencing the prevalence of the metS is not clear, therefore, further research is needed to bridge this gap.

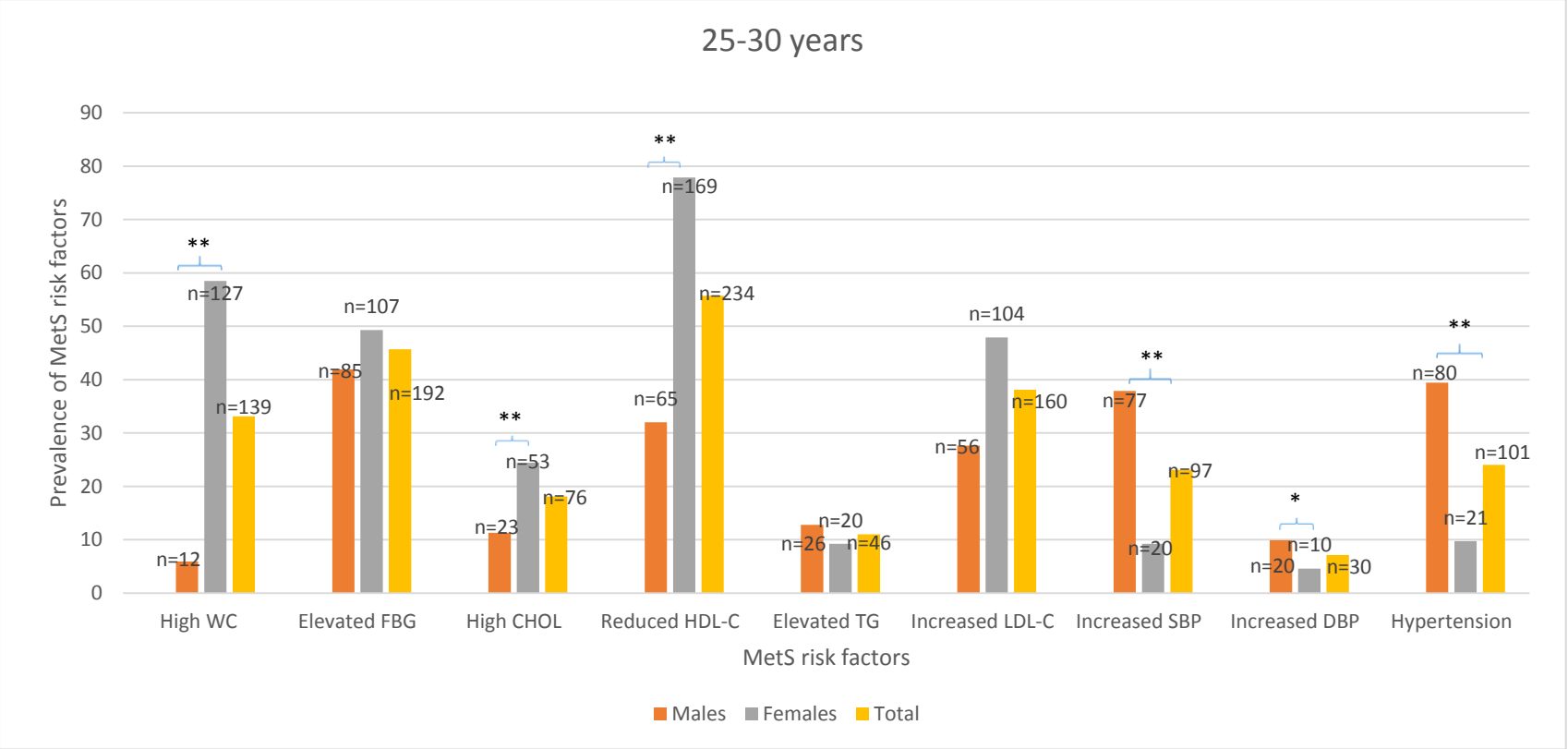


**Figure 10 Prevalence of metabolic syndrome risk factors in the total sample, males and females in age group 25-30 years of Ellisras young adults**

*n=number of participants; WC=waist circumference; FBG=fasting blood glucose; CHOL=cholesterol; TG=triglycerides; HDL-C=high-density lipoprotein cholesterol; LDL-C=low-density lipoprotein cholesterol;**p<0.001;*p<0.05*

As shown in table 6, although the study participants consumed less energy than recommended, the majority of males had high total energy, high protein and low fibre intake when compared to females (95.8% vs 17.6%, $p<0.001$; 5.9% vs 1.6%, $p<0.05$ and 99.3% vs 96.2%, $p<0.05$, respectively). Similar results were reported in other studies conducted in South Africa (Kruger and Nell, 2017; Steyn and Temple, 2012). This trend also seemed to be similar when data was disaggregated by age, such that 99.0% of males within the age group 18-24 years had high total energy intake compared to 7.9% females. Males had high total energy, high protein intake and low fibre intake, compared females in age group 25-30 years. Unfortunately, the majority of studies reported only the mean values for energy and overall dietary intakes. Females on the other hand had overall high added sugar intake compared to males (56.9% vs 48.4%, $p<0.05$) with this gender trend also observed in the majority (66.3%) of the 18–24 year old females compared to 48.5% of males ($p<0.05$). Lastly, the majority of females 25-30 years also had higher trans fatty acid intake than males (27.6% vs 17.7%). There were no significant age differences in terms of dietary intake except for protein intake, where more 25-30 year olds males consumed high levels of protein when compared to females (4.5% vs 2.0%, $p<0.05$).

Table 6 The prevalence of dietary intake of the participants

| | 18-24 years | | | 25-30 years | | | 18-30 years | | |
|---|---|---|---|---|---|---|---|---|---|
| **dietary intake** | **Males(n=103)** | **Females(n=101)** | **Total(n=204)** | **Males(n=203)** | **Females(217)** | **Total(n=420)** | **Males(n=306)** | **Females(n=318)** | **Total(n=624)** |
| | **(%)n** | **(%)n** | **(%)n** | **(%)n** | **(%)n** | **(%)n** | **(%)n** | **(%)n** | **(%)n** |
| **High energy male>10626; female>8465** | 99.0**(102) | 7.9**(8) | 53.9(110) | 94.1**(191) | 22.1**(48) | 56.9(239) | 95.8**(293) | 17.6**(56) | 55.9(349) |
| **High fatty acids ≥35%** | 18.4(19) | 19.8(20) | 19.1(39) | 23.6(48) | 24.0(52) | 23.8(100) | 21.9(67) | 22.6(72) | 22.3(139) |
| **High protein ≥35%** | 2.9(3) | 1.0(1) | 2.0*(4) | 7.4*(15) | 1.8*(4) | 4.5*(19) | 5.9*(18) | 1.6*(5) | 3.7(23) |
| **High carbohydrate ≥65%** | 40.8(42) | 50.5(51) | 45.6(93) | 48.3(98) | 44.7(97) | 46.4(195) | 45.8(140) | 46.5(148) | 46.2(288) |
| **High added sugar <25g** | 48.5*(50) | 66.3*(67) | 57.4(117) | 48.3(98) | 52.5(114) | 50.5(212) | 48.4*(148) | 56.9*(181) | 52.7(329) |
| **Low fibre male=38g; female=25g** | 99.0(102) | 97.0(98) | 98.0(200) | 99.5*(202) | 95.9*(208) | 97.6(410) | 99.3*(304) | 96.2*(306) | 97.8(610) |
| **High saturated fatty acids <10%** | 22.3(23) | 23.8(24) | 23.0(47) | 27.6(56) | 30.9(67) | 29.3(123) | 25.8(79) | 28.6(91) | 27.2(170) |
| **High monounsaturated fatty acids ≥20%** | 4.9(5) | 6.9(7) | 5.9(12) | 7.4(15) | 7.4(16) | 7.4(31) | 6.6(20) | 7.2(23) | 6.9(43) |
| **High polyunsaturated fatty acids ≥10%** | 22.3(23) | 19.8(20) | 21.1(43) | 18.2(37) | 22.6(49) | 20.5(86) | 19.6(60) | 21.7(69) | 20.7(129) |
| **High trans fatty acids <1%** | 26.2(27) | 25.7(26) | 26.0(53) | 17.7*(36) | 27.6*(60) | 22.9(96) | 20.6(63) | 27.0(86) | 23.9(149) |

*n= number of participants; **p <0.001; *p <0.05*

*The association between dietary intake and metabolic syndrome risk factors*

Table 7 shows the linear regression analysis undertaken to show the association of each log dietary intake variable with different metS risk factors. The results showed no association between log total energy, log added sugar, log SFA and log MUFA with metabolic risk factors. There was a low and negative significant association between log fibre with SBP and DBP (β:-0.004, p=0.003 and β:-0.004, p=0.046), respectively, for unadjusted. After adjusting for the potential confounding factors, log fibre was also associated with FBG (β:-0.028,p=0.046). Log PUFAs was inversely associated with FBG, HDL-C and SBP crude. Log trans fatty acids was inversely associated with WC, HDL-C and SBP crude. Both log PUFAs and log trans fatty acids were not associated with any metabolic risk factors after adjusting for potential cofounding factors. Log protein was inversely associated with SBP both crude and adjusted for potential cofounding factors.

Table 7 Regression coefficient showing the association of dietary intake with various metS risk factors of Ellisras adults

| | ENERGY (kj) | | ADDED SUGAR (g) | | FIBRE (g) | | Saturated fatty acids (%) | | PUFAs (%) | | MUFAs (%) | | Trans fatty acids (%) | | Carbohydrates (%) | | Protein (%) | | Fatty acids (%) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Crude** | | | | | | | | | | | | | | | | | | | | |
| | β | SE | β | SE | β | SE | β | SE | β | SE | β | SE | β | SE | β | SE | β | SE | β | SE |
| **Age (y)** | -0.034** | 0.009 | -0.016 | 0.014 | -0.038** | 0.009 | -0.020* | 0.009 | -0.029* | 0.009 | -0.026* | 0.01 | -0.008 | 0.006 | -0.033** | 0.01 | -0.065** | 0.016 | -0.069** | 0.02 |
| **Gender** | 0.009 | 0.036 | 0.108* | 0.055 | 0.038 | 0.035 | 0.004 | 0.038 | -0.004 | 0.037 | -0.005 | 0.042 | 0.033 | 0.025 | 0.007 | 0.039 | -0.04 | 0.064 | -0.008 | 0.07 |
| **WC (cm)** | -0.002 | 0.001 | 0.001 | 0.002 | -0.001 | 0.001 | -0.001 | 0.001 | -0.002 | 0.001 | -0.002 | 0.002 | -0.002* | 0.001 | 0.001 | 0.002 | -0.002 | 0.003 | -0.004 | 0.000 |
| **FBG (mmol/L)** | -0.020 | 0.014 | 0.013 | 0.022 | -0.03* | 0.014 | -0.007 | 0.015 | -0.016* | 0.016 | -0.012 | 0.017 | -0.005 | 0.010 | -0.016 | 0.015 | -0.025 | 0.025 | -0.031 | 0.030 |
| **TCHOL (mmol/L)** | -0.011 | 0.017 | -0.008 | 0.027 | -0.015 | 0.017 | -0.016 | 0.018 | -0.007 | 0.018 | -0.016 | 0.020 | -0.001 | 0.012 | -0.031 | 0.019 | -0.034 | 0.031 | -0.046 | 0.040 |
| **HDL-C (mmol/L)** | 0.027 | 0.053 | -0.097 | 0.082 | -0.004 | 0.052 | 0.071 | 0.056 | 0.042 | 0.055 | 0.067 | 0.062 | 0.074* | 0.037 | -0.025 | 0.057 | -0.024 | 0.094 | -0.004 | 0.110 |
| **TG (mmol/L)** | -0.033 | 0.031 | -0.027 | 0.047 | -0.026 | 0.030 | -0.032 | 0.032 | -0.024 | 0.032 | -0.022 | 0.036 | -0.03 | 0.021 | -0.015 | 0.033 | -0.043 | 0.055 | -0.058 | 0.060 |
| **LDL-C (mmol/L)** | -0.014 | 0.02 | 0.006 | 0.031 | -0.006 | 0.021 | -0.028 | 0.021 | -0.014 | 0.021 | -0.028 | 0.024 | -0.009 | 0.014 | -0.036 | 0.022 | -0.037 | 0.036 | -0.054 | 0.04 |
| **SBP (mmHg)** | -0.002 | 0.001 | 0.002 | 0.001 | -0.004* | 0.001 | -0.002 | 0.001 | -0.003* | 0.001 | -0.002 | 0.002 | -0.002* | 0.001 | -0.002 | 0.001 | -0.006* | 0.002 | -0.006* | 0.000 |
| **DBP (mmHg)** | -0.002 | 0.002 | 0.001 | 0.003 | -0.004* | 0.002 | -0.002 | 0.002 | -0.003 | 0.002 | -0.003 | 0.002 | -0.002 | 0.001 | -0.004* | 0.002 | -0.007* | 0.003 | -0.007 | 0.000 |
| **Adjusted for age, gender and energy** | | | | | | | | | | | | | | | | | | | | |
| **Age (y)** | -0.031** | 0.009 | -0.015 | 0.014 | -0.037** | 0.009 | -0.016 | 0.01 | -0.026* | 0.01 | -0.002 | 0.011 | -0.004 | 0.006 | -0.032* | 0.01 | -0.062** | 0.016 | -0.063** | 0.020 |
| **Gender** | 0.001 | 0.045 | 0.165* | 0.070 | -0.002 | 0.044 | 0.003 | 0.048 | -0.002 | 0.047 | -0.002 | 0.053 | 0.036 | 0.031 | -0.002 | 0.049 | -0.199 | 0.08 | -0.049 | 0.090 |
| **WC (cm)** | 0.001 | 0.002 | -0.002 | 0.002 | 0.001 | 0.002 | 0.001 | 0.002 | 0.001 | 0.002 | -0.001 | 0.002 | -0.001 | 0.001 | 0.002 | 0.002 | 0.002 | 0.003 | 0.001 | 0.000 |
| **FBG (mmol/L)** | -0.017 | 0.014 | 0.008 | 0.022 | -0.028 | 0.014 | -0.002 | 0.015 | -0.011 | 0.015 | -0.006 | 0.017 | -0.001 | 0.01 | -0.013 | 0.016 | -0.017 | 0.025 | -0.023 | 0.030 |
| **TCHOL (mmol/L)** | -0.198 | 0.302 | -0.436 | 0.471 | 0.017 | 0.295 | -0.217 | 0.321 | -0.009 | 0.315 | -0.203 | 0.354 | -0.124 | 0.210 | 0.245 | 0.329 | -0.251 | 0.536 | -0.226 | 0.62 |
| **HDL-C (mmol/L)** | 0.223 | 0.304 | 0.350 | 0.475 | -0.049 | 0.297 | 0.305 | 0.324 | 0.054 | 0.315 | 0.280 | 0.357 | 0.21 | 0.212 | 0.239 | 0.331 | 0.233 | 0.540 | 0.236 | 0.620 |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **TG (mmol/L)** | 0.030 | 0.069 | 0.081 | 0.107 | -0.001 | 0.067 | 0.034 | 0.073 | 0.002 | 0.072 | 0.047 | 0.081 | 0.014 | 0.048 | 0.064 | 0.075 | 0.049 | 0.122 | 0.046 | 0.140 |
| **LDL-C (mmol/L)** | 0.194 | 0.304 | 0.431 | 0.475 | -0.021 | 0.297 | 0.190 | 0.324 | 0.003 | 0.318 | 0.179 | 0.357 | 0.113 | 0.212 | 0.214 | 0.331 | 0.238 | 0.540 | 0.193 | 0.620 |
| **SBP (mmHg)** | -0.002 | 0.002 | 0.004 | 0.003 | -0.004* | 0.002 | -0.002 | 0.002 | -0.003 | 0.318 | -0.002 | 0.002 | -0.002 | 0.001 | -0.001 | 0.002 | -0.007* | 0.004 | -0.006 | 0.000 |
| **DBP (mmHg)** | 0.001 | 0.002 | -0.002 | 0.004 | 0.001 | 0.002 | 0.001 | 0.003 | 0.001 | 0.003 | 0.001 | 0.341 | 0.001 | 0.002 | -0.002 | 0.003 | 0.001 | 0.004 | 0.001 | 0.01 |

*WC=waist circumference; FPG=fasting blood glucose; TCHOL=total cholesterol; HDL-C=high density lipoprotein cholesterol; LDL-C=low density lipoprotein cholesterol; TG=triglycerides; CHO=carbohydrates; SBP=systolic blood pressure; DBP=diastolic blood pressure; SFAs=saturated fatty acids; PUFAs=polyunsaturated fatty acids; MUFAs= monounsaturated fatty acids; TFA=trans fatty acids; SE=standard error; **p<0.001; *p<0.05*

Literature on the association between metS risk factors with dietary intake is limited, particularly in poor rural populations (Narasimhan *et al*., 2016). In the current study, dietary fibre was significantly associated with SBP and DBP. The same finding was reported by Moreno Franco *et al*. (2014). An intervention study showed that increased dietary fibre intake significantly reduced both SBP and DBP (Roberts *et al*., 2002). Further association was observed between dietary fibre intake and FBG amongst the current study participants. These findings are consistent with the Giacco *et al* (2000) in that high dietary intake improves the blood glucose level. The beneficial metabolic effects of the dietary fibre intake included both an improvement in the daily blood glucose level and a reduction in the number of hypoglycemic events (Giacco *et al.,* 2000). Polyunsaturated fatty acids (PUFAs) were significantly associated with FBG, HDL-C and SBP. Food rich in PUFA increases insulin sensitivity glucose utilization, decreases insulin resistance and risk of type 2 diabetes (Carpentier *et al*., 2006; Farsi *et al*., 2014). These results show that PUFAs improve metS risk factors. Protein was also associated with SBP in the current study. There is evidence that consumption of high protein has a protective effect against the metS (Wojcik *et al*., 2016). However, this association needs to be understood with caution given that consuming higher than recommended amounts of protein is associated with increased blood pressure and hypertensive diseases (Obarzanek *et al*., 1996), however, in the management for metS, it is still controversial.

*The influence of dietary intake on developing metabolic syndrome*

Table 8 shows that, participants who had high dietary energy intake were significantly less likely to present with larger WC, low HDL-C and high LDL-C (OR: 0.250 95%CI [0.161;0.389], OR: 0.306 95%CI [0.220;0.425] and OR: 0.583 95%CI [0.418;0.812], respectively), but more likely to presents with elevated FBG, high TCHOL, high TG and hypertension (OR: 1.01 95%CI [0.735;1.386], OR: 1.039 95%CI [0.575;1.337], OR: 1.186 95%CI [0.695;2.023], OR: 5.205 95%CI [3.156;8.585], respectively) crude. After adjusting for age, gender, smoking and alcohol status, high energy intake was more likely to increase two times high the large WC and elevated FBG among study participants (OR: 2.766 95%CI [0.863;3.477] and OR: 2.227 95%CI [1.051;3.328], respectively). Furthermore, low dietary fibre intake was nearly four times more likely to increase the low HDL-C, crude. (OR: 3.864 95%CI [1.067;13.988]) crude.

Those participants who consumed high trans fats were more likely to present with high FBG (OR: 1.424 95%CI [0.985; 2.060]), however, these participants were less likely to present with LDL-C (OR: 0.540 95%CI [0.321; 0.906]) for unadjusted. However, after adding potential cofounding factors, participants with high fatty acid were less likely to present with high FBG (OR: 0.672 95%CI [0.441;1.023]). The rest of the dietary factors (protein, carbohydrates, polyunsaturated fats and monounsaturated fatty acids) were not included in the odds ratio model since they could not meet the categorical data standard.

In summary, it seems as though high total dietary energy, high added sugar intake, low fibre, high SFAs and trans fatty acids increased the likelihood of participants presenting with high WC, FBG, TCHOL, HDL-C, TG, LDL-C and hypertension.

Table 8 Binary logistic regression analysis to show dietary predictors of metS risk factors in young adults (18-30 years) of Ellisras

| | crude | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **ENERGY (kj)** | | | **ADDED SUGAR (g)** | | | **FIBRE (g)** | | | **Saturated fatty acids (%)** | | | **Trans fatty acids (%)** | | |
| | **OR** | **95%CI** | **P value** | **OR** | **95%CI** | **P value** | **OR** | **95%CI** | **P value** | **OR** | **95%CI** | **P value** | **OR** | **95%CI** | **P value** |
| **WC male≥102 cm, female≥88 cm** | 0.250 | (0.161;0.389) | <0.001 | 1.005 | (0.669;1.509) | 0.982 | 0.741 | (0.164;3.358) | 0.741 | 1.052 | (0.669;1.654) | 0.826 | 0.930 | (0.574;1.506) | 0.767 |
| **FBG ≥5.6 mmol/L** | 1.010 | (0.735;1.386) | 0.053 | 0.701 | (0.512;.964) | 0.029 | 0.638 | (0.211;1.925) | 0.425 | 1.076 | (0.756;1.532) | 0.683 | 1.424 | (0.985;2.060) | 0.052 |
| **TCHOL ≥5.1 mmol/L** | 1.039 | (0.575;1.337) | 0.009 | 1.088 | (0.714;1.659) | 0.693 | 0.830 | (0.183;3.765) | 0.809 | 0.870 | (0.537;1.412) | 0.574 | 1.215 | (0.753;1.961) | 0.425 |
| **HDL-C Men<1 mmol/L, Female<1.2 mmol/L** | 0.306 | (0.220;0.425) | <.001 | 0.686 | (0.500;.941) | 0.019 | 3.864 | (1.067;13.988) | 0.039 | 0.881 | (0.619;1.254) | 0.482 | 0.739 | (0.510;1.070) | 0.109 |
| **TG ≥1.7 mmol/L** | 1.186 | (0.695;2.023) | 0.0531 | 0.846 | (0.499;1.436) | 0.536 | 1.528 | (0.334;6.989) | 0.585 | 0.838 | (0.455;1.542) | 0.570 | 0.663 | (0.336;1.307) | 0.235 |
| **LDL-C >3 mmol/L** | 0.583 | (0.418;0.812) | 0.001 | 0.866 | (0.623;1.206) | 0.395 | 0.740 | (0.229;2.388) | 0.615 | 1.176 | (0.816;1.696) | 0.386 | 0.540 | (0.321;0.906) | 0.020 |
| **Hypertension ≥130/≥85 mmHg** | 5.205 | (3.156;8.585) | <0.001 | 1.2424 | (0.840;1.836) | 0.278 | 0.970 | (0.941;1.001) | 0.054 | 0.716 | (0.451;1.138) | 0.158 | 1.255 | (0.858;1.835) | 0.242 |
| | **Adjusted (age, gender, smoking and alcohol status)** | | | | | | | | | | | | | | |
| **WC male≥102 cm, female≥88 cm** | 2.766 | (0.863;3.477) | 0.022 | 1.014 | (0.614;1.675) | 0.957 | 0.401 | (0.084;1.903) | 0.250 | 0.919 | (0.508;1.664) | 0.780 | 1.143 | (0.618;2.115) | 0.669 |
| **FBG ≥5.6 mmol/L** | 2.227 | (1.051;3.328) | 0.033 | 0.706 | (0.504;0.988) | 0.042 | 0.641 | (0.208;1.976) | 0.439 | 1.027 | (0.689;1.530) | 0.897 | 0.672 | (0.441;1.023) | 0.053 |
| **TCHOL ≥5.1 mmol/L** | 1.145 | (0.556;2.358) | 0.714 | 1.200 | (0.756;1.903) | 0.440 | 0.803 | (0.171;3.769) | 0.781 | 1.394 | (0.788;2.467) | 0.254 | 0.680 | (0.385;1.203) | 0.185 |
| **HDL-C Men<1 mmol/L, Female<1.2 mmol/L** | 1.000 | (0.988;1.000) | 0.003 | 1.008 | (1.003;1.013) | 0.002 | 1.046 | (1.015;1.157) | 0.004 | 0.993 | (0.966;1.020) | 0.601 | 1.022 | (0.948;1.103) | 0.568 |
| **TG ≥1.7 mmol/L)** | 0.826 | (0.316;2.163) | 0.698 | 0.772 | (0.441;1.351) | 0.365 | 1.681 | (0.357;7.929) | 0.511 | 1.127 | (0.575;2.211) | 0.727 | 1.405 | (0.666;2.964) | 0.372 |
| **LDL-C >3 mmol/L** | 1.191 | (0.661;2.145) | 0.501 | 0.963 | (0.670;1.384) | 0.863 | 0.638 | (0.192;2.116) | 0.462 | 0.914 | (0.596;1.402) | 0.680 | 0.870 | (0.555;1.363) | 0.543 |
| **Hypertension ≥130/≥85 mmHg** | 1.376 | (0.618;3.065) | 0.434 | 0.950 | (0.615;1.468) | 0.818 | 0.985 | (0.953;1.017) | 0.350 | 1.131 | (0.661;1.936) | 0.653 | 1.505 | (0.824;2.748) | 0.183 |

*WC=increased waist circumference; FPG=elevated fasting blood glucose; TCHOL=high total cholesterol; HDL=low high density lipoprotein; LDL=high low density lipoprotein; TG=elevated triglycerides; SBP=increased systolic blood pressure; DBP=increased diastolic blood pressure; OR=odds ratio*

High energy intake was less likely to shown large WC, low HDL-C and LDL-C amongst the participants. Similar results were reported by Bruscato *et al* (2010). These results are possibly because the participants consumed less energy than recommended. These results have a policy implication in that they call upon urgent interventions including nutrition education in rural and poorer communities of South Africa in order to halt the escalating MetS epidemic as shown by a number of studies in the country (Mayosi *et al*., 2009; Van Zyl *et al*., 2012; Omuse *et al*., 2017). This outlines the problem the South African population is facing. Therefore, the longitudinal investigation of this syndrome is needed.

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# CHAPTER 5

## 5. SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

## 5.1. INTRODUCTION

Globally the proportion of non-communicable diseases (NCDs) deaths is predicted to rise from 59% in 2002 to 69% of all deaths by 2030 (Mathers and Loncar, 2006). The NCDs profile has been suggested to be changing rapidly globally. For example, the growing patterns of elderly, economic growth and rapid urbanization which is characterized by a shift in the dietary patterns and a lack of physical activity resulted in the increase in the NCDs morbidity and mortality worldwide (Monyeki and Kemper, 2008; Miranda *et al*., 2009; Collaboration, 2017). Furthermore, the interconnected risk factors as a result of lifestyle; such as elevated blood pressure (BP), waist circumference (WC), blood sugar and low high density lipoprotein cholesterol (HDL-C) are amongst the leading risk factors for NCDs in Africa

## 5.2. SUMMARY

Chapter 1–Non-communicable diseases (NCDs) are responsible for two out of three deaths worldwide with their profile changing from one country to another (Rahim *et al*., 2014). Africa is expected to experience the largest increase in NCD related mortality globally with about 46% of all mortality expected to be attributed to NCDs by 2030 (Rahim *et al*., 2014; Dalal *et al*., 2011). Metabolic syndrome (metS) has been considered the fastest developing NCDs syndrome worldwide (Ford, 2005). This chapter addressed the need to determine the lifestyle risk factors associated with metabolic syndrome among the Ellisras rural sample aged 18-30 years.

The objectives of this study among rural population (6-30 years) who are part of the ELS were:

i. To determine the performance of blood pressure to height ratio as a screening tool for elevated blood pressure in rural children
ii. To determine the prevalence of risk factors for metS (TCHOL, HDL-C, LDL-C, Hypertension, TG, WC and FBG levels)
iii. To determine the association between dietary intake and metS risk factors
iv. To determine the risk of developing metS

Chapter 2–The literature of some important international researchers who argued that diagnosing hypertension in children and adolescents is complicated due to

variation of blood pressure values to age, gender and height, therefore, blood pressure to height ratio can be an accurate tool for screening elevated blood pressure (Hansen *et al*., 2007; Xi *et al*., 2014). Metabolic syndrome (metS) has been considered to be one of the fastest developing NCDs entities in the world (Ford *et al*., 2004; Mottillo *et al*., 2010). Metabolic syndrome is characterized by a group of risk factors that co-exist in an individual. Therefore, metS shares similar risk factors to that of NCDs, such as elevated blood pressure, glucose intolerance and insulin resistance. These risk factors have been associated with obesity, thereby suggesting the interrelation between NCDs and metS (McKeigue *et al*., 1998). The argument that metS is also pandemic in South African population is reviewed (Motala *et al*., 2011; Van Zyl *et al*., 2012).

Chapter 3–The methodology and the statistical analysis of the data collected were described. Receiver-operating characteristics curve analysis was performed to assess the performance of blood pressure to height ratio as an accurate tool for screening elevated blood pressure. Linear regression was used to investigate the association of dietary intake with various metS risk factors. Finally, logistic regression was used to show the risk of developing risk factors of metS due to dietary intake.

Chapter 4–The results and discussion of the study were described. The discriminatory ability of optimal cut-offs of systolic blood pressure to height ratio and diastolic blood pressure to height ratio for identifying stage 1 and stage 2 hypertension were satisfactory. The cut-offs points seem to be better at predicting stage 2 hypertension of which the majority of children have been screened in the current study and consistent with the findings of other research (Lu *et al*., 2011). Furthermore, the overall prevalence of metabolic syndrome was 23.1% (8.6% males and 36.8% females) in young adults from rural Ellisras. This prevalence is lower than the prevalence shown in South African coloured participants in the Erasmus *et al*. (2012) study, but is higher than the prevalence shown in black North West residents (9.5% for females and 6.8% for males) participating in the Hoebel *et al*. (2011) study. Additionally, the current population had a higher metS prevalence than that reported in low-income black South Africans (Owolabi *et al*., 2018). However, the males in Owolabi's study had a higher prevalence than that of the current study population, even though both studies used the IDF criteria definition for metS.

Chapter 5–A summary overview of the dissertation was outlined together with recommendations of the study which will help to uproot the dynamics of metabolic syndrome among the young adult population in rural South Africa.

## 5.3. CONCLUSIONS

The conclusions of the study are provided in relation to the objectives and hypothesis set out in Chapter 1.

**Objective 1:** To determine the performance of blood pressure to height ratio as a screening tool for elevated blood pressure in rural children.

**Hypothesis 1: The performance of blood pressure to height ratio will be an accurate tool for screening elevated blood pressure in rural Ellisras children**

Xi *et al*. (2014) initially reported blood pressure to height ratio as a simple and accurate index for the screening of hypertension in 11 661 Chinese children aged 6-17 years. The optimal thresholds of blood pressure to height ratio for screening hypertension were established by these authors. The optimal thresholds for prehypertension were 0.81 in children and 0.70 in adolescents for systolic blood pressure to height ratio, while diastolic blood pressure to height ratio was 0.52 in children and 0.46 in adolescents, and were similar to the results of Ellisras children and adolescents aged 6-17 years.

The blood pressure to height ratio is an accurate tool for screening elevated blood pressure in Ellisras children aged 6-17 years. This can well help to prevent the misclassification of children and adolescent hypertension. Furthermore, this tool can be used to screen children before the development of prehypertension and hypertension. Moreover, it can be used to manage hypertension in Ellisras children, ultimately reducing the risks of developing hypertension and associated cardiovascular disease in adulthood. However, it can be used as a screening tool but should not be used as a diagnostic tool.

**In the line of the findings above, hypothesis 1 was therefore partially accepted.**

**Objective 2:** To determine the prevalence of risk factors for metS (TCHOL, HDL-C, LDL-C, hypertension, TG, WC and FBG levels).

**Hypothesis 2: The prevalence of risk factors of metabolic syndrome of Ellisras sample will be similar to those studies in other parts of the world.**

Globally, the prevalence of the metS is on the rise, and this increase is the case in developing countries, in Africa and in SA. Based on current population estimates, nearly 100 million people have metS (Roberts *et al.*, 2013). It was recently reported that, the prevalence of metS is higher in African populations than in Caucasian populations (Hoebel *at al*., 2011). These findings were supported by the African based studies which reported highest prevalence of metS in Africa (Erasmus *et al*., 2012; Peer *et al*., 2015). As compared to other areas of the world (Siminialayi and Emem-chioma, 2008; Awosan *et al*., 2013; Magalhães *et al*., 2014). As expected, the prevalence of metS in the current study was 23.1% (8.6% males and 36.8% females).

**In the line of the findings above, hypothesis 2 was therefore partially accepted.**

**Objective 3:** To determine the association between dietary intake and metS risk factors.

**Hypothesis 3: There will be a significant association between dietary intake and metabolic syndrome risk factors**

The majority of the rural population still consume less than the recommended dietary intake for both macro and micronutrients (Mchiza *et al*., 2015). Consumption of excessive food energy has been shown in other studies conducted in black ethnic communities and other rural areas in South Africa. Briefly, the total mean energy intake males (9 788 kj) and females (7 250 kj) and dietary fibre males (22%) and females (18%) of South Africans appears to lie below the DRIs (Nel and Steyn, 2002). Jaffer *et al*. (2009), reported the same trend with energy intake for males (8 600 kj) and females (7 600 kj) and dietary fibre males (18.9%) and females (16.2%). Moreover, the total mean energy in males (6 973 kj) and females (6 107 kj) and dietary fibre in males (19%) and females (17%) were observed from rural settings (Wentzel-Viljoen and Kruger, 2010). As evidenced in the current study, dietary intake of some macronutrients were associated with metS risk factors, whereas others were not, example; there was no association between log total energy, log added sugar, log SFA and log MUFA with metabolic risk factors. However, a low and negative

significant association between log fibre with SBP and DBP, crude was observed. The same finding was reported by Moreno Franco *et al*. (2014). An intervention study shown that increased dietary fibre intake significantly reduced both SBP and DBP (Roberts *et al*., 2002). After adjusting for the potential confounding factors, log fibre was also associated with FBG. These results were consistent with the results of Giacco *et al* (2000). Furthermore, log PUFAs was inversely associated with FBG, HDL-C and SBP crude. Log trans fatty acids was inversely associated with WC, HDL-C and SBP crude. Finally, log protein was inversely associated with SBP both crude and adjusted for potential cofounding factors.

**In the line of the findings above, hypothesis 3 was therefore partially accepted.**

**Objective 4:** To determine the risk of developing metS

**Hypothesis 4: The Ellisras population will be at risk of developing metabolic syndrome similar to those studies in the world.**

Participants who had high dietary energy intake were significantly less likely to present with larger WC, low HDL-C and high LDL-C, respectively). These results could possibly be due to the finding that participants consumed less energy than recommended. However, participants who had a high dietary energy intake were more likely to present with elevated FBG, high TCHOL, high TG and hypertension. Similar results were reported by Bruscato *et al*. (2010). After adjusting for age, gender, smoking and alcohol status, high energy intake was more than two times more likely to predict metS in adults with a large WC and elevated FBG among study participants. Furthermore, low dietary fibre intake was nearly four times more likely to increase the low HDL-C, crude.

Those participants who consumed high trans fats were more likely to present with high FBG, but less likely to present with high LDL-C, crude. However, after adding potential confounding factors, participants with high fatty acid were less likely to present with high FBG. These results have a policy implication in that they call for urgent interventions including nutrition education in rural and poorer communities of SA in order to halt this escalating metS epidemic as shown by a number of studies in the country (Mayosi *et al*., 2009; Van Zyl *et al*., 2012; Omuse *et al*., 2017).

**In the line with the findings above, hypothesis 4 was therefore partially accepted.**

## 5.4. RECOMMENDATIONS

Metabolic syndrome risk factors are largely preventable. However, researchers need to find the mechanism that links diet to health.

We recommend that:

1. Blood pressure to height ratio is used as a screening tool for identifying children and adolescents with elevated blood pressure. This can help to prevent the misclassification of children and adolescent hypertension. Furthermore, this tool can be used to screen children before the development of prehypertension and hypertension.
2. Further studies with a larger sample that is conveniently representative are needed in order to draw a solid conclusion on the findings
3. Other risk factors of metabolic syndrome such as tobacco smoking, alcohol consumption, physical activity and socioeconomic status should be included in the analysis. More cohort studies are needed to track the development of metabolic syndrome over time in rural communities in order to devise strategic interventions and management plans to prevent the metabolic syndrome in the rural population.
4. Diet plays a significant role as a risk factor for NCDs, therefore, sufficient and safe food from a variety of food supplies are recommended in the rural communities

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# APPENDIXES

Faculty of Science and Agriculture

APPENDIX A (DATA FORM)

Biochemical measurements and life style

| | | | |
|---|---|---|---|
| Subject Name: | | | |
| Subject Number: | | | |
| Code of the Fieldworker: ……. | | | |
| Examination Date: | | | |
| Name of School/village:……. | | | |
| School number/village number: …… | | | |
| Last Grade at school …….. | | | |
| Gender: | Male 1 | Female 2 | |
| Date of birth: | | | |

| | | | | |
|---|---|---|---|---|
| **Fasting status** | | | | |
| **Have you had anything to eat or drink in the last 12 hours? (other than water or unsweetened black tea or coffee)** | | | | |
| | yes=1 | no =2 | Uncertain=3 | |
| **Blood glucose** | | | | |
| **Time of blood specimen taken ____/____ hh:mm** | | | | |
| **Fasting blood glucose (mmol/l)** | | | | |
| **History of Diabetes (Expanded)** | | | | |
| *B 3. Have you had your blood sugar measured in the last 12 months?* | | | | |

| yes=1 | no =2 | Uncertain=3 | |
|---|---|---|---|

*B 4. Have you ever been told by a doctor or other health worker that you have diabetes?*

| yes=1 | no =2 | Uncertain=3 | |
|---|---|---|---|

*B 5. Are you currently receiving any of the following treatment for diabetes?*

| | | | | |
|---|---|---|---|---|
| *Insulin* | *yes 1* | *no 2* | *uncertain 3* | |
| *Oral drug prescribed by a doctor or other health worker*[1] | *yes 1* | *no 2* | *uncertain 3* | |
| *Special diet prescribed by a doctor or other health worker* | *yes 1* | *no 2* | *uncertain 3* | |
| *Advice or treatment to lose weight* | *yes 1* | *no 2* | *uncertain 3* | |
| *Advice or treatment to stop smoking* | *yes 1* | *no 2* | *uncertain 3* | |
| *Herbal or traditional remedy* | *yes 1* | *no 2* | *uncertain 3* | |

**Blood lipids**

Total cholesterol (mmol/l)

Triglycerides (mmol/l)

HDL cholesterol (mmol/l)

LDL cholesterol (calculated) (mmol/l)

*M 9. Have you been told by a doctor or other health worker in the past year (12 months) that you have elevated blood pressure or hypertension?*

| yes=1 | no =2 | Uncertain=3 | |
|---|---|---|---|

*M 10. Are you currently receiving any of the following treatments for high blood pressure?*

| *Drug(s) prescribed by a doctor or other health worker*[2] | yes=1 | no =2 | |
|---|---|---|---|

| | | | | |
|---|---|---|---|---|
| *Special diet prescribed by a doctor or other health worker* | | | | |
| *Advice or treatment to lose weight* | | | | |
| *Advice or treatment to stop smoking* | | | | |
| *Herbal or traditional remedy* | | | | **61** |

**Life style (Tobacco smoking and alcohol consumption status)**

| Yes=1 | No=2 | *Uncertain= 3* | |
|---|---|---|---|

| | *yes=1* | *No=2* | |
|---|---|---|---|
| *Do you smoke tobacco/ cigarette* | | | |
| *Do you drink alcohol* | | | |
| | | | |

Signature of fieldworker:_____ Date:_____
Signature of the Supervisor (name_____
Signature _____Date:_____


Faculty of Science and Agriculture

APPENDIX B (DATA FORM)

Anthropometry and blood pressure data form

DD MM YY DD MM YY

Subject number: | | | | | | | Birth Date: | | | | | | | observedate: | | | | | | |

Gender: M/F

| | | | |
|---|---|---|---|
| **Height (cm)** | | | |
| **Waist circumferences (cm)** | | | |
| **Systolic blood pressure (mmHg)** | | | |
| **Diastolic blood pressure (mmHg)** | | | |

Signature of fieldworker:_____ Date:_____

Signature of the Supervisor (name_____

Signature _____Date:_____

Faculty of Science and Agriculture

APPENDIX C (DATA FORM)
24 hour recall (Dietary intake)

# ELLISRAS LONGITUDINAL STUDY

Subject number: ☐☐☐☐☐ Birth Date: DD MM YY ☐☐☐☐☐☐ Interwdate: DD MM YY ☐☐☐☐☐☐

School name:_____ School number:_____Interviewer_____

**DIETARY INTAKE QUESTIONNAIRES (24 HR RECALL)**

**Instructions:**

Now I want you to tell me everything that you ate and drank yesterday. Lets start with when you woke up. Did you have anything to eat or drink?

- Enter each item eaten in grams under the correct interval of the day eaten.
- Make sure that the code is circled.
- Items not on the questionnaire should be looked up in the Quantity Manual or list of food codes.
- Specify fully when new items are entered and look up the code later.

**ABBREVIATIONS:**

| | | |
|---|---|---|
| Measures<br>1t = 1 rounded teaspoon<br>1T = 1 rounded tablespoon (15ml)<br>1SP = 1 rounded servingspoon (30ml)<br>C = measuring cup (250ml)<br>s/s = small size<br>m/s = medium<br>L/s = large<br>E = enriched<br>P = plain<br><br>Milk:<br>SM = skim milk<br>WN = whole milk<br>BL = blend<br>CON = condensed milk<br>ND = non-dairy | Bread<br>Wh = white<br>Br = Brown<br>Ww = wholewheat<br><br>Meat<br>F = with fat<br>FT = fat trimmed<br><br>Oil/ Fat<br>B = butter<br>HM = hard margarine<br>Med = medium fat/ light<br>PM = polyunsaturated<br>SO = sunflower oil<br>WF = white fat<br>PM = peanut butter | BR = breakfast ( Up to 09h00)<br>IS = in - between snack<br>L = lunch (midday (12h00-14h00)<br>D = dinner (evening (17h00-20h00)<br>AD = after meal<br>Comm = commercial<br>Home = homemade<br>Pot = poatato<br>Cab = cabbage<br>Carr = Carrot<br>Fill = Filling<br>Usually = at least 4x/week |

| | Dairy Fruit Mix - 2791 | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
| | + Sugar White-3989;Brown-4005;Syrup-3988; Honey-3984 | 1t = 6g | | | | | | |
| | Syrup (undiluted)-2865; Guava Syrup-2864 | 1t = 5g | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| BREAKFAST CEREALS | Maltabella: Soft-3241; Mabella: Soft-3437 | ½c = 125g | | | | | | |
| | M/Meal: Soft: Plain-3399; Enrich-4277 | 1c soft = 250g | | | | | | |
| | Stiff: Plain-3400; Enrich-4278 | 1c stiff = 250g | | | | | | |
| | Crumbly: Plain-3401; Enrich-4279 | 1c crumbly = 140g | | | | | | |
| | Sour Porridge: Maize with Vinegar-P0001, Maize Fermented- P0002 Mabella with Vinegar-P0003; Mabella Fermented-P0004 | ½c = 125g<br>1c = 250g | | | | | | |
| | Oats-3239; Tastee Wheat-3240 | ½c = 125g | | | | | | |
| | Corn Flakes-3243; Sugar Frosted-3374 | 1c = 40g | | | | | | |
| | Honey Crunch and Muesli - 3303 | ½c = 65g | | | | | | |
| | Pronutro: Great Start-3438; High Energy-3245; Wholewheat-3436 | ½c = 50g | | | | | | |
| | Puffed Wheat-3325; Sweetened-3376 (Honey Smacks) | ½c = 12g | | | | | | |
| | Raisin Bran-3373; Fruit Loops-3425 | Raisin Bran ½c = 45g<br>Fruit Loops ½c = 18g | | | | | | |
| | Special K-3322; All Bran-3242 | ½c = 25g | | | | | | |
| | Rice Crispies-3252; Cocopops-3372 | ½c = 20g | | | | | | |
| | Weetbix – 3244 | 1 = 25g | | | | | | |
| | + Fat: B -3479; HM-3484; Med-3531; PM-3496; WF-3516 | 1 t PB = 12g;<br>1 t marg/oil = 5g | | | | | | |
| | Ghee-3525; PB-3485; Butro-3523; SO-3507 | | | | | | | |
| | + Sugar White-3989;Brown-4005; Syrup-3988;Honey-3984 | 1 t sugar = 6g<br>1 t honey/syrup = 15g | | | | | | |
| | + Cond Milk:SM-2744; Cond WM-2714;Cond ND- P0042 | 1t = 10g | | | | | | |
| | + Evap WM-2715; Evap SM-2827; Evap Light-P0043 | 1t = 3g | | | | | | |
| | + Non-Dairy Creamer-2751 | 1t = 4g | | | | | | |
| | + WM Powder-2831 | 1t = 4g | | | | | | |
| | + Milk: SM-2719; WM-2718 | 125g – Instant cereal<br>60g – porridge<br>180g – Pro Nutro | | | | | | |
| | BL-2771; 2%-2772 | | | | | | | |
| | Soy-2737; Breast-2741; Goat-2738 | | | | | | | |
| | Formula (Specify):_____<br>No of Scoops/Bottle:_____ | | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| BREAD | Bread: Comm & Home: Wh-3210 | Wh + Br 10mm = 30g<br>Ww 10mm = 35g<br>Wh + Br 20mm = 60g<br>Ww 20mm = 70g | | | | | | |
| | Br-3211 | | | | | | | |
| | Ww-3212 | | | | | | | |
| | Cream Crackers-3230; Provita-3235; Tuc 3331; Crackers Ww-3391 | Cr Cracker = 8g; Tuc = 4g; Provita = 6g | | | | | | |
| | Maize Meal Bread - 3278 | m/s = 30g; L/s = 50g | | | | | | |
| | Muffins: Plain-3408; Bran-3407 | 6cm diam = 35g<br>8cm diam = 60g | | | | | | |

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|---|---|---|---|---|---|---|---|---|
| | Rolls: Wh-3210; Br-3211; Ww-3212 | Wh round (10cm) = 30g<br>Wh long (16cm) = 40g | | | | | | |
| | Roti: SO-3358; HM-3357 | s/s = 50g (Roti) | | | | | | |
| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
| | Rusks: Comm Wh-3364; Bran-3330 | Outspan = 15g; All Bran = 30g | | | | | | |
| | Comm Buttermilk: Wh-3329; | Wh = 35g; Ww = 30g | | | | | | |
| | Home Buttermilk: Wh-3215; Ww-3255; Bran & Raisins-3380 | Wh = 30g; Ww = 30g | | | | | | |
| | Scones: (Wh) SM-3411; WM-3237<br>(Ww) SM-3412; WM-3320 | 6cm diam = 35g<br>8cm diam = 60g | | | | | | |
| | Vetkoek: Wh-3257; Ww –3324; Dumpling-3210 (no yeast) | 8cm diam = 60g | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| SPREADS ON BREAD | Beef Fat-3494; Mutton Fat-3497; Lard-3495 | Thin Med Thick | | | | | | |
| | Butter-3479; Butro-3523 | 5 10 15 | | | | | | |
| | Ghee-3525; WF-3516; | | | | | | | |
| | Fishpaste-3109; Liver Spread-2922; Meat Paste-2917 | 5 7 10 | | | | | | |
| | Jam-3985; Honey-3984; Syrup-3988 | 10 20 35 | | | | | | |
| | Marg: H-3484 | 5 7 10 | | | | | | |
| | Med-3531 | | | | | | | |
| | PM-3496 | | | | | | | |
| | Marmite-4030; Meat Spread (Bovril)-4029 | 2 4 7 | | | | | | |
| | Peanut Butter-3485; Sandwich Spread-3522; ChocSpread-P0005 | 5 10 20 | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| EGGS | Eggs: Boiled/Poached - 2867 | 1 egg = 50g | | | | | | |
| | Curried - 2902 | 1 egg + sauce (IT) = 75g | | | | | | |
| | Fried: B-2868; HM-2877; PM-2878 | 1 egg = 52g | | | | | | |
| | SO-2869; Bacon Fat-2870 | | | | | | | |
| | Scrambled/Omelette: SM + B-2886; SM + HM-2887 | IT = 35g; 1SP = 80g<br>½c = 115g (± 2 eggs)<br>omelette = 60g egg (med)<br>120g (L/s) | | | | | | |
| | SM+PM-2888; SM+SO-2889; WM+B-2874 | | | | | | | |
| | WM+HM-2890; WM+PM-2891; WM+SO-2873 | | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| CHEESE | Cheddar-2722; | grated: med = 10g<br>Thick = 15g<br>1 cheezi = 20g; cubes = 30g<br>1 slice = 8g | | | | | | |
| | Gouda/Sweetmilk-2723 | | | | | | | |
| | Cheese Spread-2730 | med = 12g; thick = 25g | | | | | | |
| | Cottage Cheese; Creamed-2759; Cream Cheese-2725 | thin = 10g med = 20 | | | | | | |
| | Cottage Cheese: Fat Free-2729; Low Fat-2760 | med = 20g; thick = 30g | | | | | | |
| | Macaroni Cheese: SM-3343; WM-3301 | 1T = 45g; 1 SP = 90g;<br>½c = 115g | | | | | | |
| | Pizza (Cheese + Tomato)-3353 | S/s = 90g; L/s = 340g | | | | | | |
| | Savoury Tart+Asparagus-3367;+Vienna-3326;+Tuna-3366 | wedge: small = 65g;<br>med = 75g ; large = 110g | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| MEAT | Bacon: Fried: Lean-2915 F-2906 | 1 rasher = 10g | | | | | | |

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|---|---|---|---|---|---|---|---|---|
| | Beef:Corned/Silverside/Cold cuts:F-2924;Bully Beef-2940 | 138 x 85 x 3 = 20g<br>½c = 100g | | | | | | |
| | Lean-2962; Curry Beef-P0006 | | | | | | | |
| | Fillet: F-2933; FT-2929 | 100 x 70 x 10 = 90g | | | | | | |
| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
| M E | Mince: Pan Fried F-2910; Lean-2961; Curry-3015 | T = 40; SP = 85g<br>½c = 100g | | | | | | |
| | - Savoury (Tomato + Onion)-2987 | | | | | | | |
| | - Cottage Pie: WM + HM-3009 | | | | | | | |
| | Roast: F-2944; FT-2960 | 120 x 60 x 5 = 35g<br>120 x 60 x 10 = 70g | | | | | | |
| | Rump: Fried: F-2908; FT-2959 | S/s 130 x 70 x 15 = 125g<br>L/s 165 x 70 x 30 = 270g | | | | | | |
| | Sirlion/T-Bone: Grilled: F-2946; FT-2907 | | | | | | | |
| | Stew: Vegetables (Fat Meat)-3006 | 1 SP = 105g; ½c = 125g | | | | | | |
| | : Pot + Carrots + Peas + Onions (Lean Meat)-2909 | | | | | | | |
| | Biltong: Beef-2911; Game-2912 | grated 1SP = 10g<br>beefeater = 18g<br>sliced 1SP = 35g | | | | | | |
| | Bobotie: Lean, SM, SO-3013; F, WM, S0-2986 | 1SP = 85g; ½c = 115g | | | | | | |
| | Chicken: Boiled + Skin-2926; No Skin-2963; Curry-P0007 | breast + skin = 125g<br>thigh = 80g<br>drumstick = 42g<br>foot = 30g<br>wing = 30g<br>pie(comm)=150g home = 90g<br>liver = 30g; stomach = 20g | | | | | | |
| | Feet-2997; Giblets-2998; Heads-2999 | | | | | | | |
| | Pie (Comm)-2954 | | | | | | | |
| | Roast + Skin-2925; No Skin-2950; Fried-2925 | | | | | | | |
| | Stew: Vegetables-3005 | 1SP = 90g; ½c = 125g | | | | | | |
| | Tomato + Onion – 2985 | | | | | | | |
| | Batter Dipped-Fried eg. Kentucky-3018 | 1SP = 105g; ½c = 125g | | | | | | |
| | Burger Pattie –2950 | 1 pattie = 80g | | | | | | |
| | + Bun (4 cm diam)-3210 | 1 bun = 60g | | | | | | |
| | Cornish Pie: (Comm) - 2953 | med = 150g | | | | | | |
| | Frankfurter-2937 | 155 x 20 = 45g<br>168 x 21 = 60g | | | | | | |
| | + Roll (16 cm long)-3210 | 1 roll = 40g | | | | | | |
| | Goat meat: Stewed (plain)-4281; (+ Veg)-4282 | 120 x 60 x 5 = 35g<br>120 x 60 x 10 = 70g | | | | | | |
| | Fried F-P0008; Fried FT-P0009 | | | | | | | |
| | Grilled F-P0010; Grilled FT-P0011 | | | | | | | |
| | Ham-2967; Ham & Tongue loaf-2990 | med slice = 25g | | | | | | |
| | Heart: Beef-2968; Sheep-2969 | sheep heart = 60g<br>sheep kidney = 30g<br>beef kidney = 85g | | | | | | |
| | Kidney: Beef-2923; Sheep-2956 | | | | | | | |
| | Lung: Beef-3019 | | | | | | | |
| | Lasagne: SM-3440; WM-3261 | T = 40g; SP = 75g; ½c = 120g | | | | | | |
| | Liver: Fried : Beef-2920; Sheep-2955; Patty (Fried) -2971 | sheep = 55g<br>chicken = 30g<br>beef = 80g | | | | | | |
| | Cooked: Chicken-2970 | | | | | | | |
| | Meat Ball: F + Egg-2965; F-No Egg-2966 | 50mm = 60; 75mm = 120g | | | | | | |
| | Lean + Egg-3033; Lean, No Egg-3034 | | | | | | | |
| | Meat Loaf: F-3035; Lean-3002 | 80 x 85 x 15mm slice = 80g | | | | | | |

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|---|---|---|---|---|---|---|---|---|
| | Meat Patty: (Hamburger)-2984 | s/s = 50g; m/s = 100g | | | | | | |
| | + Bun (4 cm diam)-3210 | 1 bun = 60g | | | | | | |
| | Mutton: Chop (grilled) F-2927; FT-2934 | loin chop = 60g<br>rib chop = 40g | | | | | | |
| | Roast: F-2947; FT-2973 | s/s slice = 30g<br>med = 70g | | | | | | |
| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
| MEAT | Stew: Plain-2974; Irish-2916 (Vegetables) Curry-3039; Greenbean-3040 | 1SP = 105g; ½c = 125g | | | | | | |
| | Offal: Cooked-Tripe(Pens&Pootjies)-2951;Vetderm-P0023<br>(Specify): _____ | 1SP = 105g; ½c = 125g | | | | | | |
| | Oxtail: Stewed-2976 | | | | | | | |
| | Polony-2919 | slice 5mm thick = 8g<br>comm slice = 16g | | | | | | |
| | Pork: Chop (Grilled) F-2930; FT-2977 | chop: 115 x 80 x 20 = 100g<br>schnitzel: 115 x 80 x 20 =110g<br>roast: 110 x 65 x 5 = 30g<br>1SP = 105g; ½c = 125g<br>3 ribs = 130g | | | | | | |
| | Crumbed-2992; Spareribs-3010 | | | | | | | |
| | Rib, Braised: F-3046; FT-3045 | | | | | | | |
| | Roast: F-2958; FT-2978 | | | | | | | |
| | Salami and Russians-2948 | slice 5mm thick = 12g<br>1 Russian = 50g | | | | | | |
| | + Roll-3210 | 1 roll = 40g | | | | | | |
| | Samoosa: with Veg-3414; Meat-3355 | s/s = 42g | | | | | | |
| | Sausage: Beef: Dry-2949; Cooked-2931 (Boerewors) | thin x 200mm = 45g<br>thick x 165mm = 90g | | | | | | |
| | + Roll-3210 | 1 roll = 40g | | | | | | |
| | Pork: Cooked-2932 | med = 55g | | | | | | |
| | + Roll-3210 | 1 roll = 40g | | | | | | |
| | Roll/Meat Pie (Comm)-2939 | 25mm pie = 120g<br>roll x 135mm = 165g | | | | | | |
| | Spaghetti Bolognaise: Lean-3388; F-3260 | T=40g; SP = 75g; ½c = 100g | | | | | | |
| | Steak & Kidney: Pie-2957; Stew-2979 | comm pie = 120g (30mm)<br>1SP = 100g; ½c = 135g | | | | | | |
| | Tongue: Ox-2935; Sheep:2980 | slice 75 x 45 x 10 = 40g | | | | | | |
| | Toppers/Imana: Cooked-3196 | SP = 85g; ½c = 120g | | | | | | |
| | Veal: Cutlet (Fried): Plain-3049; Crumbed-2983 | 1 chop = 90g | | | | | | |
| | Vienna Sausage/Canned Sausage-2936 | 100mm = 30g; 150mm = 40g | | | | | | |
| | + Roll-3210 | 1 roll = 40g | | | | | | |
| | Worms/Insects:Mopani,Dried-4250;Mopani,Canned-4284;<br>Specify: _____ | | | | | | | |
| | Wild Birds, Animals; Specify: _____ | | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| FISH | Bokkems (Dry Fish)-3097 | 1 s/s = 25g (120mm)<br>L/s = 40g (135mm) | | | | | | |
| | Fatty Fish: Kipper; Galjoen; Snoek; Shad: Fried (SO)-3084; Batter-3094; Grill-3082 | small 50 x 55 x 30 = 60g<br>med 100 x 55 x 30 = 120g<br>stew 1 SP = 95; ½c = 140g | | | | | | |
| | Salted-3097; Steam-3103; Smoked-3112 | | | | | | | |
| | Stew-3076 (Tomato and Onion) / Pickled / Curried | | | | | | | |

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|---|---|---|---|---|---|---|---|---|
| | Fish Cakes: (Fried): Home-3098; Comm-3080 | 65 x 15mm = 50g | | | | | | |
| | Fish Fingers: (Fried)-3081 | 85mm = 35g | | | | | | |
| | Haddock: Smoked (Boiled)-3061 | 70 x 70 x 15 = 65g | | | | | | |
| | Mackerel Canned-3113 | 1 = 80g (15 mm) | | | | | | |
| | Pilchards: Tomato Sauce-3102; Brine-3055 | 1 = 75g | | | | | | |
| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
| | Sardines: + Sauce-3087; + Oil-3104 | s/s = 7g; L/s = 25g | | | | | | |
| | Smoorsnoek-3074 | 1SP = 55g; ½c = 80g | | | | | | |
| | Sole: Fried-3090; Grilled-3073 | baby sole: 180mm = 70g | | | | | | |
| | Tuna: Oil Pack-3093; Tuna: Water-3054; Salmon-3058 | ¼c = 50g | | | | | | |
| | White Fish: Hake, Haddock, Kingklip; Cod : Stew-3076 (Tom + On); Baked+Fat-3092; No Fat-3089 | s/s piece 50 x 55 x 30 = 60g<br>med 100 x 55 x 30 = 120g<br>stew 1 SP = 95g; ½c = 140g | | | | | | |
| | : Grilled-3079; Batter-3072; Fried-3060 | | | | | | | |
| | Other: eg Fresh Water Fish; Specify:_____ P0012 | | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| STARCH | M/Meal: Soft: Plain-3399; Enrich-4277 | T SP ½c | | | | | | |
| | Stiff: Plain-3400; Enrich-4278 | stiff 75 120 125 | | | | | | |
| | Crumbly: Plain-3401; Enrich-4279 | crum 30 75 70 | | | | | | |
| | Mabella Cornrice/Sorghum cooked (soft or stiff)-3437 | soft 75 120 125 | | | | | | |
| | Sour Porridge: Maize & Vinegar-P0001, Fermented-P0002 | | | | | | | |
| | Mabella with Vinegar-P0003; Fermented-P0004 | | | | | | | |
| | Maize Rice (Mealie Rice)-3250 | 25 45 65 | | | | | | |
| | Samp: (Cooked) –3250; Fresh Mealies-3725 | 55 125 125 | | | | | | |
| | Rice: Wh-3247; Br-3315 | 25 60 65 | | | | | | |
| | Spaghetti/Macaroni: (Cooked)-3262 | 35 70 90 | | | | | | |
| | Spaghetti + Tomato Sauce -3258 | 45 80 125 | | | | | | |
| | Stamped Wheat/Wheat Rice-3249 | 30 80 80 | | | | | | |
| | + Fat: B -3479; HM-3484; Med-3531; PM-3496; WF-3516 Ghee-3525; PB-3485; Butro-3523; SO-3507 | 1 t PB = 12g;<br>1 t marg/oil = 5g | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| LEGUMES | Baked Beans-3176 | T SP ½ c<br>50 105 135 | | | | | | |
| | Beans: (Cooked) Haricot-3185; Sugar-3205; Kidney-3183 | 50 85 135 | | | | | | |
| | Breyani: Rice + Lentils + Ghee-3194; +SO-3193 | 40 80 85 | | | | | | |
| | Lentils: Cooked/curried-3179 | 40 80 90 | | | | | | |
| | Samp and Beans (1:1)-3402; Comm-P0045 (No fat added) | 50 125 125 | | | | | | |
| | Samp & Peanuts (80:20) P0013 | | | | | | | |
| | Soup: Comm (Packets)-3165 | 125 | | | | | | |
| | Split Pea-3157; Lentil-3153; Beef + Veg-3159; Bean-3145 | 35 80 130 | | | | | | |
| | Sousboontjies (Dried Bean Salad)-3174 | 40 105 135 | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Stew: Bean + Potato + Onion-3178 | | | | | | 60 | 120 | 125 | | | | | | | | |
| | Other (Specify) _____ | | | | | | | | | | | | | | | | |
| COOKED VEGETABLES | | Boil | Fat Added (or Fried) | | | | | | | | | | | | | | |
| | | NF | B | HM | PM | SO | T | SP | ½C | | | | | | | | |
| | Gr Beans | 3696 | | 3788 | 3789 | | 25 | 60 | 60 | | | | | | | | |
| | Gr Bean Curry | 3791 | | | | | 40 | 75 | 120 | | | | | | | | |
| | GrBean+Pot+Onion | | | 3792 | | 3794 | | | | | | | | | | | |
| COOK | FOOD ITEMS | | | | | | QUANTITY (g/ml) | | | BR | IS | L | IS | D | AD | | |
| | Beetroot + Sugar | 3699 | | | | | 40 | 70 | 80 | | | | | | | | |
| | - No Sugar | 3698 | | | | | | | | | | | | | | | |
| | Brinjal | 3700 | | 3800 | | 3802 | 1 slice = 20g (70mm) + batter = 30g | | | | | | | | | | |
| | - Fried + Egg | | | | | 3803 | | | | | | | | | | | |
| | - + Tomato + Onion | | | 3796 | | 3798 | 50 | 100 | 130 | | | | | | | | |
| | Brocolli | 3701 | | 3805 | | | 25 | 60 | 75 | | | | | | | | |
| | Brussels Sprouts | 3703 | | 3808 | | | 50 | | | | | | | | | | |
| | | Boil | Fat Added (or Fried) | | | | | | | | | | | | | | |
| | | NF | B | HM | PM | SO | T | SP | ½C | | | | | | | | |
| | Cabbage | 3756 | | 3810 | | 3812 | 30 | 55 | 80 | | | | | | | | |
| | Cab + Pot + Onion | | | 3813 | | 3815 | 35 | 75 | 80 | | | | | | | | |
| | Carrots | 3757 | | 3816 | 3817 | | 20 | 50 | 80 | | | | | | | | |
| | Car + Pot + Onion | | | 3822 | | 3824 | 35 | 70 | 105 | | | | | | | | |
| | Carrot + Sugar | 3818 | | 3819 | 3820 | | 25 | 50 | 85 | | | | | | | | |
| | Cauliflower | 3716 | | 3825 | 3826 | | 40 | 65 | 80 | | | | | | | | |
| | Caul + Cheese Sauce | 3715 | | | | | 43 | 70 | 90 | | | | | | | | |
| | Marogo/imifino* Amaranth leaves | 3980 | | | | | 40 | 105 | 90 | | | | | | | | |
| | Marog + Peanuts Ratio: 80:20 | P0014 | | | | | 55 | 120 | 105 | | | | | | | | |
| | Mealies (corn) | 3725 | | | | | 30 | 60 | 95 | | | | | | | | |
| | Sweetcorn | 3726 | | | | | 55 | 125 | 135 | | | | | | | | |
| | Canned Whole Kernel | 3942 | | | | | 55 | 125 | 135 | | | | | | | | |
| | Mix Veg (Froz) | 3727 | | 3835 | 3836 | 4269 | 35 | 75 | 75 | | | | | | | | |
| | Mushroom (Sliced) | 3729 | | 3839 | | 3841 | 30 | 65 | 80 | | | | | | | | |
| | Mushroom, Raw | | | | | 3842 | 30 | 65 | 80 | | | | | | | | |
| | Onions (Sliced) | 3773 | | 3844 | | 3730 | 50 | | | | | | | | | | |
| | Onion + Batter | | | | | 3846 | rings: med = 40g | | | | | | | | | | |
| | Peas | 3719 | | 3856 | | | 30 | 65 | 85 | | | | | | | | |
| | Peas, Frozen | 4146 | | | | | 30 | 65 | 85 | | | | | | | | |
| | Peas + Sugar | 3720 | | 3859 | | | 30 | 65 | 85 | | | | | | | | |
| | Potato: + Skin | 4155 | | | | | s/s = 60; m/s = 90g | | | | | | | | | | |
| | : Baked + Skin | 3736 | | | | | s/s = 60g; m/s = 90g | | | | | | | | | | |
| | : Chips | | | | | 3740 | ½c = 50g; med = 80g | | | | | | | | | | |

8

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| : Peeled | 3737 | | 3867 | 3868 | | s/s = 60g; m/s = 90g; (90 x 60 x 40) | | | | | | | | |
| : Sauté | | | 3871 | | 3873 | 3 | 50 | 90 | | | | | | |
| Potato Cake | | | | | 3915 | 1 med = 40g (75 x 30) | | | | | | | | |
| Potato Mash (SM) | | | | 3875 | | 50 | 115 | 125 | | | | | | |
| Potato Mash (WM) | | | 3876 | | | | | | | | | | | |
| Potato (Roast): Beef Fat-3878; Chicken-3923; Lamb-3736; Pork-3956 | | | | | | 1 med = 90g | | | | | | | | |
| * If indigenous, specify local name: _____ | | | | | | | | | | | | | | |

| | FOOD ITEMS | | | | | | QUANTITY (g/ml) | | | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Boil | Fat Added (or Fried) | | | | | | | | | | | | |
| | | NF | B | HM | PM | SO | T | SP | ½c | | | | | | |
| COOKED VEGETABLES | Pumpkin (Yellow) | 4164 | | | | | 45 | 85 | 105 | | | | | | |
| | Butternut | 3759 | | | | | | | | | | | | | |
| | Pump + Sugar | 3728 | | 3893 | | | | | | | | | | | |
| | Pump Fritter | | | | | 3784 | 75 x 50 x 9 = 25g | | | | | | | | |
| | Spinach | 3913 | | 3898 | 3899 | | 40 | 105 | 90 | | | | | | |
| | Spinach + Peanuts Ratio: 80:20 | P0015 | | | | | 55 | 120 | 105 | | | | | | |
| | Spin + Pot + Onion | | | 3901 | | 3786 | 50 | 105 | 110 | | | | | | |
| | Squash –Gem | 3760 | | | | | ½ gem = 45g<br>1 SP marrow = 85g | | | | | | | | |
| | Gem Squash + Sugar | 3754 | | | | | | | | | | | | | |
| | Squash –Marrow | 4179 | | | | | | | | | | | | | |
| | Marrow + Sugar | | | 3885 | | | | | | | | | | | |
| | Sw Potato: without skin | 3903 | | | | | 50 | 110 | 145 | | | | | | |
| | Sw Potato with Skin | 3748 | | | | | | | | | | | | | |
| | Sw Pot + Sugar | | | 3749 | | | | | | | | | | | |
| | Tomato + Onion | 3925 | | | | | 35 | 75 | 140 | | | | | | |
| | Tom + Onion +Sugar | 3910 | | | | | | | | | | | | | |
| | Tomato | | | 3908 | | 3767 | 1 slice 5mm = 15g (thin); med = 25g | | | | | | | | |
| | Turnips | 3911 | | | | | 25 | 45 | 90 | | | | | | |
| | Other (Specify) | | | | | | | | | | | | | | |
| | Asparagus-3695 | | | | | | med asparagus = 15g | | | | | | | | |
| | Avocado-3656 | | | | | | ¼ avo (80 x 50mm) = 40g | | | | | | | | |
| | Beetroot (Grated) + Sugar-3699 | | | | | | 1T = 25g; SP = 65g | | | | | | | | |
| | Carrot: (Grated)+ Sugar-3721 | | | | | | 1T = 25g; | | | | | | | | |
| | + Pine + Orange - 3710; + Orange Juice = 3711 | | | | | | 1T = 35g; 1SP = 60g | | | | | | | | |
| | Coleslaw + Mayonnaise-3705 | | | | | | T = 20g; SP = 40g; ½c = 50g | | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|
| | Cucumber Raw/Pickled-3718 | med slice = 10g; thick = 15g | | | | | | | |
| | Lettuce-3723 | 1 med leaf = 30g | | | | | | | |
| | Mixed (Tom + Cucum + Lett) - No Dressing-3921 | 1T = 40g; 1SP = 85g | | | | | | | |
| | Mixed Green - No Dressing-3927 | | | | | | | | |
| | Potato Salad + Mayonnaise (Comm), Egg-3928 | T = 45g; 1SP = 105g; ½c = 120g | | | | | | | |
| | Tomato (Raw)-3750 | med = 120g; slice = 15g | | | | | | | |
| | Other (Specify) _____ | | | | | | | | |

| | FOOD ITEMS | | | | | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| DRESSINGS | French Dressing-3487 | | | | | 1t = 5g; 1T = 15g | | | | | | |
| | Mayonnaise: Home-3506; Comm-3488; Low Fat- 3489 | | | | | 1t = 10g 1T = 40g | | | | | | |
| | Oil: Olive-3509; Sunflower-3507; Canola-4280 | | | | | 1t = 5g; 1T = 15g | | | | | | |
| | Salad Dressing: Cooked-3503; Low-Oil-3505 | | | | | | | | | | | |
| FRUIT | | Canned + Sugar | Raw | Dry | Stewed | | | | | | | |
| | Apple | 3599 | 3532 | 3600 | 3603 | 1T = 60g; ½c = 120g; 1 med = 160g (52 x 66) | | | | | | |
| | Apricot | 3535 | 3534 | 3536 | 3537 | 1 med = 35g | | | | | | |
| | Banana | | 3540 | | | 1 med = 75g | | | | | | |
| | Dates | | 3543 | | | 1 med = 10g | | | | | | |
| | Figs | | 3544 | 3557 | | 1 med = 40g (45 x 44) 1 dry = 20g | | | | | | |
| | Fruit Salad | 3580 | 3605 | 3593 | 3590 | ½c = 110g (med) | | | | | | |
| | Granadilla | | 3545 | | | 1 med = 22g | | | | | | |
| | Grape Fruit | 3547 | 3546 | | | ½ med = 125g | | | | | | |
| | Grapes | 3623 | 3550 | | | med bunch = 230g; ½c =90g | | | | | | |
| | Guava | 3553 | 3551 | | | med (6cm) = 95g | | | | | | |
| | Litchi | 3631 | 3632 | | | med (3cm) = 8g | | | | | | |
| | Mango | 3633 | 3556 | | | 135mm = 350g | | | | | | |
| | Naartjie | 3635 | 3558 | | | med = (5cm) = 75g | | | | | | |
| | Orange | | 3560 | | | med (7cm) = 180g | | | | | | |
| | Pawpaw | | 3563 | | | wedge 165 x 26 x 27 = 90g | | | | | | |
| | Peach | 3567 | 3565 | 3568 | 3569 | 1 med = 160g (60 x 65) | | | | | | |
| | Pear | 3583 | 3582 | 3585 | 3586 | 1 med (80 x 65mm) = 165g | | | | | | |
| | Pineapple | 3648 | 3581 | | | 1 slice (85 x 10mm) = 40g | | | | | | |
| | Plum | | 3570 | | | 1 med = 50g (45 x 40) | | | | | | |
| | Prunes | 3676 | 4230 | 3596 | 3564 | 1T = 50g; ½c = 110g; 1 = 12g | | | | | | |
| | Raisins | | 3552 | | | handfull = 27g | | | | | | |
| | Strawberries | 3653 | 3573 | | | 1 med = 12g; ½c = 80g | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sweetmelon, Green | | 3575 | | | 1 wedge (145 x 31 x 20mm) = 60g; ¼ = 110g | | | | | | |
| | Sweetmelon, Yellow | | 3541 | | | | | | | | | |
| | Watermelon | | 3576 | | | slice (330 x 70mm) = 220g | | | | | | |
| | Wild Fruit, Berries: Specify: _____ | | | | | | | | | | | |
| | Other Fruit: | | | | | | | | | | | |

| | FOOD ITEMS | | | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|---|
| | | SM | WM | | | | | | | |
| PUDDINGS | Apple + Batter | 3345 | 3327 | med serving = 70g | | | | | | |
| | Apple Crumble | | 3334 | med serving = 70g | | | | | | |
| | Baked Pudd + Syrup | 3348 | 3312 | med serving = 30g<br>30 x 65 x 65 = 50g | | | | | | |
| | - No Syrup | 3347 | 3221 | | | | | | | |
| | Blancmange | 3282 | 3281 | SP = 75; ½c = 95g | | | | | | |
| | Egg Type eg. Bread, Sago | 3346 | 3263 | 1T = 50g; ½c = 140g;<br>SP = 100g | | | | | | |
| | Ice Cream: Commercial Regular-3483 | | | scoop = 40g; 1SP = 65g;<br>½c = 75g | | | | | | |
| | Commercial Rich-3519 | | | | | | | | | |
| | Ice Lollies-3982 | | | | | | | | | |
| | Soft Serve-3518 | | | plain = 135g; + flake = 155g | | | | | | |
| | Sorbet-3491 | | | 1SP = 65g; ½c = 75g | | | | | | |
| | Instant Pudding | 3314 | 3266 | T = 45g; SP = 95g; ½c = 145g | | | | | | |
| | Jelly-3983 | | | 1T = 35g; 1SP = 75g;<br>½c = 110g | | | | | | |
| | Jelly + Fruit-4006 | | | 1T = 40g; 1SP = 90g;<br>½c = 125g | | | | | | |
| | Jelly Whip | 2749 | 2750 | 1T = 55g; SP = 95g;<br>½c = 120g | | | | | | |
| | Pancake/Crumpets | 3344 | 3238 | 1 crumpet = 25g<br>pancake = 70g | | | | | | |
| | Trifle-3311; Vermicelli Pudding-3385 | | | ½c = 130g (med) | | | | | | |
| | Other Puddings (Specify) _____ | | | | | | | | | |
| SA UC | Cream: Plant-3492; Canned-3499 | | | 1T = 13g (not whipped) | | | | | | |
| | - Fresh (12%) -3481; Heavy (dessert, 20%)-3480 | | | 1T = 30g (whipped) | | | | | | |
| | Chocolate Sauce-3129 | | | T = 15g | | | | | | |
| | Custard: SM-2717; WM-2716 | | | T = 13g; SP = 40g | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|
| | Sugar-3989 | 1t = 6g | | | | | | | |
| | Other (Specify) _____ | | | | | | | | |
| CAKE | Banana Loaf: WM + HM-3333; SM + PM-3370 | slice = 45g, 90 x 80 x 10mm | | | | | | | |
| | Cake –Carrot-3392 | 80 x 40 x 40 = 50g | | | | | | | |
| | - Plain: SM + HM-3286; PM-3287 | single slice = 50g (75 x 75 x 20mm) double slice = 100g (plain) icing = 10g per slice | | | | | | | |
| | WM + B-3218; HM-3288; SO-3290 | | | | | | | | |
| | Cake Icing: HM-4014; PM-4015 | | | | | | | | |
| | - Chocolate (No Icing) WM-3289; SM-3339 | | | | | | | | |
| | FOOD ITEMS | QUANTITY (g/ml) | BR | | IS | L | IS | D | AD |
| CAKE | - Fruit: Comm-3291; Home-3427 | home: 70 x 85 x 15mm = 70g comm: 90 x 70 x 15mm = 35g | | | | | | | |
| | - Sponge (Plain)-3219 | 100 x 50 x 50 = 40g | | | | | | | |
| | - Swiss Roll-3292 | slice = 60g; 15cm thick | | | | | | | |
| | Cheese Cake: Baked-3293; Unbaked-3294 | slice 95 x 50 x 30mm = 70g | | | | | | | |
| | Other (Specify) _____ | | | | | | | | |
| COOKIES & SPECIAL BREADS | Comm + Fill-3217; Plain-3216; Shortbread-3296 | plain = 10g + fill = 15g | | | | | | | |
| | Home: Plain HM-3233; PM-3341 | plain = 15g + fill = 20g hertzog = 50g; cupcake = 35g shortbread = 12g | | | | | | | |
| | Jam-3295; Oats-3265 | | | | | | | | |
| | Custard Slice-3338 | 110 x 45 x 35mm = 250g | | | | | | | |
| | Date Loaf; HM-3256; PM-3340 | slice 90 x 75 x 10mm = 40g | | | | | | | |
| | Doughnuts: Jam-3423; Plain-3232 | med round = 45g med long = 90g | | | | | | | |
| | Eclairs + Cream + Chocolate-3268 | 1 = 120g (160mm) | | | | | | | |
| | Gingerbread: HM-3253; PM-3371 | 90 x 75 x 15 = 70g | | | | | | | |
| | Koeksister-3231 | 100 x 35 = 60g | | | | | | | |
| | Pumpernickel Bread-3283 | slice 85 x 100 x 10mm = 30g | | | | | | | |
| | Raisin Bread-3214 | slice 85 x 100 x 10mm = 30g | | | | | | | |
| | Rye Bread-3213 | slice 85 x 100 x 10mm = 30g | | | | | | | |
| | Sweetcorn Bread-3379 | slice 85 x 100 x 10mm = 30g | | | | | | | |
| | Other (Specify) _____ | | | | | | | | |
| [illegible] | Apple: HM-3224; PM-3352 | 50 x 50 x 50mm = 70g (med) | | | | | | | |
| | Coconut-3228 | wedge 50 x 100 x 30mm= 55g | | | | | | | |
| | Condensed: HM-3294; PM-3439 | 95 x 70 x 30mm = 90g | | | | | | | |
| | Fridge (Fruit): HM-3394; PM-3434 | | | | | | | | |
| | Lemon Meringue: HM-3226; PM-3349 | 100 x 70 x 35mm = 75g | | | | | | | |
| | Milk (Short) WM + HM-3360; SM + PM-3351 | 120 x 70 x25mm = 75g | | | | | | | |
| | Milk (Flaky) WM + B-3443; WM + HM-3229 | | | | | | | | |

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| | Savoury: Aspar-3367; Tuna-3366; Vienna-3326 | 120 x 50 x 25 = 75g | | | | | | |
| | Tipsy: HM-3323; Jam-3225 | 87 x 70 x 50mm = 90g | | | | | | |
| | Other (Specify) _____ | | | | | | | |

| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|
| SWEETS | Bubble/Chewing gum-3993 | See Manual | | | | | | |
| | Chocolates: Assorted-3992 | | | | | | | |
| | Coated Bars eg. Tex, Lunch, Chomp-3997 | | | | | | | |
| | Milk (White Chocolate)-3987 | | | | | | | |
| | Nuts/Raisins-3994 | | | | | | | |
| | Plain eg Smarties, Flake, Aero-4003 | | | | | | | |
| | Dry Fruit Sweets-3995 | | | | | | | |
| | Fruit Gums-4000 | | | | | | | |
| | Hard/Jelly Sweets eg. Sugus, Jelly Tots, Fruit Drops-3986 | | | | | | | |
| | Ice Lollies-3982 | | | | | | | |
| | Marshmallows-4001 | | | | | | | |
| | Meringues-4008 | | | | | | | |
| | Peanuts: Raw-4285; Peanut Brittle-4002; | | | | | | | |
| | Roasted, Salted-3458; Roasted Unsalted-3452 | | | | | | | |
| | Peppermints-4004 | | | | | | | |
| | Popcorn: Plain-3332; Sugar Coated-3359 | | | | | | | |
| | Potato Crisps eg. Simba, O=Gradys-3417 | | | | | | | |
| | Raisins, Seedless-4232 | | | | | | | |
| | Snacks – Fritos, Niknaks, Cheese Curls-3267 | | | | | | | |
| | Soft Sweets - Fudge, Toffees, Caramel-3991 | | | | | | | |
| | Other (Specify) _____ | | | | | | | |
| OH | Cheese Sauce: WM + HM-3125; SM + PM-3128 | SP = 65g; 1T = 25g | | | | | | |
| | Curry Sauce-3130 | 1T = 25g | | | | | | |
| | Chutney-3168; Atjar-3117; Tomato Chutney-3114 | 1T = 14g; 1T = 60g | | | | | | |
| | Gravy: Comm-3119; Meat-3122; NF-3121 | 1T = 15g; SP = 35g | | | | | | |
| | Mustard-4034 | 1t = 6g | | | | | | |

13

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|---|---|---|---|---|---|---|---|---|
| | Pickles-3866 | 1 = 10g | | | | | | |
| | Tomato Sauce (Comm)-3139 | 1t = 6g; 1T = 25g | | | | | | |
| | White Sauce: WM + HM-3142; SM + PM = 3141 | | | | | | | |

| | FOOD ITEMS | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|
| INFANT FOODS | Baby Cereals (dry): Nestum 1-2832; Nestum 2-2834 | 1t = 2g<br>1T = 8g<br>½c = 20g | | | | | | |
| | Purity:Mixed-2842;Wholewheat-2861; Rice-2862 | | | | | | | |
| | Cerelac-2836; Nestum Rice & Maize-2835 | | | | | | | |
| | Junior-2833 | | | | | | | |
| | Milk: SM-2719; WM-2718 | to drink ½c = 125ml<br>baby bottle = 250ml | | | | | | |
| | BL-2771; 2%-2772 | | | | | | | |
| | Soy-2737; Breast-2741; Goat-2738 | | | | | | | |
| | Formula (Specify):_____<br>No of Scoops/Bottle: _____ | | | | | | | |
| | + Sugar, White-3989;Brown-4005;Syrup-3988;Honey-3984 | 1t = 6g | | | | | | |
| | First Food Fruit-2852; First Food Veg-2851 | jar = 80g, 1t = 11g | | | | | | |
| | Fruit Juice (Strained)-2860; Fruit Juice-2866 | ½c = 125ml | | | | | | |
| | Infant Dinners (Dry): Beef + Veg-2841; Chicken+Veg-2840 | 1t = 5g<br>1T = 15g<br>½c = 47g | | | | | | |
| | Guava + Custard-2837; Mix Veg-2839 | | | | | | | |
| | Orange + Banana-2838 | | | | | | | |
| | Junior Food (Jar): Veg + Meat-2848; Mix Veg-2849; Pasta + Beef-2850 | jar = 200g<br>1t = 11g<br>½c = 125g | | | | | | |
| | Junior Fruit (Jar): Fruit-2863; Guava-2855 | | | | | | | |
| | Junior Pudding: Fruit+Yog-2858; Vanilla Cust-2859 | | | | | | | |
| | Strained Food (Jar): Macaroni Beef-2845; Veg+Meat-2846 | jar = 125g<br>1t = 11g<br>½c = 125g | | | | | | |
| | Fruit + Yog-2857; Fruit-2854; | | | | | | | |
| | Av. Pudding-2844; Meat Soup-2847; | | | | | | | |
| | Veg Soup-2843; Vegetables-2853; | | | | | | | |
| | Junior Fruit Guava-2856 | | | | | | | |
| OH | | | | | | | | |
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|---|---|---|---|
| 9. Did this child go to bed hungry last night? | 1 Yes | 2 No | 3 Don't Know |
| 10. Did this child eat from the same pot as the rest of the family at the main meal yesterday? | 1 Yes | 2 No | 3 Don't Know |
| 11. Did this child eat from the same plate as the siblings, at the main meal yesterday? | 1 Yes | 2 No | 3 Don't Know |

## A. SCHOOL/CRECHé FEEDING SCHEME

3. Name of School/Creché: _____

4. Address: _____

_____

5. Telephone: _____

6. Person to Contact: _____

7. Composition of the Meals/Supplements

| ITEMS | CODE | AMOUNT (g) |
|---|---|---|
| i)<br>ii)<br>iii) | | |

Description:

_____

_____

_____

## B. ADD ADDITIONAL ITEMS EATEN TO THE 24-HR RECALL QUESTIONNAIRE

### RECIPES

| NAME OF DISH | INGREDIENTS | CODES | AMOUNT (g) OR (mg) |
|---|---|---|---|
| | | | |

15

APPENDIX D

ELS Ethics committee clearance letter

**UNIVERSITY OF LIMPOPO**
**Medunsa Campus**

***MEDUNSA RESEARCH & ETHICS COMMITTEE***

***CLEARANCE CERTIFICATE***

| | |
|---|---|
| **MEETING:** | **07/2013** |
| **PROJECT NUMBER:** | **MREC/P/204/2013: IR** |
| **PROJECT:** | |
| **Title:** | Tracking determinants and risk factors of non-communicable diseases among rural South Africa population over time: Ellisras Longitudinal Study |
| **Researcher:** | Dr KD Monyeki |
| **Co-workers:** | Prof L Hay<br>Dr L Scott<br>Prof PS Mntla (Cardiology)<br>Prof Han CG Kemper (Institute for Care and Health Research in Extramural Medicine (EMGO) VU University Medical Centre Amsterdam, The Netherlands<br>Prof A Mogale |
| **Department:** | Human Physiology |
| **School:** | Pathology & Pre-Clinical Sciences |
| **Research Type:** | Independent Research |

UNIVERSITY OF LIMPOPO
Medunsa Campus
2013 -09- 05
MEDUNSA RESEARCH ETHICS COMMITTEE
MREC CHAIRPERSON

**DECISION OF THE COMMITTEE:**

MREC approved the project.

**DATE:** 05 September 2013

**PROF GA OGUNBANJO**
**CHAIRPERSON MREC**

The Medunsa Research Ethics Committee (MREC) for Health Research is registered with the US Department of Health and Human Services as an International Organisation **(IORG0004319)**, as an Institutional Review Board **(IRB00005122)**, and functions under a Federal Wide Assurance **(FWA00009419)**
Expiry date: **11 October 2016**

Note:
i) Should any departure be contemplated from the research procedure as approved, the researcher(s) must re-submit the protocol to the committee.
ii) The budget for the research will be considered separately from the protocol.
PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES.

Finding Solutions for Africa

APPENDIX E

Consent form

# UNIVERSITY OF LIMPOPO

## Department of Physiology and Environmental Health

Faculty of Science and Agriculture

**Private Bag x 1106**
**Sovenga**
**0727**
**SOUTH AFRICA**
**Tel: (015) 268 2953**
**Email: Kotsedi.monyeki@ul.ac.za**
**Website:www.ul.ac.za**

TITLE: **TRACKING DETERMINANTS AND RISK FACTORS OF NON-COMMUNICABLE DISEASES AMONG RURAL SOUTH AFRICAN POPULATION OVER TIME: ELLISRAS LONGITUDINAL STUDY**

**INFORMED CONSENT FORM**

Ellisras Longitudinal Study subjects will be requested to take part. This research is looking at the adolescents' and young adults' lifestyle risk factors and how they may affect the development of non communicable diseases, including obesity, hypertension, diabetes, coronary heart disease. If you agree to participate, you will be asked about what and how much you eat, your physical activity levels, the illnesses you or your family members may have had and your use of healthcare services and the type of support you receive from those around you. We will also ask you some questions about the number of people in your household, their ages, jobs, how much money you make or earn, some of the things that your household members may own and some of the hygiene practices that are followed in your household. We will also ask about your smoking patterns, alcohol intake, your use of drugs and your social and personal characteristics. We will measure your weight, height, arm and leg circumferences, arm and leg breadths, body fat measures along your arms and legs, your blood pressure and heart activity.

After an overnight 10 hour fast (where you do not eat or drink anything except water after your evening meal, the night before the blood sampling, and miss your breakfast the day of the test), we will place a sterile little tube in a vein in your arm and take 15 ml (3 teaspoons) of blood from this. We will give you a cup of sugar water to drink and then we will take two more blood samples (each 2 teaspoons) over the next two hours. Taking the blood sample may cause a little discomfort at the site but there are no risks for this test, other than those associated with routine blood sampling. All procedures will be supervised and carried out by appropriately trained medical personnel who will use techniques to minimise any risks of infection. This test is used routinely for medical purposes. The

blood sample will be used to determine your blood sugar, insulin, cholesterol and other additional factors that may help us learn more about diabetes and cardiovascular diseases risk factors. In addition you will be required to provide 24 hour urine which will help to assess sodium, potassium and other electrolyte in the body. This will provide us with valuable information regarding your hypertension status. You will be required to wear an accelerometer for nine consecutive days which will be used to assess your physical activity.

These measurements and interviews will take place once every two years from 2013 to 2017 (2013, 2015 & 2017).

Questions about what foods you eat and alcohol and drug use, about household or family members, how much money household members make or earn in a month and some of the things that household members may own and your living conditions will be done in your home and will take no more than 120 minutes (2 hours). All the remaining interviews and assessments will take place at a nearby school and will last no more than 180 minutes (3 hours).

**RISKS**

Choosing to be part of this study or not to part of this study will not affect your ability to get care at health if you should need them. You may feel uncomfortable when being measured or when providing all the information asked or when your blood sample is collected; however, the examiners and interviewers will make every effort to make you as comfortable as possible during the process.

**BENEFITS**

There are no benefits to you as a participant; however, your participation will help provide ideas and information about the health status and healthcare needs among adolescents and young adults in this area and will contribute to understanding the health status and health care needs of adolescents and young adults in Africa. You will not be paid for taking part.

**CONFIDENTIALITY-** Any information obtained during this study will remain secret and kept safe private and will only be shared with your permission or as required by law. Participants will be given a number instead of using their names in the study, so they cannot be identified. The hard copies of the data will be safely discarded after data entry and cleaning. The electronic version of the data will be kept under locked storage in the principal investigators office.

**PARTICIPATION-** You can chose to be in this study or not. Your participation is voluntary, and you may get out of the study at any time and for any reason. Being or not being in this study does not cost anything. You can also refuse participate in any parts of the study and still remain in the study. The researcher may remove you from the study if conditions arise and give reason to do so. If you participate in the study, you will receive a small amount of money to help cover some of the travel costs you may have incurred during the research study.

**CONTACT-**This research is being done by Prof. Kotsedi Daniel Monyeki, Physiology Department, University of Limpopo. He may be reached at the local research office and can also be reached at (015) 268 2953 for questions or to report a research-related problem. You may contact Chairman of Ethics Committee Human Ethics Committee) - at (012) 521 4414 if you have questions or comments regarding your rights as a participant in the research.

This research has been reviewed according to University of Limpopo, ethics committee procedures governing your participation in this research.

CONSENT

PART:A:(To be used by adults and fully conscious persons):

I (NAME IN FULL)…….have understood the information I have been given/ I have read.

I, …….. agree to take part in the study.

Signature……..Date ……...

Witness

(NAME)..……..……Signature……

…Date……

**PART:B: (To be used by parents/guardians of children OR guardians, parents or relatives of patients with altered level of consciousness/ altered ability to consent):**

I (NAME IN FULL)…….have understood the information I have been given/ I have read.

I, ……on behalf of …….. agree that he/she will to take part in the study

Reason of failure of (Name)…….. to consent is (TICK):

(i) Child ( )
(ii) Altered consciousness ( )
(iii) Others (specify)……

Signature of parent/ guardian/ relative……..

Mention your relationship with the participant……

Date ……...

Witness (NAME)……..……

Signature……

Date……

**Assent form for the child**: I (NAME IN FULL)…….have understood the information I have been given/ I have read.

I, …….. agree to take part in the study.

Signature……..Date ……...

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APPENDIX F

Ellisras community letter

# UNIVERSITY OF LIMPOPO

## Department of Physiology and Environmental Health

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**SOUTH AFRICA**
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29 October 2015

The Principal/Manager

……

……

Dear Sir/Madam

**ALL ELLISRAS LONGITUDINAL STUDY MEMBERS ARE KINDLY REQUESTED TO TAKE PART DURING THE PERIOD NOVEMBER 2015 TO 5 JANUARY 2016**

Diabetes, high blood pressure and cholesterol problems or high fat levels in the blood seem to be more common in the community than they were 10 or 20 years ago. This is most likely a result of both environmental and genetic factors. Environmental factors are those factors related to your lifestyle such as your diet and your physical activity levels. Genetic factors are those factors you "inherit" from your parents and grandparents. These conditions can lead to further health problems such as problems with eyesight, the heart and strokes, but the chance of having these problems can be lessened by treatment. It is my pleasure to report that the department of Physiology and Environmental Health, University of Limpopo will commence with the Ellisras Longitudinal Study (ELS) shortly. The aim of the ELS will be to track the role of lifestyle risk factors in determining adverse health outcomes. In particular, the development of non-communicable diseases, including obesity, hypertension, diabetes and coronary heart disease in a cohort of rural adolescents of South Africa over time.

All Ellisras Longitudinal Study subjects will be requested to take part. This research is looking at the adolescents' and young adults' lifestyle risk factors and how they may affect the

development of non communicable diseases, including obesity, hypertension, diabetes, coronary heart disease. If you agree to participate, you will be asked about what and how much you eaten the previous day. Anthropometric measurements will include weight, height, sitting height, leg length, skinfolds: (triceps, biceps, subscapular and supraspinale), girth (waist, hip, arm girth flexed and tense, neck, arm girth relax and calf girth), width: (femure and humerus). Your blood pressure and pulse rate will be measured.

Furthermore, after an overnight 10 hour fast (where you do not eat or drink anything except water after your evening meal, the night before) we will place a sterile little tube in a vein in your arm and take 15 mls (3 teaspoons) of blood. Taking the blood sample may cause a little discomfort at the site but there are no risks for this test, other than those associated with routine blood sampling. All procedures will be supervised and carried out by appropriately trained medical personnel from University of Limpopo, Medical Research Council, Vrije University Amsterdam, The Netherland le Sefako Makgatho Health Science University, who will use techniques to minimise any risks of infection. This test is used routinely for medical purposes. The blood sample will be used to determine your blood sugar, insulin, cholesterol and other additional factors that may help us learn more about diabetes and cardiovascular diseases risk factors.

These measurements and interviews will take place during the period November 2015 to 5 January 2016.

Please allow me to refer to PhD thesis of Monyeki (2000):

"…Bahlalerwa (cultural name of our population) I requested for your help and you responded positively. I am happy because together we could make a difference. I am appealing to all of us to support and avail ourselves to any form of research activities taking place in our area. Such activities are geared towards improving the health not only of the Bahlalerwa population but the whole South Africa if not Africa. We should keep focus and if somehow we could be blinded, with the help of the Almighty God the father of Jesus Christ, crystals like glass will fall from our eyes like what happen to Paul in the Holy Bible. Our vision will be broaden and we will no longer think and perform our duties inside the box…."

Past my regards to everybody at home.

Yours sincerely

Prof Kotsedi Daniel Monyeki
**Principal Investigator: Ellisras Longitudinal Study**

Dr Marlise van Staden
**Head of Department**

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PEER REVIEWED ARTICLES EMANATING FROM THE DISSERTATION

1. **Sekgala MD,** Monyeki KD, Mogale MA, Ramoshaba NE. Performance of blood pressure to height ratio as a screening tool for elevated blood pressure in rural children: Ellisras Longitudinal Study. *Journal of Human Hypertension*. 2017 April. 06.

2. **M.D Sekgala,** K.D Monyeki1, A. Mogale, Z.J Mchiza, W. Parker, S.R Choma, H.M Makgopa. The risk of metabolic syndrome as a result of lifestyle among Ellisras rural young adults. *Journal of Human Hypertension*, 2018.